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ABSTRACT BOOK

MEMS
Established 1981
Malaysian Endocrine
& Metabolic Society

MEMS ANNUAL CONGRESS

MAC 14 2024

Invigorating Endocrinology: Harmonising the Hormones

18 July 2024 Pre Congress | **19-21 July 2024** Congress

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MEMS
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MEMS ANNUAL CONGRESS
MAC 14 2024

Welcome Message from MAC 14 Chairperson / Organising Committee

Dear Friends and esteemed Colleagues,

Warmest Greetings from MEMS!

On behalf of the Organizing Committee, it is my great pleasure to extend a warm welcome to each and every one of you to the 14th MEMS Annual Congress 2024 (MAC 14) – a convergence of minds and expertise in the dynamic field of endocrinology which will be held from 19th July to 21st July, 2024 at Hilton and Le Meridien Kuala Lumpur, Malaysia.

Bringing the theme **'Invigorating Endocrinology: Harmonizing the Hormones',** we will bring you on a journey of exploration and collaboration, delving into the latest advancements, breakthroughs, and challenges that define the forefront of endocrine research and clinical practice. Our distinguished speakers, who are leaders in their respective fields, will share their insights and experiences, fostering intellectual discourse that is at the heart of this scientific congress.

A pre-congress workshop focussing on **'Confirming & Localising Functioning Tumours,'** has been organized for the 18th July, 2024 for endocrinologists and interested internists.

This congress also serves as a platform for the exchange of scientific knowledge, ideas and innovations that shape the future of endocrinology in Malaysia. We invite you to submit your abstracts early by 30th April, 2024 to be eligible in the running for Best Oral & Best Poster prizes.

Beyond the scientific program, there will also be plenty of networking opportunities, forging connections that may spark new collaborations and fuel the collective pursuit of excellence in endocrine research and patient care.

I look forward to welcoming you at this Invigorating congress!

Yours Sincerely,
Dr Noor Lita Adam
Chairperson, Organising Committee,
14th MEMS Annual Congress 2024 (MAC 14)

MAC 14 Organising Committee

Organising Chairperson	Dr Noor Lita Adam
Scientific Chairperson	Dr Subashini Rajoo (Chair, Adult Session)
	Dr Lim Poi Giok (Co-Chair, Paeds Session)
Committee Members	Dr Arini Nuran Md Idris
	Dr Elliyyin Katiman
	Dr See Chee Keong
	Dr Tong Chin Voon
	Dr Yusniza Yusoff
	Ms Cheah Yet Mei

MEMS ANNUAL CONGRESS MAC 14 2024

18 July 2024

Pre-Congress

Confirming & Localising Functioning Tumours

Prof Dr Norlela Sukor
Senior Consultant Endocrinologist,
Universiti Kebangsaan Malaysia

Datin Dr Malinda Abd Majid
Consultant Radiologist,
Hospital Kuala Lumpur

Dr Hairuddin Achmad Sankala
Radiologist
Hospital Kuala Lumpur

Dr Gayathri Devi Krishnan
Consultant Endocrinologist,
Hospital Kuala Lumpur

Dr Subashini Rajoo
Consultant Physician &
Endocrinologist,
Hospital Kuala Lumpur

Programme:

8.30 am - 9.00 am	Registration & Welcome Reception	
9.00 am - 9.10 am	Welcome Address	Dr Noor Lita Adam
9.10 am - 10.25 am	Pituitary Imaging	Dr Hairuddin Achmad Sankala
10.25 am – 10.40 am	Coffee/Tea Break	
10.40 am – 11.55 am	Adrenal & Pancreas Imaging	Datin Dr Malinda Abd Majid
11.55 am – 12.55 pm	IPSS Inferior Petrosal Sinus Sampling	Dr Gayathri Devi Krishnan
12.55 pm – 2.00 pm	Lunch Break	
2.00 pm – 3.00 pm	AVS Adrenal Venous Sampling	Prof Dr Norlela Sukor
3.00 pm – 4.00 pm	ASVS Arterial Selective Venous Sampling	Dr Subashini Rajoo
4.00 pm	Tea and Networking	
4.00 pm – 5.00 pm	Endocrine Fellow Town Hall	

MEMS ANNUAL CONGRESS MAC 14 2024

Faculty

Prof Dr C Rajasoorya

Senior Consultant Physician and Endocrinologist
Sengkang General Hospital

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Professor of Clinical Endocrinology,
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Paediatric Endocrinologist,
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University of Auckland

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Consultant Paediatric Endocrinologist,
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Consultant Endocrinologist,
Hospital Putrajaya

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Senior Consultant Urologist,
Universiti Kebangsaan Malaysia

Programme
Day 1 - 19th July 2024 (Friday)

	Clarke Ballroom	Ballroom B + Ballroom C	Sentral Ballroom
0730 - 0820	Registration		
0820 - 0830	**Welcome Remarks** *Noor Lita Adam, Organising Chairperson MAC 14* **Opening Speech** *Nurain Mohd Noor, President MEMS*		
0830 - 0845	**MEMS Highlights:** Changing Diabetes in Children *Nurain Mohd Noor* Introduction to Practical Guide to Endocrine Dynamic Tests *KKM Endocrine Subspecialty Service*		
0845 - 0930	**PLENARY 1:** Optimising Management for Pituitary Tumors Through Functional Imaging Chairperson: Subashini Rajoo *Mark Gurnell*		
0930 - 1015	**PLENARY 2:** Winning the War Against Diabesity Chairperson: Noor Lita Adam *Roger Chen*		
1015 - 1030	Tea Break / *Tour d'MAC*		
1030 - 1200	**Symposium 1:** **BONE METABOLISM** Chairperson: Hew Fen Lee Osteo-sarcopenia in Diabetes *Sharmila Paramasivam* Vitamin D Supplementation: Busting the Myths *Manju Chandran* CKD and Osteoporosis *Goh Bak Leong*	**Symposium 2:** **ADRENAL** Chairperson: Azraai Nasruddin Biochemical Evaluation in Pheochromocytoma and Paraganglioma *Norlela Sukor* Pseudocushing *Zanariah Hussein* Primary Aldosteronism – Time for Us to Think Differently *Mark Gurnell*	**Symposium 3:** **PAEDIATRICS: DIABETES & OBESITY** Chairperson: Meenal Mavinkurve Managing Diabetes in Resource Limited Settings *Lim Song Hai* T2DM In Children & Adolescents: What is on the Horizon? *Muhammad Yazid Jalaludin* Obesity Crisis Among Children & Adolescents in Malaysia *Nalini Selveindran*

Continue to Next Page >

1200 - 1245	**MTE 1:** 'Tricks' in DEXA *Hew Fen Lee*	**MTE 2:** Non-Insulinaemic Hypoglycemia *Yong Sy Liang*		**MTE 3:** Congenital Adrenal Hyperplasia: The Journey from Birth to Adulthood *Wu Loo Ling*
	Clarke Ballroom	**Ballroom B**	**Ballroom C**	**Sentral Ballroom**
1245 - 1345	Abbott Lab Lunch Symposium Chairperson: Nor Shaffinaz Yusoff Azmi Merican Diabetes Care: A Holistic Approach to Help Mary *Lim Lee Ling* Abbott	ZPT Lunch Symposium Chairperson: Sharifah Faradila Wan Muhamad Hatta A Lifetime of Adjustments: Unveiling the Emotional Rollercoaster of Pill to Needle *Luqman Ibrahim* ZP THERAPEUTICS making healthcare more accessible	Novo Nordisk Lunch Symposium **Obesity Talks – It All Starts Here** Decoding Obesity: Unravelling the Science Behind Obesity *Rohana Abdul Ghani* From SCALE to Real-World Experience: Managing Obesity Effectively *Jeyakantha Ratnasingam* novo nordisk	Novo Nordisk (Rare Disease Malaysia) Lunch Symposium Chairperson: Raja Aimee Raja Abdullah **Optimizing the Approach to Growth Disorders: What, Why and How?** Growth an Indicator of Health: Importance of Early Diagnosis and Treatment *Azriyanti Anuar Zaini* GH Therapy: Importance of Treatment Adherence for a Better Future *Paul Hofman* novo nordisk
1345 - 1400	Trade Exhibition *Tour d'MAC*			

Continue to Next Page >

Programme

Day 1 - 19th July 2024 (Friday)

	Clarke Ballroom	Ballroom B + Ballroom C	Sentral Ballroom
1400 - 1440	**MTE 4:** Approach to Hirsutism *Wong Ming*	**MTE 5:** Type 1 DM & CV Risk *Foo Siew Hui*	**MTE 6:** Approach to Endocrine Causes of Hypertension in Children and Adolescents *Azriyanti Anuar Zaini*
1440 - 1610	**Symposium 5:** **PITUITARY** Chairperson: See Chee Keong Acromegaly in Malaysia: Is There Data? *See Chee Keong* Medical Therapy in NFPA *Azraai Nasruddin* Water Balance Post Pituitary Surgery *C Rajasoorya*	**Symposium 4:** **OBESITY** Chairperson: Norlaila Mustafa White Fat and Brown Fat: Friend or Foe *Rohana Abdul Ghani* Role of Gut Microbiome in Obesity *Masni Mohamad* Time Restricted Feeding: How to Make it Work? *Winnie Chee Siew Swee*	**Symposium 6:** **PAEDIATRICS: CONTROVERSIES IN PAEDIATRIC ENDOCRINOLOGY** Chairperson: Raja Aimee Raja Abdullah Should Central Precocious Puberty with Onset Between 6 – 8 Years be Suppressed? Points to Ponder *Rashdan Zaki Mohamed* Should Skeletal Maturation be Manipulated for Extra Height Gain? *Muhammad Yazid Jalaludin* Pharmacotherapy in Childhood Obesity: Is it Safe and Effective? *Paul Hofman*
1610 - 1615	Solve the Quiz		
1615 - 1705		**MEMS Olympiad Final**	**Poster Presentation at Sentral Ballroom Foyer**
1705 - 1800	Trade Exhibition / *Tour d'MAC*		
1900 - 2200	MEMS *'House of Harmony'* Annual Dinner (***exclusively for members and by invitation only***) ***Sentral Ballroom, Hilton Kuala Lumpur***		

	Ballroom B + Ballroom C		
0750 - 0830	Merck Sunrise Symposium Chairperson: Alexander Tan Tong Boon Pharmacotherapy in Prediabetes Management: How Early Should We Start? *Norlaila Mustafa*		
0830 - 0915	**PLENARY 3:** Hormone & Aging – Arresting the Pause Chairperson: Nurain Mohd Noor *Chan Siew Pheng*		
0915 - 1000	**PLENARY 4:** Too Early or Too Late: Managing Puberty at the Right Time Chairperson: Janet Hong *Loke Kah Yin*		
1000 - 1020	Tea Break / *Tour d'MAC - First Draw*		
	Clarke Ballroom	**Ballroom B + Ballroom C**	**Sentral Ballroom**
1020 - 1150	**Symposium 7:** **DIABETES MELLITUS** Chairperson: Tong Chin Voon Secondary Diabetes: Not to be Missed! *Tong Chin Voon* MASD *Chan Wah Kheong* When to Consider Therapy De-Escalation in Diabetes? *Miza Hiryanti Zakaria*	**Symposium 8:** **THYROID** Chairperson: Wan Izani Wan Mohd DTC Risk Assessment Post-Thyroidectomy *Malik Mumtaz* Unconventional ATD *Mafauzy Mohamed* Uncommon DTC *Navarasi S Raja Gopal*	**Symposium 9:** **PAEDIATRICS– BONE METABOLISM & THYROID** Chairperson: Noor Arliena Mat Amin Interpretation of Abnormal TFTs *Ting Tzer Hwu* Bone Health In Chronic Illness *Arini Nuran Md Idris* Vitamin D Deficiency Epidemic *Wong Sze Lyn Jeanne*
1150 - 1230	**MTE 7:** What to do When Thyroid Function Tests Don't Make Sense *Mark Gurnell*	**MTE 8:** Pituitary Tumors & Pregnancy *C Rajasoorya*	**MTE 9:** Challenges and Pitfalls in Diagnosis and Management of Growth Hormone Deficiency in Children & Adolescents *Paul Hofman*

Continue to Next Page >

Programme
Day 2 - 20th July 2024 (Saturday)

	Clarke Ballroom	Ballroom B	Ballroom C
1230 - 1330	Boehringer Ingelheim Lunch Symposium Chairperson: Yew Shiong Shiong **U-R-INE FOR THE RENAL DEAL!** Protecting DKD Patients with SGLT2i: The Connection between T2D and CKD *Lim Soo Kun* Celebrating the New Kid(ney) on the Block: A Paradigm Shift in CKD Management *Alexander Tan Tong Boon* Boehringer Ingelheim	ZPT Lunch Symposium Chairperson: Rohana Abdul Ghani Rise to the Peak- Present and Future of Incretin Therapy *Roger Chen* ZP THERAPEUTICS making healthcare more accessible	Novo Nordisk Lunch Symposium **The R Factors - Transforming T2D Management Across Patient Journey** Holistic Management Across the T2D Patient Journey *Shireene Vethakkan* Shifting Weights and Turning Tides in the Realm of T2D with Oral GLP-1RA *Lim Lee Ling* Patient-centric Care: The Cornerstone Across Continuum of Diabetes Management *Chan Siew Pheng* novo nordisk®
1330 - 1400	Solve the Quiz / Trade Exhibition / *Tour d'MAC*		
	Clarke Ballroom	**Ballroom B + Ballroom C**	**Sentral Ballroom**
1400 - 1440	**MTE 11:** **Congenital Rickets** *Manju Chandran*	**MTE 10:** **Amiodarone and Thyroid: Friend or Foe** *Nor Azmi Kamaruddin*	**MTE 12:** **When is Too Short a Concern?** *Loke Kah Yin*
1440 - 1610	**Symposium 11:** **REPRODUCTIVE ENDOCRINOLOGY** Chairperson: Elliyyin Katiman Preserving Fertility in Oncology *Mohd Faizal Ahmad* Transition Care in Turners' *Shanty Velaiutham* Hormonal Replacement Therapy in Premature Ovarian Failure *Lim Siang Chin*	**Symposium 10:** **ENDOCRINE DISORDERS IN PREGNANCY** Chairperson: Norasyikin Abd Wahab Gestational Thyrotoxicosis: Harmful or Harmless? *Shireene Vethakkan* Metformin In Pregnancy: Is it Still Safe? *Nurain Mohd Noor* Isolated Hypothyroxinemia in Pregnancy: Should We Treat? *Florence Tan Hui Sieng*	**Symposium 12:** **NEONATAL ENDOCRINOLOGY** Chairperson: Teoh Sze Teik Neonatal Grave's Disease *Suhaimi Hussain* Neonatal Hypocalcaemia *Joyce Hong Soo Synn* Neonatal Hypoglycaemia *Lim Poi Giok*

Continue to Next Page >

	Clarke Ballroom	Ballroom B	Ballroom C	Sentral Ballroom
1610 - 1700	Young Investigator Award (Adult Track) ***presentation***	Servier High-Tea Symposium Chairperson: Chan Siew Pheng Era of Diabesity: How Does Gliclazide MR "Fit"? ***Chan Siew Pheng & Nemenio A. Nicodemus, Jr.*** SERVIER	DKSH High-Tea Symposium Chairperson: Wong Ming Optimizing Glycemic Variability with Vildagliptin: Insights from Clinical Practice ***Hew Fen Lee*** DKSH	Young Investigator Award (Paeds Track) ***presentation***
1700 - 1800	Trade Exhibition / *Tour d'MAC*			
*1700 - 1900	MEMS Annual General Meeting (Sentral Ballroom, Hilton Kuala Lumpur)			

