



Journal of the ASEAN Federation of Endocrine Societies

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MEMS ANNUAL CONGRESS

MAC15

2025

30 May - 1 June 2025

Hilton and Le Méridien Kuala Lumpur

MIND THE GAP

Translating Knowledge Into Practice



Malaysian Endocrine
& Metabolic Society



www.memsmac.org

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**Malaysian Endocrine
& Metabolic Society**



Welcome Message from MAC 15 Chairperson / Organising Committee

Dear Friends,

2025 marks the crystal anniversary of the Malaysian Endocrine and Metabolic Society's Annual Congress (MAC). Over the years MAC has bloomed to become one of the most highly anticipated national congresses not only for the adult and paediatric endocrinologist, but also for the generalist who deals with the many routine endocrine problems.

The field of endocrinology is moving at an extremely rapid pace and it is easy to fall through the many "gaps". Hence, MAC 2025 is themed "**Mind The Gap: Translating Knowledge Into Practice**" and aims to address the many "unaddressed" questions in endocrinology and equip you with the confidence to manoeuvre through your practice, backed up by sound scientific evidence.

MAC 15 which is planned for the weekend of **30th May-1st June 2025 at the Hilton and Le Meridien Kuala Lumpur**, will be a star-studded affair. This year's congress will witness the largest number of international speakers ever, with more than a dozen of them from various regions, who are really the "who's who" in their respective fields. They will be joined by our local esteemed speakers to deliver new insights via plenaries, symposiums and meet the expert sessions. The pre-congress workshop for 2025 will be held on the 29th of May 2025 at the same venue and will engage a full day multi-disciplinary discourse on the advances in the management of thyroid cancers.

We hope that 2025 will bring you the **BIGGEST MAC EVER** not only in terms of impactful scientific content, but also giving lot of fun opportunities to connect with old and new friends, win prizes for best abstracts presented, to forge local and international collaborations and for the industry to showcase the best of their best.

We also warmly welcome our friends from outside of Malaysia so you will also have a chance to enjoy the colourful city, sunshine and culinary delights of Kuala Lumpur while you are here for the congress. I am superbly excited and look forward to welcoming all of you at the **BIGGEST MAC** ever!

Yours sincerely

Assoc Prof Dr Jeyakantha Ratnasingam
Organising Chairperson
15th MEMS Annual Congress (MAC 15)



MAC 15 Organising Committee

MEMS President & MAC Advisor	: Dr Nurain Mohd Noor
Scientific Advisor	: Prof Emeritus Dr Chan Siew Pheng
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Scientific Co-Chair (Paediatrics)	: Prof Dr Muhammad Yazid Jalaludin
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	: Prof Dr Lim Lee Ling
	: Dr Luqman Ibrahim
	: Dr Sharmila Sunita Paramasivam
	: Dr See Chee Keong
	: Dr Tong Chin Voon
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Programme

Day 1 - 30 May 2025 (Friday)

BALLROOM B + BALLROOM C, HILTON KL			
0730 - 0830	Registration		
0830 - 0840	Welcome Remarks <i>Jeyakantha Ratnasingam, Organising Chair of MAC 15</i> Opening Speech <i>Nurain Mohd Noor, President of MEMS</i>		
0840 - 0925	PLENARY 1: Primary Aldosteronism: Lessons from Clinics, Cohorts & Consumers <i>Jun Yang (Australia)</i> <i>Chairperson: Norlela Sukor</i>		
0925 - 1010	PLENARY 2: Growth Hormone Use from Womb to Tomb: Are We Ready for Primetime? <i>Gudmundur Johannsson (Sweden)</i> <i>Chairperson: Hew Fen Lee</i>		
1010 - 1030	Tea Break / Trade Exhibition + <i>Tour d'MAC</i> + <i>Tour d'Sentral</i>		
	BALLROOM B, HILTON KL	BALLROOM C, HILTON KL	CLARKE BALLROOM, LE MERIDIEN KL
1030 - 1200	Symposium 1: PITUITARY <i>Chairpersons:</i> <i>Wan Juani Wan Seman</i> <i>See Chee Keong</i> Emerging Therapies for Acromegaly: New Solutions for a Giant Problem <i>Kevin Yuen (USA)</i> Radiation for Pituitary Tumours: When & Whom? <i>Muthukkumaran Thiagarajan</i> Controlling the Incurable: Updates on Medical Management of Cushing's <i>Ann McCormack (Australia)</i> <i>Supported by IPSEN Pharma</i>	Symposium 2: OBESITY <i>MEMS & MyOS Joint Symposium</i> <i>Chairperson: Rohana Abdul Ghani</i> Reframing Obesity as a Chronic, Relapsing Neurobehavioral Disease <i>Rohana Abdul Ghani</i> <i>Launch of Whitepaper on Obesity Care</i> Clinical Psychologist as a "Devil's Advocate" in Bariatric Surgery: Pre-Surgical Psychological Assessment <i>Rahmattullah Khan</i> Evidence-Based Dietary Approaches: Low Carb, Mediterranean, & Intermittent Fasting <i>Barakatun Nisak Mohd Yusof</i> Personalized Medicine in Obesity Care: Optimizing Pharmacotherapy <i>Tham Kwang Wei (Singapore)</i>	PAEDIATRICS Symposium 3: GROWTH CONCERNS <i>Chairperson: Cheng Hooi Peng</i> Short Teens, Short Adult. Consequences and Impact on Health and Society <i>Nurshadia Samingan</i> Recognising Short Stature Amongst Children, Teens & Young Adults <i>Lim Song Hai</i> Bone Health and the Risk of Early Osteoporosis in Young Adults <i>Noriyuki Namba (Japan)</i>
1200 - 1240	MTE 1: Making Sense of Nuclear Smudges <i>Nor Salita Ali</i> <i>Chairperson: Anuar Zaini Md Zain</i>	MTE 2: Diabetic Peripheral Neuropathy: Is There Any Escape? <i>Solomon Tesfaye (UK)</i> <i>Chairperson: Vijiya Mala Valayatham</i>	MTE 3: Multidisciplinary Approach to the Management of Turner Syndrome <i>Lee Yee Lin</i> <i>Chairperson: Mazidah Noordin</i>

Programme

Day 1 - 30 May 2025 (Friday)

1240 - 1340	Zuellig Pharma Lunch Symposium Hitting Right, Tipping Right with the Right Diabetes Treatment: A Case-Based Exploration <i>Hew Fen Lee</i> <i>Lim Lee Ling</i> <i>Chairperson: See Chee Keong</i> 	Novo Nordisk Pharma Lunch Symposium It's Worth Starting Early: Act Now with Obesity Obesity-CKM Nexus: Is it Worth Starting Early? <i>Tham Kwang Wei (Singapore)</i> Beyond Weight Loss for Health Gains: The Weigh to Go! <i>Jeyakantha Ratnasingam</i> Bench to Bedside: Let's Talk REAL <i>Tham Kwang Wei (Singapore)</i> <i>Jeyakantha Ratnasingam</i> <i>Chairperson: Chan Siew Pheng</i> 	Servier Malaysia Lunch Symposium Young Onset T2D: Can SU Be the Rescue? <i>Viswanathan Mohan (India)</i> <i>Chow Yok Wai</i> <i>Chairperson: Alexander Tan Tong Boon</i> 
1340 - 1400	Trade Exhibition + <i>Tour d'MAC</i> + <i>Tour d'Sentral</i>		
1400 - 1440	MTE 4: Achieving Fertility in Secondary Hypogonadism <i>Richard Quinton (UK)</i> <i>Chairperson: Jeshen Lau</i>	MTE 5: Dealing with Thyroid Diseases in Pregnancy <i>Samantha Yang (Singapore)</i> <i>Chairperson: Florence Tan</i>	MTE 6: Management of Thyrotoxicosis & Beyond: RAI vs Surgery <i>Nalini Selveindran</i> <i>Chairperson: Noor Arliena Mat Amin</i>
1440 - 1610	Symposium 4: ADRENAL <i>Chairpersons:</i> <i>Tong Chin Voon</i> <i>Subashini Rajoo</i> Primary Aldosteronism: Outcomes of Medical Therapy <i>Jun Yang (Australia)</i> Glucocorticoid-induced Adrenal Insufficiency: Updates from the 2024 Guidelines <i>Gudmundur Johannsson (Sweden)</i> Approach to Bilateral Adrenal Masses <i>Norlela Sukor</i>	Symposium 5: THYROID <i>Chairpersons:</i> <i>Serena Khoo</i> <i>Norhaliza Mohd Ali</i> Subclinical Thyroid Disease: What's New in 2025 <i>Amir S Khir</i> Graves' Ophthalmopathy: Prevention, Monitoring & New Therapies <i>Tai Lai Yong</i> Managing Graves' Across the Lifespan <i>Mafauzy Mohamed</i>	Symposium 6: MULTIENDOCRINE CONCERNS <i>Chairperson: Ting Tzer Hwu</i> Identifying Precocious Puberty: Timely Diagnosis and Early Intervention <i>Wong Sze Lyn Jeanne</i> New Treatments for Congenital Adrenal Hyperplasia (CAH): Advancements in Care and Management <i>Suhaimi Hussain</i> Neonatal Thyroid Disorders, the Hypo's and the Hyper's <i>Joyce Hong</i>

Programme

Day 2 - 31 May 2025 (Saturday)

	BALLROOM B, HILTON KL	CLARKE BALLROOM, LE MERIDIEN KL	
0750 - 0830	Novartis Corporation Breakfast Symposium  NOVARTIS Optimising Strategies in Achieving LDL Goals in Patients at Cardiovascular Risk <i>Loh Wann Jia (Singapore)</i> <i>Chairperson: Lim Lee Ling</i>	Viartis Breakfast Symposium  VIATRIS Optimal Management of Painful Diabetic Peripheral Neuropathy and the Diabetic Foot <i>Solomon Tesfaye (UK)</i> <i>Chairperson: Jeyakantha Ratnasingam</i>	
	BALLROOM B + BALLROOM C, HILTON KL (will be live broadcast to Clarke Ballroom)		
0830 - 0915	PLENARY 3: Use & Abuse of Hormones for Brawn, Beauty & Wellness <i>Kevin Yuen (USA)</i> <i>Chairperson: Jeyakantha Ratnasingam</i>		
0915 - 1000	Crossroads in Endocrinology: What Should Be First Priority in T2D: Glycemia, Weight or Organ Protection? <i>Thomas Pieber (Austria), Tham Kwang Wei (Singapore), Alexander Tan Tong Boon</i> <i>Moderator: Chan Siew Pheng</i>		
1000 - 1020	Tea Break / Trade Exhibition + <i>Tour d'MAC</i> + <i>Tour d'Sentral Draw</i>		
	BALLROOM B, HILTON KL	BALLROOM C, HILTON KL	CLARKE BALLROOM, LE MERIDIEN KL
1020 - 1150	Symposium 7: SALT & WATER <i>Chairpersons:</i> <i>Mohd Badrulnizam Long Bidin</i> <i>Fatimah Zaherah Mohamed Shah</i> Moving Away from Water Deprivation: Role of Co-Peptin in AVP-D <i>Gudmundur Johannsson (Sweden)</i> SIADH: When Fluid Restriction Fails <i>Joseph Verbalis (USA)</i> Long Term Desmopressin Replacement: Tips & Tricks <i>Ann McCormack (Australia)</i> <i>Supported by Otsuka Pharmaceutical & Ferring Pharmaceutical</i>	Symposium 8: DIABETES-THINKING OUT OF THE BOX <i>Chairpersons:</i> <i>Swarupini Ponnampalam</i> <i>Wan Mohamad Wan Bebakar</i> T1D and Ramadhan – Is Technology a Game-Changer? <i>Khadija Hafidh (UAE)</i> Uncontrolled Diabetes: Could It Be Hypercortisolism <i>Bee Yong Mong (Singapore)</i> Diabetes Heterogeneity: Are Asians Different? <i>Alice Kong (Hong Kong, ROC)</i>	PAEDIATRICS
			Symposium 9: DIABETES <i>Chairperson: Ho Sheau Chui</i> T1D: Automated Insulin Delivery Systems; Is It Time to Move Forward? <i>Luqman Ibrahim</i> T2D Pharmacotherapy: What's in Store for Children? <i>Noor Shafina Mohd Nor</i> Atypical Diabetes in Children: Are We Misdiagnosing Them? <i>Meenal Mavinkurve</i>
1150 - 1230	MTE 7: 10 Plus % of Pheochromocytomas: Challenging Cases <i>Zanariah Hussein</i> <i>Chairperson: Yong Sy Liang</i>	MTE 8: Fifty & Fabulous: Managing Menopause <i>Imelda Balchin</i> <i>Chairperson: Lim Siang Chin</i>	MTE 9: Update of National DKA Incidence & Management <i>Wu Loo Ling</i> <i>Chairperson: Ch'ng Tong Wooi</i>



Programme

Day 2 - 31 May 2025 (Saturday)

1230 - 1330	Zuellig Pharma Lunch Symposium Talk of the Town: The Time for GIP is NOW <i>Khadija Hafidh (UAE)</i> <i>Sarah Glastras (Australia)</i> <i>Lim Shueh Lin (Australia)</i> <i>Jeyakantha Ratnasingam</i> <i>Chairperson: Chan Siew Pheng</i> 	Novo Nordisk Pharma Lunch Symposium Groundbreaking New Data with Semaglutide: Beyond HbA1c and Weight Loss! Heart of the Matter in Diabetes Management <i>Thomas Pieber (Austria)</i> From Global Advances to Local Impact: The Malaysian Perspective <i>Rohana Abdul Ghani</i> <i>Chairperson: Lim Lee Ling</i> 	Abbott Diabetes Care Lunch Symposium Revolutionising Diabetes Care: Effective, Safe & Easy The Future of Continuous Glucose Monitoring (CGM) is Here <i>Foo Siew Hui</i> Are All Continuous Glucose Monitoring (CGM) Systems Made Equal? <i>Alice Kong (Hong Kong, ROC)</i> 
1330 - 1400	Trade Exhibition + Tour d'MAC		
1400 - 1440	MTE 10: To AVS or Not to AVS: Updates on PA Diagnostics <i>Jun Yang (Australia)</i> <i>Chairperson: Norasyikin Abd Wahab</i>	MTE 11: Oxytocin: The Forgotten Sibling from the Posterior Pituitary <i>Joseph Verbalis (USA)</i> <i>Chairperson: Shanty Velaiutham</i>	MTE 12: Sharing Cases on Calcium and Vitamin D Disorders <i>Noriyuki Namba (Japan)</i> <i>Chairperson: Noor Shafina Mohd Nor</i>
1440 - 1610	Symposium 10: CONTEMPORARY DEBATES IN ENDOCRINOLOGY Surgery as Desired First Line Treatment for Prolactinomas: Yes vs No <i>Kevin Yuen (USA) vs Ann McCormack (Australia)</i> <i>Moderator: Azraai Bahari Nasruddin</i> Continuing Metformin for Women with Obesity / PCOS in Pregnancy: Yes vs No <i>Norlaila Mustafa vs Nurain Mohd Noor</i> <i>Moderators: Shireene Vethakkan Sarah Glastras (Australia)</i>	Symposium 11: WOMEN'S HEALTH <i>Chairpersons: Noor Lita Adam Ijaz Hallaj Rahmatullah</i> PCOS: Old Disease, New Façade <i>Wong Ming</i> PCOS & Fertility: A Gynaecologist's Perspective <i>Mukhri Hamdan</i> Deep Dive into Female Hypogonadism <i>Richard Quinton (UK)</i>	Symposium 12: OBESITY <i>Chairperson: Nurshadia Samingan</i> Overview on Childhood Obesity: Common & Not-So-Common Causes <i>Rashdan Zaki Mohamed</i> Pharmacotherapy for Childhood Obesity: What's New? <i>Azriyanti Anuar Zaini</i> Bariatric Surgery for Adolescents: Is It Ready for Primetime? <i>Reynu Rajan</i>

Programme

Day 2 - 31 May 2025 (Saturday)

1610 - 1700	Bayer High-Tea Symposium Finerenone, The New Kid On the Block: A Pillar Therapy in CKD with T2D <i>Bee Yong Mong (Singapore)</i> <i>Alexander Tan Tong Boon</i> <i>Chairperson: Jeyakantha Ratnasingam</i> 	AstraZeneca High-Tea Symposium The Art of Balancing OAD & Injectables in Cardio Kidney Metabolic (CKM) <i>Lim Lee Ling</i> Primetime to Fight CKD <i>Chong Yip Boon</i> Lipids: The Missing Link in Diabetes Management <i>Azani Daud</i> <i>Chairperson: Chan Siew Pheng</i> 	Young Investigator Award Presentation (Adult Track) <i>Clarke Ballroom</i>
			Young Investigator Award Presentation (Paediatric Track) <i>Accord Room</i>
1700 - 1800	Trade Exhibition + <i>Tour d'MAC</i>		

* 1700 - 1900 MEMS Annual General Meeting (For MEMS members only) in Clarke Ballroom

Programme

Day 3 - 1 June 2025 (Sunday)

	BALLROOM B, HILTON KL		CLARKE BALLROOM, LE MERIDIEN KL	
0750 - 0830	<div><div><div>Taisho Pharmaceutical Breakfast Symposium</div><div></div><div>Clinical Insights on the Benefits of an SGLT2i in Treating Diabetes <i>Chan Siew Pheng</i></div><div>Chairperson: Alexander Tan Tong Boon</div></div></div>		<div><div><div>Amgen Biopharmaceuticals Breakfast Symposium</div><div></div><div>Tailored Approaches in Osteoporosis: Managing High and Very High-Risk Osteoporosis with Targeted Therapies <i>Manju Chandran (Singapore)</i></div><div>Chairperson: Jeyakantha Ratnasingam</div></div></div>	
	BALLROOM B, HILTON KL	BALLROOM C, HILTON KL	CLARKE BALLROOM, LE MERIDIEN KL	
0830 - 1000	<div><div>Symposium 13: BONE Chairpersons: Sharmila Paramasivam Loh Huai Heng</div><div>Goal Directed Therapy for Osteoporosis <i>Manju Chandran (Singapore)</i></div><div>Fracturing Heavyweights: Obesity & Fragility <i>Foo Siew Hui</i></div><div>Osteoporosis: Men Want Attention Too <i>Hew Fen Lee</i></div></div>	<div><div>Symposium 14: CV & DIABETES Chairpersons: Lim Lee Ling Yusniza Yusoff</div><div>Heart Failure & Diabetes: Should We Even Bother Screening in the Age of SGLT-2 Inhibitors? <i>Azmee Mohd Ghazi</i></div><div>BP & Diabetes: Dash, SPRINT or Marathon? <i>Abdul Rashid Abdul Rahman</i></div><div>New Kids on the Lipid Block: New Targets & Therapies <i>Loh Wann Jia (Singapore)</i></div></div>	<div><div>PAEDIATRICS Symposium 15: ADOLESCENT ENDOCRINOLOGY Chairperson: Raja Aimee Raja Abdullah</div><div>Primary and Secondary Amenorrhoea: Is it PCOS? <i>Nur Azurah Abdul Ghani</i></div><div>Transitioning Care for Growth Hormone Disorders <i>Janet Hong</i></div><div>Modelling Transitioning Care for Diabetes Service- Local Experience <i>Azriyanti Anuar Zaini</i> <i>Lim Quan Hziung</i></div></div>	
	1000 - 1020	Tea Break		
		BALLROOM B + BALLROOM C, HILTON KL		
	1020 - 1030	Tour d'MAC Draw		
1030 - 1115	<div><div>PLENARY 4: Male Hypogonadism from Assessment to Management: Men Deserve Attention Too <i>Richard Quinton (UK)</i> Chairperson: Saiful Kassim</div></div>			
1115 - 1200	<div><div><div>Abbott Nutrition / Abbott Pharma Symposium</div><div></div><div>Keep An Eye, Trust Your Gut</div><div>A New Lens on Diabetic Retinopathy Management <i>Chan Siew Pheng</i></div><div>Diabetic Dyspepsia: A Prelude to Gastroparesis <i>Chuah Kee Huat</i></div><div>Chairperson: Foo Siew Hui</div></div></div>			
1200 - 1245	<div><div>Debate: Intensifying T2D Control: CGM-Guided Lifestyle Intervention versus Upscaling Pharmacotherapy <i>Alice Kong (Hong Kong, ROC) vs Chan Siew Pheng</i> Moderator: Luqman Ibrahim</div></div>			
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Adult Oral Presentation

OP_A001

LONGITUDINAL CHANGES IN SKELETAL MUSCLE MASS AND PHASE ANGLE FOLLOWING METABOLIC BARIATRIC SURGERY: A 12-MONTH FOLLOW-UP

<https://doi.org/10.15605/jafes.040.S1.001>

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INTRODUCTION

Metabolic bariatric surgery (MBS) is an effective intervention for weight loss, but muscle retention remains a concern, as excessive muscle loss can impact metabolic health and physical function. Phase angle (PhA), derived from bioelectrical impedance analysis (BIA), is a marker of cellular integrity and quality. While previous studies have explored muscle loss post-surgery, the longitudinal trends of PhA alongside muscle retention remain underexplored. This study aims to describe trends in skeletal muscle mass (SMM) retention and PhA change over time in post MBS patients.

METHODOLOGY

This is a multicentre intervention study involving patients with obesity undergoing metabolic surgery in private centres around Klang Valley. Those with a body mass index (BMI) of ≥ 25 kg/m² included in this study. Body composition components including body fat mass (BFM), SMM and PhA were measured using a BIA (InBody S10). Statistical analysis was conducted using SPSS software version 29.

RESULT

This study involved 120 patients, with most being female (n=75, 62.5%) and Malays (n = 80, 66.7%). The overall mean age was 39.10 (SD 7.8) years. Mean preoperative values for BMI, SMM and PhA were 41.17 ± 9.40 kg/m², 32.4 ± 8.3 kg and 6.04 ± 1.0 respectively. Skeletal muscle mass significantly declined at 6 months (-5.61 kg, $p < 0.001$) and remained lower at 12 months (-6.13 kg, $p < 0.001$) compared to baseline, with minimal improvement between 6 months and 12 months ($p = 0.268$). Similarly, PhA decreased significantly at 6 months (-1.18 , $p < 0.001$), but despite a slight increase by 12 months, the change was not statistically significant ($p = 1.00$).

CONCLUSION

Significant decline in both SMM and PhA occur within the first 6 months post-surgery, with minimal recovery observed at 12 months. This suggests that early postoperative phase is critical for muscle retention and cellular health. While nutritional and physical activity interventions have been shown to support muscle preservation in previous studies, further research is needed to confirm their effect in post-MBS recovery. These findings highlight the importance of targeted approaches to prevent long-term muscle deterioration.

Adult Oral Presentation

OP_A002

CLINICAL OUTCOMES AND CHALLENGES IN PROLACTINOMA MANAGEMENT: A RETROSPECTIVE STUDY FROM PUTRAJAYA HOSPITAL

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INTRODUCTION

Prolactinomas showed significant variability in treatment response, with an overall display of dopamine agonist (DA) responsiveness in our cohort. This study evaluates the clinical and tumor characteristics separating DA-responsive and resistant patients in a Malaysian tertiary endocrine center.

METHODOLOGY

A retrospective analysis was done on 74 patients with prolactinoma treated between 2015 and 2025. Inclusion criteria encompassed hyperprolactinemia and MRI-documented adenomas. Clinical results were analyzed using established criteria: DA responsiveness, DA resistance, surgical remission, and long-term cure.

RESULT

Baseline characteristics revealed substantial gender differences, with male patients presenting at an older age (51.4 ± 15.0 vs 34.5 ± 9.6 years, $p < 0.001$), higher baseline prolactin levels ($72,274.9 \pm 100,505.4$ vs $7,105.9 \pm 9,152.7$ mIU/L, $p = 0.005$), and larger tumor size (25.1 ± 16.8 vs 11.5 ± 8.5 mm, $p = 0.002$). Male patients generally presented with more aggressive disease features and greater tumor invasiveness, including increased cavernous sinus invasion (57.1% vs 22.6%, $p = 0.0098$) and optic chiasm compression (52.4% vs 7.5%, $p = < 0.001$). When comparing treatment response, DA-resistant tumors revealed clinically relevant patterns, including higher baseline prolactin levels (median 9,642 vs. 4,111 mIU/L, $p = 0.090$) and more frequent cavernous sinus invasion (60% vs. 31.8%, $p = 0.207$). Dopamine agonist resistance rates were similar between genders. The sample demonstrated 89.2% total DA response, with comparable long-term cure rates (31.1% overall), albeit male patients required much higher doses of hormonal replacement therapy (57.1% vs. 3.8%, $p < 0.001$).

CONCLUSION

Our findings demonstrate that prolactinomas in males typically present later in life, are more aggressive and larger, and have a higher prolactin level, but the overall DA response remains good. Despite increased baseline prolactin and invasiveness in males, DA responsiveness was similar between genders. The long-term outcomes were positive, albeit more males required hormone replacement. These findings demonstrate that DA remains as effective first-line therapy for prolactinomas.

Adult Oral Presentation

OP_A003

PREVALENCE, ASSOCIATED FACTORS AND ENDOTHELIAL DYSFUNCTION OF LATENT AUTOIMMUNE DIABETES IN YOUTH AMONG PATIENTS DIAGNOSED WITH TYPE 2 DIABETES MELLITUS

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INTRODUCTION

Latent autoimmune diabetes in youth (LADY) is a slowly progressive form of autoimmune diabetes often misclassified as type 2 diabetes mellitus (T2DM). This study aimed to determine the prevalence of LADY among patients with young-onset T2DM, evaluate clinical characteristics, and assess endothelial dysfunction as a marker of vascular risk in a Malaysian cohort.

METHODOLOGY

A cross-sectional study was conducted at UiTM Sungai Buloh and Hospital Al-Sultan Abdullah, involving 125 patients aged 18-60 years diagnosed with T2DM before the age of 40. Participants were screened for anti-GAD antibodies to identify LADY cases. Clinical, biochemical, and anthropometric parameters were assessed, including c-peptide levels. Endothelial dysfunction was evaluated using flow-mediated dilation (FMD) and nitroglycerin-mediated dilation (NMD). Logistic regression was performed to identify factors associated with LADY and endothelial dysfunction.

RESULT

Of the 125 participants, 11 (8.8%) were diagnosed with LADY based on positive anti-GAD antibodies. Patients with LADY had a significantly longer diabetes duration (18 vs. 11 years, $p = 0.039$) and were predominantly female (82%). They exhibited lower c-peptide levels and required insulin more frequently (91% vs. 61% in T2DM). While LADY had lower median FMD (3.83% vs. 6.82% in T2DM), the difference was not statistically significant ($p = 0.147$). Logistic regression identified increasing age as significantly associated with endothelial dysfunction ($p = 0.016$), while LADY status showed a trend toward significance ($p = 0.071$).

CONCLUSION

Early identification and screening for LADY are essential to prevent delayed diagnosis, optimize metabolic management, and reduce the risk of endothelial dysfunction. Targeted interventions may optimize glycaemic control, mitigate vascular complications and improve long-term outcomes in this high-risk population.

Adult Oral Presentation

OP_A004

MILD AUTONOMOUS CORTISOL SECRETION IN ADRENAL INCIDENTALOMAS: CLINICAL PREDICTORS AND OUTCOMES IN A MALAYSIAN COHORT

<https://doi.org/10.15605/jafes.040.S1.004>

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INTRODUCTION

Mild autonomous cortisol secretion (MACS) represents a functional spectrum of adrenal incidentalomas (AIs) that often go underdiagnosed due to the subtle biochemical profile and variable clinical impact. While extensively studied in Western populations, data on MACS in Asian cohorts, particularly from Southeast Asia, are scarce. This study aimed to evaluate the clinical, metabolic, and radiological characteristics of patients with MACS compared to non-MACS adrenal incidentalomas, and to assess the natural history and outcomes of conservative versus surgical management.

METHODOLOGY

We conducted a retrospective, multi-center analysis of 251 patients with non-malignant AIs across three Malaysian tertiary hospitals. Patients were classified based on hormonal evaluation into MACS and non-MACS groups. Baseline demographic, clinical, metabolic, and imaging features were analyzed. Logistic regression was used to identify independent predictors of MACS. In a subset of patients with MACS, outcomes of surgical versus conservative treatment were compared over a median follow-up period of 50 months.

RESULT

Mild autonomous cortisol secretion was identified in 12.7% (n = 32) of patients. Compared to non-MACS counterparts, patients with MACS had significantly higher prevalence of diabetes mellitus, dyslipidemia, obesity, and osteoporosis, and were more likely to have bilateral adrenal lesions. On multivariate analysis, only osteoporosis (OR 4.2; $p = 0.001$) and bilateral adrenal involvement (OR 4.2; $p = 0.003$) remained independently associated with MACS. Adrenalectomy in patients with MACS resulted in significant improvement in glycemic control and reduced antihypertensive use. Conversely, those managed conservatively demonstrated tumor growth and increased antihypertensive requirement over time.

CONCLUSION

Mild autonomous cortisol secretion is a clinically significant entity associated with increased metabolic morbidity. Osteoporosis and bilateral adrenal lesions are key predictors of MACS and warrant targeted screening. Surgical intervention may confer metabolic benefits, reinforcing the importance of individualized management strategies in patients with AIs.

Adult Best Poster Presentation

BP_A001

CLINICAL AND DEMOGRAPHIC PROFILE OF MEN 1 AND MEN 2A: A 10 YEARS' EXPERIENCE

<https://doi.org/10.15605/jafes.040.S1.005>

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INTRODUCTION

Multiple endocrine neoplasia (MEN) syndromes are rare genetic disorders causing tumours in endocrine glands, with MEN 1 primarily affecting the parathyroid, pancreas, and pituitary, while MEN 2A is characterized by parathyroid tumour, medullary thyroid carcinoma (MTC) and pheochromocytoma.

METHODOLOGY

A retrospective cross-sectional study was conducted at Institut Endokrin Hospital Putrajaya. Electronic medical records of patients who attended Endocrinology or combined surgical clinic between 1st January 2015 till 31st March 2025 were reviewed. Descriptive and statistical analyses for MEN 1 and MEN 2A patients were performed using SPSS version 25.

RESULT

The cohort comprised 16 patients with MEN syndromes—10 with MEN 1 and 6 with MEN 2A. There was a balanced gender distribution (56% male, 44% female), with an age range of 19 to 70 years (mean: 48 years). Patients with MEN 1 were slightly older (mean age: 49 years) compared to those with MEN 2A (mean age: 44 years).

In the MEN 1 population, all had parathyroid involvement (100%), with 60% exhibiting pancreatic tumors and 30% adrenal or pituitary lesions. Most underwent parathyroid (80%) and pancreatic (60%) surgeries. Only 20% had family members screened for MEN genes, suggesting that family screening rate is still low.

In the MEN 2A population, all cases featured medullary thyroid carcinoma (MTC) and RET oncogene mutations, with 83% found to have parathyroid disease and 50% with pheochromocytoma. Thyroid surgery was done for all patients while 83% underwent parathyroidectomy as well. Half had access to family genetic screening, emphasizing the hereditary nature of MEN 2A.

CONCLUSION

Distinct profiles emerged from our cohort. Multiple endocrine neoplasia 1 is marked by parathyroid-pancreatic axis tumors, whereas MEN 2A is defined by MTC and RET mutations. A multidisciplinary approach, including genetic screening and tailored surgery, is critical for optimal outcomes. Genetic screening access for patient and family members can be improved to close the critical gaps in cascade testing for at-risk relatives.

Adult Best Poster Presentation

BP_A002

ARTIFICIAL INTELLIGENCE ECHOCARDIOGRAPHY BY NON-CARDIOLOGISTS FOR EARLY DETECTION OF HEART FAILURE IN TYPE 2 DIABETES

<https://doi.org/10.15605/jafes.040.S1.006>

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INTRODUCTION

Type 2 Diabetes (T2D) is a well-known risk factor for the development of heart failure. We aim to demonstrate the use of artificial intelligence (AI) echocardiography in the hands of non-cardiology-trained doctors as a useful tool for diagnosing Stage B heart failure in patients with diabetes.

METHODOLOGY

Participants with existing T2D but no known heart failure diagnosis or symptoms of heart failure were recruited. All participants had AI echocardiography (Kosmos portable ultrasound, with image analysis by Us2.AI software) done by non-cardiology-trained doctors, and were tested for NTproBNP. Cut-offs for diagnosis of heart stress or heart failure (HF) were based on 2023 ESC consensus.

RESULT

Among 36 patients (median HbA1c 7.4%; mean BMI 27.3 ± 6.0 kg/m²), 52.8% were on SGLT2i and RASi respectively, 36.1% were on beta blockers and 5.6% were on steroidal MRAs. Seven participants (19.4%) were assessed to have heart failure by AI echocardiography (3 HFpEF, 3 HFmrEF and 1 HFrEF). Out of the 7 cases, only 3 of them had raised NTproBNP. The most common echocardiography abnormality was increased relative wall thickness (RWT, 84%), followed by LV diastolic dysfunction (40.9%), LV concentric remodelling (30.4%) and a high E/e' ratio (12.5%).

CONCLUSION

We have demonstrated that AI echocardiography in conjunction with the biomarker NTproBNP can detect Stage B heart failure among patients with T2D. As most patients are not screened for heart failure until symptoms develop, increasing the availability of this technology may facilitate earlier diagnosis of heart failure and initiation of guideline-directed medical therapy.

Adult Best Poster Presentation

BP_A003

CASE SERIES OF PARATHYROID CARCINOMA: A SINGLE CENTER EXPERIENCE

<https://doi.org/10.15605/jafes.040.S1.007>

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INTRODUCTION

Parathyroid carcinoma is a rare but aggressive malignancy of the parathyroid gland, often presenting with severe hyperparathyroidism and associated complications such as hypercalcemia and nephrolithiasis. This study aims to describe the clinical presentation, biochemical profile, imaging findings and treatment modalities and outcomes of patients with parathyroid carcinoma.

METHODOLOGY

A retrospective review of patients diagnosed with parathyroid carcinoma (PC) was conducted. Data on demographic characteristics, clinical presentation, biochemical markers, preoperative imaging findings, treatment and postoperative follow-up were collected.

RESULT

A total of ten cases were identified, majority were female (70.0%) with a median age of 54 years (range: 45.1-60.9 years). Majority presented with symptomatic hypercalcemia (70.0%). One-third exhibited neck swelling, renal stones, and nephrocalcinosis. Biochemical analysis revealed markedly elevated calcium levels (median: 3.7 mmol/L, range: 3.28-3.78), hypophosphatemia (0.59 mmol/L, range: 0.503-0.698), significantly elevated intact parathyroid hormone (iPTH) levels (median: 257 pmol/L, range: 122.4-366.0) and elevated ALP levels (median: 312 g/L, range: 121.8-754.3). Preoperative imaging included ultrasonography (90.0%), parathyroid scintigraphy (70.0%) and CT neck (60.0%). Of the seven parathyroid scintigraphies performed, two (28.6%) failed to identify a hyperfunctioning parathyroid lesion. The median PC size is 2.9 cm (range: 1.90-3.33 cm). Five patients (50.0%) had metastatic PC involving lymph nodes, lungs and bones. All patients underwent surgical resection, with adjuvant radiotherapy administered in three patients (30.0%). Two patients (20.0%) experienced recurrence, occurring 4 months and 28 months postoperatively.

CONCLUSION

Parathyroid carcinoma remains a challenge to diagnose as it closely resembles benign primary hyperparathyroidism. It should be considered in the presence of severe hypercalcemia, significantly elevated iPTH and a large parathyroid lesion. Early identification of PC through biochemical markers and imaging, coupled with surgical management is essential for optimal outcomes. Larger studies are warranted to further elucidate prognostic factors and optimize therapeutic strategies for this rare disease.

Adult Best Poster Presentation

BP_A004

CLINICAL CHARACTERISTICS AND COMPLICATIONS OF PRIMARY HYPERPARATHYROIDISM AT A TERTIARY CENTRE

<https://doi.org/10.15605/jafes.040.S1.008>

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INTRODUCTION

Primary hyperparathyroidism (PHPT) is the third most common endocrine disorder, following diabetes and thyroid diseases. The most common cause of PHPT is a single parathyroid adenoma, accounting for approximately 85% of cases. In this study, we describe the characteristics and complications of PHPT observed at an endocrine center.

METHODOLOGY

This cross-sectional study retrospectively reviewed data from patients diagnosed with PHPT who underwent parathyroidectomy at Hospital Putrajaya between 2012 and 2024. Relevant demographic, biochemical, clinical characteristics and complications were extracted from electronic medical records. Secondary and tertiary hyperparathyroidism were excluded.

RESULT

We studied 160 patients, of whom 65.0% were female with a median BMI of 24.5 kg/m². Nearly half were Malays (46.3%) and almost half had hypertension (48.1%). The majority (83.8%) experienced symptoms and complications, primarily renal (45.6%), skeletal (41.9%), and abdominal (31.9%). Among the renal complications, nephrolithiasis was the most common, while bone pain and osteoporosis were the most frequently reported skeletal complications. Patients underwent parathyroidectomy at a mean age of 52.2 years, either in the same year or within one year of diagnosis in 88.8% of cases. Pre-operative serum iPTH levels ranged from 3.70 to 325.40 pmol/L. The mean serum calcium levels pre-operation and six hours post-operation were 2.93 mmol/L and 2.59 mmol/L respectively ($p < 0.05$). One-third (31.3%) underwent right inferior parathyroidectomy. Adenoma was the most common tumour type, accounting for 91.8% of cases.

CONCLUSION

Primary hyperparathyroidism predominantly affects females, with most patients experiencing symptoms and complications, particularly renal and skeletal. Parathyroidectomy was performed around the age of 52, within a year of diagnosis. Adenomas were the most common tumor type, with a significant reduction in mean serum calcium level post parathyroidectomy.

Adult E-Poster

EP_A001

TRANSIENT RENAL TUBULAR ACIDOSIS IN PREGNANCY: A TRIGGER FOR RHABDOMYOLYSIS – A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.009>

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INTRODUCTION/BACKGROUND

Hypokalemia during pregnancy is a rare yet significant condition that can predispose to rhabdomyolysis, as demonstrated in this case report of a 27-year-old female.

CASE

We report a case of hypokalemia-induced rhabdomyolysis due to RTA (renal tubular acidosis) which occurred in 2 subsequent pregnancies. During her second pregnancy at 28 weeks gestation, the patient developed severe hypokalemia-induced rhabdomyolysis secondary to transient renal tubular acidosis (RTA), a condition also observed during her first pregnancy. Clinical findings included profound hypokalemia (2.1 mmol/L), metabolic acidosis, renal potassium wasting, and markedly elevated creatinine kinase levels. Immediate management with potassium supplementation and hydration led to symptom resolution, and the patient had an uneventful postpartum recovery.

CONCLUSION

This case highlights the rarity and complexity of transient yet recurrent RTA in pregnancy in association with rhabdomyolysis, emphasizing the importance of early diagnosis and multidisciplinary management to prevent fetomaternal complications. Follow-up and preemptive care in subsequent pregnancies are recommended.

EP_A002

APATHETIC HYPERTHYROIDISM: WHEN A COMMON DIAGNOSIS COMES WITH UNCOMMON MANIFESTATIONS

<https://doi.org/10.15605/jafes.040.S1.010>

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INTRODUCTION/BACKGROUND

The classic symptoms of hyperthyroidism include heat intolerance, tremors, palpitations, anxiety, weight loss despite an increased appetite, diarrhea, and breathlessness. In this case report, we looked at a case of atypical presentation of hyperthyroidism.

CASE

A 66-year-old male with underlying diabetes mellitus, hypertension, chronic kidney disease and gout presented with 1 week history of fever, lethargy, reduced appetite and giddiness. Unintentional weight loss was noted. Vital signs were stable with no tachycardia. Thyroid eye signs, tremor, and goiter were absent. ECG showed sinus rhythm. Initial lab data were significant for ALT 78 U/L, AST 68 U/L and ALP 140 U/L. He was started on empiric antibiotics and was worked up for the presence of a possible malignancy. Total bilirubin, hepatitis screening and tumor markers (CEA, Ca 19-9, PSA, AFP) were unremarkable. Subsequent blood tests showed worsening ALT 150 U/L, AST 133 U/L and ALP 156. Abdominal ultrasound revealed no signs of intra-abdominal fluid collection. After a week of antibiotics, the patient continued to complain of lethargy. Hence, more investigations were conducted. Surprisingly, the results showed TSH <0.005 mIU/L and T4 >78.10 pmol/L, indicative of the diagnosis of apathetic hyperthyroidism. The patient was started on Lugol's iodine 10 drops TDS for 10 days, T. cholestyramine 4 g TDS and discharged with the plan to start thionamides once his liver function improves.

CONCLUSION

Apathetic hyperthyroidism is a rare presentation of thyrotoxicosis. It poses diagnostic challenges due to the lack of characteristic signs and symptoms associated with hyperthyroidism. This can be attributed to decreased adrenergic tone, changes in the autonomic nervous system, and tissue resistance to thyroid hormone effects at the cellular level. Apathetic hyperthyroidism should be suspected in an elderly patient exhibiting inexplicable lethargy, weight loss, and depression.

Adult E-Poster

EP_A003

HYPOKALEMIA-INDUCED NEPHROGENIC DIABETES INSIPIDUS IN REFEEDING SYNDROME

<https://doi.org/10.15605/jafes.040.S1.011>

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INTRODUCTION/BACKGROUND

Nephrogenic diabetes insipidus is a rare disorder in which the body produces excessive amounts of urine. It can be caused by a genetic mutation or acquired factors such as certain medications (lithium, amphotericin-B), electrolyte imbalance (hypokalemia, hypercalcemia), chronic kidney disease or obstructive uropathy. In this case report, we describe a case of refeeding syndrome followed by hypokalemia-induced nephrogenic diabetes insipidus.

CASE

A cachectic 37-year-old male with BMI of 15.0 kg/m² with underlying mild intellectual disability and history of pulmonary tuberculosis (TB) who had completed his TB treatment presented at the emergency department with 2 weeks history of vomiting and 3 weeks history of bilateral lower limb weakness, loss of appetite and weight loss. Vital signs were stable. Systemic examination revealed bilateral lower limb weakness with power of 4/5. Laboratory data were significant for hypokalemia (1.8 mmol/L), hypophosphatemia (0.48 mmol/L) and hypocalcemia (1.6 mmol/L). The patient was admitted for electrolyte correction. At the ward, patient was noted to be producing excessive amounts of urine ranging from 4 to 10 L/day. Water deprivation test was performed. It showed failure to increase urine osmolality with water deprivation and lack of response to desmopressin suggestive of nephrogenic diabetes insipidus. Spot urine potassium was low at 9.8 mmol/L. With multiple corrections of electrolytes, hypokalemia, hypophosphatemia and hypocalcemia gradually resolved. The patient was started on a low-calorie, high-protein diet for the consideration of refeeding syndrome. Following correction of electrolytes with potassium reaching a stable level of 4 mmol/L, on the 17th day of admission, the daily urine output was restored to a normal volume of approximately 2 L/day. After three days, he was discharged.

CONCLUSION

Patients with polyuria and hypokalemia should be evaluated for nephrogenic diabetes insipidus. Hypokalemia-induced nephrogenic diabetes insipidus can be reversed by correcting hypokalemia.

EP_A004

A CASE OF SEVERE HYPERCALCAEMIA SECONDARY TO PARATHYROID CARCINOMA

<https://doi.org/10.15605/jafes.040.S1.012>

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INTRODUCTION/BACKGROUND

Parathyroid carcinoma is a rare malignancy, accounting for only 0.005% of all cancers and about 0.5-1% of parathyroid disorders with similar incidence in male and females. It usually presents with manifestations of severe hypercalcaemia with bone involvement and nephrolithiasis, associated with markedly raised parathyroid hormone; although presentations can be variable.

CASE

We report a case of a 44-year-old Chinese male with underlying hypertension, chronic kidney disease stage 3A (eGFR 52 mL/min/1.73 m²) who was incidentally found to have PTH-dependent severe hypercalcaemia (corrected calcium 4.56 mmol/L), hypophosphataemia with iPTH 102.1 (>7 times the upper limit of normal) during admission for left cheek subcutaneous abscess. Hypercalcaemia was managed with calcitonin, bisphosphonate alongside aggressive saline diuresis resulting to a serum calcium of 2.85 mmol/L. Ultrasound of the neck showed a well-defined ovoid hypoechoic lesion, caudal to the lower pole of the left thyroid lobe measuring 1.4 x 1.5 x 2.0 cm consistent with left parathyroid adenoma, which was confirmed with parathyroid Sestamibi scintigraphy. Ultrasound of the kidneys revealed no nephrolithiasis while bone densitometry showed severe osteoporosis at the distal third of forearm and left neck of femur with T score -4.1 and 3.0 respectively. Surgical excision of the left parathyroid with intraoperative PTH monitoring was done with subsequent removal of the lesion leading to normalization of the PTH level. Histopathology examination revealed lymphovascular permeation with irregular nodular proliferation of parathyroid cells with transgressed boundaries which are features compatible with parathyroid carcinoma. Post-operatively, the patient was normocalcaemic but declined further surgical tumour clearance.

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CONCLUSION

This case highlights the need for a high index of clinical suspicion for the presence of parathyroid carcinoma pre-operatively in patients who exhibit severe hypercalcemia, markedly raised PTH and bone manifestations so that en bloc-resection of the parathyroid with ipsilateral partial thyroidectomy and central node dissection can be planned prior to surgery.

EP_A005

THYMIC HYPERPLASIA IN GRAVES' DISEASE: A DIAGNOSTIC AND MANAGEMENT CHALLENGE

<https://doi.org/10.15605/jafes.040.S1.013>

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INTRODUCTION/BACKGROUND

Thymic hyperplasia is a recognized but frequently underappreciated entity associated with Graves' disease (GD). It is often misinterpreted as a mediastinal mass, potentially leading to unwarranted biopsies or surgical intervention. The underlying pathophysiological mechanisms remain poorly understood. Spontaneous regression of the mediastinal mass following euthyroidism with effective thyrotoxicosis treatment supports a benign etiology. Here, we present a case of a young female with GD and an incidentally discovered anterior mediastinal mass, highlighting the diagnostic complexities that necessitated a multidisciplinary approach.

CASE

A 21-year-old female presenting with a large goiter, a thyrotoxic state (FT4 >100 pmol/L, TSH 0.01 mU/ml and anti-TSH receptor Ab >40 IU/L) with no thyroid ophthalmopathy was diagnosed with GD. Despite medical management, adequate control of her thyroid hormone levels proved to be challenging, prompting a surgical consultation for a potential thyroidectomy. To assess the extent of the goiter, computed tomography (CT) imaging was performed, revealing a grossly enlarged thyroid gland with mild tracheal narrowing and a well-defined, solid, enhancing 5.6 cm × 6.4 cm × 4.3 cm anterior mediastinal mass.

Given the initial concern for an ectopic thyroid gland or malignancy, performing an invasive biopsy was considered. However, a multidisciplinary team consisting of experts from endocrinology, surgery, respiratory medicine, radiology, and nuclear medicine reviewed the findings and concluded that the mass was most consistent with

thymic hyperplasia. Considering the high surgical risk, a conservative approach was pursued, with the patient undergoing radioiodine therapy for thyrotoxicosis and serial imaging to monitor the mediastinal mass. Long-term outcomes are yet to be seen.

CONCLUSION

This case underscores the diagnostic challenges posed by thymic hyperplasia in patients with GD and the potential for misdiagnosis as a mediastinal pathology. Awareness of this association is crucial in order to avoid unnecessary surgical interventions. A multidisciplinary approach is essential for accurate diagnosis and optimal management, promoting a conservative therapeutic strategy when appropriate.

EP_A006

GRANULOMATOUS DISEASE-INDUCED SEVERE HYPERCALCEMIA

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INTRODUCTION/BACKGROUND

Hypercalcemia is commonly seen in granulomatous disease especially in sarcoidosis in around 40-50% cases; however, lower rates of association have been reported in tuberculosis. The etiology is due to the production of extrarenal 1-alpha-hydroxylase enzymes by activated macrophages seen in the granulomas. This will then lead to inappropriately elevated 1,25-dihydroxyvitamin D causing dysregulation of calcium metabolism.

CASE

A patient with a known case of disseminated tuberculosis (TB) was admitted to critical care with an initial impression of cerebral toxoplasmosis. Throughout his admission, blood parameters were closely monitored which revealed moderate to severe hypercalcemia ranging from 2.8-4.0 mmol/L with clinical features of nephrogenic diabetes insipidus (polyuria of 5440 ml urine output per day, hyponatremia ranging 147-157 mmol/L (135-145 mmol/L) and low urine osmolality 143 mOsm/kg). However, despite treatment with hydration, severe hypercalcemia resulted in the atypical presence of J-wave or Osborn wave on electrocardiogram (ECG). Hypothermia has been ruled out as his body temperature ranges from 36.7-37 °C. There is no interruption in his TB medications and iatrogenic

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causes have also been excluded. Intact parathyroid hormone (iPTH) yielded a low result of 1.61 pmol/L (1.95-8.49 pmol/L). Overall features point to non-iPTH dependent hypercalcemia. Corticosteroid therapy with IV hydrocortisone 50 mg TDS together with subcutaneous calcitonin 100 iU TDS were initiated. Bisphosphonate therapy consisting of one dose of intravenous zoledronic acid 4 mg was added to the therapeutic regimen the next day. After 3 days of treatment, hypercalcemia resolved with corrected calcium ranging from 1.7-2.3 mmol/L. The patient unfortunately succumbed to overwhelming sepsis with multiorgan involvement.

CONCLUSION

Granuloma-induced-hypercalcemia remains a diagnostic challenge in persons with TB due to its uncommon occurrence. However, there must be a high index of suspicion to facilitate early intervention with calcium lowering drugs to avoid morbidity and mortality in such patients.

EP_A007

BEYOND THE SORE THROAT: UNVEILING THE THYROID'S HIDDEN TURMOIL

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INTRODUCTION

Subacute thyroiditis (SAT) is an inflammatory thyroid disorder often presenting with anterior neck pain and transient thyroid dysfunction. This case highlights the diagnostic challenges and evolving thyroid function tests (TFT) in a patient initially referred for prolonged throat pain.

CASE

A 50-year-old female presented with anterior neck pain persisting for two weeks, initially treated with antibiotics for suspected infection. She had no dysphagia, odynophagia, or overt hyperthyroid symptoms but reported a significant weight loss of 6 kg over a year, early satiety, and loss of appetite. Examination revealed tenderness over the thyroid gland with no palpable goiter or lymphadenopathy. Initial TFT showed suppressed TSH <0.01 mIU/L with elevated free T4 of 56.4 pmol/L.

Inflammatory markers were raised, with an ESR of 77 mm/hr and CRP of 71.5 mg/L, supporting an inflammatory process. Tumor markers, including CEA, AFP, CA-125, CA 15-3, and CA 19-9, were not elevated, reducing the suspicion for the presence of malignancy.

Follow-up TFT showed a rapid transition from hyperthyroidism (TSH <0.01, T4 56.4) to euthyroidism (TSH 1.39, T4 8.6), and subsequently, hypothyroidism (TSH 9.9, T4 7.1). The dynamic TFT pattern, absence of thyroid autoantibodies, and recent upper respiratory symptoms supported the diagnosis of subacute thyroiditis.

The patient was started on L-thyroxine 50 mcg OD due to hypothyroid progression, with plans for TFT reassessment in 4 weeks. Referral to gastroenterology for early satiety was deferred, considering the likelihood of thyroid-related symptoms.

CONCLUSION

This case underscores the importance of recognizing evolving TFT trends in subacute thyroiditis in order to avoid unnecessary interventions. A systematic approach to thyroid dysfunction in medical care is crucial for timely diagnosis and management, bridging the gap between knowledge and clinical practice.

EP_A008

POST-COVID-19 CHRONIC FATIGUE SYNDROME WITH ACUTE PANCREATITIS AND TRANSIENT HYPERZINCEMIA

<https://doi.org/10.15605/jafes.040.S1.016>

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INTRODUCTION/BACKGROUND

Chronic fatigue syndrome (CFS) often follows viral infections, including COVID-19. Long COVID is increasingly recognized as a cause of post-viral fatigue. While rare, hyperzincemia has been linked to acute pancreatitis. This case explores the interplay between post-viral fatigue, metabolic disturbances, autonomic dysfunction, and transient hyperzincemia in acute pancreatitis.

CASE

A 61-year-old male with hypertension, benign prostatic hyperplasia, hepatosteatorosis, and gallstone-induced acute pancreatitis presented with persistent fatigue

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post-COVID-19. Clinical evaluation included metabolic, endocrine, and nutritional markers, focusing on zinc, copper, ceruloplasmin, thyroid, and adrenal function. Autonomic function and post-exertional fatigue patterns were assessed.

The patient developed persistent fatigue following his third and most severe COVID-19 infection, which required hospitalization. Fatigue worsened with exertion and was not relieved by rest. Gallstone-related acute pancreatitis revealed transient hyperzinaemia (serum zinc: 153 mcg/dL, reference: 60-106 mcg/dL) with normal copper, ceruloplasmin, and adrenal function (AM cortisol: 196 nmol/L).

Possible mechanisms include transient zinc release from pancreatic tissue due to acinar cell destruction, reduced zinc excretion resulting from impaired clearance due to the presence of hepatic dysfunction and potential renal impairment, gallstone-related factors such as the presence of cholestasis leading to decreased biliary excretion, altered zinc distribution due to systemic inflammation, exogenous sources leading to contamination or artifacts arising from measurement errors.

Fatigue improved with nil by mouth but recurred post-discharge. Blood pressure fluctuations during this period suggest possible autonomic or even beginning adrenal dysfunction. Hyperzinaemia resolved with dietary modifications.

CONCLUSION

Post-COVID-19 fatigue requires a thorough metabolic, endocrine, and autonomic evaluation. This case highlights transient hyperzinaemia in acute pancreatitis and the need for cautious interpretation of trace element abnormalities. Understanding zinc metabolism and autonomic dysfunction may offer insights into post-viral fatigue syndromes.

EP_A009

THE FIRST CASE OF GUSELKUMAB-INDUCED THYROID STORM IN A YOUNG WOMAN WITH PLAQUE PSORIASIS

<https://doi.org/10.15605/jafes.040.S1.017>

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INTRODUCTION/BACKGROUND

Guselkumab is a biologic agent used to treat moderate to severe plaque psoriasis by targeting interleukin-23 (IL-23). While effective in managing psoriasis, the impact of Guselkumab on thyroid function is not well-documented. Autoimmune thyroid diseases such as Graves' disease can be triggered by several factors, including immune-modulating therapies. This case report aims to highlight a rare but severe adverse reaction of Guselkumab in a young female with a predisposition to autoimmune diseases.

CASE

We report a 20-year-old Malay female, a medical student, with plaque psoriasis on Guselkumab therapy. Her elder sister has psoriasis, Graves' disease with severe orbitopathy. Following the patient's first injection of Guselkumab, she developed a moderate-sized diffuse goiter with tenderness. Despite this, she continued with two more doses of Guselkumab over the next six months at three-month intervals.

Approximately two weeks after the fourth dose of Guselkumab, she experienced symptoms of palpitations, hand tremors, low-grade fever, and generalized malaise. She was admitted to the hospital for treatment of severe thyrotoxicosis. Serum free T4 levels were found to be three times above the upper limit of normal, T4: 59.1 pmol/l and a TSH level of <0.01 mIU/l with borderline high anti-TPO antibodies. Despite good compliance with carbimazole 30 mg daily and propranolol 60 mg three times daily for one month, her condition worsened.

Development of signs of thyroid storm, including anxiety, hyperdefecation, hand tremor, low-grade fever (37.6 °C), and sinus tachycardia (150 beats per minute) prompted consult at the emergency department, where she was found to have Burch-Wartofsky Point Scale of 45. Acute phase reactants showed a CRP level of 5, which made subacute thyroiditis unlikely. Due to the severity of her condition,

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Lugol's iodine was administered, thus precluding the performance of thyroid scintigraphy. Neck ultrasound revealed features suggestive of thyroiditis in which Graves' disease cannot be excluded, with no focal lesion of thyroid parenchyma found.

During the admission, response to intravenous hydrocortisone 100 mg tds and high dose propylthiouracil 250 mg QID was slow, thus necessitating alternative treatment with T. cholestyramine 1 g QID. Her TSH level remained static at <0.01 mIU/l and free T4 decreased from >64 pmol/l to 54.5 pmol/l then to 32.2 pmol/l.

She subsequently underwent a total thyroidectomy for severe Graves' disease with grade 3 goiter. The postoperative course was complicated by transient hypocalcemia requiring calcium and vitamin D supplementation. Psoriasis remained well-controlled but a flare developed postoperatively, prompting the reintroduction of Guselkumab.

CONCLUSION

This case underscores the importance of monitoring thyroid function in patients receiving biologic agents, especially in those with a known predisposition to autoimmune diseases. Clinicians should remain vigilant for signs of thyroid dysfunction and consider the potential of biologic agents like Guselkumab to trigger severe autoimmune reactions, including thyroid storm. Early surgical intervention enabled optimal treatment of the skin disorder while preventing further life-threatening complications.

EP_A010

SEVERE HYPERCALCAEMIA AFTER TREATMENT WITH EMPAGLIFLOZIN IN A PATIENT WITH POSTSURGICAL HYPOPARATHYROIDISM

<https://doi.org/10.15605/jafes.040.S1.018>

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INTRODUCTION/BACKGROUND

Sodium-glucose co-transporter 2 (SGLT2) inhibitors have been reported to cause hypercalcaemia in some literature. We describe a patient with postsurgical hypoparathyroidism who was on stable doses of calcium and activated vitamin D but developed severe hypercalcaemia after taking a SGLT2 inhibitor.

CASE

A 77-year-old female was admitted for a three-day history of dizziness and unsteadiness in December 2024. She had type 2 diabetes mellitus, hypertension, dyslipidaemia, ischaemic cardiomyopathy, stage 4 chronic kidney disease, as well as hypothyroidism and hypoparathyroidism post-subtotal thyroidectomy in 1974. Maintenance medications included basal bolus insulin regimen, aspirin 100 mg daily, atorvastatin 40 mg daily, bisoprolol 2.5 mg daily, furosemide 40 mg daily, levothyroxine 25 mcg daily, calcium carbonate 2 g thrice daily and alfacalcidol 1 mcg daily. Calcium level in July 2024 was 2.51 mmol/L (normal range: 2.10-2.55). In October 2024, she was prescribed empagliflozin 25 mg daily by her cardiologist. On examination, she was dry and lethargic. Blood pressure was 136/79 mm Hg with evidence of postural hypotension. Blood glucose was 12.6 mmol/L with no evidence of diabetic ketoacidosis. Physical examination was unremarkable. Severe hypercalcaemia (corrected calcium 3.59 mmol/L) and acute-on-chronic kidney disease (creatinine rose from 201 µmol/L to 231 µmol/L) were noted. Intravenous saline infusion was administered and intravenous furosemide 40 mg daily was subsequently given. Calcium carbonate, alfacalcidol and empagliflozin were withheld. Calcium level normalised and renal function returned to baseline nine days after admission, accompanied by marked clinical improvement. Calcium carbonate 1 g twice daily and alfacalcidol 1 mcg daily were reintroduced when calcium level declined to 2.53 mmol/L. Two weeks after discharge, her calcium level remained normal at 2.41 mmol/L.

CONCLUSION

Sodium-glucose co-transporter 2 (SGLT2) inhibitors potentially cause dehydration from osmotic diuresis and increased intestinal calcium absorption. Close monitoring of calcium level is recommended after initiating SGLT2 inhibitors, particularly in elderly patients who are also taking oral calcium.

Adult E-Poster

EP_A011

STEROID-INDUCED HYPERGLYCEMIA IN AN ADOLESCENT WITH OBESITY: A COMPLEX CHALLENGE IN ACUTE MENINGOENCEPHALITIS MANAGEMENT

<https://doi.org/10.15605/jafes.040.S1.019>

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INTRODUCTION/BACKGROUND

Glucocorticoids are synthetic medications mimicking cortisol, characterized by potent anti-inflammatory properties. These pharmacological agents significantly disrupt glucose metabolism, potentially leading to steroid-induced hyperglycemia, which increases the risk of developing diabetes mellitus and metabolic dysregulation. Those patients are predisposed to developing acute emergencies such as hyperglycemic hyperosmolar state or diabetic ketoacidosis. This article also tackles different mechanisms which contribute to these complications.

CASE

We reported a case of a 14-year-old Malay male with morbid obesity (BMI 40 kg/m²) who was admitted for severe meningoencephalitis secondary to a complicated ear infection. The patient became critically ill and necessitated intensive care upon revival from cardiac arrest after 14 minutes of performing cardiopulmonary resuscitation. Upon diagnosing posterior fossa meningoencephalitis, intravenous dexamethasone 8 mg three times daily was administered to mitigate cerebral edema. Such intervention triggered a significant metabolic disturbance in the form of acute hyperglycemia. Even if diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) were ruled out, the patient still required aggressive insulin management. A high-dose insulin infusion was implemented using a fixed-scale protocol, administering 20 units of insulin hourly. After three days, IV dexamethasone was discontinued due to persistent poor glycemic control. Subsequently, the fixed-scale protocol was transitioned to a sliding-scale insulin regimen over 12 hours. Eventually, the treatment was modified to basal Insulatard, resulting in gradual improvement of blood glucose control. The patient's HbA1c level was found to be 6.3% despite the acute hyperglycemia, given that he has no previous history of diabetes mellitus.

CONCLUSION

This case highlights the significant metabolic effects of corticosteroid therapy, particularly in an adolescent patient with obesity. The rapid onset of steroid-induced hyperglycemia required prompt and adaptive insulin therapy. A sedentary lifestyle, along with overweight and obesity, can increase the risk of developing insulin resistance, complicating treatment and potentially leading to more challenging management. This may, in turn, elevate the risk of increased morbidity and mortality.

EP_A012

SYNCHRONOUS PRIMARY HYPERPARATHYROIDISM AND PAPILLARY THYROID CANCER, INITIALLY PRESENTING WITH RECURRENT CHEST PAIN: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.020>

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INTRODUCTION/BACKGROUND

The coexistence of primary hyperparathyroidism (pHPT) and thyroid disease is well recognized, but the simultaneous occurrence of pHPT due to parathyroid hyperplasia and thyroid malignancy, particularly papillary thyroid carcinoma (PTC), is rare. We present a unique case of a 46-year-old female diagnosed with pHPT due to parathyroid hyperplasia and concurrent PTC affecting both thyroid lobes.

CASE

A 46-year-old female had recurrent chest pain and was evaluated by cardiology in a district hospital from 2021 to May 2024. Persistent hypercalcemia, unnoticed initially, was later identified. An exercise stress test was inconclusive, and a CT coronary angiogram showed no coronary stenosis or plaque, with a total calcium score of 0. Moderate hypercalcemia prompted referral to endocrinology.

Laboratory investigations revealed elevated corrected calcium (3.01 mmol/L), low phosphate (0.73 mmol/L), elevated intact parathyroid hormone (iPTH) (197 pg/mL), normal alkaline phosphatase (138 U/L), and low 25-hydroxy vitamin D3 (33 nmol/L), suggestive of PTH-mediated hypercalcemia. A 24-hour urine calcium-creatinine ratio was low, likely due to vitamin D deficiency. Thyroid function tests were normal.

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Neck ultrasound identified bilateral thyroid nodules, including a highly suspicious left-sided nodule (TIRADS 5). Technetium (99 mTc) sestamibi scintigraphy demonstrated a parathyroid adenoma (0.9 × 0.8 × 2.7 cm) infero-posterior to the lower pole of the left thyroid gland. Fine-needle aspiration biopsy of the thyroid nodule was suspicious for PTC. Further imaging revealed right nephrolithiasis, and a DEXA scan indicated severe osteoporosis (T-score: -3.7 at L4).

The patient underwent total thyroidectomy with left inferior parathyroidectomy and central neck lymph node dissection in October 2024. Histopathology confirmed PTC in a background of nodular hyperplasia (TNM staging: pT1b pN1a). The left inferior parathyroid gland showed hyperplasia. Postoperatively, the patient was chest pain-free and is currently on cholecalciferol with calcium carbonate supplementation.

CONCLUSION

Recognizing chest pain in the setting of PTH-mediated hypercalcemia is crucial to prevent complications of chronic hypercalcemia and avoid unnecessary cardiac investigations. This case underscores the need for thorough endocrine and metabolic evaluations in patients presenting with persistent hypercalcemia and chest pain.

EP_A013

ALCOHOL-INDUCED REVERSAL OF SEMAGLUTIDE'S GLYCAEMIC BENEFITS: A CASE STUDY

<https://doi.org/10.15605/jafes.040.S1.021>

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INTRODUCTION/BACKGROUND

Semaglutide is a GLP-1 receptor agonist widely used in the management of type 2 diabetes. Alcohol is known to interfere with glucose metabolism and insulin sensitivity. This case highlights how alcohol consumption negated the glycaemic benefits of semaglutide, with marked improvement of glycaemic control observed during periods of abstinence.

CASE

A 37-year-old male, diagnosed with type 2 diabetes in 2020, initially presented with poor glycaemic control (HbA1c 9.5%). Semaglutide was initiated in September 2022, leading to a significant improvement in HbA1c, which eventually

dropped to 5.7%. Despite this, his weight remained stable between 108–110 kg. However, by early 2025, his HbA1c had again risen to 9%, despite continued use of semaglutide. Over this period, a pattern emerged, with fluctuations in his HbA1c between approximately 6%–9%, corresponding to his drinking habits—rising during periods of active alcohol consumption and improving during months of sobriety.

The patient consumed around 20 units of whisky per week, in light of his profession in the liquor industry. Despite awareness of the risks, he struggled with abstinence. Other confounding factors such as medication adherence, diet, physical activity, and organ dysfunction were ruled out.

Chronic alcohol use is known to impair GLP-1 activity by reducing secretion and increasing degradation. Additionally, alcohol can induce insulin resistance through hepatic steatosis, systemic inflammation, and oxidative stress. Ethanol metabolism generates excess NADH, inhibiting gluconeogenesis, while alcohol-induced glucagon dysregulation may further increase hepatic glucose production. Moreover, alcohol promotes increased caloric intake, disrupts appetite regulation, and contributes to mitochondrial dysfunction.

CONCLUSION

This case underscores the importance of assessing alcohol intake in patients using GLP-1 receptor agonists. Chronic alcohol use may negate semaglutide's glycaemic lowering effects. Clinicians should actively counsel patients on alcohol's impact on diabetes management and consider strategies to encourage periods of sobriety for optimal therapeutic outcomes.

EP_A014

CHALLENGES IN THE DIAGNOSIS AND MANAGEMENT OF EXCLUSIVELY DOPAMINE SECRETING PARAGANGLIOMA

<https://doi.org/10.15605/jafes.040.S1.022>

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INTRODUCTION/BACKGROUND

Head and neck paragangliomas (PGLs) comprise 65% to 70% of all paragangliomas. Functioning head and neck paragangliomas are rare, particularly carotid body paraganglioma with solely dopamine secretion. Majority of dopamine secreting paragangliomas are poorly differentiated with locally invasive or metastatic potential.

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CASE

A 48-year-old female, with no known medical illness presented with right neck swelling for 5 months duration. The painless neck swelling progressively increased in size, with no obstructive symptoms. She did not exhibit any symptoms related to catecholamine excess. No medications were given as well. There was no other significant personal or family medical history, including familial cancer syndromes such as multiple endocrine neoplasia type 2 (MEN 2), Von-Hippel Lindau (VHL) and neurofibromatosis (NF1). She was normotensive (133/64 mm Hg), with normal heart rate (90 beats per minute). Neck examination revealed right neck swelling measuring 2.5 cm x 3 cm, well demarcated, firm and immobile. Biochemistry results showed normal metanephrine (0.43 umol/24H), normal normetanephrine (0.66 umol/24H) but elevated 24 hour urine 3-Methoxytyramine (6.66 umol/24H). Computed tomography scan and MRI of the neck demonstrated a right carotid space enhancing mass measuring 3.2 x 3.0 x 4.1 cm. Subsequently, CT scan of the thorax, abdomen and pelvis were carried out, but no adrenal nodule or mass was noted. After a week of alpha-blockade as preoperative management, she successfully underwent pre-embolization and tumor excision via transcervical approach. Intra-operatively, neither hypotension nor hypertension was noted. After the operation, she required voice rehabilitation and recovered well. Histopathology report confirmed the diagnosis of exclusively dopamine secreting carotid body paraganglioma with no extension to the lymph nodes. Post-operatively, PET scan and 24 hour urine metanephrine/normetanephrine/ 3-methoxytyramine were conducted and no biochemical or imaging evidence of recurrence or metastasis was observed.

CONCLUSION

Dopamine secreting paragangliomas are rare and difficult to diagnose. Hence as clinicians, one needs to have a high index of suspicion to enable early diagnosis and management.

EP_A015

EUGLYCEMIC DIABETIC KETOACIDOSIS: ELUSIVE, YET A DIAGNOSIS NOT TO BE OVERLOOKED IN CASES OF UNEXPLAINED METABOLIC ACIDOSIS

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INTRODUCTION

Euglycemic diabetic ketoacidosis is a rare but serious condition. The absence of hyperglycemia frequently causes a delay in diagnosis and treatment initiation. We present a case of acute coronary syndrome in cardiogenic shock in which the euglycemic DKA diagnosis was missed.

CASE

A 65-year-old female with underlying diabetes mellitus, hypertension, chronic kidney disease and ischemic heart disease presented with typical chest pain and heart failure symptoms. Patient was tachypneic with Grd 2 edema, BP 107/58 mm Hg, HR 98 beats/min, SpO₂ 89% at room air and blood glucose 6.3 mmol/L. Electrocardiogram had dynamic changes. Initial blood investigations showed urea 15.3 mmol/L, sodium 133 mmol/L, K 4.4 mmol/L, Cl 105 mmol/L, creatinine 376 umol/L, pH 7.236, lactate 6.7 mmol/L, bicarbonate 12.2 mmol/L and anion gap 16.6 mmol/L. Bedside ultrasound revealed ejection fraction of 40-50%, RWMA, plethoric IVC measuring 2.3 cm. As the patient's blood pressure dropped, noradrenaline was administered with the furosemide infusion. The patient was assessed to have acute decompensated heart failure in cardiogenic shock secondary to acute coronary syndrome and acute on chronic kidney disease. Despite optimal doses of diuretics, there was no urine output. Dialysis was initiated due to refractory fluid overload. Venous blood gas post dialysis showed pH 7.184, HCO₃ 11.5 mmol/L, glucose 7 mmol/L, lactate 1.6 mmol/L and anion gap 17 mmol/L. Despite dialysis and improved serum lactate levels, the metabolic acidosis worsened. Capillary ketone was taken for unexplained acidosis showing an alarming value of 4.3 mmol/L, confirming the diagnosis of euglycemic DKA. Insulin infusion with dextrose was initiated. Follow-up VBG indicates an improvement of pH to 7.273 and HCO₃ to 13.6 mmol/L.

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CONCLUSION

Failure to recognize euglycemic DKA may lead to catastrophic outcomes. Clinicians must maintain a high index of suspicion in high-risk populations and advocate for ketone testing in unexplained metabolic acidosis, regardless of glucose levels. Early recognition and targeted therapy can rapidly reverse acidosis and prevent morbidity.

EP_A016

CUSHING'S DISEASE AND THE COST OF DELAY: FROM METABOLIC TO SKELETAL FRAGILITY

<https://doi.org/10.15605/jafes.040.S1.024>

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INTRODUCTION/BACKGROUND

Cushing's disease, caused by an ACTH-secreting pituitary adenoma, can lead to profound metabolic disturbances, including insulin resistance, hypertension, osteoporosis, and an increased risk of fractures. Timely intervention is crucial to prevent long-term complications. Surgical removal of the pituitary adenoma via transsphenoidal surgery remains the gold standard treatment, offering potential for disease remission and metabolic recovery.

CASE

A 39-year-old female was initially investigated at age 34 for young-onset hypertension, recurrent hypokalaemia, and diabetes mellitus. Despite the absence of classical Cushingoid features, biochemical evaluation revealed persistent hypercortisolism, with an unsuppressed overnight dexamethasone suppression test (ONDST 565 nmol/L), low-dose dexamethasone suppression test (607 nmol/L), and markedly elevated 24-hour urinary cortisol (1401 nmol/L). Adrenocorticotrophic hormone (ACTH) levels were elevated (5.4 pmol/L), and a cortisol day curve confirmed the loss of cortisol and ACTH diurnal rhythm. Magnetic resonance imaging identified a left pituitary microadenoma (5.9 × 6.5 mm). However, the patient was lost to follow-up and was only reinvestigated after sustaining a T10 compression fracture from a trivial fall. Repeat biochemical testing reaffirmed hypercortisolism (ODST: 750 nmol/L, 24-hour urinary cortisol: 1544 nmol/L, ACTH: 9.7 pmol/L). Magnetic resonance imaging showed a stable pituitary lesion (6.2 × 4.0 mm), and inferior petrosal sinus sampling confirmed a pituitary source of ACTH hypersecretion, with post-DDAVP central-to-peripheral ACTH ratios >3. Ketoconazole was initiated (titrated to 400 mg BD) for biochemical control. She successfully underwent

endoscopic transsphenoidal surgery with adenomectomy and hypophysectomy in October 2024. Postoperatively, she achieved remission but developed panhypopituitarism, necessitating hormone replacement with hydrocortisone, L-thyroxine, and estradiol (Progyluton). Remarkably, she no longer required diabetes treatment, and her hypertension improved, requiring only a single antihypertensive agent.

CONCLUSION

This case highlights the challenges of diagnosing Cushing's disease in the absence of overt clinical features, the devastating skeletal consequences of delayed treatment, and the transformative impact of successful surgical intervention. Early recognition, multidisciplinary management, and timely surgical intervention remain paramount in optimizing patient outcomes.