Programme

Day 3 - 21st July 2024 (Sunday)

	Clarke Ballroom	Ballroom B + Ballroom C	Sentral Ballroom
0750 - 0830	Amgen Sunrise Symposium Chairperson: Malik Mumtaz Advancing the Management of Osteoporosis in Very High-Risk Population *Jeyakantha Ratnasingam*		
0830 - 1000	**Symposium 13:** **NEURO-DIABETES** Chairperson: Jeyakantha Ratnasingam Dementia and Diabetes *Chin Ai Vyrn* Erectile Dysfunction in Diabetes: Is There Hope? *Zulkifli Md Zainuddin* Vitamin B in Diabetic Neuropathy – Is it Placebo Effect? *Hiew Fu Liong*	**Symposium 14:** **LIPIDOLOGY & CV** Chairperson: Yusniza Yusoff Statin and Antiplatelet for Primary Prevention: Revisited *Norlaila Mustafa* Lipoprotein A – Coming Next Year? *Lim Lee Ling* Residual Risk After LDL to Target: What's Next? *Azhari Rosman*	**Symposium 15:** **ADOLESCENT ENDOCRINOLOGY** Chairperson: Nurshadia Samingan Pubertal Induction in Girls *Lee Yee Lin* T1DM in Adolescents: Overcoming Barriers to Effective Diabetes Care *Noor Shafina Mohd Nor* Graves' Disease in Adolescents: How Can We Do Better? *Annie Leong*
1000 - 1030	Tea Break / Trade Exhibition/ *Tour d'MAC – Second & Final Draw*		
1030 - 1115	**DEBATE – SEASON 2: BARIATRIC SURGERY VS GLP-1 AGONIST** Moderator: Chan Siew Pheng *Nik Ritza Kosai Nik Mahmood* **vs** *Alexander Tan Tong Boon*		
1115 - 1200	Astra Zeneca Lunch Symposium Chairperson: Lim Lee Ling Evidence Road Trip: Mapping the Patient Journey from Evidence to Real Life *Tan Li Ping*		
1200 - 1230	Prize Giving & Closing Ceremony		

Table of Contents

ADULT ORAL PRESENTATION

ADULT BEST POSTER PRESENTATION

ADULT E-POSTER

BASIC SCIENCE E-POSTER

PAEDIATRICS ORAL PRESENTATION

PAEDIATRICS BEST POSTER PRESENTATION

PAEDIATRICS E-POSTER

Adult Oral Presentation

OP_A001

METABOLICALLY HEALTHY OBESITY AND ASSOCIATED RISK FACTORS AMONG MALAYSIAN ADULTS

https://doi.org/10.15605/jafes.039.S1.001

Quan Hziung Lim,[1] Devakumaran Packrisamy,[1] Jia Sheng Ong,[1] Myann Lynn Kang,[1] Jun Kit Khoo,[1] Ying Guat Ooi,[1] Tharsini Sarvanandan,[1] Nicholas Ken Yoong Hee,[1] Sharmila Sunita Paramasivam,[1] Jeyakantha Ratnasingam,[1] Shireene Ratna Vethakkan,[1] Lee-Ling Lim[1,2,3]

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[2]*Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Hong Kong SAR, China*
[3]*Asia Diabetes Foundation, Hong Kong SAR, China*

INTRODUCTION

Metabolically Healthy Obese (MHO) describes the subset of people with obesity (PwO) who do not exhibit overt cardiometabolic abnormalities, namely dysglycaemia, dyslipidemia or hypertension. While Malaysia has one of the highest obesity rates regionally, prevalence of MHO is unclear. This study aims to investigate the local prevalence of MHO among PwO and predisposing factors to Metabolically Unhealthy Obesity (MUO).

METHODOLOGY

Subjects were recruited at Universiti Malaya Medical Centre (UMMC) via convenience sampling. Healthcare workers in UMMC with no known chronic metabolic conditions were invited to participate and were included if their BMI was 23 kg/m^2 and above. Data collected on demographics, anthropometric measurements, bioimpedance analysis (BIA), and a fasting blood sample for glucose and lipid metrics were collected. The subjects were classified as MHO if no metabolic abnormalities were detected, and otherwise as MUO. Statistical analysis was done using SPSS Version 25.

RESULT

Two hundred ninety-seven subjects were recruited. Mean age was 36.26 ± 7.37 years, majority were female (71.00%) and Malay (90.20%). Overall, 172 (57.9%) were classified as MHO. Males were more likely to be MUO, X^2 (1, N=297) =4.09, p = 0.05. The MUO subgroup, compared to MHO subgroup, had significantly higher weight [median=77.65 (IQR:19.15) kg versus 70.55 (14.91) kg; p<0.01], BMI [30.10 (6.00) kg/m^2 versus 26.80 (4.67) kg/m^2, p <0.01], waist [94.40 (4.02) cm versus 90.50 (10.00) cm, p<0.01] and hip [105.40 (5.70) cm versus 101.90 (10.38) cm, p <0.01] circumferences, and waist-to-height ratio (WHtR) [0.59 (0.09) versus 0.56 (0.07), p<0.01] but not waist-to-hip ratio [0.89 (0.11) versus 0.86 (0.09), p >0.05]. On BIA, MUO subgroup had higher body fat percentage (BFP) [42.40 (11.32) % versus 39.90 (9.87) %, p <0.01] and Fat Mass Index (FMI) [12.75 (5.72) kg/m^2 versus 10.65 (4.20) kg/m^2, p <0.01]. After adjusting for age, gender, WHtR, BMI and BFP, neck circumference remained a significant predictor for MUO status [Odds Ratio = 1.16 (95%CI 1.02-1.31), p = 0.01].

CONCLUSION

A large proportion of PwO have MUO. High neck circumference is an independent predictor of MUO status among PwO and should prompt early screening for metabolic disturbances.

OP_A002

BARIATRIC SURGERY IN A PUBLIC HOSPITAL IN MALAYSIA: THREE YEARS EXPERIENCE (2021- 2024)

https://doi.org/10.15605/jafes.039.S1.002

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INTRODUCTION

Obesity in Malaysia has reached an alarming rate and various treatment modalities are available for patients depending on the severity of their obesity. Bariatric surgery has been proven to be part of the armamentarium to combat obesity; however, only a few public hospitals in Malaysia are capable of providing such a niche service. This study reports our experience over the past 3 years in a long-standing multidisciplinary bariatric clinic that was established in 2014 and based in a public hospital.

METHODOLOGY

Retrospective analysis of a prospectively maintained database of all patients undergoing bariatric surgery from January 2021 until April 2024 was performed. We analysed patient demographics, comorbidities and peri-operative complications. Prior to surgery, patients are thoroughly assessed, management of comorbidities is optimised, and the type of procedure and its expected outcomes are discussed in detail. Patients are required to be placed on an outpatient-based very low-calorie diet (VLCD) from 10 to 14 days prior to surgery. Peri-operative deep vein thrombosis prophylaxis is generally practiced unless contraindicated.

RESULT

A total of 125 patients underwent various bariatric procedures (75 sleeve gastrectomies, 40 Roux-en-Y gastric bypasses, 7 sleeve plus procedures, and 3 revisional procedures) with a mean age of 39.5 years and a mean BMI of 49.1 kg/m^2. The percentage of patients with diabetes, hypertension, and dyslipidaemia was 28.8%, 50.4% and 32% respectively. One patient was excluded as she required a conversion back to normal anatomy after gastric bypass. Throughout the study period, 3 major complications (Clavien-Dindo Grade III and above) were observed and 2 patients required readmission within one month due to dehydration.

CONCLUSION

This study shows that a multidisciplinary team with sufficient training and experience can provide bariatric surgery within a constrained public health system.

OP_A003

PREVALENCE AND ASSOCIATED RISK FACTORS OF LOW BONE MINERAL DENSITY IN MALAYSIANS LIVING WITH HIV: A CROSS-SECTIONAL STUDY

https://doi.org/10.15605/jafes.039.S1.003

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INTRODUCTION
The global impact of HIV/AIDS has led to improved survival rates due to treatment efforts, but it has also resulted in a rise in chronic comorbidities such as low bone mineral density (BMD). Contributing factors to low BMD in HIV-positive individuals include the virus itself, treatment, and lifestyle factors. This study aims to describe the prevalence of low BMD and associated risk factors among individuals living with HIV. Findings from this research can potentially influence future screening practices.

METHODOLOGY
This cross-sectional study involved patients aged 25-45 years old with HIV undergoing follow-up in an infectious disease clinic. Participants underwent DXA scans to evaluate bone density, with T scores used to categorize BMD levels according to WHO criteria. Data were analysed to determine associations between low BMD and various risk factors.

RESULT
The study found that 65.5% (226 out of 345) of patients had low BMD, with 91.1% (206) having osteopenia and 8.2% (20) having osteoporosis. Binary logistic regression analysis revealed associations between low BMD and male gender and lower BMI. Additionally, BMD was observed to improve with a longer duration of antiretroviral therapy (ART).

CONCLUSION
The study underscores the high prevalence of low BMD among people living with HIV, emphasizing the need for vigilance, particularly among those with risk factors such as low BMI and male gender. Therefore, it is crucial to pay attention to this population subgroup, promote a healthy lifestyle, normal body weight, and adherence to antiviral medication to enhance overall bone health.

OP_A004

THE NAFLD THREAT IN HYPOTHYROID PATIENTS: A CROSS-SECTIONAL STUDY

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INTRODUCTION
Non-alcoholic fatty liver disease (NAFLD) is a rapidly progressing condition with a concerning rise in prevalence. Studies suggest a potential association between NAFLD and hypothyroidism, another prevalent condition. However, in the Indian context, not much has been reported on this association. This study aimed to determine the prevalence of NAFLD in a cohort diagnosed with hypothyroidism at our hospital.

METHODOLOGY
We conducted a hospital-based, cross-sectional observational study. Patients with confirmed hypothyroidism based on established criteria (elevated TSH with normal or low T4) were recruited. Standardized diagnostic tools for NAFLD were employed, including liver ultrasound to assess the grade of fatty infiltration and Fibro Scan® to measure liver stiffness. Descriptive statistics were used to report the prevalence of NAFLD within the hypothyroid population.

RESULT
A total of 120 patients with hypothyroidism participated (87.2% female, ages 15-66 years, majority 21-50 years). Over half (51.3%) had overt hypothyroidism. We observed a prevalence of NAFLD of 46.7% (31.6% Grade 1 fatty liver, 15% Grade 2) on ultrasound. Notably, the prevalence of NAFLD was higher in overt hypothyroidism (43.5% Grade 0, 32.3% Grade 1, 24.2% Grade 2) compared to subclinical hypothyroidism (62% Grade 0, 31% Grade 1, 7% Grade 2). Fibro Scan® revealed 65% with no fibrosis (F0-F1), 18.3% with F2 fibrosis, and 16.7% with F3 fibrosis. Statistical analysis showed a positive correlation between TSH levels and both NAFLD severity on ultrasound and liver fibrosis on Fibro Scan®.

CONCLUSION
This study investigated the co-occurrence and potential association of NAFLD in patients with hypothyroidism. We found a high prevalence of NAFLD (46.7%) and a positive correlation between TSH levels and NAFLD severity/fibrosis. These findings suggest a potential link between hypothyroidism and NAFLD. Further research is warranted to explore the underlying mechanisms and potential therapeutic implications.

OP_A005

BRAIN NATRIURETIC PEPTIDE: A PREDICTOR OF ADVERSE OUTCOMES IN THYROID STORM?

https://doi.org/10.15605/jafes.039.S1.005

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INTRODUCTION

Brain natriuretic peptide (BNP) is a cardiac biomarker used in the assessment of heart failure. Potentially, BNP levels can be used to stratify patients with thyroid storm (TS) who are at risk of complications such as heart failure and atrial fibrillation. We compared BNP levels in patients admitted with TS or impending TS (study group) with patients with newly diagnosed hyperthyroidism (control group) and to determine if BNP is a predictor of morbidity and mortality in patients with thyroid storm.

METHODOLOGY

This is a cross-sectional study conducted at University Malaya Medical Centre (UMMC) over one year (January 2023-December 2023). Fifty patients with impending TS/TS (TSG) and fifty patients with newly diagnosed hyperthyroidism (HG) were recruited and their serum BNP was measured. Patients' morbidity and mortality outcomes were recorded.

RESULT

FT4 and FT3 levels were higher in impending TS/TS (TSG) compared to newly diagnosed hyperthyroidism (HG): [FT4: 64.5 (27.4-129.1) pmol/L vs 30.5 (21.9-131.2) pmol/L, p <0.001; FT3 13.5 (7.9-45.4) pmol/L vs 13.5 (7.9 – 45.4) pmol/L, p = 0.038]. BNP levels were significantly higher in TSG [TSG vs HG:316 (2.0-8148.0) pg/ml vs 27.0 (2.0-310.0) pg/ml, p <0.001]. Those with impending TS/TS experienced higher rates of respiratory failure, atrial fibrillation, sinus tachycardia, and heart failure. In those with impending TS/TS, 80% had elevated BNP compared to 12% in HG. Those with elevated BNP also had significantly longer hospital stays [TSG vs HG: 5 (1-45) days vs 3 (2-7) days, p = 0.019]. Atrial fibrillation and sinus tachycardia were independently associated with elevated BNP detected by multivariable logistic regression. There was no mortality in either group.

CONCLUSION

This study demonstrated that BNP levels are elevated in impending/thyroid storms and can be potentially used to predict outcomes.

Adult Best Poster Presentation

PP_A001

BILATERAL ADRENAL MASSES: A SINGLE CENTER EXPERIENCE

https://doi.org/10.15605/jafes.039.S1.006

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INTRODUCTION
Bilateral adrenal masses are uncommon and present with varied clinical manifestations and aetiologies. Understanding the prevalence and characteristics of different aetiologies of adrenal masses is crucial for instituting effective management strategies.

METHODOLOGY
A retrospective analysis was conducted on 34 patients with bilateral adrenal masses evaluated at the Endocrine Centre, Hospital Putrajaya, from 2015 to 2024.

RESULT
The mean age of our cohort was 53.7 ± 16.7 years, the majority being male (58.8%). Pheochromocytoma was the most common aetiology (10 cases, 28.6%), presenting at a younger mean age of 41.2 ± 17.5 years, with half of the cases being clinically silent. The mean size of the adrenal masses was 3.47 ± 1.94 cm. Seventy percent of patients had confirmed genetic defects, and all had benign histopathology except one with features of a tumour likely to have a malignant behaviour. This patient had a positive VHL variant. Similarly, non-functioning bilateral adenomas contributed 10 cases (28.6%), which were all asymptomatic. The mean age at presentation was 64.3 ± 8.9 years, and the mean size of the adrenal masses was 1.32 ± 0.44 cm. Chronic infection was found in seven cases (20%) of bilateral adrenal masses (three tuberculosis, two histoplasmoses, one MRSA/salmonella bacteraemia and one adrenalitis from an unknown infectious agent), with 85.7% of them exhibiting symptoms of hypocortisolism. Malignancy represented a minority of cases (4, 11.4%), with one case of adrenal lymphoma and three cases of adrenal metastasis. These cases presented at an older age (mean age: 62 ± 10.3 years) with no disturbance of adrenocortical function. The mean size of the adrenal masses was 5.67 cm for lymphoma and 1.92 ± 0.48 cm for adrenal metastasis. Other etiologies observed included two cases of cortisol-secreting adenoma, one case of adrenal oncocytoma, and one case of unknown cause.