EP_A017

AN UNUSUAL SITE OF ADRENOCORTICAL CARCINOMA

<https://doi.org/10.15605/jafes.040.S1.025>

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INTRODUCTION/BACKGROUND

Adrenocortical carcinoma (ACC) is a rare malignancy with an incidence of 0.5–2 cases per million per year. Typically, ACC originates in the adrenal glands. Although exceedingly rare, ectopic presentations can occur due to developmental anomalies and rarely may arise from an adrenal rest. These adrenal rests are usually clinically silent, but on rare occasions, may undergo malignant transformation and hormonal secretion.

CASE

We report the case of a 33-year-old female with underlying hypertension and diabetes who had an incidentally discovered right adnexal mass which was asymptomatic during a routine medical checkup. She underwent complete laparoscopic tumour resection without complications. Comprehensive histopathologic evaluation revealed a low-grade ectopic ACC arising from an adrenal rest. Postoperative imaging demonstrated no residual tumor and normal adrenal glands. She remains under active surveillance.

The case highlights the diagnostic challenge posed by an ectopic ACC masquerading as an adnexal mass. Detailed histopathologic and immunohistochemical analyses are

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essential in accurately determining tumor origin, thus guiding optimal management strategies. Adrenal rests have been described within the retroperitoneum, broad ligament, testis, ovaries and inguinal region. Due to limited data, the management of ectopic ACC is generally considered similar to that of eutopic tumors. Complete surgical resection is still the mainstay of treatment for both eutopic and ectopic ACC. Long-term follow-up and close monitoring are imperative given the risk of recurrence.

CONCLUSION

This case underscores the importance of maintaining a high index of suspicion, as many ectopic adrenocortical rests are under-recognized due to their small size and low clinical relevance. Awareness of ectopic adrenal rests is crucial to correctly identify sources of adrenocortical hormone production, avoid misinterpretations in the diagnostic workup of intraabdominal masses, and to evaluate for possible malignant transformation.

EP_A018

SUCCESSFUL THYROIDECTOMY IN SEVERE GRAVES' DISEASE: A MODIFIED BLOCK-AND-REPLACE APPROACH

<https://doi.org/10.15605/jafes.040.S1.026>

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INTRODUCTION/BACKGROUND

Graves' disease is the most common cause of autoimmune hyperthyroidism. In severe cases, thyroidectomy is required. The block-and-replace regimen helps achieve euthyroidism preoperatively, but perioperative thyroid instability remains a challenge, particularly in urgent surgical settings.

CASE

A 20-year-old Malay female with severe plaque psoriasis developed a painful goiter and severe thyrotoxicosis following Guselkumab treatment, necessitating carbimazole 30 mg daily. She was initially scheduled for radioactive iodine (RAI) therapy; however, two weeks after her fourth Guselkumab dose, just before her planned RAI, she had thyroid storm. Emergency management included Lugol's iodine, high-dose propylthiouracil, corticosteroids, and cholestyramine. Due to recent iodine exposure, RAI was no longer a viable option, necessitating an alternative definitive treatment approach.

Methimazole was increased from 20 mg to 25 mg twice daily, successfully lowering free T4 from 27 to 17 pmol/L. However, on the day before her scheduled thyroidectomy, severe hypothyroidism (TSH <0.01 mIU/L, T4 <5 pmol/L) was noted. To rapidly restore euthyroidism, she received a total of 300 mcg of levothyroxine overnight while continuing methimazole. This intervention raised her T4 to 8.3 pmol/L, ensuring safe surgical conditions while mitigating the risk of recurrent thyroid storm in this difficult-to-control case.

CONCLUSION

This case highlights the challenges of perioperative thyroid management in Graves' disease. High-dose levothyroxine while maintaining methimazole facilitated urgent surgical clearance, balancing the risks of hypothyroidism and thyroid storm. This modified block-and-replace approach may be considered in select cases requiring time-sensitive surgical intervention.

EP_A019

HYPORENINAEMIC HYPOALDOSTERONISM (HH) AS THE CAUSE OF UNEXPLAINED HYPERKALAEMIA

<https://doi.org/10.15605/jafes.040.S1.027>

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INTRODUCTION

Hyporeninaemic hypoaldosteronism (HH) is a frequently overlooked cause of hyperkalaemia. In HH, juxtaglomerular apparatus dysfunction secondary to diabetes, chronic kidney disease and medications like NSAIDs, ACEI, and heparin leads to reduced renin secretion, thus decreasing aldosterone synthesis, resulting to impaired potassium excretion and H⁺ secretion. Hyperkalaemia and metabolic acidosis ensue respectively with no adrenal insufficiency.

CASE

A 57-year-old female presented with persistent and asymptomatic hyperkalaemia for a year at primary care. Hemolysis was ruled out. Electrocardiogram findings remained normal throughout. She had type 2 diabetes mellitus for 15 years, hypertension and stage 2 chronic kidney disease (CKD) (eGFR ~62 mL/min/1.73 m²) for 2 years. Diabetes was moderately controlled with metformin. Hypertension was treated with amlodipine. Additionally, she had been using NSAIDs intermittently for back pain over the last three years. Due to the presence of hyperkalaemia despite the fairly normal renal function,

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she was referred to an endocrinologist. Further evaluation included a morning serum cortisol level at 9 am, which was normal, ruling out adrenal insufficiency. However, serum aldosterone was low with inappropriately low renin levels despite elevated potassium. Mild metabolic acidosis was present. These findings strongly suggested a diagnosis of HH. The patient was managed through dietary potassium restriction, discontinuation of NSAIDs, initiation of fludrocortisone for potassium excretion, and sodium bicarbonate to correct the metabolic acidosis with a close watch on renal function and potassium levels. Within two weeks, her potassium levels normalized and remained stable thereafter. Hyperkalaemia was ultimately attributed to HH, likely caused by a combination of chronic diabetes with CKD and the use of NSAIDs, all leading to juxtaglomerular apparatus dysfunction.

CONCLUSION

This case highlights the importance of considering HH in persons with diabetes or CKD with unexplained hyperkalaemia despite a normal or mildly impaired renal function and initiating the appropriate management to prevent potentially life-threatening arrhythmias.

EP_A020

THYROTOXICOSIS WITH DISCORDANT THYROID FUNCTION TESTS: A RARE PITUITARY TUMOR PRESENTING WITH THYROTOXIC CARDIOMYOPATHY

<https://doi.org/10.15605/jafes.040.S1.028>

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INTRODUCTION

Thyrotoxicosis can lead to life-threatening complications, including thyroid storm and thyrotoxic cardiomyopathy. Discordant thyroid function tests (TFTs) in severe thyrotoxicosis raise suspicion for atypical causes such as assay interference, pituitary pathology, or ectopic thyrotropin (TSH) secretion.

CASE

A 29-year-old male presented with a two-week history of cough, dyspnea, and palpitations. On admission, he was hemodynamically stable but had bibasal fine crepitations, bilateral pedal edema and signs of thyrotoxicosis (agitation, fine tremors, and hyperreflexia). Cardiac monitoring

revealed atrial fibrillation with a heart rate >150 bpm. Thyroid function tests showed discordant TSH 14.92 mIU/L, T4 65.9 pmol/L and T3 13.47 pmol/L levels. He was treated as a case of thyroid storm with thyrotoxic cardiomyopathy.

Echocardiography confirmed heart failure with reduced ejection fraction (20%) and pulmonary artery systolic pressure of 48 mm Hg. Further workup ruled out Group 2, 3, 4 Pulmonary Hypertension (PH) and no invasive right heart catheterization was done. Assay interference was excluded, thyroid autoantibodies were negative and other pituitary hormones were normal. Pituitary MRI showed a large pituitary mass, raising suspicion for the presence of a TSH-secreting pituitary adenoma.

He is currently managed with the anti-thyroid drug methimazole, anti-heart failure medications and anticoagulation by a multidisciplinary team.

TSH-secreting pituitary adenomas (TSHomas) are rare, causing autonomous TSH secretion unresponsive to negative feedback. Unlike resistance to thyroid hormone (RTH), TSHomas typically present with overt hyperthyroidism and tumor-related symptoms (visual defects, headaches, anterior pituitary dysfunction). Atrial fibrillation and heart failure are rare in TSHomas but were prominent in this case. Transsphenoidal surgery is the preferred treatment, resulting to thyroid function normalization in 80% of cases. However, TSHomas often exhibit aggressive invasion, affecting surgical success. If surgery fails, somatostatin analogs (SSAs) can normalize TSH and reduce tumor size.

CONCLUSION

This case highlights the need to consider causes of atypical thyrotoxicosis when TFTs are discordant. Early recognition and a multidisciplinary approach are crucial for managing thyrotoxic cardiomyopathy and its underlying etiology.

EP_A021

ADULT LANGERHANS CELL HISTIOCYTOSIS WITH MULTISYSTEM INVOLVEMENT

<https://doi.org/10.15605/jafes.040.S1.029>

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INTRODUCTION/BACKGROUND

Langerhans cell histiocytosis (LCH) is a rare, heterogeneous disease with a wide range of manifestations, from unifocal lesions to multisystem involvement. Commonly affected

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sites include the bone, lungs, pituitary gland, liver, bone marrow, and skin. We report a case of adult-onset LCH presenting with unifocal bony lesions and hypophysitis.

CASE

A 34-year-old female presented in June 2022 with polyuria and polydipsia for four months. Serum sodium was 148 mmol/L, serum osmolality 309 mOsmol/kg and urine osmolality 62 mOsmol/kg. Water deprivation test confirmed central diabetes insipidus. In September 2022, she complained of amenorrhea for three months but denied headaches, visual disturbances, galactorrhea or significant weight changes. Menstrual cycles were previously regular. She had two children, with her last childbirth two years prior. Anterior pituitary function was consistent with central hypogonadism (LH: 2.1 IU/L, FSH: 5.3 IU/L). Autoimmune screening and tumour markers were negative. Magnetic resonance imaging of the pituitary revealed a thickened pituitary stalk and the absence of bright spot in the posterior pituitary. No biopsy was performed. Hence, she was treated for hypophysitis and given desmopressin as well as estradiol.

Six months later, she developed left shoulder pain. Radiographs revealed a 3.0 x 4.6 x 5.4 cm lytic lesion in the left scapula. She underwent curettage and excision, and histopathological examination confirmed LCH with neoplastic cells expressing CD1a and S100. The patient did not consent for chemotherapy. One year after diagnosis, she developed central hypothyroidism (free T4: 8.2 nmol/L, TSH: 0.12 mU/L), and was started on levothyroxine. During her latest follow-up, she was asymptomatic, with no new lesions on imaging. Cortisol axis remains intact.

CONCLUSION

Adult-onset LCH is rare and biopsy remains the gold standard for diagnosis. This case highlights the diagnostic challenge of distinguishing LCH from other causes of hypophysitis. A comprehensive systemic evaluation is crucial for accurate diagnosis and assessment of disease extent.

EP_A022

GLUCOCORTICOID-INDUCED UNMASKING OF CRANIAL DIABETES INSIPIDUS IN HYPOPHYSITIS: A CASE SERIES

<https://doi.org/10.15605/jafes.040.S1.030>

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INTRODUCTION/BACKGROUND

Hypophysitis is a rare inflammatory condition of the pituitary gland that can mimic other sellar masses. Glucocorticoid (GC) therapy remains the mainstay of treatment, but in some cases may unmask underlying posterior pituitary dysfunction such as cranial diabetes insipidus (DI). We report two distinct cases of hypophysitis in young women in which GC therapy revealed subclinical DI.

CASE

Case 1: A 25-year-old female presented with headache, visual blurring, and polyuria. Magnetic resonance imaging showed pituitary stalk thickening and a sellar mass suggestive of lymphocytic hypophysitis. Initial endocrine evaluation showed isolated hyperprolactinemia. Following high-dose GC therapy, she developed cranial DI confirmed biochemically and treated successfully with desmopressin. Follow-up MRI showed resolution of pituitary swelling.

Case 2: A 22-year-old female with bullous skin lesions and chronic otorrhea was diagnosed with multisystem Langerhans Cell Histiocytosis (LCH). Chemotherapy with vinblastine and high-dose dexamethasone led to diabetic emergencies and the onset of cranial DI. Magnetic resonance imaging revealed infundibular involvement and empty sella. Desmopressin was initiated and doses of steroids and chemotherapy were tapered, resulting to clinical improvement.

These cases highlight the phenomenon of GC-induced unmasking of DI in patients with hypophysitis. Inflammatory edema may initially obscure AVP dysfunction, which becomes apparent only after anti-inflammatory treatment. A high index of suspicion and close monitoring for polyuria following GC initiation are essential. Magnetic resonance imaging remains a valuable diagnostic tool, although radiological differentiation from other pituitary pathologies may be challenging.

CONCLUSION

Glucocorticoid therapy in hypophysitis can unmask subclinical cranial DI. Clinicians should be vigilant for evolving symptoms post-therapy. Early recognition and treatment of DI can significantly improve patient outcomes.

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EP_A023

SPONTANEOUS REMISSION OF GRAVES' DISEASE FOLLOWING SYSTEMIC LUPUS ERYTHEMATOSUS TREATMENT: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.031>

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INTRODUCTION/BACKGROUND

Hyperthyroidism secondary to Graves' disease is typically managed with thionamides, radioiodine therapy, or thyroidectomy. However, spontaneous remission is uncommon, especially after prolonged thionamide therapy. This case highlights a rare instance of hyperthyroidism remission one year after treatment with steroids for systemic lupus erythematosus, despite seven years of prior thionamide use.

CASE

A 25-year-old Malay female with type 1 diabetes mellitus (T1DM) and SLE was diagnosed with Graves' disease at age 15 and treated with carbimazole for seven years. Hyperthyroidism resolved three years before her SLE diagnosis. In 2021, she was diagnosed with Class IV/V lupus nephritis and started on high-dose corticosteroids (methylprednisolone and prednisolone) with cyclophosphamide. One year after initiating steroid therapy, thyroid function tests (TFTs) remained euthyroid without antithyroid medication. Repeat TFTs confirmed continued remission.

Several mechanisms may explain the remission of Graves' disease in this case. High-dose corticosteroids suppress autoreactive B and T lymphocytes, potentially reducing thyrotropin receptor antibody (TRAb) production and facilitating remission. Additionally, corticosteroids enhance regulatory T-cell (Treg) activity, restoring immune tolerance and reducing autoimmunity. The presence of multiple autoimmune diseases suggests a broader dysregulation of immune function, thus immunosuppressive therapy for SLE may have inadvertently suppressed the pathogenic mechanisms driving Graves' disease. Lastly, long-standing autoimmunity can lead to immune exhaustion, where autoreactive immune cells become less active over time, potentially contributing to spontaneous remission.

CONCLUSION

Although corticosteroids are not a conventional treatment for hyperthyroidism, their immunomodulatory effects may inadvertently promote disease remission in select cases. This highlights the need for further research to elucidate the potential role of immunosuppressive therapy in achieving sustained remission of autoimmune hyperthyroidism.

EP_A024

AUTOIMMUNE POLYGLANDULAR SYNDROME TYPE IIIA WITH LUPUS NEPHRITIS: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.032>

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INTRODUCTION/BACKGROUND

Autoimmune polyglandular syndrome (APS) is a rare disorder characterized by multiple autoimmune endocrinopathies. The condition is driven by T-lymphocyte-mediated and autoantibody-induced destruction of various organs. APS Type III is defined by the presence of autoimmune thyroid disease in association with other autoimmune conditions, excluding adrenal insufficiency. APS Type IIIa specifically involves autoimmune thyroid disease and type 1 diabetes mellitus (T1DM). Early recognition and multidisciplinary management are crucial for optimal outcomes.

CASE

A 25-year-old Malay female with T1DM since age seven, inactive Graves' disease, and systemic lupus erythematosus (SLE) with lupus nephritis presented with loose stools (Bristol 7), vomiting, heartburn, bloating, reduced oral intake, and oliguria of eight days duration. Though she was ambulatory, she had a 2-day history of generalized muscle weakness. There was no fever or other indicators of infection. No dietary indiscretion was noted.

Type 1 diabetes mellitus was well-controlled on an insulin regimen. She had hypertension and dyslipidemia since age ten. Graves' disease resolved three years ago after carbimazole treatment. Systemic lupus erythematosus was complicated by class IV/V lupus nephritis, initially treated with corticosteroids and cyclophosphamide, then eventually shifted to mycophenolate mofetil and bisphosphonates as maintenance therapy. Renal function corresponds to CKD stage 3 (eGFR 36 mL/min/1.73 m²).

Examination revealed central obesity, bilateral pitting edema, and striae, without overt dehydration or hyperglycemia. No anemia, acute infection, cardiac failure, or thyroid dysfunction was noted. On the basis of the presence of T1DM, autoimmune thyroid disease, and SLE, she meets APS type IIIa criteria.

CONCLUSION

This case highlights the need for heightened awareness of autoimmune polyglandular syndrome (APS), particularly APS type IIIa in patients presenting with multiple autoimmune endocrinopathies. Clinicians should maintain

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a high index of suspicion for APS in individuals with a history of multiple autoimmune disorders, allowing for early screening and intervention to prevent complications. Comprehensive autoimmune surveillance and interdisciplinary collaboration are essential for optimizing patient outcomes.

EP_A025

BILATERAL ADRENAL HISTOPLASMOSIS IN AN IMMUNOCOMPETENT ELDERLY PATIENT: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.033>

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INTRODUCTION

Histoplasmosis is a fungal infection caused by *Histoplasma capsulatum*, often acquired through inhalation of spores. Disseminated histoplasmosis with adrenal gland involvement is rare and may lead to adrenal insufficiency.

CASE

We report a case of a 70-year-old male, previously healthy, who presented with constitutional symptoms i.e. generalized lethargy, reduced appetite, and significant weight loss of 10 kilograms within 3 months. He was normotensive and no hyperpigmentation was noted. Tumor markers and viral screening were negative and HbA1c was 6.8%. There was no hyponatremia or hyperkalemia. Morning cortisol was 341 nmol/l. Computed tomography scan of the thorax, abdomen, and pelvis revealed bilateral mixed solid cystic adrenal masses and a wedge-shaped hypodense area in the spleen. Adrenal protocol of the CT showed bilateral adrenal masses measuring 5.3 x 3.7 x 3.8 cm on the right and 3.4 x 2.8 x 2.6 cm on the left. A CT-guided adrenal biopsy was performed and histopathological examination revealed highly fragmented tissue strips with huge areas of necrosis and hemorrhage composed of vague formation of epithelioid granuloma with numerous fungal spores. The fungi appearing intra-cytoplasmic in the H&E staining may suggest a histoplasmosis etiology. Synacthen test confirmed adrenal insufficiency with a peak cortisol level of

453 nmol/l. Intravenous amphotericin-B was given for two weeks followed by oral itraconazole 200 mg BD, alongside corticosteroid replacement for adrenal insufficiency. After 6 months, his condition improved significantly, and his adrenal size decreased on follow-up imaging with the largest diameter measuring 3.8 cm. We plan to complete itraconazole for a total of 12 months duration.

CONCLUSION

Bilateral adrenal histoplasmosis usually affects immunocompromised patients or those from endemic areas. This diagnosis should be taken into consideration in a patient presenting with bilateral adrenal masses. Diagnosis is often delayed due to nonspecific symptoms. Imaging, serology, and biopsy are essential for diagnosis. Prompt diagnosis and commencement of antifungal treatment are imperative to prevent adrenal crises.

EP_A026

SECRETIVE SECRETIONS, EXPLOSIVE EXCRETIONS: A RARE CASE OF VIPoma

<https://doi.org/10.15605/jafes.040.S1.034>

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INTRODUCTION/BACKGROUND

Vasoactive Intestinal Peptide (VIP)-secreting neuroendocrine tumours (VIPomas) are rare, usually presenting with profuse watery diarrhea leading to severe electrolyte imbalances. Our patient's initial presentation with obstructive jaundice and portal vein thrombosis preceded the typical presentation, complicating early diagnosis.

CASE

A 34-year-old male presented in May 2024 with progressive jaundice, tea-colored urine and pale stools but no diarrhea. Investigations revealed markedly elevated direct hyperbilirubinemia (205 µmol/L), alkaline phosphatase (742 U/L) and gamma-glutamyl transferase (612 U/L), which was suggestive of biliary obstruction. CT scan identified a 4.2 cm pancreatic mass with hepatic metastases and portal vein thrombosis. EUS visualized a hypoechoic lesion at the head of the pancreas measuring 38 x 35 mm, causing upstream dilatation of the pancreatic and common bile duct. ERCP was done for sphincterectomy and stenting. Histopathology revealed a low-grade neuroendocrine neoplasm.

Six months later, the patient developed persistent watery diarrhea for two weeks, along with fatigue and weight loss. His severe hypokalemia (as low as 1.5 mmol/L) was

Adult E-Poster

refractory despite 3 days of intravenous potassium infusion. Stool studies excluded infective causes, and 24-hour urine 5-HIAA was normal. Trial of subcutaneous octreotide, titrated up to 100 mcg, led to resolution of symptoms and hypokalaemia within a day.

His fasting serum VIP levels sent prior to initiation of therapy were elevated at 211 pg/ml (normal range <86 pg/ml). Gallium-68 DOTATATE showed somatostatin-avid disease at the pancreatic head, multiple abdominopelvic lymph nodes and both liver lobes.

Our multidisciplinary team meeting determined that curative surgery was not feasible due to extensive metastases and vascular involvement. The patient remains well and asymptomatic on octreotide long-acting release during subsequent follow-ups.

CONCLUSION

This case highlights an unusual case of VIPoma, which presented with cholestatic jaundice prior to diarrhea. Strong multidisciplinary collaboration is crucial to optimize outcomes.

EP_A027

DOEGE-POTTER SYNDROME ARISING FROM AGGRESSIVE RECURRENT FIBROUS TUMOUR OF THE LUNG: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.035>

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INTRODUCTION

Doege-Potter Syndrome (DPS) is a rare paraneoplastic syndrome characterized by hypoinsulinemic hypoglycaemia. It typically arises in patients with mesenchymal tumours, particularly fibrous tumours of the lung. DPS is secondary to ectopic secretion of high-molecular-weight insulin-like growth factor (IGF)-2 that induces hypoglycemia.

CASE

We report a 56-year-old male with an underlying solitary fibrous lung tumour that was resected in 2013, which recurred in 2023 and was deemed unresectable. He was to undergo chemotherapy. However, while waiting, he presented neuroglycopenia with a capillary blood glucose of 1.9 mmol/L, reversed with glucose administration. Imaging studies revealed a large pleural-based lesion on the left with an interval increment in size, with its

largest diameter at 20.6 cm and worsening mass effect. Histopathology report from CT-guided biopsy confirmed diagnosis of recurrent fibrous tumour with no malignant features. Renal and liver profiles were normal, and HbA1c was 4.3%. Paired random blood glucose was 2.8 mmol/L, with suppressed C-peptide and insulin levels of 31.81 pmol/L (NR 366.66-1466.65) and <1.39 pmol/L (NR 17.8-173), respectively. Serum IGF-1 was normal at 166.2 ng/ml (NR 54.3-194.2). Serum IGF-2 was 479 ng/ml (NR 333-967), with an IGF-2:IGF-1 ratio of 3, supporting the diagnosis of IGF-2-mediated hypoglycemia. We started him with oral prednisolone 0.5 mg/kg/day, and the hypoglycaemia improved. Unfortunately, he succumbed to respiratory failure due to advanced tumour progression. Given the clinical findings and available biochemical markers, this case is consistent with a diagnosis of non-islet cell tumour hypoglycaemia (NICTH).

CONCLUSION

This case emphasizes the association between solitary fibrous tumours of the lung and DPS, highlighting the potential for recurrence of the tumour and persistent paraneoplastic effects. Early recognition and appropriate management of DPS are crucial in improving patient outcomes, such as the commencement of corticosteroids, while awaiting definitive treatment.

EP_A028

ELDERLY WITH ABSOLUTE INSULIN DEFICIENCY IN A SENIOR CARE FACILITY: A TAILORED APPROACH

<https://doi.org/10.15605/jafes.040.S1.036>

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INTRODUCTION

Managing diabetes in elderly insulin-deficient patients poses significant challenges, particularly when social support is limited.

CASE

We present an elderly female with recurrent diabetic ketoacidosis (DKA) and frequent hypoglycemic episodes. Despite various insulin regimens, she experienced unpredictable glycemic fluctuations, complicated by hypoglycemia unawareness.

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A 65-year-old frail Chinese female with poor social support and underlying medical conditions such as diabetes mellitus (DM), hypertension and dyslipidemia, was first admitted due to loss of consciousness at home. She was diagnosed with DKA secondary to pneumonia. After stabilization, she was transferred back to a district hospital. Despite trials of multiple insulin regimens -basal-bolus, basal insulin plus sulfonylurea and premixed insulin, she continued to experience multiple episodes of hyperglycemia and hypoglycemia. Laboratory findings showed a low/undetectable C-peptide level, confirming insulin deficiency. After discussion with endocrinologists, she was transitioned to a basal-bolus regimen with s/c Toujeo 10 u OM and s/c Novorapid 6 u tds, leading to improved glycemic control but still unpredictable glycemic readings.

Given her planned placement in a nursing home, carbohydrate counting was impractical. Instead, we collaborated with a dietitian and elderly home nursing staff to implement a structured meal-based insulin dosing strategy based on her total daily insulin requirement (0.4 units/kg/day), insulin sensitivity ratio (1 unit: 3 mmol/l), and insulin-to-carbohydrate ratio (1 unit per 14g CHO). This approach significantly stabilized her blood sugar, preventing further hypoglycemia and DKA episodes.

CONCLUSION

This case underscores the complexity of insulin management in elderly patients with insulin deficiency. Rather than carbohydrate counting, a structured meal-based insulin regimen proved to be a viable solution in a nursing home setting, ensuring safe and effective glycemic control.

EP_A029

THE SILENT REMODELER: A CASE OF PAGET'S DISEASE OF THE BONE

<https://doi.org/10.15605/jafes.040.S1.037>

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INTRODUCTION/BACKGROUND

Paget's disease of bone (PDB) is a metabolic bone disorder characterized by excessive osteoclastic activity, leading to abnormal bone remodeling. PDB is often underdiagnosed, particularly among Southeast Asians and individuals under 40 years old. We report a rare case of PDB in a 48-year-old Malaysian woman presenting with severe complications.

CASE

The patient, with a history of hypertension, developed progressive shortness of breath and reduced effort tolerance over a week. She had a five-year history of bilateral hip and knee pain, which led her to change jobs from cashier to babysitter. Over two years, she became increasingly stooped and required a walking stick for support. She also reported bilateral hearing impairment for a week and had unintentional weight loss of 15 kg over two years. There was no family history of bone disorders, trauma, or consanguinity.

On examination, she had frontal bossing, interdental spacing, pectus carinatum, bilateral leg bowing and muscle wasting. Biochemical tests showed markedly elevated alkaline phosphatase (1073 U/L) with normal serum calcium and phosphate levels. Serum parathyroid levels were significantly raised (250 pmol/L). She had acute kidney injury (eGFR 27.5 mL/min/1.73 m²) with metabolic acidosis. Imaging revealed bowing of the femur, diploic widening and cotton wool appearance of the skull, severe kyphoscoliosis with possibility of restrictive lung disease, bilateral staghorn calculi and severe pulmonary hypertension with high output cardiac failure.

Given the late-stage presentation, she was given IV zoledronic acid (3 mg) and analgesics but succumbed to her illness after eight days of hospitalization.

CONCLUSION

This case highlights the need for clinicians to be aware of PDB, especially in rare populations. A high index of suspicion in those with characteristic clinical, biochemical and radiological features is essential, as early diagnosis and treatment can improve quality of life and survival.

Adult E-Poster

EP_A030

HYPERTHYROIDISM WITH SEVERE TRANSAMINITIS IN A PREGNANT FEMALE WITH A TOXIC NODULE

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INTRODUCTION/BACKGROUND.

Hyperthyroidism occurs in 0.1–0.4% of pregnancies, mostly due to Graves' disease. Other causes include toxic adenoma, multinodular goitre and trophoblastic tumours.

CASE

A 27-year-old primigravida at 9 weeks presented with vomiting, epigastric discomfort, anorexia, palpitations and weight loss of 8 kg in one month. Family history was significant for thyroid malignancy. On examination, she was alert but dehydrated, icteric and tachycardic (HR 120 bpm), spiking fever (38°C) with tremors present. No goitre or thyroid eye signs noted. Systemic findings were unremarkable. Investigations revealed TWC: 13 x10⁹/L, Urea: 8 mmol/L, Sodium: 119 mmol/L, TB: 31.5 µmol/L, ALP: 74 U/L, ALT: 943 U/L and AST: 630 U/L. ECG showed sinus tachycardia. She was managed symptomatically with antiemetics and intravenous fluids. However, her liver function worsened; TB: 56 µmol/L, ALP: 69 U/L, ALT: 1583 U/L and AST: 530 U/L. TFT revealed thyrotoxicosis with Free T4 >100 pmol/L and TSH 0.012 mIU/L. Neck ultrasonography showed a homogenous, non-enlarged thyroid gland with a TIRADS 5 thyroid nodule on the right. Normal vascularity was seen within the gland. Hepatic ultrasonography was unremarkable. She was initiated on oral propranolol 60 mg TDS and Lugol's iodine 10 drops TDS for five days. Thyroid autoantibodies, viral hepatitis panel and other second-line investigations for transaminitis were negative. As results improved, she was discharged with tapered Lugol's iodine (5 drops TDS for 5 days) and continued on propranolol. After two weeks, both liver and thyroid function normalised. Fine-needle aspiration cytology of the thyroid nodule revealed atypia of undetermined significance, Bethesda Category III. Postpartum hemi- or total thyroidectomy has been planned as she remains well with a stable pregnancy.

CONCLUSION

Significant transaminitis in early pregnancy mandates a broad differential, including thyroid dysfunction, as early recognition ensures favourable maternal and fetal outcomes. In selected cases, short-term iodine and beta-blockade offer a safe, effective bridge to definitive management.

EP_A031

FLORID ERUPTIVE XANTHOMAS IN A FAMILIAL HYPERTRIGLYCERIDEMIA PATIENT

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INTRODUCTION/BACKGROUND

Eruptive xanthomas can be an early indicator of lipid disorders, occurring in approximately 8.5% of patients with severe hypertriglyceridemia.

CASE

A 27-year-old Malay male, with a history of Type 2 Diabetes Mellitus and Dyslipidemia since age 19, presented for treatment after being lost to follow-up. He reported osmotic symptoms, weight loss and recurrent severe epigastric pain radiating to the back. He is a teetotaler, heavy smoker (13 pack-years), with poor dietary habits and frequent high-fat, high-carbohydrate meals. Family history includes diabetes and dyslipidemia in both parents.

On examination, BMI was 27 kg/m², waist circumference 80 cm and blood pressure 144/91 mm Hg. Crops of 2–5 mm yellow nodules were present over the elbows, buttocks, knees, lateral malleolus, and interphalangeal joints, along with yellowish papules over palms and back, suggestive of eruptive xanthomas. There was no arcus senilis or xanthelasma. Other systemic examinations were unremarkable. Fundoscopy showed no lipemia retinalis or diabetic retinopathy.

Investigations revealed markedly elevated Total Cholesterol (TC) 15.89 mmol/L, Triglycerides (68.44 mmol/L), low HDL (0.61 mmol/L), with invalid LDL values. HbA1c was 10.7%, with mild transaminitis, proteinuria and CKD stage 2 (eGFR 61 mL/min/1.73 m²). Amylase, TSH and other biochemical tests were normal. ECG and echocardiogram were normal. Abdominal imaging revealed fatty liver without chronic pancreatitis.

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He was admitted for triglyceride lowering with IV insulin infusion and started on a low-calorie diabetic diet. Medications included Dapagliflozin 10 mg OD, Metformin 1 g BD, Rosuvastatin 40 mg ON, fenofibrate 145 mg ON and Omega-3 fatty acids 3600 mg/day. At one-month follow-up, lipid levels improved (TC 3.81 mmol/L, TG 8.95 mmol/L, HDL 0.8 mmol/L), though LDL remained invalid. He is planned for PCSK9 inhibitor initiation and was referred to dermatology for xanthomas. Genetic testing is also scheduled.

CONCLUSION

Early recognition and management of severe hypertriglyceridemia is vital to reduce risks of acute pancreatitis as well as long-term cardiovascular complications.

EP_A032

MULTIMODAL MANAGEMENT OF METASTATIC INSULINOMA: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.040>

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INTRODUCTION/BACKGROUND

Pancreatic neuroendocrine tumors (pNETs) are rare, with insulinomas being the most common functional variant. Malignant insulinomas, constituting only about 10% of cases, pose significant management challenges due to refractory hypoglycemia and limited treatment options.

CASE

We present a case of a 58-year-old male with metastatic insulinoma who required a multifaceted approach to control severe, recurrent hypoglycemia. Initial investigations revealed a pancreatic head tumor with liver metastases. Despite medical therapy with diazoxide, octreotide and verapamil, the patient remained dependent on dextrose infusions. Multidisciplinary input guided the initiation of sequential local and systemic therapies, including radio-frequency ablation (RFA), transarterial chemoembolization (TACE), and peptide receptor radionuclide therapy (PRRT). These interventions improved glycemic stability, allowing for eventual weaning off dextrose infusions. He was subsequently initiated on capecitabine and temozolomide for systemic disease control.

Malignant insulinomas necessitate an individualized, multimodal approach. In this case, aggressive local tumor control strategies in combination with systemic therapies successfully mitigated hypoglycemic episodes and

improved the patient's quality of life. This report highlights the importance of early multidisciplinary intervention in optimizing outcomes for metastatic insulinoma patients.

CONCLUSION

Metastatic insulinoma remains a rare but highly morbid entity. A comprehensive, multimodal strategy integrating medical, interventional, and systemic therapies is essential to manage refractory hypoglycemia and tumor progression. This case underscores the need for early referral to specialized centers for optimal patient outcomes.

EP_A033

INITIATION OF CARBIMAZOLE WHEN BASELINE LIVER TRANSAMINASES ARE 3 TO 5 TIMES OF UPPER LIMIT OF NORMAL: A DIRE CLINICAL JUDGEMENT OR AN EVIDENCE-BASED PRACTICE

<https://doi.org/10.15605/jafes.040.S1.041>

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INTRODUCTION

Antithyroid drugs (ATDs) are the first-line treatment options for hyperthyroidism. ATDs are generally avoided when transaminases are >3-5 times the upper limit of normal. We present a case of carbimazole initiation despite transaminitis of almost 5 times the upper limit of normal (ULN).

CASE

A 69-year-old female with underlying hypertension presented with a 2-week history of worsening palpitations, poor oral intake, lethargy and vomiting. Upon review, her vital signs were stable except for a heart rate of 160 beats/min. Physical examination demonstrated warm peripheries and fine tremors. ECG revealed atrial fibrillation. IV Propranolol 1 mg was given, and the rhythm reverted to sinus. Initial blood tests showed overt hyperthyroidism, FT4 >78 pmol/L and suppressed TSH <0.005 uIU/ml. Her baseline transaminases were elevated at ALT 231 U/L (5-49 U/L), AST 162 U/L (4-39 U/L), with normal serum ALP and total bilirubin. Since liver transaminases were raised, ATD was not started, but Lugol's iodine 10 drops thrice daily and Propranolol 40 mg TDS were given. Hepatobiliary ultrasound showed fatty liver disease, while neck ultrasound showed features of Graves' disease. Static ALT readings of 203 U/L and 237 U/L were recorded later. Lugol's iodine was discontinued, and T carbimazole 10 mg

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BD was started. Close monitoring of transaminases was done. One week later, during clinic review, she was well and her ALT improved to 136 U/L with AST 71 U/L, ALP 63 U/L, and total bilirubin level 12 umol/L.

CONCLUSION

According to the American Thyroid Association (ATA), patients with transaminases >5 times the ULN should reconsider before initiating ATDs. However, ATDs can be cautiously trialed in such patients with transaminitis, provided liver function is closely monitored. In such circumstances, methimazole is recommended over PTU due to reduced hepatotoxicity risk.

EP_A034

A MULTIPRONGED APPROACH TO ACHIEVE SIGNIFICANT LDL CHOLESTEROL REDUCTION: A CASE FROM A METABOLIC CLINIC

<https://doi.org/10.15605/jafes.040.S1.042>

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INTRODUCTION/BACKGROUND

Lowering low-density lipoprotein cholesterol (LDL-C) is crucial in reducing cardiovascular disease (CVD) risk, especially in patients with metabolic syndrome and obesity. While statins remain the primary pharmacological intervention, a comprehensive approach incorporating lifestyle changes and adjunctive therapies can yield remarkable results. This case highlights the successful application of a multipronged strategy in a metabolic obesity clinic.

CASE

A 38-year-old Malay female with obesity, type 2 diabetes mellitus (T2DM), dyslipidemia, and fatty liver was followed up for lipid management. Upon her initial visit to the metabolic obesity clinic two years ago, her LDL-C was markedly elevated at 5.7 mmol/L. She was started on atorvastatin 20 mg nightly alongside lifestyle modifications.

To further improve metabolic control, Contrave (naltrexone-bupropion) was introduced initially for weight management but was sequentially switched to Rybelsus (oral semaglutide) over the past year. A structured dietary approach, including a low-calorie diet with reduced refined carbohydrates and increased fiber intake, was implemented along with gradual exercise initiation.

Over two years, her LDL-C dropped dramatically from 5.7 mmol/L to 1.6 mmol/L. Concurrently, triglycerides improved, HDL-C increased, and her HbA1c decreased from 7.2% to 5.6%. She also achieved clinically significant weight loss, from 91 kg to 86 kg. This comprehensive intervention led to substantial cardiometabolic benefits.

CONCLUSION

This case demonstrates that a multipronged approach integrating statins, novel glucose-lowering agents and lifestyle modifications can achieve exceptional LDL-C reduction and broader metabolic improvements. Clinicians should consider a patient-centered, holistic strategy to optimize lipid control and long-term cardiovascular outcomes.

EP_A035

FAHR'S SYNDROME SECONDARY TO NON-SYNDROMIC PRIMARY HYPOPARATHYROIDISM

<https://doi.org/10.15605/jafes.040.S1.043>

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INTRODUCTION/BACKGROUND

Fahr's syndrome is a rare neurological disorder characterized by abnormal calcium deposits in the brain, particularly in the basal ganglia. The aetiology can be primary or secondary, with endocrinopathies being the most common cause. We report a case of Fahr's syndrome in which the patient developed seizures and ECG changes due to severe hypocalcemia.

CASE

A 29-year-old female with underlying type 2 diabetes, psoriasis, and cognitive delays presented with an episode of generalized tonic-clonic seizure along with perioral numbness, skin redness and peeling for one week. Medical records showed her corrected calcium was less than 1.9 mmol/L for over a decade. There was no history of neck surgery or radiation, nor similar conditions in her family. She had no dysmorphic features but was septic with a capillary glucose of 29.5 mmol/L. ECG revealed prolonged QT interval of 516 Msec. Laboratory results showed profound hypocalcemia of 1.28 mmol/L, hypomagnesemia

Adult E-Poster

of 0.65 mmol/L and normal phosphate level. Intact PTH and 25-hydroxy-vitamin D levels were low, at 0.485 pmol/L and 24.5 nmol/L, respectively. Her brain CT scan showed cerebral atrophy with extensive intracranial calcifications, features which were consistent with Fahr's syndrome. Other evaluations did not suggest infiltrative or autoimmune disorders. There was no cataract or nephrolithiasis as a result of prolonged hypocalcemia. A multidisciplinary team managed her in the ICU with a diagnosis of severe sepsis secondary to erythrodermic psoriasis with superimposed bacterial infection. One week later, she was discharged well with calcium carbonate 1 gram thrice daily and calcitriol 0.5 mcg twice daily. No genetic test was performed due to financial constraints.

CONCLUSION

This case underscores the importance of timely diagnosis of primary hypoparathyroidism to prevent long-term complications. There are no established guidelines for the radiological surveillance intervals in Fahr's syndrome, and individualized management remains crucial in caring for patients with this condition.

EP_A036

PEMBROLIZUMAB INDUCED DIABETES MELITUS IN AN ELDERLY WOMEN WITH NON-SMALL CELL LUNG CANCER

<https://doi.org/10.15605/jafes.040.S1.044>

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INTRODUCTION/BACKGROUND

Immune checkpoint inhibitor (ICI)-induced diabetes mellitus is rare, with an incidence of 0.9 to 2%. As ICI usage increases, awareness of associated endocrinopathies, particularly diabetes, is crucial.

CASE

We describe a rare case of a 72-year-old non-diabetic female with NSCLC (non-small cell lung carcinoma) who presented with diabetic ketoacidosis after initiation of an immune checkpoint inhibitor.

Diagnosed with advanced NSCLC in 2023, she enrolled in a clinical trial and received a three-weekly regimen which included Pembrolizumab. She completed three cycles without major side effects, with fasting blood glucose between 5–6 mmol/L.

During her fourth trial visit, she complained of lethargy, with a glucometer reading of 28 mmol/L. Further testing

indicated diabetic ketoacidosis. She was hospitalized and started on the standard DKA fluid and insulin regimen. She was phenotypically lean, with no evidence of insulin resistance, and HbA1c taken at the time was 6.9%, indicating the glucose spike to be recent. Controlling her glucose levels in the ward was challenging. Eventually, despite resolution of DKA, she required high insulin doses (>1 u/kg/day) upon discharge.

Blood investigation at the time did not show evidence of other endocrinopathies, renal or liver impairment, and pancreatic enzymes were not significantly elevated. Her insulin autoantibody tests (ICA/anti-GAD/IAA) were negative. However, her C-peptide levels were markedly depleted at <6.67 pmol/L, indicating absolute endogenous insulin deficiency. After three cycles of ICI, repeated scans showed progression of her disease, and she was eventually withdrawn from the clinical trial. Her diabetes persisted despite cessation of her immunotherapy, requiring lifelong insulin.

CONCLUSION

The onset of ICI-induced diabetes here aligns with the reported median presentation times. Anti-PD-1 immune events are not contraindications and correlate with better progression-free survival. However, insulin therapy is often lifelong, highlighting the importance of early detection, prompt insulin initiation and regular endocrinopathy monitoring in affected patients.

EP_A037

PEMBROLIZUMAB-INDUCED HYPOPHYSITIS WITH CENTRAL DIABETES INSIPIDUS: A RARE IMMUNE-MEDIATED ADVERSE EVENT

<https://doi.org/10.15605/jafes.040.S1.045>

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INTRODUCTION/BACKGROUND

With the growing use of immune checkpoint inhibitors, hypophysitis is gaining increased clinical recognition while remaining a formidable diagnostic and therapeutic challenge. Pembrolizumab, a PD-1 inhibitor, is a breakthrough therapy that enhances the immune system's attack on tumours but comes with the risk of immune-related adverse events.

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CASE

We report the case of a 65-year-old male undergoing pembrolizumab treatment for renal cell carcinoma who presented with profound lethargy 18 months after treatment initiation. Hormonal evaluation upon admission revealed panhypopituitarism, characterized by critically low random cortisol (<14 nmol/L), ACTH deficiency (<5 pg/mL), and biochemical findings suggestive of secondary hypothyroidism (TSH: 1.68 mIU/L [0.55–4.78], free T4: 10.7 pmol/L [11.5–22.7]). The gonadal function was preserved (testosterone: 21.5 nmol/L; LH: 5.5 IU/L [1.5–9.3]; FSH: 15.8 IU/L [1.4–18.1]), while prolactin levels were mildly elevated (315 mIU/L). The autoimmune screening was ANF positive but only with titre 1:80, normal anti-dsDNA, and normal C3C4 and tumour markers were unremarkable.

The patient was promptly initiated on intravenous hydrocortisone, followed by a tapering regimen of oral hydrocortisone and thyroxine replacement. Shortly after glucocorticoid initiation, he developed polyuria and polydipsia. Further evaluation confirmed cranial diabetes insipidus (DI), with low urine osmolality (101 mOsm/kg) and elevated serum osmolality (287 mOsm/kg). Subcutaneous desmopressin was initiated, leading to rapid symptom resolution and stabilization. A pituitary MRI showed no evidence of adenoma or stalk enlargement. Although pembrolizumab-induced hypophysitis is a known immune-related adverse event, arginine vasopressin (AVP) deficiency remains a rare complication of checkpoint inhibitor therapy.

CONCLUSION

This case highlights the spectrum of pembrolizumab-induced hypophysitis, which can manifest as panhypopituitarism and, in rare cases, cranial diabetes insipidus. Clinicians should maintain a high index of suspicion for hypophysitis in patients with new-onset fatigue post-ICI therapy, as timely hormonal replacement is crucial in preventing life-threatening adrenal insufficiency and associated complications.

EP_A038

DIABETIC MASTOPATHY IN A PATIENT WITH TYPE 1 DIABETES MELLITUS

<https://doi.org/10.15605/jafes.040.S1.046>

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INTRODUCTION/BACKGROUND

Diabetic mastopathy is a rare fibroinflammatory condition that predominantly affects long-standing type 1 diabetes mellitus. It commonly presents as firm and painless breast masses, mimicking malignancy. The diagnosis is often based on clinical evaluation, imaging studies and pathological correlation. While the exact pathophysiology remains unclear, it is hypothesized to involve an autoimmune mechanism, leading to lymphocytic infiltration and stromal fibrosis in the breast tissue.

CASE

We present the case of a 30-year-old primigravid at 16 weeks of gestation, with a background of poorly controlled long-standing type 1 diabetes mellitus complicated by diabetic nephropathy and retinopathy. She presented with a painless lump in her left breast. Clinical examination found a 3 × 2 cm mass in the upper outer quadrant of the left breast, which was firm, mobile and non-tender. There were no overlying skin changes. Breast ultrasound revealed multiple irregular hypoechoic masses with pronounced posterior shadowing. Histopathological examination (HPE) of the mass showed dense stromal keloidal type fibrosis with moderate lymphocytic infiltration around the periductal, peri-lobular and perivascular regions. The diagnosis of diabetic mastopathy was made, and reassurance was given to the patient.

CONCLUSION

Diabetic mastopathy is a benign self-limiting breast lesion with a high risk of recurrence after surgical intervention, hence it generally does not require treatment. The awareness of this rare condition may avoid unnecessary surgical intervention, mental distress, as well as diagnostic uncertainty.

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EP_A039

UTILITY OF BRONCHOSCOPIC INTRA-TUMORAL ALCOHOL INJECTION (ITAI) IN MEDIASTINAL PARAGANGLIOMA: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.047>

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INTRODUCTION/BACKGROUND

ITAI is an emerging bronchoscopic modality to restore airway patency in malignant airway obstruction, particularly in primary lung cancers and metastatic thoracic diseases. Its use in mediastinal paragangliomas, however, remains under-researched. Mediastinal paragangliomas present unique challenges for complete resection due to their high vascularity and proximity to critical structures.

CASE

A 35-year-old female presented to the respiratory clinic with chronic cough, hemoptysis and dyspnea. Her past surgical history included a right adrenalectomy done 15 years ago for pheochromocytoma. She was in biochemical remission up to 3 years post-operatively, then defaulted. Recently, she also experienced paroxysmal symptoms of headache, palpitation and diaphoresis, coinciding with new-onset hypertension. CT thorax revealed a middle mediastinal mass compressing the right bronchus intermedius and right lower lobe bronchus, causing segmental lung collapse. Her 24-hour urine metanephrines were 26 times elevated for normetanephrines at 58950 nmol/L (606-2287), and endobronchial biopsy confirmed paraganglioma. Multi-disciplinary team discussions concluded that complete surgical resection was impossible, and multimodal therapies including systemic, radionuclide and local therapies, were indicated for disease control. Bronchoscopic interventions using ITAI combined with cryoablation, argon plasma coagulation and balloon dilation were performed in two separate sessions, following at least 10 days of preprocedural alpha blockade. The patient underwent the procedure without experiencing a catecholamine crisis but required a pint of packed red blood cell transfusion due to a drop in hemoglobin levels from post-procedural hemoptysis. Subsequent chest radiographs demonstrated improvement with better symptom control.

CONCLUSION

ITAI is a promising adjunctive debulking modality for managing mediastinal paragangliomas, particularly in alleviating disabling obstructive symptoms caused by endobronchial obstruction. ITAI induces tumor cell necrosis and microcirculatory embolization, thereby inhibiting tumor growth and reducing blood supply to the treated area. It may also serve as a bridge to systemic, radionuclide, or targeted therapies in cases where surgical resection is not feasible.

EP_A040

YOUNG-ONSET DIABETES MELLITUS: A DIAGNOSTIC AND MANAGEMENT DILEMMA

<https://doi.org/10.15605/jafes.040.S1.048>

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INTRODUCTION/BACKGROUND

Type 1 diabetes mellitus is typically characterized by severe insulin deficiency with diabetes-related autoantibodies, though up to 5% of patients may have insulin deficiency without these autoantibodies. Diagnosing and managing diabetes in young adolescents can be particularly challenging. We present a case of a 12-year-old male diagnosed with diabetes after presenting with diabetic ketoacidosis (DKA), facing significant management challenges.

CASE

A 12-year-old male presented with severe DKA requiring ventilatory support in December 2021. His parents had no history of diabetes, but two older siblings had type 2 diabetes. After successful DKA management, he was discharged with basal-bolus insulin therapy. His HbA1c was 15%, and investigations revealed absent autoantibodies and a low C-peptide level (<3.33pmol/L), suggesting insulin deficiency. Despite treatment, he experienced frequent nocturnal and postprandial hypoglycaemic episodes, as confirmed by continuous glucose monitoring (CGM). Reducing insulin doses did not resolve the hypoglycaemia. His condition was further complicated by non-compliance due to peer pressure and reluctance to take insulin while attending boarding school.

A trial of basal insulin combined with sulfonylurea and close glucose monitoring resulted in over six months of stable glucose levels without hypoglycaemia. However, after a subsequent hospitalization for *Klebsiella* bacteraemia and uncontrolled glucose, basal-bolus therapy was reinstated in

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October 2024. During this hospitalization, C-peptide levels increased to 287 pmol/L. Given his ongoing difficulties with insulin compliance, a new trial with basal insulin and a DPP-IV inhibitor was initiated.

CONCLUSION

The initial diagnosis of Type 1B diabetes was suggested by low C peptide and negative autoantibodies. Persistent hypoglycaemia despite low insulin doses and the challenge of non-compliance led to consideration of MODY variants, prompting trials with oral glucose-lowering drugs. Further genetic studies are needed for a definitive diagnosis.

EP_A041

FASTING, FEASTING, AND FALLING GLUCOSE: A CASE OF NON-INSULINOMA PANCREATOGENOUS HYPOGLYCEMIA SYNDROME FOLLOWING WEIGHT LOSS AND KETOGENIC DIET DISCONTINUATION

<https://doi.org/10.15605/jafes.040.S1.049>

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INTRODUCTION/BACKGROUND

Non-insulinoma pancreatogenous hypoglycemia syndrome (NIPHS) is a rare cause of endogenous hyperinsulinemic hypoglycemia, distinct from insulinoma, often linked to β -cell dysfunction after bariatric surgery in adults.

CASE

This case report describes a previously well 31-year-old male with recurrent hypoglycemia symptoms following intentional weight loss of 44 kg (120 to 76 kg) from practising ketogenic diet for one year. The patient experienced recurrent episodes of giddiness, palpitations and syncope, with lowest capillary blood glucose levels documented as 1.8 mmol/L. These symptoms emerged after resuming a regular carbohydrate diet, after he developed severe constipation with ketogenic diet. The episodes were erratic but reported to be more common after prolonged fasting. Initial evaluation revealed a normal HbA1c (4.7%) with normal hemoglobin (16.5 g/dL), normal morning cortisol and renal and hepatic functions. A supervised 72-hour fast demonstrated symptomatic venous hypoglycemia (2.7 mmol/L) at the 36th hour with inappropriately elevated insulin (5.67 μ IU/mL) and C-peptide (496 pmol/L) levels, confirming endogenous hyperinsulinemia. Prolonged oral glucose tolerance testing (OGTT) revealed exaggerated insulin secretion (peak insulin: 43.92 mIU/L at 1 hour; glucose: 8.1 mmol/L), followed by non-suppression of

insulin (5.65 mIU/L) and C-peptide (1244 pmol/L) at 4 hours despite low blood glucose of 3.5 mmol/L. Pancreatic CT was normal, supporting a diagnosis of NIPHS. Patient's symptoms improved following small frequent meals and avoidance of large amounts of simple carbohydrates.

CONCLUSION

This case highlights NIPHS as a consequence of altered β -cell function following prolonged ketogenic diet, likely exacerbated by rapid dietary carbohydrate reintroduction.

EP_A042

BILATERAL OSTEOPOROTIC FEMUR FRACTURES IN A YOUNG WOMAN: AN AFTERMATH OF EMPTY SELLAR SYNDROME

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INTRODUCTION/BACKGROUND

Empty sella syndrome (ESS) is characterized by the radiological finding of a flattened pituitary gland within the empty sella turcica due to subarachnoid space expansion, commonly associated with hormonal deficiencies. We report a rare case of panhypopituitarism due to primary ESS in a young female who presented with bilateral osteoporotic femur fractures.

CASE

A 38-year-old aboriginal female was first brought to the orthopedic team for persistent right hip pain and a limping gait for several years. There was no prior history of trauma, surgery or irradiation. CT of bilateral hip joints showed generalized osteopenia with non-union bilateral femur fracture. Bone mineral density revealed osteoporosis of the lumbar spine and left radius with Z-score of -4.3 and -6.6, respectively. Further evaluation for secondary osteoporosis revealed short stature with a low BMI of 17 kg/m². Notably, she has primary amenorrhea and delayed puberty, with Tanner stage 1 breast and pubic hair development. Family history was unremarkable except for one younger sister with short stature. Anterior pituitary hormone profile revealed central hypothyroidism (TSH: 2.05 mIU/L, fT4: 6.7 pmol/L), hypogonadotrophic hypogonadism (serum estradiol <5.1 pmol/L, FSH: 0.3 IU/L, LH <0.2 IU/L), low

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prolactin (35.7 mIU/L) and low IGF-1 level of 11.9 ng/ml (69 -227 ng/ml) suggestive of growth hormone deficiency. Additionally, her vitamin D level was insufficient, 68.79 nmol/L. Short Synacthen test revealed adequate cortisol response. MRI of pituitary reported features of empty sella, confirming the diagnosis of primary ESS. She received hormonal replacement therapy, including estradiol, for pubertal induction. She was counselled for right total hip replacement, but she was not keen.

CONCLUSION

Panhypopituitarism observed in ESS affects bone remodeling, leading to early osteoporotic fracture. Treatment with hormonal replacement is essential to restore secondary sexual characteristics for psychosocial well-being as well as to improve bone health to reduce the risk of further fracture.

EP_A043

RACING HEART UNDER STORMY SKIES: A JOURNEY THROUGH AGRANULOCYTOSIS TO THYROID STORM

<https://doi.org/10.15605/jafes.040.S1.051>

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INTRODUCTION/BACKGROUND

Thyroid storm is a rare but life-threatening exacerbation of thyrotoxicosis characterised by multi-system dysfunction. Its shared features with sepsis may pose a significant diagnostic challenge. We describe a patient with Graves' disease who developed carbimazole-induced agranulocytosis followed by thyroid storm necessitating therapeutic plasma exchange (TPE) and urgent thyroidectomy.

CASE

A 31-year-old female with newly diagnosed Graves' disease on carbimazole 40 mg daily presented with palpitations and right hypochondriac pain. Initial investigation showed elevated FT4 at 31 pmol/L (7.86-14.41 pmol/L), suppressed TSH at 0.03 uIU/mL (0.38-5.33 uIU/mL) with cholestatic transaminitis. On day 3 of admission, she developed agranulocytosis with an absolute neutrophil count (ANC) of $0.06 \times 10^9/L$, hence carbimazole was withheld. Granulocyte-colony stimulating factor was initiated along with a broad-spectrum antibiotic to cover for neutropenic sepsis. The ANC normalised after three days, but she developed spiking fever up to 40.8°C, associated with persistent vomiting followed by hypotension and tachycardia.

Biochemical tests revealed rising FT4 to 44.5 pmol/L and hyperbilirubinemia. Initiation of glucocorticoids upon withholding carbimazole was delayed due to concern of sepsis. Diagnosis of thyroid storm was made and urgent TPE was initiated along with high-dose intravenous glucocorticoids and esmolol infusion. She showed immediate clinical improvement with defervescence and stabilization of hemodynamic parameters after the first cycle of TPE. Total thyroidectomy was performed after two cycles of TPE, and she was discharged well on day 30.

CONCLUSION

This case highlights the challenge of distinguishing thyroid storm from sepsis in the setting of carbimazole-induced agranulocytosis. It underscores the importance of prompt recognition and timely intervention of thyroid storms to prevent morbidity and mortality. Early initiation of TPE as bridging therapy before definitive therapy in the setting where antithyroid drug was contraindicated provided rapid control of thyrotoxicosis and was well tolerated.

EP_A044

THROUGH THE EYES OF LUPUS: LIPAEMIA RETINALIS AS A RARE OCULAR MANIFESTATION OF HYPERTRIGLYCERIDEMIA

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INTRODUCTION/BACKGROUND

Lipemia retinalis is a rare but striking ocular finding caused by extreme hypertriglyceridemia. It is typically associated with primary dyslipidemias but may also occur secondary to autoimmune disease such as systemic lupus erythematosus (SLE). We describe a case of newly diagnosed SLE with lupus nephritis, incidentally found to have lipemia retinalis, leading to the diagnosis of severe hypertriglyceridemia.

METHODOLOGY

A 12-year-old female presented with two months of intermittent fever and constitutional symptoms. Investigations revealed normochromic normocytic anemia, raised inflammatory markers, positive ANA (speckled pattern) and anti-dsDNA with low complement levels. Proteinuria was present, and subsequent renal biopsy confirmed class III lupus nephritis. Fundoscopy revealed an incidental finding of lipemia retinalis, and lipid profile showed severe

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hypertriglyceridemia with a serum triglyceride (TG) of 11.7 mmol/L. Other secondary causes of hypertriglyceridemia, including hyperglycemia, hypothyroidism, alcohol or dietary fat, were excluded from biochemistry and clinical history. The lipid profiles of first-degree relatives were unremarkable.

She was started on high-dose omega-3 fish oil, a very low-carbohydrate diet and fenofibrate 145 mg every other day along with immunosuppression therapy for SLE. Her serum TG dropped markedly to 4.1 mmol/L within 3 weeks. However, she had transient bradycardia leading to temporary cessation of fenofibrate and hydroxychloroquine, and her serum TG rebounded to 16.5 mmol/L. After ruling out other causes of bradycardia, fenofibrate was resumed without recurrence of bradycardia, followed by normalization of the TG level. Fundoscopic examination two months later showed resolution of lipaemic retinalis. She completed six cycles of cyclophosphamide with steroid tapering, and her serum TG remained normal at 0.5 mmol/L.

CONCLUSION

This case highlights lipaemia retinalis secondary to severe hypertriglyceridemia as a rare manifestation in newly diagnosed SLE. Early recognition, aggressive lipid-lowering therapy, along with immunosuppressive treatment for the underlying SLE led to rapid triglyceride reduction with complete resolution of lipemia retinalis.

EP_A045

WEIGHT REBOUND POST GLP-1 RA CESSATION: THE IMPORTANCE OF GRADUAL TAPERING AND PATIENT EDUCATION

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INTRODUCTION/BACKGROUND

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have demonstrated significant efficacy in weight management. However, abrupt discontinuation often leads to an uncontrollable appetite rebound and subsequent weight regain. This phenomenon underscores the need for structured tapering protocols and comprehensive patient education to ensure sustainable weight management post-therapy cessation.

CASE

A 54-year-old female with obesity (BMI 33.8) was initiated on subcutaneous Saxenda (liraglutide) for weight management. She successfully escalated to a 3 mg daily dose, tolerating mild gastrointestinal side effects. Despite an initial weight reduction (94.5 kg >92.4 kg), she discontinued treatment due to injection site reactions. Three months post-discontinuation, her weight increased to 96.7 kg (BMI 35.95) with increased appetite and dietary non-compliance. Upon restarting therapy, a gradual dose escalation was advised to minimize adverse effects and improve adherence. The patient also received structured education on medication tapering, dietary modifications, and lifestyle interventions.

CONCLUSION

This case highlights the challenges of abrupt GLP-1 RA discontinuation and the subsequent weight rebound. A strategic tapering plan is essential to mitigate appetite dysregulation and sustain weight loss. Moreover, patient education on the physiological impact of cessation, proper injection techniques, and behavioral strategies is crucial in optimizing long-term obesity management outcomes. Health practitioners must emphasize these aspects to ensure adherence and enhance treatment success.

EP_A046

FROM PANIC DISORDER TO CARCINOID SYNDROME IN AN EXPECTING MOTHER

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INTRODUCTION/BACKGROUND

Carcinoid syndrome occurs in ~10% of neuroendocrine tumours (NET). It indicates advanced disease with liver metastasis associated with lower survival. However, it is often misdiagnosed as other gastrointestinal, respiratory or dermatologic conditions, with a median delay in diagnosis of 3.4 years because of its rarity.

CASE

We present a case of a 32-year-old female at 10 weeks gestation presenting with abdominal distension. Physical examination revealed hepatomegaly and a pansystolic murmur. Ultrasound showed an enlarged liver with multiple solid lesions. Liver biopsy confirmed a well-differentiated grade 2 NET. Further history revealed a 2-year history of progressive facial flushing and diarrhoea that had been diagnosed as panic attacks. Endoscopic ultrasound showed

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a 2.3 cm pancreatic mass. Echocardiography showed moderate tricuspid regurgitation with an enlarged right atrium. A provisional diagnosis of carcinoid syndrome secondary to pancreatic NET with liver metastases was made. A multidisciplinary team decision was made to terminate the pregnancy to allow further evaluation and treatment. Computed tomography of the thorax, abdomen and pelvis confirmed the pancreatic head lesion with liver metastasis. Biochemistry showed elevated 24-hour urinary 5-hydroxyindoleacetic acid and serum chromogranin A, confirming the diagnosis of carcinoid syndrome. Histopathology of the biopsied pancreatic mass was consistent with grade 2 NET with a Ki-67 of 3-4%. Gallium-68 DOTATE and fluorodeoxyglucose positron emission tomography demonstrated concordant disease involving the pancreatic head, liver, lymph nodes and bone. The tumour was deemed inoperable and the patient was commenced on somatostatin analogue, followed by peptide receptor radionuclide therapy given the predominant Gallium-68 DOTATE-avid disease.

CONCLUSION

This case highlights the delay in diagnosis of carcinoid syndrome due to the lack of awareness of NET, leading to a heavy, inoperable tumor burden with guarded prognosis. A concerted effort is required to educate all healthcare providers on NET to minimise delay in diagnosis and improve patient outcomes.

EP_A047

HABBATUS SAUDA OIL-INDUCED SEVERE HYPERTRIGLYCERIDEMIA IN A PATIENT WITH DIABETES MELLITUS

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INTRODUCTION/BACKGROUND

Hypertriglyceridemia (HTG) is a significant risk factor for cardiovascular disease, pancreatitis, and metabolic complications, particularly in patients with multiple comorbidities. While herbal supplements like Habbatus Sauda (*Nigella sativa*) oil are often used for their purported health benefits, emerging reports suggest potential adverse metabolic effects. This case highlights severe HTG potentially triggered by Habbatus Sauda oil in a patient with T2DM, dyslipidaemia and psoriatic arthritis (PsoA).

CASE

A 50-year-old Malay male, a research officer, presented with poorly controlled diabetes (HbA1c: 11%), dyslipidemia and severe HTG. He had a five- to six-year history of T2DM, previously on oral hypoglycemic agents (OHA) but discontinued, along with hypertension and PsoA managed with methotrexate (MTX). His triglyceride (TG) levels fluctuated significantly (2.1 → 13 → 8 mmol/L), with worsening levels temporally associated with the consumption of Habbatus Sauda oil. No other dietary or medication changes could fully explain the lipid surge.

The patient was advised to discontinue Habbatus Sauda oil and implement strict lifestyle modifications. Pharmacological interventions included metformin XR, gliclazide MR, vildagliptin, dapagliflozin (self-purchased), fenofibrate and atorvastatin. Despite adherence challenges, TG levels improved from 13 mmol/L to 8 mmol/L following supplement cessation and medication optimization. Hyperkalemia (K: 6.0 mmol/L) was incidentally detected, requiring urgent potassium-lowering therapy. The patient remained resistant to injectable anti-diabetic therapy and exhibited inconsistent compliance with diet and medications.

CONCLUSION

This case highlights a potential link between Habbatus Sauda oil and worsening hypertriglyceridemia, underscoring the need for vigilance in patients with pre-existing metabolic disorders. While herbal supplements are widely perceived as beneficial, they may have unintended metabolic consequences, particularly in high-risk individuals. Clinicians should actively inquire about supplement use when evaluating unexplained dyslipidemia and provide comprehensive patient education on adherence and supplement safety. A multidisciplinary approach is essential to optimizing long-term cardiovascular and metabolic outcomes.

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EP_A048

HIDDEN IN PLAIN SIGHT: MULTIFOCAL PARAGANGLIOMA IN AN ADOLESCENT WITH HYPERTENSION

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INTRODUCTION/BACKGROUND

Pheochromocytomas and paragangliomas (PPGLs) are rare neuroendocrine tumors. The high incidence of multifocality, recurrence and metastatic disease complicates the management of paraganglioma in adolescents.

CASE

A previously healthy 14-year-old male presented with a one-month history of occipital headache associated with blurring of vision. He had no chest pain, dyspnoea, diaphoresis or syncope. There is no family history of hypertension in the young. His mother has hyperthyroidism.

On arrival, his blood pressure was 242/167 mm Hg, heart rate was 127 bpm, and SpO₂ was 100% on room air. His capillary blood glucose was normal at 4.8 mmol/L. Physical examination showed no signs of goitre, cushingoid features, or acromegalic traits. The patient is overweight, with a BMI of 25.6 kg/m². He showed no stigmata of neurofibromatosis. Fundoscopy showed bilateral optic disc swelling and macular edema, consistent with grade IV hypertensive retinopathy.

Electrocardiogram revealed sinus tachycardia with T-wave inversion in lead V2-V6. Laboratory investigations, including complete blood count, calcium and renal profile, were unremarkable. His endocrine workup confirmed a diagnosis of pheochromocytoma with elevated 24-hour urinary normetanephrine (90.75 umol/day, 36.5x ULN) and 3-methoxytyramine (4.02 umol/day, 2.8x ULN).

Adrenal CT imaging revealed a large, lobulated, heterogeneously enhancing mass measuring 5.0 × 6.1 × 5.4 cm (AP × W × CC) along the left margin of the abdominal aorta. The bilateral adrenal glands are normal.

Neck and Thorax CT showed a well-defined, round, homogeneously enhancing lesion at the base of the skull measuring 1.0 × 1.1 × 1.4 cm (AP × W × CC). Therefore, he was diagnosed with paraganglioma.

His blood pressure is currently controlled with three anti-hypertensive medications, including an alpha blocker. Given his multifocal disease, germline genetic testing is warranted, and functional imaging should be considered preoperatively to exclude metastasis. He was referred to an endocrine center for further management.

CONCLUSION

Pediatric PPGLs are more often extra-adrenal, multifocal/metastatic, and recurrent, likely due to a stronger genetic predisposition. Hence, timely diagnosis is crucial to prevent morbidity and mortality.

EP_A049

INSULIN AUTOIMMUNE SYNDROME OR INSULINOMA? UNRAVELLING THE CAUSE OF HYPERINSULINEMIC HYPOGLYCEMIA IN A PATIENT WITH A PANCREATIC CYST

<https://doi.org/10.15605/jafes.040.S1.057>

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INTRODUCTION/BACKGROUND

Insulin Autoimmune Syndrome (IAS) constitutes a rare aetiology of non-diabetic endogenous hyperinsulinemic hypoglycaemia, with a prevalence of 4.9–11.7%. We report a case of a 61-year-old Chinese female who was confirmed to have endogenous hyperinsulinemic hypoglycaemia. Subsequent imaging revealed a cystic pancreatic lesion, while insulin autoimmune antibodies (IAA) were mildly elevated. This case highlights the challenge of distinguishing between insulinoma and IAS.

CASE

A 61-year-old Chinese female, with no prior diabetes, presented in April 2024 with symptoms suggestive of Whipple's triad. She experienced both fasting and postprandial hypoglycemia (2.0–3.0 mmol/L) and postprandial hyperglycemia (up to 16 mmol/L). She had no significant drug history except recent glucosamine use two weeks prior. A 72-hour fasting test confirmed endogenous hyperinsulinemia with elevated insulin (245 U/mL) and C-peptide (13.3 ng/mL) at a plasma glucose of 2.6 mmol/L, with a molar insulin-to-C-peptide ratio of 0.4. Sulfonylurea screening was negative, but IAA was mildly elevated at 17.9 U/mL (<2.4).

Pancreatic CT scan revealed a 9 mm non-enhancing hypodense lesion in the pancreatic body, and endoscopic evaluation found a 7×5 mm pancreatic cyst. Ga68DOTATATE

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PET-CT showed no uptake, and arterial stimulation venous sampling (ASVS) was negative for insulinoma or nesidioblastosis. PEG precipitation of random insulin indicated low insulin recovery (7.5%), confirming IAA interference. She was started on diazoxide and a low-glycemic diet with small frequent meals. Her hypoglycaemia spontaneously resolved within three months, even after stopping diazoxide prior to ASVS. Given the negative ASVS, positive IAA, and spontaneous resolution, IAS was diagnosed. She was commenced on acarbose 50 mg TDS and remains well, with continuous glucose monitoring showing infrequent hypoglycemia and milder postprandial hyperglycemia.

CONCLUSION

This case underscores the diagnostic complexity of differentiating IAS from cystic insulinoma in a patient with endogenous hyperinsulinemic hypoglycemia, a cystic pancreatic lesion, and elevated IAA.

EP_A050

MINIMALLY INVASIVE MANAGEMENT OF PARATHYROID ADENOMA: A CASE OF SUCCESSFUL THERMAL ABLATION IN A HIGH-RISK ELDERLY PATIENT

<https://doi.org/10.15605/jafes.040.S1.058>

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INTRODUCTION/BACKGROUND

Primary hyperparathyroidism (PHPT) due to parathyroid adenoma often requires parathyroidectomy. However, surgery may not be feasible in high-risk patients. Thermal ablation techniques, such as microwave ablation (MWA), offer a minimally invasive alternative. We present a case of an elderly woman who was successfully treated with microwave ablation for severe hypercalcemia caused by a parathyroid adenoma.

METHODOLOGY

An 81-year-old semi-dependent female was diagnosed with parathyroid hormone (PTH)-dependent hypercalcemia in 2020 with a calcium level at 3.4 mmol/L (2.2-2.6) and an iPTH level at 69.7 pmol/L (1.96-8.49). Parathyroid ultrasound and 99m Tc MIBI parathyroid scintigraphy confirmed the presence of a right upper pole parathyroid adenoma, measuring 1.7 x 1.1 x 2.4 cm. Preoperative evaluation revealed an ectatic ascending thoracic aorta and

aortic arch, causing tracheal deviation and restrictive lung disease, which placed her at high surgical risk.

Initially, she was managed conservatively with cinacalcet 25-50 mg bd and denosumab 30-60 mg every 3-6 months. Unfortunately, her condition worsened despite intensified medical therapy, resulting in frequent hospitalizations due to severe hypercalcemia (calcium >3.5 mmol/L). Her iPTH levels increased to 204.7 pmol/L, and the adenoma grew to 2.1 x 1.9 x 3.1 cm. Given her deteriorating condition, she underwent ultrasound-guided microwave ablation of the adenoma.

Two days after the procedure, her iPTH levels dropped by 80% to 11.3 pmol/L and stabilized between 35-40 pmol/L in the outpatient setting. Her post-procedural calcium level was within the mild hypocalcaemia range (2.8-3.0 mmol), and she no longer needed cinacalcet or pain medication. She experienced significant improvements in her physical function and could engage in static exercise. A follow-up ultrasound one month post-procedure revealed a 56% reduction in the adenoma's volume.

CONCLUSION

Ultrasound-guided microwave ablation is an effective non-surgical treatment for PHPT in high-risk patients. It provides clinically significant improvements, reduces medication requirements, and enhances the quality of life.

EP_A051

WHEN TREATMENT BACKFIRES: SEVERE HYPOTONIC HYPONATREMIA INDUCED BY ANGIOTENSIN RECEPTOR BLOCKERS

<https://doi.org/10.15605/jafes.040.S1.059>

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INTRODUCTION/BACKGROUND

Angiotensin receptor blockers (ARBs) are commonly used antihypertensive medications. ARBs may cause worsening of renal function and hyperkalemia, necessitating renal profile monitoring after their initiation. We report a case of severe hypotonic hyponatremia in an elderly patient who was started on valsartan.

CASE

A 70-year-old Malay female with underlying hypertension was recently prescribed valsartan 80 mg OD by her primary care (PC) doctor for blood pressure optimization. Notably, she had a history of adrenal insufficiency secondary to

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exogenous steroid use but had successfully discontinued oral hydrocortisone after an adequate Synacthen test response 2 years ago. Four days after starting valsartan, she presented to the Emergency Department (ED) with dizziness and vomiting. Laboratory results revealed severe hypotonic hyponatremia (serum sodium 110 mmol/L, serum osmolality 259 mOsm/kg, urine osmolality 247 mOsm/kg, urine Na 71 mmol/L) and hyperkalemia (serum potassium 7.0 mmol/L). In the ED, she was given a lytic cocktail and 150 cc of 3% saline. Prior to starting valsartan, her serum sodium at the PC clinic was 135 mmol/L. She denied using any over-the-counter or traditional medications. Her blood pressure and blood glucose levels were normal throughout hospitalization, making adrenal insufficiency less likely. Further investigations, including morning serum cortisol (500.4 nmol/L) and TSH (0.54 mIU/L, NR 0.4-4.0 mIU/L), were normal. Thus, the diagnosis of severe hypotonic hyponatremia secondary to valsartan was made. After withholding valsartan, her symptoms resolved, the serum sodium and potassium normalized, and she was discharged well 4 days later.