CONCLUSION
This retrospective analysis reveals diverse aetiologies of adrenal masses, including pheochromocytoma, nonfunctioning adenomas, infections, and malignancies. Variability in presentation, age, and size underscores the need for comprehensive evaluation and tailored management strategies for this complex patient population.

PP_A002

ADRENAL INSUFFICIENCY AND ITS ASSOCIATED FACTORS AMONG MULTI-ETHNIC PEOPLE LIVING WITH HIV/AIDS CONSULTING AT TERTIARY HOSPITALS

https://doi.org/10.15605/jafes.039.S1.007

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INTRODUCTION
Adrenal insufficiency (AI) is one of the most common potentially life-threatening endocrine complications in people living with HIV/AIDS. This study aims to describe AI among them, where data on this topic are scarce.

METHODOLOGY
A cross-sectional study was conducted at the Infectious Diseases clinics of two tertiary centres. A total of 110 stable adults living with HIV/AIDS, aged 18 to 80 years old from the outpatient setting, were recruited. Clinical and laboratory data were collected and cosyntropin stimulation tests were performed. Adrenal antibody was analysed among those who had an inadequate response.

RESULT
The mean age of the studied population was 37.5 ± 10.6 years. Majority of the subjects were male (81.8%). Only 23.6% of subjects had a normal BMI, with 10.9% classified as underweight and 64.5% as overweight/obese. The mean disease duration was 6.6 ± 6.2 years. All subjects were receiving highly active antiretroviral therapy (HAART), with a mean treatment duration of 4.9 ± 4.2 years. Among these, 22 individuals (20%) experienced treatment failure, and 46 individuals (41.8%) had a history of opportunistic infections. Five individuals (4.5%) had autoimmune disease. The mean nadir CD4 count among the studied population was 202 cells/mm^3, with 60% of them having a nadir CD4 <200 cells/mm^3. At the time of the cosyntropin stimulation test, the majority of individuals, 97 (88.2%), had a viral load (VL) <40 copies/ml, and 89 (89.1%) had a CD4 count >200 cells/mm^3. In the assessment for adrenal insufficiency, 7 individuals (6.4%) exhibited symptoms suggestive of adrenal insufficiency. However, only 2 individuals (1.8%) were found to have an inadequate response to cosyntropin. Adrenal antibodies were negative, and ACTH was not elevated in these two patients.

CONCLUSION
This study reveals a low prevalence of AI (1.8%) among stable patients with HIV/AIDS on antiretroviral therapy. Individualized screening and ongoing monitoring are crucial. These findings emphasize the importance of tailored care strategies for this population.

PP_A003

EARLY METABOLIC AND WEIGHT LOSS OUTCOMES IN LAPAROSCOPIC SLEEVE GASTRECTOMY PLUS PROXIMAL JEJUNAL BYPASS (SLEEVE PJB)

https://doi.org/10.15605/jafes.039.S1.008

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INTRODUCTION

Sleeve plus procedures are sleeve gastrectomy variations that include numerous procedures such as SADI-S (single anastomosis duodenoileal bypass with sleeve), loop duodenojejunal bypass with sleeve gastrectomy (Loop DJB) and sleeve gastrectomy plus proximal jejunal bypass (Sleeve PJB). We have performed Sleeve PJB for selected patients in our centre.

METHODOLOGY

We did a retrospective analysis of patients who underwent laparoscopic sleeve PJB in Hospital Raja Permaisuri Bainun Ipoh in 2023. We analysed demographics, weight, BMI and HbA1c. All patients had a sleeve gastrectomy utilizing our standard operative technique. For the proximal jejunal bypass (PJB), jejunum was transected at 50 cm distal to the ligament of Treitz. Subsequently, another 270 cm to 300 cm of jejunum was measured from the divided region and a side-to-side jejuno-jejunostomy was done.

RESULT

A total of 6 patients underwent this procedure. All patients were not amenable to gastric bypass and wanted a more durable surgery in terms of weight loss and metabolic outcomes. Four of them had pre-diabetes and two patients had diabetes based on HbA1c. Cumulative mean age, weight and BMI were 44 years, 109 kg and 44 kg/m^2 respectively. The HbA1c for the prediabetes and diabetes group preoperatively were 6% and 8% respectively. Postoperatively at 6 months, the HbA1c was 5.4% and 5.9% respectively for each group. Cumulative postoperative mean current weight and mean percentage of total weight loss (%TWL) at 6 months were 91.5 kg and 20.57%.

CONCLUSION

This study shows that early outcomes for sleeve PJB are favourable and likely a non-inferior option for patients who are not amenable to a standard gastric bypass.

PP_A004

IMPACT OF METABOLIC SURGERY ON CARDIOMETABOLIC RISK FACTORS IN OBESE PATIENTS WITH TYPE 2 DIABETES, PREDIABETES AND NORMOGLYCAEMIA

https://doi.org/10.15605/jafes.039.S1.009

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INTRODUCTION

Metabolic surgery has shown promising results in managing obesity-associated conditions, including type 2 diabetes mellitus (T2DM). Despite its efficacy, its impact on obese individuals at different stages of T2DM remains underexplored, particularly in Malaysia. This study aimed to assess the effect of bariatric surgery on cardiometabolic risk factors in obese patients and compare outcomes among patients with T2DM, prediabetes, and without diabetes over 12 months.

METHODOLOGY

This study included 86 obese patients from various centres in Klang Valley, Malaysia, who underwent metabolic surgery procedures such as laparoscopic sleeve gastrectomy, Roux-en-Y gastric bypass, mini-gastric bypass, and one-anastomosis gastric bypass. They were stratified into three groups based on baseline HbA1c measurements according to Malaysia's CPG of Management of Type 2 Diabetes Mellitus 6th Edition: no diabetes (n = 42), prediabetes (n = 25), and diabetes (n = 19). Changes in weight, HbA1c, lipid profiles, and liver function markers (ALT, AST, and GGT) were evaluated at 6 and 12 months. Postoperative changes in cardiometabolic risk factors were analysed using the generalized estimating equations.

RESULT

The patients were mostly female (67.4%) and Malay (65.3%), with a mean age of 39.0 ± 7.7 years and a mean BMI of 39.7 ± 8.1 kg/m^2. Significant improvements were observed in weight, HbA1c, HDL-C, triglycerides, AST, ALT, and GGT levels after 12 months across all groups (p <0.05), with the diabetes group showing the most substantial improvement in most variables. Comparison between groups highlighted significant differences in mean HbA1c and TG levels between the diabetes and prediabetes groups compared to the normoglycemia group (p <0.01).

CONCLUSION

Metabolic surgery improves cardiometabolic risk factors in obese patients, particularly in those with T2DM. Its potential benefits extend to individuals at high risk of developing diabetes, underscoring the need for further research to assess their eligibility for surgical intervention.

PP_A005

VITAMIN D DEFICIENCY IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE AND TYPE 2 DIABETES MELLITUS

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INTRODUCTION

Low vitamin D levels have been associated with type 2 diabetes mellitus and non-alcoholic fatty liver disease. Insulin resistance, inflammation and oxidative stress have been suggested to be responsible for the development of NAFLD. These mechanisms are linked to vitamin D's anti-inflammatory effect. Vitamin D deficiency is highly prevalent among patients with T2DM and NAFLD and is an independent risk factor for developing NAFLD in patients with T2DM.

METHODOLOGY

The study aimed to determine the prevalence and associated risk factors of vitamin D deficiency in patients with NAFLD and T2DM. We conducted a cross-sectional study in patients with T2DM (n=110). The patients were divided into 2 groups: NAFLD (n=86) and no NAFLD (n = 24). The patients within the NAFLD group were further divided into 2 groups (vitamin D deficient and non-deficient). Serum total 25 (OH) D3 was analysed using electrochemiluminescence immunoassay (Roche), and deficiency was defined as a level of <50 nmol/L. Diagnosis of NAFLD was based on the abdominal ultrasound performed by 2 experienced radiologists. Steatosis was defined as increased liver echogenicity.

RESULT

The prevalence of vitamin D deficiency in patients with T2DM and NAFLD was 52.3% (45/86), as compared to only 33.8% (8/24) in those without NAFLD. Vitamin D level was significantly lower in patients with NAFLD as compared to those without NAFLD (51.53 ± 19.68 vs 60.61 ± 20.25, p <0.05). There was no difference in the age (57.74 ± 8.42 vs 57.96 ± 7.97 years, p = 0.91), gender (male 55.7% vs 70.8%, p = 0.18), BMI (28.99 ± 3.80 vs 28.24 ± 3.74 kg/m^2, p = 0.40), and diabetes duration (12.43 ± 7.92 vs 10.42 ± 6.51, p = 0.25), between the 2 groups. Multivariate analysis demonstrated that HbA1c (AOR 1.89; 95%CI 1.15-3.09; p = 0.01) and vitamin D deficiency were the independent risk factors for NAFLD (AOR 3.15 95%CI 1.10-9.04; p = 0.03), after adjustment for age, gender, diabetes duration and eGFR.

CONCLUSION

This study demonstrated a high prevalence of vitamin D Deficiency in patients with NAFLD and T2DM. In patients with T2DM, those with vitamin D deficiency were three times more likely to develop NAFLD. Vitamin D levels of <50 nmol/L and higher HbA1c were the two independent risk factors for developing NAFLD.

PP_A006

T1DM PATIENTS IN HOSPITAL SELAYANG: CLINICAL CHARACTERISTICS AND ASSOCIATION WITH RECURRENT DKA

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INTRODUCTION

Type 1 Diabetes Mellitus (T1DM) patients in Malaysia are characterized by poor glycaemic control and with high prevalence of diabetic ketoacidosis (DKA) among children and adolescents. This study aimed to examine the clinical characteristics of adult T1DM patients and to explore the association with recurrent DKA.

METHODOLOGY

All adult T1DM patients under Endocrine Unit follow-up in Selayang Hospital were included. Socio-demographic data associated with co-morbidities, macrovascular and microvascular complications, acute complications such as hypoglycaemia or DKA, metabolic profile and anthropometric measurements were extracted from medical records. Diabetes distress was evaluated using the diabetic distress scale (DDS).

RESULT

This cohort included 97 patients with a median age of 28 (IQR 15) and 64.9% of them were male. The ethnic distribution was Malay (51.5%), Chinese (29.9%) and Indian (15.5%). Majority (57.9%) received only primary or secondary school education while 75.8% were from the B40 (bottom-tier household income) category. The median disease duration was 10.0 (IQR 12.0) years. Seventy-one percent of Insulin users were on an analogue insulin-based regimen. Mean HbA1c was 9.9 ± 2.9%. Diabetes complications, predominantly retinopathy and diabetic kidney disease were present in 29.9% of the cohort.

Of the cohort, 35.1%, had recurrent DKA in the past 5 years. This was associated with Indian ethnicity (p = 0.047), lower education group (p = 0.023), higher diabetes distress (p = 0.013), higher HbA1c (p = <0.001), non-obese (p = 0.017) and patients with diabetes complications such as retinopathy (p = 0.012) and diabetic kidney disease (p =<0.001). The association remained significant in the adjusted analysis for higher HbA1c (OR 1.352, 95%CI 1.073, 1.703), Indian ethnicity (OR 12.956, 95%CI 1.962, 85.56), retinopathy (OR 8.087, 95%CI 1.309, 49.95), diabetic kidney disease (OR 9.173, 95%CI 1.888, 44.56) and non-obese (OR 44.543, 95%CI 2.644, 750.43).

CONCLUSION

Our T1DM cohort had poor glycaemic control with a high burden of recurrent DKA associated with higher HbA1c, greater diabetic distress, microvascular complications and a non-obese state. Further research is warranted to explore the causal association between these factors to develop effective strategies to improve glycaemic control and the burden of DKA.

Adult E-Poster

EP_A001

ADRENAL INCIDENTALOMA: THE CLUES TO AID DIAGNOSIS

https://doi.org/10.15605/jafes.039.S1.012

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INTRODUCTION/BACKGROUND
Adrenal incidentaloma (AI) is found while imaging for a different purpose when there are no overt signs of adrenal illness. A thorough history and examination of a patient with adrenal insufficiency may provide more hints to help diagnose and narrow the differentials.

CASE
The first patient is a 25-year-old male with hypertension and chronic diarrhoea. Blood investigation showed deranged liver function. Ultrasound of the abdomen revealed a heterogenous hyperechoic mass at the right suprarenal region measuring 6.9 x 5.8 x 7.5 cm (APxWxCC) which is compressing the adjacent right liver lobe. Twenty-four-hour urinary-free metanephrine demonstrated that metanephrine and normetanephrine levels were twenty times higher than the upper limit of the reference value.

For the second patient, a 49-year-old female with hypertension and asthma presented with acute asthma exacerbation at the ED and POCUS showed an incidental finding of a right liver mass. Ultrasound of the abdomen showed a well-defined, heterogeneous hypoechoic, mixed solid-cystic lesion superior to the right kidney measuring 8.0 x 7.3 x 9.4 cm, suggestive of a right adrenal mass. Twenty-four-hour urinary-free metanephrine showed elevated normetanephrine 37.40 umol/24H (0.88-2.88).

The third patient is a 49-year-old female who presented with abdominal discomfort, anorexia and weight loss of 3 kg over 3-4 months. Colonoscopy and OGDS yielded normal results. Ultrasound of the abdomen showed a large, heterogeneous lobulated lesion seen in the left retroperitoneal region measuring 11.9 x 5.4 x 6.2 cm. Twenty-four-hour urine-free metanephrine was normal. Corticoadrenal carcinoma was ruled out. Serial CT of the adrenal done two months apart showed a rapid increase in the size of the left adrenal mass with multiple enlarged lymph nodes. CT-guided biopsy of the left adrenal revealed primary diffuse large B cell lymphoma.

CONCLUSION
Hypertension is common in patients with adrenal insufficiency. Symptoms and blood investigation can give clues to the specific adrenal hyperfunction present which can help narrow down the differentials, thus reducing the cost of work-up in a resource limited centre. A patient with phaeochromocytoma might be asymptomatic and a low level of urine metanephrine could be due to a necrotic tumour. Computed tomography of the adrenal is essential to assess the characteristics of the lesion to further risk stratify the patient.

EP_A002

TWO'S COMPANY - UNEXPECTED MEN 2 PRESENTING AS INCIDENTALOMA

https://doi.org/10.15605/jafes.039.S1.013

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INTRODUCTION/BACKGROUND
Bilateral pheochromocytomas are uncommon, classically described as occurring in 10% of cases. Such presentation should raise suspicion of the presence of a hereditary syndrome or predisposing genetic mutations.

CASE
A 43-year-old female presented with renal colic. A computed tomography scan showed nonobstructing renal calculi and bilateral adrenal incidentaloma measuring ~3.8 cm. Serum calcium was normal (2.16 mmol/L). Twenty-four-hour urinary metanephrines were elevated sevenfold above normal with normal normetanephrines. Physical examination was unremarkable except for labile blood pressure (SBP 135-170 mmHg). She only reported symptoms of occasional palpitations and mild headache. Family history was significant for the death of unknown cause of her mother and 2 elder siblings before the age of 60 years. MEN 2A was suspected. Ultrasound of the neck revealed four TIRADS-5 thyroid nodules measuring 0.7-1.3 cm, FNAC confirmed medullary thyroid carcinoma (MTC). Carcinoembryonic antigen was 13.6 ng/ml (<5).

She underwent bilateral retroperitoneoscopic adrenalectomy uneventfully following adrenergic blockade. Post-operatively, she was normotensive and received hydrocortisone and fludrocortisone replacement. Histopathological examination confirmed bilateral pheochromocytoma with

no malignant features. She awaits total thyroidectomy. Her kindred were advised to undergo screening for MEN 2, albeit the lack of genetic study due to financial constraints.

CONCLUSION

Genetic testing for RET proto-oncogene would be useful to guide management and screening in MEN 2. Medullary thyroid carcinoma is the most common manifestation of MEN 2 with 100% penetrance and should be actively sought for in patients suspected of having MEN 2.

EP_A003

RIFAMPIN-INDUCED ADRENAL CRISIS

https://doi.org/10.15605/jafes.039.S1.014

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INTRODUCTION/BACKGROUND

Rifampicin is an essential first-line anti-tuberculosis drug. It is crucial for medical practitioners practicing in countries such as Malaysia where tuberculosis is endemic to recognize that rifampicin, an enzyme inducer, can have serious drug-drug interactions and needs to be used cautiously.

CASE

We describe a case of a 30-year-old male who sustained a mild traumatic brain injury with cerebrospinal fluid leakage in 2022. His injury was complicated by panhypopituitarism and secondary adrenal insufficiency, which required hydrocortisone 10mg/5mg BD and desmopressin replacement. He was compliant to hormonal replacement and remained asymptomatic throughout regular follow-up. In February 2024, he presented with submandibular swelling that turned out to be tuberculous lymphadenitis with pulmonary tuberculosis. He was started on first-line antituberculosis medications (Akurit-4), containing rifampicin, isoniazid, pyrazinamide and ethambutol with his usual dose of hydrocortisone. Three days after the initiation of anti-tuberculosis medication, the patient presented with vomiting, fever with postural dizziness without polyuria. Blood pressure was 102/64 mmHg, with postural hypotension and hypoglycaemia. The patient was diagnosed with adrenal insufficiency secondary to rifampicin.

The patient was started on intravenous hydrocortisone 50 mg QID. Laboratory investigations revealed serum cortisol of <27 nmol/L with adrenocorticotropic hormone level of 0.36 pmol/L. After adequate hydrocortisone replacement, the patient had polyuria with a gradual reduction of serum sodium to 125 mmol/L, unmasking the presence of central diabetes insipidus. Desmopressin was started and the patient clinically improved with normalisation of serum sodium.

CONCLUSION

In patients with pre-existing adrenal insufficiency, initiation of an anti-tuberculosis regimen containing rifampicin may increase the metabolism of cortisol by inducing cytochrome CYP3A4 activity and precipitate an adrenal crisis. Before initiation of anti-tuberculosis medications, drug-drug interaction should be reviewed. In such cases, dose adjustment of hydrocortisone is necessary to prevent adrenal insufficiency. Increasing the hydrocortisone dose gradually and close monitoring of the patient's biochemical and clinical state are important to reduce the risk of adrenal crisis and mortality.

EP_A004

THE RIFAMPICIN RED FLAG

https://doi.org/10.15605/jafes.039.S1.015

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INTRODUCTION/BACKGROUND

Rifampicin is an essential first-line anti-tuberculosis (TB) drug which exhibits potent hepatic enzyme-inducing properties. It has significant drug interactions with an array of other medications, including hydrocortisone as we report in this case.

CASE

A 65-year-old male, HIV positive, treatment-naive, with concurrent primary adrenal insufficiency (Synacten done: Cortisol 0 hour 247.8 nmol/L, 60 minutes 316 nmol/L, and normal ACTH 7.76 pmol/L) on hydrocortisone 10 mg/5 mg replacement for 4 months was admitted for prolonged fever and lethargy. He was diagnosed to have extrapulmonary TB by urine lipoarabinomannan (LAM) test and was started on isoniazid, rifampicin, pyrazinamide plus ethambutol – HREZ regime.

On Day 12 of HREZ, he exhibited hypoglycaemia, postural hypotension, and hyponatremia. Serial monitoring of his sodium levels showed a decreasing trend from a normal level initially of 135 mmol/L to a nadir of 116 mmol/L on day 21 of rifampicin. A diagnosis of adrenal insufficiency secondary to rifampicin was made. Rifampicin accelerates cortisol metabolism resulting in low levels of serum cortisol.

The patient was started on IV hydrocortisone 50 mg QID. He responded well to treatment with amelioration of symptoms and normalization of sodium levels. Steroids were then tapered to oral hydrocortisone with the lowest replacement dose of 20 mg/10 mg daily (double the usual physiological dose) given the ongoing use of rifampicin. The patient was started on hydrocortisone tablet 20/10 mg daily and with no further dose reduction planned while concurrently on rifampicin. The hydrocortisone dosage will be gradually reduced to the standard physiological dose upon the patient's completion of rifampicin treatment.

CONCLUSION

Prompt identification of drugs that can affect cortisol metabolism is essential to for patients on hydrocortisone replacement therapy. Close monitoring, multidisciplinary collaboration, personalized dose adjustments and careful tapering of hydrocortisone with biochemical and clinical correlation are paramount in navigating the challenges posed by rifampicin-hydrocortisone interaction.

EP_A005

A DECADE OF INITIAL EXPERIENCE IN ADMINISTRATION OF METAIODOBENZYLGUANIDINE THERAPY FOR ADVANCED STAGE PARAGANGLIOMA AND PHEOCHROMOCYTOMA

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INTRODUCTION/BACKGROUND

Metaiodobenzylguanidine (MIBG) labeled with radioactive iodine can be utilised for imaging and therapy in advanced stage paraganglioma and pheochromocytoma. Our centre became a local pioneer and started to offer MIBG therapy in 2013. Patients received 200 mCi of Iodine-131 MIBG for each therapy session. We present a case series to highlight the clinical complexity of these rare endocrine neoplasms and our early experience with MIBG therapy.

CASE

The first case involves a 57-year-old male with a large, right pheochromocytoma diagnosed in 2013. Recurrence was noted post-debulking surgery and chemoembolisation. He had 2 MIBG therapies between 2015 and 2016. Surveillance showed a stable underlying tumour and decreasing urine metanephrine level. However, he developed a metastatic pleural nodule and multiple abdominal nodes in 2021. The third MIBG therapy was given in October 2022. Stable disease was noted on a follow-up MIBG scan in April 2023 with markedly decreasing serum Chromogranin A (CgA).

For the second case, a 74-year-old male diagnosed with retroperitoneal paraganglioma in 2002 underwent surgery but presented back with metastatic lesions involving the liver and right ilium in 2012. He received 3 cycles of MIBG therapy between 2015 and 2017. Unfortunately, he deteriorated over the subsequent 18 months due to progressive multiple liver, abdominal nodes, lungs and skeletal metastases.

The third case is a 50-year-old male with subhepatic paraganglioma diagnosed in 2017. Transarterial embolisations were done as surgery was deemed infeasible. He had 3 MIBG therapies between 2018 and 2020. Surveillance in 2021 showed a stable, focal upper abdomen lesion and decreasing CgA level. However, he developed metastatic disease progression with rebound CgA elevation in February 2022. Fourth MIBG therapy was given in September 2022. A MIBG-avid subhepatic mass was seen with extensive skeletal and cervical, thoracic and abdominal node metastases.

CONCLUSION

MIBG therapy may offer potential palliative benefit in pheochromocytoma and paraganglioma as seen among cases with a solitary large lesion or oligometastasis. However, advanced stage diseases especially those with bone metastasis show a poorer prognosis.

EP_A006

ANCIENT SCHWANNOMA: A GREAT MIMICKER OF ADRENAL TUMOUR

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INTRODUCTION/BACKGROUND

Retroperitoneal schwannoma is a benign neoplasm arising from the neural crest cells. Pre-operative diagnosis of this rare tumour is often difficult due to its enormous size at the time of presentation and the lack of distinctive imaging phenotypes. We share a case of an incidentally discovered huge right periadrenal ancient schwannoma in an elderly patient who suffered from an underlying nasopharyngeal carcinoma.

CASE
A 70-year-old male was found to have a 15-cm right suprarenal mass when he underwent CT scan for the staging of nasopharyngeal carcinoma. He had no paroxysmal symptoms or hypertension. There were no features of Cushing syndrome. Endocrine evaluation showed no evidence of functioning pheochromocytoma or adrenocortical carcinoma. The patient underwent open adrenalectomy and tumour excision uneventfully. Pathology examination revealed a large ancient schwannoma consisting of spindle cells with nuclear and cytoplasmic S-100 positivity.

CONCLUSION
The pre-operative diagnosis of retroperitoneal schwannoma remains challenging despite the advances in imaging modalities. Th definitive diagnosis relies on biopsy or resection.

EP_A007

THE FAST AND FURIOUS CUSHING'S SYNDROME

https://doi.org/10.15605/jafes.039.S1.018

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INTRODUCTION
Ritonavir is a commonly prescribed protease inhibitor for human immune deficiency (HIV) treatment. It is a potent inhibitor of hepatic cytochrome P450 (CYP450) enzyme. Interaction between ritonavir and corticosteroids induces iatrogenic Cushing's Syndrome. We share a case of an acute onset of Cushing's Syndrome in a young female with HIV.

CASE
A 25-year-old female with stable retroviral disease on ritonavir along with tenofovir, emtricitabine and atazanavir developed Cushing's syndrome within 2 weeks of receiving injectable hydrocortisone from a general practitioner's clinic for skin itchiness. Facial swelling, hirsutism, abdominal striae, body acne, weight gain and proximal myopathy were noted. Early morning cortisol was 28 nmol/L and the 24-hour urine-free cortisol was 45 nmol/day. She was diagnosed with iatrogenic Cushing's syndrome with suppression of the hypothalamic-pituitary adrenal (HPA) axis secondary to drug interaction between ritonavir and intravenous hydrocortisone. She was started on oral hydrocortisone 20 mg in the morning and 10 mg in the evening. Throughout her hospitalization and upon discharge, she remained clinically well. She is planning for a Synacthen test on an outpatient basis to reassess her HPA axis.

CONCLUSION
Drug interaction between ritonavir and corticosteroids may result in increased levels of plasma corticosteroids, potentiated by the CYP450 metabolism which prolongs the half-life of hydrocortisone, that can lead to Cushing's syndrome. This highlights the importance of a thorough review of the patient's medications to prevent drug-to-drug interaction. If corticosteroid administration cannot be avoided, the patient needs to be monitored for symptoms of Cushing's syndrome.

EP_A008

A CASE OF CLINICALLY AND BIOCHEMICALLY SILENT GIANT PHEOCHROMOCYTOMA

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INTRODUCTION/BACKGROUND
With the widespread use of computed imaging and genetic testing, up to 60% of pheochromocytomas are diagnosed in the presymptomatic stage, particularly when the lesion is smaller than 3 cm. We report a rare case of clinically and biochemically silent giant pheochromocytoma.

CASE
A 44-year-old Malay male with a two-year history of hypertension was initially admitted to the surgical team for gallbladder empyema. However, abdominal CT showed a lobulated, heterogeneously enhancing mass with an area of necrosis at the right peritoneal region measuring 11 x 13.5 x 15.2 cm. Subsequent ultrasound-guided biopsy of the mass revealed pheochromocytoma. He was then referred to the Endocrine team for further management. No paroxysmal symptoms were reported by the patient and his blood pressure was well-controlled on a single antihypertensive. Laboratory workup including 24-hour urine catecholamines and 24-hour urine metanephrine were not elevated. Thus, 68Ga/Dotatate scan was performed, which demonstrated evidence of somatostatin receptor avid malignancy in the abdominal mass with no evidence of regional or distant metastasis. Following the scan, serum chromogranin A (CgA) was sent and was found to be elevated (2682.4 ng/ml, normal range: 27-94 ng/ml). After adequate alpha- and beta-blockade, he successfully underwent right adrenalectomy with complete removal of the mass with no complications intra- and postoperatively. The HPE of the mass reported the presence of a well-circumscribed tumour focally encapsulated by a thin fibrous capsule, with the absence of necrosis and invasion

of the vascular, adrenal capsular and periadrenal soft tissue, with a Ki-67 proliferative index of 1%. A repeat abdominal CT done two months post-operatively showed no evidence of local recurrence and a normal CgA level (85.8 ng/ml).

CONCLUSION
Surgery is the primary treatment for pheochromocytoma, and pre-operative alpha- and beta-blockade are essential regardless of tumour size and biochemical status. In patients without elevated levels of catecholamines, CgA is the alternative functional diagnostic and surveillance marker.

EP_A009

PHEOCHROMOCYTOMA: AN OVERLOOKED CONDITION IN HYPERTENSIVE DISORDER IN PREGNANCY

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INTRODUCTION/BACKGROUND
Pheochromocytoma is a rare tumour derived from chromaffin cells of the adrenal medulla or extra-adrenal paraganglia. It is a rare cause of secondary hypertension and is commonly overlooked in pregnancy due to limitations in investigation during pregnancy. It occurs in 0.1-1% of the hypertensive population and is even more rare in pregnancy.

CASE
A 33-year-old pregnant female with a parity of 8, diagnosed with chronic hypertension and with a history of severe preeclampsia in her previous pregnancy, presented again at 34 weeks of gestation with severe preeclampsia. Blood pressure was 179/124, and heart rate ranged from 100 to 120 bpm. Urinary examination revealed proteinuria. Despite treatment with conventional antihypertensives, her blood pressure remained uncontrolled. Thus, she was planning for emergency C-section. Intubation was done due to cardiorespiratory compromise, which was complicated by cardiac arrest. She was successfully resuscitated. Computed tomography (CT) of the adrenal glands showed a large, heterogeneously enhancing right adrenal lesion measuring 7.4 x 7 x 8 cm. Twenty-four-hour urinary catecholamine levels were elevated, with normetanephrine at 67.80 umol/day (0-2.13), metanephrine at 97.30 umol/day (0-1.62), and 3-methoxytyramine at 7.60 umol/day (0.1-1.79). The classical presentation of pheochromocytoma with paroxysmal hypertension, headaches, sweating, and palpitations may not be simultaneously present, especially during pregnancy. Labile BP and difficult to control hypertension should raise suspicion for pheochromocytoma, to prompt appropriate investigations that will facilitate an early diagnosis. Measurements of urinary or plasma catecholamines have reasonable sensitivity for detecting most pheochromocytomas, particularly in patients with sustained hypertension. Radioisotope scans, including iodine 131-labeled metaiodobenzylguanidine scanning, should be avoided during pregnancy due to foetal concerns and, if required, should be postponed until the postpartum period.

CONCLUSION
Pheochromocytoma in pregnancy is a life-threatening condition. Early suspicion and recognition are essential to prevent fetomaternal morbidity and mortality.

EP_A010

DISSEMINATED HISTOPLASMOSIS WITH BILATERAL ADRENAL INFILTRATION AND PRIMARY ADRENAL INSUFFICIENCY

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INTRODUCTION/BACKGROUND
Histoplasmosis is a fungal disease caused by *Histoplasma capsulatum* and characterized by two forms: pulmonary and disseminated histoplasmosis. In the latter form, adrenal infiltration is a common feature, resulting in detection of bilateral adrenal masses radiologically. Bilateral extensive destruction of the adrenal glands results in primary adrenal insufficiency (PAI), which occurs in 5–71% of adrenal histoplasmosis. We present a case of PAI with adrenal histoplasmosis.

CASE
A 71-year-old male, with underlying diabetes, hypertension, and pulmonary tuberculosis, presented with bloody diarrhea and thrombocytopenia. Multiple ulcers were observed over the dorsal surface of the tongue. The histopathological examination (HPE) of the tongue was consistent with histoplasmosis. Colonoscopy examination was unremarkable. The patient was referred to an infectious disease team and was prescribed a course of itraconazole for six weeks.