CONCLUSION

ARBs can lead to severe hyponatremia by blocking the angiotensin II receptor, which inhibits renal tubular sodium reabsorption. This effect is particularly pronounced in the elderly and individuals on concomitant thiazide therapy. Although rare, ARB-associated hyponatremia should be considered in patients with hypotonic hyponatremia when other causes have been ruled out.

EP_A052

A SILENT THREAT: LARYNGEAL INVOLVEMENT IN PAGET'S DISEASE LEADING TO AIRWAY COMPROMISE

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INTRODUCTION/BACKGROUND

Paget's disease of bone (PDB) is a chronic skeletal disorder characterized by disorganized bone remodelling, often affecting the skull, spine, pelvis and long bones. While complications such as fractures, arthritis and hearing loss are well-documented, laryngeal involvement leading to acute airway obstruction is exceptionally rare. To our knowledge, no previous case reports have described PDB affecting the thyroid and arytenoid cartilages, resulting in airway compromise.

CASE

A 45-year-old male with hypertension and eczema was diagnosed with a variant of PDB (normal alkaline phosphatase) in 2018, following an evaluation for right knee and ankle pain that began in 2016. Extensive investigations, including a bone biopsy, revealed nonspecific sclerosis, normal ALP, and mildly elevated bone formation markers (P1NP). A Tc-99m MDP bone scan showed multiple hot spots involving the skull, clavicles, ribs, L5, right elbow, both knees and both ankles. He was initiated on yearly intravenous zoledronate (4 mg). In 2022, he sustained a low-impact distal third right ulna fracture, necessitating a locking plate. The fracture site biopsy confirmed Paget's disease.

In 2024, he presented with acute upper airway obstruction. A CT neck scan revealed expansile lytic lesions involving thyroid and arytenoid cartilages, causing significant airway narrowing. An emergency tracheostomy was performed to secure his airway. A repeated Tc-99m MDP bone scan demonstrated disease progression, with worsening involvement of the thyroid and cricoid cartilages.

CONCLUSION

This case highlights a rare and potentially fatal complication of PDB, with airway obstruction due to expansile lytic lesions of the laryngeal cartilages. Given the absence of prior reports on this manifestation, clinicians should remain vigilant for atypical presentations of PDB, particularly in patients with progressive disease. Early diagnosis and intervention are critical to preventing life-threatening outcomes.

EP_A053

MARINE-LENHART SYNDROME: A RARE CASE OF AUTOIMMUNE HYPERTHYROIDISM AND FUNCTIONAL THYROID NODULE

<https://doi.org/10.15605/jafes.040.S1.061>

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INTRODUCTION/BACKGROUND

Marine-Lenhart syndrome is a rare thyroid disorder characterized by the presence of Graves' disease and autonomously functioning thyroid nodules. This dual

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pathology poses a diagnostic and therapeutic challenge as it combines features of autoimmune hyperthyroidism and toxic multinodular goiter. The prevalence is estimated to be 0.8-4.1% among patients with Graves' disease. Patients with Marine-Lenhart Syndrome generally have lower remission rates with thionamide therapy, unlike typical Graves' disease, and frequently require definitive treatment such as radioiodine ablation or total thyroidectomy.

CASE

A 31-year-old female presented with persistent tremors, palpitations, heat intolerance and an unintended 5 kg weight loss over three months. She also reported progressive bilateral eye bulging over the past year. Upon physical examination, tachycardia, fine tremors, a diffusely enlarged thyroid gland with palpable nodules, and mild exophthalmos with lid lag were observed. Laboratory evaluations revealed suppressed thyroid-stimulating hormone level (<0.01 mIU/L; reference range 0.35-4.94) and elevated free thyroxine level (>64.35 pmol/L; reference range 0.70-1.48). The thyroid receptor antibody level was significantly elevated at 11.1 U/L, indicating Graves' disease. Thyroid ultrasound showed a diffusely enlarged, hyperplastic gland with multiple mixed cystic and solid nodules bilateral (TI-RADS 1) and solid nodule size $1 \times 0.9 \times 0.8$ cm in the right thyroid (TI-RADS 3). Thyroid scintigraphy demonstrated diffusely increased uptake (35.3%; normal 1-5%) with multiple hot nodules. The presence of autoimmune hyperthyroidism alongside functioning nodules confirmed the diagnosis of Marine-Lenhart syndrome. She was initially treated with thiamazole, propranolol, selenium and ocular lubricants. She underwent radioiodine ablation as definitive treatment.

CONCLUSION

This case highlights the importance of considering a diagnosis of Marine-Lenhart syndrome in patients presenting with hyperthyroidism and thyroid nodules. Delay or failure to recognize it may lead to misdiagnosis and inadequate treatment, potentially extending symptoms and increasing the risk of complications. A comprehensive clinical, biochemical and imaging examination is needed for accurate diagnosis and proper management.

EP_A054

BEYOND OSMOTIC DIURESIS: DIAGNOSING ARGININE VASOPRESSIN DEFICIENCY (AVP-D) IN A PATIENT WITH UNCONTROLLED DIABETES

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INTRODUCTION/BACKGROUND

Polyuria and polydipsia in patients with poorly controlled diabetes mellitus are often attributed to osmotic diuresis. However, concurrent (AVP-D) is a rare but critical differential diagnosis that requires careful evaluation.

CASE

A 57-year-old female with hypertension and poorly controlled diabetes mellitus, with HbA1c 10.6%, presented with polyuria, polydipsia and significant weight loss. She had no history of fever, infective symptoms, head surgery or head trauma. She denied any pertinent family history. There was no evidence of hyper- or hypopituitarism symptom-wise. She was hemodynamically stable, and systemic examinations were unremarkable. Her initial investigations showed sodium 138 mmol/L, potassium 4.1 mmol/L, creatinine 56 μ mol/L, random blood sugar 18.3 mmol/L, corrected calcium 2.5 mmol/L and phosphate 1.15 mmol/L. Chest X-ray, KUB ultrasound and brain CT were unremarkable. In the ward, the patient was commenced on insulin therapy to optimize blood glucose control. Nevertheless, despite controlling the blood glucose, she had persistent polyuria up to 9L/day, with hypernatraemia 149 mmol/L and low urine SG 1.000, even with urine glucose 4+. Hence, modified water deprivation test was performed, revealing an inappropriately low urine osmolality of 97 mOsm/kg despite elevated plasma osmolality of 319 mOsm/kg, with a significant increase of urine osmolality to 426 mOsm/kg post-desmopressin, confirming AVP-D. Pituitary MRI showed a normal posterior pituitary bright spot without structural abnormalities. The patient was initiated on oral desmopressin, which resulted in marked improvement in clinical symptoms. Her pituitary hormonal assessment showed FSH 42.4 IU/L, LH 24.7 IU/L, estradiol 96.0 pmol/L, TSH 0.54 mIU/L, FT4 12.46 pmol/L, AM cortisol 217 nmol/L, prolactin 93.3 uIU/mL. Her tumor markers, beta HCG, Mantoux test and anti-TPO antibodies were negative, which would be mostly idiopathic AVP-D.

CONCLUSION

AVP-D should be considered in patients with diabetes who have persistent polyuria and polydipsia despite glucose

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normalization, particularly when urine osmolality is unexpectedly low. The patient's osmotic diuresis from hyperglycemia may have initially masked AVP-D, complicating the diagnosis.

This case highlights the need to differentiate AVP-D from osmotic diuresis in a patient with diabetes with persistent polyuria. Identifying the condition early and treating it with desmopressin, while optimizing blood sugar control, can help prevent future complications.

EP_A055

T3 THYROTOXICOSIS AS A PARANEOPLASTIC MANIFESTATION OF METASTATIC EXTRAGONADAL NONSEMINOMATOUS GERM CELL TUMOUR

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INTRODUCTION/BACKGROUND

Nonseminomatous germ cell tumours (NSGCTs) are rare malignancies that arise from gonadal or extragonadal sites and comprise various histological subtypes. In 90% of cases, β -human chorionic gonadotropin (β -hCG) is elevated, with extreme levels occasionally inducing thyrotoxicosis via TSH receptor cross-reactivity.

CASE

We report a case of metastatic extragonadal NSGCT presenting with T3 thyrotoxicosis. A 22-year-old Malay male with no prior medical history developed progressive abdominal pain, nausea, vomiting and a 20 kg weight loss over four months. On arrival at the emergency department, he was hypertensive (153/120 mm Hg) and tachycardic (132 bpm). Examination revealed a 3 × 3 cm left cervical lymph node but no signs of hyperthyroidism. Initial thyroid function tests showed suppressed TSH (0.017 mU/L), normal free T4 (20.82 pmol/L), and elevated T3 (6.6 mU/L), consistent with T3 thyrotoxicosis. He was initiated on carbimazole 20 mg OD. TSH receptor antibody was negative. He required intensive care admission for heart failure, where echocardiography revealed global hypokinesia with a left ventricular thrombus. Further evaluation with a contrast-enhanced CT scan of the neck, thorax, abdomen and pelvis showed extensive cervical, mediastinal and abdominal lymphadenopathy, as well

as a large lobulated left suprarenal mass (6.7 × 6.5 × 6.4 cm) with necrosis. Workup for adrenal hyperfunction was negative, and a markedly elevated β -hCG (250,573.0 U/L) led to a revised diagnosis of metastatic extragonadal NSGCT with paraneoplastic thyrotoxicosis. A cervical lymph node biopsy confirmed the diagnosis. Antithyroid therapy was tapered to achieve normal T3 levels. He was then referred for chemotherapy. His thyroid function normalised following treatment and carbimazole was discontinued, coinciding with a decline in β -hCG levels.

CONCLUSION

This case highlights the importance of considering paraneoplastic thyrotoxicosis in patients with unexplained hyperthyroidism and systemic symptoms, particularly in the context of extreme β -hCG elevations. Early recognition and appropriate oncological management are crucial for optimising outcomes.

EP_A056

PRIMARY ADRENAL INSUFFICIENCY SECONDARY TO BILATERAL ADRENAL TUBERCULOSIS DURING ANTI-TUBERCULOSIS TREATMENT

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INTRODUCTION/BACKGROUND

Adrenal tuberculosis (TB) is a rare but serious form of extrapulmonary TB, accounting for 7% to 20% of primary adrenal insufficiency (PAI) cases worldwide. It typically results from haematogenous spread, leading to granulomatous inflammation, caseous necrosis and progressive adrenal destruction. Despite appropriate anti-TB therapy, PAI can develop weeks to months later due to ongoing adrenal damage.

CASE

A 68-year-old Malay male with type 2 diabetes mellitus, hypertension and ischaemic heart disease was recently diagnosed with miliary TB and had been on anti-TB treatment (EHRZ regimen) for 43 days. He presented with a two-day history of lethargy, poor oral intake and postural giddiness. Upon arrival, he appeared cachectic, with hyperpigmentation over the knuckles, a blood pressure of 88/71 mm Hg, and a heart rate of 99 bpm. Given his persistent hypotension despite fluid resuscitation, adrenal crisis was suspected, and intravenous hydrocortisone was

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initiated. His laboratory tests showed a low random cortisol level of 21 nmol/L and an elevated ACTH level of 143 pmol/L (reference range: 1.6–13.9 pmol/L), confirming PAI. A Computed Tomography (CT) scan of the adrenal glands revealed bilateral adrenal enlargement with peripheral enhancement and central necrosis, consistent with adrenal TB. Anti-TB treatment was continued, and hydrocortisone was gradually tapered to 20 mg in the morning and 10 mg in the evening. He required a higher maintenance dose due to concurrent rifampicin therapy.

CONCLUSION

This case highlights the importance of early recognition of adrenal insufficiency in TB patients. Delayed-onset PAI can occur despite ongoing therapy, necessitating a high index of suspicion and prompt glucocorticoid replacement to prevent adrenal crisis. Additionally, clinicians should be mindful of rifampicin-induced glucocorticoid metabolism, which often necessitates higher maintenance doses of glucocorticoids in affected patients.

EP_A057

CONFRONTING THE ELUSIVE GIANT: A RARE CASE OF GIANT CYSTIC PARATHYROID ADENOMA

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INTRODUCTION/BACKGROUND

Giant cystic parathyroid adenomas are an uncommon cause of primary hyperparathyroidism and may result in severe hypercalcemia. Due to their cystic nature, they can evade detection by conventional imaging such as Sestamibi scans, posing diagnostic challenges. We report a case of a 60-year-old female with a giant cystic parathyroid adenoma, where conventional imaging failed to identify the lesion.

CASE

A 60-year-old female with hypertension and stage 4 chronic kidney disease presented with a three-month history of diffuse goitre and asymptomatic hypercalcemia (corrected calcium 3.11–3.77 mmol/L). Investigations showed elevated iPTH (160.3 pmol/L), low phosphate (0.75 mmol/L), low vitamin D (33.25 nmol/L), with normal thyroid function. Neck ultrasound detected a benign thyroid nodule (TIRADS 1), but no parathyroid lesion. A Sestamibi scan was negative for hyperfunctioning or ectopic parathyroid tissue and showed only cystic changes in the thyroid.

Due to persistent hypercalcemia, CT imaging was performed and revealed a large cystic mass on the left neck (4.2 × 6.2 × 10.8 cm), suggestive of a cystic parathyroid adenoma. The patient had osteopenia and required multiple pamidronate infusions. She underwent a left parathyroidectomy, during which a large cystic parathyroid tumor was removed. Postoperative calcium levels normalized, and histopathology confirmed cystic parathyroid adenoma.

Sestamibi scans may not detect cystic parathyroid adenomas due to poor radiotracer uptake in cystic tissue. CT imaging played a key role in identifying the lesion in this case. Awareness of false-negative imaging results is essential to avoid delayed treatment and complications.

CONCLUSION

Negative Sestamibi scans do not exclude parathyroid pathologies, particularly in the presence of cystic adenomas. Clinicians should maintain a high index of suspicion and use complementary imaging modalities to avoid delays in treatment.

EP_A058

A RARE CASE OF ECTOPIC LINGUAL THYROID WITH SUBCLINICAL HYPOTHYROIDISM

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INTRODUCTION

Ectopic thyroid tissue may be found in locations other than the anterior neck region, and lingual thyroid accounts for 90% of the ectopic cases. It is an embryological aberration where the thyroid gland fails to descend from the foramen cecum to the lower part of the neck. Individuals with lingual thyroid are usually asymptomatic, but local obstructive symptoms may develop. Subclinical hypothyroidism is a common manifestation in patients with an ectopic lingual thyroid without a co-existing orthotopic thyroid gland. We present a case of ectopic lingual thyroid with subclinical hypothyroidism.

METHODOLOGY

A 68-year-old female presented with progressive voice changes for many years, associated with intermittent shortness of breath upon lying flat. Physical examination and transnasal scope showed a mass at the base of the tongue pushing on the epiglottis with oedematous bilateral arytenoids. Tracheostomy, direct laryngoscopy and tele-

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bronchoscopy were performed, and tumour fluid for cytology was negative for malignant cells. MRI of the neck revealed an ectopic lingual thyroid with nodular goitre and haemorrhagic cystic component causing oropharyngeal luminal narrowing, with absence of orthotopic thyroid gland. She is clinically euthyroid but biochemically subclinical hypothyroidism (TSH 11.87 uIU/mL, FT4 13.07 pmol/L). Anti-TPO antibodies were negative; a neck ultrasound showed no normal thyroid tissue in the anterior neck. Thyroxine hormone replacement commenced, and her TFT levels normalized 4 months later. Repeat neck CT showed no reduction in the size of the lingual thyroid. However, she declined surgical intervention.

CONCLUSION

Lingual thyroid is extremely rare but remains an important differential for patients presenting with a mass at the tongue base. Treatment with thyroxine should be initiated to prevent hypothyroidism and progressive growth of the ectopic tissue. Surgical intervention is indicated when the patient presents with severe respiratory tract obstruction, limited size-reduction despite thyroxine replacement or malignancy.

EP_A059

TRANSIENT REMISSION OF ACROMEGALY FOLLOWING PITUITARY APOPLEXY AND EARLY RELAPSE: A CASE REPORT

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INTRODUCTION/BACKGROUND

Acromegaly is caused by excessive growth hormone (GH) secretion and secondary elevation of insulin-like growth factor-1 (IGF-1). Elevated serum IGF-1 level is a useful screening tool for acromegaly. However, IGF-1 levels may appear normal in conditions such as liver disease, malnutrition, uncontrolled diabetes mellitus and pituitary apoplexy. When serum IGF-1 levels are normal, it is easy to miss the diagnosis of acromegaly without a high index of suspicion and/or a GH suppression test. We report a case of an acromegaly patient with pituitary apoplexy and initially normal IGF-1 level.

CASE

A 24-year-old young female presented initially with severe headache, blurring of vision, vomiting and sudden onset of reduced consciousness. Brain CT showed intratumoral haemorrhage of sellar and suprasellar mass causing

cerebral oedema and mass effect, suggestive of pituitary apoplexy. Emergency craniectomy and tumour excision were performed, and HPE revealed a pituitary adenoma. Hormonal workup prior to surgery showed central hypothyroidism with hyperprolactinemia, likely caused by stalk effect. Other parameters were unremarkable, including normal IGF-1. Postoperatively, she developed panhypopituitarism, bilateral eye blindness and scar epilepsy. IGF-1 was rechecked 6 months postoperatively for spade-like hands, but the result was not found. It was only after four years, following family concerns about gradual acral enlargement, that her post-op IGF-1 was found markedly elevated. GH suppression test subsequently confirmed acromegaly. She was offered repeat surgery due to the persistent sellar mass from MRI surveillance. However, the patient was not keen and medical therapy with a somatostatin receptor ligand was initiated, with referral to oncology for radiotherapy.

CONCLUSION

Transient remission of acromegaly after pituitary apoplexy can occur. However, a high index of suspicion of relapse is crucial especially in those patients with acromegaloid features. Hence, repeating IGF-1 testing or GH suppression test is advisable if the diagnosis is uncertain.

EP_A060

HAIRY PREGNANCY: A RARE CASE OF GESTATIONAL HYPERANDROGENISM

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INTRODUCTION/BACKGROUND

The role of testosterone in pregnancy is usually overlooked. Studies have shown that pregnancy can result in physiological elevation of testosterone, as high as 2 to 3 times the upper limit of normal, but this does not result in the virilisation of the mother. We present a rare case of virilisation during pregnancy with a complete resolution of symptoms post-delivery.

CASE

This is the case of a 27-year-old female referred for increased hair on her limbs and face, requiring her to shave every 1-2 weeks (Ferriman-Gallway 5). In history, she has had irregular menses starting at 18 years old. She is obese, with a pre-pregnancy weight of 84 kg and a BMI of 35 kg/m². She was treated for polycystic ovarian syndrome (PCOS) by the Gynaecology team. Ovarian ultrasound showed no cysts. The hormonal profile revealed an elevated

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testosterone level of 5.74 nmol/L (NR: <1.67). Other blood investigations were normal. Further investigation for the hyperandrogenism from possible androgen-producing tumour was postponed till post-delivery. However, during the 6-month follow-up post-delivery, she had regularised menses and reduced facial hair, which minimised regular shaving. Her repeat testosterone level taken 4 months post-delivery was 0.49 nmol/L.

CONCLUSION

Elevated testosterone during pregnancy is a normal physiological response vital for maintaining pregnancy and initiation of parturition. It is caused by the increased production and reduced clearance of testosterone. Excess testosterone during pregnancy does not cause clinical hyperandrogenism as a result of increased SHBG, which binds the androgens, and placental aromatase, which converts excess testosterone to estradiol. However, PCOS can result in a diminished protective effect of the placenta aromatase, resulting in clinical hyperandrogenism during pregnancy. Our patient had a pre-pregnancy PCOS diagnosis, which worsened her hyperandrogenism intrapartum. This condition was similar to a few published case reports. The resolution of PCOS symptoms post-delivery can transiently happen due to the stabilisation of hormones postpartum; unfortunately, PCOS symptoms may recur later on.

EP_A061

WHEN THE THYROID AND STOMACH COLLIDE: APS TYPE 3B BEHIND CARDIAC SYMPTOMS

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INTRODUCTION/BACKGROUND

Autoimmune Polyglandular Syndromes (APS) are a group of disorders characterised by the simultaneous or sequential occurrence of multiple autoimmune-mediated diseases affecting endocrine glands. Pernicious anaemia is commonly part of this broader spectrum of autoimmune conditions.

CASE

We report the case of a 65-year-old male with a seven-year history of megaloblastic anaemia treated with cyanocobalamin, who presented with severe anaemia-induced non-ST-elevation myocardial infarction (NSTEMI) that manifested as chest pain, reduced exercise tolerance

and profound fatigue. Initial investigations revealed pancytopenia, with a haemoglobin level of 5.0 g/dL, elevated mean corpuscular volume (142.2 fL), platelet count of $26 \times 10^9/L$, white cell count of $0.9 \times 10^9/L$, and significantly elevated troponin I levels (initially 2069 ng/L and rising to over 25,000 ng/L). Iron studies showed low serum iron (9.3 $\mu\text{mol/L}$), marginally elevated ferritin (325.3 ng/mL) and reduced total iron-binding capacity (40.84 $\mu\text{mol/L}$). Vitamin assays confirmed severe vitamin B12 deficiency (59 pmol/L) with elevated folate (49.2 nmol/L). Given the profound B12 deficiency, immunological testing revealed the presence of anti-parietal cell antibodies and elevated intrinsic factor IgG, which confirmed the diagnosis of pernicious anaemia. Given the clinical features suggestive of hypothyroidism, thyroid function testing was performed, revealing a free T4 of 4.3 pmol/L, TSH of 108.96 mIU/L and anti-thyroid peroxidase antibodies >600 IU/mL, consistent with Hashimoto's thyroiditis. Levothyroxine and cyanocobalamin replacement therapy were initiated subsequently. These findings led to a diagnosis of APS type 3b, characterised by the coexistence of pernicious anaemia and Hashimoto's thyroiditis.

CONCLUSION

Hashimoto's thyroiditis (HT) and autoimmune gastritis (AIG) often coexist. Studies have shown that HT is present in 10–40% of patients with gastric disorders, and about 40% of those with AIG also have HT. This case emphasizes the need to screen for coexisting autoimmune conditions.

EP_A062

OVARIAN OVERDRIVE: FUNCTIONING GONADOTROPH ADENOMA LEADING TO SPONTANEOUS OVARIAN HYPERSTIMULATION

<https://doi.org/10.15605/jafes.040.S1.070>

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INTRODUCTION/BACKGROUND

Functioning gonadotroph adenomas (FGAs) are rare pituitary tumours characterised by the hypersecretion of biologically active gonadotrophs. We report a case of a 22-year-old Malay female diagnosed with FGA with ovarian hyperstimulation syndrome (OHSS), highlighting her clinical presentation, management, and post-operative outcomes.

CASE

The patient first presented at age 19 with acute abdominal pain and irregular menstruation. An abdominal ultrasound

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showed large bilateral multiloculated ovarian cysts, the largest cyst measuring 12 x 15 cm. She underwent laparotomy for left salpingo-oophorectomy and right ovarian cyst aspiration with histopathological examination (HPE) that ruled out ovarian malignancy.

Post-operatively, she remained amenorrheic with a thin endometrial wall despite progestin therapy. Follow-up scans showed persistent large ovarian cysts. Hormonal work-up revealed elevated estradiol (13422 pmol/L, NR 110-1468 pmol/L), unsuppressed FSH (31.8 IU/L, NR 3-8 IU/L) and raised prolactin (1551 mIU/L, NR 70-566 mIU/L) levels. She also had intermittent headaches but no visual disturbances, galactorrhea or hirsutism. MRI of the pituitary reported pituitary macroadenoma (1.8 x 2.6 x 2.9 cm) with suprasellar extension compressing onto the optic chiasm.

The patient successfully underwent transsphenoidal surgery (TSS) of the pituitary adenoma. Tissue HPE stained positive for synaptophysin (+), FSH (+) and LH (+), with a low Ki-67 index of 0.1%, confirming the diagnosis of FGA with stalk effect. Post-TSS, her gonadotropin level normalised, menstruation resumed and ovarian cyst size decreased. Follow-up MRI showed no residual tumour or recurrence.

CONCLUSION

FGAs are a rare differential diagnosis that needs to be considered in females presenting with spontaneous OHSS, accompanied by elevated serum estradiol and unsuppressed FSH. Early diagnosis and prompt transsphenoidal surgery can restore normal menstruation, improve fertility, and potentially avoid ovarian surgery.

EP_A063

UNMASKING SYNDROMIC HYPOPARATHYROIDISM IN PREGNANCY: A CASE OF BARAKAT SYNDROME

<https://doi.org/10.15605/jafes.040.S1.071>

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INTRODUCTION/BACKGROUND

Hypoparathyroidism (HypoPT) is a rare endocrine disorder that presents unique challenges in pregnancy due to altered calcium homeostasis. While 75% of cases follow neck surgery, 25% arise from autoimmune, genetic or other causes. During pregnancy, elevated Parathyroid hormone-related peptide (PTHrP) suppresses PTH, while increased

1,25-(OH)₂-D₃ enhances calcium absorption, reducing maternal calcium requirements. Maintaining stable calcium levels is essential to prevent fetal hypocalcemia and secondary hyperparathyroidism.

CASE

A 24-year-old female at 37 weeks of gestation was incidentally found to have asymptomatic hypocalcemia. Two years earlier, during her first pregnancy, she experienced severe postpartum hypocalcemia accompanied by bilateral lower limb weakness. Investigations revealed a corrected calcium level of 1.65 mmol/L (reference range: 2.18–2.60), phosphate of 1.73 mmol/L (0.78–1.65), iPTH of 0.84 pmol/L (1.56–6.03) and a total 25-hydroxy vitamin D level of 28.88 nmol/L (≥75 nmol/L), leading to a diagnosis of vitamin D deficiency and HypoPT. Unfortunately, she was lost to follow-up. During her current pregnancy, blood tests showed a corrected calcium of 2.04 mmol/L, phosphate of 1.32 mmol/L and iPTH of 2.86 pmol/L. She remained clinically asymptomatic with no signs of hypocalcemia. The goal of management was to maintain calcium levels within the lower normal range until delivery. Further history revealed no prior neck surgery, but she had long-standing bilateral sensorineural hearing loss since childhood. Notably, her parents and siblings also had congenital deafness. Although rare, syndromic causes of HypoPT, such as Barakat syndrome (HDR syndrome), should be considered in patients with hearing impairment, renal disease, or congenital anomalies, especially with a strong family history.

CONCLUSION

This case highlights the importance of recognising syndromic HypoPT in pregnancy. Barakat syndrome, caused by GATA3 mutations, is characterised by HypoPT, deafness and renal disease, with the full triad in 62.3% of cases and HypoPT with deafness in 28.6%.

EP_A064

UNMASKING GASTRIC VOLVULUS IN THE SHADOW OF HYPOTHYROIDISM: A CASE OF ACUTE MESENTERO-AXIAL ROTATION

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INTRODUCTION

Gastric volvulus is a rare, life-threatening condition caused by an abnormal stomach rotation, potentially leading to obstruction and strangulation. The mesentero-axial type

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is the most common, comprising about one-third of cases. This report discusses a case of acute mesentero-axial gastric volvulus likely caused by severe hypothyroidism.

CASE

A 77-year-old female with a history of degenerative spine disease, hypertension, hypothyroidism post-thyroidectomy and hyperlipidemia presented with a three-day history of abdominal pain, distension and vomiting. Clinically, her abdomen was distended and tender. Abdominal CT reported mesentero-axial gastric volvulus, and endoscopy showed a distended stomach with undigested food. Barium swallow revealed oesophageal motility disorders but no mass. Further tests showed elevated TSH levels (>51.9 mIU/L) and low free T4 (6.6 pmol/L), indicating severe hypothyroidism, likely due to missed levothyroxine (LT4) doses.

Due to her advanced age and initial presentation of sinus arrhythmia, the plan was to administer LT4 rectally with serial monitoring of TFT. The patient was prescribed a daily dose of 150 mcg (4 mcg/kg/day) of LT4, titrated up to 200 mcg daily (6 mcg/kg/day) and later switched to intravenous LT4 50 mcg daily before undergoing gastropexy. Following thyroid hormone replacement, her condition improved.

CONCLUSION

Severe hypothyroidism can impair gastrointestinal motility, and while gastrointestinal symptoms are common, gastric volvulus is rare. This case emphasises the importance of recognising thyroid dysfunction as a differential cause of gastric volvulus, particularly in patients with thyroid disorders. Early thyroid hormone replacement is essential to prevent recurrence and severe complications.

EP_A065

DEBILITATING NEUROGLYCOPENIA SECONDARY TO HIRATA DISEASE ACHIEVING REMISSION SPONTANEOUSLY

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INTRODUCTION/BACKGROUND

Insulin Autoimmune Syndrome (IAS), also known as Hirata's disease, is a rare cause of hyperinsulinaemic hypo-

glycaemia. It is characterised by spontaneous hypoglycemia associated with extremely high circulating insulin levels and positive anti-insulin antibodies. Hypoglycaemic episodes usually occur in the post-prandial state and are commonly associated with other autoimmune conditions, such as Graves' disease, systemic lupus erythematosus and rheumatoid arthritis.

CASE

We report a case of a 62-year-old male with a background history of hypertension and cerebrovascular accident who presented with reduced consciousness. His capillary blood glucose levels ranged from 1.7 to 2.2 mmol/L and hypoglycaemia symptoms resolved following intravenous dextrose administration. The patient denied any consumption of oral hypoglycemic agents, exogenous insulin or traditional medications. Hypoglycaemia episodes occurred in both fasting and postprandial states. There was no weight gain to suggest insulinoma, and there were no constitutional symptoms to suggest underlying malignancy. No associated autoimmune conditions were noted. Systemic examination was unremarkable. Laboratory results revealed random blood glucose levels of 1.7 mmol/L, with marked elevation of insulin level of >6944 pmol/L and C-peptide level of 3496 pmol/L. Renal and liver profile and septic parameters were all within normal range. Insulin autoantibody (IAA) titers were elevated at 175 IU/mL (positive >20 IU/mL). Sulfonylurea levels were undetectable, and tumour markers were within normal limits. Localisation studies with CT, MRI and endoscopic ultrasound of the pancreas showed a normal pancreaticobiliary system. He was initially managed with oral prednisolone 10 mg twice daily and responded well. The dose was tapered off over two months with no recurrent episodes of hypoglycaemia afterwards.

CONCLUSION

IAS should be considered in a patient with postprandial hypoglycaemia with marked elevation of insulin levels. The diagnosis can be confirmed by elevation of insulin autoantibody titres. Most cases are self-remitting but may be managed with low-carbohydrate meals, steroids and steroid-sparing immunosuppressants.

Adult E-Poster

EP_A066

LANGERHANS HISTIOCYTOSIS-RELATED HYPOPHYSITIS: A DISTINCT CAUSE OF CRANIAL DIABETES INSIPIDUS

<https://doi.org/10.15605/jafes.040.S1.074>

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INTRODUCTION/BACKGROUND

Cranial diabetes insipidus (DI) is a rare manifestation of hypothalamo-pituitary axis disorders. Langerhans Cell Histiocytosis (LCH), a clonal disease of dendritic cells, can infiltrate multiple organs, including the pituitary gland. When the posterior pituitary is involved, LCH can cause secondary hypophysitis, leading to cranial DI. Due to overlapping symptoms with more common conditions, diagnosis can be delayed.

CASE

A 22-year-old female with a one-year history of bullous skin lesions and oral ulcers was initially diagnosed with pemphigus vulgaris and treated with prednisolone. However, a skin biopsy later confirmed LCH. Imaging, including CT of the brain, neck and thorax-abdomen-pelvis, showed multisystem involvement (skin, ear, thyroid, and thymus). Hence, she underwent chemotherapy.

After the first cycle of chemotherapy with vinblastine and high-dose dexamethasone, she developed vomiting and lethargy. Laboratory tests revealed a hyperosmolar hyperglycemic state (serum osmolality 323 mOsm/kg). Despite normalisation of her blood glucose, she developed marked polyuria (>13,000 mL/day). Further testing showed serum osmolality of 294 mOsm/kg and urine osmolality of 54 mOsm/kg. A desmopressin trial resulted in a >50% increase in urine osmolality (398 mOsm/kg at 2 hours; 511 mOsm/kg at 4 hours), confirming cranial DI. MRI revealed a 1.1 × 1.3 × 1.0 cm lesion in the pituitary infundibulum, consistent with LCH-related hypophysitis, along with empty sella syndrome, likely secondary to chronic pituitary involvement and glucocorticoid therapy. She was treated with sublingual desmopressin and high-dose steroids, showing clinical improvement.

CONCLUSION

This case highlights the need to consider cranial DI as a manifestation of systemic LCH, especially in young adults with multisystem disease. Given the overlap of symptoms, early recognition of cranial DI is crucial for timely management.

EP_A067

DISCORDANT THYROID FUNCTION TESTS: DIAGNOSTIC CHALLENGES IN A PATIENT WITH A TSH-SECRETING PITUITARY ADENOMA

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INTRODUCTION/BACKGROUND

Discordant Thyroid Function Tests (TFTs) may present a diagnostic challenge in clinical practice. Assay interference must be excluded before proceeding to further investigations. Rarely, the underlying diagnosis may be a TSH-secreting pituitary adenoma (TSHoma) or resistance to thyroid hormone (RTH). Making a diagnostic distinction between these two conditions is important as their clinical management varies significantly.

CASE

A 47-year-old engineer was referred to the Endocrine clinic with symptoms of hyperthyroidism and discordant TFTs. These symptoms improved with propranolol and carbimazole. Only his paternal aunt has a goitre on family history. On physical examination, there was no palpable goitre. His TFT was discordant before starting carbimazole [TSH 4.32 mIU/L (NR:0.27-4.20), fT4 34.1 pmol/L (NR: 12.0-22.0), fT3 11.6 pmol/L (NR: 3.10-6.80)]. Discordance persisted despite using two different immunoassays after stopping carbimazole. Alpha-subunit was raised (2.47 IU/L; NR: 0.0-0.7). A pituitary MRI showed pituitary macroadenoma. Pituitary hormones were within normal limits except for an elevated IGF-1. An oral glucose tolerance test was inconsistent with acromegaly as nadir growth hormone was 0.8 mIU/L (0.27 g/L). Several tests distinguishing a TSHoma from RTH were unavailable, so the patient underwent a somatostatin test. Following octreotide, TSH was suppressed with low-normal free thyroid hormone levels, highly suggestive of a TSHoma.

Our patient underwent an endoscopic transsphenoidal hypophysectomy. The histology report confirmed TSH and growth hormone-secreting adenoma.

CONCLUSION

The case illustrates challenges in establishing the diagnosis of a TSHoma with resource limitations and supports using intramuscular octreotide LAR as a diagnostic tool. Appropriate evaluation of discordant TFT is paramount

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to avoid unnecessary investigations and treatments. The somatostatin test can be useful and practical in differentiating TSHoma from resistance to thyroid hormone.

EP_A068

LATENT AUTOIMMUNE DIABETES IN YOUTH PRESENTING AS YOUNG-ONSET TYPE 2 DIABETES: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.076>

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INTRODUCTION/BACKGROUND

Latent autoimmune diabetes forms a continuous age-related spectrum from LADY to LADA, where LADY exhibits greater autoimmunity. Latent autoimmune diabetes in youth (LADY) is diagnosed in individuals aged 15 to 29 years. A high prevalence of LADY is observed among youth with the T2DM phenotype. The TCF7L2 rs12255372 polymorphism is linked to an increased risk of developing T2DM at a young age and is associated with lower levels of GADA in individuals with either T2DM or latent autoimmune diabetes.

CASE

A 28-year-old female with no history of T2DM was admitted to the emergency room due to altered mental status. She had experienced a weight loss of approximately 10 kg and frequent nocturnal urination; however, she had never consulted a physician. Both her parents and her sister have a known history of T2DM. A physical examination revealed excess body weight and acanthosis nigricans. Laboratory results indicated elevated glucose levels, a high A1C level, a normal fasting C-peptide, negative ketones in the urine and a high HOMA-IR score. Following the patient's clinical improvement, we transitioned from insulin to oral hypoglycemic drugs. After several weeks, we identified a positive anti-GAD result and the TCF7L2 gene polymorphism, rs12255372 (G/T).

CONCLUSION

The clinical diagnosis of latent autoimmune diabetes (LAD) can be quite challenging. Young individuals exhibiting a T2DM phenotype should undergo assessment for pancreatic islet cell autoantibodies. Common TCF7L2 gene polymorphisms are linked to T2DM and latent autoimmune diabetes but not type 1 diabetes.

EP_A069

SUSPECTED LEFT ADRENOCORTICAL CARCINOMA LATER DIAGNOSED AS EXTRA-ADRENAL COMBINED SCHWANNOMA AND GANGLIONEUROMA

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INTRODUCTION/BACKGROUND

Ganglioneuromas and schwannomas are both rare benign tumours. They arise from different types of nerve cells: ganglioneuromas from autonomic ganglion cells and schwannomas from nerve sheath cells (Schwann cells). A combined adrenal ganglioneuroma and schwannoma is extremely rare, representing 1.4% of adrenal incidentalomas.

CASE

We present a 41-year-old male with an underlying left ureteric calculus who was referred for left adrenal incidentaloma from CT urography (CTU). He had no history of paroxysms of headache, sweating or palpitations. There were no symptoms to suggest Cushing's or underlying malignancy. On examination, he was normotensive and there were no discriminatory features of Cushing's. Laboratory evaluation showed normal potassium (4.3 mmol/L), and the overnight dexamethasone test (ODST) was appropriately suppressed (12 nmol/L). 24-hour urine metanephrine was within normal range. Testosterone and DHEAS were within the normal range with 19 nmol/L and 3.830 umol/L levels, respectively. Initial CTU reported a left adrenal mass measuring 4.0 x 3.1 x 3.8 cm. The adrenal CT demonstrated a left adrenal mass measuring 4.1 x 3.2 x 4.0 cm, with a 39 Hounsfield unit, an absolute washout of 28% and a relative washout of 16%. These findings indicate an indeterminate adrenal mass with a differential of adrenocortical carcinoma or pheochromocytoma. He underwent a left adrenalectomy and was discharged well. Histopathological examination showed an encapsulated biphasic tumour. There were Verocay bodies, and the neoplastic cells were narrow, elongated and wavy with tapered ends, interspersed with collagen fibres, which are distinct characteristics of schwannomas. At the periphery of the tumour, a separate proliferation of spindle cells

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with multiple ganglion cells was seen, indicative of ganglioneuromas. Sections of the adrenal gland show an uninvolved cortex and medulla.

CONCLUSION

In our case, ganglioneuromas may have arisen from the paravertebral sympathetic plexus located retroperitoneally. This rare condition may mimic adrenal malignancy radiologically, and the modality of treatment is surgical excision.

EP_A070

A CASE REPORT OF THYROTOXIC PERIODIC PARALYSIS: AN ENDOCRINE EMERGENCY CAUSE OF PARAPARESIS IN YOUNG ADULTS AND ITS REVIEW OF PATHOPHYSIOLOGY

<https://doi.org/10.15605/jafes.040.S1.078>

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INTRODUCTION/BACKGROUND

Thyrototoxic periodic paralysis (TPP) is a potentially life-threatening clinical manifestation of thyrotoxicosis predominantly seen in those of Asian descent between the ages of 20 and 40 years. The attack is characterised by acute and reversible severe muscle weakness with hypokalemia that resolves with the treatment of hyperthyroidism.

CASE

A 22-year-old Chinese male with no previous medical illness presented to the emergency department with sudden onset bilateral lower limb weakness associated with intermittent palpitations for the past month. Lower limbs neurological examination revealed proximal muscle weakness but preserved tone, reflexes and sensation. There was a small diffuse goitre and fine tremors on the bilateral hands. He did not have features of thyroid eye disease or a thyroid bruit. Additionally, he denied any family history of thyroid disorders. Electrocardiogram showed sinus tachycardia, flattened T-waves and generalised U-waves. Laboratory assessments showed severe hypokalemia with a serum potassium level of less than 1.5 mmol/L (3.4-4.5). He was given intravenous potassium correction (KCl) twice (4 g in total) and 4 pints maintenance drips at 1.5g KCl per pint. Thyroid function tests and TSH receptor antibodies were suggestive of Graves' Disease. He was discharged home with carbimazole and propranolol and remains well after discharge.

CONCLUSION

Thyrototoxic periodic paralysis should be considered in the differential diagnosis of neuromuscular weakness in the context of hypokalaemia by the treating physicians. In TPP, hypokalaemia results from an intracellular shift of potassium induced by thyroid hormone sensitisation of the Na⁺/K⁺-ATPase pump, triggering muscle weakness and paralysis. The importance of prompt recognition, early diagnosis and treatment of the condition can prevent severe complications, such as cardiac dysrhythmia and respiratory failure. The addition of non-selective beta-blockers, such as propranolol, is utilised to treat and prevent paralytic attacks by mitigating hyperadrenergic activity and improving hypokalaemia.

EP_A071

A CASE REPORT AND LITERATURE REVIEW OF SUBCUTANEOUS LEVOTHYROXINE ABSORPTION TESTING IN A PATIENT WITH REFRACTORY PRIMARY HYPOTHYROIDISM

<https://doi.org/10.15605/jafes.040.S1.079>

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INTRODUCTION/BACKGROUND

We present a case of refractory primary hypothyroidism in which the patient failed an oral levothyroxine (LT4) absorption test under optimised conditions. Given limited formulary options and the patient's complex clinical background, an off-label trial of subcutaneous LT4 was initiated as an alternative treatment strategy.

CASE

A 51-year-old male underwent total thyroidectomy with right central neck dissection and radioactive iodine ablation for papillary thyroid carcinoma. He was maintained on a supraphysiologic dose of oral LT4 (approximately 3.16 mcg/kg/day) with suppressed TSH 0.24 mIU/L and fT4 20.6 pmol/L. He was admitted for encapsulating sclerosing peritonitis, requiring two paracentesis, diagnostic laparoscopy, intravenous antibiotics and systemic corticosteroids. During admission, thyroid function progressively worsened (TSH >100 mIU/L and fT4 9.7 pmol/L) despite adherence to increasing oral LT4 doses. An oral LT4 absorption test confirmed malabsorption. Given his ischemic heart disease, weekly high-dose intravenous or intramuscular LT4

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posed a potential cardiac risk. Daily intravenous LT4 was logistically impractical, and daily intramuscular injections were deemed unsuitable due to patient discomfort and medication wastage. A subcutaneous LT4 absorption test was performed using 100 mcg (1 mL) of IV LT4 (Fresenius Kabi) administered subcutaneously with a 25-gauge needle at a 45-degree angle. Free T4 levels were measured at baseline and 2-, 4-, 6-, and 48-hour post-injection (6.1, 8.7, 9.4, 12.4, and 7.2 pmol/L respectively). A peak increase in free T4 of 103.3% at 6 hours confirmed effective subcutaneous absorption. The LT4 dose was escalated to 150 mcg thrice weekly, resulting in biochemical improvement (TSH: 20.31 mIU/L; fT4: 9.1 pmol/L).

CONCLUSION

This case highlights subcutaneous LT4 as a viable off-label alternative in patients with confirmed malabsorption. Pharmacokinetic assessment revealed an estimated bioequivalence of 59.3% compared to intravenous LT4 (AUC calculated via trapezoidal method), consistent with findings from prior literature (Sharpe et al.).

EP_A072

A CASE OF LYMPHOCYTIC HYPOPHYSITIS WITH HYPOCORTISOLISM AND CRANIAL DIABETES INSIPIDUS

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INTRODUCTION/BACKGROUND

Lymphocytic Hypophysitis (LH) is an autoimmune pituitary gland disorder that can result in arginine vasopressin deficiency. Low cortisol levels may stimulate antidiuretic hormone (ADH) secretion and promote renal water reabsorption, which can be suppressed by exogenous corticosteroids. We report a case of LH with cranial diabetes insipidus (CDI), initially masked by concurrent hypocortisolism.

CASE

A 26-year-old female presented with a sudden-onset blurring of vision in the left eye, headache and polyuria. The ophthalmologic evaluation revealed optic neuropathy in the left eye, along with bitemporal hemianopia. Pituitary MRI demonstrated a mass measuring $1.1 \times 1.2 \times 1.6$ cm with associated thickening and enhancement of the pituitary infundibulum. The normal posterior pituitary bright spot was also absent.

On admission, her serum sodium was within the normal range, with a serum osmolality of 294 mOsm/kg and a urine osmolality of 793 mOsm/kg. Following the initiation of intravenous methylprednisolone, she developed polyuria. Paired osmolality testing showed a decrease in serum osmolality to 289 mOsm/kg and a drop in urine osmolality to 77 mOsm/kg, consistent with steroid-unmasked CDI. Desmopressin was initiated, resulting in an increase in urine osmolality to 760 mOsm/kg, confirming complete CDI and leading to symptomatic improvement.

Her autoimmune screening and infection markers were negative. She was discharged on oral prednisolone and sublingual desmopressin. At follow-up one month later, her symptoms and vision had significantly improved.

CONCLUSION

Although rare, the onset of polyuria following steroid initiation raises concern for the unmasking of CDI, particularly in patients with concurrent hypocortisolism. Since corticosteroids are the mainstay of medical treatment for LH, recognising this phenomenon is clinically important for timely diagnosis and appropriate management.

EP_A073

THE ADRENAL PARADOX: DECODING A CASE OF PRIMARY HYPERALDOSTERONISM WITH DISCORDANT DIAGNOSTIC CLUES

<https://doi.org/10.15605/jafes.040.S1.081>

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INTRODUCTION/BACKGROUND

Primary hyperaldosteronism (PHA) is a frequently overlooked cause of secondary hypertension, particularly in younger adults. If untreated, it can lead to serious cardiovascular complications. Diagnosis may be challenging when investigations produce conflicting results. We present a case of resistant hypertension due to PHA, successfully treated surgically despite discordant imaging and sampling findings.

CASE

A 45-year-old male with a history of type 2 diabetes, dyslipidaemia and obstructive sleep apnoea was referred for evaluation of hypertension, first diagnosed at age 31. He had persistent hypokalaemia (2.2–2.6 mmol/L) and proteinuria (urine protein-creatinine ratio: 112.9 mg/dL). Initial work-up, including hormonal, cardiac, and renal assessments, showed no significant abnormalities.

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However, a positive aldosterone-renin ratio (ARR) of 36, with elevated aldosterone levels (1076 pmol/L) and direct renin (29.5 mU/L), along with a positive saline suppression test (post-infusion aldosterone 910 pmol/L), all confirmed the diagnosis of PHA.

CT imaging showed a small (0.6 × 0.7 cm) nodule in the left adrenal gland and a normal right gland. However, adrenal venous sampling (AVS) revealed lateralisation to the right adrenal gland, indicating it as the source of aldosterone excess. Given the patient's resistant hypertension, large pill burden (including five antihypertensives and high-dose potassium supplements), surgical management was preferred. Following a multi-disciplinary discussion, a right adrenalectomy was performed. Post-operatively, the patient showed significant clinical improvement, reducing his antihypertensive regimen from five to three medications, and potassium supplementation was no longer needed.

CONCLUSION

This case highlights the critical role of accurate ARR sampling and strict adherence to the diagnostic pathway in evaluating PHA. Relying solely on CT imaging can be misleading, particularly with small adrenal lesions, making AVS essential for precise localisation. A systematic, stepwise approach is key to achieving optimal treatment outcomes.

EP_A074

FLORID ECTOPIC CUSHING SYNDROME FROM AN UNRESECTABLE MEDIASTINAL NEUROENDOCRINE TUMOUR

<https://doi.org/10.15605/jafes.040.S1.082>

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INTRODUCTION/BACKGROUND

Ectopic adrenocorticotrophic hormone (ACTH) Cushing syndrome (ECS) is rare but frequently a severe condition because of the intensity of hypercortisolism. 50% of ECS originates primarily from neuroendocrine tumours (NETs)

of the lung. NETs from the mediastinum are extremely rare; they often arise from the thymus gland or paraganglionic structures.

CASE

A 46-year-old male presented with altered behaviour and fatigue. On examination, the patient was hypertensive at 184/91 mm Hg, lean with a BMI of 23 kg/m² and with physical examination findings of hyperpigmented palmar crease, acanthosis nigricans, and generalised acne. Laboratory investigations revealed severe hypokalemia (1.6 mmol/L) and metabolic alkalosis (pH- 7.755, HCO₃⁻ 62.5). ODST was not suppressed (1519 nmol/L) and 24-hour urine cortisol was elevated at 16,198 nmol. ACTH was increased at 70.40 pmol/l (1.6-13.9) and HbA1c was 5.1%. No pituitary adenoma was noted from the pituitary MRI. The whole body CT reported an anterior mediastinal mass with the largest diameter at 9.2 cm and a T8 vertebrae compression fracture. Functional PET-CT showed predominant avidity in the FDG-PET compared to the Gallium-PET scan. CT-guided biopsy confirmed an intermediate-grade NET (atypical carcinoid). The mass was unresectable as it encased the great vessels. We commenced oral ketoconazole to control his hypercortisolemic state and IM Octreotide LAR 30 mg four times weekly. He responded well; his repeat morning cortisol ranged between 252 and 327 nmol/L. We titrated down his anti-hypertensives, ketoconazole, and potassium replacements. However, four months later, he was readmitted for symptomatic severe hypokalemia and raised cortisol level (1453 nmol/l). The repeat imaging showed progressive disease, now with metastasis to the lung, scapula and tumour thrombosis. Chemotherapy with Etoposide and Carboplatin was initiated. Unfortunately, the patient succumbed to sepsis after his second cycle of chemotherapy.

CONCLUSION

The primary treatment of ECS is surgical resection of the ACTH-secreting tumour. Other treatment options are chemotherapy, somatostatin analogues and radiotherapy. Medical therapy with adrenal enzyme synthesis inhibitors may be needed to control the degree of hypercortisolemia.

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EP_A075

DEFYING THE PROGNOSIS: LONG-TERM SURVIVAL IN ADVANCED ADRENOCORTICAL CARCINOMA WITH MULTIMODAL THERAPY

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INTRODUCTION/BACKGROUND

Adrenocortical carcinoma (ACC) is a rare and aggressive malignancy with poor prognosis. Long-term survival is challenging due to high recurrence rates and limited treatment options. A multimodal treatment strategy includes incorporating surgical resection, systemic therapy and radiotherapy. Stereotactic body radiotherapy (SBRT), a highly precise form of radiation therapy, targets tumours effectively with minimal surrounding damage.

CASE

A 33-year-old Chinese female presented with a one-month history of low back pain and constitutional symptoms. The abdominal CT scan revealed a 15 x 15 cm left adrenal mass with inferior vena cava (IVC) thrombosis and lung metastases. She underwent extensive surgery with complete surgical resection of the adrenal tumour, including left adrenalectomy, IVC thrombectomy, splenectomy and lung metastasectomy. Histopathologic examination confirmed metastatic ACC with Ki-67 proliferation of 40%. One month post-operatively, mitotane was initiated and titrated to a maximum dose tolerable at 3 g daily. Sorafenib was trialed but discontinued after four months due to adverse effects. Seven months post-operation, the PET scan revealed FDG-avid in the right upper lobe lung nodule with active IVC thrombus. A multidisciplinary team deemed the thrombus inoperable. Hence, she underwent 10 cycles of SBRT. A repeated FDG PET scan 8 months later showed a right upper lobe nodule and IVC thrombus resolution. At 4- and 6-years post-surgery, the PET dotatate scan revealed a dotatate-avid lesion at the right upper and left upper lobes. Hence, we proceeded with a biopsy, and the HPE examination showed only benign findings. Subsequent FDG-PET/CT scans revealed FDG-avid hypermetabolic activity in the lungs, consistent with bronchiectasis and plate atelectasis, but no signs of local recurrence.

CONCLUSION

Despite the typically poor prognosis of advanced ACC, this patient achieved long-term survival beyond eight years through a comprehensive, individualised treatment strategy, including complete surgical resection, systemic therapy, targeted SBRT and close multidisciplinary follow-up. This case highlights the potential role of SBRT in managing ACC and underscores the importance of coordinated, patient-specific oncologic care.

EP_A076

HYPOKALEMIA AS A HIDDEN CAUSE OF CUSHING DISEASE

<https://doi.org/10.15605/jafes.040.S1.084>

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INTRODUCTION/BACKGROUND

Cushing disease is caused by an adrenocorticotrophic hormone (ACTH)-secreting pituitary adenoma, and it comprises 70% of endogenous Cushing syndrome. Cushing disease is rare and is associated with high morbidity and mortality. The diagnosis is often delayed due to its manifestations of variable clinical features. Cortisol also has mineralocorticoid activity, and hypokalemia occurs when severe hypercortisolism occurs.

CASE

A 34-year-old female with a past medical history of type 2 diabetes mellitus, hypertension and polycystic ovarian syndrome. She was admitted for the first time 5 years ago for uncontrolled diabetes mellitus and complained of subacute onset of recurrent bilateral lower limb weakness and excessive weight gain. There was no history of neck trauma, gastrointestinal losses, thyrotoxic symptoms and treatment with corticosteroids. Physical examination revealed classical cushingoid features with truncal obesity, thin limbs, moon face, facial acne, dorsocervical fat pad and purplish abdominal striae. She was hypokalemic with serum potassium of 2.6-2.8 mmol/L and alkalotic with a bicarbonate level of 32 mmol/L. The thyroid function test and serum magnesium were normal. She received both enteral and parenteral potassium supplementation. Cushing syndrome was considered and further evaluation confirmed ACTH-dependent Cushing syndrome with a non-suppressed overnight dexamethasone test with raised serum cortisol, 24-hour urinary cortisol and ACTH. The pituitary MRI showed a microadenoma (8.2 x 9.4 x 8.3 mm). She was started on steroidogenesis inhibitors (ketoconazole) preoperatively. She underwent trans-

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sphenoidal surgery (TSS), and histopathology confirmed a pituitary adenoma. However, she still had persistent Cushing disease post-operatively with non-suppressed serum cortisol, poor glycemic control with HbA1c of 11-13% and mild hypokalemia. A repeat pituitary MRI was scheduled, and a repeat TSS is likely warranted.

CONCLUSION

Although hypokalemia is not a determining feature of CD, it can be a significant presentation. Hence, a high index of clinical suspicion of the possible etiologies in evaluating hypokalemia is essential.

EP_A077

A RARE CASE OF THIOAMIDE-INDUCED PANCYTOPENIA

<https://doi.org/10.15605/jafes.040.S1.085>

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INTRODUCTION

Thioamides play a central role in the management of hyperthyroid disorder due to their efficacy and relatively lower risk of adverse events. While serious adverse effects are relatively uncommon, the more frequently reported are agranulocytosis, hepatotoxicity and vasculitis. Notably, propylthiouracil has been associated with a higher incidence and severity of agranulocytosis and hepatic dysfunction compared to carbimazole. We report a case of a patient with toxic multinodular goitre who developed pancytopenia shortly after initiation of various thioamide agents.

CASE

A 72-year-old female with toxic multinodular goitre developed recurrent neutropenic sepsis following exposure to multiple thioamides. She was initially treated with carbimazole but was complicated with neutropenic sepsis after 2 weeks of treatment; hence, she was switched to cholestyramine and prednisolone. Due to a lack of clinical response, propylthiouracil was introduced, resulting in initial improvement but with subsequent pancytopenia. Iodine therapy was then attempted but failed to produce clinical benefit. A low dose of methimazole was initiated as a final medical option, which eventually precipitated a third episode of neutropenic sepsis. In all three episodes, she was treated with appropriate antibiotics and received granulocyte-colony stimulating factor (G-CSF) support, leading to hematologic recovery. Extensive work-up excluded other potential causes of pancytopenia. Eventually, despite persistently elevated thyroid hormone levels and

being at a high risk of intra-operative thyroid crisis, she underwent a successful semi-emergency total thyroidectomy following a multi-disciplinary team discussion.

CONCLUSION

This case highlights a rare and potentially life-threatening complication associated with thioamides, distinct from more commonly observed isolated agranulocytosis, emphasising the need for heightened vigilance when prescribing these medications.

EP_A078

PROLONGED HYPOTHYROIDISM AS A RARE COMPLICATION AFTER ANTITHYROID TREATMENT FOR A PATIENT PRESENTING WITH THYROID STORM

<https://doi.org/10.15605/jafes.040.S1.086>

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INTRODUCTION/BACKGROUND

Hypothyroidism rarely occurs following anti-thyroid therapy (ATT). We present a case of prolonged hypothyroidism following ATT for thyroid storm.

CASE

A 50-year-old female presented to the emergency department with a 3-week history of failure symptoms, 10 kg weight loss and diarrhoea. She was in respiratory distress, hypotensive with a high fever and had atrial fibrillation in rapid ventricular response (170 beats/min) with congestive heart failure. She had no goitre or ophthalmopathy. She was diagnosed with thyroid storm (Burch-Wartofsky Score 90) with free T4 79.9 pmol/L and TSH <0.005 IU/L. Despite prompt initiation of carbimazole, IV hydrocortisone, Lugol's iodine, non-invasive ventilation, IV amiodarone and electrical cardioversion, she suffered cardiorespiratory arrest. She was revived after cardiorespiratory resuscitation, intubation and triple inotropic support. Her 21-day ICU stay was eventful with multiorgan failure (ischaemic hepatitis, cardiogenic shock, oliguric kidney injury) complicated by nosocomial infection, critical illness myopathy and bed sore. She spent three months in the hospital, including one month of inpatient rehabilitation. Thyroid-wise, she responded to ATT with fT4 dropping to 36 pmol/L on day 3 of admission. All ATT was discontinued on day 11 when fT4 was reduced to 3.64 pmol/L and TSH <0.005 IU/L. On Day 28, her fT4 remained suppressed, reaching a nadir of 1.36 pmol/L (TSH 0.084 IU/L, fT3 1.60 pmol/L [normal

Adult E-Poster

3.1-6.8 pmol/L)). Her fT4 rebounded to 47.80 pmol/L (TSH<0.005 IU/L) after 6 weeks (or one month from the last dose of carbimazole). Carbimazole was reintroduced and continued up to her recent follow-up at 2 months post-discharge.

CONCLUSION

Hypothyroidism can occur with ATT for primary hyperthyroidism due to overdosage or increased individual sensitivity, but it is usually short-lived. Prolonged hypothyroidism shortly after presentation of thyroid storm is unusual. Possible explanations include the presence of TSH blocking or stimulating antibodies, sick euthyroid syndrome and the elusive “shock thyroid.” A thyroid storm due to a thyrotoxic phase of thyroiditis is unlikely here due to the subsequent relapse of thyrotoxicosis. Careful clinical assessment and monitoring are essential to guide treatment direction.

EP_A079

UNMASKING MACRO-TSH: A CASE SERIES

<https://doi.org/10.15605/jafes.040.S1.087>

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INTRODUCTION/BACKGROUND

Discrepancies between biochemical findings and clinical presentation—particularly isolated elevations in thyroid-stimulating hormone (TSH) with normal free thyroxine (FT4) and the absence of hypothyroid symptoms should prompt the consideration of assay interference. Macro-TSH is one of the important possible causes that should be considered. Failure to recognise macro-TSH can result in unnecessary investigations and inappropriate treatment. We describe two middle-aged male patients, both without a family history of thyroid disorders, who were referred for evaluation of discordant thyroid function tests.

CASE

Case 1. A 52-year-old male with long-standing Type 2 diabetes and chronic kidney disease Stage 3a was referred for an abnormal thyroid function test (TFT). His TSH was 7.83 uIU/L (0.35-4.94), while free T4 (FT4) was within the normal limit at 16.59 pmol/L (9-19.05). Polyethylene glycol (PEG) precipitation was 0.67 uIU/mL, with a recovery rate of 93% and a confirmed diagnosis of macro-TSH.

Case 2. A 29-year-old male had been treated for hypothyroidism with levothyroxine for 10 months following an initial TSH of 12.37 uIU/mL and free T4 of 13.27 pmol/L. Despite adherence to treatment and titrating doses of thyroxine, his TSH persistently rose to 86.06 uIU/mL with free T4 of 11.64 pmol/L. He remained clinically euthyroid. PEG precipitation revealed pre-precipitation TSH of 76.46 uIU/mL with 84% recovery and post-precipitation TSH of 11.88 uIU/mL. These findings confirmed the presence of macro-TSH and led to the cessation of thyroxine treatment.

CONCLUSION

These cases underscore the importance of considering macro-TSH in patients with elevated TSH and normal FT4 who lack clinical symptoms of hypothyroidism. Failure to recognise this phenomenon may result in misdiagnosis and inappropriate treatment. PEG precipitation testing is a valuable tool in confirming macro-TSH and guiding appropriate clinical decision-making.

EP_A080

MUSCLE UNDER SIEGE: A CASE OF POST-BARIATRIC SURGERY RHABDOMYOLYSIS

<https://doi.org/10.15605/jafes.040.S1.088>

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INTRODUCTION/BACKGROUND

Rhabdomyolysis after bariatric surgery is rare and under-recognised. It can lead to acute kidney impairment with an associated 25% risk of mortality. We report a patient with rhabdomyolysis after sleeve gastrectomy.

CASE

A 48-year-old male patient who has class III obesity (body mass index of 70 kg/m²) was admitted for bariatric surgery. His medical history was significant for hypertension, gouty arthritis and moderate obstructive sleep apnea, with an American Society of Anesthesiologists (ASA) III physical status. He received 3 weeks of in-patient meal replacement therapy with a very low-calorie liquid diet and resistance exercise program before his operation. Intra-operatively, he was placed in a reverse Trendelenburg position. Initially, laparoscopic sleeve gastrectomy was planned, but a switch to open surgery was made due to technical difficulties. The total duration of surgery was 554 minutes. Post-operatively, the patient had a blister and grade II pressure injury at the left gluteus. He was oliguric (urine output less than 0.1 ml/kg/day) with elevated blood creatine kinase

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(>22000 U/L at 36th-hour post-op) and stage 3 acute kidney injury (serum creatinine 360 $\mu\text{mol/L}$). He was diagnosed with rhabdomyolysis and was co-managed with the nephrology team, whereby aggressive fluid replacement with diuresis was initiated. He did not require kidney replacement therapy throughout his course of recovery. On day 10 post-op, the laboratory findings normalised and the patient was discharged home fully recovered.

CONCLUSION

Postoperative rhabdomyolysis is a severe complication of bariatric surgery, which is potentially life-threatening. Creatine kinase testing should be performed in high-risk patients after bariatric surgery for timely diagnosis and interventions.

EP_A081

NON-ISLET CELL TUMOR SECONDARY TO MALIGNANT PHYLLODES TUMOR OF BREAST

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INTRODUCTION/BACKGROUND

Non-islet cell tumour-induced hypoglycaemia (NICTH) is a rare but important cause of recurrent hypoglycaemia in patients with non-pancreatic tumours. Unlike insulinomas that cause hypoglycaemia through excess insulin secretion, NICTH is associated with large mesenchymal or epithelial tumours producing high-molecular-weight insulin-like growth factor 2 (IGF-2), leading to insulin-independent hypoglycaemia. We report a case of NICTH in a patient with a malignant phyllodes tumour of the breast.

CASE

A 50-year-old female was found unresponsive at home with a blood glucose level of 2.3 mmol/L. She regained consciousness following the administration of IV glucose. She had no history of diabetes or use of glucose-lowering agents. Examination revealed a large, firm 20 × 20 cm left breast mass. Hypoglycaemia work-up showed a random glucose level of 3.0 mmol/L, C-peptide of 35 pmol/L and insulin <2.78 pmol/L, suggesting hypoinsulinaemic hypoglycaemia. IGF-1 was within the normal range. She was treated with glucocorticoids while awaiting surgery. She underwent a left mastectomy, which revealed a 16 × 12.5 × 22.5 cm 7.6-kg malignant phyllodes tumour. Histopathology examination confirmed a malignant phyllodes tumour with high mitotic activity and a high risk of recurrence. An oncology referral was made for adjuvant therapy. At

one-month follow-up, she remained asymptomatic with no hypoglycemia.

CONCLUSION

NICTH should be considered in patients with large tumours presenting with hypoglycemia. Corticosteroids may help manage hypoglycaemia before surgery, which remains the definitive treatment. A multi-disciplinary approach is essential for optimal care.

EP_A082

NOCTURNAL HYPOGLYCEMIA: THE TUMOR YOU DON'T SEE, BUT YOUR BLOOD SUGAR DOES

<https://doi.org/10.15605/jafes.040.S1.090>

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INTRODUCTION/BACKGROUND

Non-islet cell tumour hypoglycemia (NICTH) is a rare but potentially life-threatening complication of malignancy, often driven by tumour overproduction of insulin-like growth factor 2 (IGF-2). Diagnosis can be challenging due to non-specific symptoms and limited access to specialised assays.

CASE

We report the case of an 87-year-old female with no known medical history who presented with reduced consciousness and was found to have symptomatic hypoglycemia with capillary glucose 2.1 mmol/L. She had experienced unexplained hypoglycemic episodes over the past 3 months. During hospitalisation, she showed a pattern of nocturnal hypoglycemia that temporarily resolved with continuous dextrose infusion, fulfilling Whipple's triad. The laboratory work-up revealed low serum insulin, low C-peptide, low insulin-like growth factor, negative serum sulfonylurea screen and normal random serum cortisol. Unfortunately, IGF-2 measurement was not available. A contrast-enhanced CT (CECT) of the thorax and abdomen exposed a large left lung mass with features suggestive of malignancy. The patient was initiated on glucocorticoid therapy, which led to partial improvement, although nocturnal hypoglycemic episodes persisted. Given her advanced age and overall condition, she declined surgical intervention and opted for conservative management.

CONCLUSION

This case underscores the importance of considering NICTH in elderly patients with recurrent, unexplained

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hypoglycemia, particularly when occurring nocturnally. IGF-2 overproduction, typically by large mesenchymal or epithelial tumours, is the underlying reason behind these events. Although surgical resection of the tumour remains the definitive treatment, glucocorticoids and frequent glucose supplementation are used in conservative management.

EP_A083

MEN TYPE 2B SYNDROME IN A NORMOTENSIVE YOUNG FEMALE WITH INCIDENTALLY DISCOVERED PHAECHROMOCYTOMA

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INTRODUCTION/BACKGROUND

Phaeochromocytomas (PC) account for up to 5-25% of adrenal incidentalomas. Some PC patients, especially those with an adrenal incidentaloma, are asymptomatic and have normal blood pressure. The frequency of incidentally discovered normotensive phaeochromocytomas is increasing owing to better accessibility of imaging procedures. These tumours may be linked to certain genetic syndromes, such as Multiple Endocrine Neoplasia (MEN) type 2B, a rare condition caused by the RET proto-oncogene mutation, and includes a range of clinical manifestations such as phaeochromocytomas, medullary thyroid carcinoma and mucosal neuromas.

CASE

A 34-year-old female who had undergone total thyroidectomy for medullary thyroid carcinoma (MTC) was referred for an incidental right adrenal mass from CT staging. She had no paroxysms of headache or palpitations, no family history of MTC, pheochromocytoma and MEN-related diseases. She was normotensive. Physical examinations revealed mucosal neuromas on the tongue, buccal mucosa, lips, and eyelids. No marfanoid habitus present. Laboratory results showed normal serum calcium (2.57 mmol/L), but a 24-hour urine metanephrine was four times the upper limit of normal, along with borderline elevation of normetanephrines. The adrenal CT revealed an indeterminate right adrenal mass measuring 2 x 2 x 3 cm with peripheral calcifications suggesting pheochromocytoma.

Left lymph node and carotid sheath biopsy were reported as features consistent with ganglioneuroma. The patient is scheduled for a right retroperitoneoscopic adrenalectomy. The unifying clinical presentations are consistent with MEN 2B Syndrome. However, genetic panel testing was not done due to financial constraints.

CONCLUSION

This case underscores the importance of considering genetic syndromes, such as MEN type 2B, in patients with incidental findings of pheochromocytomas, even when the patient is normotensive. Early diagnosis and genetic testing can help guide management, including surveillance for other tumours associated with MEN type 2B and early intervention. Further research is needed to explore the clinical presentation of pheochromocytomas in normotensive patients.

EP_A084

PSEUDOACROMEGALY IN A PATIENT WITH PACHYDERMOPERIOSTOSIS

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INTRODUCTION/BACKGROUND

Patients who are clinically suspected of acromegaly are usually referred to an endocrinologist. Biochemical evaluation is necessary to confirm the presence of an abnormality in the growth hormone (GH) axis. There are some conditions with physical features mimicking acromegaly in the absence of GH excess. Given the heterogeneity of conditions that can cause pseudoacromegaly, this posed a diagnostic challenge.

CASE

A 32-year-old male was referred to the Endocrine clinic for evaluation of suspected acromegaly. He had an incidental finding of pancytopenia and chronic excessive sweating when he presented to his General Practitioner. Following this, he was diagnosed with myelofibrosis by a haematologist. He reported having large hands and feet since his teens. He was also referred to a dermatologist for generalised skin thickening and itchiness and treated for photodermatitis.

Clinically, he has clubbing of the fingers of his hands and feet and furrowing of skin on his forehead but no other phenotypical features of acromegaly. Biochemically, IGF-1 and GH levels were normal.

Adult E-Poster

He had a left leg X-ray followed by a CT of the left tibia and fibula for a swelling on his left lower leg, which showed wavy periosteal thickening in the tibia and fibula suggestive of hypertrophic osteoarthropathy. With a suspicion for primary hypertrophic osteoarthropathy (PHO), it was confirmed through genetic analysis that he has homozygous pathogenic variants identified in SCLO2A1 associated with an autosomal recessive PHO.

CONCLUSION

Primary hypertrophic osteoarthropathy, or pachydermo-periostosis (PDP), is a rare genetic disorder characterised by digital clubbing, periostosis and pachydermia. Myelofibrosis is a complication of PDP where bone marrow becomes scarred and fibrotic. In patients with features of hypertrophic osteoarthropathy and acromegaly, PDP should be considered as part of the differential diagnoses.

EP_A085

ECTOPIC CUSHING'S SYNDROME: THE LONG HUNT FOR THE ELUSIVE CULPRIT

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INTRODUCTION/BACKGROUND

Ectopic Cushing's Syndrome (ECS), caused by non-pituitary ACTH-secreting tumours, is a rare but life-threatening form of hypercortisolism. Diagnosis and management can be challenging due to the small, indolent tumours of variable locations. We present two cases highlighting the complexity of diagnosing and managing ECS.

CASE

A 70-year-old male presented with hypertensive urgency, hypokalemia (K^+ 1.9 mmol/L), and new-onset diabetes mellitus (HbA1c 7.1%). He required four antihypertensives, dual oral antidiabetic therapy and potassium supplementation. Investigations revealed markedly elevated cortisol (3026 nmol/L), non-suppressible with dexamethasone (1750 nmol/L), and high ACTH (500 pg/mL) consistent with ACTH-dependent Cushing's Syndrome (CS). Initial Thorax-Abdomen-Pelvis CT, pituitary MRI and Gallium-68 PET scans were unremarkable. Treatment with ketoconazole and spironolactone led to clinical improvement, allowing discontinuation of antihypertensives, antidiabetics and potassium supplements. Serial CT TAP

later detected an enlarging 1.2 cm right middle lobe lung nodule. Surgical resection confirmed an ACTH-positive carcinoid tumour. The patient remained in remission for 6.5 years post-operatively.

A 59-year-old female with poorly controlled hypertension and diabetes was found to be cushingoid during hospitalisation for a finger abscess. Cortisol was 1164 nmol/L, ACTH 19.5 pmol/L, with non-suppression to dexamethasone. Conventional imaging (CT TAP, pituitary MRI, PET scan) showed no significant abnormality. However, IPSS confirmed an ectopic ACTH source. She exhibited cyclical CS, which was marked by fluctuations in blood pressure, glucose, potassium levels, weight and oedema. Management required a block-and-replace regimen using ketoconazole and hydrocortisone. A Ga-68-DOTATATE PET scan two years later revealed a DOTATATE-avid right lung nodule, but the biopsy was inconclusive. The patient declined further procedures.

CONCLUSION

These cases highlight the diagnostic complexity of ECS, which has required multimodal and serial imaging over the years due to elusive lesions. Biochemical control can be challenging due to cyclical CS demanding balance to avoid complications. Persistent localisation efforts remain essential as surgical resection is potentially curative.

EP_A086

LEFT ADRENAL TUBERCULOSIS MIMICKING PHAECHROMOCYTOMA POSSIBLY DUE TO RIFAMPICIN INTERFERENCE IN URINE METANEPHRINES

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INTRODUCTION/BACKGROUND

Phaeochromocytoma classically presents with uncontrolled hypertension and paroxysms of headache, diaphoresis and palpitations. The measurement of 24-hour urinary metanephrines is one of the standard first-line tests for detecting phaeochromocytoma. False elevation results may

Adult E-Poster

be brought about by various factors such as urine volume, medication interference and certain foods.