A year later, he had recurrent bloody diarrhea, and repeated colonoscopy revealed inflamed rectal mucosa. Histopathological examination revealed chronic proctitis with noncaseating granulomas that were consistent with fungal infection. Intravenous amphotericin B

was administered, followed by oral isavuconazole and itraconazole. Computed tomography (CT) scan demonstrated enlarged bilateral adrenal glands with the right side measuring 4.9 x 2.6 x 6.8 cm, and the medial limb of the left adrenal gland measuring 4.3 x 2.5 cm. The lateral limb of the left adrenal gland was 3.6 x 2.1 cm. Ten weeks after antifungal therapy was started, CT scan revealed a smaller left adrenal lesion, but the right adrenal lesion remains unchanged. Short synacthen test showed PAI with peak cortisol 246 nmol/L, ACTH 20.3 pmol/L (1.6-13.9). He is awaiting adrenal biopsy pending urinary metanephrines. Glucocorticoid replacement was initiated. Antifungal therapy would be continued for no less than one year.

CONCLUSION

Adrenal histoplasmosis is common and histopathological analysis is crucial in managing such cases. It is important to be vigilant about infections like histoplasmosis as a potential cause of PAI. Delay in treatment could result in life-threatening consequences.

EP_A011

MALIGNANT PARAGANGLIOMA IN AN ADOLESCENT

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INTRODUCTION/BACKGROUND

Pheochromocytomas (PCC) and paragangliomas (PGL) (PPGL) are rare neuroendocrine tumours occurring in children and adolescents. Nevertheless, they are the most common endocrine tumours in the paediatric population and account for 0.5–1% of paediatric hypertensive cases. We describe a 16-year-old female with malignant paraganglioma.

CASE

A previously healthy 16-year-old female presented with a one-month history of intermittent headaches associated with palpitations and presyncopal attacks. The first blood pressure reading revealed that she was hypertensive, with a BP of 159/116. She had no chest pain, shortness of breath, diaphoresis, abdominal pain, or diarrhoea. There is no family history of hypertension in the young or endocrine disorder. The patient is lean with a BMI of 14.2 kg/m^2. No goitre, cushingoid or acromegalic features were present. The hormonal workup done was consistent with phaeochromocytoma (normetanephrine: 55.30 umol/day) (35.5 X ULN)). Other forms of work-up for secondary hypertension were unremarkable. Adrenal CT imaging revealed an enhancing mass at the left pararenal space measuring 4.1 x 4.7 x 4.7 cm with local infiltration to the tail and body of the pancreas complicated by a left renal infarct. Therefore, she was diagnosed with left paraganglioma with local infiltration. Preoperatively, she was started on oral prazosin 1 mg, 6 hourly and oral bisoprolol 2.5 mg daily and successfully underwent open resection of the left paraganglioma. Unfortunately, she remained hypertensive post-surgery, indicating a possible malignant paraganglioma. Thus, she was restarted on antihypertensive medications. Gallium-DOTATE scan and genetic testing have been arranged to aid further management.

CONCLUSION

Diagnosis of pheochromocytoma and paraganglioma is paramount during the evaluation of secondary hypertension in the paediatric population. Although they are uncommon, possible curative surgery can be offered. All children should be subjected to genetic testing given the high rate of inheritance of these tumours. Subsequently, all patients with genetic mutations ought to be under lifelong surveillance in view of the risk of recurrence and malignancy.

EP_A012

A RARE CASE OF UNILATERAL ADRENAL LYMPHOMA WITH LYMPHADENOPATHY

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INTRODUCTION/BACKGROUND

Adrenal lymphoma is an extremely rare and highly invasive malignant disease. We report a rare case of unilateral adrenal lymphoma with lymphadenopathy.

CASE

A 68-year-old male presented with abdominal discomfort, polyuria and weight loss of 15 kg over 6 months. Physical examination revealed a thin elderly man with fullness over the left lumbar and inguinal lymph nodes. Laboratory tests showed markedly elevated lactate dehydrogenase (LDH) levels of >690 UI/L (<248) and hypercalcemia. A computed tomography (CT) scan revealed a large left adrenal mass (11.6 x 8.3 x 9.6 cm) with multiple matted abdominal lymph nodes, raising a suspicion of adrenal malignancy. Following this, hormonal profile was done which showed normal cortisol and catecholamines. An ultrasound-guided trucut biopsy of the right inguinal lymph nodes was performed. The microscopic examination showed a malignant tumour composed of mononuclear cells with pleomorphic nuclei with high mitotic figures. On immunohistochemistry, the tumour cells were positive for vimentin, CD20, CD10,

BCL-6, leucocytes common antigen (LCA) and Ki67 proliferating index was 60% in the tumour cells. A diagnosis of diffuse large B cell lymphoma (DLBCL) was made. The patient was referred to the haematology team and started on chemotherapy.

CONCLUSION

Primary adrenal lymphoma is a rare entity with a generally poor prognosis. They usually involve both adrenal glands, but unilateral adrenal involvement can occur in about one-third of patients. In patients with large adrenal masses and constitutional symptoms, the initial dilemma is to differentiate between adrenal carcinoma versus other forms of malignancies or chronic infections. Adrenal biopsy is generally avoided in suspected adrenal carcinoma as it may be harmful because it can lead to tumour seeding. In our patient, the presence of multiple lymphadenopathy which was accessible for biopsy helped clinch the diagnosis of adrenal lymphoma. The presence of lymphadenopathy with large adrenal masses, even if unilateral, should prompt suspicion of the diagnosis of adrenal lymphoma.

EP_A013

CT STAGING THAT UNVEILS A MYSTERY – ASYMPTOMATIC PHEOCHROMOCYTOMA ASSOCIATED WITH NEUROFIBROMATOSIS TYPE 1

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INTRODUCTION/BACKGROUND

Pheochromocytomas and paraganglioma (PPGL) are catecholamine-secreting tumours, derived from chromaffin cells. The classical triad comprises paroxysms of headache, palpitation, and diaphoresis. About 10% of patients are asymptomatic. At least one-third of patients with PPGLs have hereditary disease caused by germline mutations. This includes neurofibromatosis type 1 (NF-1) which may predispose patients to pheochromocytoma and occurs in 0.1-5.7% of cases.

We report a case of a 57-year-old female with hypertension and diabetes who was referred from the surgical team after an incidental finding of a right adrenal mass on abdominal CT performed for rectal adenocarcinoma staging. She denied paroxysms or other symptoms that suggest catecholamine or cortisol excess. Blood pressure was well controlled with a single agent. Examination revealed axillary freckling, multiple cafè au lait spots and generalized cutaneous nodules which were present since adolescence.

CASE

Serial abdominal CT scans showed increasing size of right adrenal mass measuring 4.4 x 5.4 x 5.9 cm (previously 4.4 x 5.1 x 5.6 cm) with presence of fluid-fluid level within, with HU ranging from HU 20 (anteriorly) and HU 70 (posteriorly). Metanephrine 5.42 umol/L (0.33-1.53), normetanephrine 8.0 umol/L (0.88-2.88) and 3-methoxytyramine 1.16 umol/L (0.66-2.60) were elevated on 24-hour urine collection. Thyroid function test and serum calcium were normal. Histopathological examination of the cutaneous nodule confirmed neurofibromas. She underwent open right adrenalectomy and HPE was consistent with pheochromocytoma. Three months post adrenalectomy, urine metanephrines had normalized, and there was no tumor residual or recurrence on CT imaging. She no longer requires any anti-hypertensive drug, and we were able to withdraw insulin therapy.

CONCLUSION

Though rare, the combination of NF-1 with pheochromocytoma in our patient is an offbeat presentation of adrenal incidentaloma in a patient with multiple cutaneous nodules, hypertension and diabetes. Screening for pheochromocytoma should be done in a patient with NF-1 and hypertension. Prompt treatment will alleviate the detrimental effect of catecholamine excess and improve the patient's quality of life.

EP_A014

CORTISOL DAY CURVE TO GUIDE GLUCOCORTICOID REPLACEMENT IN A PATIENT WITH ADRENAL INSUFFICIENCY ON ANTI-TUBERCULOSIS THERAPY

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INTRODUCTION/BACKGROUND

Hydrocortisone in divided doses (typically 15 – 25 mg/day) is the most common form of glucocorticoid replacement regimen in patients with adrenal insufficiency (AI). However, this may be inadequate for patients on CYP3A4 inducers

which affect glucocorticoid metabolism. While there are no specific guidelines on dose adjustment, cortisol day curve (CDC) could be used to guide optimal replacement. We report a case of a female with AI secondary to pituitary tuberculosis requiring hydrocortisone dose adjustment following initiation of anti-tuberculosis therapy (anti-TB).

CASE

A 45-year-old female presented with a worsening headache for a month, without any constitutional symptoms. Imaging revealed a heterogenous sellar mass (1.3 x 1.5 x 2.1 cm) without chiasmal compression or cavernous involvement. Blood investigations showed serum cortisol <14 nmol/L (reference interval 145-619), FT4 10 pmol/L (reference interval 11.5 – 22.7) and TSH <0.01 m IU/L (reference interval 0.55-4.78). Other blood investigations were normal. She was commenced on hydrocortisone 10 mg BD (8 am, 2 pm) and levothyroxine 50 mcg OD. Trans-sphenoidal resection was performed, and histopathology revealed necrotising granulomatous inflammation with caseating necrosis. The diagnosis of pituitary tuberculosis was made, and first line anti-TB drugs (rifampicin, isoniazid, pyrazinamide, and ethambutol) were initiated. Despite pre-emptively increasing hydrocortisone to 20 mg BD, she experienced postural hypotension, lethargy, and nausea soon after initiation of anti-TB medications, especially in the early afternoon and evening. Cortisol day curve was done by measuring serum cortisol hourly from 8 am – 8 pm while she took her regular hydrocortisone 20 mg BD. Serum cortisol levels were <14 nmol/L (8 am), 1009 nmol/L (9 am), 664 nmol/L (10 am), 386 nmol/L (11 am), 217 nmol/L (12 pm), 88 nmol/L (2 pm), 761 nmol/L (3 pm), 857 nmol/L (4 pm), 521 nmol/L (5 pm), 256 nmol/L (6 pm), and 85 nmol/L (8 pm). Hydrocortisone was adjusted to 20 mg (8 am), 10 mg (1 pm) and 5 mg (6 pm) to counter the trough levels. This led to a marked improvement in her symptoms.

CONCLUSION

CYP3A4 inducers (anti-TB) affect glucocorticoid metabolism and replacement in patients with AI. The cortisol day curve could be used as a guide to tailor therapy in situations where adequate replacement doses and timing are not easily identified.

EP_A015

A PERPLEXING MIXED CORTICAL AND MEDULLARY ADRENAL NEOPLASM PRESENTING WITH INDETERMINATE ADRENAL MASS

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INTRODUCTION

A mixed cortical and medullary adrenal neoplasm originating in the adrenal gland is an exceedingly rare tumour characterized by the coexistence of cell nests from both the adrenal cortex and medulla within a singular mass, which results in the production of adrenocortical steroid hormones and catecholamines (CA). We report a case of mixed cortical medullary adrenal neoplasm presenting with indeterminate adrenal mass.

CASE

A 51-year-old female, with a known case of left breast mucinous carcinoma, underwent a right mastectomy and axillary clearance and completed 6 cycles of systemic chemotherapy. The computed tomography of the thorax, abdomen and pelvis (CT TAP) surveillance post-treatment revealed left adrenal indeterminate incidentaloma. Computed tomography adrenal protocol showed that the left adrenal mass was heterogeneous with a size of 1.8 x 2.2 x 2.4 cm, with a plain HU of 18 HU, absolute contrast washout of 21%, and relative contrast washout of 15%. Biochemically, both the overnight dexamethasone suppression test (ODST) and low dose dexamethasone suppression test (LDDST) were not suppressed with values of 81 nmol/L and 119 nmol/L, respectively. A 24-hour urine metanephrine collection demonstrated results within the normal range: metanephrines 0.4 umol/day (0.1-6.2), normetanephrine 1 umol/day (0.2-1.3) and 3-methoxytyramine 0.8 umol/day (0.1-1.79). Given that the left adrenal indeterminate incidentaloma had autonomous cortisol secretion, a left adrenalectomy was performed. Remarkably, the histopathological examination (HPE) unveiled a mixed corticomedullary adrenal tumour.

CONCLUSION
A mixed cortical and medullary adrenal neoplasm is extremely rare. Therefore, knowledge about long-term clinical course and prognosis is limited. Long-term follow-up is recommended to assess the recurrence in the contralateral adrenal gland.

EP_A016

A SINGLE METASTATIC LARGE ADRENAL MASS MIMICKING ADRENOCORTICAL CELL CARCINOMA

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INTRODUCTION
Invasive breast ductal carcinoma commonly metastasizes to the lungs, liver, bones and brain. Solitary adrenal metastasis from invasive ductal carcinoma is extremely rare. Hence, a single large adrenal metastasis can mimic a primary adrenal tumour such as adrenocortical carcinoma. We report a case of a rare single metastatic large adrenal mass from invasive ductal carcinoma mimicking adrenocortical carcinoma.

CASE
A 49-year-old female, with a known case of invasive ductal carcinoma of the right breast underwent right mastectomy and axillary clearance and completed 6 cycles of systemic chemotherapy and 15 cycles of radiotherapy. The FDG-PET scan surveillance post-treatment showed FDG-avid left adrenal mass suggestive of metastasis or primary malignancy with no other solid organ or bone metastasis. Subsequent computed tomography (CT) scan also showed a large, irregular lobulated solid left adrenal mass measuring 7.3 x 4.8 x 5.4 cm. Plain HU was 30 HU and the calculated absolute washout was 55%. A large left adrenal mass with the absence of other solid organs and bone metastasis made adrenocortical carcinoma one of the possible aetiologies. Biochemical investigation to assess adrenal tumour functionality, namely, overnight dexamethasone suppression test (ODST), aldosterone renin ratio (ARR) and 24-hour urine metanephrines all yielded normal results. Left open adrenalectomy was performed and histopathological examination (HPE) confirmed left adrenal metastasis from the breast carcinoma.

CONCLUSION
Single large solitary adrenal metastasis from invasive breast ductal carcinoma is rare and can mimic adrenocortical carcinoma. Early recognition and adrenalectomy will probably lead to improved patient survival.

EP_A017

A RARE CASE OF ANTIPHOSPHOLIPID SYNDROME PRESENTING AS ADRENAL CRISIS AND BILATERAL ADRENAL HEMORRHAGE

https://doi.org/10.15605/jafes.039.S1.028

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INTRODUCTION/BACKGROUND
Bilateral adrenal haemorrhage is a rare condition with potentially life-threatening consequences due to adrenal crisis. It can be the first presentation of antiphospholipid syndrome (APLS). We report a rare case of APLS presenting as an adrenal crisis and bilateral adrenal haemorrhage.

CASE
A 37-year-old male with a history of a motor vehicle accident 2 weeks ago presented with abdominal pain for 1 week, accompanied by nausea, vomiting, and lethargy. During the accident, where his motorbike skidded, he sustained an open fracture of the proximal phalanx of his little finger. Wound debridement was performed and a K-Wire was inserted. Clinical examination showed left-hand cellulitis, tenderness over the bilateral flank, and slightly low blood pressure (90/46 mmHg). Blood investigations revealed thrombocytopenia, hyponatremia, prolonged APTT that did not correct in the coagulation mixing study, and a low cortisol level (67 nmol/L). Abdominal CT scan revealed bilateral adrenal haemorrhage without any other solid organ and bowel injury. He was treated for acute adrenal crisis and subsequently started on hydrocortisone and fludrocortisone, resulting in significant clinical improvement. However, the left-hand cellulitis triggered digital artery thrombosis, leading to left-hand gangrene. Despite anticoagulation and ilioprost administration, he ended up with a left transradial amputation. Antiphospholipid syndrome was suspected in this patient based on the bilateral adrenal haemorrhage, digital artery thrombosis and abnormal coagulation profile. A full autoimmune work-up confirmed the presence of anticardiolipin, lupus anticoagulant, and anti-beta-2 glycoprotein. The anti-nuclear antibody was also positive 1:320, with a speckled pattern. C3, C4, anti-double

stranded DNA and extractable nuclear antibodies were otherwise negative. He was discharged well on warfarin, hydrocortisone and fludrocortisone.

CONCLUSION

Adrenal insufficiency is a rare manifestation of APLS. This case highlights the importance of maintaining a high index of suspicion for APLS in patients presenting with bilateral adrenal haemorrhage and thrombotic events. Failure to diagnose or treat this condition promptly may lead to significant morbidity and mortality.

EP_A018

CLINICAL CONUNDRUM OF STEROID RESISTANT TESTICULAR ADRENAL REST TUMOURS (TARTS): CASE SERIES

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INTRODUCTION/BACKGROUND

Testicular adrenal rest tumours (TARTs) are benign intratesticular masses that occur in male patients with congenital adrenal hyperplasia (CAH), with more than 90% of cases caused by a deficiency of 21-α-hydroxylase. The presence of TART is an important complication leading to irreversible gonadal dysfunction and infertility. TARTs appear to be associated with poor hormonal control with concomitant elevated ACTH. The current mainstay of therapy is intensified glucocorticoid therapy. We describe two challenging cases of steroid-resistant TARTs.

CASE

Case 1 is a 25-year-old male with classical CAH diagnosed at 7 weeks of life. At age 12, he was diagnosed with TARTs. With a 17-OHP elevated to >60.6 nmol/L, his glucocorticoid dose was intensified with dexamethasone 0.5 mg ON for 5 years together with fludrocortisone. The latest 17-OHP was 436.7 nmol/L (1-10), ACTH 80.6 pmol/L (1.6-13.9), Direct renin >550 mU/L (5.3-99.1) and testosterone 21.64 nmol/L (0.58 - 31.28). Testicular ultrasonography demonstrated unresolved TARTs 1.0 x 1.1 x 1.4 cm (right) and 1.3 x 1.2 x 2.3 cm (left). Dexamethasone was subsequently switched to prednisolone 2.5 mg BD together with a referral to the urology team for consideration of testicular-sparing surgery or semen cryopreservation.

Case 2 is a 20-year-old male with classical CAH diagnosed as a one-month-old infant and diagnosed with TARTs at age 13 years. Apart from fludrocortisone, his glucocorticoid therapy was intensified with dexamethasone 2.5 mg ON (tapering dose) for 5 years with his latest 17-OHP 116.4 nmol/L, testosterone 23.06 nmol/L, ACTH 128.9 pmol/L and Renin 47.2 mU/L. Testicular ultrasonography revealed increased size of TARTs 2.0 x 1.9 x 3.3 cm (right) and 1.7 x 1.5 x 2.9 cm (left). His case was complicated by exogenous Cushing syndrome secondary to dexamethasone. Dexamethasone was switched to prednisolone 2.5 mg BD with a referral to the urology team for co-management.

CONCLUSION

Intensified glucocorticoid therapy has led to tumour size reduction and improved testicular function in only a subset of patients. However, this approach potentially leads to serious side effects. Further research should aim to identify pharmacological alternatives that can effectively prevent the development of TARTs and treat existing TARTs to improve fertility outcomes.

EP_A019

MANAGING STEROIDS IN A FEMALE WITH NCCAH COMPLICATED WITH COVID-19 IN PREGNANCY

https://doi.org/10.15605/jafes.039.S1.030

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INTRODUCTION

We report a case of a female with Non-Classical Congenital Adrenal Hyperplasia (NCCAH) which was complicated by COVID-19 during her pregnancy. This is the second such case to be reported so far; the first being in Italy in 2020. The role of steroid management throughout her pregnancy is highlighted.

CASE

This is a case of a 28-year-old female diagnosed with NCCAH since age 21 and maintained on oral prednisolone 7.5 mg OD pre-pregnancy. At the 20th week of gestation, she was switched to oral hydrocortisone 10 mg BD as she developed mild Cushingoid features. Consequently, she presented at 30 weeks of gestation to the ED with symptoms of breathlessness, dehydration and compensated shock and was diagnosed with COVID-19 Category 5. She received fluid boluses and low-dose inotrope and was placed under ICU care due to worsening hypoxia. She was initiated on maintenance fluids and Remdesivir for COVID-19 and intravenous hydrocortisone 100 mg bolus with maintenance of 50 mg QID to prevent an adrenal crisis. In the ICU, her condition stabilized with inotropic and oxygen support weaned off. Her intravenous hydrocortisone was maintained at 50 mg QID. Throughout her stay, no foetal compromise was observed. She was discharged

well on day 5 of illness with a supraphysiological dose of hydrocortisone 20 mg/10 mg. Further steroid adjustments were planned at the outpatient clinic on follow-up.

CONCLUSION

Managing acute COVID-19 infection during late pregnancy, especially with an underlying adrenal condition, poses significant challenges due to therapeutic uncertainties. A multidisciplinary team and close ICU monitoring were vital for ensuring a successful outcome for both mother and child. Steroid management in NCCAH patients, particularly during pregnancy, is critical. Early treatment with appropriate antivirals and steroids can mitigate illness progression and severity, reducing morbidity and mortality associated with COVID-19.

EP_A020

SPECTRUM OF ADRENAL INFECTIONS – FROM SOFT TO HARD: CASES OF ADRENAL ABSCESS AND CALCIFICATION

https://doi.org/10.15605/jafes.039.S1.031

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INTRODUCTION

Adrenal infections can present in a spectrum from adrenalitis to abscess and calcification. It may either affect the adrenals unilaterally or bilaterally. We present two cases of adrenal infections: a unilateral adrenal abscess following ERCP and a bilateral adrenal calcification due to Histoplasmosis.

CASE

The first case is a 54-year-old male, with a history of alcoholism and diabetes, who presented with abdominal pain. Initial CT-abdomen showed chronic pancreatitis with a right adrenal lesion measuring 4.8 cm. ERCP done showed pancreatitis with infected pseudocyst. However, he presented back 2 months later with fever, abdominal pain and constitutional symptoms. CT-abdomen revealed a small liver abscess and a right adrenal abscess measuring 9 cm with an average HU of 61. He was treated with antibiotics for 8 weeks and underwent abscess drainage. The hormonal work-up was within normal range with adrenal insufficiency ruled out. All bacterial, tuberculous and fungal work-up were negative. A repeat CT of the abdomen after 4 months showed a residual adrenal abscess measuring 4.8 cm and a right adrenalectomy was scheduled.

The second case is a 45-year-old male, a smoker with hypertension, who presented with constitutional symptoms, skin darkening, fever and features of adrenal crisis. Steroids and antibiotics were started. CT of the abdomen showed enlarged and calcified bilateral adrenals measuring 5 cm. Primary adrenal insufficiency was confirmed biochemically. CT-guided biopsy showed fibrous and necrotic tissue and PAS, GMS and Ziehl-Nielsen stains were negative. Adrenal tissue PCR was positive for *Histoplama capsulatum* and a diagnosis of adrenal histoplasmosis was made. The patient underwent a two-week treatment with Amphotericin B and continued with oral Itraconazole-planned for 1 year. He showed improved general health and increased weight. Repeat CT of the adrenals after 3 months showed no significant change.

CONCLUSION

Adrenal infections have various presentations and can affect both immunocompetent and immunocompromised patients. Treatment of the underlying organism with antimicrobial therapies and steroid replacement is key to avoiding significant morbidity and mortality.

EP_A021

CARNEY COMPLEX: THE CASE OF A RARE ENDOCRINE SYNDROME

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INTRODUCTION/BACKGROUND

Carney complex (CNC) is a rare multiple endocrine neoplasia characterized by spotty skin pigmentation, myxomas and endocrine overactivity. We report a case of a young female with multiple typical manifestations of CNC over the past 12 years, including bilateral primary pigmented nodular adrenocortical disease, bilateral breast ductal adenoma, cardiac myxoma and thyroid nodule.

CASE

This female first presented at age 28 for secondary amenorrhea, weight gain and uncontrolled hypertension. She had pigmentation over her lips and features of Cushing syndrome, such as facial plethora, purple striae and proximal myopathy. She had a right breast fibroadenoma at age 17 and young-onset hypertension at age 25 on past medical history. There were no familial diseases noted. The initial work-up was suggestive of ACTH-independent Cushing syndrome. The adrenal CT showed a 2.3 x 1.2 cm right adrenal adenoma and a normal left adrenal gland. She underwent a right adrenalectomy with a tissue histopathology suggestive of pigmented nodular adrenal-

cortical hyperplasia. She was then subjected to a left adrenalectomy as she remained hypercortisolemic after the initial surgery. She went into remission after the bilateral adrenalectomy. At age 29, a surveillance scan showed a left solitary thyroid nodule and multiple bilateral breast lumps with a tissue biopsy suggestive of ductal adenoma. Excision of atrial myxoma was done at age 33 following the detection of cardiac myxoma from an echocardiogram when she complained of palpitations. Unfortunately, she was diagnosed with left breast carcinoma at age 38, requiring a left mastectomy. A recent tissue biopsy of a right breast lump showed intraductal papilloma.

CONCLUSION

The diagnosis of CNC is often delayed owing to its rarity and complexity. Clinical and biochemical screening are the gold standard for the diagnosis of CNC. This patient requires a lifelong follow-up for the recurrence of cardiac myxoma and other associated manifestations of CNC.

EP_A022

ASSESSING THE POTENTIAL OF DULAGLUTIDE IN DE-INTENSIFICATION OF BACKGROUND ORAL GLUCOSE-LOWERING DRUG (OGLD) AND INSULIN THERAPY IN MALAYSIANS WITH TYPE 2 DIABETES MELLITUS

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INTRODUCTION/BACKGROUND

Many Malaysian T2DM patients are on multiple glucose-lowering drugs (i.e. ≥2 OGLDs ± insulin). Dulaglutide, a once-weekly GLP-1RA, has been shown to significantly lower HbA1c levels in T2DM patients. However, there is a lack of real-world data to show the reduction of background treatment after patients start dulaglutide.

METHODOLOGY

This study aims to assess the potential of dulaglutide in de-intensifying background OGLDs and total daily dose (TDD) of insulin in T2DM patients in a real-world clinical setting. This is a retrospective study of 45 T2DM patients who initiated dulaglutide in 3 Ministry of Health (MOH) hospital-based endocrinologist-led diabetes clinics conducted in Hospital Putrajaya, Hospital Selayang and Hospital Tuanku Ja'afar. The primary outcome was a change in OGLDs and insulin therapy at 6 and 12 months of dulaglutide therapy.

RESULTS

At baseline, 91% (n = 41) of patients were on ≥2 OGLDs, while 82% (n = 37) were on insulin therapy with a mean baseline TDD of 64 units. After 6 months of dulaglutide therapy, 18% (n = 8) of the patients had at least one of their OGLD doses reduced, 38% (n = 17) of patients were able to stop one OGLD, and 4% (n = 2) of patients were even able to stop two OGLDs. At 12 months, 22% (n = 10) of patients had at least one of their OGLD doses reduced, 40% (n = 18) of patients were able to stop one OGLD, 9% (n = 4) of patients were able to stop two OGLDs from baseline, 56% (n = 25) of insulin-treated patients on dulaglutide had a TDD reduction of 23 units (-36%) at 6 months and 19 units (-30%) at 12 months.

CONCLUSION

Dulaglutide, with its once-weekly dosing, can effectively simplify patients' diabetes treatment by allowing the reduction of OGLDs and TDD of insulin. This de-intensification of medication could reduce the medication burden on patients and lessen the total drug cost for T2DM patients.

EP_A023

ASSESSING THE REAL-WORLD EFFICACY OF DULAGLUTIDE IN MALAYSIAN MOH PATIENTS WITH TYPE 2 DIABETES MELLITUS

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INTRODUCTION/BACKGROUND

An estimated 70% of Type 2 Diabetes Mellitus (T2DM) patients treated in Ministry of Health (MOH) hospital-based diabetes clinics are still unable to achieve HbA1c targets despite combination glucose-lowering drugs. Moreover, more than 80% of these patients are overweight or obese. In Malaysia, dulaglutide, a once-weekly GLP-1RA, was approved in 2018 for use in patients with T2DM. Accessibility to GLP-1RA therapy is much limited in MOH hospitals.

METHODOLOGY

This study aims to assess the glycaemic and weight-lowering efficacy of dulaglutide at 6 and 12 months in T2DM patients treated in a real-world clinical setting. We conducted a retrospective study of 69 T2DM patients who initiated dulaglutide in 4 MOH endocrinologist-led

hospital-based diabetes clinics (Hospital Putrajaya, Hospital Selayang, Hospital Kuala Lumpur, and Hospital Tuanku Ja'afar). The primary outcome was HbA1c reduction at 6 months of dulaglutide therapy, while the secondary outcomes were HbA1c reduction at 12 months and weight loss at 6 and 12 months.

RESULTS

In this study, the patients' mean baseline age, HbA1c and weight were 54 years old, 8.33% and 91.2 kg, respectively. The mean absolute reduction of HbA1c at 6 months was -0.93% and -0.87% at 12 months. The percentage of patients that achieved ≥2%, 1-2% and 0.5-<1% HbA1c reductions were 16%, 28% and 21% at 6 months, respectively, and 17%, 33% and 11%, at 12 months, respectively. For the secondary outcome analyses, patients experienced a mean weight loss of 3.73 kg at 6 months, and 4.83 kg at 12 months. The percentage of patients that achieved ≥10 kg, 5-10 kg and 1-<5 kg weight reductions at 6 months were 13%, 25%, and 34%, respectively; and at 12 months, 16%, 22% and 41%, respectively.

CONCLUSION

Dulaglutide therapy was shown to be effective in reducing HbA1c and weight at 6 and 12 months of therapy in Malaysian patients with type 2 diabetes currently treated with at least two or more OGLDs, with or without insulin.

EP_A024

MINIMED™ 780G ADVANCED HYBRID CLOSED-LOOP SYSTEM IN TYPE 1 DIABETES DURING RAMADAN: A SINGLE CENTRE EXPERIENCE

https://doi.org/10.15605/jafes.039.S1.035

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INTRODUCTION/BACKGROUND

The MiniMed™ 780G automated insulin delivery system has improved outcomes for people with type 1 diabetes. In Putrajaya Hospital, most patients with type 1 diabetes fall in the high-risk category and are advised to avoid fasting during Ramadan, yet many still choose to observe it.

METHODOLOGY

We aim to review the effectiveness and safety of the MiniMed™ 780G use during Ramadan. We report a prospective observational, single-centre study of Type 1 diabetes patients using the MiniMed™ 780G during Ramadan 2024. Four patients were selected and had their CareLink personal data extracted before and during Ramadan to examine safety and glycemic metrics. Changes were made to their pump settings when necessary.