CASE

We report a case of a 67-year-old male with Hepatitis B and smear-positive pulmonary tuberculosis on maintenance treatment with rifampicin and isoniazid, who was referred for left adrenal incidentaloma from CT of the hepatobiliary system. He denied any paroxysmal symptomatology of pheochromocytoma and was normotensive. 24-hour urinary metanephrines revealed significantly elevated normetanephrine (14 times the upper limit of normal [30.15 µmol/day]), with normal metanephrine and 3-methoxytyramine levels. The adrenal CT demonstrated a left adrenal mass measuring 2.7 x 1.4 x 2.6 cm, 32 Hounsfield units (HU), with absolute and relative washout of 62.8% and 19.6%, respectively, indicating an indeterminate adrenal mass. The patient was diagnosed with left pheochromocytoma and underwent laparoscopic left adrenalectomy with Phenoxybenzamine cover. However, the histopathological findings revealed multiple granuloma formation, with special stains negative for acid-fast bacilli, suggestive of chronic changes of right adrenal tuberculosis (non-active) and no features of pheochromocytoma. Thoracic and abdominopelvic CT scans showed no evidence to suggest paraganglioma, which might contribute to elevated normetanephrine levels. A post-operative repeat 24-hour urine metanephrine came back normal. This repeated sample was taken after the patient completed tuberculosis treatment (including rifampicin). Some reports recognised rifampicin interference with urinary metanephrine measurement as it is eluted with normetanephrine, causing significantly elevated levels. These findings correlate with this patient as urine normetanephrine returned to normal once he was off rifampicin.

CONCLUSION

Histopathological findings of the left adrenal mass were suggestive of post-adrenal tuberculosis rather than pheochromocytoma. Rifampicin was found to be an interferent in urine metanephrines measurement, which led to falsely elevated normetanephrine levels with no catecholaminergic signs or symptoms.

EP_A087

A RARE CASE OF TURNER MIMICKER

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INTRODUCTION/BACKGROUND

Primary amenorrhea and delayed puberty are frequently encountered in primary care, prompting suspicion of Turner syndrome, especially in cases with short stature. This case underscores the importance of considering Swyer syndrome even when significant growth impairment is present.

CASE

A 28-year-old phenotypic female, born of a non-consanguineous union, presented with primary amenorrhea and a short stature of 1.31 meters. Physical examination revealed absent secondary sexual characteristics (Tanner stage 1). External genitalia were unambiguously female. Bone age assessment identified significant delay, corresponding to a 15-year-old. The hormonal evaluation showed hypergonadotropic hypogonadism. Thyroid function and insulin growth factor-1 levels were normal. Pelvic MRI demonstrated an atrophic uterus, absent fallopian tubes and ovaries. Karyotype analysis confirmed a 46, XY genotype, consistent with Swyer syndrome. Following pubertal induction for 3 months, she developed regular menstruation and progression to Tanner stage 3.

Swyer syndrome is a rare disorder of sex development featuring female phenotype, hypergonadotropic hypogonadism and streak gonads. While 15-20% of cases result from SRY gene mutations impairing testis-determining factor function, other genes have also been implicated. Swyer syndrome classically causes tall stature from estrogen-deficient delayed epiphyseal fusion. However, our case exhibited profound short stature and severely delayed bone age, explained by the complete prepubertal estrogen deprivation abolishing both growth spurt and fusion. Additional factors, like SHOX gene variations, may have contributed to her growth impairment. Diagnostic complexity arose from initial Turner syndrome overlap; however, the absence of other Turner stigmata and 46, XY karyotype confirmed Swyer syndrome. This emphasises karyotyping's diagnostic importance in primary amenorrhea with hypergonadotropic hypogonadism, regardless of phenotype. Hormone replacement therapy remains crucial for puberty induction, bone health and cardiovascular protection.

Adult E-Poster

CONCLUSION

Swyer syndrome may mimic Turner syndrome in cases of primary amenorrhea with short stature. Accurate diagnosis requires comprehensive hormonal, imaging and genetic evaluation beyond clinical phenotype alone.

EP_A088

UNRAVELLING THE MYSTERY: A CASE OF ATYPICAL DIABETES WITH HEPATIC AND RENAL CLUES TO HNF1B DEFICIENCY

<https://doi.org/10.15605/jafes.040.S1.096>

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INTRODUCTION/BACKGROUND

Hepatocyte nuclear factor 1 β (HNF1B) deficiency associated with MODY-5 is increasingly recognised as a multifaceted syndrome with diverse manifestations. We present a suspected case initially misdiagnosed as type 1 diabetes with autoimmune hepatitis.

CASE

A 14-year-old male with learning disability was admitted for insulin initiation when he presented with osmotic symptoms with hyperglycaemia and ketonuria. He reported no family history of diabetes. HbA1c was 18.5% and LFTs were deranged (AST 74, ALT 209 and ALP 451 IU/L). He has some dysmorphic facial features. Despite good glycemic control on intensive insulin therapy, his liver enzymes remained elevated (8-17x ULN) with normal ferritin, ceruloplasmin and viral panel. Abdominal ultrasound showed normal liver and spleen but detected bilateral medullary nephrocalcinosis. The liver biopsy showed mild periportal hepatitis. He was treated for autoimmune hepatitis with prednisolone and azathioprine. Subsequent investigations revealed negative diabetes (anti-GAD, ICA, IA2), hepatic (ANA, smooth muscle, LC1, LKM and mitochondrial) autoantibodies and normal serum immunoglobulins. The absence of diabetes-related autoantibodies, coupled with multisystem involvement (pancreas, liver, kidney, neurocognitive and dysmorphism), raised the suspicion of HNF1B mutation. Although genetic confirmation was not feasible, further investigation with elevated C peptide (1652 pmol/L) and persistent hypomagnesemia (0.4 to 0.55 mmol/L) further substantiated this hypothesis. Immunotherapy was withheld. He remained well with fluctuating liver function on follow-up 5 years since the initial presentation.

CONCLUSION

This case underscores the diagnostic complexity of HNF1B deficiency, a rare monogenic diabetes subtype accounting for ~6% of MODY. Despite an autosomal dominant inheritance pattern, de-novo mutation accounts for 50% of cases. Lack of family history does not preclude the diagnosis. Diagnostic clues include multisystem involvement, which is rarely found in other MODY subtypes. Hypomagnesemia is another common feature. Early recognition is essential for individualised management, avoidance of mismanagement, monitoring for other organ involvement or complications and genetic counselling.

EP_A089

TWIN-TWIN TRANSFUSION SYNDROME ASSOCIATED MATERNAL HYPERTHYROIDISM

<https://doi.org/10.15605/jafes.040.S1.097>

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INTRODUCTION/BACKGROUND

Pregnancies complicated by twin-twin transfusion syndrome (TTTS) are associated with elevated human chorionic gonadotropin (hCG) compared to uncomplicated twin pregnancies. Studies have shown a positive correlation between hCG and free thyroxine (FT4) in TTTS, thereby increasing the risk of maternal hyperthyroidism. This case report describes a twin pregnancy complicated by TTTS, where maternal hyperthyroidism developed prior to fetoscopic laser ablation (FLA).

CASE

We present a 36-year-old female with a twin pregnancy complicated by TTTS. She was diagnosed with gestational transient thyrotoxicosis (GTT) at 10 weeks of gestation with thyroid stimulating hormone (TSH) of 0.01 mIU/L, FT4 of 24.8 pmol/L and triiodothyronine (T3) of 3.8 pmol/L. She had negative thyroid-stimulating hormone receptor antibodies and a normal neck ultrasound. Clinically, she has no goitre or thyroid eye disease. At 15 weeks of gestation, her FT4 decreased to 14.3 pmol/L while TSH remained suppressed. She did not receive any anti-thyroid drugs (ATDs) during the first trimester. She was admitted at 22 weeks of age of gestation for FLA due to TTTS stage 1. Upon admission, she complained of palpitations, and the cardiac monitor showed sinus tachycardia with a heart rate of 123 bpm. Her TSH was <0.008 mIU/L, FT4 was increased to 21 pmol/L and hCG of >225,000U/L. Due to hyperthyroid symptoms, she was treated with carbimazole and beta-blocker prior to FLA. Her carbimazole dose was reduced at 25 weeks of gestation as FT4 dropped to 13.2 pmol/L. It was then

Adult E-Poster

discontinued at 28 weeks of gestation (FT4 11.25 pmol/L; TSH 0.11 mIU/L). She underwent emergency hysterectomy at 28 weeks of gestation due to TTTS progression to stage 4.

CONCLUSION

GTT in twin pregnancies typically resolve by the end of the first trimester. A sustained FT4 increase should raise suspicion for TTTS. ATDs should be considered due to the risk of TTTS-associated maternal hyperthyroidism, as it may persist until successful FLA.

EP_A090

WHEN LIGHTNING STRIKES TWICE: A CASE OF METACHRONOUS INVASIVE BREAST CARCINOMA AND PAPILLARY THYROID CARCINOMA IN A FEMALE FILIPINO PATIENT

<https://doi.org/10.15605/jafes.040.S1.098>

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INTRODUCTION/BACKGROUND

Triple-negative invasive ductal carcinoma is a more aggressive type of breast cancer that poses therapeutic challenges. Papillary thyroid carcinoma is generally indolent but has shown aggressive behaviour among Filipinos. As primary carcinomas, both tend to have a good prognosis with early detection and management. However, failure to anticipate a secondary malignancy, when one occurs after the other, can turn a treatable journey into a devastating outcome.

CASE

A 45-year-old female presented with a movable left breast lump 3 years ago. Biopsy confirmed invasive ductal carcinoma with a negative ER/PR/HER2 on immunohistochemistry. She eventually underwent a modified radical mastectomy of the left breast and staged as 2B (T2N1M0) due to the absence of lympho-vascular space invasion and distant metastasis. She completed eight cycles of adjuvant chemotherapy with Doxorubicin and Cyclophosphamide. Post-chemotherapy surveillance confirmed the absence of metastasis. A 2 x 2 cm thyroid nodule was detected on the left anterior neck two years later during routine follow-up. Ultrasound revealed a lobulated solid hypoechoic wider-than-tall nodule in the superior pole of the left lobe (TI-RADS 5). The patient was clinically and biochemically euthyroid. Ultrasound-guided fine needle biopsy identified the presence of Papillary Thyroid Carcinoma (Bethesda Category VI). As such, the patient underwent a total thyroidectomy. Final histopathologic

studies confirmed a classic subtype of Papillary Thyroid Carcinoma (ATA Low Risk) without lymphatic, perineural, extrathyroidal invasion and regional lymph node metastasis. Post-operative high-dose radioactive iodine was administered to eliminate any residual thyroid tissue. She was then maintained on levothyroxine suppression and continuously monitored for tumour recurrence.

CONCLUSION

As better understanding of tumorigenesis has revolutionised cancer screening and management, the metachronous coexistence of breast and thyroid carcinoma highlights the importance of multidisciplinary care and vigilant screening for secondary malignancies. Overexpression of estrogen and progesterone, together with shared environmental and genetic factors in breast cancer, have been shown to promote thyroid tumorigenesis and progression.

EP_A091

EXPERIENCE OF CINACALCET TREATMENT DURING PREGNANCY IN PRIMARY HYPERPARATHYROIDISM

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INTRODUCTION/BACKGROUND

Primary hyperparathyroidism (PHPT) in pregnancy is rare but associated with high maternal (68.6%) and foetal (80%) complications, including pre-eclampsia, miscarriage and intrauterine growth restriction. The risks are directly related to the severity of the disease and the serum calcium level. We describe two cases with differing clinical outcomes based on the timing of diagnosis and intervention.

CASE

A 30-year-old gravida 3, para 2, presented with maternal tachycardia at 27 weeks of gestation. On work-up, the patient was incidentally found to have hypercalcaemia. ECG showed a shortened QTc. Biochemically, her calcium was 2.99 mmol/L (Reference Value [RV]: 2.2-2.7 mmol/L), phosphate 0.7 mmol/L (RV: 0.8-1.45 mmol/L) and intact PTH level of 12.3 pmol/L (RV: 1.58-6.03), suggestive of parathyroid (PTH) dependent hypercalcaemia. Ultrasound showed an enlarged right parathyroid gland. Despite IV hydration, hypercalcaemia persisted, leading to cinacalcet initiation at 29 weeks. At 30 weeks, calcium was highest at 3.05 mmol/L. She was treated with subcutaneous salmon calcitonin (5 mg/kg/dose), which was given twice daily, and cinacalcet was titrated up to 75 mg/day. Her calcium

Adult E-Poster

decreased to 2.44 mmol/L after six doses of calcitonin. Eventually, she delivered vaginally at 38 weeks with no complications; the neonate weighed 2.58 kg.

A 33-year-old primigravida developed hypertension at 22 weeks, progressing to preeclampsia with pulmonary oedema and blurred vision at 27 weeks, requiring emergency caesarean section. The neonate weighed 980 g; placental histology showed vascular malperfusion and retroplacental haematoma. Hypertension persisted post-delivery. Retrospectively, there was an unrecognised hypercalcaemia detected from 26 weeks gestation. Postpartum laboratory showed calcium 3.04 mmol/L, phosphate 0.67 mmol/L and intact PTH 20.3 pmol/L. Ultrasound and Tc-99m Sestamibi confirmed a left inferior parathyroid adenoma. She underwent a left parathyroidectomy, and she was immediately weaned off antihypertensives.

CONCLUSION

PHPT in pregnancy poses significant maternal and foetal risks. Medical therapy options are limited during pregnancy. Early recognition and tailored management are crucial to minimise maternal and foetal complications.

EP_A092

WHEN THE CURE BITES BACK: A CASE REPORT ON CARBIMAZOLE- INDUCED MYOSITIS

<https://doi.org/10.15605/jafes.040.S1.100>

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INTRODUCTION/BACKGROUND

Musculoskeletal complaints, including myositis, are common in thyroid disorders. However, it is a rare and non-classical side effect of antithyroid drugs.

CASE

A 35-year-old male with Graves' disease was admitted for symptomatic thyrotoxicosis despite treatment with high-dose carbimazole, lithium and dexamethasone for direct observed therapy (DOTS). On examination, he exhibited signs of thyrotoxicosis, including tremors, sweaty palms, proptosis and a large goitre. Thyroid function tests revealed overt thyrotoxicosis (TSH <0.001 mIU/L, T4 122.5 pmol/L). He responded well to DOTS on the same treatment, with T4 decreasing to 27 pmol/L by Day 10 of admission. However, he developed severe proximal muscle aches on day 10. Laboratory investigation revealed an elevated creatinine

kinase (CK) level of 5662 IU/L. A diagnosis of carbimazole-induced myositis was made, prompting the discontinuation of carbimazole and initiation of intravenous hydration and cholestyramine while continuing lithium and steroids. The myositis improved, but there was a rebound in his thyrotoxicosis (T4 52pmol/L), prompting a trial of propylthiouracil, which led to a flare of the myositis (CK 1328 IU/L). He was planned for urgent total thyroidectomy then and was started on Lugol's iodine.

Myalgia and elevated CK levels are rare but recognised manifestations of hyperthyroidism. These can be due to direct effects of thyrotoxicosis on skeletal muscle, thyrotoxic hypokalemic periodic paralysis and drug-induced myositis by carbimazole and propylthiouracil. The pathophysiology of drug-induced myositis may be due to the abrupt decrease in circulating thyroid hormones, leading to a relative hypothyroid state in the peripheral tissues and resulting in myositis. Thioamides may also have a direct toxic effect on myocytes and trigger a local immune response at myocytes, leading to myositis.

CONCLUSION

This case highlights myositis as a rare side effect of anti-thyroid drugs and can cause significant morbidity. Early recognition and close monitoring are essential for managing this condition.

EP_A093

A RARE CASE OF HYPONATREMIA AS A LEADING SIGN OF EMPTY SELLA SYNDROME

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INTRODUCTION/BACKGROUND

Hyponatremia is a common electrolyte disturbance that can be life-threatening. Careful evaluation of hyponatremia may reveal significant underlying conditions and may lead to the diagnosis of a rare multiple hormone deficiency, with detrimental consequences for the patient if left untreated. We present an interesting case of a patient who presented with symptoms of chronic hyponatremia, and the results of the examination led us to the discovery of partial empty sella syndrome as the aetiology.

CASE

A 61-year-old female presented with a six-year history of general weakness, nausea, vomiting and cold intolerance.

Adult E-Poster

She was diagnosed with hyponatremia and has routinely received sodium supplementation therapy. However, the patient's complaints have worsened over the past year. There is history of severe vaginal bleeding and shock during childbirth 22 years ago, which required a hysterectomy to be performed. Physical examination showed no abnormalities. Laboratory data showed severe hyponatremia with a serum sodium of 115 meq/L and low serum osmolality, but urine osmolality and urine sodium were elevated. Renal examination showed no abnormality. Thyroid function showed a low FT4 level at <0.4ng/dL (Reference Value [RV]: 0.7-1.48 ng/dL) and normal TSH at 1.882uIU/mL (RV: 0.35-4.94 uIU/mL). A low AM cortisol level of <1.0 mcg/dL (RV: 3.7 – 19.4 mcg/dL), normal adrenocorticotrophic hormone (ACTH) level of 18.9pg/mL (RV: 7.2-63.3 pg/mL) suggested secondary adrenal insufficiency. She had low LH (6.6 mIU/mL), low FSH (11.7 mIU/mL) and low estradiol (5 pg/mL). The brain magnetic resonance imaging (MRI) confirmed the diagnosis of partial empty sella. Treatment with steroids and levothyroxine led to symptoms and laboratory resolution in a few months.

CONCLUSION

Empty sella syndrome is a rare pituitary condition that can be primary or secondary. It can potentially cause hypopituitarism that may be symptomatic or asymptomatic. This case emphasises the need for a comprehensive work-up of hyponatremia, awareness of secondary adrenal insufficiency, panhypopituitarism and recognition of the life-threatening potential of partial empty sella syndrome if left untreated.

EP_A094

AN UNUSUAL CASE PRESENTATION: MALIGNANT THYROID NODULE IN A PATIENT WITH LUNG ADENOCARCINOMA

<https://doi.org/10.15605/jafes.040.S1.102>

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INTRODUCTION/BACKGROUND

Lung adenocarcinoma is a prevalent subtype of lung cancer known for its capacity to metastasize to various organs. In patients diagnosed with this cancer, thyroid nodules pose diagnostic challenges, as they may indicate metastasis or

represent distinct conditions such as multinodular goiter or other thyroid malignancies. Notably, lung adenocarcinoma has a higher tendency to metastasize to the thyroid, with an incidence rate of 1-3%.

CASE

A 53-year-old female, undergoing chemotherapy for lung adenocarcinoma, presented to the Internal Medicine Clinic at RSUP M. Djamil Padang with a secondary thyroid nodule suspected to be metastatic. Other differential diagnoses were primary thyroid tumor and subclinical hyperthyroidism. Her history included a neck mass, hoarseness, pain while swallowing, weight loss, fatigue, and tremors. Physical examination revealed multiple nodules in the anterior neck with defined borders that moved during swallowing. Thyroid ultrasound indicated bilateral multinodular goiter (TIRADS III) without lymph node enlargement. Scintigraphy showed a cold nodule. Fine needle aspiration biopsy (FNAB) confirmed thyroid carcinoma consistent with metastasis from lung adenocarcinoma. The management plan included continuing chemotherapy and oncology consultation.

CONCLUSION

This case highlights the critical importance of early detection and management of secondary cancers to enhance patient outcomes and emphasizes the need for clinician awareness of thyroid metastasis in lung cancer, particularly lung adenocarcinoma.

EP_A095

POSTMENOPAUSAL VIRILIZATION: THE TELLTALE SIGN OF A RARE OVARIAN TUMOR

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INTRODUCTION/BACKGROUND

Postmenopausal virilization describes the occurrence of male secondary characteristics in a postmenopausal woman, contributed by excess androgen that originating from either the ovaries or adrenal glands. Relative androgen excess could be due to menopausal transition or polycystic ovarian syndrome. However, with virilizing symptoms, further investigation is warranted to look for ovarian hyperthecosis or androgen-secreting ovarian or adrenal tumors. Sertoli-Leydig cell tumors are considered significantly rare, accounting for less than 1% of all primary ovarian tumors. Diagnosis of these rare tumors can be challenging.

Adult E-Poster

CASE

We report a case of a 68-year-old woman of Indian ethnicity who first presented to us at 60 years old in 2017 with postmenopausal hirsutism. Treatment with co-cyprindiol (Diane-35) for a year and spironolactone did not alleviate her symptoms but instead worsened them with other virilizing symptoms such as deepening of voice, breast atrophy and androgenic alopecia. Testosterone levels were persistently elevated [43.757 nmol/L (December 2016) – 48 nmol/L (July 2017) - >52 nmol/L (October 2019)]. Computed tomography imaging done in 2020 showed an enlarged right ovary. She was referred to Gynecology and was given one dose of Leuporelin (Lucrin) on 11/7/2020, with the intention to assess ovarian suppression; however, elevated testosterone levels persisted at >52 nmol/L. The patient eventually underwent total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAHBSO) in May 2021 and histopathology revealed a right ovarian Sertoli-Leydig cell tumor. Post-operation testosterone levels showed rapid reduction to normal at 0.3 nmol/L and remained normal at <0.1 nmol/L in September 2021.

CONCLUSION

This case emphasizes the importance of thorough evaluation in women with postmenopausal virilization, which can be the only sign of rare ovarian tumors. Additionally, this condition can be distressing to patients and affect their quality of life, especially social interactions. The delay in her diagnosis and surgery highlights the need to increase awareness of this condition among clinicians.

EP_A096

MALIGNANT STRUMA OVARIII IN A PATIENT WITH GRAVES' DISEASE: A CASE REPORT

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INTRODUCTION/BACKGROUND

Malignant struma ovarii (MSO) is a rare ovarian teratoma containing malignant thyroid tissue, accounting for <5% of all struma ovarii cases. Papillary carcinoma is the most common histological subtype, followed by follicular carcinoma. Diagnosis may be challenging, especially when coexisting with thyroid nodules or autoimmune thyroid diseases such as Graves' disease, due to overlapping histologic and functional features.

CASE

A 58-year-old female with longstanding hyperthyroidism due to Graves' disease presented with an abdominal mass measuring 15 × 10 × 9 cm. Laparotomy in September 2023 revealed a right ovarian tumor. Histopathology showed adenomatous struma with focal atypia. Immunohistochemistry revealed BRAF V600E positivity with partial CD56 and CK19 expression, and negative HBME1 and cyclin D1. Although non-classical, this staining pattern can be observed in thyroid carcinoma with oncocytic or clear cell features.

Thyroid ultrasound showed a bilateral multinodular goiter with a TIRADS 4 nodule; FNAB was benign. A thyroid scan revealed diffusely increased uptake (55.3%), and elevated TRAb (4.43 IU/L), consistent with Graves' disease. Total thyroidectomy in June 2024 revealed adenomatous struma with chronic inflammation and no malignancy, likely representing a degenerating nodule in the context of treated Graves' disease.

This case highlights the complexity of diagnosing MSO in the setting of autoimmune thyroid disease. Total thyroidectomy was performed to exclude primary thyroid carcinoma and to support future surveillance or radioactive iodine therapy.

CONCLUSION

MSO should be considered when ovarian tumors contain thyroid tissue with atypia. In patients with Graves' disease, degenerating thyroid nodules may mimic malignancy. A multidisciplinary approach using imaging, histopathology, immunohistochemistry, and autoantibody testing is essential for accurate diagnosis and long-term management.

EP_A097

ALPHA LIPOIC ACID-INDUCED INSULIN AUTOIMMUNE SYNDROME (IAS): A REPORT OF TWO CASES

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INTRODUCTION/BACKGROUND

Insulin Autoimmune Syndrome (IAS) is a rare cause of hypoglycemia. Alpha-lipoic acid (ALA), found in Bionerv (Vitamin B supplement), induces IAS by modifying insulin structure, leading to insulin autoantibody (IAA) production in genetically susceptible individuals. Most cases are self-limiting. We present two IAS cases, emphasizing diagnostic and management challenges.

Adult E-Poster

CASE

A 49-year-old man presented with recurrent hypoglycemia. During his first clinical consult, a computed tomography (CT) scan of the pancreas was done and showed normal findings. Prolonged fasting test was consistent with endogenous hyperinsulinemic hypoglycemia. He was later referred to our center. In further history, he revealed that his symptoms started 10 days after taking Bionerv. A mixed meal test showed fasting hypoglycemia with late postprandial hypoglycemia, and markedly elevated serum insulin levels. His IAA was positive, and his sulfonylurea screen was negative. A diagnosis of ALA-induced IAS was made. Despite stopping ALA and dietary modifications, his symptoms persisted, requiring diazoxide and prednisolone. The patient was monitored with continuous glucose monitoring (CGM), which revealed episodes of alternating hyper and hypoglycemia.

A 69-year-old man with a history of thyrotoxicosis presented with symptoms of hypoglycemia. Prolonged fasting confirmed endogenous hyperinsulinemia. Abdominal CT and endoscopic ultrasound (EUS) were normal, while Gallium-68 DOTATATE imaging showed mild uptake at a 10 mm pancreatic tail nodule. He was suspected of having insulinoma and was referred to us for further assessment. Further history revealed that the symptoms started after 1 week of taking Bionerv. His IAA was positive. In view of the temporal relationship with ALA, an IAS diagnosis was made. Symptoms improved after discontinuation of ALA, dietary modification and medical therapy. His CGM showed predominant hyperglycemia with late evening hypoglycemia.

CONCLUSION

Although ALA is generally safe, emerging case reports demonstrate its potential to trigger IAS. Detailed drug history and clinical suspicion is crucial to avoid the misdiagnosis of insulinoma and unnecessary interventions.

EP_A098

OBSTRUCTIVE JAUNDICE FOLLOWING MIBG THERAPY IN MALIGNANT PHEOCHROMOCYTOMA: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.106>

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INTRODUCTION/BACKGROUND

Malignant pheochromocytoma is a rare neuroendocrine tumor with potential for local invasion and distant meta-

stasis. In inoperable cases, nonsurgical options include I-131 metaiodobenzylguanidine (MIBG) therapy. MIBG-related complications may occur, especially in patients with bulky or anatomically complex tumors. We describe a case of post-MIBG ascending cholangitis due to tumor-related biliary obstruction.

CASE

A 49-year-old Kadazan Muslim female with hypertension, type 2 diabetes, dyslipidaemia, and previous strokes was diagnosed in August 2022 with inoperable malignant right adrenal pheochromocytoma. She presented with right hypochondriac pain, weight loss, palpitations, diaphoresis, and postural hypotension. Imaging revealed a right suprarenal mass (6.3 × 4.9 × 7.5 cm) invading the inferior vena cava and right renal vein. Elevated urine normetanephrine and positive DOTATATE, FDG-PET, and MIBG scans confirmed a functional tumor. Due to high surgical risk, she declined surgery and underwent right adrenal artery embolization with stable disease on follow-up. In July 2024, she received high-dose I-131 MIBG therapy (211 mCi) for palliative intent. Eight days post-therapy, she developed fever and jaundice. Imaging revealed intrahepatic biliary dilatation secondary to tumor compression at the porta hepatis. She was diagnosed with ascending cholangitis complicated with gram-negative sepsis and thrombocytopenia. She was managed with intravenous antibiotics, biliary stenting and supportive care.

CONCLUSION

Obstructive jaundice is a rare but serious complication following MIBG therapy. In this case, tumor compression near the porta hepatis likely exacerbated by post-therapy inflammation or necrosis, led to biliary obstruction. Although preoperative biliary stenting is standard in pancreaticobiliary malignancies, its use in neuroendocrine tumors, including pheochromocytoma, is not well defined. This case supports the potential role of pre-emptive biliary decompression in select high-risk patients undergoing MIBG therapy. Multidisciplinary planning is essential for risk stratification and outcome optimization.

Adult E-Poster

EP_A099

PRIMARY AMENORRHEA AND ANOSMIA IN A YOUNG WOMAN: A LATE DIAGNOSIS OF KALLMANN SYNDROME

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INTRODUCTION/BACKGROUND

Kallmann syndrome (KS), a genetic form of congenital hypogonadotropic hypogonadism (CHH), is characterized by delayed or absent puberty in combination with anosmia or hyposmia. It is more frequently diagnosed in males and often overlooked in females due to subtler clinical features. Underrecognition of anosmia and the absence of routine olfactory assessment contribute to diagnostic delays. Early diagnosis and hormone replacement therapy are essential for initiating pubertal development and supporting reproductive planning.

CASE

A 25-year-old female was referred for evaluation of primary amenorrhea and absent secondary sexual characteristics. She reported a lifelong inability to perceive odors. She was born full-term with no perinatal complications or congenital anomalies. Physical examination showed Tanner stage I breast and pubic hair development. Hormonal evaluation revealed low estradiol, luteinizing hormone (LH), and follicle-stimulating hormone (FSH), consistent with hypogonadotropic hypogonadism. Prolactin and thyroid hormones were within normal limits. Pelvic ultrasound revealed hypoplastic uterus and ovaries. Olfactory testing confirmed anosmia. Brain MRI demonstrated bilateral atrophy of the olfactory bulbs and non-visualization of the olfactory sulci, with normal pituitary anatomy. Bone mineral density (BMD) screening revealed osteoporosis. A diagnosis of Kallmann syndrome was established. Estrogen therapy was initiated, resulting in early breast development.

CONCLUSION

This case highlights the need to consider Kallmann syndrome in women presenting with primary amenorrhea, particularly when anosmia is present. Olfactory testing and neuroimaging are essential components of the diagnostic workup. BMD screening plays a crucial role in evaluating long-term complications of untreated hypogonadism. Timely hormonal therapy supports secondary sexual development, improves bone health, and may enhance psychosocial and reproductive outcomes.

EP_A100

VIRILISATION SECONDARY TO LEYDIG CELL OVARIAN TUMOR IN A POSTMENOPAUSAL WOMAN WITH PRIMARY HYPERPARATHYROIDISM

<https://doi.org/10.15605/jafes.040.S1.108>

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INTRODUCTION/BACKGROUND

Ovarian sex cord-stromal tumors, which include steroid cell tumors, comprise 5–8% of all ovarian tumors. Less than half are androgen-secreting. Leydig cell tumors are a type of steroid cell tumor. They are usually androgen-secreting, unilateral, and can be either benign or malignant. We report a case diagnosed in a postmenopausal woman during follow-up for primary hyperparathyroidism.

CASE

A 66-year-old female was noted to have significant hirsutism during a follow-up consult for hyperthyroidism, which reportedly started in the past 5 years. She had primary hyperparathyroidism secondary to right inferior parathyroid adenoma for the last 10 years, not fulfilling criteria for surgery. Her other comorbidities were rheumatoid arthritis diagnosed at age 50; as well as diabetes mellitus, hypertension, dyslipidaemia and fatty liver disease, diagnosed between 54 to 62 years of age. She had 3 children and experienced early menopause at age 42. She had frontal balding and terminal hair growth on chest, back, abdomen, face and limbs. Systemic examination was unremarkable.

Testosterone level was 14.8 nmol/L (normal range 0.24–1.70) while dehydroepiandrosterone sulfate (DHEAS) level was normal. CT imaging noted an enhancing focus in the left ovary (0.8 × 1.1 × 1.4cm). She underwent exploratory abdominal hysterectomy with bilateral salpingo-oophorectomy, appendectomy and omental biopsy which revealed Leydig cell tumor of the left ovary, with intact capsule. Two months post-operatively, testosterone was undetectable (<0.087 nmol/L) and the patient reported reduced facial hair growth.

CONCLUSION

The development of true hirsutism, alopecia, and/or acne in postmenopausal women should not be disregarded, and assessment for causes of postmenopausal hyperandrogenism should be undertaken. This case illustrates a rare co-existence of an androgen-secreting ovarian tumour with concomitant long-standing primary hyperparathyroidism.

Adult E-Poster

EP_A101

THYROID-ASSOCIATED ORBITOPATHY IN HASHIMOTO'S THYROIDITIS: A RARE AUTOIMMUNE OVERLAP

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INTRODUCTION/BACKGROUND

Thyroid-Associated Orbitopathy (TAO), or Graves' Orbitopathy (GO), is an immune-mediated inflammatory disorder of the orbit most commonly associated with hyperthyroidism in Graves' disease. It is primarily driven by TSH receptor antibodies (TRAb), which stimulate orbital fibroblasts and induce tissue remodelling. In contrast, Hashimoto's thyroiditis is characterized by gland-destructive autoimmunity, with elevated anti-thyroid peroxidase (TPO) antibodies and progressive hypothyroidism. The occurrence of GO in patients with overt hypothyroidism due to Hashimoto's thyroiditis is rare and represents a unique overlap of autoimmune thyroid diseases.

CASE

We present the case of a 56-year-old male with no prior history of thyroid disease who presented with progressive, bilateral eye discomfort, photophobia, eyelid swelling, and intermittent diplopia over the preceding eight months. He also reported experiencing fatigue, cold intolerance, dry skin, myalgia, weight gain, and constipation. The patient's medical history included hypertension, and he was an active smoker; both are known risk factors for orbitopathy.

Physical examination revealed eyelid lag, dry skin, and bilateral exophthalmos. His Clinical Activity Score (CAS) indicated active Graves' orbitopathy (GO). No goitre or tremor was noted. Thyroid function tests confirmed overt hypothyroidism (TSH 19.515 μ IU/mL; FT4 0.59 ng/dL), with significantly elevated anti-thyroid peroxidase (anti-TPO) antibodies (9,307.99 IU/mL) and borderline-positive TSH receptor antibodies (TRAb) (1.85 IU/L). Thyroid ultrasound demonstrated reduced thyroid volume and heterogeneous echotexture, consistent with Hashimoto's thyroiditis. Orbital computed tomography (CT) showed bilateral rectus muscle thickening, further supporting the diagnosis of Graves' orbitopathy. Clinical improvement was observed following treatment with levothyroxine and selenium.

CONCLUSION

This case underscores the concept of autoimmune thyroid disease existing on a spectrum, wherein features of both Hashimoto's thyroiditis and Graves' disease can coexist. Early recognition of this overlap is crucial for accurate diagnosis, appropriate treatment guidance, and prevention of long-term ocular complications.

EP_A102

CLOTS AND CRACKS: OSTEOPOROSIS AS A CONSEQUENCE OF PROTEIN C DEFICIENCY AND WARFARIN USE

<https://doi.org/10.15605/jafes.040.S1.110>

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INTRODUCTION/BACKGROUND

Activated protein C is essential in anticoagulation. Protein C deficiency results in inappropriate blood clot formation due to dysregulated coagulation. We report a case of osteoporosis secondary to protein C deficiency and warfarin use.

CASE

A 30-year-old male initially presented with a superior sagittal sinus thrombosis, complicated with a left frontal lobe venous infarct at the age of 21. He reported a family history significant for venous thromboembolism. A thrombophilia screen done during presentation revealed a moderately low protein C activity at 44.6% (reference interval 70 – 140) with normal protein S levels. Autoimmune workup including anticardiolipin, and lupus anticoagulants were negative. Long term warfarin was initiated for the treatment of the cerebral venous thrombosis (CVT).

Three years after CVT and warfarin use, old compression fractures involving the T5 and T7 vertebrae were found on routine X-rays done during an admission for rhabdomyolysis. A bone mineral densitometry (BMD) showed a Z score of -2.8 at the femoral neck, and -1.5 at the L1-L4 vertebrae. Screening for causes of secondary osteoporosis, specifically hyperparathyroidism, hyperthyroidism, hypogonadism, acromegaly, chronic kidney disease, and Cushing syndrome were negative. The patient's inability to attain peak bone mass would likely be due to severe illness (CVT) suffered at a young age.

Adult E-Poster

Upon diagnosis of osteoporosis, warfarin was replaced with rivaroxaban for anticoagulation and vitamin D replacement and calcium supplements were started, while no anti-osteoporosis medications were initiated. Annual BMD was done, and the latest imaging showed an improvement of 2.4% in the femoral neck compared to the previous year. Apart from the previously noted vertebral compression fractures, no new fractures were appreciated during follow-up. BMD monitoring will continue every 2 years.

CONCLUSION

Osteoporosis in the young should be thoroughly investigated and managing the underlying condition is key to proper treatment.

EP_A103

LIPOPROTEIN X-MEDIATED PSEUDOHYPONATREMIA IN A PATIENT WITH TYPE 2 DIABETES

<https://doi.org/10.15605/jafes.040.S1.111>

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INTRODUCTION/BACKGROUND

Pseudohyponatremia is a lab abnormality commonly caused by hypertriglyceridemia, hyperglycemia or hypergammaglobulinemia. Lipoprotein X (LpX) is an abnormal lipoprotein that most commonly appears in the plasma of patients with cholestasis. LpX mediated pseudohyponatremia is rare but has been described in the literature. We report a patient with type 2 diabetes mellitus (T2DM) and LpX-mediated pseudohyponatremia due to severe cholestatic hepatitis.

CASE

A 31-year-old female was admitted with newly diagnosed T2DM and severe DKA secondary to bilateral calf abscesses. She was treated with insulin and intravenous cefazolin as intraoperative tissue culture grew MSSA. Three days after starting cefazolin she developed progressively worsening severe cholestasis [peak total bilirubin (TB) 245 umol/L (reference interval (RI) <17), conjugated bilirubin 175 umol/L (RI <6), peak ALP 1027 U/L (RI 45-129), with normal

to marginally elevated transaminases] with negative viral and autoimmune serologies including AMA. Malignancy, biliary stones, and extra-hepatic cholestasis were excluded by imaging including CECT liver. Liver biopsy showed non-caseating granulomatous hepatitis, consistent with drug-induced liver injury secondary to cefazolin.

Concurrently, she developed hyponatremia despite adequate glycemic control on insulin therapy, that was established to be secondary to severe hypercholesterolemia [nadir serum sodium (sNa) 125 mmol/L (RI 136-145), serum osmolality 308 mmol/kg (RI 275-295), total cholesterol (TC) 30.6 mmol/L (RI <5.2), triglyceride 5.3 mmol/L]. Serum protein electrophoresis showed a supernumerary peak between albumin and alpha-1 region, suggestive of the presence of LpX. Cefazolin was discontinued and she was given a course of ursodeoxycholic acid (UDCA) for three months. Subsequently, TB and ALP dramatically improved, TC gradually declined and serum sodium became normal. During her most recent follow-up, her liver panel and serum sodium remained normal. TC, triglyceride, and LDL, while markedly improved, remained slightly elevated, compatible with her diagnosis of metabolic syndrome.

CONCLUSION

Recognition of the relationship of cholestasis, elevated LpX and pseudohyponatremia is important to avoid mismanagement of hyponatremia. Electrophoresis confirms the diagnosis of LpX and diagnosed patients should subsequently be monitored for hyperviscosity secondary to hypercholesterolemia.

EP_A104

A CASE OF LATE-ONSET HYPOPARATHYROIDISM FOLLOWING RECURRENT ANTERIOR NECK SURGERY RESULTING IN RHABDOMYOLYSIS

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INTRODUCTION/BACKGROUND

Hypoparathyroidism is a known complication of anterior neck surgery, with 1.5% becoming permanent. Delayed-onset hypoparathyroidism can manifest years postoperatively due to progressive scar tissue formation. It is often overlooked, causing complications. We present such a patient complicated by rhabdomyolysis and renal failure.

Adult E-Poster

CASE

A 78-year-old female with poorly-controlled diabetes mellitus presented with recurrent episodes of generalized weakness, lethargy and gastrointestinal symptoms since March 2024. She had undergone a total thyroidectomy in 2009 for multinodular goiter and neck surgery in 2022 for extensive neck abscess. Her calcium was normal in 2019 but no other postoperative monitoring was done.

She was admitted in March, May, and September 2024 with increasing myalgia, breathlessness, elevated creatine kinase (CK) (500 to 3000 U/L) and progressive renal dysfunction [creatinine: 93 mmol/L (March), 175 mmol/L (May), 422 mmol/L (September)]. Thyroid function tests were normal. Urinalysis showed proteinuria and hematuria. Extensive investigations for autoimmune myositis and renal failure were unremarkable, resulting in a presumed diagnosis of diabetic nephropathy.

In September, amid worsening renal function and persistent CK elevation, severe hypocalcemia (1.30 mmol/L*) was finally recognized. Retrospectively, hypocalcemia (1.47 mmol/L) was first detected in May 2024, treated with intravenous calcium bolus, but not investigated. Immediate calcium infusion with oral calcium and calcitriol supplementation led to a significant CK reduction from 1233 U/L to 286 U/L, creatinine level decreased from 422 mmol/L to 315 mmol/L, with marked improvement of her symptoms and she was discharged without residual weakness. Subsequent follow-up showed further improvement in creatinine to 187 mmol/L and a stabilized CK level (235 U/L). Ultimately, hypoparathyroidism was confirmed to have an undetectable iPTH level.

CONCLUSION

This case highlights the importance of recognizing delayed hypoparathyroidism and its presentation with severe rhabdomyolysis. Unawareness of this complication and a low index of suspicion can lead to prolonged misdiagnosis and exacerbate complications. Prompt recognition and treatment are crucial.

EP_A105

BLINDED BY METASTASIS: A RARE CASE OF RENAL CELL CARCINOMA IN THE PITUITARY

<https://doi.org/10.15605/jafes.040.S1.113>

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INTRODUCTION/BACKGROUND

Renal cell carcinoma (RCC) is the most common primary kidney tumor, accounting for 1-3% of adult malignancies. Metastasis of RCC to the pituitary gland is extremely rare, with only a few reported cases. The time interval from primary tumor diagnosis to pituitary metastasis ranges from 3 months to 27 years, with a median interval of 1 year. Surgical resection is the treatment of choice in cases where vision deteriorates due to optic nerve compression. Adjuvant therapies may also be used, including radiotherapy, chemotherapy, immunotherapy, or targeted therapy. Here, we report a case of RCC metastasis to the pituitary presenting with impaired vision.

CASE

A 62-year-old healthy male presented with progressive blurring of vision in both eyes, where the left eye was completely blind, and the right eye had tunnel vision. Constitutional symptoms occurred four months after undergoing right nephrectomy for RCC stage III. Magnetic resonance imaging revealed an enlarged sella with a solid lesion extending into the suprasellar region, compressing the bilateral optic chiasm and abutting both anterior cerebral arteries. He underwent transsphenoidal surgery, but the procedure was incomplete due to significant bleeding from the vascularized tumor. Two months later, a second decompression surgery was performed to preserve both the optic nerve and chiasm. Postoperatively, he developed panhypopituitarism and required hormone replacement therapy with thyroxine and hydrocortisone. Histopathology examination confirmed metastasis of clear cell renal carcinoma. Hence, radiotherapy and the tyrosine kinase inhibitor (TKI) Pazopanib were used as adjuvant therapies. Following treatment, the patient's vision remained stable, with neither improvement nor further deterioration.

Adult E-Poster

CONCLUSION

This case underscores the rarity of pituitary metastases from renal cell carcinoma and emphasizes the need for clinicians to consider this complication among patients with unexplained neurological symptoms. A multidisciplinary treatment approach with radiotherapy and TKI has potential benefits in challenging cases with incomplete surgical resection.

EP_A106

A RARE ENCOUNTER: UNVEILING THE CLINICAL SPECTRUM OF SUBACUTE THYROIDITIS

<https://doi.org/10.15605/jafes.040.S1.114>

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INTRODUCTION/BACKGROUND

Subacute thyroiditis (SAT) is a rare, self-limiting inflammatory thyroid disease. It usually presents with neck pain, transient thyrotoxicosis and systemic dysfunction. It predominantly affects women aged 20-50 years and is commonly associated with viral infections or autoimmune responses. We report an unusual case of SAT with atypical presentation, highlighting its diagnostic challenges and management.

CASE

A 38-year-old female presented with fever and painless anterior neck swelling with significant weight loss of 9 kg for two weeks, preceded by left otalgia for one week. She denied any significant past medical history, was not taking any medications and reported no family history of thyroid diseases. On examination, the patient was calm, and except for a high-grade fever, there were no other signs of sepsis. She had a palpable, non-tender, diffuse goiter without thyroid eye signs or fine tremors.

Laboratory investigations showed low thyroid-stimulating hormone (TSH) at 0.05 mIU/L, with elevated free T4 (51.6 pmol/L) and free T3 (14.1 pmol/L), yielding a T3/T4 ratio of <0.3. C-reactive protein (CRP) was markedly elevated at 162 mg/L, though white cell count remained normal. Anti-thyroid antibodies were negative. Thyroid ultrasound revealed a multinodular goiter (TIRADS 3), while thyroid scintigraphy demonstrated low uptake, confirming SAT. These findings indicated the hyperthyroid phase of SAT.

Based on clinical symptoms, laboratory results, and imaging findings, a diagnosis of subacute thyroiditis was made.

The patient was treated with corticosteroids to reduce inflammation alongside symptomatic treatment. The patient responded well, with resolution of symptoms within four weeks. Follow-up thyroid function tests normalized after two months, with no recurrence of symptoms or persistence of hyper/hypothyroidism was noted.

CONCLUSION

This case emphasizes that SAT should be considered in patients presenting with fever and elevated free T4 who lack typical thyrotoxic features, especially following a recent infection. The painless goiter and significantly elevated free T4 in this case represented atypical features that could have easily led to misdiagnosis.

EP_A107

INDIVIDUALIZED MANAGEMENT STRATEGIES FOR VERY SEVERE HYPERTRIGLYCERIDEMIA: A CASE SERIES

<https://doi.org/10.15605/jafes.040.S1.115>

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INTRODUCTION/BACKGROUND

Very severe hypertriglyceridemia (HTG) is defined by the Endocrine Society as a serum triglyceride concentration ≥ 22.6 mmol/L. Management typically involves dietary modification, pharmacotherapy such as fibrates combined with statins, insulin therapy, and plasmapheresis in select cases. We report two cases of non-familial very severe HTG secondary to poorly controlled type 2 diabetes mellitus, each managed using different therapeutic strategies.

CASE

Case 1: A 23-year-old female with type 2 diabetes mellitus and class I obesity (BMI 29.3 kg/m²) presented with diabetic ketoacidosis and acute pancreatitis. She had a history of poor adherence to insulin therapy. Her serum triglyceride level was markedly elevated at 64 mmol/L. She was treated with a fixed-rate intravenous insulin infusion (0.1 units/kg/hour) and kept on nothing per ore, resulting in a significant reduction of triglyceride levels to 2.5 mmol/L within three days.

Case 2: An 83-year-old female with type 2 diabetes mellitus, stage 4 chronic kidney disease, hypertension, neurocognitive disorder and osteoporosis who was incidentally found to have severe HTG (25.6 mmol/L) during routine screening. Despite being asymptomatic, she was started on a variable-rate intravenous insulin infusion to reduce the

Adult E-Poster

risk of pancreatitis. Her triglyceride levels decreased to 16.8 mmol/L but then plateaued. Insulin was discontinued, and a low-carbohydrate, low-fat diet with intermittent fasting (from lunch until the next day's breakfast) was initiated. This led to a reduction in her triglyceride level to 9.01 mmol/L within three days.

Both patients were discharged with premixed insulin, rosuvastatin, omega-3 fatty acid supplementation and additional fenofibrate for Case 1 only, as Case 2 has CKD stage 4.

CONCLUSION

These cases highlight the importance of individualized treatment strategies in managing very severe non-familial HTG. While insulin infusion and dietary interventions were effective in both patients, the choice of therapy should be guided by clinical context.

EP_A108

INTERPRETING THYROID HORMONE LEVELS IN A PATIENT WITH GRAVES' DISEASE ON ENOXAPARIN

<https://doi.org/10.15605/jafes.040.S1.116>

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INTRODUCTION/BACKGROUND

Enoxaparin, a low molecular-weight heparin, can interfere with free T4 measurements by displacing thyroid hormones from their binding proteins, potentially giving rise to misleading results. Diagnosis becomes particularly challenging in Graves' disease when this interference occurs, as fluctuating thyroid function caused by shifting TSH receptor antibodies is rare in this condition.

CASE

We report a 38-year-old Malay female who was diagnosed with Grave's disease in May 2024 and treated with carbimazole. Five months later, she was readmitted for dyspnea and hypoxia and diagnosed with a severe pulmonary embolism. Treatment included thrombolysis with alteplase, followed by anticoagulation therapy using enoxaparin.

Upon admission, her anti-thyroid therapy was withheld due to subclinical hypothyroidism, as evidenced by a slightly low free T4 (12.8 pmol/L) and elevated TSH (6.65 iU/L). During her prolonged hospital stay, reassessment

revealed discordant thyroid hormone levels, with both free T4 and TSH being elevated, coinciding with the development of hypothyroid symptoms. Assay interference was ruled out through thyroid function tests performed in other laboratories. While anti-TSH receptor antibody was positive, anti-thyroid peroxidase antibody was normal. A multidisciplinary discussion between physicians and biochemical pathologists concluded that the discordant thyroid function test results were likely due to enoxaparin-induced interference (falsely elevated free T4) in the context of her underlying hypothyroid state (elevated TSH). Oral levothyroxine was initiated. Subsequently, her pulmonary embolism treatment was switched to oral rivaroxaban, and further thyroid function tests showed normal free T4 and TSH levels, corresponding to a clinically euthyroid state.

CONCLUSION

This case emphasizes that when managing Graves' disease with low molecular-weight heparin, clinicians should be aware of potential laboratory interference when interpreting discordant thyroid function test results.

EP_A109

ACARBOSE: AN UNEXPECTED ALLY IN MANAGING REACTIVE HYPOGLYCEMIA IN PREGNANCY

<https://doi.org/10.15605/jafes.040.S1.117>

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INTRODUCTION/BACKGROUND

Reactive hypoglycemia, or postprandial hypoglycemia, which can occur during pregnancy due to physiological changes such as altered insulin sensitivity and heightened metabolic needs, presents unique management considerations. Acarbose, an alpha-glucosidase inhibitor, offers a potential therapeutic strategy by slowing the digestion and absorption of carbohydrates, thereby helping to regulate postprandial glucose levels and prevent hypoglycemic episodes in pregnant women.

CASE

We present the case of a 25-year-old pregnant female diagnosed with overt diabetes mellitus at 10 weeks of gestation via oral glucose tolerance test (OGTT). Initially she was started with insulin and metformin, however despite dietary adjustments and titration of medications, she experienced recurrent hypoglycemic episodes. These episodes occurred 1-2 hours after meals and were refractory to conventional management. Acarbose was then initiated

Adult E-Poster

at 28 weeks of gestation. During her third trimester, she required basal bolus insulin regimen with adjustment of acarbose dose. Throughout the pregnancy, the patient's blood glucose and fetal well-being were closely monitored and her glucose levels remained well-controlled. She delivered at 37 weeks of gestation, and both mother and baby were discharged in good health.

CONCLUSION

Acarbose may be a valuable addition to the treatment plan for pregnant women experiencing postprandial hypoglycemia, particularly when conventional treatments such as insulin therapy and dietary modification are inadequate. Although further research is needed to establish the safety and efficacy of acarbose in pregnancy, this case suggests that acarbose could be an effective and safe option for managing postprandial hypoglycemia in diabetic pregnancies.

EP_A110

UNMASKING ATYPICAL FEMORAL FRACTURES IN OSTEOPOROSIS: A CASE SERIES OF HIGH-RISK PATIENTS

<https://doi.org/10.15605/jafes.040.S1.118>

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INTRODUCTION/BACKGROUND

Atypical femoral fractures (AFFs) are rare but serious complications associated with long-term antiresorptive therapy for osteoporosis. These fractures often occur in the femoral diaphysis, particularly in the subtrochanteric or mid-shaft regions, and may arise without trauma or with minimal trauma. Corticosteroid use and other comorbidities, such as chronic inflammation, further increase the risk of AFFs in susceptible individuals.

CASE

We report three cases of AFFs in female patients aged 61 to 75 years. The mean duration of bisphosphonate use prior to AFF was 5 years and 8 months. Two out of three patients had a history of chronic glucocorticoid use and one had rheumatoid arthritis who was on methotrexate. Only one patient had prodromal thigh pain. All patients presented with complete AFF and one patient with contralateral incomplete AFF. Most complete AFFs were sustained from a simple fall while one case was atraumatic. All complete AFFs were treated with intramedullary nailing and recovered well postoperatively. The patient with contralateral incomplete AFF was counselled for prophylactic surgical intervention which she declined. Anti-osteoporotic

treatment was switched from bisphosphonate to anabolic agents following surgery.

CONCLUSION

Early recognition of AFFs and proactive intervention can prevent further fractures. Clinicians should consider drug holidays or switching therapies for patients on long-term bisphosphonates, especially those with additional risk factors, to optimize bone health.

EP_A111

CULTURAL SILENCE: UNVEILING PITUITARY APOPLEXY IN A MAN WITH CHRONIC ERECTILE DYSFUNCTION AND COEXISTING PROSTATIC ABSCESS

<https://doi.org/10.15605/jafes.040.S1.119>

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INTRODUCTION/BACKGROUND

Pituitary apoplexy is an endocrine emergency caused by hemorrhage or infarction within a pituitary tumor, resulting in acute pituitary dysfunction. Triggers can include major surgeries, angiography, intracranial hypertension, or distant infections. In this case, fever and signs of increased intracranial pressure revealed a previously undiagnosed pituitary macroadenoma, initially presenting as chronic erectile dysfunction, which ultimately led to a diagnosis of pituitary apoplexy.

CASE

A 43-year-old male with obstructive sleep apnoea presented with acute fever, vomiting, headache, postural dizziness, and lethargy. He had long standing erectile dysfunction, previously uninvestigated. Initially treated for gastroenteritis, he was drowsy (Glasgow Coma Scale, GCS: Eye 3, Verbal 4, Motor 6), with left temporal hemianopia but no signs of meningism or focal neurological deficits.

Laboratory tests showed leukocytosis ($12 \times 10^9/L$) predominantly neutrophilia, elevated C-reactive protein (105 mg/L), hyponatremia (125 mmol/L), hypokalemia (3 mmol/L) and negative blood cultures. Abdominal ultrasonography revealed a small prostatic abscess. Neuroimaging (CT brain and MRI pituitary) confirmed a pituitary macroadenoma ($2.0 \times 2.9 \times 3.7$ cm) with hemorrhagic apoplexy compressing the optic chiasm. Hormonal evaluation showed secondary hypogonadism, hypocortisolism, and hypothyroidism.

Adult E-Poster

Treatment with antibiotics and intravenous hydrocortisone, followed by thyroxine and testosterone therapy, resulted to clinical improvement, including improvement of GCS with resolution of the prostatic abscess, visual field deficit, and erectile dysfunction. Follow-up imaging showed a decrease in the size of the sellar mass ($1.5 \times 1.7 \times 1.5$ cm), hence the neurosurgery team opted for conservative management with continued endocrine follow-up.

CONCLUSION

The importance of addressing male sexual dysfunction without stigma is highlighted by this case. Non-functioning pituitary macroadenomas commonly affect the hypothalamic-pituitary-gonadal axis, and concurrent fever may mask an underlying infection. This infection, in turn, could trigger pituitary apoplexy. Clinicians should maintain a high index of suspicion and intervene promptly in patients presenting with elevated intracranial pressure to ensure optimal outcomes.

EP_A112

A CASE OF PROLACTIN AND GROWTH HORMONE CO-SECRETING PITUITARY MACROADENOMA

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INTRODUCTION/BACKGROUND

Prolactin (PRL) and growth hormone (GH) co-secreting pituitary macroadenomas are relatively rare and exhibit a multifaceted clinical presentation. Optimal management of PRL/GH co-secreting pituitary adenomas remains clinically challenging. We report a case of a PRL/GH co-secreting pituitary macroadenoma managed with cabergoline monotherapy.

CASE

A 25-year-old male without comorbidities presented with a chronic headache for eight years. Pituitary MRI revealed a pituitary macroadenoma measuring $1.7 \times 2.5 \times 1.8$ cm compressing the optic chiasm, with minimal extension into the right cavernous sinus and mild sellar widening. He had markedly elevated prolactin at 11,170 mIU/L and was started on Cabergoline 0.25 mg twice weekly and transferred care to our centre for further management of functioning pituitary macroadenoma. Notable physical examination findings included frontal bossing and prognathism, but no macroglossia, skin tags, or tremors were seen. A visual

field assessment revealed no defects. Hormonal workup exhibited elevated GH of 4.75 μ g/L (5x the upper limit of normal), IGF-1 of 418.9 ng/mL, prolactin of 3,433 mIU/mL and failure of GH suppression on OGTT, consistent with a GH and prolactin-secreting pituitary adenoma. The multidisciplinary team reached a consensus that curative surgery is not feasible due to tumor extension into the right cavernous sinus. They recommended increasing his Cabergoline dosage to 3-4 mg weekly. The patient remains asymptomatic and his prolactin level is 784 mIU/mL while on Cabergoline 3.5 mg weekly. A follow-up MRI shows a smaller pituitary lesion with resolution of the optic chiasm mass effect. Currently, the patient is not inclined to consider surgery.

CONCLUSION

Surgical intervention is typically the first-line treatment for mixed co-secreting pituitary adenomas. However, cabergoline is known to effectively normalize prolactin levels in patients with hyperprolactinemia caused by mixed adenomas. In this particular case, cabergoline monotherapy appears to be successful at controlling both tumor growth and prolactin levels.

EP_A113

EFFECTIVE MEDICAL THERAPY FOR MULTIPLE ENDOCRINE NEOPLASIA TYPE 1-ASSOCIATED METASTATIC VIPoma

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INTRODUCTION/BACKGROUND

Pancreatic islet cell tumors occur in 40% of MEN 1 patients. VIPoma is a rare functioning pancreatic neuroendocrine tumor characterized by excessive secretion of vasoactive intestinal peptide (VIP), with an annual incidence of fewer than 1 in 10 million in the general population. Most cases are sporadic, but about 5% are linked to multiple endocrine neoplasia type 1 (MEN 1) syndrome. We describe a patient with MEN 1 associated metastatic VIPoma.

CASE

A 56-year-old female was first diagnosed with a benign insulinoma in 2005 and underwent successful laparoscopic tumor excision. There was no prior or family history of endocrine tumors. In 2019, she developed intermittent abdominal pain and chronic diarrhea which progressed over a 3-year period.

Adult E-Poster

In 2022, investigations confirmed elevated vasoactive intestinal peptide (VIP) levels of 276 pg/mL (normal reference: <75 pg/mL) and a pancreatic body tumor with diffuse pancreatic infiltration, as well as liver and nodal metastases were identified on multimodal imaging. Extensive debulking surgery was recommended, but the patient declined.

In March 2023, she presented to our hospital with hypovolemic shock resulting from profuse diarrhea and severe hypokalemia. Intramuscular octreotide long-acting release (LAR) 30 mg was initiated, leading to prompt symptom control, and she continued to receive monthly injections. Genetic studies confirmed multiple endocrine neoplasia type 1 (MEN 1), prompting subsequent screening of family members.

By March 2024, the patient was asymptomatic, had experienced significant weight gain (from 50 to 65 kg), and her VIP levels had normalized to 66 pg/mL. A Gallium-68 (Ga-68) DOTATATE PET/CT scan in March 2024 demonstrated stable pancreatic and nodal disease but progressive liver metastases. The neuroendocrine multidisciplinary team offered the option of peptide receptor radionuclide therapy (PRRT), but she declined this treatment.

CONCLUSION

This case highlights the effectiveness of medical management with Octreotide LAR for control of symptoms and tumor-related hormonal hypersecretion in inoperable metastatic VIPoma regardless of disease progression. A personalized multidisciplinary approach is a pre-requisite for achieving optimal outcomes.

EP_A114

THE SHRINKING HYPOTHALAMIC LESION: SERIAL MRI-GUIDED MANAGEMENT OF REFRACTORY XANTHOMATOUS HYOPHYSITIS

<https://doi.org/10.15605/jafes.040.S1.122>

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INTRODUCTION/BACKGROUND

Xanthomatous hypophysitis is a rare inflammatory condition affecting the pituitary gland, characterized by the presence of lipid-laden macrophages (xanthoma cells) and chronic inflammation.

CASE

We describe a 26-year-old female with a 12-year history of xanthomatous hypophysitis complicated by panhypo-

pituitarism and diabetes insipidus. Initial presentation at age 13 years with growth failure led to transcranial tumor debulking in 2016, with histopathology confirming chronic xanthogranulomatous inflammation. Due to recurrent disease activity, she received two courses of high-dose prednisolone (2018 and 2019-2020), followed by maintenance azathioprine (initiated 2020, currently 25 mg daily).

Serial MRI surveillance showed a gradual regression of the lesion over time. In July 2023, the hypothalamic lesion measured 1.3 × 0.8 × 0.8 cm, a reduction from its previous size of 1.5 × 1.3 × 1.1 cm in 2021. This regression was accompanied by decreased compression on the optic chiasm. The pituitary gland remained thinned, and the posterior pituitary bright spot is absent, consistent with long-standing structural damage. These findings suggest ongoing inflammatory control under azathioprine.

Hormonal management included levothyroxine (37.5 mcg/day), sublingual desmopressin (60 mcg/day for DI), and cyclic estrogen-progestin (Progyluton). Adrenal function recovered post-steroid withdrawal, confirmed by a normal Synacthen test (peak cortisol 624 nmol/L, 2022). Complications included secondary osteoporosis (spine T-score -1.8, hip -2.5) managed with calcium/vitamin D, and microcytic anemia (Hb 9.4 g/dL, MCV 29.8 fL), likely due to iron deficiency, now treated with oral iron and folate.

CONCLUSION

This case highlights the chronic relapsing-remitting nature of xanthomatous hypophysitis, necessitating long-term immunosuppression and meticulous endocrine surveillance. The latest MRI findings (2023) confirm disease stability under azathioprine, supporting its role in preventing progression. However, residual hypothalamic involvement underscores the need for continued monitoring. A multidisciplinary approach (endocrinology, neurosurgery, rheumatology) remains essential to manage hormonal deficits, bone health, and potential disease recurrence. Future considerations include azathioprine tapering if imaging remains stable, emphasizing the importance of serial MRI in guiding therapy.

Adult E-Poster

EP_A115

PARANEOPLASTIC HYPOGLYCEMIA IN HEPATOCELLULAR CARCINOMA: A REPORT OF TWO CASES

<https://doi.org/10.15605/jafes.040.S1.123>

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INTRODUCTION/BACKGROUND

Hepatocellular carcinoma (HCC) is the second most common cause of non-islet cell tumor hypoglycemia (NICTH), with a reported prevalence varying from 4% to 27%. It can also be an early presentation of HCC, even before the tumour is large enough to be detected on imaging. We report two cases of NICTH in patients with viral hepatitis-associated HCC.

CASE

Case 1: A 64-year-old male presented with severe hypoglycemia (glucose 1.8 mmol/L) during an admission for spontaneous bacterial peritonitis. He was newly diagnosed with Child-Pugh C liver disease from chronic hepatitis C. Hypoglycemia persisted despite resolution of sepsis, requiring continuous dextrose 50% infusion. Physical examination revealed a hard mass in the right upper quadrant. Laboratory findings included elevated AFP at >830 IU/mL, low albumin at 24 g/L, INR of 1.5, bilirubin of 25 µmol/L, and platelet count of 180 x10⁹/L. CT imaging showed a cirrhotic liver with a segment II mass (4.2 x 4.0 x 4.0 cm). Laboratories done during hypoglycemia showed low levels of insulin at <0.5 mIU/L (normal 3–25), low C-peptide at 46 pmol/L (298–2350), low IGF-1 at 10.5 ng/mL (42–179), and HbA1c of <3.4% (Hb 9.6 g/dL). IGF-2 testing was unavailable.

Case 2: A 48-year-old female with chronic hepatitis B and a large HCC (14 x 10 x 9 cm) and elevated AFP at 8,925 IU/mL. She presented with symptomatic hypo-glycemia (glucose 2.5 mmol/L). Prolonged fasting revealed hypoglycemia at 4 hours (glucose 2.1 mmol/L), with insulin of <2 µIU/mL, C-peptide of <33 pmol/L, IGF-1 of 17 ng/mL (normal 90–249), and cortisol at 509 nmol/L. The presence of inappropriately low insulin and low c-peptide during an episode of hypoglycemia favors the diagnosis of NICTH.

Both cases were managed with oral prednisolone 1 mg/kg which allowed for successful weaning from dextrose. Despite transient stabilization, both patients succumbed within one month.

CONCLUSION

Paraneoplastic hypoglycemia should be considered in HCC patients with unexplained hypoglycemia. Early recognition and glucocorticoid therapy may improve short-term outcomes, though prognosis remains poor.

EP_A116

PSEUDOACROMEGALY: A CASE OF SEVERE INSULIN RESISTANCE WITH ACROMEGALIC FEATURES AND LOW IGF-1

<https://doi.org/10.15605/jafes.040.S1.124>

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INTRODUCTION/BACKGROUND

Severe insulin resistance (SIR) is typically associated with well-established metabolic conditions such as central obesity, glucose intolerance, hypertension, dyslipidaemia and metabolic dysfunction-associated fatty liver disease (MAFLD). We present a case of SIR with prominent dermatologic and acral manifestations mimicking acromegaly, highlighting the importance of recognizing skin and soft tissue changes as possible indicators of underlying insulin resistance.

CASE

A 35-year-old male with a history of obesity (weight 128.7 kg, height 1.71 m, BMI 44 kg/m²), hypertension (on amlodipine) and hyperlipidaemia (on atorvastatin) was referred for evaluation of hypokalemia (2.35 mmol/L). Clinical examination revealed acromegalic features, including macroglossia, rhinophyma, spade-like hands and marked dermatologic findings with acanthosis nigricans, pachyderma, multiple epidermoid cysts and dense axillary skin tags. Visual fields were normal. Anterior pituitary function was normal except for low IGF-1 at 78 ng/mL (normal: 88.3–246 ng/mL). Liver function tests revealed mild abnormalities of bilirubin (51 µmol/L), aspartate aminotransferase 71 U/L (normal: <32 U/L), alanine aminotransferase 23 U/L (normal: <33 U/L) and alkaline phosphatase 136 U/L (normal: 40–129 U/L). The patient denied alcohol use. Fasting insulin was 647 pmol/L (normal: 17.8–173 pmol/L). A diagnosis of pseudoacromegaly secondary to SIR was considered, supported by a significantly elevated HOMA-IR of 27.3 (normal: 0.5–1.4). HbA1c was 8.5%. He was initiated on metformin and lifestyle modification.

Adult E-Poster

CONCLUSION

This case highlights a rare presentation of SIR with prominent dermatological manifestations mimicking acromegaly. This phenotype of insulin-mediated pseudo-acromegaly is thought to arise from a selective post-receptor insulin signaling defect, where metabolic pathways are impaired while mitogenic signaling remains relatively intact. Recognizing these atypical dermatologic clues is vital for early identification of insulin resistance syndromes, enabling timely diagnosis and appropriate intervention.

EP_A117

MASQUERADING INSULINOMA: A RARE CASE OF ENDOCRINE TUMOR AND COMPLEX CLINICAL PRESENTATION

<https://doi.org/10.15605/jafes.040.S1.125>

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INTRODUCTION/BACKGROUND

Recurrent hypoglycemia poses a diagnostic challenge, particularly when multiple endocrine disorders are present. Adrenal insufficiency, characterized by deficient cortisol production, impairs glucose homeostasis by reducing gluconeogenesis and increasing insulin sensitivity. Insulinoma, a rare pancreatic β -cell tumor, causes unregulated insulin secretion, leading to symptomatic hypoglycemia. Diagnosis relies on Whipple's triad and specific biochemical markers, including elevated insulin, C-peptide, and proinsulin levels during fasting. The concurrent presence of insulinoma and adrenal insufficiency is rare and requires a high index of suspicion.

CASE

A 54-year-old male with type 2 diabetes (on metformin), obstructive sleep apnea, and a history of traditional medicine use presented with recurrent dizziness and documented hypoglycemia. Examination and electrolytes were unremarkable. A short Synacthen test showed an inadequate cortisol response, confirming adrenal insufficiency. He was started on oral hydrocortisone, with subsequent stabilization of glucose levels and was discharged.

Three months later, he presented again with symptomatic hypoglycemia (as low as 1.2 mmol/L) despite compliance with medications. Biochemical tests revealed elevated insulin (296 pmol/L) and C-peptide (2,595 pmol/L), suggesting endogenous hyperinsulinemia. A 72-hour fast confirmed persistent hypoglycemia with inappropriately high insulin and C-peptide levels. CT imaging showed a distal pancreatic lesion consistent with insulinoma and

he then underwent distal pancreatectomy. Hypoglycemia resolved after surgery. He remains stable on hydrocortisone (10 mg three times daily).

Cortisol deficiency initially explained the patient's hypoglycemia, which improved with steroid therapy. However, persistent episodes prompted further workup, revealing an insulinoma. Surgical removal resulted in resolution, confirming the dual etiology of his hypoglycemia.

CONCLUSION

This case highlights the rare coexistence of insulinoma and adrenal insufficiency. Cortisol deficiency can obscure insulinoma symptoms, emphasizing the need for a thorough and systematic diagnostic approach to recurrent hypoglycemia.

EP_A118

GROWTH RETARDATION THERAPY IN PATIENTS WITH MARFAN SYNDROME: A CASE REPORT AND LITERATURE REVIEW

<https://doi.org/10.15605/jafes.040.S1.126>

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INTRODUCTION/BACKGROUND

Marfan syndrome is an autosomal dominant disorder affecting connective tissues, with an incidence of 2-3 per 100,000 individuals. It involves mutations of the gene coding for fibrillin-1, and is characterized by ocular, cardiovascular, and skeletal manifestations. Excessive linear growth and tall stature are common features, which may lead to aortic dilatation, scoliosis, and social adjustment problems. Hence, height control treatment is considered in some patients.

CASE

We report the case of a 15-year-old male who was referred to the Endocrine clinic for possible growth-reductive therapy. His height was 198 cm (97th percentile) and his weight was 69.5 kg (50th percentile). He experienced rapid growth starting at age 7; however, he did not receive any treatment for height reduction. Physical examination revealed a high-arched palate, a systolic murmur at the mitral area, arachnodactyly, and mild upper thoracic scoliosis. An echocardiogram showed mild dilation of the left atrium and left ventricle, mild to moderate mitral regurgitation, and mitral valve prolapse. His aortic root was not dilated.

He maintains a normal social life, although he occasionally feels excluded due to limitations on physical activities resulting from loose ligaments. A review of available

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treatment options indicated that estrogen-based therapies have demonstrated efficacy in females when initiated before age 11, while the use of testosterone or high-dose estrogen in males remains less well-studied. Given the patient's advanced pubertal status (Tanner stage V) and chronological age, therapy was considered unlikely to be effective. Nevertheless, a shared decision-making process emphasizing supportive management, monitoring, and counseling was undertaken with the patient and his family.

CONCLUSION

The case highlighted the importance of early identification and timely referral for growth-reductive therapy in patients with Marfan Syndrome. Height reduction strategy has been promising, however data is limited with relatively small sample size, with inconclusive evidence on growth-suppressing therapies among male patients.

EP_A119

EXPLORING HYPERGLYCEMIA-RELATED SEIZURES: A CASE SERIES

<https://doi.org/10.15605/jafes.040.S1.127>

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INTRODUCTION/BACKGROUND

Hyperglycemia-related seizures, though rare, represent a serious complication of uncontrolled diabetes, often occurring in the context of Hyperosmolar Hyperglycaemic State (HHS). We present a retrospective case series detailing the clinical characteristics, metabolic parameters and outcomes of patients admitted with hyperglycemia-related seizures.

CASE

Seven patients (4 females, 3 males) were included, with a median age of 64 years (range 16–76). All except one, a known defaulter, were on insulin therapy. Glycemic control was poor, with a median HbA1c of 9.9% (range 7.4 – 19.8). Random blood glucose levels at presentation ranged from 15 to 48.8 mmol/L. Serum sodium ranged from 120 to 150 mmol/L and serum osmolality ranged from 292.2 to 361.1 mOsm/kg. Three had HHS and one had overlapping diabetic ketoacidosis. Generalised tonic-clonic (GTC) seizures were the most common presentation (n = 6), while one had focal seizures.

Only one patient had a prior history of stroke; none had known epilepsy. Three patients required intubation for airway protection. Potential confounders included

dementia (n = 2), hypertensive crisis (n = 1), liver cirrhosis (n = 2, including one with history of substance abuse) and sepsis (n = 1). Brain CT most commonly showed cerebral atrophy with small vessel disease (n = 4); two had concurrent multifocal infarct. Two EEGs were performed, showing no epileptiform changes. Most patients achieved seizure control following normalization of blood glucose. Three patients were started on antiepileptic medications, and two of these patients were discharged on the same medications. The mortality rate was high, with three deaths occurring during the study period. One of these patients developed a total anterior circulation infarct.

CONCLUSION

Our findings suggest that GTC seizures are more prevalent in patients with severe hyperglycemia. The absence of epileptiform activity on EEG supports a metabolic etiology. Early recognition, aggressive glycemic management and comprehensive post-discharge follow-up are important. These measures may improve neurological outcomes and reduce the high mortality associated with this complication.

EP_A120

PRIMARY ALDOSTERONISM IN PREGNANCY: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.128>

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INTRODUCTION/BACKGROUND

Primary aldosteronism (PA) in pregnancy is a rare and potentially severe disorder that poses significant challenges for diagnosis and treatment. Compared to essential hypertension, PA is associated with increased risks of preterm delivery, fetal growth restriction, and preeclampsia due to hypertension and hypokalemia.

CASE

We present a 34-year-old primigravida with confirmed PA of ten years duration marked by recurrent episodes of hypokalemia and hypertensive urgency. Despite the initiation of mineralocorticoid receptor antagonist (MRA) treatment, the patient's blood pressure remained poorly controlled, and the patient also had irregular follow-up. She presented at 8 weeks of gestation with uncontrolled hypertension. She required multiple antihypertensive medications with maximal doses as pregnancy progressed, including methyldopa, labetalol, and nifedipine, but BP control remained suboptimal. Imaging revealed a left adrenal nodule, leading to retroperitoneoscopic adrenal-

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ectomy in the second trimester. Postoperatively, her blood pressure improved moderately, but she developed severe preeclampsia at 26 weeks, necessitating an emergency caesarean delivery and her premature infant did not survive. She remained hypertensive post-adrenalectomy and post-partum, suggesting concomitant essential hypertension.