RESULTS

All patients were able to fast for more days with the MiniMed™ 780G compared to previous years, with a mean of 13.5 days (8-20 days) on the pump vs 8.3 days (3-12 days) on basal-bolus insulin. All our patients demonstrated hyperglycemia after Iftar which needed 4-5 hours to resolve. Three of 4 patients developed hypoglycemia 1-4 hours before iftar requiring intervention. One patient developed one episode of severe hypoglycemia requiring hospitalization. No patients developed diabetic ketoacidosis. The average TIR was 72% before Ramadan and 70% during Ramadan.

CONCLUSION

The MiniMed™ 780 G increased the number of days of completion in fasting among our patients. However, incidences of hypoglycemia and hyperglycemia persisted requiring adjustments in the pump settings throughout Ramadan. This system allowed our patients to fast confidently and safely. Improvement in the outcome is to be expected with continuous experience in the future.

EP_A025

RECURRENT HYPOGLYCEMIA IN A TEENAGER WITH OBESITY: A CASE REPORT

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INTRODUCTION

Reactive hypoglycaemia is a condition of postprandial hypoglycemia occurring within 2 to 5 hours after meal intake. This condition is characterised by inappropriately increased blood insulin levels due to pancreatic over-activity to carbohydrates, most often refined sugars, thus producing hypoglycaemic symptoms. Recent studies have shown that the prevalence of T2D in obese children and adolescents is 0.18–7.9%, which is five times that in normal-weight individuals.

CASE

This is a case of a 16-year-old female with a BMI of 34.28 kg/m^2 who presented with frequent symptoms of hypoglycaemia, mainly palpitations, sweating, giddiness and syncopal attacks since January 2023. The frequency of symptoms was noted 5 to 6 times per week, commonly occurring 4 hours post-meal and after strenuous activity.

She practices a heavy dietary intake 4 times per day with refined carbohydrates at each meal. Clinically, the patient exhibits signs of insulin resistance such as acanthosis nigricans. She underwent a mixed-meal tolerance test in November 2023 which showed no clinical and biochemical evidence of hypoglycaemia. Following that, continuous glucose monitoring was arranged for a week which showed hypoglycaemic episodes ranging from 3.5 to 3.9 mmol/L in the afternoon of one of the days. She was prescribed Acarbose but declined treatment due to gastrointestinal intolerance. She opted for a high fibre, low glycaemic index diet with frequent small meals which showed improvement of the symptoms.

CONCLUSION

Lifestyle modifications are the mainstay of management and prevention of the development of diabetes mellitus for patients with reactive hypoglycaemia. Furthermore, studies have shown that the addition of metformin or acarbose also plays a vital value in preventing reactive hypoglycaemia.

EP_A026

GLYCEMIC CONTROL AND BODY WEIGHT EFFECTS OF SGLT2 INHIBITORS (EMPAGLIFLOZIN 25 MG, EMPAGLIFLOZIN 12.5 MG AND DAPAGLIFLOZIN 10 MG) IN THE TREATMENT OF TYPE 2 DIABETES MELLITUS: A SINGLE CENTRE STUDY

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INTRODUCTION/BACKGROUND

Sodium-glucose co-transporter 2 (SGLT2) inhibitors have emerged as promising therapeutic agents for the management of type 2 diabetes mellitus (T2DM), offering a novel mechanism of action that targets renal glucose reabsorption.

METHODOLOGY

This study investigates the glycaemic control and body weight effects of SGLT2 inhibitors, specifically empagliflozin 25 mg, empagliflozin 12.5 mg and dapagliflozin 10 mg, in the context of their availability within Hospital Teluk Intan. This is a cross-sectional study which involved patients who had been prescribed SGLT2 inhibitors for a duration exceeding one year. Inclusion criteria encompassed patients meeting the specified duration of SGLT2 inhibitor use, while exclusion criteria comprised individuals with less than one year of SGLT2 inhibitor therapy, those procuring SGLT2 inhibitors independently, those admitted within one year of commencing SGLT2 inhibitors and those lacking documented body weight data due to mobility constraints. Patient records were systematically reviewed to extract demographic details and pertinent clinical parameters, including pre- and post-initiation measurements of glycated haemoglobin (HbA1c), body weight and insulin dosage.

RESULT

The study included 24 patients taking dapagliflozin 25 mg, 14 patients on empagliflozin 12.5 mg and 3 patients on dapagliflozin 10 mg, all meeting the inclusion criteria with available data. Among those on empagliflozin 25 mg, there was no significant reduction in HbA1c or weight. In the empagliflozin 12.5 mg group, while HbA1c reduction was not significant, there was a notable decrease of 3.1 kg in body weight. Similarly, in the dapagliflozin 10 mg group, HbA1c reduction was not significant, but there was a weight reduction of 2.7 kg post-treatment.

Initial observations from the enrolled participants suggest significant improvements in body weight, indicating a potential benefit of SGLT2 inhibitors, particularly empagliflozin 12.5 mg and dapagliflozin 10 mg, in fostering weight loss among T2DM patients. However, further examination is necessary to determine the statistical significance of these results and understand the extent of the effect across various doses and types of SGLT2 inhibitors.

CONCLUSION

This study offers valuable insights into the impact of SGLT2 inhibitors, including empagliflozin and dapagliflozin, on glycaemic control and body weight management in T2DM patients. The findings highlight the potential of SGLT2 inhibitors in addressing weight concerns, albeit without significant effects on glycaemic control.

EP_A027

GAPS IN THE MANAGEMENT OF TYPE 2 DIABETES MELLITUS AMONG WOMEN LIVING WITH HIV IN AN ACADEMIC MEDICAL CENTRE

https://doi.org/10.15605/jafes.039.S1.038

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INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) poses significant cardiovascular risks. Individuals with HIV face compounded risks due to accelerated aging, chronic inflammation and certain antiretroviral therapies (ART). Postmenopausal women have increased cardiovascular risk. However, data on women living with HIV (WWH) and T2DM in Malaysia is scarce.

METHODOLOGY

This study examines gaps in the management of T2DM and cardiometabolic health among WWH undergoing routine HIV care in a tertiary hospital. We reviewed the electronic medical records of WWH with T2DM attending the Infectious Diseases (ID) Clinic at Universiti Malaya Medical Centre (UMMC) in 2023. We extracted HIV demographics, anthropometrics, latest HbA1c, fasting plasma glucose (FPG) and lipid levels, and defined targets for control as per the 6th Malaysian Clinical Practice Guidelines for T2DM. Gaps in care were defined as the proportion not achieving targets for control of metabolic parameters.

RESULTS

We collected data from 33 WWH with T2DM, representing 17.8% of all WWH in ID UMMC. Their median age was 54 years (IQR 49, 61) and the median duration since HIV diagnosis was 19 years (IQR 14, 25). All were on ART, with 30 (90.9%) having suppressed viral loads. Menopause status was recorded in 57.6% (n=19), with 78.9% (n=15) being menopausal. Four (12.1%) were active smokers. For T2DM management, 26 (96.3%) were on oral antidiabetic drugs (OADs) and 1 (3.7%) was on insulin. The most used OADs were metformin (65.7%), followed by sulfonylurea (25.7%) and SGLT2i (2.9%). Overall, 60.6% (n = 33) met the HbA1c target of <7% and 66.7% had an FPG within 4.4-7.0 mmol/L (n = 30/33). For lipids, 54.8% (n = 31/33) had triglycerides ≤1.7 mmol/L, 67.7% (n=31/33) HDL >1.2 mmol/L, and 56.7% (n = 30/33) LDL ≤2.6 mmol/L. 72.7% were on statins. For BP, only one (0.03%) had readings within the target range. 36.4% were on an ACE inhibitor or angiotensin-receptor blocker. Only 33.4% had an ideal BMI.

CONCLUSION

There are significant gaps in managing T2D among WWH. Addressing these gaps requires interdisciplinary collaboration for integrated care solutions.

EP_A028

ANALYSIS OF DIABETIC KETOACIDOSIS CASES IN HOSPITAL TELUK INTAN IN 2023

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INTRODUCTION

There is an increase in the incidence of diabetes mellitus (DM) in Malaysia and worldwide. Diabetic ketoacidosis (DKA) is one of the most serious acute complications of diabetes and is even the first presentation of diabetes in some patients.

METHODOLOGY

This study aims to evaluate the incidence and outcome of DKA patients in a district hospital Hospital Teluk Intan (HTI) in 2023. This is a retrospective audit which included every patient who was admitted for DKA in HTI from January 2023 until December 2023. The data was collected from clinical notes and electronic medical records.

RESULTS

Forty subjects were included in this audit which comprised 20 males and 20 females. The median age of the study population was 53.5 years and most patients were Malay (82.5%), followed by Indian (10%) and Chinese (7.5%). Most of them had type 2 DM [34 (87.5%)] whereas the rest had type 1 DM [4 (10.3%)] and newly diagnosed DM [1 (2.6%)]. The most common causes of DKA were sepsis [22 (55%)] and non-compliance to medications [16 (40%)]. The mean HbA1c during admission was 13.9% and the median length of stay was 5.5 days. Twelve patients (30%) had severe DKA and 17 patients (42.5%) required ICU/HDU admission in which 2 patients (5%) required intubation. About 2/3 of patients [24 (60%)] had DKA resolution within 24 hours of admission and most patients were discharged home [37 (92.5%)]. The low mortality rate could be due to early diagnosis and high admission to ICU/HDU.

CONCLUSION
The high number of DKA cases is mainly caused by sepsis and non-compliance to medication, which are critical areas to address to prevent DKA occurrences. While infections may be inevitable, it is essential to counsel diabetes patients on the importance of strict adherence to their medications to avoid serious complications.

EP_A029

A CLINICAL AUDIT ON STATIN THERAPY AMONG TYPE 2 DIABETES MELLITUS PATIENTS ATTENDING PUSAT PERUBATAN ANGKATAN TENTERA (PPAT), SUNGAI BESI, MALAYSIA

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INTRODUCTION/BACKGROUND
Type 2 Diabetes Mellitus (T2DM) patients are at higher risk of developing atherosclerotic cardiovascular disease (ASCVD), which leads to increased morbidity, mortality and use of healthcare resources. Therefore, the primary prevention of ASCVD can be achieved by prescribing the appropriate doses of statin therapy depending on the patient's risk. This is a clinical audit on the use of statin therapy among type 2 Diabetes Mellitus patients at PPAT, Sungai Besi.

METHODOLOGY
This clinical audit aims to improve the quality of care of adult patients with T2DM in preventing ASCVD by utilizing the T2DM Clinical Practice Guidelines (CPG) 2020. A retrospective clinical audit was conducted using a convenient sampling method that involved 32 medical records from PPAT, Sungai Besi. Adults aged above 40 years with T2DM diagnosed for more than six months, and under active follow-up, were included. The criteria were based on the T2DM Clinical Practice Guidelines (CPG) 2020, with standards set at 90% and 50% based on literature reviews.

RESULT
A total of 32 medical records were audited. Most subjects were male (53%), with a median age of 54 years. 90.63% of T2DM adults were on statin therapy. The usage of statin therapy in T2DM patients above 40 years old is satisfactory and achieved the standard of 90%. However, only 20.69% were on high-intensity statin therapy and did not achieve the standard of 50%.

CONCLUSION
These issues need to be addressed by training healthcare providers. Enhancing clinic protocols to address relevant issues is imperative to enhance overall diabetes care, particularly ensuring appropriate utilization of statin therapy in T2DM patients who are either at high risk or very high risk of ASCVD.

EP_A030

UNVEILING EARLY CARDIOVASCULAR DISEASE PREDICTION IN TYPE 2 DIABETES: POTENTIAL ROLE OF CARDIOMETABOLIC BIOMARKERS

https://doi.org/10.15605/jafes.039.S1.041

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INTRODUCTION/BACKGROUND.
Type 2 diabetes individuals are at higher risk of developing cardiovascular disease compared to the general population. Cardiovascular disease remains the leading cause of death in type 2 diabetes despite vigilant monitoring. Early detection of type 2 diabetes patients predisposed to cardiovascular complications is important to reduce the disease burden.

METHODOLOGY
This study aimed to investigate the potential role of cardiometabolic biomarkers in cardiovascular risk prediction among type 2 diabetes patients. A case-control

study consisting of type 2 diabetes with cardiovascular disease outcome, type 2 diabetes without cardiovascular complications and healthy control group was conducted in 221 participants. We employed a machine learning algorithm to develop a cardiovascular risk prediction model.

RESULTS

A combination of sociodemographic, anthropometry and routine biochemical data was assessed using ensemble classifier as the base model for predicting cardiovascular risk (84.8% accuracy, 76.5% positive predictive value in high-risk). The predictive ability was improved when serum ferritin, vitamin D and NT-proBNP (89.4% accuracy, 83.3% positive predictive value in high-risk) were added to the model.

CONCLUSION

As cardiometabolic biomarkers may potentially improve cardiovascular prediction, further analysis can be performed to validate their clinical utility in diverse type 2 diabetes individuals.

EP_A031

UNVEILING A RARE PRESENTATION: LARGE RENAL ABSCESS IN A TEENAGER WITH NEWLY DIAGNOSED DIABETES

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INTRODUCTION/BACKGROUND

Type 2 diabetes mellitus (T2D), once considered a predominantly adult-onset disease, has witnessed a concerning surge in prevalence among adolescents worldwide emerging as a significant public health challenge. Studies have suggested that young-onset T2D might represent with more severe and rapidly progressive disorder than adults. We illuminate the clinical journey of a teenage patient who presented with a renal abscess as a rare complication concurrent with the diagnosis of diabetes.

CASE

A 13-year-old Indian female, with no known medical illness, presented with fever and osmotic symptoms for 1 month. Otherwise, she had no other infective symptoms. Upon presentation, she was hemodynamically stable and systemic examinations were unremarkable. Her BMI was 20 kg/m^2, with weight of 45 kg and height of 150 cm. She had acanthosis nigricans, capillary blood sugar of 13.2 mmol/L, serum ketone of 0.4 mmol/L, and no metabolic acidosis. Her investigations showed total white cells of 18.2x103/uL, c-reactive protein 146.9 mg/L and HbA1c 13.1%. Because of persistent fever, an ultrasound of the abdomen was done which revealed a right upper pole renal nephronia (3.1 x 2.5 x 1.8 cm) and a large left lower pole renal abscess (5.4 x 8.5 x 10.1 cm). The renal abscess was removed with pigtail drainage and the abscess culture & sensitivity grew Klebsiella pneumonia, sensitive to amoxicillin-clavulanate. After 6 weeks of adequate antibiotics and intensive insulin therapy, repeated imaging showed a resolved renal abscess. Her pancreatic auto-antibodies panel was positive for anti-Islet cell [42.9 IU/ml, (reference range <28)], and negative for anti-IA2 and anti-GAD. Distinguishing between the types of diabetes can be challenging in this age group. As she had clinical features of insulin resistance, high c-peptide level (1764 pmol/L) and parental history of T2D, she was diagnosed as young T2D with positive pancreatic autoantibody. During subsequent follow-up, her glycaemic treatment was de-intensified to basal insulin and metformin. In addition to dietary and lifestyle modification, her HbA1c improved to 6.0% with good glycaemic control.

CONCLUSION

There is an increasing prevalence of T2D in adolescents. However, renal abscess remains a rare presentation in teenagers with newly diagnosed diabetes. Successful management involved timely diagnosis, implementation of imaging, source control, adequate antibiotics and optimal glycaemic control.

EP_A032

DIABETES CONTROL AMONG ELDERLY DIABETIC PATIENTS IN KUANTAN, MALAYSIA

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INTRODUCTION/BACKGROUND

The transformation of the aging population in Malaysia carries a significant healthcare burden in chronic diseases like Type 2 Diabetes Mellitus (T2D).