Managing PA in pregnancy is difficult because MRAs have adverse effects in pregnancy, and other antihypertensive drugs have limited ability to lower aldosterone-mediated hypertension. This case illustrates the problems of achieving tight blood pressure control in pregnancy and consequent maternal and fetal complications. Surgical adrenalectomy may not completely alleviate hypertension during pregnancy because of ongoing vascular remodelling from chronic aldosterone excess. Compared with essential hypertension, PA in pregnancy carries a larger risk of unfavourable outcomes, including preeclampsia, IUGR and placental insufficiency due to aldosterone's direct endothelial and pro-inflammatory effects. Despite adrenalectomy, this patient still developed preeclampsia, emphasizing the persisting vascular dysfunction even after surgery.

CONCLUSION

Careful management of primary aldosteronism (PA) during pregnancy is crucial to reduce complications. Adrenalectomy may improve blood pressure control, but it does not ensure protection from adverse outcomes. Multidisciplinary care and continuous monitoring are therefore necessary.

EP_A121

PITUITARY HYPOPLASIA PRESENTING WITH HYPOPITUITARISM: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.129>

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INTRODUCTION/BACKGROUND

Hypopituitarism is a deficiency of one or more hormones secreted by the anterior or posterior pituitary gland. It is a rare condition, with a prevalence of 46 cases per 100,000 population. It can be caused by several conditions, but it is rarely caused by pituitary hypoplasia.

CASE

A 19-year-old female presented to the hospital with concerns of short stature and delayed puberty. She reported never having experienced menstruation and a lack of breast development. The patient denied headache and there was no reported history of hormonal abnormalities or previous medication use. Her intellectual abilities were noted to be well-developed.

On examination, the patient's height was 135 cm and her weight was 32 kg. Her genetic height potential was estimated to be between 142.5 and 159.5 cm. She exhibited no signs of puberty (Tanner stage I).

Laboratory results revealed: LH <0.09 mIU/mL, FSH 0.69 mIU/mL, estradiol <10 pg/mL, TSH 4.1 mIU/mL, FT4 5.52 pmol/L, IGF-1 21 ng/mL, and cortisol 1.8 µg/dL. Bone age was assessed as equivalent to a 13-year-old female, with an open epiphyseal plate. Gynecological ultrasound showed a small uterus measuring 5.47 x 2.33 cm. Brain MRI revealed pituitary hypoplasia (6.9 x 3.9 x 7.5 mm) with no other identified abnormalities.

Based on these findings, the patient was diagnosed with pituitary hypoplasia and hypopituitarism (hypogonadism, hypothyroidism, central hypothyroidism, adrenal insufficiency). Treatment was initiated with estradiol valerate 2 mg, levothyroxine 25 mcg, and hydrocortisone 20 mg. Within six months, the patient experienced menstruation and breast development.

CONCLUSION

We have treated a patient with hypopituitarism secondary to pituitary hypoplasia. We hypothesize that a genetic defect caused pituitary hypoplasia in this patient. The patient has had a positive outcome and continues to receive routine follow-up care at the hospital for hormone replacement therapy.

EP_A122

REASSESSING MEN 1 P.Ala541Thr: NON-DELETERIOUS POLYMORPHISM OR UNDERESTIMATED RISK?

<https://doi.org/10.15605/jafes.040.S1.130>

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INTRODUCTION/BACKGROUND

Multiple endocrine neoplasia type 1 (MEN 1) is an autosomal dominant hereditary tumor syndrome caused by inacti-

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vating mutations in the MEN 1 tumor suppressor gene. Germline mutations in MEN 1 show high penetrance and account for approximately 70–80% of diagnosed MEN 1 cases. Several polymorphisms have also been identified within the MEN 1 gene region, with at least 12 benign variants reported in the general population. While these variants are typically considered non-pathogenic, the c.1621A>G variant has been reported in some studies as potentially contributing to a low-penetrance MEN 1 phenotype in certain carriers.

CASE

We report a case of a 56-year-old female who presented with a 3-month history of painless jaundice and anorexia. She had no personal or family history of malignancy or endocrine disorders. Investigations revealed cholestatic jaundice (bilirubin 89 $\mu\text{mol/L}$), and hypercalcemia (2.88 mmol/L). Imaging showed a solitary 1.7 cm enhancing pancreatic head lesion. Biochemical workup indicated primary hyperparathyroidism (intact-PTH 22.9 pmol/L [normal range; 1.96 – 8.49], calcium/creatinine clearance ratio 0.04). She underwent Whipple's procedure, and histopathology confirmed a 2.1 cm grade 1 pancreatic neuroendocrine tumor (T2N0). Gallium-68 PET/CT showed no distant disease but identified a right lower thyroid lobe focus, suggestive of a parathyroid adenoma. Pituitary MRI was unremarkable.

She met the 2 hallmark features for MEN 1; primary hyperparathyroidism and a pancreatic neuroendocrine tumor, although her presentation occurred later than is typical for MEN 1 cases. The whole exome sequencing showed no pathogenic MEN 1 mutation but detected a c.1621A>G variant that is classified as non-deleterious polymorphism. Interestingly, pathogenic variants in TP53 and BRCA1 were identified without phenotypic expression to date.

CONCLUSION

This case raises questions about the possible pathogenic role of the MEN 1 c.1621A>G variant, especially considering previous reports linking it to low-penetrance MEN 1. Its coexistence with mutations in TP53 and BRCA1 further suggests potential gene-gene interactions or modifier effects, warranting further investigation.

EP_A123

METASTATIC POORLY DIFFERENTIATED THYROID CANCER: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.131>

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INTRODUCTION/BACKGROUND

Poorly differentiated thyroid carcinoma (PDTC) is an aggressive subtype of thyroid cancer, representing 0.23%–2.6% of cases.¹ Due to its rarity, the role of thyroglobulin (Tg) monitoring and the effectiveness of radioactive iodine (RAI) ablation have not been clearly defined. Elevated Tg levels in PDTC are associated with higher recurrence suggesting prognostic significance. While RAI avidity is variable, 25% of PDTC cases maintain the ability to uptake iodine. In such cases, RAI ablation significantly improves survival after thyroidectomy.

CASE

A 51-year-old female presented to a private hospital with a one-year history of neck swelling in April 2023. Initial blood investigations, including thyroid function tests were normal and she was advised that no surgical intervention was necessary. There was progressive enlargement of the neck, and by January 2024, she developed airway compression. CT scan showed a large multinodular goiter, an ill-defined hypodense mass in the left thyroid lobe and pulmonary nodules measuring 0.5–2 cm suggesting metastases. She underwent total thyroidectomy and histopathology confirmed PDTC with lymphovascular spread (pT3aNx, high risk).

She was referred to Endocrinology post-thyroidectomy and was started on TSH suppression therapy and given RAI ablation (150 mCi) in April 2024. Baseline stimulated Tg was >500 ng/mL with negative anti-Tg antibodies.

CONCLUSION

This is the first case of PDTC in our center. A multidisciplinary team was important in management. Our case highlights the prognostic role of Tg, the need for more evidence on the efficacy of each treatment modality and the importance of a standardized treatment algorithms for PDTC.

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EP_A124

METASTATIC PULMONARY NEUROENDOCRINE NEOPLASM WITH CARCINOID SYNDROME COMPLICATED BY BOWEL PERFORATION

<https://doi.org/10.15605/jafes.040.S1.132>

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INTRODUCTION/BACKGROUND

Carcinoid syndrome (CS) results from hormone-secreting neuroendocrine neoplasms (NENs) releasing bioactive substances into systemic circulation. NENs are most commonly found in the gastrointestinal tract and less frequently in the lungs. CS develops in about 19% of patients with NENs, with 20% presenting with distant metastases. Rarely, mesenteric fibrosis in CS can lead to ischemia and perforation.

We present a case of metastatic bronchial NEN with CS, complicated with bowel perforation and subsequent death.

CASE

A 46-year-old male with previous history of spinal surgery for a post-traumatic compression fracture presented with gradual bilateral lower limb weakness and back pain, followed by chronic diarrhea and significant weight loss. Spinal MRI revealed extensive metastatic bone disease. Oesophagogastroduodenoscopy (OGDS) and colonoscopy were unremarkable; however, CT imaging identified a solitary endobronchial mass in the left lower lobe (3.1 x 2.6 x 2.8 cm), associated with left hilar lymphadenopathy and liver metastases.

Biopsy of the lung mass revealed a grade 1 neuroendocrine tumor (Ki67 1%). Urinary 5-hydroxyindolacetic acid (5-HIAA) levels were markedly elevated at 854.6 µmol/day. Gallium-68 DOTATATE PET-CT demonstrated somatostatin receptor-avid disease involving the left lung, with mediastinal nodes, liver and extensive skeletal metastasis. A diagnosis of CS was established based on clinical presentation, elevated 5-HIAA, imaging, and histopathology.

The patient was initiated on octreotide, a somatostatin analogue. However, he struggled to come to terms with the diagnosis and self-discharged against medical advice. He was later readmitted with severe hypokalemia, acute kidney injury, metabolic acidosis and acute abdomen. CT imaging revealed pneumoperitoneum consistent with a perforated duodenum. Due to hemodynamic instability, surgical intervention was not feasible, and palliative care was given.

CONCLUSION

This case illustrates a rare and potentially fatal complication of CS, underscoring the importance of early diagnosis and prompt treatment. Maintaining a high index of suspicion is crucial for timely identification of CS.

EP_A125

TRAPPED IN THE HYPOGLYCEMIA LOOP: A RARE CASE OF RAPIDLY PROGRESSIVE METASTATIC INSULINOMA

<https://doi.org/10.15605/jafes.040.S1.133>

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INTRODUCTION/BACKGROUND

Malignant insulinomas are rare and account for approximately 10-15% of all insulinomas. Most metastatic insulinomas are not curable with surgery alone and necessitate a multimodal approach encompassing medical, locoregional, targeted, systemic, and supportive therapies. The optimal treatment sequence should be individualized to each patient.

CASE

A 21-year-old male presented with a one-month history of recurrent hypoglycemic episodes characterized by neuroglycopenic symptoms. Subsequent evaluation confirmed endogenous hyperinsulinemic hypoglycemia. Computed tomography (CT) imaging revealed a 2.1 x 2.0 x 1.8 cm lesion in the pancreatic tail, multiple liver lesions in both lobes (largest measuring 3.8 x 4.1 x 4.1 cm), and intra-abdominal lymphadenopathy (largest measuring 1.1 x 1.8 cm). Gallium-68 (Ga-68) DOTATATE PET/CT and fluorine-18 fluorodeoxyglucose (18F-FDG) PET/CT demonstrated predominantly somatostatin receptor-avid disease.

Following multidisciplinary team discussion, the tumor was deemed inoperable. Medical management was rapidly escalated, involving diazoxide, hydrochlorothiazide, corticosteroids, octreotide followed by pasireotide, and dextrose infusion, guided by continuous glucose monitoring. Endoscopic ultrasound-guided fine needle biopsy (EUS-FNB) of the pancreatic tail lesion revealed a high-grade, well-differentiated neuroendocrine tumor (Ki67 50%, G3).

While awaiting access to systemic therapies, including everolimus, peptide receptor radionuclide therapy (PRRT), and chemotherapy, the patient underwent radiofrequency ablation (RFA) of the pancreatic tail lesion and transarterial embolization (TAE) of the hepatic lesions. However,

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repeat imaging two months later demonstrated rapid progression, with diffusely scattered and enlarging liver metastases throughout both lobes (largest measuring 7.9 x 16.8 x 15.2 cm).

The patient experienced frequent, severe hypoglycemic episodes requiring prolonged hospitalization, high-concentration dextrose administration via multiple central venous catheters, high-dose corticosteroids, and further escalation of medical therapy. Over the course of his hospitalization, he developed recurrent sepsis and multi-organ dysfunction, ultimately leading to his death.

CONCLUSION

This case illustrates the aggressive nature, management complexities and therapeutic challenges of metastatic insulinomas. Several studies demonstrated that early administration of systemic chemotherapy in high-grade insulinomas has been associated with improved survival. Early consideration of advanced therapies like everolimus, PRRT and chemotherapy may be crucial in managing malignant insulinomas.

EP_A126

A RARE ENCOUNTER: HIRSUTISM UNMASKING ADRENAL ONCOCYTIC NEOPLASM IN A YOUNG WOMAN

<https://doi.org/10.15605/jafes.040.S1.134>

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INTRODUCTION/BACKGROUND

Adrenal oncocytic neoplasms (AONs) are rare tumors, with fewer than 300 cases reported since their first description in 1986. Most AONs are benign, non-secretory, and discovered incidentally. Hormone-secreting AONs are exceptionally uncommon. We present a case of a testosterone- and cortisol-secreting AON in an 18-year-old woman with primary amenorrhea and hirsutism.

CASE

An 18-year-old female presented with increased hair growth and primary amenorrhea. She had a history of

unsuccessful hormonal therapy for amenorrhea since age 15. Physical examination revealed signs of hyperandrogenism, including hirsutism (Ferriman-Gallwey score 19), androgenic alopecia, deepened voice, and clitoromegaly. Pelvic ultrasound showed a small uterus with non-visualized ovaries. Laboratory investigations revealed elevated hematocrit (56%) and hormonal profiles indicative of hyperandrogenism and hypercortisolism. Abdominal computed tomography (CT) identified a 7.5 cm right adrenal mass with heterogeneous enhancement. A provisional diagnosis of a cortisol- and androgen-secreting adrenal tumor was made.

The patient underwent open right adrenalectomy with perioperative steroid coverage. Gross pathological examination was consistent with an AON. The tumor exhibited capsular and sinusoidal invasion but lacked vascular invasion, aberrant mitosis or necrosis. Based on Lin-Weiss-Bisceglia criteria, the tumor was classified as an AON of uncertain malignant potential.

Postoperatively, the patient experienced spontaneous menstruation five months after surgery. Follow-up CT at 15 months showed no recurrence or metastases, and hormonal profiles showed resolution of hyperandrogenism and hypercortisolism.

CONCLUSION

This case highlights a rare functional AON presenting with hyperandrogenism and hypercortisolism. Experienced pathologists play a crucial role in aiding accurate diagnosis. Complete surgical excision led to hormonal resolution and menstrual recovery, reinforcing the importance of considering adrenal tumors in young women with unexplained hyperandrogenism and amenorrhea.

EP_A127

AORTOCAVAL PARAGANGLIOMA IN VON HIPPEL-LINDAU DISEASE: A RARE EXTRA-ADRENAL PRESENTATION WITH DISTINCT BIOCHEMICAL AND CLINICAL PROFILE

<https://doi.org/10.15605/jafes.040.S1.135>

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INTRODUCTION/BACKGROUND

Pheochromocytomas and paragangliomas (PPGLs) are catecholamine-secreting tumors derived from chromaffin cells, with approximately 40% linked to germline mutations. One of the most common genetic associations is Von Hippel-Lindau (VHL) disease. VHL-related PPGLs typically arise in

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the adrenal glands, with only 10–20% occurring in extra-adrenal sites. Here we describe a patient who presented with VHL-associated aortocaval paraganglioma.

CASE

A 34-year-old female with poorly controlled hypertension on triple therapy presented with paroxysmal symptoms and worsening renal function since December 2023. She was admitted in May 2024 for hypertensive urgency and renal impairment. Ultrasonography and CT scan of the abdomen revealed a 6 cm retroperitoneal aortocaval mass. Biochemical tests confirmed elevated normetanephrine levels with normal metanephrine levels, and Ga-68 DOTATATE PET/CT imaging showed a somatostatin receptor-avid paraganglioma. Her family history was notable for a sibling with pheochromocytoma. She received adequate alpha-blockade, followed by successful surgical excision of the tumour. Histology confirmed a paraganglioma with low proliferative activity (Ki-67 <1%). Genetic testing revealed a VHL missense variant, confirming a diagnosis of VHL disease. Subsequent surveillance for other VHL-related manifestations revealed no additional tumours, and she is currently in remission for PPGL. Family screening identified five other individuals, including her young son, with the same genetic mutation; all are now undergoing regular follow-up.

CONCLUSION

VHL-associated PPGLs present at a younger age than sporadic cases and primarily secrete noradrenaline due to reduced PNMT expression, resulting from impaired hypoxic pathways caused by VHL loss. Patients often present with chronic hypertension and tachycardia. Functional imaging with 18F-DOPA PET/CT is preferred for its high sensitivity. These tumors have a low metastatic risk (5–8%) and rarely require systemic therapy. Given the high mutation penetrance of VHL disease (~90% by age 65), lifelong surveillance is essential. Early genetic and clinical monitoring enables timely detection in patients and at-risk relatives.

EP_A128

PERMANENT CENTRAL DIABETES INSIPIDUS IN A POST TRANSSPHEOIDAL SURGERY PATIENT: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.136>

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INTRODUCTION/BACKGROUND

Diabetes insipidus (DI) is a disorder characterized by the excretion of large volumes of hypotonic urine. Four types of DI must be differentiated: central DI (cDI), nephrogenic DI (nDI), gestational DI, and primary polydipsia. Central DI can be transient, particularly as a complication of pituitary surgery. Permanent central DI is a rare complication of pituitary surgery.

CASE

A 48-year-old female was referred from the neurosurgery department with a nine-month history of polyuria (urination exceeding 3 liters daily), polydipsia (excessive thirst), fatigue, constipation, and cold intolerance. Nine months prior, she underwent transsphenoidal surgery for a pituitary macroadenoma. She reported no prior history of hormonal abnormalities or use of antithyroid medication. Her body mass index (BMI) was 24.5 kg/m², and her thyroid gland was not enlarged.

Laboratory results showed: TSH 0.25 mIU/mL, FT4 9.5 pmol/L, cortisol 312 µg/dL, prolactin 268 ng/mL, LH 2 mIU/mL, FSH 11.35 mIU/mL, estradiol 62.3 pg/mL, sodium 143 mmol/L, urine osmolality 100 mOsm/kg, serum osmolality 292 mOsm/kg. A water deprivation test (WDT) revealed a urine osmolality of 212 mOsm/kg, which increased to 499 mOsm/kg after desmopressin administration.

The diagnoses were central diabetes insipidus (cDI) (post-operative, permanent), central hypothyroidism, and hypogonadotropic hypogonadism. Treatment consisted of desmopressin 0.6 mg once daily and thyroxine 50 mcg once daily. After six months, her signs and symptoms improved.

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CONCLUSION

Permanent central DI occurs when the hypothalamus and/or pituitary stalk is irreversibly injured. It is important to monitor DI in post pituitary surgery. One of the diagnostic tests used is WDT.

EP_A129

PYCNODYSTOSIS IN A YOUNG ADULT PRESENTING WITH FRAGILITY FRACTURE AND HIGH BONE MINERAL DENSITY: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.137>

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INTRODUCTION/BACKGROUND

Pycnodysostosis is a rare skeletal dysplasia and a subtype of osteopetrosis caused by deficient activity of the lysosomal protease cathepsin K (CTSK) gene. We present a case of pycnodysostosis exhibiting characteristic clinical features alongside a fragility fracture—an association that has been rarely reported.

CASE

A 33-year-old Malay female, born of a consanguineous marriage, presented with a subtrochanteric fracture of the proximal left femur following a trivial fall and underwent open reduction with locking compression plate fixation. She was noted to have a short stature (height: 145 cm). Further examination revealed frontal bossing, maxillary hypoplasia, and brachydactyly of both hands and feet. Other systemic examinations including neurological assessment were unremarkable. A family history of similar physical traits was noted in her late paternal grandmother, though medical records were unavailable. Her elder sister exhibited similar abnormalities. Laboratory investigations showed a low insulin-like growth factor 1 (IGF-1) level, but her insulin tolerance test was normal, excluding growth hormone deficiency. Other blood parameters, including complete blood count, serum calcium, phosphate, 25-hydroxy vitamin D, alkaline phosphatase, thyroid function tests, and cortisol were within normal limits. Bone mineral density (BMD) analysis revealed an elevated Z-score of +5.5 at the total hip and +0.9 at L1-L4. Genetic analysis identified a homozygous variant of uncertain significance in the CTSK gene, which is associated with autosomal recessive pycnodysostosis. Given the lack of a specific treatment, symptomatic management was initiated, focusing on fracture prevention, oral hygiene, and psychological support.

CONCLUSION

This case underscores the need to consider rare genetic skeletal dysplasias like pycnodysostosis in individuals presenting with fragility fractures, distinct craniofacial and skeletal features, and unusually high bone mineral density. Prompt recognition, aided by genetic testing, is crucial for guiding patient counseling, monitoring, and coordinated multidisciplinary care. Although no curative treatment exists, early diagnosis can help reduce complications and support a better quality of life.

EP_A130

A SURPRISING TWIST: HYPONATREMIA INDUCED BY ZOLEDRONIC ACID – A RARE CLINICAL PUZZLE

<https://doi.org/10.15605/jafes.040.S1.138>

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INTRODUCTION/BACKGROUND

Zoledronic acid is a potent bisphosphonate commonly used in the management of severe hypercalcemia, particularly in cases related to malignancy or metabolic bone disease. While hypocalcemia is a well-documented side effect following zoledronic acid administration especially in patients with primary hyperparathyroidism, there are no widely reported instances of hyponatremia in this patient population. We present a unique case of significant hyponatremia associated with zoledronic acid in a patient with primary hyperparathyroidism.

CASE

A 58-year-old female with a background of severe hypercalcemia secondary to primary hyperparathyroidism complicated by nephrocalcinosis and severe osteoporosis presented with recurrent hospital admissions due to severe hypercalcemic episodes. Over a two-month period, she received three doses of intravenous zoledronic acid infusion, followed by a single subcutaneous injection of denosumab. During follow-up in the clinic, laboratory investigations revealed significant hyponatremia (sodium: 113 mmol/L) and hypophosphatemia (phosphate: 0.52 mmol/L). There was no glycosuria, no hypokalemia, no hypouricemia and venous blood gas was not acidotic which excludes Fanconi syndrome. She was asymptomatic and denied any episode of vomiting or diarrhea. Secondary causes of adrenal insufficiency, syndrome of inappropriate antidiuretic hormone secretion (SIADH), hypothyroidism and diuretic use were excluded. With

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appropriate fluid management, her sodium levels gradually normalized.

CONCLUSION

This case highlights a rare but clinically significant adverse effect of zoledronic acid therapy. Hypocalcemia remains the more commonly expected metabolic complication. A few cases of hyponatremia associated with severe diarrhea or vomiting following zoledronic acid administration have been reported in the literature. However, our patient did not exhibit such gastrointestinal symptoms. Although the exact mechanism by which zoledronic acid contributes to hyponatremia remains unclear, early recognition is crucial to prevent potential complications.

EP_A131

A SEPTIC MASQUERADE: MULTIFOCAL SEPTIC ARTHRITIS REVEALING DISSEMINATED MELIOIDOSIS IN A YOUNG PATIENT WITH TYPE 1 DIABETES

<https://doi.org/10.15605/jafes.040.S1.139>

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INTRODUCTION/BACKGROUND

Melioidosis, caused by *Burkholderia pseudomallei*, is a potentially fatal infection endemic to Southeast Asia and northern Australia. While often linked to type 2 diabetes mellitus, disseminated melioidosis in patients with type 1 diabetes mellitus (T1DM) is exceedingly rare, more so if with musculoskeletal involvement. We report a case of disseminated melioidosis presenting with multifocal septic arthritis, a thigh abscess, pulmonary infection, and splenic microabscesses in an adolescent with T1DM, highlighting the need for heightened vigilance in endemic regions.

CASE

An 18-year-old Indian female with T1DM since age of 13 (HbA1c 10.3%), on insulin aspart and detemir, presented with five weeks of fever, one week of painful left thigh swelling, and three days of cough. She was admitted with severe diabetic ketoacidosis and was empirically treated with intravenous ampicillin-sulbactam. Ultrasound of the left thigh revealed an abscess, which was drained; pus culture was positive for *Burkholderia pseudomallei*. Antibiotics were escalated to intravenous ceftazidime and trimethoprim-sulfamethoxazole.

Despite treatment, she remained febrile and required intubation on day eight of admission due to respiratory compromise. Blood and respiratory cultures also isolated *Burkholderia pseudomallei*. Computed tomography of the thorax, abdomen, and pelvis showed pulmonary infection (patchy ground-glass opacities, bilateral consolidation, minimal pleural effusion) and splenic microabscesses. Joint ultrasound revealed bilateral knee effusions and a complex right ankle effusion. Emergency arthrotomies and washouts of all affected joints yielded the same organism.

Her fever resolved with marked clinical improvement following complete source clearance. She was discharged ambulatory after six weeks of intravenous ceftazidime and a five-month oral eradication course, with optimized glycemic control. Follow-up imaging confirmed resolution of all lesions.

CONCLUSION

This case highlights that the aggressive and atypical presentation of disseminated melioidosis in T1DM may delay diagnosis. Persistent fever in endemic areas warrants prompt reevaluation. Early antibiotic escalation, timely surgical intervention, and multidisciplinary care were keys to recovery.

EP_A132

A CASE SERIES OF THREE CHINESE-MALAYSIAN PATIENTS WITH VARIED CHARACTERISTICS OF LATENT AUTOIMMUNE DIABETES IN ADULT (LADA)

<https://doi.org/10.15605/jafes.040.S1.140>

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INTRODUCTION/BACKGROUND

Latent autoimmune diabetes in adults (LADA) is an autoimmune diabetes typically present in adulthood with initial insulin independence and positive anti-glutamic acid decarboxylase (GAD) antibodies. Most progress to insulin dependence within six months of diagnosis.

We present three Chinese-Malaysian patients with LADA, each demonstrating varied presentations and management, all culminating in diabetic complications.

CASE

Case 1. A 53-year-old lean male with a 22-year history of presumed Type 2 Diabetes Mellitus (T2DM), initially

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managed with oral agents for ten years and later transitioned to premixed insulin, presented with recurrent diabetic ketoacidosis (DKA). His first DKA was at 51 and subsequently required titration of insulin therapy during follow-up. Two years later, he experienced another DKA episode. Autoimmune testing revealed high-titer anti-GAD antibodies, confirming LADA.

Case 2. A 68-year-old male with a 15-year history of presumed T2DM presented with recurrent DKA following insulin interruption and initiation of an SGLT2 inhibitor. Initially managed with two oral agents, he experienced progressive glycemic deterioration after eight years, necessitating insulin therapy. He was positive for anti-GAD antibodies, confirming the diagnosis of LADA.

Case 3. A 71-year-old female with a three-year history of presumed T2DM was initiated on premixed insulin alongside oral agents due to poor glycemic control. Despite this, she experienced recurrent DKA, triggered by brief interruptions in insulin therapy. This raised suspicion for LADA despite her advanced age. Autoantibody was positive for anti-GAD antibodies, confirming the diagnosis.

CONCLUSION

These cases highlight the variable and delayed presentation of LADA, which is frequently misclassified as T2DM. A higher rate of LADA is observed amongst the ethnic Chinese population in Malaysia, mirroring the high prevalence amongst T2DM patients in China. Features such as early treatment failure, recurrent DKA and insulin sensitivity in Chinese ethnicity should raise clinical suspicion, as timely antibody testing is crucial for accurate diagnosis and management.

EP_A133

MACROPROLACTINOMA IN A POST-MENOPAUSAL WOMAN: A RARE CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.141>

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INTRODUCTION/BACKGROUND

Prolactinoma is a type of benign pituitary tumor that secretes prolactin derived from lactotropes and constitutes 50% of all pituitary adenomas. Microprolactinoma (diameter <10 mm) is the more common type and rarely develops into macroprolactinoma (diameter ≥10 mm). Prolactinomas are predominantly diagnosed in premenopausal women and postmenopausal cases are uncommon, often presenting with atypical symptoms.

CASE

A 63-year-old woman came to M Djamil General Hospital with complaints of narrowed visual fields and recurrent headaches. The patient had no history of malignancy. She had not menstruated for 15 years. There were no signs or symptoms of endocrine disorders. Laboratory tests revealed neutropenia (30%) and lymphocytosis (52%). Pituitary hormone examination showed the following results: prolactin level of 42.78 uIU/mL (normal range: 5.13–26.53), luteinizing hormone level of 3.65 uIU/mL (normal range: 0.58–14), follicle-stimulating hormone level of 23.98 uIU/mL (normal range: 1.38–5.47), and thyroid-stimulating hormone level of 3.75 uIU/mL (normal range: 0.25–5). An MRI scan of the head revealed an intrasellar tumor extending into the suprasellar region, suggestive of a pituitary macroadenoma, measuring approximately 42.61 × 28.06 × 45.1 mm, along with bilateral maxillary sinusitis. The patient was started on low-dose bromocriptine therapy at 0.625 mg orally once daily, with regular monitoring of treatment response. After three months of therapy, prolactin levels decreased significantly to <0.6 uIU/mL. A follow-up MRI scan was performed six months after therapy, revealing a reduction in tumor size (36.7 × 22.8 × 45 mm).

CONCLUSION

Prolactinoma diagnosed in postmenopausal women is less common due to hormonal changes. The absence of typical hyperprolactinemia symptoms due to the cessation of ovarian function makes the diagnosis challenging

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and often delayed. Despite their size and invasiveness, macroprolactinomas in postmenopausal women generally respond well to treatment with dopamine agonists.

EP_A134

CHARCOT ARTHROPATHY IN A CONTROLLED DIABETIC PATIENT: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.142>

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INTRODUCTION/BACKGROUND

Charcot arthropathy is a severe complication of diabetes which is often diagnosed late, characterized by a red, warm, and swollen foot with bone abnormalities on imaging. Most studies report elevated HbA1c as a risk factor in Charcot patients, but there are rare cases with normal HbA1c. If not promptly diagnosed and treated, the condition can lead to deformity, foot ulcers, amputation, and death.

CASE

A 54-year-old male came to M Djamil General Hospital with complaints of ulcers around the right ankle. The patient has a history of diabetes mellitus (13 years). We found deformity with ulcers and pus in the right ankle joint. We did several examinations to confirm the diagnosis. The laboratory results are random blood glucose 152 mg/dL; fasting blood glucose 65 mg/dL; two-hours postprandial glucose 111 mg/dL; HbA1c 7.0%. CT scan of the lower extremities found osteomyelitis of the tarsal bones with cellulitis; histopathology found chronic and acute inflammation with granulation tissue. The working diagnosis was Charcot arthropathy of the right distal tibia Brodsky Type 3A, and Type 2 Diabetes Mellitus. We performed immobilization, external fixation, sequestrectomy and boot casting and controlled glycemia with medical nutrition therapy and rapid acting insulin for perioperative management. We used antibiotics and analgesics to treat infection and pain. The results were good and the patient was advised to use ankle foot orthosis.

CONCLUSION

This is a rare case report of Charcot arthropathy in a patient with normal HbA1c. This condition may be associated with rapid HbA1c normalization, which can trigger acute episodes, and the duration of diabetes. Clinicians should assess glycemic history and neuropathic risk factors. Target HbA1c between 7.0 – 8.0% during treatment can facilitate wound healing without increasing mortality.

EP_A135

SEVERE HYPOTHYROIDISM-INDUCED RHABDOMYOLYSIS IN THE ABSENCE OF A TRIGGERING FACTOR

<https://doi.org/10.15605/jafes.040.S1.143>

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INTRODUCTION/BACKGROUND

Thyroid disorders are among the most common endocrine diseases globally, with hypothyroidism affecting approximately 3.4% of the Malaysian population. Muscle-related symptoms, such as fatigue, cramps, and myalgia are frequently observed in hypothyroidism and usually present with mild to moderate elevations of the muscle enzymes. However, rhabdomyolysis due to hypothyroidism, particularly in the absence of other apparent causes, is rare and is more frequently associated with Hashimoto's thyroiditis. The exact mechanism remains unclear, but it is hypothesized that hypothyroidism disrupts muscle metabolism, leading to prolonged oxidative damage and subsequently rhabdomyolysis.

CASE

We report a case of a 32-year-old male with no prior medical history who presented with one month of weight gain and lethargy, associated with facial puffiness for 2 weeks. He denied systemic symptoms, strenuous activity, trauma, alcohol use, or recent medications. No family history of thyroid or autoimmune disease was noted. Examination showed mild facial puffiness, no muscle weakness, and normal reflexes. Laboratory investigations revealed elevated creatinine kinase (CK) levels of 2,527 U/L (55-170), aspartate transaminase (AST) of 130.3 U/L (8-33), alanine transaminase (ALT) of 118.1 U/L (7-56) and acute kidney injury with urea 7.3 mmol/L (7-12), creatinine 182 µmol/L,

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and eGFR 37.6 mL/min. Thyroid function tests confirmed severe hypothyroidism with free T4 7.60 pmol/L (NR: 7.86-14.41), and TSH >49.40 mIU/L (NR: 0.38-5.33) with positive thyroid peroxidase antibody, confirming Hashimoto's thyroiditis. No other causes for rhabdomyolysis were identified and autoimmune hepatitis screening was negative. The patient was managed with aggressive intravenous hydration and levothyroxine replacement therapy, resulting in clinical and biochemical resolution.

CONCLUSION

This case underscores the importance of considering hypothyroidism in the differential diagnosis of unexplained rhabdomyolysis, especially in the absence of conventional triggers. Prompt recognition and early treatment are essential in preventing complications and ensuring optimal patient outcome.

EP_A136

MEMBRANOUS NEPHROPATHY IN A PATIENT WITH ELEVATED CARCINOEMBRYONIC ANTIGEN: AN UNUSUAL PRESENTATION OF MEDULLARY THYROID CARCINOMA

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INTRODUCTION/BACKGROUND

Membranous nephropathy is an important cause of nephrotic syndrome where immune complexes are deposited at the subepithelial space of the glomerular basement membrane. Membranous nephropathy is classified into primary and secondary causes, including infections, autoimmune, neoplasms, drugs or idiopathic. Medullary thyroid carcinoma (MTC) is a relatively rare malignant tumour subtype originating from the parafollicular cells of the thyroid gland, producing tumour markers such as calcitonin, carcinoembryonic antigen (CEA) and chromogranin A. The co-occurrence of membranous nephropathy with MTC is extremely uncommon, and such an association may reflect a paraneoplastic manifestation or an underlying monoclonal gammopathy.

CASE

We report the case of a 68-year-old woman who presented with progressive shortness of breath, bilateral lower limb edema, frothy urine, and periorbital puffiness. She denied orthopnea, paroxysmal nocturnal dyspnea, constitutional symptoms, or features suggestive of autoimmune disease. Initial workup revealed nephrotic-range proteinuria, and a renal biopsy demonstrated early membranous nephropathy.

Notably, her CEA level was persistently elevated; however, upper and lower gastrointestinal endoscopies showed only benign findings, including a hiatal hernia and sigmoid colon diverticulum. Contrast-enhanced CT imaging revealed a retrosternal goitre, while FDG-PET scanning identified an FDG-avid lesion in the left thyroid lobe with ipsilateral cervical lymphadenopathy. Thyroid ultrasound showed a TIRADS 4 nodule, and subsequent core needle biopsy confirmed MTC. She underwent total thyroidectomy and modified neck dissection without complications. At one-month postoperative follow-up, her proteinuria had slightly improved.

CONCLUSION

This case underscores the importance of a thorough malignancy workup in atypical presentations of nephrotic syndrome and highlights a rare paraneoplastic link between MTC and glomerular disease. In the context of raised CEA with negative findings despite extensive investigations for gastrointestinal tract causes, one might need to consider other non-gastrointestinal related causes for raised CEA such as medullary thyroid carcinoma.

EP_A137

NOT JUST TYPE 2 DIABETES: SEVERE INSULIN RESISTANCE WITH ATYPICAL FAT DISTRIBUTION SUGGESTS LIPODYSTROPHY SYNDROME

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INTRODUCTION/BACKGROUND

Lipodystrophy syndromes are rare disorders of adipose tissue distribution, often leading to severe insulin resistance and metabolic complications. We present a case of a young woman initially diagnosed with type 2 diabetes, unresponsive to standard insulin therapy, who was ultimately diagnosed with familial partial lipodystrophy (FPLD).

CASE

A 26-year-old female, with diabetes diagnosed 4 years ago, was referred for uncontrolled capillary blood glucose levels persistently ranging from 20–25 mmol/L despite high-dose basal-bolus insulin and oral hypoglycemic agents. Her body mass index (BMI) was 22 kg/m² with an HbA1c of 12.2%. Autoantibody screening (GAD, ICA, IA2 antibodies) was negative, and C-peptide was markedly elevated (2193 pmol/L). Her metabolic profile showed hypertriglyceridemia, raised liver enzymes suggestive

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of metabolic-dysfunction associated steatotic liver disease, proteinuria due to diabetic kidney disease, and oligomenorrhea.

She had a well-defined muscular appearance in her limbs with prominent veins, raising suspicion of partial lipodystrophy. Fat loss was noted in the trunk, hips, and gluteal regions, contrasting with fat accumulation in the face, neck, and viscera. Mild acanthosis nigricans were present, but there was no significant hirsutism.

She denied antiretroviral therapy use and autoimmune features were absent. While her family history was unremarkable for diabetes or consanguinity, her mother had died from renal failure at the age of 40. She acknowledged a distinct body habitus compared to her siblings.

Whole-exome sequencing confirmed the presence of a heterozygous pathogenic p.Arg482Trp variant in the LMNA gene, diagnostic of autosomal dominant FPLD type 2. Her management included increased insulin doses and the addition of pioglitazone to enhance adiponectin levels and insulin sensitivity.

CONCLUSION

This case highlights the need to consider lipodystrophy syndromes in young patients with severe insulin resistance and atypical fat distribution. Early diagnosis enables targeted therapy and better metabolic control.

EP_A138

DORSAL PANCREATIC AGENESIS PRESENTING AS NEW-ONSET TYPE 3C DIABETES IN A YOUNG MALAYSIAN ADULT: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.146>

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INTRODUCTION/BACKGROUND

Type 3C diabetes mellitus (DM), secondary to exocrine pancreatic disease, is uncommon. Dorsal pancreatic agenesis (DPA), a rare congenital absence of part of the pancreas, can lead to both exocrine insufficiency and DM. We present a unique case of new-onset Type 3C DM due to DPA in a young Malaysian adult.

CASE

A 26-year-old Malaysian male with no known medical illness presented with a 6-month history of worsening loose

stools, significant 15 kg weight loss, increased hunger, foot numbness, and blurred vision. His initial blood glucose was very high (58.7 mmol/L), leading to a diagnosis of new-onset DM. However, his weight loss and diarrhea were atypical. Tests for viral hepatitis, HIV, and diabetes autoantibodies were negative. Colonoscopy was normal. A CT scan of the abdomen revealed findings suggestive of DPA. Consequently, a diagnosis of Type 3C DM secondary to DPA and likely exocrine pancreatic insufficiency-related diarrhea was made. He was started on insulin, and his gastrointestinal symptoms improved moderately with diet and lifestyle changes.

DPA is a rare cause of DM, especially in young adults. The absence of typical autoimmune markers and the presence of significant exocrine symptoms were key in identifying this unusual etiology. The development of diabetes in DPA is thought to be due to reduced pancreatic beta-cell mass. This case highlights the importance of considering rare causes like DPA in atypical diabetes presentations. Thorough evaluation, including imaging, is crucial for accurate diagnosis and management. While insulin therapy was initiated, dietary modifications provided some relief for his gastrointestinal issues.

CONCLUSION

This case demonstrates a rare instance of Type 3C DM secondary to DPA in a young Malaysian adult. It emphasizes the need for awareness of such unusual associations in young patients with new-onset diabetes and unexplained gastrointestinal symptoms. Further research on DPA-related diabetes in Malaysia is warranted.

EP_A139

CASE REPORT: BEYOND THE TOXICOLOGY SCREEN: RECOGNIZING THYROID STORM IN A PATIENT INITIALLY SUSPECTED OF SUBSTANCE-INDUCED CARDIOMYOPATHY

<https://doi.org/10.15605/jafes.040.S1.147>

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INTRODUCTION/BACKGROUND

Thyroid storm is a life-threatening endocrine emergency characterized by exaggerated hyperthyroidism. Its diverse clinical manifestations can sometimes mimic other acute conditions, leading to diagnostic challenges. We present a unique case of a young adult with thyroid storm whose initial presentation strongly suggested substance-induced cardiomyopathy.

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CASE

A 29-year-old Malay male with a history of active smoking and drug abuse presented with a one-week history of non-productive cough and sudden onset of shortness of breath after physical exertion. On initial assessment, he exhibited marked restlessness, diaphoresis, and irregular narrow complex tachycardia on electrocardiogram (ECG). Urine toxicology was positive for amphetamine and methamphetamine, leading to an initial suspicion of substance-induced cardiomyopathy complicated by rapid atrial fibrillation. He was managed with anti-arrhythmics and non-invasive ventilation. However, persistent tachycardia and clinical deterioration necessitated intubation. A chest X-ray revealed cardiomegaly.

Interestingly, routine thyroid function tests, which were ordered due to the patient's unexplained tachycardia, returned with a significantly suppressed thyroid-stimulating hormone (TSH) of <0.008 mIU/L and an elevated free thyroxine (FT4) of 64 pmol/L. This, coupled with a Burch-Wartofsky score of 65, strongly indicated thyroid storm. The initial diagnosis was revised accordingly. Despite aggressive management for thyroid storm, including anti-thyroid medications, beta-blockers, and supportive care, the patient developed acute infarcts in the right middle cerebral artery territory with subsequent hemorrhagic transformation and significant cerebral edema on serial computed tomography (CT) scans of the brain. Neurosurgical intervention was considered but declined by the family due to the guarded prognosis. The patient eventually succumbed to death due to massive cerebral infarct.

This case highlights the importance of considering thyroid storm in the differential diagnosis of young adults presenting with acute cardiac symptoms and agitation, even in the presence of positive toxicology screens. The initial clinical picture and positive drug screen misleadingly pointed towards a primary cardiac etiology. The significantly abnormal thyroid function tests were crucial in establishing the correct diagnosis. While the exact mechanism of the cerebral infarction in this context remains unclear, it could be a rare complication of severe thyroid storm, potentially exacerbated by underlying substance abuse or other unidentified factors. This case underscores the need for a broad differential diagnosis and timely thyroid function testing in patients with unexplained acute cardiovascular symptoms, particularly when atypical features are present.

CONCLUSION

This case serves as a reminder of the protean manifestations of thyroid storm and the potential for diagnostic confusion with other acute conditions. A high index of suspicion and prompt laboratory investigations are essential for timely and accurate diagnosis, which is critical for improving patient outcomes in this life-threatening endocrine emergency.

EP_A140

DELAYED DIAGNOSIS OF LYMPHOCYTIC HYPOPHYSITIS PRESENTING AS CHRONIC HEADACHES: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.148>

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INTRODUCTION/BACKGROUND

Lymphocytic hypophysitis (LH) is a rare autoimmune inflammatory disorder of the pituitary gland, often presenting with non-specific symptoms leading to diagnostic delays. This case highlights the challenges in the timely diagnosis of LH and the consequences of treatment default in a young male.

CASE

A 37-year-old male with a three-year history of chronic headaches, initially managed symptomatically, presented with recurrent episodes of worsening headaches, vomiting, and chest pain requiring multiple emergency department visits over two years. After 2 years of intermittent visits, a non-contrast computed tomography (CT) brain, performed due to persistent and escalating headaches, revealed a possible pituitary fossa mass. Subsequent urgent magnetic resonance imaging (MRI) confirmed a bulky pituitary gland with minimal suprasellar extension. Hormonal evaluation revealed panhypopituitarism. Based on clinical and radiological findings, a diagnosis of lymphocytic hypophysitis was suspected. The patient was commenced on hydrocortisone and thyroid hormone replacement. Regrettably, the patient defaulted on follow-up and discontinued his prescribed medications in favour of traditional treatment.

This case underscores the insidious presentation of LH, where chronic headaches can be the predominant initial symptom, leading to significant delays in diagnosis. The patient's repeated emergency department visits for non-specific symptoms highlight the need for a high index of suspicion for underlying endocrine disorders in patients with persistent and evolving complaints. The eventual radiological findings of a pituitary mass and subsequent confirmation of panhypopituitarism were crucial for suspecting LH. The patient's decision to discontinue conventional treatment and opt for traditional remedies emphasizes the importance of patient education, adherence strategies, and culturally sensitive approaches in managing chronic endocrine conditions. The potential long-term sequelae of untreated panhypopituitarism warrants concern.

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CONCLUSION

This case serves as a reminder of the diverse clinical manifestations of lymphocytic hypophysitis and the importance of considering pituitary pathology in patients with chronic headaches. Early radiological investigation in cases of persistent or worsening headaches, coupled with hormonal evaluation, is crucial for timely diagnosis and management. Furthermore, addressing factors influencing treatment adherence, including patient beliefs and preferences, is essential for optimal outcomes in chronic endocrine disorders.

EP_A141

CUTANEOUS TUMOUR IN MULTIPLE ENDOCRINE NEOPLASIA TYPE 1

<https://doi.org/10.15605/jafes.040.S1.149>

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INTRODUCTION/BACKGROUND

Multiple Endocrine Neoplasia (MEN) 1 syndrome is a genetic disease characterized by pituitary adenoma, parathyroid hyperplasia, and pancreatic tumors. Cutaneous manifestations of the syndrome are rare. We report a case of MEN 1 syndrome with collagenoma, initially misdiagnosed as neurofibromatosis.

CASE

A 48-year-old male with hypertension and gouty arthritis was diagnosed with Neurofibromatosis type 1 10 years ago based on neurofibromas on his neck and abdomen. He defaulted follow-up until admission in June 2024 for hypertensive emergency. During admission, incidental findings of asymptomatic moderate PTH-dependent hypercalcemia (corrected calcium: 3.02 mmol/L, phosphate: 0.37 mmol/L, iPTH: 31.9 pmol/L [Normal range 1.58 - 6.03]) and a suspicious right hilar mass on chest X-ray, prompting further workup. Calcium-creatinine clearance ratio was 0.046. His tumor marker levels, including B-HCG, were normal. CT of the thorax, abdomen, and pelvis showed a solid anterior mediastinal mass with a superior mediastinal lesion and an enhancing pancreatic mass. A DOTATATE scan confirmed somatostatin receptor avid disease in the pancreas and anterior mediastinal mass with nodal, liver, and bone metastases. CT-guided biopsy of anterior mediastinal mass followed by anterior mediastinal mass resection and endoscopic guided biopsy of pancreatic mass confirmed neuroendocrine tumor. His clinical condition

was suggestive of MEN 1. It is exceptionally rare for two autosomal dominant syndromes to coexist. There was no other diagnostic manifestation for neurofibromatosis type 1 except for skin lesions. Skin biopsy was done and reported as collagenoma, supporting MEN 1. Genetic study is ideal but limited by financial constraints.

CONCLUSION

Without the luxury of genetic testing, tactful correlations of clinical manifestations are essential to diagnose rare inheritable syndromes. Relevant investigations may help identify clinical signs and their association with the syndrome. In our patient, the initial diagnosis of neurofibromatosis was later revised to collagenoma with MEN 1 after HPE result from skin biopsy.

EP_A142

MYXOEDEMA MADNESS: WHEN HYPOTHYROIDISM TURNS PSYCHOTIC

<https://doi.org/10.15605/jafes.040.S1.150>

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INTRODUCTION/BACKGROUND

Myxoedema madness is a rare but serious neuropsychiatric manifestation of severe hypothyroidism. We report a case of overt hypothyroidism following radioactive iodine (RAI) therapy, presenting with paradoxical psychotic symptoms.

CASE

A 43-year-old Malay male with underlying ischemic heart disease, severe mitral regurgitation, and toxic multinodular goiter (MNG) was admitted for acute behavioral changes. Diagnosed with toxic MNG in 2019, he was initially treated with carbimazole before undergoing his first RAI therapy in January 2024. He reported mood swings that improved after starting carbimazole, not needing psychiatric evaluation. A second RAI therapy was administered in January 2025 after failed first therapy. Two months post-RAI therapy, his thyroid function test (TFT) showed a TSH of 7.6 mIU/L and a free T4 of 7.8 pmol/L; hence, carbimazole 2.5 mg daily was withheld, and he was scheduled for follow-up. One month after stopping carbimazole, he presented to the Emergency Department with a five-day history of disorganized behavior, irrelevant speech, and bizarre ideations. Clinically, he was restless, requiring physical restraint. There were no signs of meningism or hypothyroidism. His inflammatory markers and brain CT were normal, and both urine drug toxicology and infective screening were negative. A repeat TFT confirmed overt

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hypothyroidism post-RAI therapy (TSH >49.9 mIU/L, free T4 3.9 pmol/L) and levothyroxine 50 mcg daily was started. He was also started on risperidone by the psychiatric team for acute delirium secondary to hypothyroidism. Following treatment, he became calmer and more manageable.

CONCLUSION

Myxoedema madness has been reported in patients with untreated or inadequately treated hypothyroidism, particularly post-thyroidectomy or RAI therapy, and in patients with psychiatric comorbidities. Symptoms such as hallucinations, delusions, and disorganized behavior are typically reversible with appropriate treatment, including thyroid hormone replacement. Clinicians should maintain vigilance for myxoedema madness in hypothyroid patients presenting with acute behavioral changes.

EP_A143

A CASE OF PANHYPOPHYSITIS THAT MYSTERIOUSLY DISAPPEARED

<https://doi.org/10.15605/jafes.040.S1.151>

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INTRODUCTION/BACKGROUND

Panhypophysitis is a rare inflammatory condition that affects the entire pituitary gland, predominantly affecting women of reproductive age. Presentation is often vague, complicating diagnosis and management. We report a possible lymphocytic panhypophysitis that resolved with corticosteroids given for another indication.

CASE

A 31-year-old Indonesian female with an underlying diabetes mellitus presented with lethargy, polyuria, and polydipsia for four months. Previously, she had 3 uneventful deliveries. She was admitted for hyperosmolar hyperglycaemic state and noted to have persistent hyponatraemia with urine output of 3-4 litres daily. Further investigations were consistent with central diabetes insipidus (urine osmolality: 74 mOsm/kg, serum osmolality: 337 mOsm/kg, serum sodium: 152mmol/L), and responded to desmopressin. Anterior pituitary hormones showed central hypothyroidism (TSH 0.14 mIU/L, T4: 7.8 pmol/L), hypogonadotropic hypogonadism (LH 0.9 IU/L, FSH 3.4 IU/L, estradiol 108 pmol/L) and secondary hypocortisolism (18 nmol/L). She received hormonal replacement. MRI pituitary reported a homogeneously-enhancing pituitary lesion extending into the suprasellar region, which abuts

the chiasm, with loss of the posterior pituitary bright spot, concerning for panhypophysitis. Further investigations for secondary hypophysitis were negative. Later, she was admitted for bilateral lower limb weakness and sensory deficit, with initial concern of transverse myelitis, and she was started on IV methylprednisolone for 3 days. Subsequent MRI spine revealed no spinal cord pathology, and the diagnosis was revised to diabetic neuropathy. Follow-up MRI pituitary after 9 months showed complete resolution of the pituitary lesion and normalization of the infundibulum. Her clinical condition improved, and the desmopressin dosage was reduced.

CONCLUSION

The resolution of the pituitary lesion after high-dose corticosteroids in our case supports a diagnosis of lymphocytic hypophysitis, the most common form of hypophysitis. High-dose steroids likely halted the inflammatory process, resulting in structural and functional recovery. A trial of medical therapy may be considered in similar cases before opting for surgical intervention.

EP_A144

DIFFERENT CLINICAL PRESENTATIONS OF PARAGANGLIOMA FROM TWO DIFFERENT ORIGINS: A CASE SERIES

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INTRODUCTION/BACKGROUND

Paragangliomas are rare neuroendocrine tumours that arise from extra-adrenal paraganglia. Presentation can vary based on the anatomic origin. Sympathetic paragangliomas typically manifest with classic adrenergic symptoms. Here we present two cases of functional paraganglioma from two different origins.

CASE

A 25-year-old Malay female was diagnosed with pregnancy-induced hypertension during her last pregnancy 3 years ago, necessitating admission for impending eclampsia at 37 weeks. She complained of palpitations and chest pain. Post-partum, she remained hypertensive. A workup for secondary hypertension revealed marked elevation of 24-hour urine normetanephrines, 36 times the upper limit of normal (95.42 umol/day) and 2.5 times the elevation of

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3-Methoxytyramine (4.46 $\mu\text{mol/day}$). Adrenal CT showed a well-defined, enhancing lesion at the aortocaval region, measuring 5.1 \times 5.8 \times 7.0 cm. CT scans of the neck and thorax were unremarkable. A Gallium-68 PET scan demonstrated SSTR-avid uptake in the aortocaval mass, with no evidence of SSTR-avid disease elsewhere. Currently, she requires three antihypertensive agents to control her blood pressure while awaiting surgical intervention.

A 67-year-old Malay female with underlying hypertension, diabetes, and ischaemic stroke had multiple admissions for urosepsis. Ultrasound revealed a bladder mass suspicious for malignancy, a left ureteric stone, and hydronephrosis. CT and MRI showed a 4.0 \times 3.6 \times 3.7 cm heterogeneously enhancing mass arising from the right lateral bladder wall. She underwent transurethral resection of bladder tumour (TURBT); intraoperatively, her blood pressure was labile with systolic BP of 65-320 mm Hg. Histopathology confirmed paraganglioma. Post-operative 24-hour urine normetanephrines were four times the upper limit of normal. Due to her poor performance status, she was managed conservatively. Her blood pressure is currently controlled on double antihypertensives.

CONCLUSION

Paragangliomas can present variably depending on their anatomical origin and catecholamine-secreting status. A high index of suspicion, appropriate biochemical testing, and functional imaging are key to diagnosis. Individualized management is essential, especially in patients with comorbidities or poor performance status.

EP_A145

STERIOD-UNMASKED CENTRAL DIABETES INSIPIDUS IN A PATIENT WITH PITUITARY METASTASIS FROM BREAST CARCINOMA: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.153>

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INTRODUCTION/BACKGROUND

Pituitary metastases are rare but clinically significant, most commonly originating from breast or lung cancers. Diabetes insipidus (DI) is the most frequent manifestation of posterior pituitary involvement. We describe a case of pituitary metastasis presenting with panhypopituitarism and central diabetes insipidus (CDI), initially unmasked by adrenal insufficiency.

CASE

A 68-year-old female with metastatic left breast carcinoma, post-mastectomy and on hormonal therapy, presented with a generalized tonic-clonic seizure and a Glasgow Coma Scale (GCS) score of 4. She exhibited persistent hypoglycemia requiring repeated dextrose corrections, along with hypotensive episodes.

Brain CT revealed a well-defined iso-to-hyperdense lesion in the sellar and suprasellar regions (2.0 \times 2.5 \times 3.0 cm). Subsequent pituitary MRI showed a heterogeneously enhancing lobulated mass (2.2 \times 2.5 \times 3.0 cm) with loss of normal anterior pituitary architecture.

Laboratory tests confirmed adrenal and thyroid insufficiency, with a random cortisol level of 284 nmol/L, TSH at 0.072 $\mu\text{IU/mL}$, and free T4 below 3.20 mmol/L. Hydrocortisone therapy was initiated, leading to a significant increase in serum sodium from 132 to 160 mmol/L. Serum and urine osmolality measured 318 and 183 mOsm/kg, respectively, with urine sodium under 10 mmol/L, raising suspicion for CDI. Desmopressin was commenced, resulting in improved sodium (145 mmol/L) and osmolality levels (serum 335 mOsm/kg, urine 646 mOsm/kg). Gonadotropin levels (FSH, LH) and estradiol were also low, indicating panhypopituitarism.

A multidisciplinary team confirmed pituitary metastasis secondary to breast carcinoma. The patient was transitioned to palliative care with hormone replacement: hydrocortisone, desmopressin, and levothyroxine.

CONCLUSION

Hypocortisolism in breast cancer patients should raise suspicion for pituitary metastasis. Polyuria after steroid therapy may indicate underlying central diabetes insipidus. Prompt diagnosis and hormone replacement can significantly enhance symptom management and patient well-being.

EP_A146

A CURIOUS CASE OF RECURRENT HYPOGLYCAEMIA IN NEUROFIBROMATOSIS

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INTRODUCTION/BACKGROUND

Neurofibromatosis type 1 (NF-1) is commonly associated with neural tumors such as pheochromocytomas, para-

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gangliomas, and duodenal somatostatinomas. However, its association with insulinoma is extremely rare, with only a few cases reported.

CASE

A 66-year-old female with longstanding NF-1 presented with a six-month history of recurrent symptomatic hypoglycaemia. She was non-diabetic, lived in a nursing home, and had no history of hypoglycaemic agent use. Her episodes, typically occurring during fasting, were associated with intense hunger and resolved with food intake. Capillary glucose readings ranged from 1.7 to 3.1 mmol/L. She was admitted after being found unconscious with a glucose level of 1.7 mmol/L. Clinical examination revealed multiple dermal neurofibromas and café-au-lait spots. In the ward, paired samples during spontaneous hypoglycaemia (glucose 1.6 mmol/L) showed inappropriately elevated insulin (53.9 pmol/L) and C-peptide (465 pmol/L). After intramuscular glucagon (1 mg), her blood glucose rose from 3.0 to 6.4 mmol/L over 60 minutes. Morning cortisol (450 nmol/L) and IGF-1 (113.5 ng/mL) were normal. β -hydroxybutyrate, IGF-2, and sulphonylurea levels were not tested due to financial limitations. These findings confirmed the presence of endogenous hyperinsulinaemia. A pancreatic CT scan was scheduled to localize the suspected insulinoma but was missed twice. She later re-presented with a seizure, likely secondary to hypoglycaemia, as both brain CT and EEG findings were unremarkable. To better control her hypoglycaemia, diazoxide and a calcium channel blocker were initiated. Imaging was subsequently rescheduled to aid in localizing the insulinoma.

CONCLUSION

Although rare, insulinoma should be considered in NF-1 patients presenting with recurrent hypoglycaemia. Early recognition and appropriate investigation are crucial to prevent serious complications.

EP_A147

POLYGLANDULAR AUTOIMMUNE SYNDROME TYPE 3: THE UNEXPECTED TRILOGY

<https://doi.org/10.15605/jafes.040.S1.155>

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INTRODUCTION/BACKGROUND

Polyglandular autoimmune syndromes (PGAS) are a rare group of disorders characterized by the presence of two or more autoimmune endocrine diseases. Polyglandular

autoimmune syndrome type 3 (PGAS-3) is characterized by the presence of autoimmune thyroid disease associated with other autoimmune diseases excluding adrenal insufficiency and hypoparathyroidism. This case report focuses on a patient who developed a sequential presentation of pernicious anemia, Graves' disease, and later type 1 diabetes mellitus (T1DM), raising suspicion for PGAS-3.

CASE

A 30-year-old male presented in 2019 with fatigue, weight loss, and palpitations. Initial investigations revealed pancytopenia with Hb 4 mmol/L, WBC 3.99 mmol/L, PLT 87 mmol/L and a critically low vitamin B12 level, alongside positive anti-parietal cell antibodies, indicative of pernicious anemia. Oesophagogastroduodenoscopy (OGDS) was done later in 2023, showing pangastritis. Concurrently, the patient was found to have hyperthyroidism with positive thyroid antibodies, consistent with Graves' disease. He was treated with subcutaneous cyanocobalamin and antithyroid medications, resulting in partial improvement. He underwent radioactive iodine therapy for Graves' disease. Four years later, the patient was hospitalized for uncontrolled diabetes mellitus. Insulin autoantibodies were requested and results of anti-islet cell antibodies, anti-glutamic acid decarboxylase and anti-insulinoma-associated antigen-2 were positive, leading to the diagnosis of T1DM. Adrenocorticotrophic hormone (ACTH) and early morning serum cortisol were normal, excluding adrenal involvement. The coexistence of Graves' disease, pernicious anemia, and T1DM fulfilled the diagnostic criteria for PGAS-3. Genetic testing and further autoimmune screening were recommended for a more comprehensive understanding of the underlying pathophysiology.

CONCLUSION

The patient's progression from pernicious anemia to Graves' disease followed by T1DM is consistent with polyglandular autoimmune syndrome type 3. Clinicians should be vigilant in identifying PGAS in patients with multiple autoimmune endocrine disorders to ensure appropriate diagnosis and treatment, mitigating its potential long-term consequences.

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EP_A148

CASE REPORT: RECURRENT UNILATERAL ALDOSTERONE-PRODUCING ADRENAL ADENOMA

<https://doi.org/10.15605/jafes.040.S1.156>

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INTRODUCTION/BACKGROUND

Primary aldosteronism (PA) is the most common cause of secondary hypertension. While adrenalectomy can be curative in unilateral cases, recurrence after total adrenalectomy is exceedingly rare. We describe a rare case of recurrent PA requiring two adrenalectomies, eight years apart.

CASE

A 43-year-old male was diagnosed with hypertension at age 30 and initially required four antihypertensive agents for blood pressure control. In 2012, biochemical screening confirmed PA, with an aldosterone-renin ratio (ARR) of 1991 and a post-saline suppression aldosterone level of 600 pmol/L. Adrenal CT revealed a hypodense lesion in the lateral limb of the left adrenal gland measuring 0.9×1.3 cm with HU of -4 to 20. Adrenal venous sampling (AVS) was performed but yielded inconclusive results due to failed cannulation of the left adrenal vein. In 2015, he underwent retroperitoneoscopic left adrenalectomy, with histopathology confirming an adrenal cortical adenoma. Postoperatively, his blood pressure improved and was maintained on a single antihypertensive agent.

Over the following years, his blood pressure gradually increased, requiring multiple medications. In 2023, repeat screening showed an ARR of 238 and adrenal CT showed a recurrent lesion in the left adrenal bed. A second left adrenalectomy was performed in December 2024. Postoperatively, his blood pressure normalized without the need for antihypertensives.

This case highlights the rare occurrence of recurrent PA after unilateral adrenalectomy. Possible mechanisms include residual hyperfunctioning adrenal tissue or the development of a new aldosterone-producing lesion in the ipsilateral adrenal bed. Some studies suggest that patients with certain genetic mutations such as KCNJ5 may be predisposed to developing multiple aldosterone-producing nodules, either at the time of initial surgery or later in the remaining adrenal tissue.

CONCLUSION

Recurrent PA after unilateral adrenalectomy is rare but clinically significant. Lifelong monitoring of blood

pressure post adrenalectomy is essential. Repeat surgical intervention can achieve biochemical remission and restore blood pressure control in cases of recurrence.

EP_A149

WHEN NUMBERS DON'T ADD UP: DISCORDANT THYROID FUNCTION IN HIV INFECTION

<https://doi.org/10.15605/jafes.040.S1.157>

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INTRODUCTION/BACKGROUND

Thyroid function test (TFT) abnormalities in retroviral disease (RVD) are well documented, ranging from isolated low fT4 levels to overt hypothyroidism. However, careful evaluation of TFTs is essential as it presents a diagnostic challenge.

CASE

This is a case of a 28-year-old male with hypertension and end-stage kidney disease on peritoneal dialysis. He was diagnosed with vertical transmission of HIV since childhood, and the viral load is suppressed by regular antiretroviral therapy; oral lamivudine 500 mg daily, oral efavirenz 600 mg daily and oral abacavir 600 mg daily. He was referred for abnormal TFT, fT4 7.88 (9.01-19.05 pmol/L), TSH 1.99 (0.35-4.94 mIU/L). TFT was done for a pre-cadaveric renal transplant workout. Clinically, he is euthyroid without any palpable goiter. He denied consuming biotin-containing supplementation.

The results of the serial TFT showed a similar pattern. The pituitary hormonal workout excludes a central cause. Total T4 was normal at 68.1 (66-181 nmol/L). TSH assay interference was subsequently evaluated. Of the three different analyzers used (Lab A, B, and C), Lab A analyser displayed normal TFT results: TSH 1.93 (0.4-4 mIU/L), fT4 10.8 (10-26 pmol/L), while the other lab shows low fT4 and normal TSH. Macro-TSH, heterophile antibody and rheumatoid factor interference run by Lab B were negative.

CONCLUSION

A low fT4 level combined with a normal TSH level may be affected by multiple factors, such as antiretroviral therapy (ART) and assay interference, as seen in this patient's case. Due to limited resources and testing capacity, the specific

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antibody or causative agent cannot be identified. It is essential to take the necessary actions to eliminate other causes for the discordant TFT results and to prevent unnecessary thyroxine replacement. For this patient, any future TFT testing should be conducted at Lab A to rule out any potential assay interference with upcoming samples, if needed.

EP_A150

THE PARADOX OF PLENTY: WHEN GLUCOCORTICOID RESISTANCE SYNDROME MEETS SYSTEMIC LUPUS ERYTHEMATOSUS

<https://doi.org/10.15605/jafes.040.S1.158>

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INTRODUCTION/BACKGROUND

Glucocorticoid resistance syndrome (GRS) is a rare condition characterized by biochemical hypercortisolism without the typical clinical manifestations of Cushing's syndrome. Patients with GRS exhibit elevated serum cortisol, increased 24-hour urinary free cortisol, normal to elevated ACTH, non-suppressed low-dose dexamethasone-suppression test results and preserved circadian rhythm, which are findings that help distinguish it from Cushing's disease. It is associated with various mutations in the NR3C1 gene, which encodes the glucocorticoid receptor. Clinical presentations can vary from being asymptomatic to exhibiting features of mineralocorticoid or androgen excess such as hypertension with hypokalemia or hyperandrogenism.

CASE

A 51-year-old female with type-2 diabetes mellitus, hypertension, and dyslipidemia presented with bilateral lower limb edema and intermittent facial flushing. Her BMI was within normal range, and her blood pressure and blood glucose were well-controlled. Notably, she had persistent hypokalemia and elevated cortisol levels. MRI of the pituitary revealed a partial empty sella with a suspected right-sided pituitary adenoma. Her bone mineral density was also normal. Inferior petrosal sinus sampling confirmed ACTH-dependent hypercortisolism. However, in the absence of clinical features of Cushing's syndrome, diagnosis of GRS was made.

She was started on dexamethasone, leading to significant reduction in cortisol levels over nine months. However,

her condition was complicated by recurrent infections, soft tissue abscesses, and a newly diagnosed systemic lupus erythematosus (SLE) with concomitant lupus nephritis. Frequent steroid adjustments were necessary to manage autoimmune flares, which, in turn, increased her risk for opportunistic infections, culminating in severe *Pneumocystis jirovecii* pneumonia.

CONCLUSION

This case illustrates the diagnostic and therapeutic challenges of managing GRS, particularly when complicated by autoimmune disease and infection risk. While dexamethasone is effective in suppressing the HPA axis in GRS due to its glucocorticoid receptor affinity and mineralocorticoid-sparing properties, its use in patients with concurrent immunosuppressive conditions like SLE requires careful balance to avoid immunosuppression-related complications. Individualized steroid management is crucial to optimize outcomes and minimize adverse events.

EP_A151

DIAZOXIDE-INDUCED HYPERGLYCAEMIC CRISIS IN AN ELDERLY: A TRAP FOR THE UNWARY

<https://doi.org/10.15605/jafes.040.S1.159>

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INTRODUCTION/BACKGROUND

Diazoxide inhibits pancreatic insulin secretion and is a well-established pharmacological agent for management of hypoglycaemia in insulinoma. Hyperglycemic emergencies associated with its use are rare, being mostly reported in the elderly and in children.

CASE

An 88-year-old female with hypertension and dyslipidaemia presented to the emergency room with syncope and was noted to be hypoglycaemic with capillary glucose of 2.6 mmol/L. She reported a year-long history of recurrent presyncopal episodes and early morning hunger pangs. Renal profile, 8 am cortisol, thyroid and liver function tests were normal. Laboratory tests confirmed endogenous hyperinsulinemia (random blood glucose: 1.7 mmol/L, serum insulin 373 pmol/L, C-peptide 3054 pmol/L) with negative sulfonylurea screening. CT imaging revealed a 0.4 x 0.9 cm hypodense lesion in the proximal pancreas. She was started on diazoxide and was advised glucose

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monitoring and dietary modifications. Her capillary blood glucose remained stable (5–7 mmol/L) on follow-up. However, weeks later, she presented again with reduced responsiveness. Investigations revealed overlapping diabetic ketoacidosis and hyperosmolar hyperglycaemic state with acute kidney injury (glucose 32 mmol/L, ketones 7.5 mmol/L, pH 7.2, HCO₃ 15mmol/L, Na 162 mmol/L, urea 27 mmol/L, creatinine 309 mol/L, osmolality 362 mOsm/L). CXR showed right lower zone consolidation. She was treated with antibiotics and insulin, requiring up to 30 units per day when steroid was added for bronchospasm. After recovery and weaning of steroids, insulin was tapered off. However, she experienced further episodes of hypoglycaemia despite being off all glucose lowering medication. Diazoxide was resumed at 100 mg every other day. Family opted for nonsurgical management and she remained well with normal home glucose profile on follow up 3 months later.

CONCLUSION

This case highlights the rare but potentially life-threatening side-effect of diazoxide. The risk is heightened in the elderly, especially when confounded by renal impairment, high doses, intercurrent illness or steroid use. Awareness and vigilant monitoring are essential in the vulnerable to avoid adverse outcome.

EP_A152

THE ROLE OF DAPAGLIFLOZIN AS AN ADJUNCTIVE THERAPY IN SIADH-INDUCED HYPONATREMIA

<https://doi.org/10.15605/jafes.040.S1.160>

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INTRODUCTION/BACKGROUND

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) leads to impaired water excretion and dilutional hyponatremia. Sodium-glucose cotransporter 2 inhibitors (SGLT2i), which were initially developed for diabetes and heart failure, have shown promise as a novel treatment for chronic SIADH-related hyponatremia based on recent studies.

CASE

We report the case of a 66-year-old male with comorbidities of systemic lupus erythematosus, heart failure and adrenal insufficiency on steroid replacement. His heart failure medications included furosemide, spironolactone, and dapagliflozin, which was initiated in May 2024. Prior to admission, his serum sodium levels ranged from 130–135

mmol/L. During his current hospitalization, he was treated for pneumonia and incidentally noted to be hyponatremic with a sodium level of 128 mmol/L. At this point, diuretics and dapagliflozin were withheld. He responded to fluid boluses given, showing an initial improvement in his serum sodium, which then plateaued, followed by a declining trend to a nadir of 115 mmol/L. Paired serum and urine samples sent were consistent with SIADH. Hormonal workup taken showed normal thyroid and cortisol level. The patient was then given hypertonic saline to correct the initial severe hyponatremia, followed by fluid restriction and oral salt. Despite an initial improvement, this effect was not sustained, with sodium levels remaining static at 125–126 mmol/L. Dapagliflozin was then reintroduced, resulting in progressive improvement in his serum sodium, which allowed for discontinuation of oral sodium supplementation. He showed progressive clinical improvement and was discharged well with a serum sodium of 138 mmol/L.

CONCLUSION

This case illustrates the potential benefit of SGLT2 inhibitors in managing SIADH-related hyponatremia. Reintroduction of dapagliflozin led to a sustained rise in sodium levels, even after discontinuing salt supplementation. SGLT2i may enhance free water clearance and could be considered as adjunctive therapy in chronic SIADH, alongside fluid restriction and sodium supplementation.

EP_A153

UNMASKING A HORMONAL CHAMELEON: TSHoma WITH HIDDEN ACTH CO-SECRETION

<https://doi.org/10.15605/jafes.040.S1.161>

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INTRODUCTION/BACKGROUND

TSH-secreting pituitary adenomas (TSHomas) are rare and often misdiagnosed due to overlapping features with primary thyroid disorders. Even rarer are plurihormonal pituitary adenomas that co-secrete TSH and ACTH. We report a unique case where initial evaluation suggested a TSHoma, with ACTH co-secretion only suspected perioperatively based on clinical features and was later confirmed histologically.

CASE

A 41-year-old woman with a two-year history of hypertension and primary infertility presented with palpitations, heat intolerance, and insomnia. She had a history of

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menstrual irregularities, progressing to amenorrhoea after right oophorectomy. Thyroid function tests (TFT) revealed mildly elevated FT4 (22.5 pmol/L) with normal TSH (1.37 mIU/L), prompting a diagnosis of thyrotoxicosis and treatment with carbimazole was started.

Additional hormonal assessment revealed hyperprolactinemia (2138 mIU/L), hypogonadotropic hypogonadism and morning cortisol was 528 nmol/L. Pituitary MRI showed a 1.5 × 1.8 × 1.9 cm sellar-suprasellar mass compressing the optic chiasm.

The discordant TFT in the presence of a sellar lesion raised suspicion for TSHoma, although SHBG was normal 41.9 nmol/L (ref: 16.8-125.2 nmol/L). She was referred for surgery. Perioperative examination revealed Cushingoid features – facial hirsutism, centripetal obesity, and dorsocervical fat pad. ACTH co-secretion was suspected. She underwent endoscopic transsphenoidal resection, during which a fungal ball was incidentally discovered in the sphenoid sinus and was managed accordingly.

Postoperatively, the patient developed adrenal insufficiency with hypotension (random cortisol 24 nmol/L) requiring hydrocortisone. Histopathology confirmed a pituitary neuroendocrine tumor positive for both TSH and ACTH on immunostaining, alongside synaptophysin and chromogranin positivity, with a low Ki-67 index (1%). Postoperative thyroid and prolactin levels normalized.

CONCLUSION

This case highlights the diagnostic complexity of plurihormonal pituitary tumors. Although initially suspected to be a TSHoma based on discordant TFT, perioperative recognition of Cushingoid features led to the diagnosis of ACTH co-secretion confirmed via immunostaining. Careful clinical evaluation and histological confirmation are critical in such rare presentations.

EP_A154

A CASE SERIES OF DRUG-INDUCED THYROIDITIS

<https://doi.org/10.15605/jafes.040.S1.162>

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INTRODUCTION/BACKGROUND

Drug-induced thyroiditis is a relatively rare condition which is characterised by the inflammation of thyroid gland

after exposure to certain medications, with contrast agents and amiodarone being our main focus in this case series.

CASE

Case 1. A 40-year-old female with temporal bone squamous cell carcinoma and no prior thyroid disorder was undergoing radiotherapy and cisplatin-based chemotherapy and lost 5kg within a month. Clinically, the patient was euthyroid. Initial thyroid function test (TFT) showed TSH <0.01 mIU/L (0.27-4.20) and fT4 35 pmol/L (12-22). Carbimazole 20 mg OD and propranolol 20 mg BD were started. Despite optimising carbimazole dose to 40 mg OD, repeated TFT after 10 days showed TSH <0.01 mIU/L, fT3 8.6 pmol/L (3.1-6.8), fT4 62 pmol/L. Anti-TSH Receptor and anti-TPO antibodies were negative. Thyroid ultrasonography showed bilateral spongiform thyroid nodules (TR1). With a history of CT-simulation radiotherapy with 21000 mg iodine-based contrast given 1 month prior, a diagnosis of contrast-induced thyroiditis was made. Prednisolone 40 mg OD (1 mg/kg) was initiated while carbimazole was tapered off over a month. Patient became biochemically euthyroid after three months of corticosteroids.

Case 2. A 75-year-old man with no prior thyroid disorder and a recent history of coronary angiography presented with multiple episodes of ventricular tachycardia, requiring repeated synchronised cardioversion and multiple boluses of IV amiodarone 150 mg. Patient had palpitations but no signs of hyperthyroidism. TFT revealed TSH 0.1 mIU/L, fT3 5.5 pmol/L, fT4 33 pmol/L. Carbimazole 20 mg OD was started. Anti-TSH receptor and anti-TPO antibodies were negative. Diagnosis of type 2 amiodarone-induced thyroiditis was made; thus the patient was started on prednisolone 25mg OD (0.5 mg/kg). Carbimazole was subsequently stopped, while prednisolone was gradually tapered off. Patients became biochemically euthyroid after one month of corticosteroids.

CONCLUSION

These cases are two types of drug-induced thyroiditis—contrast-induced thyroiditis and type 2 amiodarone-induced thyroiditis. Both cases showed hyperthyroidism biochemically but were clinically asymptomatic. It is crucial to make an accurate diagnosis to ensure appropriate treatment. Steroids played a major role in the treatment, while antithyroid drugs are less effective.

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EP_A155

VENTRICULAR ARRHYTHMIA POST I-131-METAIODOBENZYLGUANIDINE (MIBG) THERAPY IN AN INOPERABLE RIGHT RETROPERITONEAL PARAGANGLIOMA

<https://doi.org/10.15605/jafes.040.S1.163>

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INTRODUCTION/BACKGROUND

Paraganglioma is a rare neuroendocrine tumour arising from extra-adrenal paraganglia. First line treatment involves surgical resection; however, a proportion of patients present with unresectable tumour. (131)I-MIBG therapy has emerged as a systemic treatment option for inoperable disease. Though rare, MIBG therapy can potentially cause ventricular arrhythmia due to catecholamine surge via tumour cell lysis. This case describes a patient presenting 3 weeks after therapy with ventricular tachycardia.

CASE

A 49-year-old male with underlying hypertension, diabetes mellitus, ischaemic heart disease post-angioplasty and chronic kidney disease was diagnosed with retroperitoneal paraganglioma when he presented with abdominal mass with elevated urine normetanephrine and 3-methoxytyramine. CECT Abdomen revealed a large lesion arising from inferior vena cava (IVC) measuring 9.3 x 9.0 x 13 cm. Biopsy was consistent with moderately differentiated paraganglioma with functional scan evidence of MIBG-avid disease. Multidisciplinary team meeting deemed the tumour to be inoperable in view of huge tumour with high vascular risk, while chemotherapy with temozolomide was not suitable in view of comorbidities. Consensus opted for 131-Metaiodobenzylguanidine (MIBG) therapy. A baseline echocardiography showed good left ventricular systolic function. He received one dose of 200 mCi of I-131 MIBG and was stable throughout admission. However, the patient later presented on day 21 post-therapy with chest pain and palpitation with a heart rate of 225 beats per minute. ECG showed monomorphic ventricular tachycardia which reverted to sinus rhythm with intravenous infusion of amiodarone. A subsequent cardiac MRI reported normal left ventricular function and ejection fraction with no features of myocardial infarction or infiltration.

CONCLUSION

This case highlights a rare but significant complication of I-131 MIBG therapy in the form of ventricular arrhythmia, likely triggered by catecholamine release from tumour lysis

manifesting three weeks post therapy. Close cardiac monitoring is essential especially in patients with pre-existing cardiovascular comorbidities undergoing MIBG treatment.

EP_A156

PARATHYROID ADENOMA WITH PATHOLOGICAL FRACTURE IN YOUNG ADULT: A CASE REPORT

<https://doi.org/10.15605/jafes.040.S1.164>

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INTRODUCTION/BACKGROUND

Primary hyperparathyroidism is a condition of hypercalcemia caused by an increase in parathyroid hormone levels. The most common cause of primary hyperparathyroidism is parathyroid adenoma (85%). Primary hyperparathyroidism often goes undiagnosed, particularly in fracture cases, which can lead to complications.

CASE

A 24-year-old female presented to the endocrinology clinic with complaints of worsening back pain and inability to walk over the past three months. The patient had a history of a fall six months prior and was treated in the orthopedic department. MRI of the lumbosacral region revealed multiple thoracolumbar vertebral fractures suspected to be due to osteoporosis. Laboratory tests indicated hypercalcemia (11.4 mg/dl) and hyperparathyroidism (PTH 791 ng/dl). Neck MRI suggested the presence of a left parathyroid adenoma. The patient was diagnosed with primary hyperparathyroidism due to a parathyroid adenoma and underwent parathyroidectomy. Two months after the procedure, her back pain was relieved and daily activity returned to normal. Laboratory test showed the improvement of serum calcium level (8.7 mg/dl).

CONCLUSION

The presence of a pathological fracture in a young adult must raise suspicion for a possible parathyroid adenoma. The diagnosis of primary hyperparathyroidism is established based on clinical findings and laboratory tests of serum calcium and parathyroid hormone levels. Early diagnosis and definitive management of parathyroidectomy are required in patients with primary hyperparathyroidism to reduce complications and improve quality of life.

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EP_A157

OSTEOPOROSIS IN ACROMEGALY: A PARADOXICAL COMPLICATION WITH MULTIFACTORIAL MECHANISMS

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INTRODUCTION/BACKGROUND

Acromegaly results from prolonged exposure to elevated levels of growth hormone (GH) and insulin-like growth factor-1 (IGF-1), which contribute to increased bone turnover. Despite IGF-1's known anabolic effects on bone, patients with acromegaly paradoxically face a higher risk of developing osteoporosis and vertebral fractures. This case series highlights the significance of early evaluation of bone health in managing acromegaly.

CASE

We evaluated four patients with confirmed acromegaly—three females and one male—ranging in age from 27 to 56 years, who underwent bone mineral density (BMD) testing via dual-energy X-ray absorptiometry (DXA). Half of the patients were diagnosed with osteoporosis, one had osteopenia, and one had normal BMD with borderline values.

A 27-year-old male, diagnosed with acromegaly at the age of 13, exhibited severe osteoporosis with lumbar Z-score -4.3, hip Z-score -3.6, and radius Z-score -3.3, and also exhibited panhypopituitarism and skeletal deformities. A 56-year-old postmenopausal female, diagnosed at 41 years, had osteoporosis with spinal T-score -2.7 and radius T-score -2.9. A 34-year-old female, diagnosed at age 29 and with secondary amenorrhea, had osteopenia (radius Z-score -2.2) despite near-normal lumbar and hip values. A 34-year-old male, diagnosed at 33 with hypogonadotropic hypogonadism, had overall normal BMD, though his radius showed a borderline Z-score of -0.5. Longer disease duration and hormonal deficiencies appeared to correlate with lower BMD, especially in trabecular-rich regions.

While GH and IGF-1 stimulate bone formation, chronic excess may disrupt bone remodeling balance, leading to

increased resorption, deterioration of trabecular structure, and higher cortical porosity. Local IGF-1 resistance and hypogonadism further impair bone integrity. These changes contribute to bone fragility even when BMD appears normal, suggesting that skeletal damage may precede densitometric findings.

CONCLUSION

Osteoporosis is a frequent but underrecognized complication in acromegaly. Bone fragility may develop early due to increased bone turnover, trabecular deterioration, and hypogonadism—despite normal or elevated BMD.

EP_A158

UNVEILING THE UNEXPECTED: A RARE PARAOVARIAN PARAGANGLIOMA MASQUERADING AS AN ADNEXAL MASS

<https://doi.org/10.15605/jafes.040.S1.166>

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INTRODUCTION/BACKGROUND

Paragangliomas are rare neuroendocrine tumors that arise from extra-adrenal paraganglionic tissue, typically associated with the autonomic nervous system. While they are most commonly found in the adrenal medulla (as pheochromocytomas) or along the sympathetic and parasympathetic chains, their occurrence in the paraovarian region is extremely rare.

CASE

We present the case of a 42-year-old female with a history of left external iliac and common femoral vein thrombosis, who presented with progressive abdominal distension over the past six months. She was normotensive and exhibited no constitutional symptoms, features of catecholamine excess, or compressive symptoms. Blood investigations revealed an elevated CA125 level while other tumor markers were within the normal range. A Computed Tomography (CT) scan of the thorax, abdomen, and pelvis revealed a large intra-abdominal cystic mass measuring 15.2 × 20.8 × 24.5 cm, likely originating from the left ovary with significant mass effect.

The patient underwent an extrafascial hysterectomy with bilateral salpingo-oophorectomy (EHBSO), left pelvic lymph node dissection (PLND), omentectomy, appendicectomy, and adhesiolysis. Histopathological examination of the left

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ovarian tumor and fallopian tube favored a diagnosis of extra-adrenal paraganglioma as the immunohistochemical staining was positive for S-100, synaptophysin, and chromogranin. Retrospectively, the tumor was likely a non-functioning paraganglioma, as the patient underwent surgery without complications.

CONCLUSION

Paraovarian paraganglioma is an exceptionally rare entity that presents significant diagnostic challenges due to its atypical location and non-specific clinical features. This case highlights the importance of considering paraganglioma in the differential diagnosis of adnexal masses as the perioperative management may differ.

EP_A159

46,XY DSD WITH RETAINED MÜLLERIAN STRUCTURES AND GENDER TRANSITION IN ADULthood: A STEPWISE DIAGNOSTIC APPROACH

<https://doi.org/10.15605/jafes.040.S1.167>

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INTRODUCTION/BACKGROUND

Disorders of Sex Development (DSD) are congenital conditions marked by atypical chromosomal, gonadal, or anatomical sex development. A structured diagnostic approach—starting from phenotype assessment through to chromosomal and molecular studies—is essential, particularly in 46,XY DSD where clinical presentations may vary widely. This report discusses a young adult with delayed-diagnosed 46,XY DSD who transitioned to male gender, analyzed through a stepwise framework.

CASE

A 20-year-old individual, assigned female at birth, presented with progressive virilization since early adolescence. The patient had no breast development or menstrual history. Instead, a deepened voice, facial and body hair, spontaneous erections, and wet dreams were reported. The patient urinated from an orifice beneath the clitoral area in a squatting position.

Physical examination revealed masculine features, gynecomastia, and clitoromegaly measuring approximately 3 cm in length. A bifid scrotum resembling labia majora was observed, with no palpable testes. Tanner staging was M2P4.

Hormonal analysis showed hypergonadotropic hypogonadism: LH 29.7 mIU/mL, FSH 47.8 mIU/mL, testosterone 21.1 nmol/L, estradiol 27.5 pmol/L. Karyotyping confirmed a 46,XY complement. MRI revealed bilateral gonads in the inguinal canals suspected as testes, and a uterine-like structure between the bladder and rectum. No ovaries or prostate were identified. FISH and SRY gene sequencing were performed; SRY was positive, and no pathogenic variants were found.

The presence of virilized phenotype, retained Müllerian structures, and undescended testes in a 46,XY individual suggests a disorder in androgen action or synthesis. While partial androgen insensitivity syndrome (PAIS) or 5 α -reductase deficiency are possible, definitive diagnosis awaits further molecular studies such as SRD5A2 or AR gene sequencing.

CONCLUSION

This case illustrates the complexity of evaluating 46,XY DSD and emphasizes the utility of a stepwise diagnostic algorithm. Clinicians should remain vigilant to consider rare etiologies in late-presenting cases and provide multidisciplinary, gender-affirming care tailored to the patient's identity and needs.

EP_A160

REFINING THE DIAGNOSIS: A CASE REPORT ON THE ROLE OF FISH IN DETECTING SUBTLE MOSAIC KLINEFELTER SYNDROME

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INTRODUCTION/BACKGROUND

Mosaic forms of Klinefelter syndrome (KS) can pose a diagnostic challenge, particularly in patients with a normal male phenotype and unremarkable hormonal profiles. While conventional karyotyping is a widely used first-line tool for detecting chromosomal abnormalities, its sensitivity

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is limited in identifying low-level mosaicism, which may result in false-negative or misleading interpretations. Fluorescence in situ hybridization (FISH), offering higher resolution and the ability to analyze hundreds of interphase nuclei, can uncover subtle chromosomal abnormalities that significantly impact clinical decision-making.

CASE

A 34-year-old male presented with concerns about delayed pubertal development, minimal axillary and facial hair, and poor muscularity despite strength training. He reported onset of wet dreams at 16 years, with gradual deepening of the voice and scant body hair. Physical examination revealed a tall stature (187 cm), soft body habitus, mild gynecomastia, and bilaterally normal-sized testes (20 mL). Hormonal assays showed normal levels of testosterone (22.58 nmol/L), LH (3.6 mIU/mL), and FSH (4.0 mIU/mL). Initial chromosomal analysis revealed a mosaic 45,X[1]/46,XY[99] pattern, suggesting a potential diagnosis within the chromosomal disorders of sex development (DSD) spectrum. However, the discrepancy between the karyotype and the patient's unequivocally male phenotype prompted further investigation using FISH. Analysis of 300 nuclei revealed 6% 47,XXY cells and 94% 46,XY cells, confirming a diagnosis of mosaic Klinefelter syndrome. Semen analysis demonstrated severe oligoasthenoteratozoospermia with only 1% morphologically normal sperm.

CONCLUSION

This case illustrates the limitations of conventional karyotyping in detecting low-level mosaicism and underscores the diagnostic value of FISH in cases where clinical findings and cytogenetic results appear discordant. By providing higher sensitivity, FISH can uncover clinically significant mosaic patterns, facilitating accurate classification within the DSD spectrum and informing appropriate counseling and reproductive planning.

EP_A161

FUNGAL SHADOWS: DIAGNOSTIC AND MANAGEMENT CHALLENGES OF ADRENAL HISTOPLASMOSIS IN AN IMMUNOCOMPETENT ADULT

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INTRODUCTION/BACKGROUND

Histoplasmosis is a fungal infection caused by *Histoplasma capsulatum*. Disseminated histoplasmosis involving bilateral adrenal glands and resulting in adrenal insufficiency is rare, particularly in immunocompetent individuals. The non-specific symptoms often mimic other diseases, making timely diagnosis difficult, especially in resource-limited settings. We report a case of disseminated histoplasmosis with adrenal insufficiency in an immunocompetent individual presenting with bilateral adrenal masses.

CASE

A 69-year-old previously healthy male presented with generalized body weakness, intermittent fever and significant weight loss of 20 kg over three months. There were no other remarkable symptoms. Initial PET-CT scan revealed large bilateral adrenal masses with hypermetabolic rims and central metabolism (right: 8.3 x 6.6 x 8.8 cm; left: 8.6 x 6.6 x 9.7 cm) as well as hepatosplenomegaly and right lung changes suggestive of infection or malignancy. The investigations for tuberculosis and HIV were negative. Tissue biopsies of the masses revealed acute granulomatous lesions indicative of fungal infection. The patient was started on intravenous Amphotericin B, followed by maintenance therapy with oral itraconazole. An ACTH stimulation test showed inadequate adrenal response, and steroid replacement therapy was initiated. Despite nine months of antifungal therapy, he showed minimal clinical improvement. Repeat imaging demonstrated increased adrenal mass size, prompting bilateral adrenal drainage following a multidisciplinary team discussion. Histopathology confirmed ongoing fungal infection, and fungal sequencing identified *Histoplasma capsulatum*.

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A second course of IV liposomal Amphotericin B was administered, followed by itraconazole. The patient's fever resolved, and follow-up imaging showed reduction in adrenal mass size. He remains on drainage and long-term antifungal therapy, planned for at least 18 months.

CONCLUSION

This case highlights the diagnostic challenges of adrenal histoplasmosis in immunocompetent individuals presenting with vague systemic symptoms and large bilateral adrenal masses. Early recognition and a multidisciplinary approach are crucial for timely diagnosis and optimal management.

EP_A162

BEYOND THE YELLOW: UNMASKING PHEOCHROMOCYTOMA IN A JAUNDICED PATIENT

<https://doi.org/10.15605/jafes.040.S1.170>

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INTRODUCTION/BACKGROUND

Pheochromocytomas and paragangliomas (PPGLs) are rare neuroendocrine tumours (NET) arising from chromaffin cells. Bilateral pheochromocytomas are extremely rare, constituting 7–10% of all pheochromocytoma cases, and 60%–90% of them possess a germline mutation.

CASE

A 17-year-old male presented initially with epigastric pain and obstructive jaundice. He is not hypertensive. ERCP revealed choledocholithiasis and a dilated common bile duct (CBD). A contrast-enhanced CT of the liver showed an enhancing CBD lesion causing biliary obstruction and incidental bilateral adrenal tumours. A CT Adrenal protocol confirmed a left adrenal lesion measuring 5.4×4.8×5.1cm with unenhanced attenuation 35.4 Hounsfield Units (HU) and a right adrenal lesion measuring 1.2 × 1.1 × 1.6 cm with unenhanced attenuation 30.7HU, both with delayed contrast washout, consistent with pheochromocytomas. Biochemical evaluation showed elevated 24-hour urinary normetanephrine at 18,558 nmol/24h (497–2489), which is seven times the upper limit of normal, confirming catecholamine excess with normal levels of Metanephrine. Other hormonal investigations were unremarkable.

He underwent open cholecystectomy and choledochectomy with biliary reconstruction. Histopathology confirmed a well-differentiated Grade I neuroendocrine tumour (NET) of the CBD, positive for synaptophysin, chromogranin A, and CD56, with a Ki-67 <3%. Surgical margins and lymph nodes were negative. Thyroid ultrasound was normal. ⁶⁸Ga-DOTATATE Positron Emission Tomography (PET) confirmed bilateral pheochromocytomas with no extra-adrenal paraganglioma or metastatic disease. He underwent bilateral adrenalectomy after adequate alpha-blockade and was discharged well with hydrocortisone and fludrocortisone replacement. He is awaiting genetic testing.

CONCLUSION

This case highlights a rare pheochromocytoma with obstructive jaundice, lacking the classical triad of headache, palpitations, and sweating. Bilateral pheochromocytomas are commonly seen in Multiple Endocrine Neoplasia types 2A and 2B, von Hippel–Lindau disease, and rarely with MAX and TMEM127 mutations, though they can also occur sporadically. Genetic testing is crucial for diagnosing and managing bilateral pheochromocytoma, as it aids in treatment decisions, recurrence prediction, and family screening.

EP_A163

TREACHEROUS JOURNEY OF ADVANCED PAPILLARY THYROID CARCINOMA IN PREGNANCY

<https://doi.org/10.15605/jafes.040.S1.171>

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INTRODUCTION

Papillary thyroid carcinoma (PTC) is the most common thyroid malignancy and generally exhibits a favourable prognosis, but it can manifest with metastasis in advanced stages. Pregnancy complicates the management of such cases particularly when radioactive iodine (I-131) therapy is indicated.

CASE

A 26-year-old presented in her second trimester with acute exacerbation of bronchial asthma requiring mechanical ventilation. During intubation, a 6 × 4 cm anterior neck swelling was found. A computed tomography showed diffuse heterogeneous thyroid enlargement with tracheal narrowing, cervical lymphadenopathy, and pulmonary

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metastasis. Thyroid function tests were normal. She underwent debulking thyroidectomy with bilateral modified radical neck dissection. Patient required a tracheostomy due to tumour invasion into the trachea. Histopathology confirmed multifocal (>5 foci) classical variant PTC with the largest nodule measuring 25 mm. The tumour showed lymphovascular invasion, regional nodal metastases, and invasion into adjacent skeletal muscle indicating an advanced stage with a high risk of recurrence as per the American Thyroid Association (ATA) risk stratification.

Postoperatively, we started her on levothyroxine with a TSH target of below 0.1mIU/L. At 33 weeks gestation, an elective lower segment caesarean section was performed. Post delivery, cabergoline was given to suppress lactation in preparation for I-131 therapy. After consultation with nuclear medicine, I-131 therapy was scheduled at 10 weeks postpartum. Levothyroxine was withheld one month prior.

CONCLUSION

This case highlights the challenges of managing advanced PTC with metastasis during pregnancy. Thorough multidisciplinary planning of surgery and postpartum I-131 timing is essential to ensure a seamless delivery and safety of mother and child. To safeguard breast tissue from radiation exposure, breastfeeding should be entirely discontinued at least six weeks prior to I-131 therapy. Breastfeeding should not be resumed after I-131 administration to shield the infant from radiation exposure and avert harm to the infant's thyroid gland. Breastfeeding is not contraindicated in subsequent pregnancies.

EP_A164

TEMOZOLOMIDE THERAPY IN RECURRENT METASTATIC PHEOCHROMOCYTOMA: A CASE-BASED REVIEW

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INTRODUCTION

Metastatic pheochromocytoma is rare and the management is complex, requiring multifaceted, multidisciplinary management. Primarily palliative, treatment focuses on tumor control, symptom management, and quality of life. While historically associated with a poor prognosis, improved diagnosis and management, including surgery, chemotherapy and targeted therapies, are extending survival for some patients.

CASE

A 52-year-old female was initially diagnosed in 2013 with non-functioning pheochromocytoma with liver, spleen, and pancreatic tail metastases. She underwent left adrenalectomy, splenectomy, distal pancreatectomy, and local resection of metastatic liver lesions, followed by trans-arterial chemoembolization of liver metastases. Subsequent follow-up imaging revealed recurrent disease, necessitating further surgical intervention. This included left hemihepatectomy, left nephrectomy, segmental resection of the colon and splenic flexure and excision of a posterior abdominal tumor. Due to the extensive nature of her disease progression, the patient received 4 cycles of peptide receptor radionuclide therapy (PRRT) as well as palliative radiotherapy to left thoracoabdominal mass and T9 till L1 vertebrae. Despite undergoing PRRT, the disease continued to progress. A multidisciplinary team discussion led to the initiation of temozolomide treatment in March 2023. The patient has received 22 cycles of temozolomide from 2023 to date, with recent follow-up imaging demonstrating partial response to the treatment.

CONCLUSION

This case report illustrates therapeutic efficacy of temozolomide in metastatic pheochromocytoma. In recent years, temozolomide has shown good outcomes in some metastatic pheochromocytoma patients, especially those with SDHB germline mutation. Temozolomide treatment has been generally considered to have a low toxicity profile, however few studies have noted the development of severe myelosuppression. While the current evidence base is still developing and primarily relies on retrospective data and case reports, ongoing clinical trials are anticipated to yield more definitive conclusions regarding its efficacy and optimal clinical application in metastatic pheochromocytoma.

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EP_A165

THE VOICE WITHIN: ADULT LARYNGOMALACIA AS A RARE COMPLICATION OF ACROMEGALY

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INTRODUCTION/BACKGROUND

Acromegaly is a chronic disorder caused by excess growth hormone (GH) and insulin-like growth factor 1 (IGF-1), most often due to a GH-secreting pituitary adenoma. Adult-onset laryngomalacia is rarely reported.

CASE

A 69-year-old male with a history of hyperthyroidism and colon cancer presented with progressive left eye blurring. He was initially treated for herpetic keratouveitis. During follow-up, coarse facial features suggestive of acromegaly—thickened skin, enlarged jaw, tongue, hands, and feet—were noted. He had noisy breathing, prompting ENT referral. Flexible laryngoscopy revealed redundant mucosa over the arytenoids prolapsing into the laryngeal inlet during inspiration, consistent with adult-onset laryngomalacia. Biochemical evaluation confirmed acromegaly (GH >50 ng/mL; IGF-1: 973.5 ng/mL) with secondary hypogonadism. MRI showed a 1.6 × 2.2 × 1.6 cm pituitary macroadenoma compressing the left optic nerve. He was started on intramuscular Octreotide LAR and underwent supraglottoplasty.

Laryngomalacia is typically a pediatric condition caused by dynamic supraglottic collapse during inspiration. In adults, it is uncommon and may result from structural abnormalities or acquired soft tissue redundancy, as seen in acromegaly. Chronic GH and IGF-1 excess leads to hypertrophy of soft tissues, including the larynx, epiglottis, aryepiglottic folds, and arytenoids, contributing to narrowing of the upper airway. Awake fiberoptic laryngoscopy is the diagnostic gold standard. Findings include inspiratory collapse of supraglottic structures, which may cause stridor, dysphonia, or sleep-disordered breathing. In acromegaly, cartilage overgrowth and mucosal thickening reduce airway diameter and alter tissue compliance. The hyoepiglottic ligament may also lose tensile strength, further predisposing to dynamic airway obstruction. Laryngomalacia may be misdiagnosed or attributed to obstructive sleep apnea, a

common comorbidity in acromegaly and distinct anatomical distortion should prompt ENT evaluation.

CONCLUSION

Laryngomalacia should be considered in acromegalic patients presenting with stridor or noisy breathing. Early recognition and surgical management can prevent airway complications and improve patient outcomes.

EP_A166

HYPERTHYROIDISM MASQUERADING AS ACUTE MYOCARDIAL INFARCTION

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INTRODUCTION/BACKGROUND

Troponin-Positive Non-Obstructive Coronary Arteries (TpNOCA) are conditions characterized by elevated troponin levels accompanied by absent obstructive coronary artery disease (CAD) as observed on coronary angiography. It encompasses both coronary and noncoronary causes of myocardial injury.

CASE

A 31-year-old female with no known medical illness presented with fever, vomiting and diarrhea for 4 days. She did not have any features or family history of Graves' Disease. She had no goiter and denied any biotin supplements or illicit drugs. On arrival she had a fever of 38.5°C, palpitations with a pulse ranging between 110-130 beats/min, and a Blood Pressure of 89/47. She was intubated due to impending respiratory distress. Initial Electrocardiogram (ECG) done showed Atrial Fibrillation (AF). Repeated ECG showed ST-segment Elevation over the Lateral leads with reciprocal ST depression. High-Sensitivity Troponin-T taken on arrival was markedly raised at 957 ng/L, and repeated 2 hours later was 3089 ng/L. Patient was rushed for an emergency angiogram by the cardiology team which revealed unobstructed coronaries. Echocardiography performed was normal.

A thyroid function test (TFT) taken due to AF revealed Free T4 of 33 pmol/L with a suppressed TSH of 0.02 mIU/L. Alanine Aminotransferase (ALT) taken was 383 U/L attributable to ischemic hepatitis. Patient was commenced on carbimazole 10 mg daily with careful daily ALT monitoring. Lugol's Iodine 10 drops TDS and IV hydrocortisone 100 mg TDS were given for 5 days. Patient eventually improved with normalization of TFT and liver profile with tapering

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carbimazole dose. TSH-receptor Antibodies (TRAb) taken was negative and we referred this patient for an outpatient Thyroid Ultrasonography to rule out toxic adenoma.

CONCLUSION

TpNOCA may be induced by hyperthyroidism due to heightened oxygen demand and coronary vasospasm leading to Type-2 Myocardial Infarction in the presence of unobstructed coronary arteries. Prompt identification and management of hyperthyroidism is crucial to avert severe complications and ensuring a favourable outcome.

EP_A167

A CASE OF HEART FAILURE UNVEILING HIDDEN ACROMEGALY

<https://doi.org/10.15605/jafes.040.S1.175>

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INTRODUCTION/BACKGROUND

Acromegaly is a rare disease caused by hypersecretion of growth hormone. Cardiovascular disease is the most common comorbidity in acromegaly and constitutes a leading cause of mortality. However, there is currently limited direct literature addressing heart failure with preserved ejection fraction (HFpEF) in acromegaly. We present a case of acromegaly presenting with heart failure.

CASE

At a district hospital in Kedah, a 46-year-old female with a known case of hypertension since the age of 23 years old presented with dyspnoea on exertion, orthopnoea, and bilateral leg swelling. She had significant weight gain following her hypertension diagnosis. Her physical examination showed a weight of 121 kg, height of 1.75 m, and body mass index of 46.7 kg/m². Her blood pressure was 141/89 mm Hg with a heart rate of 90 beats/min. Lung examinations revealed coarse crepitations with bilateral pitting oedema. A comprehensive physical examination revealed spade-like hands and feet, prominent supraorbital ridges, widening of teeth spaces with thick lips, and an enlarged nose. Given the characteristic clinical findings, we suspected the provisional diagnosis of acromegaly. Chest radiography showed cardiomegaly with congestive features. Echocardiogram revealed an ejection fraction of 57%, mildly dilated left atrium with grade 1 diastolic dysfunction which is consistent with HFpEF. Laboratory workup showed elevated insulin-like growth factor 1 level of 278.4ng/ml (normal 56.8-194.5 ng/ml). Subsequently, she

was referred to an endocrinologist in a tertiary centre for further investigation and treatment.

CONCLUSION

This case highlights the critical importance in recognizing acromegaly as a rare underlying cause of cardiac manifestations. The clinical suspicion based on physical examination can facilitate prompt diagnosis to prevent early cardiovascular death in acromegaly patients. Clinicians should maintain a high index of suspicion for endocrine disorders that may present with cardiovascular manifestations.

EP_A168

A RARE PRESENTATION OF MEDULLARY THYROID CARCINOMA: A CASE REPORT

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INTRODUCTION/BACKGROUND

Medullary thyroid carcinoma (MTC) is a rare neuro-endocrine tumour arising from the parafollicular C cells of the thyroid gland, accounting for approximately 4% of all thyroid malignancies. We present a case of MTC with an unusual and life-threatening initial manifestation — cardiac tamponade — which led to the diagnosis.

CASE

A 63-year-old Kadazan male with a medical history of myocardial infarction with non-obstructive coronary arteries (MINOCA) in 2017, intracranial haemorrhage in 2018, polycythaemia rubra vera, dyslipidaemia, hypertension, and type 2 diabetes mellitus, presented with a three-day history of exertional dyspnoea and chest tightness. He also reported a gradual neck swelling and unintentional weight loss over the past year.

Initial chest radiography revealed a right lower zone lung opacity, and he was empirically treated for pneumonia. However, a neck ultrasound demonstrated a right thyroid nodule categorized as TIRADS 4, raising suspicion for malignancy. A contrast-enhanced CT (CECT) of the thorax revealed a suspicious right thyroid nodule with bilateral cervical, supraclavicular, and mediastinal lymphadenopathy, multiple pulmonary nodules, a segment VIII liver lesion, and a significant global pericardial effusion measuring 2.8 cm. Fine needle aspiration cytology (FNAC) of the right thyroid nodule and left cervical lymph node confirmed medullary thyroid carcinoma, with positive staining for calcitonin and amyloid deposits identified

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via Congo red staining. Transthoracic echocardiography showed right atrial and right ventricular collapse, consistent with cardiac tamponade. Emergency pericardiocentesis was performed, and cytology of the pericardial fluid confirmed metastatic MTC.

Further laboratory evaluation revealed markedly elevated serum calcitonin and carcinoembryonic antigen (CEA), along with raised urinary levels of normetanephrine, metanephrine, and 3-methoxytyramine, suggesting a paraneoplastic neuroendocrine profile. Germline RET mutation analysis could not be performed due to resource limitations.

Given the presence of distant metastases and extensive locoregional disease, the patient was scheduled for systemic therapy with Cabozantinib with plans for total thyroidectomy following tumour debulking.

CONCLUSION

This case highlights a rare and aggressive presentation of medullary thyroid carcinoma (MTC), manifesting as cardiac tamponade — a life-threatening complication seldom associated with thyroid malignancies. The diagnosis was confirmed through cytological evaluation and supported by elevated tumour markers and imaging. This case underscores the importance of considering metastatic MTC in patients with unexplained pericardial effusion and systemic symptoms, especially in the presence of a suspicious thyroid lesion. Prompt recognition and multidisciplinary management are crucial in optimizing outcomes in such advanced and atypical presentations.

EP_A169

ECTOPIC ACTH SYNDROME SECONDARY TO METASTATIC NEUROENDOCRINE CARCINOMA FROM A PRIMARY MEDIASTINAL TUMOUR

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INTRODUCTION/BACKGROUND

We report a case of ectopic ACTH syndrome secondary to metastatic neuroendocrine neoplasm of the anterior mediastinum.

CASE

A 26-year-old male was diagnosed at age 23 to have ectopic ACTH syndrome secondary to neuroendocrine tumour of mediastinum, size 7 x 6 cm. Gallium-68 DOTATATE PET-

CT revealed somatostatin receptor (SSTR) avid disease in mediastinum only, Krenning 3. He underwent surgical excision and achieved remission postoperatively. HPE reported ACTH-producing typical mediastinal carcinoid with nodal involvement, Ki67 ~ 10%, mitosis count 1 per 10 high power field, and metastatic typical carcinoid of the excised para-aortic lymph node. 6 months later, ACTH was noted to be increasing in trend although he was not Cushingoid clinically. FDG and Dotatate PET-CT scan revealed metastatic lymphadenopathy to the left supraclavicular fossa and mediastinum with low SSTR affinity (Krenning score 1 and 2). He was referred to the surgical and oncology team for further treatment. However, he opted for a second opinion in an overseas institution and started proton therapy and everolimus there, which was discontinued within weeks due to side effects.

He presented again a year later, not overtly Cushingoid, but he then developed more prominent Cushingoid signs and hypokalaemia within months. Biochemical investigation showed persistent disease with increasing ACTH. Ketoconazole was initiated. Dotatate and FDG PET-CT imaging revealed progressive metastatic lymphadenopathy involving cervical, supraclavicular, mediastinal and coeliac regions. The lesions had concordant FDG and Dotatate avidity but were more FDG-avid (Dotatate avidity Krenning 2). Multidisciplinary team discussion concluded a diagnosis of neuroendocrine carcinoma with progressive disease, thus requiring chemotherapy. He was referred to oncology team but remained undecided about proceeding further.

CONCLUSION

Neuroendocrine tumours can have heterogeneity in grade within a given lesion, in different sites, and over time. SSTR PET imaging aids in stratifying tumour differentiation thus guiding diagnostic and therapeutic decisions, as illustrated in this case.

EP_A170

THE MAN WITH MALIGNANT INSULINOMA: CHALLENGE IN MANAGEMENT

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INTRODUCTION/BACKGROUND

Insulinoma is an uncommon pancreatic neoplasm that results in excessive insulin production. Excessive insulin

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production and recurrent hypoglycemia render it a potentially lethal condition.

CASE

A 41-year-old male with comorbid hypertension and dyslipidaemia presented with right upper quadrant pain for two days, altered bowel habits, decreased appetite, and weight loss. He underwent assessment to rule out malignancy. During hospitalisation, he developed recurrent asymptomatic hypoglycaemia despite the administration of dextrose infusion. He was confirmed to have endogenous hyperinsulinism, evidenced by increased blood C-peptide levels of 689 pmol/L during a hypoglycaemic episode with plasma glucose 3.0 mmol/L. CECT of the abdomen and pelvis demonstrated rim-enhancing hypodense lesions in the liver, with the largest lesion measuring 8.1 x 7.5 x 8.8 cm (AP x W x CC). There was also a hypodense lesion in the body of pancreas (0.7 x 0.8 cm). The liver biopsy revealed poorly differentiated neuroendocrine carcinoma. He was co-managed by the surgical and oncology teams. The lesion was unresectable, and he was also not a suitable candidate for arterial embolisation. He was prescribed diazoxide tablets to alleviate hypoglycaemia. Despite the regular intake of diazoxide, his hypoglycaemia worsened, finally resulting in his death.

CONCLUSION

Insulinoma is a neoplasm with a 10% likelihood of malignancy. Individuals with insulinoma often present with Whipple's triad, characterised by hypoglycaemia symptoms, plasma glucose concentrations below 3.1 mmol/L, and symptom relief after ingestion of a high-glucose meal. Insulinoma is best treated with surgical excision. The main and challenging management issue in insulinoma is addressing recurrent hypoglycaemia in patients with unresectable tumours. Octreotide, a somatostatin analogue, is often used to reduce insulin production. Diazoxide, on the other hand, inhibits insulin secretion and stimulates glycogenolysis to enhance glucose release from the liver. In the end, a patient with malignant insulinoma may die from severe hypoglycaemia or metastatic malignancy.

EP_A171

ATYPICAL PARATHYROID TUMOR: CHALLENGES OF DIAGNOSIS AND MANAGEMENT

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INTRODUCTION/BACKGROUND

Differentiating between benign and malignant parathyroid nodules is a clinical and pathological challenge. Unlike other tumours, parathyroid carcinoma lacks definitive preoperative biomarkers. The diagnosis is frequently made retrospectively based on surgical pathology.

CASE

A 55-year-old female came to our hospital with progressive bilateral weakness of lower extremities 6 months ago, resulting in partial immobilization, bone pain, and contracture of lower extremity. Laboratory results showed high serum calcium (13.9 mg/dL) and intact PTH (2,729 pg/mL). Neck ultrasound revealed a left parathyroid nodule mass. The MIBI scan showed a negative result. Bone X-ray of vertebra and extremities showed multiple lytic lesions and osteopenia. She was treated with zoledronic acid and scheduled for subtotal parathyroidectomy. After parathyroid resection, iPTH levels decreased significantly. However, she had low calcium levels, indicating hungry bone syndrome, and we managed with oral and IV calcium to restore normal calcium levels. The histopathology of parathyroid nodules showed neoplasms of parathyroid origin that show atypical histologic features but without unequivocal capsular, vascular, or perineural invasion. During hospitalizations, she had a pathological closed fracture of the right tibia. The orthopedic surgeon placed an internal fixation in the right tibiae and did a bone biopsy, no malignant cells from bone histopathology. We diagnosed her as atypical parathyroid tumor (APT) with metabolic bone disease and gave her IV Denosumab. After routine physiotherapy, she could mobilize around the bed and was discharged eventually with normal calcium levels. We planned for serum calcium and PTH evaluation 3-6 months after surgery.

CONCLUSION

APT is a distinct and enigmatic entity between benign adenoma and malignant carcinoma of parathyroid tumour. These tumours exhibit some worrisome histologic features of malignancy but lack definitive evidence of parathyroid carcinoma. APT is usually indolent. The prognosis is

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generally favourable if the tumour is completely resected and closely monitored.

EP_A172

HIGH PREVALENCE OF PREDIABETES AND VITAMIN D DEFICIENCY IN IBB GOVERNORATE, YEMEN: A CROSS-SECTIONAL STUDY

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INTRODUCTION

Prediabetes and vitamin D deficiency are growing health concerns linked to diabetes progression and its complications, yet their prevalence and association remain underexplored, specifically in Yemen. This study aims to assess the prevalence of prediabetes and vitamin D deficiency among individuals in the Ibb Governorate, Republic of Yemen.

METHODOLOGY

A cross-sectional study involving 1046 participants who met study criteria recruited from various centres in the Ibb Governorate, Yemen, including Jiblah University for Medical and Health Sciences, Medical City Complex, and Al-Noor Hospital. They underwent HbA1c and level of vitamin D determination. Prediabetes was defined based on the American Diabetes Association, using HbA1c between 5.7% and 6.4%. Vitamin D levels of <20 ng/mL and 20-30 ng/mL are defined as deficiency and insufficiency, respectively.

RESULT

The prevalence of prediabetes was 25.7% (269). The mean HbA1c and age were 5.9 ± 0.2 % and 41.4 ± 10.2 years, respectively. From the prediabetes population, 71.4% (189) had vitamin D deficiency, while 20.7% (54) had vitamin D insufficiency, with a total of 93.1% (243) of prediabetes participants. The mean age for deficiency, insufficiency and sufficiency is 40.80 ± 9.9 ng/mL, 41.3 ± 10.0 ng/mL and 47.8 ± 9.0 ng/mL, respectively. The prevalence of low vitamin D levels (vitamin D deficiency and insufficiency) was slightly higher in males (126, 48.27%) compared to females (117, 44.83%). A significant age difference was observed for the sufficient group compared to both deficiency ($p = 0.01$) and insufficiency ($p = 0.04$) groups. The Chi-square test revealed a significant association between gender and the vitamin D status groups ($\chi^2 = 8.266$, $p = 0.01$).

CONCLUSION

The significant prevalence of prediabetes and vitamin D deficiency in the Ibb Governorate underscores the need for comprehensive interventions addressing both conditions. Correcting vitamin D deficiency may reduce the progression from prediabetes to diabetes, potentially improving metabolic health outcomes and mitigating associated complications.

EP_A173

INSULIN INITIATION IN T2DM: OUTCOMES ON GLYCAEMIC CONTROL, BODY WEIGHT, AND HYPOGLYCEMIA RISK

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INTRODUCTION

The prevalence of diabetes in Malaysia is rising due to urbanization and socioeconomic changes. Despite evidence supporting good glycaemic control, maintaining optimal control remains challenging, with mean HbA1c levels ranging from 7.9% to 8.1%. Progressive beta-cell failure leads to secondary OAD failure, necessitating insulin therapy. This study evaluates the effect of a simple insulin therapy protocol on glycaemic control in T2DM patients with secondary OAD failure.

METHODOLOGY

A prospective study was conducted in outpatient clinics at Hospital Kuala Lumpur and Hospital Serdang. Patients with T2DM and secondary OAD failure (HbA1c >9%, fasting plasma glucose ≥ 9 mmol/L) were recruited. The intervention

Adult E-Poster

group received bedtime NPH insulin (Insulatard® Penfill®) with dose titration, while the control group continued OAD therapy. HbA1c, fasting blood glucose (FBG), insulin dosage, and hypoglycaemia events were assessed at baseline, Month 3, and Month 6.

RESULT

Forty-five patients (mean age 55.1 ± 9.3 years, diabetes duration 9.7 ± 8.1 years) were included. At Month 6, HbA1c in the intervention group was significantly reduced by 1.28%, with 26.7% achieving HbA1c $<8\%$. Mean FBG decreased by 3.6 mmol/L. Insulin dose stabilized at 0.33 ± 0.13 IU/kg/day. Mean body weight change was +1.78 kg in the intervention group. One patient experienced mild hypoglycaemia.

CONCLUSION

A patient-guided insulin titration protocol improved glycaemic control in T2DM patients with secondary OAD failure, supporting its feasibility in local primary care settings.

EP_A174

A RANDOMIZED CONTROLLED TRIAL TO EVALUATE THE EFFECTS OF DIGITAL HEALTH INTERVENTIONS ON GLYCEMIC CONTROL FOR WOMEN REQUIRING INSULIN THERAPY DURING PREGNANCY

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INTRODUCTION

There are good evidences advocating the use of digital health intervention (DHI) to complement diabetes care, but this management approach remains under-utilized in our country.

METHODOLOGY

In this prospective interventional study, pregnant women with Type 2 diabetes (T2D) and a gestational age of less than 24 weeks were randomly assigned to either the DHI (intervention) or the usual care group (control). Participants

from the DHI group were enrolled in the online BioTective™ Disease Resource Center, a digital platform that included a Bluetooth-enabled glucometer connected to a smartphone application. The investigator reviewed the glucose data remotely and provided guidance on insulin titration. The primary outcome was to compare changes in glucose readings between the two groups at five time points. Pre-meal and post-meal glucose readings were analyzed at each time point, and changes in mean HbA1c levels before and after the study completion were also evaluated.

RESULT

The results presented are from an interim analysis. We have forty-two females randomized equally between the two groups. Most participants were Malay ($n = 30$, 71.4 %), with a mean age of 33 ± 34.9 years, and more than half were multigravida. Thirty-six participants (85.7 %) were receiving basal-bolus insulin therapy, and the mean HbA1c at recruitment was 7.3 ± 1.4 %. Throughout the study period, the intervention group could achieve pre-meal glucose readings below 5.3 mmol/L at all time points, while the control group had glucose readings above the target at 3 out of the 5 time points ($p = 0.565$). Both groups had post-meal glucose readings below 6.7 mmol/L at all time points ($p = 0.473$). The reduction in mean HbA1c was more pronounced in the intervention group; however, the difference compared to the control group was not statistically significant (intervention: 6.02 ± 0.67 % vs. control: 6.46 ± 0.88 %; $p = 0.774$).

CONCLUSION

Our interim analysis suggested DHI is comparable to the usual care in managing T2D women requiring insulin therapy during pregnancy.

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EP_A175

EXPLORING THE IMPACT OF INSULIN DEINTENSIFICATION ON BODY WEIGHT AND GLUCOSE CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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INTRODUCTION

Deintensification of insulin regimens and doses has potential to prevent overtreatment and hypoglycemia. This study aims to identify the characteristics and reasons for deintensification in Type 2 Diabetes Mellitus (T2DM) patients; to evaluate glycemic efficacy and to examine changes in body weight following deintensification and factors influencing these outcomes.

METHODOLOGY

A retrospective cohort observational study was conducted among T2DM patients from Hospital Tuanku Ja'afar and Hospital Canselor Tuanku Mukhriz. Data were collected from patient records receiving insulin deintensification from January 2020 to January 2024 using a data collection form with six sections.

RESULT

A total of 134 patients from two hospitals were included in this study, with 75 patients from HTJ and 59 patients from HCTM. The mean age was 57.25 ± 14.02 years, with an equal distribution of male and female participants. The majority were Malay ($n = 69$, 51.5%), followed by an equal number of Chinese and Indian ($n = 32$, 23.9% each), with most patients on a basal bolus regimen ($n = 69$, 51.5%), followed by a premixed ($n = 63$, 47%) and a basal ($n = 2$, 1.5%). The mean duration of diabetes was 17.54 ± 8.28 years. Baseline HbA1c was $9.38 \pm 1.86\%$ and most patients used insulin four times a day ($n = 59$, 44%). The mean total daily insulin dose decreased from 77.99 ± 30.18 units to 60.11 ± 25.39 units. Hypoglycemia events reduced from 98 to 11 episodes. The main reason for deintensification was hypoglycemic events ($n = 98$, 73.1%). HbA1c reduced from $9.38 \pm 1.86\%$ to $8.72 \pm$

1.78% ($t(133) = 5.57$, $p < 0.001$), and weight decreased from 77.27 ± 15.83 kg to 75.80 ± 15.75 kg ($t(133) = 6.19$, $p < 0.001$). Factors significantly associated with changes in HbA1c include baseline HbA1c ($p < 0.001$), use of basal-only insulin ($p = 0.002$), and reduction in insulin injection frequency by one ($p = 0.002$) and two ($p = 0.004$) times per day.

CONCLUSION

Insulin deintensification significantly improves glycemic control and reduces body weight in T2DM patients. Key factors influencing these improvements include baseline HbA1c levels and the type and frequency of insulin used. Monitoring for signs of overinsulinization and hypoglycemia, particularly those with high HbA1c, is crucial for optimizing diabetes management.

EP_A176

THE PREVALENCE OF COGNITIVE IMPAIRMENT AMONG ADULTS WITH TYPE 2 DIABETES MELLITUS: A MULTI-CENTER CROSS-SECTIONAL STUDY

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INTRODUCTION

Cognitive impairment is increasingly recognized as a significant complication of type 2 diabetes mellitus (T2DM), affecting memory, executive function, and processing speed. Despite its clinical relevance, cognitive impairment in T2DM often remains underdiagnosed, leading to poor disease management, reduced adherence to treatment, and diminished quality of life.

METHODOLOGY

This cross-sectional study evaluated the prevalence of cognitive impairment among T2DM patients attending the Medical Outpatient Department (MOPD) and Integrated Diabetes Clinic at Hospital Sultanah Bahiyah and Hospital Sultan Abdul Halim, Kedah. Eligible participants were adults with T2DM, selected through convenience sampling. Cognitive function was assessed using the Montreal Cognitive Assessment (MoCA), and demographic data were collected.

RESULT

A total of 144 participants were included, comprising 88 females (61.1%) and 56 males (38.9%). The median age of participants was 56 years old. The majority were of

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Malay ethnicity (n = 125, 86.8%), followed by Indian (n = 14, 9.7%), Chinese (n = 4, 2.8%), and Thai (n = 1, 0.7%). Most participants (n = 90, 62.5%) were from low-income backgrounds. Educational attainment was limited, with 16% having no formal education or only primary-level education. Cognitive function assessment revealed that only 49 participants (34%) had normal cognitive function, while 72 participants (50%) exhibited mild cognitive impairment. Moderate cognitive impairment was observed in 21 participants (14.6%), and severe cognitive impairment was identified in 2 participants (1.4%).

CONCLUSION

This study reveals a strikingly high prevalence of cognitive impairment among individuals with T2DM, underscoring an urgent need for early detection and proactive intervention. As cognitive decline directly influences disease self-management, medication adherence, and overall quality of life, its integration into routine diabetes care is imperative.

EP_A177

TREATMENT OF DYSLIPIDEMIA IN TYPE 2 DIABETES MELLITUS PATIENTS AT THE DIABETES CLINIC, HOSPITAL SULTAN HAJI AHMAD SHAH: A CLINICAL AUDIT

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INTRODUCTION

Dyslipidemia is a major risk factor for cardiovascular disease in patients with Type 2 Diabetes (T2D) and requires aggressive management. The aim of this clinical audit is to assess the appropriateness of dyslipidemia treatment in T2D patients attending the diabetes clinic at Hospital Sultan Haji Ahmad Shah, Temerloh, Pahang.

METHODOLOGY

All T2D patients attending the diabetes clinic from June to July 2024 were included in this clinical audit. Electronic medical records were reviewed for demographic data, comorbidities, lipid profiles, cardiovascular disease risk assessments, and statin prescription patterns.

RESULT

A total of 102 patients were included, with a mean age of 53.2 years, 55.9% being female, and 59.8% having a diabetes duration of more than 10 years. The majority of patients had high to very high cardiovascular risk. Among the patients,

37.3% had chronic kidney disease and 32.4% had ischemic heart disease. The LDL-C control at the latest follow-up was suboptimal, with a mean LDL-C of 2.71 mmol/L. Additionally, 33.3% of patients were not initiated on the appropriate statin intensity, and 12% did not receive any lipid-lowering therapy. 20% of patients were on high doses of atorvastatin (60-80 mg), with limited use of combination therapy. Despite recognizing the patients' cardiovascular risk, there was clinical inertia in intensifying treatment.

CONCLUSION

This clinical audit highlights weaknesses in adherence to clinical guidelines and clinical inertia in dyslipidemia treatment. There is a greater need for continuous education and a stronger emphasis on achieving treatment goals in the management of T2D patients. Additionally, a reassessment of the budget for the availability of combination therapy options is necessary.

EP_A178

OBESITY TREATMENT: IMPACT OF BLOOD GLUCOSE, LIPID AND NON-ANTIOBESITY DRUGS ON MUSCLE MASS

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INTRODUCTION

While obesity is often linked to excess muscle mass, emerging data reveal a paradoxical relationship between metabolic parameters and sarcopenia. This study examines the interplay between blood glucose regulation, lipid metabolism, and muscle mass retention in metabolic obesity.

METHODOLOGY

A cross-sectional study was conducted at Hospital Sultan Abdul Aziz Shah (HSAAS), Serdang, Selangor, to identify factors influencing muscle mass changes in metabolic obesity. Adults (≥ 18 years) with BMI ≥ 27 kg/m² and at least two comorbidities were included, while those with bariatric surgery or conditions causing intentional weight loss were excluded. Clinical data, including BMI, metabolic parameters, and medication use, were collected. Sample size was determined using a correlation formula.

RESULT

Among 35 individuals (BMI ≥ 26.5 kg/m²), hyperglycemia (HbA1c $> 6.5\%$) and hypertriglyceridemia (≥ 1.7 mmol/L) correlated with muscle loss, whereas normoglycemia

Adult E-Poster

and favorable lipid profiles (HDL ≥ 1.2 mmol/L, LDL < 2.6 mmol/L) were protective. Moderate obesity (BMI 26.5–39.9) was universally associated with muscle gain, whereas severe obesity (BMI ≥ 40) showed mixed outcomes. Beta-blockers and hormones promoted muscle retention, while statins and protease inhibitors correlated with muscle decline.

CONCLUSION

Metabolic control, rather than BMI alone, plays a critical role in muscle retention among obese individuals. Glycemic and lipid optimization may be key in mitigating sarcopenia risk in metabolic obesity.

EP_A179

EFFECTS OF SGLT2 INHIBITOR INITIATION ON INSULIN-TREATED TYPE 2 DIABETES PATIENTS: A SINGLE CENTRE EXPERIENCE

<https://doi.org/10.15605/jafes.040.S1.187>

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INTRODUCTION

Sodium-glucose co-transporter-2 (SGLT2) inhibitors have revolutionized the management of type 2 diabetes mellitus (T2DM) by enhancing glycaemic control, promoting modest weight loss, and providing proven cardiovascular and renal benefits. The impact of SGLT2 inhibitors on insulin-treated T2DM patients has also been highlighted in major clinical trials. This study examines the effects of SGLT2 inhibitors in insulin-treated T2DM patients in a dedicated diabetes clinic, focusing on HbA1c, insulin dosage and regimen, and weight changes after six months of treatment.

METHODOLOGY

This retrospective study was conducted at the diabetes clinic of Hospital Sultan Haji Ahmad Shah. Insulin-treated T2DM patients who were initiated on SGLT2 inhibitors between June and August 2024 were included in the study. Patients on concomitant GLP-1 receptor agonist therapy were excluded. Electronic medical records were reviewed for patient follow-up records.

RESULT

Fifty patients were included in the study, with a mean age of 52.32 years, and a predominance of female patients (64%). 74% of the patients were initiated on empagliflozin. The initiation of SGLT2 inhibitors resulted in a 12% reduction in basal-bolus therapy, with insulin treatment being de-intensified to premixed insulin therapy. There was a modest reduction in total daily dose (TDD) of insulin

use (mean reduction 1.12 units, SD 19.4), HbA1c (mean reduction 0.36%, SD 1.8), and weight (mean reduction 1.02 kg, SD 7.5). 34% of patients experienced a reduction in TDD insulin use of more than 5 units, and 66% showed a reduction in HbA1c levels. In the empagliflozin-treated group, there was a greater reduction in TDD insulin and weight, while the dapagliflozin-treated group showed a greater reduction in HbA1c.

CONCLUSION

Initiation of SGLT2 inhibitors in insulin-treated T2DM patients has shown promising effects, supporting the initiative for insulin deintensification. However, further exploration and investigation are needed to assess the long-term metabolic effects and durability of SGLT2 inhibitor treatment.

EP_A180

DIABETIC KETOACIDOSIS MANAGEMENT IN HOSPITAL SULTAN HAJI AHMAD SHAH (HOSHAS): A CLINICAL AUDIT

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INTRODUCTION

Diabetic ketoacidosis (DKA) is a serious condition and improper initial assessment and management may lead to undesirable outcomes and even death. This clinical audit aimed to evaluate adherence to DKA management in HoSHAS according to standardized national and local guidelines. The standards pre-determine by local standards were: (1) Severe DKA patients should be managed in an ICU/HDW setting; (2) All patients should be treated according to standardized guidelines (fluid and insulin therapy, observation) and achieve resolution of DKA within 24 hours of diagnosis; (3) All patients should be assessed by diabetes educators prior to discharge; (4) All patients should have a well-documented discharge and follow-up plan.

METHODOLOGY

This audit was conducted from November to December 2024, involving all adult patients (aged 18 years and above) who met the diagnostic criteria for DKA. Patients with concurrent cardiac disease, ESRD, elderly patients, and pregnancy were excluded. Patient demographics and clinical data were collected from electronic medical records.

Adult E-Poster

RESULT

Thirty patients were included with predominant male (56.7%) and Malay ethnicity (90%) and median age of 54 years. Majority had underlying T2DM (93.3%) and presented with sepsis (96.7%). The median length of hospital stay was 5.5 days. 33.3% of patients had severe DKA, but only 30% were managed in an ICU/HDW setting. All patients were treated according to standardized guidelines and had resolution of DKA within 24 hours. Only (55.5%) of patients were assessed by diabetes educators before discharge. Majority of patients had a well-documented discharge plan, but only (70%) had follow-up care arranged within the hospital.

CONCLUSION

The audit revealed good adherence to standardized DKA management protocols. However, there is room for improvement in the prioritization of severe DKA cases for ICU/HDW admission. The importance of diabetes educators in the post-recovery phase of DKA should be better recognized, and follow-up care process needs to be enhanced.

EP_A181

DIABETES EDUCATOR STATUS AND WELL-BEING STUDY IN PAHANG

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INTRODUCTION

Diabetes educators (DEs) are essential in shared decision-making for diabetes care. Psychological and social factors influence their well-being, motivation, and efficiency in delivering care.

METHODOLOGY

This is a cross-sectional study conducted from June to November 2024, involving DEs in Pahang using the QPS Nordic questionnaire. Responses were measured on a Likert scale (1: Very Seldom/Never to 5: Very Often/Always), assessing job demands, role clarity, role conflict, job predictability, mastery perception, leadership support, social climate, work-life balance, and motivation.

RESULT

Seventy-five DEs participated in the study (mean age: 39.3 years, SD: 5.5). Fifty-seven percent were hospital-based (80% in wards, 20% in clinics), and 43% worked in primary care. 65% had over five years of experience. The respondents had a neutral view of their quantitative job

demands (Mean score: 3.09) and had good role clarity in their institution (Mean score: 3.96). However, they faced significant conflict in completing their responsibilities and inadequate resources in their daily work. Job predictability was perceived as neutral in both short-term and long-term aspects. Most respondents were positive about their work quality and were satisfied with their ability to complete tasks. The respondents viewed their immediate superiors positively, particularly in listening to their work-related problems. Despite this, the respondents had a neutral view on superiors' empowering leadership or creating space to voice opinions. Respondents felt there was no job interference with their personal life (Mean score: 2.71). They were highly motivated to achieve success, recognition, and security in their careers, with a focus on a peaceful and healthy working environment.

CONCLUSION

Diabetes educators in this study demonstrated high motivation to excel in their profession but faced significant difficulties in fulfilling their roles. Emphasizing empowering leadership and fostering a supportive work environment are crucial to improving diabetes educators' motivation, learning, and efficiency.

EP_A182

A RETROSPECTIVE REVIEW OF ADRENAL INCIDENTALOMAS IN MALAYSIA: CLINICAL CHARACTERISTICS AND NATURAL HISTORY

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INTRODUCTION/BACKGROUND

Adrenal incidentalomas (AI) are frequently detected during imaging for unrelated conditions, with the majority being benign and non-functional. Numerous studies on AIs have been conducted in the Western countries but there is a lack of data from this region.

METHODOLOGY

This retrospective study aimed to describe the clinical and radiological characteristics, as well as the natural history of AIs in a Malaysian cohort. Medical records of 251 AI patients from three tertiary hospitals were reviewed. Baseline demographic data, imaging characteristics, hormonal evaluations and histopathological findings were collected. Follow-up data on serial imaging, hormonal re-evaluations and eventual outcome were captured.

Adult E-Poster

RESULT

The median age of the cohort was 58 years, with almost equal gender distribution and most tumors being non-malignant (92%) and non-functioning (72%).

Bilateral AIs were present in 24 (9.6%) patients. Ten were functioning with mild autonomous cortisol secretion (MACS) being the commonest subtype (80%).

Among the non-malignant AIs, 27% were functioning. Of these, the majority (51%) were diagnosed with MACS, followed by pheochromocytoma (27%), primary hyperaldosteronism (21%), and one case of overt Cushing's syndrome. Functioning AIs were associated with higher rates of hypertension and osteoporosis, larger tumour size, greater tumour density, and a lower rate of absolute contrast washout.

Adrenalectomy was performed more frequently in the functioning group, primarily for MACS and pheochromocytoma. The majority (94%) of non-functioning benign AIs were managed conservatively. No malignant transformation was observed, and only one case became functional over a median follow-up of 30 months.

Of the 20 malignant AIs, 12 were primary adrenal cancers. The malignant AIs were more likely to present with weight loss, overt Cushing's, larger tumor size, higher density, reduced contrast washout, and significantly higher mortality compared to non-malignant AIs.

CONCLUSION

This study suggests that most benign non-functioning AIs pose minimal risk of progression, supporting reduced follow-up in stable cases.

EP_A183

INSIGHTS FROM A 2024 CLINICAL AUDIT OF THYROID STORM CASES AT HOSPITAL SULTAN ISMAIL, JOHOR BAHRU

<https://doi.org/10.15605/jafes.040.S1.191>

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INTRODUCTION/BACKGROUND

Thyroid storms require immediate medical intervention due to the risk of rapid multi-organ failure and high mortality. Therefore, a retrospective audit of thyroid storm

management at Hospital Sultan Ismail, Johor Bahru (HSIJB) was done, to ascertain strengths and identify areas for improvement.

METHODOLOGY

This audit analyzed all 17 thyroid storm cases admitted to HSIJB in 2024 (1st January 2024 to 31st December 2024). Data was extracted from electronic medical records.

RESULT

Most were female (70%, n = 12) with mean age of 48 years (range 26 to 75 years). All had Bursch-Wartofsky Point Scale of at least 45 (range 45 to 140). The commonest presentation was cardiovascular manifestations (100% tachycardia, 76% atrial fibrillation, 58% heart failure), followed by gastrointestinal-hepatic dysfunction (53%) and CNS effects (47%). All 5 ventilated patients were co-managed in the ICU. Predominant etiology was Graves' disease (88%, n = 15), with a case of gestational trophoblastic disease. Main precipitants were medication non-adherence (50%, n = 8), infection (23%, n = 4), and new thyroid diagnosis (29%, n = 5). Treatment was initiated within 6 hours of presentation in 82% of cases (n = 14). In the remaining 3 cases, treatment was delayed by up to 9 hours while awaiting TFT results, as these patients had no prior history of thyrotoxicosis. Aside from one death within 3 days due to thyroid storm and tubo-ovarian abscess, there was no other mortality at up to 180 days after discharge.

CONCLUSION

Timely intervention in thyroid storms is critical to optimize patient outcomes. However, diagnosis can be challenging, particularly in patients without known thyroid disorders, which may result in delayed treatment. As such, it is essential to initiate therapy promptly based on strong clinical suspicion, even prior to laboratory results. Additionally, addressing issues related to treatment non-adherence through targeted patient education is vital to reduce the incidence of thyroid storm.

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EP_A184

PLASMAPHERESIS FOR THYROTOXICOSIS: EXPERIENCE FROM A MALAYSIAN ACADEMIC MEDICAL CENTRE

<https://doi.org/10.15605/jafes.040.S1.192>

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INTRODUCTION

Plasmapheresis is a therapeutic option for patients with severe thyrotoxicosis as a bridging therapy to urgent thyroidectomy, or when conventional antithyroid medications are ineffective, contraindicated, or cause adverse reactions. This retrospective study aims to describe the indications of plasmapheresis, clinical outcomes, and the periprocedural thyroid hormone level changes among patients with thyrotoxicosis in our local setting.

METHODOLOGY

Electronic medical records of all patients who required plasmapheresis in thyrotoxicosis for various indications at Universiti Malaya Medical Centre from April 2022 to April 2025 were reviewed. Clinical outcomes were recorded, and periprocedural FT4 levels were tabulated.

RESULT

There was a total of 11 patients in the study – nine (81.82%) were females, with a mean age of 49.9 ± 22.2 years. Only five (45.45%) fulfilled criteria for thyroid storm, whereas four (36.36%) required urgent thyroidectomy for other reasons, two (18.18%) required urgent reversal of thyrotoxicosis for other surgeries. Five (45.45%) patients died - three of whom were due to thyroid storm, and two due to other concurrent acute illnesses. Four (36.36%) patients developed periprocedural complications including allergic reaction and tachyarrhythmias. Overall, each patient underwent an average of 3.72 ± 2.33 cycles of plasmapheresis, whereas the survivors required 4.17 ± 3.13 cycles to achieve their therapeutic goals. All patients used fresh frozen plasma for exchange, with an average of 1.3 ± 0.3 times total plasma volume. Over 33 cycles in between all patients, the average reduction of FT4 per liter of plasma exchanged was 2.26 ± 5.09 pmol/L, and the average percentage reduction of FT4 after each treatment was 11.0 ± 28.1 %.

CONCLUSION

Patients in thyrotoxicosis who require plasmapheresis usually have complex or severe diseases. Apart from

currently recognised indications, another indication would be patients who require urgent lowering of thyrotoxicosis for other interventional procedures. There is a need for standardization of care to facilitate early recognition and timely implementation of this life-saving procedure.

EP_A185

VITAMIN D TESTING IN ADULT PATIENTS: AN AUDIT IN HOSPITAL TELUK INTAN

<https://doi.org/10.15605/jafes.040.S1.193>

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INTRODUCTION

Vitamin D deficiency is a prevalent condition associated with various health issues, including osteoporosis, fractures, metabolic disorders, and electrolyte imbalances like hypocalcemia. Despite its clinical importance, Vitamin D testing in Hospital Teluk Intan (HTI) is limited, requiring samples to be outsourced to Hospital Raja Permaisuri Bainun, with a turnaround time of approximately one week. This audit aims to assess the number of tests conducted, Vitamin D level distribution, and clinical indications for testing.

METHODOLOGY

This retrospective audit reviewed Vitamin D tests performed from January to December 2024. Adult patients (≥ 18 years) with completed test records were included, while pediatric patients and incomplete records were excluded. Data were collected from laboratory reports and clinical records.

RESULT

A total of 28 adult tests were conducted, with 24 females and 4 males. Among these, 75% were deficient (<50 ng/mL), 10.7% insufficient (50–74 ng/mL), and 14.3% sufficient (>75 ng/mL). The main clinical indications for testing were osteoporosis, rheumatoid arthritis, and thalassemia. Rheumatology had the highest test requests, followed by orthopedics and general medicine.

CONCLUSION

The audit highlights a low number of Vitamin D tests, with a high deficiency rate among patients. Limited test availability and delayed results may contribute to underdiagnosis. Raising awareness among healthcare providers, expanding test access, and integrating Vitamin D screening into routine care for high-risk patients could improve outcomes.

Adult E-Poster

EP_A186

TSH RECEPTOR ANTIBODY (TRAb) TESTING IN NON-PREGNANT ADULTS: AN AUDIT IN HOSPITAL TELUK INTAN

<https://doi.org/10.15605/jafes.040.S1.194>

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INTRODUCTION

Determining the etiology of hyperthyroidism is essential for appropriate management, as different causes, such as Graves' disease, toxic multinodular goiter (MNG), and toxic adenoma, require distinct treatment approaches. Among these, Graves' disease is the most common autoimmune cause of hyperthyroidism, and TSH receptor antibody (TRAb) testing plays a crucial role in its diagnosis.

At Hospital Teluk Intan, TRAb testing is not available on-site and must be outsourced to Hospital Kuala Lumpur (HKL), located approximately 160 km away. Samples are sent twice a week, and results typically take up to two weeks. This audit aims to evaluate the number of TRAb tests performed in non-pregnant adults, analyze positive and negative results, and assess the workload and clinical significance of outsourcing this test.

METHODOLOGY

A retrospective audit was conducted from January to December 2024, including all non-pregnant adult patients (≥12 years) who underwent TRAb testing. Pregnant patients, pediatric cases, and those with incomplete data were excluded. Data was retrieved from laboratory records and patient files, focusing on test requests, positivity rates, and processing issues.

RESULT

A total of 111 TRAb tests were sent during the audit period. Of these, 64 (59.8%) were positive, supporting a diagnosis of Graves' disease, while 43 (40.1%) were negative. Four samples were rejected due to incomplete clinical summaries or the absence of a specialist's countersignature.

Analysis showed that TRAb levels did not correlate with disease severity when compared with thyroid function test (TFT) results.

CONCLUSION

This audit highlights the high demand for TRAb testing and the significant proportion of positive results. Given its diagnostic importance in differentiating Graves' disease

from other causes of hyperthyroidism, in-house TRAb testing at Hospital Teluk Intan would reduce delays and improve patient management. Establishing local testing capabilities could enhance efficiency, facilitate timely diagnosis, and optimize treatment planning.

EP_A187

RENIN-ALDOSTERONE RATIO: AN AUDIT ON SAMPLES AND RESULTS IN HOSPITAL TELUK INTAN

<https://doi.org/10.15605/jafes.040.S1.195>

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INTRODUCTION

Primary aldosteronism (PA) is an important yet often under-diagnosed cause of secondary hypertension. The renin-aldosterone ratio (RAR) serves as a key screening test for PA. This audit evaluates the number of RAR samples sent, their outcomes, and their implications for clinical practice over a three-year period from 2022 to 2024. As RAR testing is not available in Hospital Teluk Intan, all samples must be outsourced to Hospital Putrajaya, with an average turnaround time of approximately one month. The objective of this audit is to determine the number of RAR tests performed in Hospital Teluk Intan. Additionally, it seeks to analyze the proportion of positive and negative results and assess the adequacy of PA screening among hypertensive patients.

METHODOLOGY

This retrospective audit was conducted over a three-year period from January 2022 to December 2024. The study included all patients tested for RAR in Hospital Teluk Intan, while cases with incomplete results or missing data were excluded. Data was collected from laboratory records and patient files.

RESULT

Over the study period, a total of 48 RAR tests were conducted. In 2022, two cases tested positive while 18 were negative. In 2023, two cases were positive and 11 were negative. In 2024, one case was positive and 14 were negative. Among the five patients with positive results, two underwent adrenalectomy and subsequently recovered. Two patients declined further treatment due to advanced age and concerns about worsening kidney disease, while one patient remains under investigation.

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CONCLUSION

Although a significant number of RAR tests were performed, the majority yielded negative results, reinforcing the fact that primary aldosteronism remains a relatively rare condition compared to primary hypertension. The audit findings suggest that approximately one in ten tested cases were diagnosed with PA. These results highlight the importance of targeted screening to ensure appropriate patient selection for RAR testing, thereby optimizing resource utilization and timely intervention.

EP_A188

MELIOIDOSIS AND DIABETES MELLITUS IN HOSPITAL TELUK INTAN: AN AUDIT OF OUTCOMES AND THEIR ASSOCIATION WITH GLYCEMIC CONTROL

<https://doi.org/10.15605/jafes.040.S1.196>

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INTRODUCTION

Melioidosis, caused by *Burkholderia pseudomallei*, is endemic in tropical climates and poses a heightened risk to individuals with diabetes mellitus, particularly those with poor glycemic control. Diabetes is a well-established risk factor for severe melioidosis, often resulting in worse clinical outcomes. This audit aims to assess the prevalence, clinical outcomes, and the relationship between glycemic control and melioidosis in patients treated at Hospital Teluk Intan in 2024. Notably, 42 cases of melioidosis were reported in Perak during the year.

METHODOLOGY

A retrospective audit was conducted on all confirmed melioidosis cases from January to December 2024. Data was collected from medical records and laboratory databases. Key variables include patient demographics, HbA1c levels, site of infection, length of hospital stay, ICU admission, and mortality. Patients were stratified based on HbA1c levels to assess glycemic control.

RESULT

In 2024, a total of 16 melioidosis cases were reported at Hospital Teluk Intan. The majority of patients were male (13), with 3 female patients. The mean age was 55 years. Of the cases, 12 patients were Malay, 1 was Indian, and 3 were foreign nationals. The mortality rate was 37.5% (6

patients), while 10 patients survived, aligning with reported mortality rates of 10–40% for melioidosis.

Among survivors, the average hospital length of stay was approximately 20 days. Of the 6 mortality cases, only 1 patient received ICU care due to limited bed availability. Type 2 diabetes mellitus (T2DM) was present in 13 out of 16 cases, with a mean HbA1c of 12.2%. Notably, 3 patients were newly diagnosed with diabetes during admission.

Melioidosis was diagnosed through blood cultures in 13 cases, while 3 cases were confirmed via tissue or swab cultures. Only 6 patients reported working in soil-related occupations, while the rest were pensioners or unemployed. Beyond T2DM, 2 patients had chronic kidney disease (CKD), and 1 patient had an underlying gastrointestinal malignancy.

CONCLUSION

This audit provides valuable insights into the burden of melioidosis in diabetic patients and the impact of glycemic control on disease severity. The findings may inform future clinical practices, emphasizing the importance of optimizing diabetes management to reduce melioidosis-related morbidity and mortality.

EP_A189

UNRAVELLING AN UNRECOGNIZED CAUSE OF DIABETES DISTRESS AMONGST DIABETES PATIENTS DURING WORLD DIABETES DAY 2024 SCREENING INITIATIVE

<https://doi.org/10.15605/jafes.040.S1.197>

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INTRODUCTION

In observance of World Diabetes Day 2024, with the theme Diabetes and Well-being: Physical, Mental, and Societal Health, a screening was conducted to assess diabetes distress among patients at the diabetes clinic of Hospital Tengku Ampuan Rahimah, Klang.

METHODOLOGY

A total of 34 patients participated in the Diabetes Distress Scale (DDS-17) screening, either self-administered or assisted by diabetes educators with informed consent. DDS-17 assesses four subscales: Emotional Burden, Physician-Related Distress, Regimen-Related Distress, and Interpersonal Distress. A mean score ≥ 3 indicated signifi-

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cant distress. Patients with significant Emotional Burden were further screened for depression and anxiety using the Patient Health Questionnaire-9 (PHQ-9) and General Anxiety Disorder-7 (GAD-7).

RESULT

The cohort comprised 8 males and 26 females, with a mean age of 47.5 years. The median DDS-17 score was 2.0, with Emotional Burden having the highest median score (2.81), followed by Regimen-Related Distress (2.40), Interpersonal Distress (1.67), and Physician-Related Distress (1.25). Notably, 13 patients (38.2%) reported significant Emotional Burden, of whom 8 (61.5%) screened positive for depression (PHQ-9 ≥ 5) and anxiety (GAD-7 ≥ 5). Among them, 4 had mild depression and anxiety (PHQ-9 and GAD-7: 5–9), 1 had moderate depression and mild anxiety (PHQ-9: 10–14, GAD-7: 5–9), 1 had moderate depression and anxiety (PHQ-9 and GAD-7: 10–14), and 2 had severe depression and anxiety (PHQ-9 and GAD-7: 20–27). These patients were referred for psychiatric assessment. Additionally, 6 patients (17.6%) had significant diabetes distress (DDS ≥ 3).

CONCLUSION

This screening revealed a significant emotional burden among diabetes patients, previously unrecognized. Further studies need to be conducted for future recommendations for screening for diabetes distress and emotional burden in diabetes patients to improve patient outcomes and well-being.

EP_A190

AUDIT ON ALDOSTERONE-TO-RENIN SAMPLING IN SCREENING FOR PRIMARY ALDOSTERONISM: SINGLE-CENTER, TERTIARY DISTRICT HOSPITAL EXPERIENCE

<https://doi.org/10.15605/jafes.040.S1.198>

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INTRODUCTION

Aldosterone-to-renin ratio (ARR) sampling is the first line investigation for detection of hyperaldosterone-driven hypertension. Clinical practice guidelines (CPG) advocate testing the ARR in specific indications with special consideration in confounding factors, especially types of antihypertensive medicine. We aimed to determine the adherence of ARR sampling as outlined by CPG.

METHODOLOGY

We retrospectively evaluated ARR requests taken from January 2020 till December 2024 in Hospital Sultan Haji

Ahmad Shah. Demographic data associated with or without hypertension, indication for screening, interfering medications and outcomes were extracted from medical records.

RESULT

Out of 287 tests retrieved, only 222 were qualified for analysis. The median age was 34 (interquartile range, IQR 11) with 133 (59.9%) males. The medical duration of hypertension was 5 years (IQR 7). The majority of ARR sampling was sent for onset of hypertension less than age 40 ($n = 150$, 67.6%). Other indications were resistant hypertension ($n = 28$, 12.6%), hypertension with hypokalemia ($n = 28$, 12.6%), hypertension with adrenal incidentaloma ($n = 4$, 1.8%) and family history with hypertension onset of less than 40 or cardiovascular disease (CVD), $n = 4$, (1.8%). The ARR were found to be positive or indeterminate in 23 samples (10.4%); highest among cohort of hypertension with hypokalemia, $n = 12$ (42.9%) then adrenal incidentaloma and family history of young onset hypertension/ CVD (25% each) and later was resistant hypertension, $n = 4$ (14.3%). Hypertension onset of less than 40 only yields a 3.3% positivity rate ($n = 5$). Interfering medicines did not significantly impact ARR results. Of 23 samples, 15 (65.2%) were confirmed primary hyperaldosteronism.

CONCLUSION

ARR sampling was overly investigated among hypertensive less than 40 years old. Adherence to indications as per guideline recommendations needs to be strengthened to prevent wasteful resources.

EP_A191

A CLINICAL AUDIT OF DIABETES CARE AMONG OLDER ADULTS ADMITTED TO MEDICAL WARDS: A SINGLE CENTRE EXPERIENCE

<https://doi.org/10.15605/jafes.040.S1.199>

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INTRODUCTION

Diabetes mellitus is a global health concern, the proportion of people living with diabetes mellitus increases every year, particularly in older adults. The adherence to inpatient glycaemic care guidelines is low. This study aims to assess compliance with inpatient glycaemic care guidelines in medical wards in a single centre and identify areas for improvement. Secondary objectives include assessing the association of achievement of glycaemic targets with 30-day readmission and inpatient mortality rates.

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METHODOLOGY

A retrospective audit of the medical records was conducted for 348 patients living with diabetes admitted to wards 13 u and 11 u between 1st of April 2023 to 30th July 2023. Point of care standard was set based on Malaysia Endocrine and Metabolic Society inpatient glycaemic management guidelines. The definition of achievement of inpatient glycaemic target is when $\geq 80\%$ of blood glucose reading lies between 7.0 mmol/L and 10.0 mmol/L during the hospital stay. Statistical analyses were performed using SPSS version 28.0.

RESULT

Of the 348 patients, 142 (40.8%) of patients were Chinese, 101 (29%) Indians, 97 (27.9%) Malays with a mean age of 77. Overweight was seen in 151 (43.3%) of patients. Adherence to guidelines while using intravenous insulin was poor, highest at 40.6%. Inpatient glycaemic targets were achieved in 39% of patients admitted. Patients who did not achieve glycaemic goal targets had a higher rate of 30-day readmissions (67.3 % versus 32.7 %) and inpatient mortality (69.2% vs 30.8%), although statistically insignificant.

CONCLUSION

This study highlights low rates of compliance towards glycaemic care guidelines in medical wards 13 u and 11 u. There was inadequate monitoring, and more than half were found to be non-adherent to guidelines while patients were on continuous insulin infusion. This underscores the need for staff training and improved screening for diabetes complications. Future audits should focus on addressing these deficiencies to enhance patient outcomes.

EP_A192

A SURVEY ON PRACTICE OF INSULIN THERAPY AMONG HEALTHCARE PROVIDERS

<https://doi.org/10.15605/jafes.040.S1.200>

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INTRODUCTION

Effective insulin therapy is crucial in the management of type 2 diabetes mellitus (T2DM). While initiation and intensification are commonly practiced, insulin de-intensification—reducing or simplifying insulin regimens—remains less well described in clinical settings.

METHODOLOGY

This is a retrospective review of a cross-sectional survey conducted among healthcare providers who attended the

“Insulin Workshop for Healthcare Professionals 2024” at Hospital Putrajaya on November 14, 2024. All participants who completed the self-administered questionnaire were included. Data on professional background, insulin therapy practices, and pre- and post-workshop knowledge scores were analyzed.

RESULT

A total of 167 respondents participated in the survey, with 43% from hospitals and 57% from primary care. Most respondents had over 10 years of working experience (51.5%), followed by 5–10 years (38.3%), and less than 5 years (10.2%). The majority reported initiating insulin in outpatient settings (87.4%), intensifying therapy when appropriate (97%), and practicing insulin de-intensification (90%) in their practice. Additionally, 73.7% stopped insulin in selected patients. The mean knowledge score improved from 76.77% pre-test to 89.39% post-test following the workshop.

CONCLUSION

Insulin initiation and intensification are commonly practiced among local healthcare providers. A high proportion is also aware about insulin de-intensification and reported practicing insulin de-intensification and discontinuation, suggesting growing awareness of individualized diabetes care. The significant improvement in post-test scores highlights the effectiveness of structured educational interventions in enhancing knowledge related to insulin therapy and T2DM management. Despite limitations of the retrospective, self-reported design and lack of sample generalizability, these findings support the value of ongoing training to promote safe and evidence-based insulin use and promote safe de-intensification in clinical practice.

EP_A193

PATIENTS' OUTCOME AND COMPLIANCE IN OBESITY CLINIC HOSPITAL KAJANG – A RETROSPECTIVE AUDIT OF WEIGHT LOSS INTERVENTIONS

<https://doi.org/10.15605/jafes.040.S1.201>

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INTRODUCTION

The primary goals of this audit were to assess outcome and compliance with the Obesity Clinic program Hospital Kajang. The outcomes were measured by weight change, blood pressure, low density lipoprotein (LDL), and glycaemic control. Compliance was assessed by the

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number of defaulters and adherence to prescribed dietary recommendations.

METHODOLOGY

The audit was conducted by the Endocrine Team of Hospital Kajang starting from January till December 2024. A total of 37 patients who attended two or more sessions were included. Data was collected from patient records, including anthropometric, laboratory investigations and treatment adherence.

RESULT

The average baseline BMI of patients is 44.47 kg/m² and 37% of patients have diabetes. A total of 75.7% of patients achieved at least 5% weight loss throughout the follow-up. Mean weight reduction was 2.997 kg ($p = 0.012$). Systolic blood pressure ($p = 0.385$) and LDL ($p = 0.894$) did not show significant changes throughout follow-up. HbA1c among diabetes patients also showed no significant changes ($p = 0.243$). Adherence to treatment was reported in 75.7%, while 97.3% of patients returned for follow-up.

CONCLUSION

This audit demonstrates that the Obesity Clinic at Hospital Kajang is effective in facilitating weight reduction. While most patients achieved weight loss, clinical improvements in metabolic parameters were not statistically significant. Future interventions should focus on improving adherence to reinforce long-term outcomes.

EP_A194

AUDIT OF POST-THYROIDECTOMY COMPLICATIONS AT HOSPITAL TELUK INTAN

<https://doi.org/10.15605/jafes.040.S1.202>

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INTRODUCTION

Thyroidectomy is a common surgical procedure performed for various thyroid conditions. While generally safe, evaluating post-thyroidectomy complications is crucial for improving patient outcomes. This study audits the incidence and types of complications following thyroidectomy at Hospital Teluk Intan.

METHODOLOGY

This retrospective audit analyzed medical records of patients who underwent thyroidectomy at Hospital Teluk Intan from January to December 2024. Data collected included patient demographics, surgical types, and complications within one year after surgery. Complications assessed were hypocalcemia, hemorrhage, vocal cord palsy, wound infection, and other rare events.

RESULT

A total of 33 thyroidectomy procedures were performed in 2024, comprising 21 total thyroidectomies and 12 hemithyroidectomies. The majority of patients were female (29 cases, 88%), with a mean age of 47 years.

The leading indication for surgery was suspicious nodules on fine-needle aspiration cytology (FNAC) (23 cases, 70%), followed by multinodular goiter (6 cases, 18%) and compressive symptoms (4 cases, 12%).

Histopathological analysis identified papillary carcinoma as the most common malignancy (12 cases, 36%), followed by follicular carcinoma (5 cases, 15%), secondary thyroid metastasis (1 case, 3%), and Hürthle cell carcinoma (1 case, 3%). Among benign cases, 12 were nodular hyperplasia (36%) and 2 were benign adenomas (6%).

Post-thyroidectomy complications occurred in 51% of cases, predominantly after total thyroidectomy. Hypocalcemia due to hypoparathyroidism was the most frequent complication (13 cases, 39%), with a strong correlation to thyroid mass size (9 cases). Vocal cord palsy occurred in 4 cases, with 2 involving larger thyroid mass. No cases of significant postoperative bleeding or infection requiring reoperation were reported. A Chi-square test revealed a significant association between the type of surgery, with total thyroidectomy being more frequently associated with postoperative hypocalcemia.

CONCLUSION

This audit highlights that post-thyroidectomy complications remain a significant concern, particularly following total thyroidectomy, with hypocalcemia being the most common adverse outcome. The findings underscore the importance of thorough preoperative assessment, surgical precision, and vigilant postoperative monitoring—especially for patients with larger thyroid masses or those undergoing total thyroidectomy.

Adult E-Poster

EP_A195

EARLY REAL-WORLD EVIDENCE FOR THE USE OF ORAL SEMAGLUTIDE IN A TERTIARY CENTRE IN MALAYSIA

<https://doi.org/10.15605/jafes.040.S1.203>

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INTRODUCTION

The efficacy of oral semaglutide has been well-demonstrated in the randomized controlled trials (RCT) of the PIONEER programme, but real-world evidence is lacking. We aimed to analyze the effects of oral semaglutide after six months of use in a real-world setting.

METHODOLOGY

In this prospective single-centre study, we analyzed the metabolic and renal outcomes of patients commenced on oral semaglutide for six months. Patients were seen in specialized diabetes clinics, and all other targets were managed according to the standard of care.

RESULT

A total of 177 patients were analyzed (mean age 56.3 ± 12.82 years, 46% male, mean duration of diabetes 16.2 ± 4.8 years, 38.3% had ASCVD, mean eGFR 46.3 ± 14.8 mL/min/1.73 m², 58% on insulin, 78% on SGLT2i, 67% on RAAS blockade). Baseline HbA1c, FPG, weight, BMI, and UACR were $7.91 \pm 1.60\%$, 7.53 ± 2.78 mmol/L, 85.1 ± 21.2 kg, 33.5 ± 12.6 kg/m², and 9.8 mg/mmol (IQR 2.1-40) respectively. 80.2% tolerated semaglutide at a full dose of 14 mg daily, whilst 18.1% tolerated 7 mg daily. The discontinuation rate was 8.5%, mainly due to gastrointestinal side effects. Significant weight reduction, SBP, and HbA1c were seen with a mean difference of 3.12 kg (95% CI: 1.1 – 4.2, $p < 0.01$), 3.76 mm Hg (95% CI: 0.6 – 6.9, $p = 0.02$) and 0.31% (95% CI: 0.1 – 0.4, $p = 0.04$) respectively. FPG and UACR showed an improving trend despite missing statistical significance, FPG: -0.15 mmol/L (95% CI: -0.3 – 0.6 , $p = 0.053$) and UACR: -9.8 to 7.7 mg/mmol (IQR -4.1 to -20 , $p = 0.19$).

CONCLUSION

Most patients tolerated oral semaglutide at 14 mg well, with follow-up data showing significant improvement in weight, SBP, and HbA1c, comparable to that of RCTs.

EP_A196

PRE-RAMADAN COUNSELLING IN ADULTS WITH TYPE 2 DIABETES (T2D) IN INSTITUT ENDOKRIN HOSPITAL PUTRAJAYA

<https://doi.org/10.15605/jafes.040.S1.204>

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INTRODUCTION

Pre-Ramadan counselling is essential for safe fasting in Muslims with Type 2 Diabetes (T2D). Structured education on risk stratification, glycaemic monitoring, and medication adjustments reduces hypoglycaemia, hyperglycaemia, and dehydration risks. We audited outcomes before and after implementing pre-Ramadan continuous medical education (CME) for clinicians at Institut Endokrin Hospital Putrajaya (IEHPJ) to assess its impact on patient counselling.

METHODOLOGY

We conducted a retrospective study to audit pre-Ramadan counselling practices before (December 2024) and after (February 2025) CME implementation at IEHPJ using universal sampling from electronic medical records. We retrieved and reviewed records of all Muslim patients attending T2D clinics during the study period.

RESULT

This study included 693 patients, 309 from the pre-CME period and 384 in the post-CME period. There were 48.3% male patients with a mean age of 57.5 years (± 12). Diabetes duration was >10 years at 62.5%. The mean HbA1c of our patients was 8.4% (± 2.0). The majority of patients (91.6%) had eGFR >30 mL/min/1.73m². Regarding medications, 20.3% were on non-sulphonylurea oral glucose-lowering drugs with or without GLP-1-RA, while the remaining were on insulin and/or sulphonylurea. 16.1% were on a basal insulin regime, and 48.9% were on more complex multiple daily injection regimes.

Pre-Ramadan counselling significantly increased from 33.9% (105/309) pre-CME to 58.4% (230/394) post-CME ($p < 0.001$). Even though most patients were on treatment regimens that predisposed them to hypoglycaemia during fasting, IDF-DAR risk scoring assessment remained low in both groups. Although IDF-DAR risk scoring improved from 13 to 42 patients after the CME, this was not statistically significant.

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CONCLUSION

CME improved counselling rates in our clinics, but gaps in risk assessment persist. Implementing structured protocols in the future could further reduce risks during Ramadan fasting.

EP_A197

VALIDATION OF IDF-DAR RISK SCORE FOR FASTING IN RAMADAN FOR ADULTS WITH DIABETES MELLITUS IN PRIMARY CARE

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INTRODUCTION

Fasting during Ramadan is a religious obligation for Muslims but poses health risks for individuals with diabetes mellitus. The International Diabetes Federation–Diabetes and Ramadan Alliance (IDF-DAR) introduced a risk stratification tool in 2021 to guide clinicians, though its utility in primary care settings remains limited.

METHODOLOGY

We conducted a retrospective cohort study on adults with diabetes attending government health clinics in Malaysia from April 15 to June 15, 2024. Medical records of those who attempted fasting during Ramadan were reviewed. The primary outcome was a composite of hypoglycaemia, hyperglycaemia, diabetes-related hospitalization, or dehydration leading to breaking the fast. The discriminative performance of the IDF-DAR tool was evaluated using the area under the receiver operating characteristic curve (AUC). Calibration was assessed via the Hosmer-Lemeshow test.

RESULT

A total of 310 patients were included (99% with type 2 diabetes). The mean age was 61 years, and the median diabetes duration was 7 years. Adverse fasting outcomes were observed in 18.4% of patients, with hypoglycaemia

being the most common (13.5%). The IDF-DAR risk stratification tool demonstrated good discriminative ability, achieving an area under the ROC curve (AUC) of 0.78 (95% CI: 0.72–0.84). At the recommended cut-off for distinguishing low-moderate from high-risk categories, the tool achieved a sensitivity of 92.9% and a specificity of 40.9%. The Hosmer-Lemeshow goodness-of-fit test indicated poor agreement between observed and predicted adverse outcomes, with a statistically significant result ($p < 0.05$).

CONCLUSION

The IDF-DAR risk stratification tool identifies high-risk patients fasting during Ramadan in primary care. However, its poor calibration highlights the need to refine the model to improve its predictive accuracy. Enhancing the tool's calibration could allow for better individual risk estimation and more precise clinical decision-making in diverse primary care settings.

EP_A198

INCIDENCE OF HYPOGLYCEMIA FOLLOWING "INSULIN CHASE:" A SINGLE-CENTER CLINICAL AUDIT

<https://doi.org/10.15605/jafes.040.S1.206>

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INTRODUCTION

Hyperkalemia poses a significant threat due to its potential to induce fatal cardiac arrhythmias. "Insulin chase," or a combination of intravenous insulin, calcium gluconate, and dextrose, is given to rapidly lower serum potassium levels. Potential risks of this regime include hypoglycemia. This study aims to determine the incidence of hypoglycemia following the administration of "insulin chase" in our center, explore associated risk factors, and assess adherence to blood glucose (BG) monitoring when this regime is administered.

METHODOLOGY

This was a retrospective observational study. Medical records of all adult patients who received insulin chase treatment at Hospital Putrajaya between January 1, 2023, and December 31, 2024, were retrieved and reviewed.

RESULT

A total of 187 patients received insulin chase during the study period. The mean age was 58 years (SD 15.9). The

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majority were male patients (63.1%), and more than half (58.8%) had background diabetes mellitus. The incidence of hypoglycemia post-insulin chase was 16.6%. Patients who developed hypoglycemia had a significantly higher median creatinine level (678 $\mu\text{mol/l}$ vs. 349 $\mu\text{mol/l}$, $p = 0.005$). Prior use of sulfonylurea was also significantly associated with an increased risk of hypoglycemia (26.3% vs. 8.8%, $p = 0.031$). Factors such as age, gender, race, presence of diabetes mellitus, and prior insulin use were not found to be significantly associated with the development of hypoglycemia. Almost one-quarter of patients (23.5%) did not have BG checked prior, and only 40.6% had BG monitoring planned post-insulin chase.

CONCLUSION

This audit demonstrated a 16.6% incidence of hypoglycemia post-insulin chase. Renal impairment and prior sulfonylurea use were significant risk factors. There is a need to improve the planning and implementation of pre- and post-treatment glucose monitoring to prevent hypoglycemia.

EP_A199

PRESCRIBING PATTERNS OF SGLT2 INHIBITORS IN TYPE 2 DIABETES MANAGEMENT AT A TERTIARY CARE CENTER IN MALAYSIA

<https://doi.org/10.15605/jafes.040.S1.207>

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INTRODUCTION

Type 2 diabetes (T2D) continues to pose a significant public health challenge in Malaysia, affecting approximately 20% or 4.4 million adults. Among the newer treatment options, sodium-glucose co-transporter-2 inhibitors (SGLT2-i) have gained increasing attention due to their proven glycaemic and cardiorenal benefits. Despite the increasing adoption of SGLT2-i due to established glycaemic and cardiorenal benefits, Malaysia-specific prescribing patterns remain understudied, particularly in tertiary healthcare environments. This study aimed to evaluate the prescribing patterns of SGLT2-i and its relationship with antidiabetic utilization in T2D patients at Hospital Kuala Lumpur.

METHODOLOGY

This retrospective cohort study used patient data from the Pharmacy Information System (PhIS) and Lab Management System (LMS). Patients aged 18–70 years diagnosed with

T2D and prescribed empagliflozin or dapagliflozin between January - December 2023 were included. Data on demographics, initiation date of SGLT2-i, medication history, and HbA1c levels were extracted. Adherence to prescribing guidelines was assessed by comparing initiation criteria, dosing, and drug combinations against recommendations outlined in the Malaysian Clinical Practice Guidelines (6th edition). Medication adherence was measured using the Medication Possession Ratio extracted from PhIS. HbA1c levels before and after SGLT2-i treatment in adherent patients were compared using the Wilcoxon signed-rank test, with a significance level set at 0.05.

RESULT

Among the 256 patients analyzed, 77.3% of prescriptions adhered to national guidelines, with excellent dose compliance (97.3%). Combination therapy was predominant (98%), with metformin (82%), vildagliptin (54%), and gliclazide (20%) most frequently prescribed concurrently. Additionally, 60.2% received insulin concurrently with SGLT2-i. Notably, adherent patients experienced significant HbA1c reductions from a median baseline of 7.5% to 7.2% post-treatment ($p = 0.019$).

CONCLUSION

SGLT2-i prescribing practices at HKL closely align with national guidelines, primarily involving combination therapy. The observed significant improvement in glycaemic control among adherent patients underscores the value of SGLT2-i in routine clinical management of T2D. Future research should focus on long-term clinical outcomes and economic implications of widespread SGLT2-i use.

EP_A200

EFFECTS OF SODIUM-GLUCOSE COTRANSPORTER-2 INHIBITORS ON HEMATOCRIT AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS: A TERTIARY CENTER EXPERIENCE

<https://doi.org/10.15605/jafes.040.S1.208>

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INTRODUCTION

Sodium-glucose cotransporter-2 inhibitors (SGLT2i) have become integral to managing Type 2 Diabetes Mellitus (T2DM) due to their cardiorenal benefits. They promote osmotic diuresis, leading to hemoconcentration and increased erythropoiesis, which may theoretically raise

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thromboembolic risk. Emerging data suggests these changes reflect improved renal function and support their benefits. This study aimed to assess the impact of SGLT2 inhibitors on haematocrit in our patients with T2DM.

METHODOLOGY

This retrospective observational study involved patients with T2DM initiated on SGLT2 inhibitors at our center between January 2024 and September 2024. Patients were included if they received continuous empagliflozin for more than 3 months. We collected data on hemoglobin (Hb) and hematocrit (Hct) levels at baseline and up to 6 months post-initiation. Erythrocytosis was defined according to the 2016 WHO criteria: Hb >16.5 g/dL and/or Hct >49% in men and Hb >16 g/dL and/or Hct >48% in women.

RESULT

This study included 88 patients with T2DM (45 men [51.1%], 43 women [48.9%]) with a median age of 62.0 years (IQR 53-70). The cohort had a median diabetes duration of 10.0 years (IQR 4-19) and a median baseline HbA1c of 7.9% (IQR 6.7-9.8). After a median follow-up of 6.0 months (IQR 6-9), we observed significant increases in hematologic parameters: hemoglobin (12.9 ± 1.8 to 13.5 ± 1.6 g/dL, $p < 0.001$), hematocrit ($40.10 \pm 5.3\%$ to $41.3 \pm 4.7\%$, $p < 0.001$), and RBC count (4.75 ± 0.77 to $4.98 \pm 0.72 \times 10^{12}/L$, $p < 0.001$). HbA1c decreased by a median of 0.5% (IQR -1.0 to 0.0, $p < 0.001$). Despite these hematologic changes, post-treatment erythrocytosis prevalence remained low at 5.7% (5/88), and no treatment discontinuation was required.

CONCLUSION

These findings demonstrate that while SGLT2 inhibitors predictably increase hematologic indices, the risk of clinically significant erythrocytosis remains low. The observed hematologic changes likely represent adaptive physiological responses contributing to empagliflozin's cardiorenal protective effects.

EP_A201

WILL YOU CARE FOR ME: PROTOCOL AND BASELINE CHARACTERISTICS OF THE PILOT CARDIOLOGY-RENAL-METABOLIC (CaReMe) INTEGRATED CLINIC IN MALAYSIA

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INTRODUCTION

At least one in three Malaysians living with type 2 diabetes (T2D) develops cardio-renal complications. Current management strategies are fragmented. The pilot, Cardiology-Renal-Metabolic (CaReMe) Clinic in Malaysia, aims to integrate care of patients with T2D by focusing on patient-centred, guideline-directed medical therapy (GDMT) use.

METHODOLOGY

This is a pilot CaReMe clinic in Southeast Asia that was established at our centre. Patients with T2D and renal or cardiovascular complications were recruited for weekly virtual clinics. These clinics involve multidisciplinary meetings between cardiology, nephrology, and endocrinology specialists. Patients' cases and current issues were high-lighted, medications reviewed, and management plans formulated. Patients were followed up every six months with data collected at baseline and every six months for two years to assess metabolic, cardiovascular, and renal outcomes, including patient-related quality-of-life measures (SF 12). Patients in the CaReMe cohort were compared against standard care by propensity score matching methods.

RESULT

One hundred forty-two patients have been recruited (mean age: 62.9 ± 11.4 years, 55.6% men). The mean baseline HbA1c was $8.9 \pm 1.7\%$, with a mean duration of diabetes of 18.2 ± 10.0 years. The mean BMI and waist circumference were 29.6 ± 6.9 kg/m² and 101.5 ± 15.3 cm, respectively. Majority of patients had hypertension (96.4%) and dyslipidaemia (95.0%). In terms of ASCVD, 28.2% had coronary artery disease, 12.0% had stroke, while 2.8% had peripheral arterial

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disease. Within this cohort, 66.2% had CKD with a mean eGFR of 61.4 ± 30.5 mL/min/1.73 m² and a median uACR of 30.8 (6.05 – 111.3) mg/mmol. 19.4% had heart failure, 16.2% had retinopathy, and 3.5% had peripheral neuropathy. A large proportion of patients were on insulin (71.1%), and GDMT uptake were as follows: SGLT2- inhibitors (90.1%), GLP1-RA (28.1%), RAAS-blockade (88.0%), and statins (93.0%).

CONCLUSION

The pilot Malaysian CaReMe clinics adopt a holistic, patient-centred implementation of integrated care to address gaps and improve outcomes in T2D. These virtual multi-disciplinary clinics can easily be implemented within resource-limited settings.

EP_A202

GLYCAEMIC OUTCOMES FOLLOWING INSULIN DE-INTENSIFICATION IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: A RETROSPECTIVE OBSERVATIONAL STUDY IN A MALAYSIAN TERTIARY CENTRE

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INTRODUCTION

Insulin deintensification is the reduction, simplification, or cessation of insulin therapy. Despite its potential benefits, there is limited local data on real-world insulin deintensification practices. We investigated the impact of insulin deintensification on glycemic control among patients with type 2 diabetes mellitus (T2DM) attending outpatient follow-up at a Malaysian tertiary hospital.

METHODOLOGY

We conducted a retrospective observational study at the outpatient clinic of Hospital Sungai Buloh from January to December 2024. Adults with T2DM who underwent insulin deintensification were included. Patients with type 1 diabetes, gestational diabetes, or incomplete follow-up data were excluded. Primary outcomes were changes in HbA1c and pre-breakfast blood glucose (BG) levels. Secondary outcomes examined associations between diabetes duration and baseline HbA1c with glycemic outcomes. Paired t-tests and correlation coefficient tests were used for statistical analyses.

RESULT

A total of 33 patients were included. Most (n = 22) were initially on a basal-bolus regimen and subsequently de-intensified to premixed human insulin (n = 12), premixed analogue insulin (n = 7), oral agents (n = 2), or basal-only insulin (n = 1).

Among nine patients initially on human premixed insulin, five were switched to a premixed analogue regimen, two to oral agents, one to basal-only insulin, and one to basal analogue. Of the two patients on basal insulin, one transitioned to oral agents and the other to a premixed analogue regimen.

Nineteen patients had complete paired HbA1c data, with mean HbA1c improving from 10.36% (SD 2.70) to 8.93% (SD 2.49) (mean change -1.43%, $p = 0.003$).

Fifteen patients had complete pre-breakfast BG data, showing a mean reduction from 12.07 mmol/L (SD 3.86) to 9.90 mmol/L (SD 3.22) ($p = 0.037$).

Baseline HbA1c strongly correlated with follow-up HbA1c ($r = 0.76$, $p < 0.001$). Meanwhile, diabetes duration showed no significant association ($r = -0.24$, $p = 0.365$).

CONCLUSION

Insulin deintensification was associated with significant improvements in HbA1c and pre-breakfast BG levels, supporting its safe implementation in selected patients with T2DM. Baseline HbA1c was a strong predictor of post-intervention control. These findings highlight the potential for regimen simplification with structured follow-up and monitoring.

EP_A203

AN AUDIT OF CONTINUOUS GLUCOSE MONITORING ON GLYCAEMIC CONTROL AND METABOLIC PROFILES OF PATIENTS WITH TYPE 1 DIABETES MELLITUS AT A TERTIARY CENTRE

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INTRODUCTION

Type 1 Diabetes Mellitus (T1DM) is a chronic autoimmune disorder that results in absolute insulin deficiency and an elevated risk of both microvascular and macrovascular complications. Achieving optimal glycemic control is essential for preventing these complications. Continuous

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Glucose Monitoring (CGM) has been demonstrated to enhance glycemic control compared to conventional self-monitoring of blood glucose (SMBG). This audit aims to assess glycemic control and metabolic profiles among patients with T1DM at the Endocrine Institute, Putrajaya Hospital, comparing those using CGM with those relying on SMBG to determine whether CGM leads to improved metabolic outcomes.

METHODOLOGY

A retrospective cross-sectional study was conducted at the Endocrine Institute, Putrajaya Hospital. Electronic medical records of patients who attended the T1DM clinic between April 1, 2024, and March 31, 2025, were reviewed. Descriptive and statistical analyses of glycaemic control and metabolic profiles between CGM users and those using SMBG were performed using SPSS version 25.

RESULT

A total of 150 patients were included in the study. Overall, the population exhibited poor glycemic control and metabolic profiles, with a mean HbA1c of 9.0%. Additionally, 55% of the patients were overweight or obese. Seventy-one percent had elevated LDL-c levels (>2.6 mmol/L), with a mean LDL-c of 3.2 mmol/L. Of the patients, 24.7% used CGM for glycemic monitoring and had a significantly lower HbA1c (-1.2%) than the SMBG group. The study also demonstrated a significant reduction in HbA1c (-0.8%) after switching to CGM for monitoring. However, no significant differences were observed in BMI or LDL-c levels between the CGM and SMBG groups.

CONCLUSION

This study showed that the use of CGM contributed to better glycemic control in patients with T1DM. However, achieving optimal glycemic control alone is insufficient for effective weight management and improving lipid profiles. Therefore, lifestyle interventions, weight management strategies, and pharmacological treatments for lipid reduction are also necessary.

EP_A204

EFFECTIVENESS AND PERSISTENCE OF GLP-1 RECEPTOR AGONIST TREATMENT AMONG PEOPLE WITH TYPE 2 DIABETES

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INTRODUCTION

People with diabetes (PwD) have increased morbidity, mortality, and healthcare costs. Glucagon-like Peptide-1 Receptor Agonists (GLP-1RA) have revolutionized diabetes management by optimizing weight and glycemic control while providing cardiorenal protection. This study is designed to evaluate the effectiveness of GLP-1RA and identify predictors of treatment persistence among PwD.

METHODOLOGY

This retrospective cohort study at the Universiti Malaya Medical Centre included adult PwD prescribed with GLP-1RA between 2018-2023, excluding those with malignancy or post-bariatric surgery. Data on demographics, anthropometrics, comorbidities, biochemistry, and adverse events were extracted from electronic health records from the initiation of GLP-1RA until the last visit before December 31, 2023. A prescription refill gap of <90 days was classified as the persistent group (PG), while the remainder were categorised as the non-persistent Group (NPG). Generalised Linear Model (GLM) was used to determine factors associated with treatment persistence.

RESULT

Among 470 PwD analysed, the mean age was 59.1 ± 13.0 years, and 54.3% were female with a baseline BMI of 32.3 ± 6.6 kg/m². 91% remained persistent with GLP-1RA. The majority were prescribed injectable semaglutide (55.7%), followed by injectable dulaglutide (27.4%), and oral semaglutide (13.2%). The PG had significantly greater reductions in both weight [-1.9 kg, 95%CI: -5.1,0.1; $p = 0.03$], and BMI [-0.78 kg/m², 95%CI: -1.94,0.04; $p = 0.02$] compared to the NPG. No significant differences were observed in HbA1c or blood lipid levels. Gastrointestinal side effects were more common among the NPG (37.2% vs 15%). Concomitant usage of SGLT2 inhibitors was the strongest predictor of treatment persistence (+16.2 weeks), with lower baseline HbA1c and urine albumin-creatinine ratio also linked to treatment persistence.

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CONCLUSION

Our findings imply that PwD already on SGLT2 inhibitors, those with better glycaemic control and milder proteinuria at baseline are more likely to persist with their GLP-1 RA therapy. Further research incorporating mixed-model analyses and patient perspectives is needed to elucidate the underlying reasons for these associations.

EP_A205

REASSESSING CARBIMAZOLE DOSING STRATEGIES: ASSOCIATION BETWEEN INITIAL DOSE AND SIX-WEEK THYROID FUNCTION

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INTRODUCTION

Carbimazole is a commonly used antithyroid medication for the treatment of hyperthyroidism. Dosage recommendations typically vary based on the severity of biochemical hyperthyroidism, particularly free thyroxine (FT4) levels. However, real-world dosing practices may deviate from guideline-based recommendations, potentially leading to suboptimal outcomes such as persistent hyperthyroidism or iatrogenic hypothyroidism. This study evaluates whether adherence to recommended dosing based on initial FT4 levels is associated with appropriate thyroid function outcomes at six weeks.

METHODOLOGY

We conducted a retrospective observational study involving 125 patients with confirmed hyperthyroidism. Patients were categorized based on whether their initial carbimazole dose was lower than, consistent with, or higher than the recommendations outlined in the American Thyroid

Association guidelines, as determined by their initial FT4 levels. Thyroid function outcomes at 6 weeks were classified as euthyroid, hypothyroid, or persistent hyperthyroid based on repeat thyroid function tests. A chi-square test was performed to evaluate the association between dosing appropriateness and thyroid outcome.

RESULT

The mean age of participants was 48.9 years (SD = 15.0). Based on initial FT4 values, 23.2% were within 1–1.5× upper normal limit (UNL), 23.2% were 1.5–2× UNL, and 53.6% were >2× UNL. Among the 63 patients who received a correct dose, 58.6% became hypothyroid, and 35.0% became euthyroid. In contrast, 52.5% of those given a lower dose achieved euthyroidism, while only 17.2% became hypothyroid. Higher-than-recommended doses resulted in 24.1% hypothyroid outcomes. The chi-square test demonstrated a statistically significant association between dose category and thyroid outcome ($p = 0.003$).

CONCLUSION

Initial carbimazole dosing based on FT4 levels is significantly associated with short-term thyroid outcomes. Interestingly, lower-than-recommended doses were more likely to achieve euthyroidism without excessive hypothyroidism. These findings suggest the need to re-evaluate dosing strategies to optimize early treatment outcomes and reduce the risk of overtreatment in hyperthyroid patients.

EP_A206

A DESCRIPTIVE COST ANALYSIS OF HOSPITALISATIONS AT A DISTRICT HOSPITAL FOLLOWING INSULIN DISCONTINUATION

<https://doi.org/10.15605/jafes.040.S1.214>

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INTRODUCTION

A nationwide shortage of human insulin in Ministry of Health (MOH) facilities has forced primary care clinicians

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to delay initiation, de-intensify, or temporarily discontinue insulin therapy in patients with Type 2 Diabetes (T2D), prioritising oral glucose-lowering drugs. Reduced insulin use may have compromised glycaemic control, increasing the risk of acute complications. This study aims to estimate the direct medical costs of hospitalisations for acute hyperglycaemic events in a district hospital.

METHODOLOGY

Adult patients admitted to Hospital Jempol with acute T2D complications – uncontrolled diabetes (UD), diabetic ketoacidosis (DKA), and hyperosmolar hyperglycaemic state – linked to insulin discontinuation from September 1, 2024, to February 28, 2025, were identified. An activity-based micro-costing approach was applied to quantify resource utilisation through medical records review. Cost components included ward stays, diagnostic procedures, laboratory investigations, pharmaceuticals, and consumables. Unit costs were sourced locally. Mean per-event costs were estimated for each complication type and expressed in 2025 Malayan Ringgits (RM).

RESULT

Twelve patients (mean age \pm SD: 62.8 \pm 8.3) with hospitalisations temporally linked to insulin discontinuations were identified, including 10 UD and 2 DKA cases, with total costs of RM61,877. The median length of stay (LOS) for UD was 4 days (range: 1-11), and it was longer for DKA (6-11 days). The mean cost per UD admission was RM3,637 \pm 2,200 (RM1,763-8,753), while DKA admissions were more costly (RM6,108-19,398). Higher costs are correlated with longer stays. Daily mean costs were RM918 (\pm 309) for UD, and RM1,391 \pm 527 for DKA. Procedures and laboratory investigations were the largest cost drivers (62.3%), followed by ward stays (25.2%), and inpatient drugs/consumables (12.4%).

CONCLUSION

Inpatient management of acute hyperglycaemic events is resource intensive. This study provides unit cost estimates for UD and DKA admissions, which, when combined with nationwide LOS data, can assess the financial impact of the insulin shortage on the MOH.

EP_A207

CLINICAL CONSEQUENCES OF INSULIN DEPRESCRIBING IN TYPE 2 DIABETES: INSIGHTS FROM A DISTRICT HOSPITAL IN MALAYSIA

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INTRODUCTION

Type 2 diabetes (T2D) is characterised by insulin resistance and progressive beta-cell dysfunction, leading to failure on oral glucose-lowering drugs (OGLDs). Insulin deprescribing requires individualisation, considering factors like residual beta-cell function, disease duration and insulin dosage. This study explores the characteristics and outcomes of patients with T2D who had insulin deprescribed at a local health clinic before hospitalisation.

METHODOLOGY

We performed a retrospective medical records review involving adult patients with T2D admitted to Hospital Jempol from September 2024 to April 2025. These patients had their insulin deprescribed within 12 months prior to admission. Basic demographics, Charlson Comorbidity Index (CCI), baseline HbA1c, admission random blood glucose (RBS), interval from insulin discontinuation to admission, length of stay (LOS) in the ward, insulin deprescribing success (OGLDs maintained at discharge) and factors favoring deprescribing were explored.

RESULT

Among 14 patients with a median age of 62.6 years (range: 41-78), 85.7% had a CCI \geq 3. All had OGLDs with insulin (basal bolus 71.4% or premixed 28.6%), with median total daily dose (TDD) of 57 units (range: 28-98) pre-admission. Duration of insulin discontinuation to admission was 3-31 weeks. Median baseline HbA1c was 11.5% (range: 6.4-14%), while median admission RBS was 19.6 mmol/L (range: 6.8 – 31 mmol/L). Infections accounted for 57% of them, in which 5 were complicated (2 DKA, 1 respiratory failure, 1 gram-negative bacteremia, 1 septic shock and intubated). Other indications for admission included symptomatic hyperglycemia (28.6%), decompensated heart failure (7.1%), and hypertensive emergency (7.1%). The

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median LOS was 4 days (range: 1–11). 28.6% transitioned successfully to OGLDs while 71.4% resumed de-intensified insulin regime. Successful deprescribing was noted in older patients (median: 68 vs 64; p -value 0.178), patients with lower baseline HbA1c (median: 8.7 vs 12; p -value 0.288) and higher RBS (median: 20.4 vs 18.2 mmol/L, p -value=1.00).

CONCLUSION

Although statistically insignificant, lower HbA1c may favour deprescribing success. These preliminary trends may inform future studies on safer deprescribing practices to prevent adverse outcomes and hospitalisations.

EP_A208

DIABETES REMISSION POST-BARIATRIC SURGERY: A SABAH PERSPECTIVE

<https://doi.org/10.15605/jafes.040.S1.216>

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INTRODUCTION

Bariatric surgery is not only effective for weight loss but also improves obesity-related complications, including inducing diabetes remission. We aimed to investigate the effects of bariatric surgery on diabetes remission in our centre.

METHODOLOGY

We conducted an observational retrospective study of patients with type 2 diabetes who underwent bariatric surgery (Laparoscopic Sleeve Gastrectomy, Laparoscopic Sleeve Gastrectomy with Proximal Jejunum Bypass, Roux-en-y Gastric Bypass or Mini Gastric Bypass) between March 2022 and February 2024 at Queen Elizabeth Hospital 2. We gathered data on the patients' preoperative weight, body mass index (BMI), HbA1c, antidiabetic medications, diabetes duration, postoperative weight loss and percentage total weight loss (%TWL). Diabetes remission at 1-year post-surgery was defined as having an HbA1c of <6.3% without antidiabetic medications.

RESULT

Thirty-five patients were recruited with mean preoperative weight of 122.0±23.2 kg, BMI of 47.0±7.5 kg/m², HbA1c 7.7±1.7%, and median diabetes duration of 4.38 years (range 0.3–19.9). Average postoperative weight loss at 1 year was 34.7±13.6 kg with mean %TWL of 27.8±7.6%. Diabetes remission was achieved in 17 patients (49%).

Factors significantly associated with remission were shorter diabetes duration (median 1.92 years [IQR: 1–4.5], p <0.001) and absence of insulin use (Crude OR 4.8, 95% CI: 1.1–20.1). No significant associations were found for preoperative HbA1c, BMI, type of surgery, or %TWL. Multivariate analysis identified diabetes duration as the sole independent predictor of remission.

CONCLUSION

Our findings support the effectiveness of bariatric surgery in achieving diabetes remission in patients with obesity, aligning with evidence from the STAMPEDE trial and DiaRem score studies. Shorter diabetes duration emerged as the strongest predictor of remission, while the types of surgery were of comparable benefit. Longer-term follow-up is warranted to assess the durability of remission.

EP_A209

ACUPUNCTURE AS AN ADJUNCT THERAPY FOR INSULIN RESISTANCE IN TYPE 2 DIABETES: A RANDOMIZED CONTROLLED TRIAL

<https://doi.org/10.15605/jafes.040.S1.217>

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INTRODUCTION

Type 2 diabetes (T2D) remains a major global health challenge, including in Malaysia. Pharmacological treatments often face issues such as poor adherence and clinical inertia. This study aimed to evaluate the effects of acupuncture on insulin resistance in patients with T2D

METHODOLOGY

Forty-six patients with T2D were recruited and randomized into either the acupuncture group or the placebo control group. Both groups received 10 sessions of acupuncture therapy using press needles or placebo needles applied to the abdominal area over a period of six weeks, while continuing their standard T2D treatment regimen. Insulin resistance, measured by HOMA-IR, was assessed at baseline and post-intervention. Adverse events were monitored at every visit. The trial adhered to The Consolidated Standards for Reporting of Trials Statement (CONSORT) reporting guideline.

RESULT

The mean age was 55.67 ± 9.41 years, and the mean duration of diabetes was 7.58 ± 5.85 years. Acupuncture significantly reduced insulin resistance by 31.74% (mean HOMA-IR 4.12 ± 1.08) compared to the placebo control group, which

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showed a 12.01% increase (mean HOMA-IR 5.57 ± 1.16) ($p < 0.05$), regardless of baseline adjustments. Within-group analysis also showed a significant reduction in HOMA-IR in the acupuncture group (from 6.08 ± 1.27 to 4.12 ± 1.08 , $p < 0.05$). However, no significant between-group differences were observed in the prevalence of poor HOMA-IR post-intervention, possibly due to the short intervention duration and the shorter needle lengths used. No adverse events were reported, except for one case of mild pain at the needle insertion site.

CONCLUSION

Acupuncture may serve as an effective adjunct therapy for improving insulin resistance in patients with T2D. Future studies with extended treatment duration and longer needles are recommended to validate these findings.

EP_A210

CLINICAL OUTCOMES OF A MULTIMODAL APPROACH COMBINING LOW-CARBOHYDRATE DIET AND PHARMACOTHERAPY FOR OBESITY MANAGEMENT

<https://doi.org/10.15605/jafes.040.S1.218>

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INTRODUCTION

Obesity and metabolic syndrome increase the risk of cardiovascular disease and type 2 diabetes. Effective weight management strategies are essential, including dietary modifications and pharmacologic interventions. This study evaluates the impact of a structured low-carbohydrate diet combined with pharmacologic therapy on metabolic parameters in patients attending the Low-Carb Clinic at Hospital Al-Sultan Abdullah, Universiti Teknologi MARA (UiTM), Puncak Alam.

METHODOLOGY

Forty-six participants (mean age 50 years, BMI 43.68 kg/m^2) attended the clinic for 11.6 months. The intervention involved a low-carbohydrate diet (less than 130 g/day). Pharmacologic treatments included GLP-1 receptor agonists (Ozempic, Saxenda, Rybelsus, Trulicity) and weight loss agents (Duromine, Orlistat) in selected cases. Key assessments included anthropometric indices, glycaemic control, lipid and renal profiles, liver function tests, and blood pressure.

RESULT

The participants' mean age was 50 ± 12 years. 56.5% were female. Thirty-nine patients received GLP-1 receptor agonists; while twelve of them received other weight loss agents (Duromine and Orlistat), and two underwent bariatric surgery. Post-intervention, participants showed significant weight loss (mean -6.49 kg , $p < 0.01$) and BMI reduction (-2.47 kg/m^2 , $p < 0.01$). Central adiposity decreased, including waist (-5.5 cm , $p < 0.01$) and neck circumference (-1.8 cm , $p < 0.01$). HbA1c dropped by 0.35% ($p = 0.05$). ALT decreased (-6.41 mmol/L , $p = 0.011$), indicating improved liver function. LDL increased by 0.36 mmol/L ($p = 0.04$), possibly due to increased fat intake. Fasting glucose, triglycerides, and blood pressure remained unchanged.

CONCLUSION

A low-carbohydrate diet combined with pharmacologic therapy, particularly GLP-1 receptor agonists, significantly improved weight, glycaemic control, and liver function. These findings support combining dietary and pharmacologic strategies for sustainable obesity management.

EP_A211

SUCCESSFUL WEIGHT LOSS POST-BARIATRIC SURGERY: A RETROSPECTIVE STUDY

<https://doi.org/10.15605/jafes.040.S1.219>

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INTRODUCTION

This study aims to determine the percentage of patients who achieved successful weight loss at 12 months post-bariatric surgery. Successful weight loss is defined as achieving at least 50% excess weight loss (EWL) within one to two years.

METHODOLOGY

We conducted a retrospective review on patients who underwent bariatric surgery at Hospital Raja Permaisuri Bainun Ipoh from 2015 to 2024. Patients' data were obtained from laboratory databases and medical records.

RESULT

A total of 298 patients underwent bariatric surgery between 2015 and 2024 in our center. Only 108 patients with weight recorded at month 12 post-operation were included in our

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analysis, with 51.8% (n = 56) receiving sleeve gastrectomy (SG), 42.6% (n = 46) Roux-en-Y gastric bypass (RYGB), and 5.6% (n = 6) sleeve-plus procedure. The mean age was 41.79 years, with 81.5% females. 56.5% had hypertension, 33.6% diabetes mellitus, 38.9% obstructive sleep apnea, and 38% dyslipidaemia. The mean preoperative weight was 122.08 kg and mean BMI was 47.85 kg/m². Overall, 49.1% (n = 53) of patients achieved successful weight loss in 12 months, with percentage distribution as follows: 45.3% had RYGB, 47.2% had SG, and 7.5% had sleeve-plus procedure. At 12 months, 66.7% (n = 4) of sleeve-plus patients, 52.2% (n = 24) of RYGB patients, and 44.6% (n = 25) of SG patients had successful weight loss. However, no significant difference in success rates was observed among the three surgical procedures ($p = 0.484$). No other factor significantly impacted the outcome of successful weight loss.

CONCLUSION

Bariatric surgery effectively facilitates weight loss, with nearly half of patients achieving successful weight loss at 12 months. While the sleeve-plus procedure showed the highest success rate, differences among procedures were not statistically significant.

EP_A212

OUTCOMES OF RADIOACTIVE IODINE THERAPY IN HYPERTHYROID PATIENTS ON EXISTING STEROID THERAPY: A RETROSPECTIVE STUDY

<https://doi.org/10.15605/jafes.040.S1.220>

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INTRODUCTION

Radioactive iodine (RAI) is a common and effective treatment for hyperthyroidism. Corticosteroids are sometimes given as adjunct therapy for hyperthyroidism, or as treatment for thyroid eye disease (TED). Data is lacking on whether pre-existing steroid therapy affects the efficacy of RAI.

METHODOLOGY

We performed a retrospective review of thyrotoxic patients attending our clinic who underwent RAI between Dec 2017 and June 2024 and identified patients who were on corticosteroid therapy prior to RAI. Parameters including age, gender, diagnosis, steroid dosage, fT₄, RAI treatment episodes and remission were evaluated. For patients who underwent multiple RAI treatments, only the first RAI treatment was evaluated. Remission was defined as euthyroid or hypothyroid status at least 6 months post-

RAI, without the need for additional RAI treatment. Chi-squared test was performed to compare the remission rate with or without prior steroids.

RESULT

Four hundred fifty-two patients underwent RAI, 17 were on steroids. For the steroid group, the mean age was 41.8 ± 9.8 years. Graves' was the predominant diagnosis (76.5%). Mean fT₄ pre-RAI was 18.4 ± 9.7 pmol/L, and mean prednisolone dose was 23.8 ± 9.3 mg. Indications for steroids included TED (41.2%), antithyroid drug allergy (17.6%), refractory Graves' (11.8%), agranulocytosis (11.8%), other autoimmune diseases (11.8%) and liver injury (5.9%). Patients with TED were often commenced on steroid therapy for two weeks pre-RAI.

Mean RAI dose was 20.2 ± 4.5 mCi for the steroid group and 17.8 ± 4.3 mCi for the non-steroid group ($p = 0.028$). Remission rate after first RAI treatment was 58.8% in the steroid group vs 73.6% in the non-steroid group. ($\chi^2 = 3.5$, $p = 0.06$)

CONCLUSION

We did not find any statistically significant difference in the post-RAI remission rates between the steroid and non-steroid groups. However, there was a trend towards reduced response to RAI in the steroid group. The study was limited by the small number of patients on steroids, and the difference in RAI doses used between groups. Analysis of a larger number of patients is warranted.

EP_A213

ALDOSTERONE-RENIN RATIO: ASSESSING APPROPRIATENESS IN DIAGNOSTIC PRACTICE

<https://doi.org/10.15605/jafes.040.S1.221>

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INTRODUCTION

Clinical guidelines recommend the aldosterone-renin ratio (ARR) as a screening tool for primary aldosteronism (PA); however, results may be influenced by pre-analytical factors such as posture, timing, salt intake, and medications.

METHODOLOGY

We conducted a retrospective evaluation of ARR requests at University Malaya Medical Centre from August 2022 to August 2024. The study aimed to determine testing indications, review interfering medications, and assess test

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outcomes using data extracted from the laboratory and hospital information systems (LIS and HIS).

RESULT

Out of 882 ARR cases, 428 were reviewed. Excluded cases included those with patients aged over 70 or under 16, tests taken outside UMMC, incomplete data, and patients who chose follow-up elsewhere or underwent 4-hour post-saline suppression tests. The cohort consisted of 47.4% females (203) and 52.6% males (225). Confirmed primary aldosteronism (PA) was identified in 13.1% of ARR cases on two antihypertensives, 9.6% on three, and 10.5% on four or more antihypertensives, compared to 0.9% in those not on treatment. PA was also more frequent among those with adrenal incidentalomas (10.1% vs. 4.3%) and those with hypokalemia (20.0% vs. 1.5%). A total of 333 ARR tests were performed in patients taking concomitant medications known to interfere with ARR results. Additionally, 113 ARR tests (26.4%) were performed without concomitant potassium measurements, among which 4 cases with confirmed PA were identified. While ARR testing is recommended to be performed in the morning, this protocol was not consistently followed in our cohort. Of the 428 ARR tests conducted, 24 (5.6%) were confirmed to have primary aldosteronism (PA).

CONCLUSION

Our findings highlight the variability in ARR testing practices, particularly regarding the timing of tests and the influence of interfering medications. The results underscore the importance of adhering to clinical guidelines to optimize the diagnostic accuracy of ARR testing for primary aldosteronism.

EP_A214

FINDINGS OF GLYCAEMIC CONTROL AND OTHER PARAMETERS AFTER INSULIN DEINTENSIFICATION EXERCISE AMID INSULIN SUPPLY INADEQUACY IN A TERTIARY CARE CENTRE

<https://doi.org/10.15605/jafes.040.S1.222>

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INTRODUCTION

Our country has recently experienced a period of human insulin supply challenges which prompted different centres all over the country to come up with plans to reduce insulin usage while avoiding diabetes management failure.

An assessment after adhering to this plan is necessary to decide the efficacy and safety of such actions.

METHODOLOGY

We reviewed the records of patients with Type 2 Diabetes Mellitus managed under the diabetes clinic Hospital Tengku Ampuan Afzan, Kuantan from July 2024 to November 2024. These patients underwent insulin deintensification based on the Pahang insulin deintensification guideline 2024. Their weight, fasting blood sugar (FBS), HbA1c and total daily insulin dose were recorded at baseline and subsequent follow-up. Data were compared and analysed using Microsoft Excel 2024 and IBM SPSS Statistics Data Editor Version 23.

RESULT

Nine patients were included. They had a median diabetes duration of 12 (1-21) years. Mean HbA1c was 9.6 (± 2.6)% and median total daily insulin dose (TDD) was 28 (10-86) units. After a median follow-up duration of 4 (3-9) months, there was an improvement in mean HbA1c to 8.6 (± 1.6)% while TDD dropped by 42.8%. There was no improvement in FBS. Patients' weight also dropped by 4%. None of the patients were admitted for diabetic ketoacidosis but two of them had episodes of mild hypoglycaemia after initiation of sulphonylureas.

CONCLUSION

While insulin remains the most potent treatment in resolving hyperglycaemia in Type 2 DM, in some patients, there is always a risk of over-insulinisation which may sometimes impair their supply. Despite being limited due to small sample size and short duration of follow-up, these findings provide a glimpse into the potential benefits in glycaemic control by optimising use of oral glucose lowering drugs subsequently reducing the reliance on insulin. However, cautious use of sulphonylurea is vital as some patients may still have normal insulin reserves.

Paediatrics Oral Presentation

OP_P001

PREVALENCE AND FACTORS ASSOCIATED WITH STUNTING AND FALTERING GROWTH AMONG INFANTS WITH VERY LOW-BIRTHWEIGHT AGED 16-24 MONTHS AT A TERTIARY CENTER

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INTRODUCTION

There is limited study in Malaysia on the prevalence of faltering growth and stunting among babies with very low birth weight (VLBW) during the first 24 months of life. This study aimed to evaluate the prevalence of faltering growth and stunting in infants with VLBW after NICU discharge and identify factors related to post-discharge faltering growth and stunting.

METHODOLOGY

We conducted a retrospective study at a tertiary center in Selangor among infants with VLBW who completed three follow-up consults at corrected age intervals (CA) of 3 to <9 months, 9 to <16 months, and 16-24 months old.

RESULT

The prevalence of post-discharge faltering growth was 39.6%, 31.8%, and 24.8% at the respective age intervals. At CA 16 to 24 months, the prevalence of stunting was 23.2%. The percentage of infants with faltering growth who experienced stunting at CA 3 to <9 months rose from 51% to 70% at CA 16 to 24 months. Small for gestational age (SGA), extremely low birth weight (ELBW), male gender, faltering growth at discharge, and length of stay >66 days (LOS) were all significant risk factors for faltering growth at all age intervals. Infants on steroids for chronic lung disease and intraventricular hemorrhage had an increased risk of faltering growth only at CA 3 to <9 months old. Among these risk factors, SGA, ELBW and male gender were identified as predictors for both faltering growth and stunting at 16-24 months of age. Maternal characteristics did not show significant association with post-discharge faltering growth and stunting.

CONCLUSION

Most of the infants who had faltering growth also experienced stunting at 16 to 24 months old. This study highlights the importance of adequate nutrition early on targeting at-risk groups to prevent faltering growth and reduce the likelihood of stunting in infants with VLBW.

Paediatrics Oral Presentation

OP_P002

PREVALENCE OF GROWTH HORMONE DEFICIENCY (GHD) USING TWO DIAGNOSTIC CUT-OFFS IN UNIVERSITY MALAYA MEDICAL CENTRE (UMMC)

<https://doi.org/10.15605/jafes.040.S1.224>

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INTRODUCTION

Growth hormone deficiency (GHD) in children is diagnosed in stunting and confirmed with two stimulation tests showing low growth hormone (GH) levels. Internationally, a peak GH level <7 ng/mL is used, while Malaysia's 2010 guideline defines GHD as <10 ng/mL. This study aims to determine the prevalence of GHD using both thresholds in children undergoing insulin tolerance test (ITT) or glucagon stimulation test (GST).

METHODOLOGY

This retrospective cross-sectional study was conducted over five years (2020–2024). Data on demographics and GH stimulation results were collected from children with stunting who underwent ITT or GST. The ITT dose was 0.1–0.15 U/kg based on pubertal status, while GST was administered at 0.3 mcg/kg (max 1 mg).

RESULT

In a cohort of 133 children undergone GH stimulation testing, 103 had ITT and 30 GST. The mean age at diagnosis was 10.4 ± 2.1 years (girls: 10.2 ± 2.0 , boys: 10.6 ± 2.1). Mean height was 125.6 ± 14.3 cm for girls and 125.97 ± 11.63 cm for boys. The mean ITT dose was 0.11 U/kg. Using a GH cut-off of <7 ng/mL, 48% (n = 64) were diagnosed with GHD; this increased to 67% (n = 89) using a <10 ng/mL threshold. An additional 23.3% (n = 31) had GH levels between 10–20 ng/mL, while only 9.7% (n = 13) had GH response (>20 ng/mL). Most patients were not primed, as they either presented early (<9 years) or had already entered puberty.

CONCLUSION

There is an increase in the number of GHD cases when the cut-off point is made at 10 ng/mL. This finding highlights the diagnostic impact of threshold selection. While the lower cut-off identifies more severe cases, the higher threshold captures an additional 25 children, nearly one in five, who may otherwise be overlooked. This emphasizes that GH secretion exists on a continuum, and a single rigid cut-off may underestimate clinically relevant cases.

Paediatrics Oral Presentation

OP_P003

A REVIEW OF CONGENITAL HYPOTHYROIDISM AND ROLE OF THYROID SCINTIGRAPHY IN UNIVERSITY MALAYA MEDICAL CENTRE (UMMC)

<https://doi.org/10.15605/jafes.040.S1.225>

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INTRODUCTION

Thyroid scintigraphy (TS) is the gold standard investigation to determine the underlying cause of congenital hypothyroidism (CHT), which can be due to thyroid dysgenesis (agenesis/hypoplasia/ ectopic), dysmorphogenesis (TDH) or transient hypothyroidism (TH). In Malaysia, thyroid scan is done at the age of 3 years old. Some centres may opt to do ultrasound instead.

METHODOLOGY

This is a retrospective study from 2020 to 2023. TS in UMMC uses radioactive tracer technetium-99m. All patients suspected of having congenital hypothyroidism (CHT) or neonatal-onset hypothyroidism were subjected to TS and reassessment of thyroid function (TFT) at the age of 3 years. Children with abnormal TS and TFT were diagnosed with thyroid dysgenesis. Those with normal TS but abnormal TFT were diagnosed with thyroid dysmorphogenesis (TDH), and those with normal TS and TFT were regarded as having TH. Data pertaining to sociodemographic factors, biochemical markers and treatment were reviewed.

RESULT

A total of 120 patients underwent TS during the study period. Nine patients had incomplete data. Amongst the 111 children, 58 (52.3%) were females and majority were Malays (n = 71, 64%). Seventeen (14.7%) had Down syndrome and 7 (6%) were born premature. TS confirmed normal findings in 103 (92.8%) cases, 3 (2.7%) had ectopic thyroid, 3 (2.7%) had thyroid agenesis, 2 (1.8%) had thyroid dysgenesis/hypoplasia. Among those with normal TS, 12 (11.7%) were diagnosed with TDH and the rest had TH (n = 91, 88.3%). The mean cord TSH were 71.3 mIU/L, 104.3 mIU/L and 17.1 mIU/L respectively for those with thyroid dysgenesis, ectopic thyroid and TDH. Among those who started on thyroxine late after the age of 3 months old, 11/14(78.6%) of them were subclinical/transient causes of hypothyroidism. Two (14.3%) had TDH, while 1/14 (7.1%) had ectopic thyroid.

CONCLUSION

Transient hypothyroidism (TH) is highly prevalent in our population. Thyroid scan is important in determining TH, hence reducing prolonged and unnecessary treatment. Alternative investigation using ultrasound may not be able to detect an ectopic or hypoplastic thyroid gland.

Paediatrics Oral Presentation

OP_P004

ASSOCIATION OF ANTHROPOMETRIC AND BIOELECTRICAL IMPEDANCE ANALYSIS MEASUREMENTS WITH METABOLIC PROFILES IN OVERWEIGHT AND OBESE CHILDREN

<https://doi.org/10.15605/jafes.040.S1.226>

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INTRODUCTION

Childhood obesity has become a global epidemic. Waist circumference (WC) measures abdominal fat but cannot distinguish between subcutaneous and visceral fat. Bioelectrical impedance analysis (BIA) measures visceral fat area (VFA) and percentage body fat (PBF), but its effectiveness in children is unclear. We aim to evaluate the relationship of anthropometric and BIA measurements with the metabolic profiles of overweight and obese children.

METHODOLOGY

Eighty-two overweight and obese children aged 7 to 18 years were recruited over a one-year period at the Paediatric Endocrine Clinic. Sociodemographic and lifestyle factors were collected. Anthropometric measurements i.e., body mass index (BMI), waist circumference (WC), and waist-to-height ratio (WHR) were taken. BIA measurements (PBF and VFA) were also performed using Inbody S10 (BioSpace, Seoul, Korea). The relationship of anthropometric and BIA parameters with obesity-related clinical factors i.e., blood pressure (BP), fasting blood sugar, HOMA- index, HbA1c and fasting lipid profile were evaluated.

RESULT

The majority of the patients were female 43/82 (52.4%) with mean age of 11.21±2.70 years. The median BMI z-score was 1.95 (1.71, 2.34). The predominant ethnicity was Malay 72/82 (87.8%) and 57/82 (69.5%) of mothers completed tertiary education. 47/82 (57.3%) patients engaged in physical activity less than three days per week and the majority (82.9%) had more than one hour of recreational screen time.

On multiple linear regression of the association of anthropometric and BIA measurements with metabolic profiles, only WC had significant positive association with systolic BP ($\beta = 0.33$, 95% CI 0.167-0.498, $p < 0.001$) while VFA had a positive association with diastolic BP ($\beta = 0.058$, 95% CI 0.024-0.092, $p = 0.001$). Only VFA was positively associated with HOMA index ($\beta = 0.011$, 95% CI 0.002-0.021, $p = 0.02$). WC was associated with higher TG when adjusted for PBF ($\beta = 0.015$, 95% CI 0.005-0.025, $p = 0.004$).

CONCLUSION

While VFA measurement by BIA was better than BMI measurement, it was not superior to WC in predicting hypertension, insulin resistance, and hypertriglyceridemia in overweight and obese children.

Paediatrics Best Poster Presentation

BP_P001

FACTORS AFFECTING GLYCEMIC CONTROL AMONG PAEDIATRIC AND ADOLESCENT PATIENTS WITH TYPE 1 DIABETES: EXPERIENCE FROM A TERTIARY HOSPITAL IN SARAWAK

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INTRODUCTION/BACKGROUND

HbA1c is one of the primary tools to assess glycemic control in diabetes. This study aims to determine factors affecting glycemic control among children and adolescents with type 1 diabetes (T1DM) in Sarawak General Hospital (SGH).

METHODOLOGY

We conducted a retrospective cross-sectional study among patients with T1DM aged 18 years and below who attended the Paediatric Endocrine Clinic, SGH for the year 2024. Data analysis was performed with SPSS version 25. The effects of different factors on glycemic control were analysed using t-test and Multi-factorial Repeated Measures ANOVA test.

RESULT

A total of 21 T1DM youths were recruited, with a mean age of 12.14 ± 4.37 years and mean disease duration of 4.55 ± 4.02 years. The patients were predominantly female and secondary school adolescents. More than 60% of the patients who were initially using fixed-dose basal-bolus insulin were switched to carbohydrate-based basal-bolus insulin regimen for the past year. The mean HbA1c had improved from $10.0 \pm 2.5\%$ on the former regimen to $9.6 \pm 2.2\%$ on the latter regimen ($p < 0.05$). Patients with lower maternal education ($10.3 \pm 1.9\%$ vs $7.7 \pm 1.8\%$), moderate and poor adherence to insulin ($9.9 \pm 0.5\%$ vs $9.4 \pm 0.6\%$) and those on fixed-dose basal-bolus insulin regimen ($9.9 \pm 1.2\%$ vs $9.0 \pm 1.2\%$) had higher recent HbA1c as compared to the initial HbA1c reading in year 2024; although the results were not statistically significant likely due to the small sample size.

CONCLUSION

Glycemic control among children and adolescents with T1DM is still suboptimal. Improving diabetes education is essential with special attention to parents with limited education and patients with medication adherence issues.

Paediatrics Best Poster Presentation

BP_P002

RESPONSE TO PAMIDRONATE THERAPY AND PHARMACOGENETICS IN PATIENTS WITH OSTEOGENESIS IMPERFECTA

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INTRODUCTION/BACKGROUND

Osteogenesis imperfecta (OI), is a genetically heterogeneous connective tissue disorder associated with skeletal fragility, deformity and growth deficiency. Intravenous bisphosphonate therapy is the mainstay of medical treatment of this condition. Due to the scarcity of regional data, this study aimed to investigate the genetic profile and clinical response to bisphosphonate therapy in a cohort of Malaysian patients.

METHODOLOGY

Genetic analysis was performed on 14 children (6 females, 8 males) with OI at Hospital Putrajaya. Three children with Type I and eleven with Type III OI. All patients received bisphosphonate therapy. Clinical, biochemical and radiological data were gathered prior to initiation of treatment and at subsequent intervals during treatment. Targeted gene sequencing using the Ion AmpliSeq platform on the Ion Torrent™ system identified genetic mutations which were validated using Sanger sequencing. In silico analysis evaluated their potential impact at the protein level.

RESULT

All patients had involvement of long bone fractures, with the addition of thoracic or lumbar vertebrae involvement in 50% of patients. A positive family history was noted in 28% of patients. Bisphosphonate therapy was started at a median age of 4 [3.1 - 10.7] years with 42.8% of patients starting treatment before the age of 2. The median duration of treatment was 6.4 (2.8-14.9) years. All patients showed a significant reduction in fracture rate while on intravenous bisphosphonate therapy, with annual fracture rates decreasing from 2.4 to 0.8 fractures per year on average. Additionally, all patients reported an improvement in bone pain. There was no significant difference in fracture rate between COL1A1 and COL1A2 positive patients.

CONCLUSION

This study highlights the genetic heterogeneity of OI patients with COL1A1 and COL1A2 in the Malaysian population and supports the efficacy of pamidronate therapy in improving skeletal outcomes.

Paediatrics Best Poster Presentation

BP_P003

PREVALENCE AND FACTORS ASSOCIATED WITH THYROID DISEASE AMONG CHILDREN WITH DOWN SYNDROME IN TWO TERTIARY HOSPITALS

<https://doi.org/10.15605/jafes.040.S1.229>

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INTRODUCTION/BACKGROUND

Children with Down syndrome (DS) have an increased risk of thyroid disease (TD). The study aimed to evaluate the prevalence and profile of TD among children with DS, and examine the association between demographic, clinical, genetic, maternal factors, and TD.

METHODOLOGY

We conducted a retrospective cohort study among patients with DS, aged 0-18 years, under paediatric follow-up at the two hospitals. We retrieved their data from the hospital electronic medical records.

RESULT

There were 299 children (150 boys, 149 girls) with DS with mean age of 8.8 years \pm 4.66 years. The majority were Malays (254/299, 84.9%). TD was detected in 49.8% (149/299). Of the 149 patients with TD, 75.8% (113/149) had congenital hypothyroidism (CH) diagnosed within age 6 months. Acquired hypothyroidism (AH), diagnosed after age 6 months, were detected in 22.8% (34/149), only 2 (1.3%) had hyperthyroidism. Subclinical hypothyroidism was the most common TD, detected in 105/113 (92.9%) of CH and 27/34 (79.4%) of AH. Almost half (74/149, 49.7%) were diagnosed in the neonatal period, another 32.9% (49/149) during infancy. Congenital cardiovascular anomalies were present in 221/299 (73.9%) patients. There were significant associations between cardiovascular anomalies ($p = 0.038$), congenital skeletal anomalies ($p = 0.038$) and TD. There were no significant associations between age, gender, ethnicity, birth weight, chromosomal abnormality, maternal age and parity, and TD.

CONCLUSION

TD is prevalent among children with Down syndrome, with subclinical hypothyroidism as the most common TD. These findings highlight the importance of ongoing surveillance and biochemical screening for early detection of TD in children with DS.

Paediatrics Best Poster Presentation

BP_P004

SAFETY AND EFFICACY OF LIRAGLUTIDE IN A SMALL PEDIATRIC OBESITY COHORT

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INTRODUCTION/BACKGROUND

Childhood obesity is a rising health concern across the globe, with the risk of persisting in adulthood. Lifestyle modification remains the cornerstone of first-line management; however, achieving favourable weight or BMI outcomes remains challenging. The use of liraglutide, a glucagon-like-peptide 1 receptor agonist, has been shown to be safe and effective in children. This study describes the use of liraglutide in six paediatric patients with obesity, examining its effects on weight, BMI, and associated side effects.

METHODOLOGY

Data of six patients who were started on liraglutide in 2024 in the Paediatric Obesity Clinic were obtained through the hospital electronic medical records. Clinical and anthropometry details at the start of liraglutide and at the last clinic visit were recorded. Associated side effects of the medication were asked post-initiation of liraglutide.

RESULT

All six patients were males and extremely obese (BMI above 120% of 95th percentile). Patients were between 12.5 to 17.8 years old (median 15.5 years). Weight ranged from 75.8 kg to 115.2kg (z-score 1.99 to 2.95) with median 101.9 kg. BMI ranged from 29.98 kg/m² to 40 kg/m² (z-score 2.14 to 2.67) with a median 35.8 kg/m². Patients were followed up for a minimum of 2 months and a maximum of 9 months (median 5 months). The median and percentage for weight and BMI reduction were -3.7 kg (4%) and -2.36 kg/m² (7%), respectively. Half of the patients were on a full dose of liraglutide (3 mg). No weight gain was observed, although 2 patients' weight did not differ remarkably at the last visit. Highest weight and BMI reduction were -9.3 kg and -3.48 kg/m² respectively, seen in 2-months period in a 15.5-year-old male on 1.8 mg daily dose of liraglutide. Half of the patients experienced nausea, abdominal cramps and bloating.

CONCLUSION

The use of liraglutide in this small cohort showed some positive evidence to treat paediatric patients with extreme obese. However, the long-term sustenance of maintaining favourable weight and BMI needs to be examined in the future.

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EP_P001

COMPARING BEHAVIOURAL PROBLEMS AMONG OBESE AND NON-OBESE CHILDREN IN HOSPITAL RAJA PEREMPUAN ZAINAB II, KOTA BHARU

<https://doi.org/10.15605/jafes.040.S1.231>

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INTRODUCTION

Childhood obesity is an increasing public health concern, both globally and in Malaysia. Beyond physical health risks, it has been associated with behavioural and psychological issues such as anxiety, depression, aggression, and low self-esteem. However, this association remains underexplored in the Malaysian context. This study aims to compare behavioural problems between obese and non-obese children at Hospital Raja Perempuan Zainab II, Kota Bharu, and assess their competency characteristics.

METHODOLOGY

A comparative cross-sectional study was conducted from September to December 2024 at the general paediatric clinic and ward of Hospital Raja Perempuan Zainab II, Kota Bharu. Participants were children aged 6–12 years, categorized as obese (BMI $\geq 95^{\text{th}}$ percentile) or non-obese (BMI $< 95^{\text{th}}$ percentile) using CDC BMI-for-age percentiles. Behavioural problems were assessed using the validated Child Behavior Checklist (CBCL/6–18), which measures internalizing, externalizing, and total behavioural problems. T-scores classified behavioural concerns as normal, borderline, or clinical range.

RESULT

A total of 85 participants (44 obese, 41 non-obese) completed the study. We found significant associations between BMI status and hobby participation ($p = 0.016$), number of friends ($p = 0.001$), and school performance ($p = 0.010$). However, we did not find any statistically significant differences in behavioural domains, including internalizing ($p = 0.781$), externalizing ($p = 0.131$), social ($p = 0.344$), thought ($p = 0.108$), attention ($p = 0.341$), and total problems ($p = 0.085$).

CONCLUSION

We did not find any significant behavioural differences between groups. However, with a study power of 52%, the results may reflect a limited sample size rather than an absence of association. We recommend larger-scale studies with refined methodologies.

EP_P002

METABOLIC BONE DISEASE OF PREMATURITY – SURVEY OF CURRENT NEONATAL INTENSIVE CARE APPROACHES

<https://doi.org/10.15605/jafes.040.S1.232>

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INTRODUCTION

Metabolic bone disease of prematurity (MBDP) requires both calcium and phosphate for prevention and treatment. However, clinicians often focus on phosphate and vitamin D supplementation, neglecting calcium deficiency in nutrition. This study evaluates MBDP management in the neonatal care unit in a quaternary hospital in Malaysia.

METHODOLOGY

We conducted a retrospective review of MBDP cases referred to paediatric endocrinologists at University Malaya Medical Centre from 2019 to 2025. We extracted data from electronic medical records and monitoring charts, and we obtained input from pharmacists and dietitians regarding mineral supplementation in enteral and parenteral nutrition.

RESULT

The study involved 22 subjects, primarily infants with extremely low birth weight (ELBW), with a median birth weight of 705 grams (interquartile range: 600–833 grams). All infants were born before 32 weeks of gestation, and most were born before 28 weeks. The mean age at referral was 66.3 ± 33.43 days. Upon referral, all subjects exhibited low phosphate and high alkaline phosphatase levels. Only 15 subjects had their parathyroid hormone (PTH) levels checked, and PTH testing was conducted more frequently after referrals to endocrinology. Fifteen subjects showed radiological evidence of MBDP, and six of them had fractures. Routine screening revealed hypophosphatemia, hyperphosphatasia, and normocalcemia at least one month prior to referral. Most subjects were presumed to be treated with oral phosphate and vitamin D supplements; however,

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many later showed elevated PTH levels, suggesting secondary hyperparathyroidism. None of the subjects were found to be vitamin D deficient. Prolonged fasting was identified as a significant risk factor for severe MBDP. Most subjects received low calcium levels alongside relatively high phosphate in parenteral nutrition. Less than 25% of the infants received Human Milk Fortifier (HMF) despite being primarily breastfed. Only two subjects received calcium supplements.

CONCLUSION

The study highlights gaps in understanding mineral supplementation in MBDP and the underutilization of PTH screening. Routine phosphate supplementation without addressing calcium deficiency worsens secondary hyperparathyroidism and MBDP. The study recommends routine HMF usage, earlier PTH screening, and standardized guidelines to improve MBDP management.

EP_P003

HEALTH SCREENING ANALYSIS OF HIGH-RISK PRIMARY SCHOOL STUDENTS OF SK SEKSYEN 7, BANGI

<https://doi.org/10.15605/jafes.040.S1.233>

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INTRODUCTION

Childhood obesity and metabolic disorders are growing concerns globally. This study examines the health status of primary school children in SK Seksyen 7, Bangi.

METHODOLOGY

We conducted a cross-sectional study on 116 high-risk primary school students (aged 10 to 12 years old). Anthropometric measurements (weight, height, BMI, waist circumference) and physiological parameters (blood pressure, blood glucose) were recorded. We also performed BMI classification and assessed their metabolic risk.

RESULT

Among the students, 78% were classified as obese, 13% were overweight, and only 9% had a normal body mass index (BMI). The glycemic results indicated that 97% of the students had normal glucose levels, while 3% were prediabetic. Additionally, only 20% of the students had a normal waist circumference measurement.

CONCLUSION

The findings highlight the importance of routine health screenings to detect early endocrine disorders in children. Early intervention strategies, including lifestyle modifications, are essential to prevent future metabolic complications.

EP_P004

VALIDATION OF DATA QUALITY IN THE MALAYSIAN PATIENT REGISTRY INFORMATION SYSTEM FOR TYPE I PAEDIATRIC DIABETES CASES

<https://doi.org/10.15605/jafes.040.S1.234>

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INTRODUCTION

Type 1 Diabetes Mellitus (T1DM) is a significant chronic condition in children, yet Malaysia lacks comprehensive population-based data on its prevalence and clinical features. To address this, the Patient Registry Information System – Non-Communicable Disease (PRIS-NCD) was developed within the Malaysian Health Data Warehouse (MyHDW) to enable longitudinal data collection. This study aimed to evaluate the validity of PRIS-NCD data following pilot implementation in a national paediatric referral centre.

METHODOLOGY

One hundred twenty paediatric patients with T1DM under follow-up in Hospital Putrajaya were retrospectively notified into the PRIS-NCD registry. We assessed data validity by comparing 47 variables between registry entries and abstracted electronic medical records (EMR). The analysis focused on exact agreement rates and missing data percentages to determine concordance and completeness.

RESULT

Of 120 cases, 115 were included in the analysis. The mean exact agreement between the registry and EMR data was 95.4% at diagnosis and 94.7% at follow-up. Most variables showed agreement rates exceeding 90%, except for BMI at diagnosis (86.8%), insulin test at diagnosis (88.6%), and microalbuminuria at follow-up (68.3%). Missing data were generally low in both datasets, with registry data showing slightly fewer missing values compared to EMR data despite being a secondary source.

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CONCLUSION

The PRIS-NCD registry demonstrated high validity in recording T1DM patient data, supporting its use for epidemiological insights and healthcare planning. Rigorous data entry procedures likely aided high agreement rates. However, a broader evaluation of completeness, comparability, and timeliness will require nationwide implementation and real-time data capture. We recommend continued improvements in data entry practices and user education to sustain and enhance data quality.

EP_P005

PAIN ASSESSMENT AMONGST CHILDREN TREATED WITH DEEP INTRAMUSCULAR INJECTION OF GnRHa IN UMMC

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INTRODUCTION

Trained nurses in our clinic routinely administer deep intramuscular injections of gonadotropin agonist (IM GnRHa). Before the injections, we offer patients options for pain relief prophylaxis. However, we have not regularly assessed the type of pain or the effectiveness of pain relief prophylaxis.

METHODOLOGY

This cross-sectional study was conducted from January to March 2025, involving all children who received IM GnRHa in the paediatric endocrine clinic at UMMC. The Wong-Baker pain scale, ranging from 0 to 10 (0 indicates no pain and 10 represents the most severe pain), was used to assess pain levels. The pain relief options offered included EMLA cream, ethyl chloride spray, ice packs, or any combination of these methods, based on patient preference. Cases were categorized as follows: New cases involved children who received three injections or less; intermediate cases involved those who received between four and nine injections; and long-term cases comprised those who received ten injections or more.

RESULT

A total of 80 children received injections during the study period. The majority were female, 73 (91%). The mean age was 10.0 ± 1.41 years. There were 24 (30%) new cases, 24

(30%) intermediate cases, and 32 (40%) chronic or long-term cases. The mean pain scores were 3.37 ± 2.44 for new cases, 3.25 ± 1.62 for intermediate cases, and 2.62 ± 1.64 for chronic/long-term cases. The majority (68 or 85%) of patients chose a combination of EMLA and ethyl chloride spray. Other preferences included the spray alone (8 patients, or 10%), EMLA cream only (3 patients, or 3.75%), and EMLA cream with ice packs (1 patient, or 1.3%). Notably, only two patients in the chronic/long-term category opted for single therapy and reported a higher mean pain score of 5.

CONCLUSION

We conclude that the more injections the patients received, the less pain they experienced, and that combination prophylaxis appears to be most beneficial. Further randomized studies are needed to determine the best pain relief method for children undergoing deep intramuscular injections in a clinical setting.

EP_P006

SECULAR TRENDS IN THE DIAGNOSIS AND MANAGEMENT OF TURNER SYNDROME: A SINGLE CENTRE 20-YEAR EXPERIENCE

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INTRODUCTION

Turner syndrome (TS) is a sex chromosome disorder with one intact X chromosome and complete or partial absence of the second chromosome. It often involves multiple organ systems, predominantly various endocrinopathies, through all stages of life.

METHODOLOGY

We performed a descriptive cross-sectional study in the Pediatric Endocrinology Unit Hospital Putrajaya. We retrieved records of all patients with TS managed in our unit between January 2005 and March 2025 from the electronic database system.

RESULT

Over the past two decades, our unit has treated 72 patients with TS, with monosomy X being the most common karyotype abnormality, affecting 40 patients.

In the first decade, from 2006 to 2015, the median age at diagnosis was 5 years (IQR: 0.2 – 10.7 years). 63.3% (n = 19) were diagnosed during childhood, with short stature being

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the most common presenting complaint. The median age of referral to our unit was 9.5 years (IQR: 4.8 – 12.1). 83% (n = 25) had received recombinant growth hormone treatment (rhGH), and the median age at initiation of rhGH therapy was 11.1 years (IQR: 7.0 – 13.3). The median age of pubertal induction was 14.6 years (IQR: 13.1 – 15.3).

In contrast, during the second decade, from 2016 to 2025, patients were diagnosed earlier, with a median age of 2.6 years (IQR: 0.2 – 10.6). Notably, 54.7% (n = 23) were diagnosed antenatally or during infancy due to typical TS features. However, the median age at referral was 7.7 years (IQR: 4.0 – 11.6). During this period, 45.2% (n = 19) began rhGH treatment, with the median age for initiation at 9.0 years (IQR: 5.7 – 11.3). The median age for pubertal induction was 13.8 years (IQR: 13.6 – 14.8).

All the patients underwent complete screening for associated abnormalities.

CONCLUSION

Referrals to a paediatric endocrinologist for Turner Syndrome are often delayed due to a lack of awareness of its various endocrinopathies. Early recognition of its salient features and prompt referral allows for timely intervention and management, predominantly growth hormone and sex hormone treatment, ultimately improving quality of life.

EP_P007

PREVALENCE AND FACTORS ASSOCIATED WITH THYROID DYSFUNCTION AMONG PREMATURE BABIES IN A SELECTED TERTIARY CENTRE IN MALAYSIA

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INTRODUCTION

Premature babies have a higher risk of developing thyroid dysfunctions due to immaturity of hypothalamic-pituitary-thyroid (HPT) axis. Rescreening of thyroid function is recommended in Malaysia among preterm babies ≤34 weeks gestation since 2022 to improve detection of thyroid dysfunction with delayed TSH rise. This study aims to analyse the prevalence and factors associated with thyroid dysfunction in preterm babies ≤34 weeks gestation and to evaluate its progression and outcome.

METHODOLOGY

A retrospective study was performed among premature babies ≤34 weeks of gestation born between January 2019 until August 2024 in a selected NICU in Malaysia. Infants who had at least one repeated thyroid function test (TFT) after birth were included in the study. Data on the demographic factors and clinical characteristics were collected from the medical records. The TFT of the study population and its progression were analysed.

RESULT

There were 14% (46/320) infants with thyroid dysfunction. The majority of infants with thyroid dysfunction had subclinical hypothyroidism 84.7% (39/46), followed by thyroid hypothyroxinemia of prematurity (THOP) 8.7% (4/46) and primary hypothyroidism 6.5% (3/46). Out of the 46 patients with thyroid dysfunction, 18/46 (39.1%) were detected at <2 weeks of life, 20/46 (43.5%) were detected at 2-4 weeks old and 8/46 (17.4%) were detected after 4 weeks old. In the evaluation of factors associated with thyroid dysfunction, only small for gestational age (SGA) was significantly associated with thyroid dysfunction compared to infants without SGA (28.2% vs 14.2%, $p = 0.017$). Only 15/46 (32.6%) of infants with thyroid dysfunction required levothyroxine replacement, all of whom had primary and subclinical hypothyroidism. All infants with THOP had spontaneous resolution of thyroid dysfunction without treatment.

CONCLUSION

The prevalence of thyroid dysfunction in preterm babies ≤34 weeks was 14%. The majority were detected between 2-4 weeks old. SGA was significantly associated with thyroid dysfunction in this study population.

EP_P008

EVALUATING OUTCOMES OF CHILDHOOD OBESITY MANAGEMENT: A 2-YEAR FOLLOW-UP STUDY

<https://doi.org/10.15605/jafes.040.S1.238>

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INTRODUCTION

Childhood obesity (CO) clinic has served as screening and intervention center. Weight management programs in

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children have not been fully established. This report is to understand the outcome after 2 years of follow-up in the same clinic.

METHODOLOGY

Children who were newly referred to the clinic from 2020-2023 (4 years) were identified. Patient demographic and anthropometric (weight, height, BMI, blood pressure and waist circumference) data at point 0 (first visit) and point 1 (2 years from the first clinic visit) were collected.

RESULT

A total of 78 new patients were included. Majority, 51 (65%), were males. A total of 27 (35%) did not come back for their second follow-up. Another 22 (28%) defaulted 2 years before. Only 29 (37%) completed follow-up for 2 years. At time point 0, the overall mean age was 12.1 ± 3.05 years with 11.96 ± 2.9 and 12.16 ± 3.07 for females and males, respectively. The mean height, weight and BMI were 143.6 ± 23.6 cm, 70.46 ± 22.75 kg and 30.3 ± 6.9 kg/m² for females and 150.66 ± 20.4 cm, 70.95 ± 23.39 kg and 30.0 ± 5.0 kg/m² for males. At time point 1, the mean age for females was 13.45 ± 4.1 years-old and 14.2 ± 3.1 for males. The mean height and weight were 150.0 ± 19.3 cm and 76.8 ± 21.5 kg for females and 163.4 ± 19.6 cm and 83.7 ± 26.1 kg for males. The mean BMI were 32.4 ± 5.59 kg/m² and 31.3 ± 6.1 kg/m² for females and males, respectively. Amongst those completed 2 years follow-up, 6/29 (20.6%) had weight loss and their mean weight and BMI losses were -6.8 kg and 2.745 kg/m². Amongst those who gained weight, the BMI gain was 2.165 kg/m². Six developed hypertension and 4 were diagnosed with pre-diabetes while under follow-up.

CONCLUSION

The expectation of weight loss while attending CO clinic may be overestimated. In the real-world data, the majority would fail to lose weight further and may develop other complications instead. Weight loss programs dedicated for children are needed to help these high-risk populations.

EP_P009

THYROID FUNCTION ABNORMALITIES IN PRETERM INFANTS: A COHORT STUDY IN A CHILDREN'S HOSPITAL

<https://doi.org/10.15605/jafes.040.S1.239>

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INTRODUCTION

Congenital hypothyroidism (CH) is a significant condition included in our national newborn screening programs based on raised cord thyroid stimulating hormone (TSH). However, in preterm infants, initial screening may miss elevated TSH. Hence, re-screening is recommended in most CH screening guidelines. This study aims to evaluate thyroid function abnormalities in preterm infants taken during re-screening.

METHODOLOGY

This is a retrospective study. All preterm infants admitted to our neonatal intensive care unit (NICU) between June 1, 2024 to February 1, 2025, with at least one thyroid function test (TFT) done will be included. Clinical parameters were extracted from the department's electronic medical record, and TFTs were retrieved from electronic laboratory records. The TFTs, including TSH and free thyroxine (fT4), were performed according to our NICU protocol, whereby:

- Infants <32 weeks: initial TFT at 4 weeks postnatally, repeated fortnightly.
- Infants ≥32 weeks: initial TFT at 36 weeks corrected age, repeated biweekly.

RESULT

There were 5,561 live births during the study period, of which 190 were preterm. A total of 120 preterm infants (55% male and 44% female) had at least one TFT done, of which 25 infants (20.8%) had abnormal TFTs. Transient hyperthyrotropinemia was the most common abnormality (15.8%), followed by transient hypothyroxinemia (3.3%). One case of primary hypothyroidism (1.7%) was diagnosed at the postnatal age of 40 weeks and required thyroxine treatment, giving rise to CH incidence of 1:120 in this cohort.

CONCLUSION

The majority of preterm infants with abnormal thyroid function had transient conditions and did not warrant treatment. Our CH incidence among preterm infants is high, which may be due to a smaller cohort. Our findings support the ongoing re-screening for CH in preterm infants. However, TFTs should be interpreted with caution to avoid over-treatment of transient thyroid dysfunction.

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EP_P010

A DESCRIPTIVE ANALYSIS OF CHILDREN WITH GRAVES' DISEASE ATTENDING PAEDIATRIC ENDOCRINE CLINIC, SARAWAK GENERAL HOSPITAL

<https://doi.org/10.15605/jafes.040.S1.240>

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INTRODUCTION

Graves' Disease (GD) remains the most common cause of hyperthyroidism in children. Understanding the clinical and sociodemographic factors associated with disease progression and hormonal control may provide early insights for better disease management. This study aims to provide descriptive data of children with GD attending Paediatric Endocrine clinic, Sarawak General Hospital.

METHODOLOGY

A retrospective cross-sectional study was conducted. Medical records of all GD children attending Paediatric Endocrine Clinic, Sarawak General Hospital in 2024 were reviewed. Sociodemographic profile, laboratory findings, treatment and response were described. SPSS v30 was used for data analysis.

RESULT

A total of 11 patients were identified, with mean age at diagnosis of 8.4 ± 2.1 years. Majority of patients were female (90.9%). Majority were Chinese (36.45), followed by Malays (27.3%), Iban (27.3%) and Bidayuh. About 54.5% had positive family history of thyroid disorders and 18.2% had Down's syndrome. The mean thyroid stimulating hormone receptor antibodies (TRAb) level at diagnosis was 24.2 ± 11.4 IU/L and the median FT4 at diagnosis was 100 pmol/L (IQR 75-100). Carbimazole was the primary treatment of all patients with mean initial dose of 0.6mg/kg/day and highest dose of 0.7mg/kg/day. Propranolol was prescribed to 72.7% of patients. Normalisation of FT4 was achieved in 82% of patients with an average duration of 76 days. None of the patients achieved remission.

CONCLUSION

While this descriptive analysis provides insight into the clinical characteristics and management patterns of children with GD, further research with larger sample size is needed to identify predictors of remission and draw definitive conclusions regarding long-term outcomes and optimal treatment strategies.

EP_P011

CASE SERIES OF CONGENITAL HYPERINSULINISM IN A TERTIARY MEDICAL CENTER

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INTRODUCTION

Congenital hyperinsulinism (CHI) is the most common cause of persistent hypoglycemia in infants and children. Early and appropriate recognition and treatment of hypoglycemia is vital to minimize neurocognitive impairment. This case series is to study the molecular diagnosis, medical management and outcome of children with CHI.

METHODOLOGY

Medical data of 17 patients who were diagnosed with CHI from 2008 - 2024 in Hospital Putrajaya was retrieved from a medical electronic report.

RESULT

Hypoglycaemia occurred within the neonatal period in 70% of patients. The remaining 30% presented at infancy with the oldest age of presentation at 11 months. The highest glucose infusion rate required was 20 mcg/kg/min. Genetic test was done for 11 patients. Of these, 54.5% of patients had ABCC8 mutation while 9% had GLUD 1 and KCNJ11 gene mutation each. Gene testing was negative in 27%. In this cohort, 78.5% were responsive to diazoxide therapy, and the involved genes were ABCC8 and GLUD 1. All patients who were diazoxide non-responders responded to octreotide therapy. The genes involved were ABCC8 (80%) and KCNJ11 (20%) mutation. Neurocognitive evaluation revealed developmental delay in 11% (n=2) of patients.

CONCLUSION

Identification of the etiology of CHI helps guide management decisions. Prompt and effective management of patients is critical to ensure a good quality of life and neurological outcome. Molecular genetic study is useful in the management of CHI and effective management with good compliance must be ensured.

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EP_P012

BASELINE ASSESSMENT OF SELF-CARE PRACTICES AND PSYCHOLOGICAL WELL BEING AMONG YOUNG ADULTS WITH TYPE 1 DIABETES IN A WARRIOR CAMP SETTING

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INTRODUCTION

Psychological well-being and effective self-care are critical in the management of young adults with Type 1 Diabetes (T1DM). The T1DM Warrior Camp conducted under the MEMS-CD1C initiative is a unique focused camp to empower young adults with T1DM on advocacy and peer leadership. This study aimed to evaluate baseline levels of depression, anxiety and stress in young adults attending a T1DM camp using the Depression Anxiety Stress Scales-21 (DASS-21) and the self-care practices using the Summary of Diabetes Self-Care Activities (SDSCA) questionnaire with a follow-up evaluation planned six months post intervention.

METHODOLOGY

Fourteen young adults with T1DM (aged 18–25) attended a structured 3-day Warrior Camp focused on diabetes education, lifestyle management and peer engagement. Baseline assessment was done using the DASS-21 and SDSCA questionnaire.

RESULT

Mean haemoglobin A1c and SDSCA score were 9.34 (\pm SD 2.43)%. Participants showed the highest adherence in blood glucose monitoring (5.93 ± 1.2), while foot care had the lowest adherence (3.1 ± 1.1). Dietary and exercise behaviours showed moderate adherence. The average stress score was 7.9 ± 1.2 , corresponding to mild stress. Participants also reported moderate anxiety and mild depression, based on mean subscale scores.

CONCLUSION

This initial assessment highlights key areas of strength and opportunity in self-care practices among young adults with T1DM. Notably, blood glucose monitoring was a strong

domain, whereas foot care requires greater emphasis. These young adults on assessment report mild to moderate symptoms of psychological distress, highlighting the importance of mental health support in this population. Camps may serve as a valuable setting for monitoring and addressing psychosocial needs in young adults with T1DM. The impact of the Warrior Camp intervention on these two critical areas will be reassessed after six months to evaluate long-term changes.

EP_P013

FAMILIAL MIDFACIAL HYPOPLASIA WITH CONGENITAL HYPOPHYTUITARISM – A CASE REPORT

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INTRODUCTION

Congenital hypopituitarism is defined as deficiency of one or more pituitary hormones due to abnormal pituitary gland development. Manifestations can be nonspecific such as poor weight gain, short stature, hypoglycemia or they may be associated with midline defects.

CASE

A 10-year-6-month-old female was diagnosed with congenital hypopituitarism at the age of 5 years whereby she presented with septicemic shock secondary to bronchopneumonia with hypernatremic dehydration. Detailed physical examinations showed that she was dysmorphic with short stature, poor muscle bulk, global developmental delay and features of midfacial hypoplasia such as right cleft lip/palate and septo-optic dysplasia. Investigations of the pituitary hormones revealed hypothyroidism, hypocortisolism, growth hormone deficiency and diabetes insipidus. Cranial MRI showed hypoplastic corpus callosum, absent septum pellucidum and thickened pituitary stalk with absence of bright spots of the posterior pituitary. She was started on pituitary hormone replacement including L-thyroxine, oral desmopressin, oral hydrocortisone and somatotrophic injection. Clinical response to treatment was satisfactory in which she had gained 6 cm of height for the past year with normalized thyroid hormone and cortisol levels. Analyzing her family history, we noticed that her mother also had features of midfacial hypoplasia. Her elder sister is having a learning disability attending special school. This raised the possibility of genetic mutation in familial congenital

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hypopituitarism such as PROP1 gene. Even with similar mutation, individuals can have different levels of hormone deficiencies and be affected differently.

CONCLUSION

Careful evaluation of a dysmorphic child with features of midfacial hypoplasia is crucial to avoid missing congenital hypopituitarism. Early identification with comprehensive hormonal work-up is important to initiate hormonal therapy.

EP_P014

IT IS NOT WHAT IT SEEMS

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INTRODUCTION

Elevated thyrotropin-releasing hormone (TRH) in response to very low thyroxine (T4) level can lead to pituitary gland hyperplasia. This condition can mimic a pituitary adenoma, making it radiographically difficult to differentiate from hyperplasia.

CASE

A 10-year-8-month-old male presented to medical attention due to poor growth and learning difficulties. He was noted to be small since he was 5 years old. There were no significant medical or dietary issues. He was not dysmorphic, but growth parameters corresponded to a 4-year old male. He was prepubertal and there was no goiter noted. Investigations initially showed a normal thyroid function, low IGF-1 and a bone age which corresponds to a 6-month-old. An MRI of the brain was arranged several months later which revealed a pituitary macroadenoma compressing / indenting the optic chiasm. A repeat pituitary panel showed elevated prolactin which did not change post PEG. There was also new evidence of primary hypothyroidism. As the biochemical results exclude a macroadenoma, it was postulated that high TRH as a response to a low FT4 leads to the stimulation of the pituitary thyrotroph and lactotroph cells resulting in pituitary gland enlargement. This is thought to be rare in children but documented to occur in those with severe primary hypothyroidism with a TSH >50 mIU/L. This could be misdiagnosed as a macro-adenoma especially when thyroid function test was not performed prior to an MRI of the pituitary gland.

CONCLUSION

This case illustrates the importance of differentiating a macroadenoma from pituitary hyperplasia. The treatment differs with invasive surgery for macroadenoma and thyroxine replacement in pituitary hyperplasia.

EP_P015

A CASE OF FAMILIAL GLUCOCORTICOID RESISTANCE SYNDROME PRESENTING WITH HYPOKALEMIC PARALYSIS AND HYPERTENSION

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INTRODUCTION

Familial glucocorticoid resistance syndrome (FGRS) is a rare condition leading to compensatory ACTH hypersecretion and excess adrenal steroid production. Patients often present with mineralocorticoid and androgen excess but without features of Cushing's syndrome.

CASE

A 17-year-old, male, Malay was referred for recurrent episodes of acute paralysis secondary to hypokalaemia and concomitant hypertension since the age of 7 years. Investigations showed persistent hypokalaemia with metabolic alkalosis. Clinically, he was not dysmorphic, taller for his age, increased skin pigmentation and was in pubertal with testicular volume of 6 ml and stretched penile length of 7 cm. Laboratory investigations showed a very marked increase in random serum cortisol of more than 2000 with elevated ACTH level. Luteinizing hormone-releasing hormone (LHRH) test confirmed a diagnosis of peripheral precocious puberty. Adrenal ultrasound did not show any suspicion of malignancy. He was started on oral dexamethasone and anti-hypertensive. He showed some improvement clinically and biochemically with no further history of paralysis and improvement in serum cortisol and potassium levels.

This patient presentation is consistent with FGRS where impaired cortisol signaling leads to compensatory increase in ACTH causing excess mineralocorticoid and sex hormones. Management focuses on reducing ACTH stimulation using high-dose dexamethasone and addressing complications such as hypertension and electrolyte imbalances.

CONCLUSION

Careful evaluation of a child presenting with unexplained hypertension, hypokalaemia and hyperpigmentation is

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very important. Early diagnosis is crucial for appropriate management and genetic counselling.

EP_P016

WHEN GENITAL AMBIGUITY LEADS TO GENETIC DISCOVERY: A CASE OF NR5A1-RELATED DISORDERS OF SEXUAL DEVELOPMENT

<https://doi.org/10.15605/jafes.040.S1.246>

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INTRODUCTION

Disorders of sexual development (DSD) encompass a broad, heterogeneous groups of congenital conditions characterized by atypical development of genetic, gonadal, or phenotypic sex accompanied by abnormal development of internal and/or external genitalia. Early diagnosis is crucial to preserve fertility, ensure normal sexual function and support appropriate sex assignment, which significantly impact psychosocial well-being.

CASE

A child assigned female at birth was referred to a Paediatric Endocrinologist at 6 weeks old for evaluation of ambiguous genitalia. Clinical examination revealed penoscrotal hypospadias, rugated labioscrotal folds, palpable gonads with phallus size of 2 cm. The child is the youngest of 2 siblings, with no family history of consanguinity. Notably, the father had hypospadias, which was surgically corrected in childhood. Pelvic ultrasound revealed bilateral oval echogenic structure within labial fold, suggestive of testes, with no visible uterine structure. Hormonal investigations revealed a high testosterone level (13.1 nmol/L) and an antimüllerian hormone level of 103 pmol/L, indicating normal Sertoli cell function. Karyotyping confirmed 46,XY genotypes. Further genetic testing identified a heterozygous variant of uncertain significance in the NR5A1 gene. The child was treated with monthly intramuscular testosterone for three months, resulting in phallus growth to 3 cm.

Thorough genital examination during newborn assessment is essential to prevent missed diagnoses of DSD. This patient was diagnosed with an undervirilized male phenotype associated with an NR5A1 mutation – a principal genetic alteration implicated in DSD. The NR5A1 gene plays a crucial role in early gonadal development, testis determination and steroidogenesis.

CONCLUSION

This case highlights the importance of early recognition and management of DSD. Genetic testing for NR5A1 mutation should be considered in cases of 46,XY DSD with ambiguous genitalia, particularly when accompanied by a family history of hypospadias.

EP_P017

LATE DIAGNOSIS OF OVO-TESTICULAR DISORDER

<https://doi.org/10.15605/jafes.040.S1.247>

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INTRODUCTION

Ovo-testicular disorder of sex development (OT-DSD), formerly known as true hermaphroditism is a rare condition characterized by the presence of both ovarian and testicular tissue in an individual.

CASE

A 16-year-old Malay, female, was initially referred at the age of 9 years for further evaluation of ambiguous genitalia. She was born with ambiguous genitalia and was raised as a female. However, the family defaulted follow-up due to logistic issues. She had no history to suggest adrenal crises or progressive skin hyperpigmentation. Clinically, she was short and underweight for age (<3rd percentile), not dysmorphic, with normal hydration. Detailed genital examination revealed penoscrotal hypospadias with no palpable gonads. Biochemically, 17-OHP was normal, testosterone was elevated with evidence of germ cell failure having elevated LH and FSH. Her chromosomal analysis revealed 2 populations of cells: 46,XX (27) -77% and 46,XY (8)-23%. PCR-based molecular analysis for the SRY gene confirmed the absence of SRY gene. Genitogram at 10 years old showed no demonstrable urogenital fistula. She underwent diagnostic laparoscopy and HPE. The right gonads showed features consistent with ovotestis (true hermaphrodites) and left gonad features compatible with streak gonads. Her serial hormonal workups showed primary gonadal failure with elevated FSH (51.44) and LH(14.86) with low testosterone (<0.087) and estradiol (<18.35). She was started on estradiol valerate while waiting for her vaginal construction operation.

OT-DSD is rare and most reported cases occurred in individuals with 46,XX karyotype. However, 46,XY and mosaic karyotypes(46,XX/46 XY) have also been observed.

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CONCLUSION

Early and accurate diagnosis is essential for optimal management requiring a multidisciplinary approach including endocrinologists and surgeons.

EP_P018

ATYPICAL PRESENTATION OF SEVERE PROGNATHISM IN PATIENT WITH CONGENITAL ADRENAL HYPERPLASIA

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INTRODUCTION

Congenital adrenal hyperplasia (CAH) is a group of autosomal recessive disorders due to mutation in the genes that regulate adrenal steroidogenesis. The commonest form is due to 21-OH enzyme deficiency in which the classic form is divided into salt-losing or simple virilizing types.

CASE

A 20-year-old Malay male has been under our follow-up since his early infancy. He was diagnosed to have a salt-losing form of CAH in the neonatal period as he had adrenal crises associated with skin hyperpigmentation. The boy was treated with oral hydrocortisone 10-15 mg/m²/day and oral fludrocortisone 150 mcg to 200 mcg per day. However, starting from the age of five, he experienced medication adherence issues due to inadequate supervision and logistical challenges. At the age of 15 years, he was diagnosed to have testicular adrenal rest tumour. He later complained of progressive difficulty chewing his food due to the development of mandibular hyperplasia or prognathism. He was treated with high dose glucocorticoid and ketoconazole to control his hyperandrogenism and referred to the maxillofacial team for further management.

CONCLUSION

Despite being detected early and managed promptly, the outcome of treatment relies strongly on the compliance of the patient. Non-adherence to medication may lead to unforeseen detrimental complications which could worsen the long-term prognosis.

EP_P019

AN ADOLESCENT WITH UNEXPLAINED DIABETES MELLITUS AND ASSOCIATED CONGENITAL GENITOURINARY ANOMALIES: A CASE REPORT

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INTRODUCTION

Maturity-onset diabetes of the young (MODY) is a rare form of diabetes found in Malaysia and worldwide, with at least 14 recognized types linked to different genetic mutations. MODY Type 5 (MODY 5) is caused by mutations in the HNF1 gene, which encodes hepatocyte nuclear factor 1 beta. This condition is characterised by diabetes and various extra-pancreatic features, including abnormalities in the kidneys and urogenital system.

CASE

We present a young female patient who initially presented with diabetes mellitus, later diagnosed with congenital renal anomalies, including a right single kidney and Müllerian anomalies. Her strong family history of diabetes and renal issues underscores the importance of recognising this diagnosis. A diagnosis of a monogenic form of diabetes was suspected since she had an onset of diabetes at the age of 13 years, absent acanthosis nigricans, positive family history with onset less than 30 years old and renal/Müllerian duct abnormalities. A targeted gene panel, whole exome sequencing panel, was performed to test for MODY. The panel indicated a positive result for the HNF1B gene mutation c.766C>T (p. Pro256Ser), resulting in an amino acid change at codon 256 from proline to serine (p. Pro256Ser).

CONCLUSION

This case highlights the importance of recognising the potential overlap between diabetes, renal disorders and Müllerian anomalies, particularly in young patients without a clear family history. Genetic testing is the gold standard for diagnosing MODY, but access and affordability can be challenging for patients in Malaysia.

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EP_P020

PAEDIATRIC PRIMARY HYPERPARATHYROIDISM PRESENTING WITH BILATERAL SLIPPED UPPER FEMORAL EPIPHYSES: A CASE REPORT

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INTRODUCTION

Primary hyperparathyroidism (PHPT) is a rare endocrine disorder in children and adolescents with a prevalence of 2-5 in 100,000. PHPT in this age group is often due to single parathyroid adenoma, whereby surgery remains the definitive treatment. Postoperative transient hypocalcemia is a common complication. Nevertheless, severe hungry bone syndrome (HBS) in paediatric is uncommon and is a challenge in the post-operative management of PHPT.

CASE

A 14-year-old Malay, male presented with trivial falls and subsequently developed bilateral lower limb pain for 2 months, which led to an abnormal, painful gait for 1 week. He also had nausea, intermittent vomiting, abdominal pain, loss of weight and appetite.

Biochemical investigations were consistent with primary hyperparathyroidism. He had severe hypercalcemia, hypophosphatemia with elevated alkaline phosphatase and intact parathyroid hormone (iPTH). Pelvic x-ray revealed bilateral slipped upper femoral epiphysis (SUFE) and periosteal bone resorption at the pelvic bones. A neck ultrasound showed a hypoechoic nodule located posterior-inferior to the right lobe of the thyroid gland. A Tc-99m sestamibi parathyroid scan detected an avid lesion inferior to the right thyroid lobe.

His severe hypercalcemia was managed by hydration and loop diuretics. For preoperative optimisation, he received intravenous zoledronate and subcutaneous calcitonin. He underwent a right-focused parathyroidectomy and histopathology confirmed the diagnosis of parathyroid adenoma. Post-operatively, he developed severe HBS. He had symptomatic hypocalcemia post-parathyroidectomy for which he required continuous calcium gluconate infusion, calcitriol, calcium carbonate and cholecalciferol. Continuous intravenous calcium and intravenous alfacalcidol were given for four weeks and stopped when serum phosphorus and alkaline phosphatase levels returned to normal limits.

CONCLUSION

PHPT in children and adolescents often presents with non-specific symptoms leading to a delay in diagnosis. A diagnosis of PHPT should be considered when they present with bone pain or skeletal deformity associated with radiological imaging of osteolytic lesions.

EP_P021

THE HIDDEN THREAT: DIABETES MELLITUS IN A CHILD WITH CONGENITAL RUBELLA SYNDROME

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INTRODUCTION

Congenital rubella syndrome arises from maternal infection with rubella virus, particularly during the first trimester of pregnancy. While rubella is primarily associated with ocular, cardiac and auditory defects, its effects on the endocrine system, particularly in relation to diabetes mellitus, are seldom reported. This case report underscores the necessity of close monitoring in children with a history of rubella exposure, given the potential risk for the subsequent development of diabetes mellitus.

CASE

A 1-year 9-month-old male had right corneal clouding and absent red reflex. He was born at term with a birth weight of 2.6 kg. His mother had a multinodular goitre with no history of fever and rashes during pregnancy. He was diagnosed with right eye glaucoma and left uveitic cataract. He underwent enucleation of the right eye at 4 months and left lens surgery at 6 months.

The patient presented recently with lethargy, excessive thirst, frequent urination and weight loss. He had global developmental delays and showed signs of dehydration during the examination. His growth was within percentile. Investigations revealed blood glucose of 46 mmol/L, positive serum ketone and metabolic acidosis (pH, 7.126; HCO₃⁻, 10.1 mmol/L; base excess -22.7 mmol/L; PaCO₂, 20 mm Hg). He had no skin lesion and other systemic examinations were unremarkable.

He was diagnosed with diabetic ketoacidosis and was treated with intravenous fluids and insulin. Following metabolic stabilization, he was transitioned to subcutaneous insulin.

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There was an increased incidence of insulin-dependent diabetes mellitus with congenital rubella syndrome. Pathogenesis is multifactorial, potentially involving the viral destruction of pancreatic β -islet cells and autoimmunity. Rubella virus peptides mimic glutamic acid decarboxylase (GAD) peptides in the pancreas. This activates T-cell-mediated autoimmune destruction and progressive loss of insulin-producing pancreatic beta-cells due to cross reaction.

CONCLUSION

This case highlights a significant endocrine complication associated with congenital rubella syndrome and emphasizes the importance of early diagnosis and management.

EP_P022

PERICARDIAL EFFUSION SECONDARY TO SEVERE HYPOTHYROIDISM IN DOWN'S SYNDROME

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INTRODUCTION

Hypothyroidism is a recognized cause of pericardial effusion. Among children with Down's syndrome, hypothyroidism may be an associated feature.

METHODOLOGY

We report a case of a 4-year-old female with Down's syndrome and severe pericardial effusion secondary to hypothyroidism. She was born with no history of maternal thyroid disease. The diagnosis of Down's syndrome was made postnatally. She was diagnosed with congenital hypothyroidism and was started on treatment during her stormy neonatal period. She had a recurrent lung infection, developed chronic lung disease and worsening pulmonary hypertension. Due to multiple hospital admissions, she was non-compliant to her thyroid medications. She has been asymptomatic apart from failure to grow and mild constipation which was attributed to poor nutrition and presumed gastroesophageal reflux disease. At the age of 3 years and 6 months, she was noted to have muffled heart sounds. Her vitals were normal for age, but ECG showed a relative bradycardia with a rate of 65 bpm with low

voltage and flattening of the T-wave. Her echocardiogram showed large pericardial effusion. Her thyroid-stimulating hormone (TSH) was 1085.52 mIU/L and free thyroxine (FT4) of <1.3 pmol/L, confirming severe hypothyroidism. She was started on intravenous levothyroxine for five days before changing to oral levothyroxine to a maximum dose of 100 mcg (8 mcg/kg/day) daily. She did not require pericardiocentesis and was discharged well. Three months later, her thyroid function test showed normalization of TSH and FT4. Repeated echocardiogram showed smaller pericardial effusion.

CONCLUSION

This case report highlights a rare presentation of significant pericardial effusion secondary to severe primary hypothyroidism in a young female with Down's syndrome. Furthermore, it emphasizes the need for vigilant monitoring of thyroid function in this population and timely intervention to prevent potentially serious complications.

EP_P023

ANDROGEN INSENSITIVITY SYNDROME: A FAMILY CASE SERIES

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INTRODUCTION

Androgen insensitivity syndrome (AIS) is a rare X-linked recessive disorder caused by mutations in the androgen receptor. In Malaysia, only four cases of complete androgen insensitivity syndrome (CAIS) have been reported.

CASE

We present three biological cousins born to two sisters from the same maternal lineage, presenting with varying degrees of genitalia ambiguity.

Cousin A. A 1-year-and-5-month-old child presented with ambiguous genitalia at 1 month old. Physical examination revealed a 3 cm genital tubercle, penoscrotal hypospadias and fused symmetrical scrotal labia, with both testes retractile in the inguinoscrotal region. Antimüllerian hormone level was elevated, and an HCG stimulation test showed an increase in testosterone response. Karyotyping confirmed a 46, XY karyotype and whole exome sequencing identified a hemizygous pathogenic variant in the AR gene: p. Arg841His. Gender was assigned as male.

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Cousin B and C. Two siblings with 46, XY karyotype and ambiguous genitalia presented at different points following birth.

Sibling 1. The elder sibling, aged 4 years and 2 months, presented with ambiguous genitalia and a left inguinal hernia at 1 month old. Physical examination revealed a single opening, incomplete labioscrotal fusion and palpable gonads bilaterally at the inguinal region. Laboratory tests showed high testosterone and anti-Müllerian hormone levels, with absent uterus and ovaries.

Sibling 2. The younger sibling, aged 2 years and 6 months had ambiguous genitalia with bilateral palpable gonads in the inguinal region at birth. Despite having a male genotype, they exhibited predominantly female phenotypic traits and were raised as females. The family declined genetic testing due to financial constraints.

CONCLUSION

Three cousins with varying presentations of the AIS highlight the phenotypic diversity of the condition and the challenges in sex assignment and management, underscoring the need for genetic counselling and multidisciplinary care.

EP_P024

CHROMOSOME 9p DUPLICATION AND SHORT STATURE: A CASE REPORT

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INTRODUCTION

Chromosome 9p duplication, also referred to as partial duplication syndrome, is a rare chromosomal disorder with fewer than 200 cases reported worldwide. It is characterized by duplication of a segment of the short arm of chromosome 9 and is frequently associated with short stature, craniofacial dysmorphism and intellectual disability.

CASE

We present the case of a 6-year-old male born at term via emergency lower segment cesarean section (EMLSCS) at 37 weeks due to intrauterine growth restriction (IUGR). Since infancy, he exhibited persistent failure to thrive and subtle dysmorphic features. His height and weight remained consistently below the 3rd percentile despite adequate nutritional intake. Physical and systemic examinations were otherwise unremarkable.

Initial workup, complete blood count, renal profile were normal, Hormonal work out showed the following: thyroid stimulating hormone: 5.36, free T4: 19.85, luteinizing hormone: <0.1 IU/L, follicular stimulating hormone: 1.23 IU/L, cortisol: 168.5 nmol/L growth hormone: 0.8 ug/L and IGF-1: 266.4 ng/mL. Gross karyotyping (46,XY) and radiological imaging yielded normal results. Chromosomal microarray analysis subsequently revealed a microduplication in the 9p12-p11.2 region, establishing the diagnosis of chromosome 9p duplication syndrome.

CONCLUSION

This case underscores the importance of considering chromosomal microduplication in children presenting with unexplained short stature and developmental concerns. The role of growth hormone therapy in this population remains uncertain due to limited evidence regarding its efficacy in this specific genetic condition.

EP_P025

THE VARIED FACES OF NEONATAL THYROID DYSFUNCTION IN THE CONTEXT OF MATERNAL GRAVES DISEASE: A CASE SERIES

<https://doi.org/10.15605/jafes.040.S1.255>

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INTRODUCTION

Maternal hyperthyroidism, most commonly due to Graves' disease (GD), can cause a range of thyroid dysfunction in the fetus and neonate. Neonatal thyroid function is influenced by factors such as maternal disease activity, levels of TSH receptor antibodies (TRAb) and in-utero exposure to antithyroid drugs (ATDs). We report two neonatal cases illustrating this variability.

CASE

Case 1. A term male neonate was born to a mother diagnosed with GD during the first trimester, who was well-controlled on oral carbimazole. She had a positive TRAb with hyperaemic thyroiditis on ultrasound. The infant had a normal cord thyroid stimulating hormone (TSH) at birth but TRAb measured at one week was 2-fold above the upper limit of normal. A thyroid function test (TFT) on Day 5 showed subclinical hypothyroidism which resolved spontaneously by one month without treatment.

Case 2. A female neonate was delivered at 34 weeks' gestation to a mother with a six-year history of GD,

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complicated by thyroid storm during the current pregnancy. Her TRAb levels prior to conception were over 23-fold above normal. The infant was born with suppressed cord TSH and markedly elevated TRAb level which is 13-fold above normal. She developed symptoms of neonatal hyperthyroidism in the second week of life and was started on carbimazole and propranolol, which were weaned off by the third week. Subsequent TFTs showed a phase of subclinical hyperthyroidism followed by hypothyroidism by two months of age requiring thyroxine replacement.

CONCLUSION

These cases highlight the diverse presentation of neonatal thyroid dysfunction associated with maternal GD, ranging from transient hypothyroidism to biphasic thyroid disturbances following neonatal hyperthyroidism. High maternal TRAb levels, as seen in Case 2, may serve as a predictor of a more severe case of evolving neonatal thyroid disease. Continuous postnatal monitoring is essential, as thyroid dysfunction may not be evident at birth and can evolve over time. Timely diagnosis and appropriate management are key to prevent complications and supporting optimal neurodevelopmental outcomes.

EP_P026

FAMILIAL DYSALBUMINEMIC HYPERTHYROXINEMIA: A RARE CAUSE OF EUTHYROID HYPERTHYROXINEMIA

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INTRODUCTION

Euthyroid hyperthyroxinemia is a common clinical conundrum. It requires careful assessment to establish an accurate diagnosis. Differential diagnosis of euthyroid hyperthyroxinemia include assay interference, thyroid hormone resistance syndrome, familial dysalbuminemic hyperthyroxinemia (FDH) and TSH-oma.

CASE

A 7-month-old male was referred for incidental finding of persistent euthyroid hyperthyroxinemia. His birth history was unremarkable. Antenatally, his mother did not have any thyroid disorder. His paternal grandmother has been undergoing treatment for hyperthyroidism. Thyroid

stimulating hormone (TSH) was elevated at 16.89 mIU/L. Routine prolonged jaundice investigations revealed free thyroxine (FT4) of 33.7 pmol/L and TSH of 7.4 mIU/L. Other investigations were normal. Clinically, he was euthyroid, not dysmorphic, no goitre and thriving well with normal developmental milestones. Repeated thyroid function test (TFT) via standard immunoassay at 2, 3 and 5 months of age showed similar results of high FT4 with unsuppressed TSH. FT3 was not available. TFT using a different assay was not done. Thyroid antibody screening was normal. He was initially suspected of having thyroid hormone resistance syndrome.

Family screening showed similar TFT pattern for his father and sister who were clinically euthyroid. His mother's TFT was normal. His family was referred for confirmatory genetic testing. Whole exome sequencing (WES) for his father identified a pathogenic missense mutation in albumin gene, resulting in the replacement of an arginine with a histidine (p.Arg242His) that is associated with FDH. No genetic testing was done for the children.

CONCLUSION

FDH is a rare cause of euthyroid hyperthyroxinemia. It is an autosomal dominant disorder characterized by an abnormally increased affinity of a mutant albumin molecule to serum thyroxine causing elevated total thyroxine (T4) and elevated or normal FT4 with normal TSH level. Genetic analysis is important to establish diagnosis, to avoid further unnecessary laboratory testing and even inappropriate treatment in FDH.

EP_P027

ATYPICAL GENITALIA IN SILVER-RUSSELL SYNDROME

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INTRODUCTION

Silver-Russell Syndrome (SRS) is a clinically heterogeneous disorder which is often associated with growth restriction.

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Genital abnormalities can be present but are not part of the diagnostic criteria. We describe one case of Silver-Russell Syndrome with atypical genitalia.

CASE

A 4-month-old male was referred to our clinic for atypical genitalia. He was born premature at 36 weeks 1 day, small for gestational age (SGA) with birth weight of 1.34 kg (<3rd centile), length of 41 cm (<3rd centile) and relative macrocephaly with head circumference of 32 cm (50th centile).

On further examination, he had dysmorphic features with prominent forehead, frontal bossing and triangular facies. His limbs were asymmetrical with hemihypertrophy of the left upper and lower limbs and bilateral 5th finger clinodactyly.

Examination of the genitalia revealed underdeveloped scrotum with no scrotal fusion, micropenis with stretched penile-length 0.5 cm (<10th percentile), penoscrotal hypospadias and non-palpable gonads. The EMS score was 0, in support of undervirilization.

Investigations revealed intact mini puberty with LH, FSH and testosterone of 1.6 U/L, 14.4 U/L and 2.95 nmol/L, respectively. Other anterior pituitary hormones were normal and 17-OHP was not elevated. Karyotype was normal with 46,XY. Short beta-hCG stimulation test revealed good testosterone level, with normal testosterone to androstenedione (T:A) ratio and testosterone to dihydrotestosterone (T:DHT) ratio, excluding both 17-hydroxysteroid deficiency and 5-alpha reductase deficiency respectively.

He was assessed by the genetic team and was noted to fulfill all the six NH-CSS criteria for clinical diagnosis of Silver-Russell syndrome. SRS methylation testing was sent to determine the molecular mechanism for future recurrence risk counselling.

CONCLUSION

Although SRS is primarily a growth disorder, it may present with atypical genitalia along with growth failure and dysmorphic features. Hence, it should be considered in the differential diagnosis of a newborn with dysmorphic features, SGA and atypical genitalia.

EP_P028

UNRAVELING THE MANIFESTATION OF VITAMIN D-DEPENDENT RICKETS TYPE 1 IN PREMATURE INFANT

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INTRODUCTION

Vitamin D-dependent rickets type 1 (VDDR1) is one of the genetic causes of calciopenic rickets. This rare autosomal recessive disorder is due to the defective 1- α hydroxylase which results in deficient active vitamin D or 1,25-dihydroxyvitamin D. It manifests as stunted growth, skeletal deformities and bone pain in young children. Diagnosing this uncommon disease requires a high index of clinical suspicion and is confirmed through genetic testing.

CASE

A seven-month-old female was born prematurely at 24 weeks of gestation with birth weight of 600 grams. Both parents were non-consanguineous. She had a stormy neonatal period with prolonged ventilation due to severe respiratory distress syndrome. In early neonatal phase, she had hypocalcaemia and hypophosphataemia, with subsequent gradual increment of alkaline phosphatase (ALP) – the overall picture initially pointing towards osteopaenia of prematurity. With time, she developed severe skeletal deformities which were bowing of the limbs, palpable widening of distal radius and double malleoli, Harrison's groove and long bones fracture. Apart from low calcium (1.88 mmol/L) and phosphate (1.32 mmol/L), other bone profiles showed: 25-hydroxyvitamin D levels were insufficient (47 nmol/L), both parathyroid hormone (PTH, 47.9 pmol/L) and serial ALP (1008 U/L) were elevated. A 1,25-dihydroxyvitamin D level was not investigated in view of financial limitations. Radiological imaging revealed rickets changes over the metaphyseal plate, including Looser's zone at the humerus and tibia. Considering the severe clinical manifestations of rickets, but inconsistent with insufficient level of stored 25-hydroxyvitamin D, this indicates deficient active vitamin D level that is consistent with clinical VDDR1. The whole exome sequencing was negative, but further workup for more expensive genetic study such as whole genome sequencing will incur additional costs.

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CONCLUSION

Osteopaenia of prematurity with nutritional deficiency is commonly observed in preterm infant. Nonetheless, the presence of severe rickets with inconsistent bone profile warrants further work-up for other alternative diagnoses, including VDDR1.

EP_P029

EXPLORING THE SPECTRUM OF HORMONAL DEFICIENCY IN PITUITARY STALK INTERRUPTION SYNDROME AND ITS OUTCOME WITH GROWTH HORMONE THERAPY: CASE SERIES FROM A TERTIARY PEDIATRIC ENDOCRINOLOGY CENTER IN MALAYSIA

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INTRODUCTION

Pituitary stalk interruption syndrome (PSIS) is a rare congenital condition characterized by either isolated or combined pituitary hormone deficiency. This paper presents 6 cases of pituitary stalk interruption syndrome diagnosed and managed in a tertiary Pediatric Endocrinology Center.

CASE

Analytical review of the medical records of patients followed up in Putrajaya Hospital, Malaysia from year 2017–2024 revealed 6 male patients with confirmed diagnosis of PSIS.

Among the cohort, 50% of them had significant perinatal events including severe neonatal jaundice, prolonged non-invasive ventilation support, sepsis or hypoxic events. A total of 17% were delivered via emergency caesarean section and the rest were born via unremarkable spontaneous vaginal delivery. Clinical presentation varied with 50% of patients presenting at birth with ambiguity of genitalia, another 50% of patients presented in adolescents with short stature and delayed puberty. Features of soft dysmorphism were observed in 67% of them. All patients have growth hormone deficiency, with 83% of them having additional pituitary hormone deficiency. Half of them have multiple pituitary hormone deficiencies. None of the patients in the cohort had clinical manifestations of diabetes insipidus. MRI imaging revealed absence of pituitary stalk on all patients. All patients who have been treated with growth hormone therapy showed improvement in height velocity with a mean of 10 (± 2.5) cm per year.

CONCLUSION

Children with PSIS often have a very broad spectrum of clinical and biochemical presentations. Screening and evaluation of the pituitary-hypothalamic hormone axis is critical to guide management. This clinical entity often presents with growth retardation and thus early diagnosis is critical to allow for timely management of these patients with growth hormone therapy.

EP_P030

A CASE SERIES OF POTENTIAL CONSEQUENCES FOLLOWING INTRAMUSCULAR INJECTIONS IN CHILDREN

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INTRODUCTION

Apart from regular vaccinations, there are limited instances where deep intramuscular (IM) injections are given to children. IM gonadotropin agonist (GnRHa) is used for the treatment of central precocious puberty (CPP). It is given deep IM at the upper outer quadrant of the buttock. We describe three cases to demonstrate complications of this procedure.

CASE

Case 1. An 8-year-old female was diagnosed with CPP at the age of 5 when she presented with breast development. She has been receiving 3-monthly IM GnRHa since then. In the clinic, parents informed that she had a 1-week history of upper respiratory tract infection. She was afebrile and was given an injection as usual. Patient came back a week later complaining of pain and swelling at injection site. After inspection, a diagnosis of sterile abscess was made. She was treated with local and oral antibiotics.

Case 2. An 8-year-3-month-old female was diagnosed with CPP at the age of 7 years and 5 months. She was due for her 3rd IM GnRHa. She has been anxious about injection pain and needed comfort from parents/nurses at each visit. She insisted on more pain relief prophylaxis. She came back after 1 week with circular erythematous rash and blister formation around injection site. Diagnosis of superficial burn secondary to ethylchloride spray was made.

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Case 3. An 8-year-old female with underlying mild autism was started on GnRHa injections for CPP. At every clinic visit, she will cry, shout and throw tantrums which were attributed to injection anxiety. Parents and nurses had a lot of difficulty getting her ready for injections. On her last visit, the nurse who gave her the injection reported bite marks and bruises on her arm because the patient bit her.

CONCLUSION

Although rare, one should always take extra precautions when dealing with IM injections in children. Repeated procedures carry higher risk as mentioned in this case series.

EP_P031

PITUITARY HYPERPLASIA SECONDARY TO PRIMARY HYPOTHYROIDISM – A CASE REPORT

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INTRODUCTION

Primary hypothyroidism in children can present insidiously and mimic other systemic conditions, including neurological symptoms. In rare cases, it may lead to pituitary hyperplasia due to lack of negative feedback on thyrotrophs. Timely recognition is essential to prevent complications and avoid unnecessary neurosurgical intervention.

CASE

We report a case of an 8-year-old female who presented with chronic headaches, cold intolerance, constipation and frontal scalp hair thinning. Symptoms were insidious, with persistent headaches noted since the age of six. Despite multiple outpatient visits, including private pediatric and ophthalmology consultations, no clear diagnosis was made. Ophthalmological evaluations were also normal.

Due to persistent symptoms, neuroimaging was done to exclude intracranial mass or raised intracranial pressure. MRI of the brain and pituitary revealed enlarged pituitary gland measuring 7.7 mm (AP) x 12.4 mm (width) x 9.7 mm

(height), with normal posterior pituitary bright spot and pituitary stalk. Other surrounding structures were normal. Thyroid function test (TFT) performed revealed an elevated TSH of 150 mIU/L with low fT4 at 8.7 pmol/L, consistent with primary hypothyroidism. Anti-thyroid peroxidase (TPO) and thyroglobulin antibodies were positive, confirming Hashimoto's thyroiditis. Other pituitary hormones were normal. She was initiated on levothyroxine, and serial TFTs demonstrated gradual improvement. MRI features were consistent with pituitary hyperplasia secondary to long-standing hypothyroidism (PHPH), and no neurosurgical intervention was warranted. A repeat MRI scan performed 10 months after commencement of treatment showed normal study with a pituitary gland measuring 6.7 mm (AP) x 12.2 mm (width) x 5.7mm (height). Her latest TFT has normalised with TSH of 3.76 mIU/L and fT4 of 18.5pmol/L on levothyroxine 37.5 mcg qd Monday to Friday, and 50 mcg qd on weekends.

CONCLUSION

PHPH is an uncommon cause of pituitary enlargement in children. This case highlights the importance of comprehensive endocrine assessment in children with chronic headaches. Early diagnosis and thyroid hormone replacement can lead to complete resolution of symptoms and regression of pituitary enlargement, avoiding misdiagnosis and overtreatment.

EP_P032

AN UNCOMMON CAUSE OF PERSISTENT HYPERCALCAEMIA WITH NEPHROCALCINOSIS IN INFANCY

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INTRODUCTION

Hypercalcaemia with nephrocalcinosis in infants is commonly caused by excessive calcium or vitamin D supplementation, neonatal primary hyperparathyroidism, subcutaneous fat necrosis or various genetic disorders.

CASE

We present a 6-month-old Indian male infant who was born preterm at 33 weeks, via elective LSCS for polyhydramnios with weight of 1.33 kg, length of 46 cm and head circumference of 27 cm. His mother had severe polyhydramnios, requiring amnioreduction thrice. Both parents were consanguineous. During his 3-month-stay at NICU, he had persistent hypercalcaemia with intermittent polyuria. Serum calcium ranged: 2.5-2.9 mmol/L,

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phosphate: 1.85-3.05 mmol/L, alkaline phosphatase (ALP): 500-800 IU/L and 25(OH)D3: 200 nmol/L. He also had hypochloraemic hypokalemic metabolic alkalosis, hyperreninemia (>550 mU/L) and hyperaldosteronemia (>3656 pmol/L). Ultrasound at 2-month-old demonstrated bilateral renal medullary nephrocalcinosis and cholelithiasis. Skeletal survey revealed no significant bony abnormalities. There were episodes of hyponatremia and hypokalemia, which improved spontaneously. Clinically, he had prominent forehead, triangular face, right hand pre-axial polydactyly and bilateral short distal phalanx of the 4th and 5th fingers with nail hypoplasia. His weight gain was poor with delayed motor development and hypotonia. At 6-month-old, his care was shared by paediatric nephrologist and endocrinologist. He was 3.57 kg with a length of 57.8 cm. His iPTH later resulted in relatively inappropriately raised level, (Ca: 2.81mmol/L, PO4: 1.87 mmol/L, ALP: 770 IU/L, iPTH: 68.3 pg/mL). Urinalysis showed profound natriuresis and hypercalciuria (24-hour urine Ca: 5.3 mg/kg/day). Ultrasound of the thyroid exhibited no abnormality. The parents' calcium profiles were normal. Pamidronic acid (1 mg/kg/dose) was given (when serum calcium >3.0 mmol/L) but the hypercalcemia only transiently improved. Eventually, he was treated with indomethacin and free water supplement. The whole exome sequencing revealed a heterozygous pathogenic variant in ROR2 gene and a homozygous variant of uncertain significance in KCNJ1 gene.

CONCLUSION

Antenatal Bartter syndrome presents insidiously during neonatal period, typically with polyhydramnios, IUGR, prematurity, polyuria and failure to thrive. It can present with nephrocalcinosis accompanied by features resembling primary hyperparathyroidism. Genetic testing enhances the diagnostic precision of various Bartter syndrome subtypes.

EP_P033

BALANCING SUGAR AND STRAIN: LIVING WITH TYPE 1 DIABETES AND CHRONIC KIDNEY DISEASE

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INTRODUCTION

Effective glycaemic control is essential in the type 1 diabetes mellitus (T1DM) to prevent both acute and chronic complications of the disease. This case explores the challenges faced in daily glucose regulation and highlights the critical role of tight glycaemic control in ensuring long term health outcomes for individuals living with the disease.

CASE

A 13-year-old male arrived at the emergency department in impending diabetic ketoacidosis (DKA). He was diagnosed with T1DM at the age of 3, though his antibody work-up was negative. His medical history revealed poor adherence to medical appointments and treatment, with multiple hospitalizations for DKA between the ages of 3 and 10. At 11 years old, he was completely lost to follow-up and was managing his insulin doses independently, without proper blood sugar monitoring. After 2 years without medical supervision, he was admitted with disseminated methicillin-sensitive *Staphylococcus aureus* (MSSA) infection, bilateral renal abscesses and stage 3a chronic kidney disease. His HbA1c at the time of admission was 14%.

CONCLUSION

This case highlights the serious consequences of poor glycemic control in T1DM. Persistent hyperglycemia and inadequate disease management likely contributed to immune dysfunction, heightened infection risk and progressive kidney damage, ultimately leading to his critical condition. Consistent diabetes management and early medical intervention are essential to prevent such life-threatening complications.

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EP_P034

A HEAVY DIAGNOSIS: CUSHING'S SYNDROME SECONDARY TO ADRENAL CORTICAL ADENOMA

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INTRODUCTION

Cushing's syndrome (CS) is very rare in childhood and adolescence. It may present as a diagnostic dilemma among clinicians.

CASE

We report an 11-year-old male with hypertensive emergency, congestive heart failure, pulmonary oedema and acute kidney injury. He had a two-year history of rapid weight gain and symptoms of obstructive sleep apnoea. He was obese with a body-mass-index (BMI) of 47 kg/m² (weight 109 kg, >95th centile; height 152 cm, 90th centile). He appeared depressed with severe acanthosis nigricans, truncal obesity, dorsocervical fat pad, striae and virilized.

He required intravenous labetalol and four antihypertensives on admission. Echocardiography revealed left ventricular impaired function (ejection fraction of 45%). Abdominal ultrasonography showed a left suprarenal lesion without renal artery stenosis. Abdominal computed tomography confirmed a lesion at the left suprarenal region (4.9 x 6.3 x 4.7 cm) and a right simple renal cyst. Urine biogenic amines and metanephrines were normal. He had elevated urinary cortisol at 3,574 nmol/24 hour (160-1,112) with loss of diurnal variation on salivary cortisol [midnight: 13.4 nmol/L (<11.3) and morning: 0.9 nmol/L (<24.1)] and on serum cortisol (midnight: 341.4 nmol/L and morning: 360.8 nmol/L). Further tests revealed suppressed ACTH: <0.33 pmol/L, and elevated serum dehydroepiandrosterone sulphate (DHEAS): 7.210 umol/L (0.660-6.700). There was no suppression on low- and high-dose dexamethasone suppression tests, consistent with ACTH-independent CS.

He underwent laparoscopic adrenalectomy, revealing an 8 x 5 cm, well-encapsulated left adrenal tumour. Preliminary histopathological analysis suggests adrenal adenoma. Perioperatively, he required stress-dose hydrocortisone (100 mg/m²/day) and tapered to a physiological dose (7 mg/m²/day) upon discharge. At follow-up, he was on two antihypertensive medications, demonstrated improved cardiac function (EF: 55%) and weight reduction (97 kg).

CONCLUSION

Although CS is rare in children, high levels of suspicion should be applied to those presenting with rapid onset obesity, hypertension and/or virilisation. Diagnosis of CS involves multiple investigative steps to guide treatment.

EP_P035

OCCULT MOSAICISM OF KARYOTYPING IN 45,X / 46,XY DSD

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INTRODUCTION

Disorder of sex development (DSD) with 45, X/46, XY mosaicism is a rare disorder. The prevalence is estimated to be less than 1:20,000.

CASE

A term baby, born with a good Apgar score at Hospital Sultan Abdul Halim. Genital examination showed atypical appearance with genital tubercle measuring 1.8 cm, bilateral labio-scrotal folds partially fused with single opening at perineum and no palpable gonads. External genitalia score (EGS) was 5/12. Ultrasound assessment revealed right inguinal lesion, equivocal for testis or inguinal hernia and small fluid-filled tubular structure posterior to the bladder which could represent either vagina or urogenital sinus. Genitogram report was consistent with vagina and complementary visualization of the uterus. Salt wasting was not observed during NICU stay. A 17-OHP screen on day-7-of-life reported 41.7 nmol/L. Urgent karyotyping initially reported 46, XY (cells analyzed 18, counted 10). On further assessment at the Hospital Sultanah Bahiyah, hormonal profile showed elevated gonadotrophins (FSH: 112.8i U/L, LH: 2.74i U/L and testosterone: 8.07 nmol/L). Follow-up 17-OHP was 82.8 nmol/L, and a short Synacthen test showed peak cortisol measuring 863.6 nmol/L. Serum AMH was 19.2 nmol/L (NV: 235.5-1125.9 for males and ≤31.2 for females). HCG stimulation showed increased testosterone: 2.00 nmol/L (Day 1) and 8.4 nmol/L (Day 3). Secondary analysis of initial chromosome samples with 48 cells analyzed and 15 counted [total 63], revealed mosaicism 45,X [14], 46,XY [49]. Surgical evaluation by 1-year-old reported presence of uterus with left streak gonad (removed) and right fimbriae-like-structure (biopsy) and suspected ovotestes (2 x 2 cm). HPE results are still pending by the time of report.

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CONCLUSION

Chromosomal analysis with adequate cells is crucial in identifying subtypes of DSD. When mosaicism is suspected, a larger number of cells (at least 30) should be analyzed to accurately detect and characterize these conditions.

EP_P036

WHEN WATER BECOMES A FRENEMY: A CASE SERIES ON THIRSTY CHILDREN AND LITERATURE REVIEW

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INTRODUCTION

Polydipsia is defined as excessive thirst causing the consumption of large amounts of fluids, more than 2 liters/m²/day in children, with consequential polyuria. It is of paramount importance to distinguish between diabetes insipidus (DI) and primary polydipsia as treatment differs, and inappropriate use of desmopressin can be detrimental in patients with primary polydipsia.

CASE

We present 3 children referred to the Paediatric Endocrine Clinic who exhibited a long history of excessive drinking.

Case 1. A 9-year-old male presented with an unquenchable thirst, drinking 6 to 8 L per day that required him to wake up 3-4 times nightly to drink water. A water deprivation test was performed, yielding inconclusive results, hence needed further investigation.

Case 2. A 9-year-old male's excessive drinking during school hours concerned his teachers, prompting an investigation. A subsequent water deprivation test confirmed primary polydipsia.

Case 3. A 2-year-old toddler presented with a progressive history of excessive drinking. Although his water deprivation test showed equivocal findings, his cranial MRI confirmed the diagnosis of central DI.

Fortunately, our patients did not demonstrate any red flags, such as dehydration, visual field loss, recurrent vomiting, headache or altered consciousness. Our school-going patients denied a history of school bullying or truancy. None of the children were on medication and there was no family history of similar symptoms.

CONCLUSION

These cases underscore the importance and limitations of a water deprivation test in diagnosing polydipsia and polyuria in children. Inconclusive results must be interpreted with caution and necessitate further investigation, as baseline clinical and biochemical variables cannot substitute for the water deprivation test.

EP_P037

THYROID CHANGES IN INFANTS OF MOTHERS WITH GRAVES' DISEASE: A CASE SERIES

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INTRODUCTION

Maternal Graves' disease (GD) can affect neonatal thyroid function. Maternal factors such as timing of diagnosis, TSH-receptor Ab (TRAb) titre, anti-thyroid medications and prior radioiodine therapy will affect outcome.

CASE

We describe six infants born to mothers with GD (2 mothers diagnosed before pregnancy and 4 mothers during pregnancy) in Hospital Sultanah Bahiyah in 2023-2024. All mothers had elevated TRAb, from 3.34 IU/L to >40 IU/L, taken at 16-35 weeks of gestation. Five were treated with carbimazole (10-40 mg daily). Four started treatment during pregnancy and one prior to pregnancy. One mother had RAI before pregnancy and her infant had negative TRAb. Two (2/6) neonates had low birth weight and four (4/6) were premature. One neonate had fetal goiter and required elective LSCS via EXIT procedure by paediatric ORL. This neonate's goitre resolved following L-thyroxine initiation and was extubated within 3 days. Four neonates had elevated TRAb ranging 11.21 U/L to 39.51 U/L. Within 1st week, five had hyperthyroidism, of whom, one was symptomatic for moderate tachycardia. Two required low dose carbimazole for 4-6 weeks. The highest fT4 was 61.24 pmol/L. One patient with no thyrotoxicosis initially developed central hypothyroidism by 1-month-old. Of those with initial transient hyperthyroidism, three (3/5) developed central hypothyroidism thereafter requiring L-thyroxine. Two of them (2/3) had transient central hypothyroidism that resolved between 2-month-old and 1-year-7-month-old. By the time of report, three (3/6) infants still require L-thyroxine of whom two (2/3) had central hypothyroidism with prior hyperthyroidism. All these infants have appropriate growth and development during follow-up.

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CONCLUSION

It is crucial to ensure mothers with GD have early diagnosis and adequate monitoring during pregnancy to prevent neonatal complications. Infants of maternal GD should be monitored closely and at regular intervals, to detect alteration of thyroid function which is important for brain development.

EP_P038

THE ECLIPSE HAS PASSED

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INTRODUCTION

Primary adrenal insufficiency (PAI) can be misdiagnosed as other life-threatening conditions. Clinical signs of PAI are based on the deficiency of both glucocorticoids and mineralocorticoids. We report an infant with generalised hyperpigmentation and PAI.

CASE

A 10-month-old female, born via spontaneous vaginal delivery, with poor Apgar Score (1^{13510}), was admitted for severe hypoxic-ischemic-injury (HIE). She had multiple episodes of seizures and required cooling therapy. Her parents are non-consanguineous. Clinically, she had generalised skin hyperpigmentation and normal female genitalia.

She had severe metabolic acidosis (pH: 6.7, HCO_3^- : 8.3 mmol/L, lactate: 14.1 mmol/L). She developed an adrenal crisis at day 5 of life with lowest sodium: 125 mmol/L (134-142) and highest potassium: >7 mmol/L (3.5-5.6). The lowest blood glucose was 3.2 mmol/L.

Investigations at day 4 of life revealed low serum cortisol: 37.6 nmol/L (NV: 185-624), detectable testosterone: 0.9 nmol/L, normal 17-hydroxyprogesterone (17-OHP): 1.89 nmol/L (NV: <19.1), inappropriately low aldosterone: <103 pmol/L (NV: 471-4272) with high renin: >550 mU/L (NV: 4.00-89.00). The karyotype was 46 XX and the inborn error of metabolism study was non-diagnostic. Adrenocorticotrophic hormone (ACTH) was normal at 5.16 pmol/L (NV: 1.60-13.90) but was done after initial doses of hydrocortisone. Pelvic ultrasonography (USG) showed Mullerian structures and cranial USG was normal.

She was treated with stress dose of hydrocortisone 2.5 mg *qid* (45 mg/m²/day), then weaned to oral hydrocortisone 1.5 mg *tid* (16 mg/m²/day), fludrocortisone 150 mcg *od*

and sodium chloride 0.5 grams *bid*. On follow-up, she had markedly reduced skin pigmentation. She had serum renin <1.80 mU/L and normal renal profiles. She was thriving with a weight of 7.4 kg (25-50th percentile), length of 65 cm (5-10th percentile) with appropriate developmental milestones.

CONCLUSION

Early diagnosis is crucial for effective management of PAI. Hyperpigmentation is a pathognomonic physical examination finding because ACTH shares the same affinity with α -melanocyte-stimulating hormone (MSH). Our patient's normal ACTH level was misleading due to hydrocortisone suppression.

EP_P039

WHEN THYROID STIMULATING HORMONE AND FREE THYROXINE MISMATCH: A CASE REPORT

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INTRODUCTION

Thyroid hormone resistance (THR) is characterized by lack of end-organ responsiveness to thyroid hormone with high serum free thyroxine (FT4) with inappropriately high thyroid stimulating hormone (TSH).

CASE

We report a 3-month-old male, born term via spontaneous vaginal delivery was referred to us at day 13 of life for inappropriately high TSH: 7.69 mIU/L (NV: 0.39-7.0) and high FT4: 57.5 mIU/L (NV: 8.7-16.2). His birth weight was 3.5 kg. Antenatally, his mother has no thyroid disorder. He is the only child of non-consanguineous parents.

The cord TSH was 22.01 mIU/L. At day 3 of life, TSH was 15 mIU/L and FT4 was insufficient. At day 23 of life, thyroid function test (TFT) revealed TSH: 6.29 mIU/L, FT4: 47.9 pmol/L and free triiodothyronine (FT3): 6.6 pmol/L (NV: 3.1-10.6). Clinically he had persistent left parieto-occipital swelling and tachycardia. He was started on carbimazole (0.1 mg/kg/day) at 2 months of life.

Thyroid autoantibodies were negative with anti-TSH receptor (TRAb) <0.8 IU/L (NV: <1.75), anti-thyroid peroxidase <9 IU/ml (NV: <35) and antithyroglobulin 14 IU/mL (NV: <115). Pituitary hormones taken on day 76 of life revealed prolactin 1,553.2 mIU/L (NV: 70.81-566.64) with mini puberty (testosterone: 4.3 nmol/L, FSH: 1.8 IU/L

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and LH: 3.3 IU/L). At day 82 of life, prolactin was normal at 322 mIU/L and IGF-1 at 45.6 ng/ml (NV: 27-157). FBC, renal profile and liver function test were normal. Skull x-ray revealed no calcification and cranial ultrasound was normal. During follow-up, he has normal developmental milestones, weight: 5.3 kg (25th percentile), length: 55 cm (2nd percentile) and COH: 40 cm (75th percentile). Genetic confirmation testing is pending while awaiting funds. We plan to stop Carbimazole during follow-up.

CONCLUSION

We started carbimazole for tachycardia and suspicion of craniosynostosis. Treatment for THRB is not needed mostly, because the hyposensitivity to thyroid hormones seems to be adequately compensated by the increase in secretion of T4 and generation of T3.

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