METHODOLOGY

This study aims to measure the patients' diabetes control and to identify its related factors among elderly diabetic patients in Kuantan. This cross-sectional study was conducted in six selected government health clinics in Kuantan (chosen from stratified random sampling) where 300 elderly patients with T2D were recruited via proportionate random sampling. The related factors studied were sociodemographic profile, health and treatment characteristics and quality of life using the RV-DQOL13 questionnaire. The data were analysed using SPSS version 26.

RESULTS

The mean age of the patients was 68.1 years old (SD=6.009). Most patients were female (57.3%), Malay (70.3%), married (66%), living independently (Modified Barthel Index = 99%) and from B40 (96.3%). The prevalence of good diabetes control was 59.3% (cut-off point = HbA1c ≤7.5%). The significant predictors for good diabetes control identified were non-Malay (aOR = 3.92, 95%CI 1.907-8.060, p <0.001), treatment with insulin injection (aOR = 0.193, 95%CI 0.094-0.395, p <0.001), abnormal capillary blood glucose (CBG) (aOR = 0.655, 95%CI 0.489-0.878, p <0.001), having higher LDL-C (aOR = 0.655, 95%CI 0.489-0.878, p = 0.005), and poor satisfactory impact from RV-DQOL13 (aOR = 0.919, 95%CI 0.884-0.954, p <0.001).

CONCLUSION

Elderly diabetic patients in Kuantan have good diabetes control. However, follow-up for this group needs to be emphasized among Malay patients, those on insulin treatment, poor CBG during TCA, high LDL-C and those who are unsatisfied with diabetes care to maintain good diabetes control prevalence.

EP_A033

PREVALENCE OF DIABETES DISTRESS AMONG PATIENTS IN AN OUTPATIENT ENDOCRINE CLINIC IN A TERTIARY HOSPITAL: A CROSS-SECTIONAL STUDY

https://doi.org/10.15605/jafes.039.S1.044

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INTRODUCTION

Diabetes distress (DD) among diabetes mellitus patients is becoming a major challenge for healthcare providers. Studies have shown that patients with diabetes distress tend to have poorer glycaemic control. The Diabetes Distress Scale is a validated tool featuring 3 major domains: emotional burden (EB), physician distress (PD) and therapeutic support distress (TSD). Our study aimed to investigate the prevalence of diabetes distress among our patients and to identify risk factors associated with this condition.

METHODOLOGY

This is a cross-sectional study involving type 1 and type 2 diabetes patients seen in the HRPB Endocrine Clinic from February-March 2024. Patients who fulfilled the inclusion criteria (n=91) answered the validated Malay version diabetes distress scale questionnaire (MDDS-17). The Total mean score (TS) and the mean score of the 3 domains were analysed using univariate analyses via SPSS. A mean item score >3.0 denotes significant diabetes distress.

RESULT

Median TS is 1.94 (1.59-2.47). 16.5% of the patients had a TS score ≥3. Significant scores in the other domains were: 27.5% for EB, 12.1% for PD and 17.6% for TSD. Those with HbA1c >8.5% had higher median TS scores versus those with HbA1c <6.5% and 6.6-8.4% (2.24 vs 1.71, p = 0.028; 2.24 vs 1.82, p = 0.023) respectively. Patients with HbA1c >8.5% also had higher median TSD scores versus those with HbA1c <6.5% and HbA1c 6.6-8.4% (2.5 vs 1.9, p = 0.03, 2.5 vs 2.06, p = 0.041), respectively. Patients aged between 12-29 had lower median PD scores versus those aged 30-49 and 50-69 (1 vs 1.5, p = <0.001, 1 vs 1.5, p = 0.009), respectively. Patients with retinopathy had higher median PD scores versus those without (1.63 vs 1.0, p = 0.015). There were no significant differences in scores for gender, ethnicity, type of DM, duration of disease, socioeconomic status and other DM complications.

CONCLUSION

The prevalence of diabetes distress is 16.5%. Patients with poor glycaemic control, the middle-aged group and those with retinopathy had significantly higher diabetes distress scores. Efforts should be made to identify these groups of patients for timely intervention.

EP_A034

EUGLYCEMIC DIABETIC KETOACIDOSIS PRECIPITATED BY HYPERTRIGLYCERIDEMIA-INDUCED PANCREATITIS, LIVER ABSCESS AND SODIUM-GLUCOSE COTRANSPORTER-2 INHIBITOR USE IN A PATIENT WITH FAMILIAL HYPERTRIGLYCERIDEMIA

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INTRODUCTION/BACKGROUND

Euglycemic diabetic ketoacidosis (EDKA) has a worse outcome than typical DKA as it is relatively rare and remains a diagnostic challenge. Conditions such as sepsis, pancreatitis, use of sodium-glucose cotransporter-2 inhibitors (SGLT-2i), pregnancy and starvation are known to be associated with EDKA. We report a case of a patient with Type 2 Diabetes Mellitus (T2DM) and familial hypertriglyceridemia on SGLT-2i who presented with hypertriglyceridemia-induced pancreatitis (HTGP) concurrently with EDKA.

CASE

A 31-year-old female presented with epigastric pain, vomiting and lethargy. The clinical exam revealed tender epigastrium with no guarding and negative Murphy's. Serum amylase was 242 U/L (Imrie score 2, BISAP 1) and C-reactive protein (446 mg/L). The ultrasound of the abdomen revealed an ill-defined collection (2.3 x 3.2 cm) at segment V of the liver with findings suggestive of chronic pancreatitis. She had three prior admissions due to acute pancreatitis and once complicated by an infected pancreatic pseudocyst. She was diagnosed with T2DM and familial hypertriglyceridemia five years ago, with poorly controlled glucose and lipid profile (HbA1c 8.4%, triglycerides 33.4 mmol/L). She is on an SGLT2 inhibitor, amongst other medications, which she continued taking despite her illness. She developed EDKA in the ward (pH 7.43, PCO_2 20, HCO_3 14, serum ketone 3.6, lactate 0.8). She was started on DKA treatment, then continued with variable rate insulin sliding scale, fasting, statin, fibrates and intravenous antibiotics. Dietary and lifestyle advice were reinforced. She was discharged well after two weeks (triglyceride 4.2 mmol/L, C-reactive protein 2 mg/L) with resolved symptoms and liver lesions.

CONCLUSION

EDKA should be a well-recognised diagnosis in an era where there is growing use of SGLT2i, especially in patients with multiple precipitating factors. Physicians must have a high clinical suspicion in patients who are on SGLT-2i in acute illness. In addition, we need to consider that EDKA can precipitate HTGP and vice versa. In both conditions, early initiation of continuous intravenous insulin infusion can improve outcomes.

EP_A035

A REVIEW OF CLINICAL PROFILE AND GLYCEMIC CONTROL OF PATIENTS WITH YOUNG-ONSET TYPE 2 DIABETES MELLITUS ON INTENSIVE INSULIN THERAPY

https://doi.org/10.15605/jafes.039.S1.046

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INTRODUCTION/BACKGROUND

Young-onset type 2 diabetes mellitus (T2D) is a more aggressive subgroup of T2DM with rapid disease progression and rate of complications. Many patients progress to intensive insulin therapy early in the disease process due to decompensation and poor glycaemic control.

METHODOLOGY

We aimed to review the demographic profile, glycaemic control, and prevalence of complications in patients with young T2DM on intensive insulin therapy at the Endocrine Institute of Hospital Putrajaya. A retrospective audit was conducted using electronic medical records. Patients with T2DM between the age of 18-40 years on basal-bolus insulin therapy attending the outpatient diabetes clinic between January 2022 – March 2024 were included. Data about the demographic profile, insulin therapy, glycaemic control and complications were collected. A descriptive analysis using SPSS version 25.0 was performed.

RESULTS

The analysis involved a total of 72 cases, with a mean age of 33.7 years. Females comprised two-thirds (68.1%), with Malays being the majority (81.9%). The mean weight was 85.3 kg and the mean BMI was 32.1 kg/m^2. The mean duration of diabetes was 10.1 years. Among them, 62.5% have comorbidities such as hypertension and dyslipidaemia, and 48.6% are obese. The average duration of insulin therapy was 5.9 years. The mean HbA1c was 10.3% before insulin therapy and 9.5% on current intensive insulin therapy. Microvascular complications were prevalent (73.6%), with

nephropathy being the most common (59.6%), followed by retinopathy and neuropathy. Approximately 5% of patients had macrovascular disease. More than two-thirds (70.8%) were on statin and half (56.9%) were on anti-proteinuria therapy.

CONCLUSION

Most patients with young-onset T2DM have poor glycaemic control despite being on intensive insulin therapy. Most patients fit the phenotype of obesity with metabolic syndrome suggesting possible insulin resistance, as opposed to depletion, as the key factor driving disease progression. Treatment strategies employed should focus on intensive lifestyle intervention and pharmacotherapy targeting weight reduction and insulin resistance as opposed to excessive insulin in this subgroup.

EP_A036

SCREENING AND TREATMENT OF DIABETIC KIDNEY DISEASE IN TYPE 2 DIABETES MELLITUS (T2DM) PATIENTS: A CLINICAL AUDIT AT HOSPITAL SULTAN HAJI AHMAD SHAH TEMERLOH, MALAYSIA

https://doi.org/10.15605/jafes.039.S1.047

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INTRODUCTION/BACKGROUND

Diabetic kidney disease (DKD) is a global health challenge that has garnered increasing attention due to its significant impact on individuals and healthcare systems worldwide. In Malaysia, DKD accounted for the majority of new dialysis patients, increasing cardiovascular risk and hence, escalating healthcare expenses.

METHODOLOGY

This clinical audit aims to assess the screening and treatment of DKD among T2DM patients in Hospital Sultan Haji Ahmad Shah (HOSHAS), Temerloh, Pahang. All T2DM patients attending the diabetes clinic in HOSHAS from June to July 2023 were included in this clinical audit. Electronic medical records were assessed for demographic data, blood pressure and glycaemic targets, screening and treatment of macro- or microalbuminuria.

RESULTS

We included 141 patients in this audit. Of those, 63.8% were females, with a mean age of 52.8 ± 15.0 years and an average duration of diabetes of 13.0 ± 8.4 years. The screening rate for albuminuria was high (93.6%) but only 25.5% of the patients had further quantification of albuminuria. Overall, 31.9% achieved a blood pressure target of below 140/80 mmHg but only 19.0% with albuminuria achieved a BP target of below 130/80 mmHg. A total of 19.1% of patients achieved HbA1c of less than 7%. Among the patients with albuminuria, 71.2% were on ACE-i/ARB and 39% were prescribed SGLT2 inhibitors.

CONCLUSION

This audit highlights the importance of early detection and appropriate management of DKD in T2DM patients. Microalbuminuria assessment, optimal blood pressure and renal-modulation therapy are essential in preventing the progression of albuminuria and reducing the risk of ESKD in patients with diabetes.

EP_A037

GLUTAMIC ACID DECARBOXYLASE (GAD) ANTIBODIES-ASSOCIATED LIMBIC ENCEPHALITIS AND DIABETES: A CASE REPORT

https://doi.org/10.15605/jafes.039.S1.048

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INTRODUCTION/BACKGROUND

Glutamic acid decarboxylase (GAD) is an enzyme involved in producing the major inhibitory neurotransmitter Gamma-Aminobutyric Acid (GABA). GAD antibodies have been implicated in the pathogenesis of insulin-dependent diabetes mellitus (IDDM) and a few neurological diseases such as the case below.

CASE

A 24-year-old male presented with a one-week history of fever, gradual memory impairment, behavioural changes and seizure. On arrival, he was confused and disoriented. His blood glucose was 18 mmol/L, HbA1c of 12.3% with acidosis at pH 7.30, bicarbonate of 19.7, serum osmolarity of 282 mmol/L and urine FEME showed ketone 2+, glucose 3+.

The lumbar puncture CSF sample was acellular with normal cerebrospinal fluid protein. Serum autoimmune and paraneoplastic panels were negative. EEG showed seizure activity at the right frontotemporal region with clinical evidence of piloerection. His brain MRI was abnormal with hyperintensity and swelling of the right medial temporal lobe. Correlating the history, EEG and radiological changes, his diagnosis is supportive of limbic encephalitis with newly diagnosed diabetes mellitus. Intravenous

methylprednisolone and intravenous immunoglobulin (IVIg) were given with marked improvement of symptoms. He was discharged with regular insulin and a tapering dose of oral steroids. He had positive GAD antibodies with two readings noted at 24.45 and 18.65 IU/ml. During subsequent clinic follow-up, his HbA1c improved to 10.8%

CONCLUSION

Limbic encephalitis (LE) is characterized by acute or subacute development of seizure, memory impairment, irritability, hallucinations and psychiatric symptoms. Its pathogenesis is related to an inflammation of the medial temporal lobes. Non-paraneoplastic LE related to GAD antibodies should be suspected if the patient has concomitant diabetes mellitus.

EP_A038

EFFECTIVE MULTI-FACETED APPROACH TO SEVERE HYPERTRIGLYCERIDEMIA IN DIABETES AND HYPERTENSION: A CASE REPORT

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INTRODUCTION/BACKGROUND

Diabetes mellitus, a metabolic disorder characterized by insulin deficiency, leads to increased release of free fatty acids and amino acids. This triggers glycogenolysis and gluconeogenesis, resulting in elevated production of very low-density lipoproteins (VLDL) and subsequent hypertriglyceridemia. Hypertriglyceridemia heightens the risk of atherosclerotic cardiovascular disease (ASCVD).

CASE

This case details the successful management of severe hypertriglyceridemia in a 45-year-old male with T2DM and hypertension. Despite non-compliance with treatment, the patient presented with asymptomatic severe hypertriglyceridemia, with a level of 45.4 mmol/L. A comprehensive approach involving pharmacological intervention, lifestyle modifications and laboratory consultation was implemented. After a thorough discussion on the impact of hypertriglyceridemia, the patient accepted the treatment regime. This included atorvastatin alongside existing anti-diabetic and anti-hypertensive medications, dietary counselling emphasizing a low-fat and high-fibre diet, regular exercise and treatment concordance. The patient demonstrated notable adherence to the prescribed regimen and incorporated smoking cessation and increased physical activity.

Over three months, significant improvements were observed in serum triglyceride levels, glycaemic control and blood pressure, reflecting the efficacy of the management approach. Additionally, laboratory consultation aided in interpreting lipid profiles and identifying pseudo-hyponatremia secondary to analytical interference from lipemic samples.

CONCLUSION

This case highlights the efficacy of a holistic strategy in addressing hypertriglyceridemia in individuals with diabetes and hypertension. Integrating atorvastatin therapy, dietary adjustments and lifestyle modifications resulted in significant enhancements in triglyceride levels, glycaemic management and blood pressure, thereby reducing cardiovascular risks.

Additionally, it highlights the crucial role of laboratory consultation in interpreting lipid profiles and identifying related anomalies in the test results, reinforcing the clinical significance of comprehensive patient care.

The comprehensive strategy for addressing diabetic dyslipidaemia is delineated, incorporating medication, counselling, lifestyle modifications, and enhanced laboratory collaboration.

EP_A039

A SNAPSHOT OF TYPE 1 DIABETES CARE AMONG ADULTS IN MALAYSIA: DATA FROM A SINGLE ACADEMIC MEDICAL CENTRE

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INTRODUCTION/BACKGROUND

There is a dearth of data on adults living with Type 1 Diabetes (T1D) in Malaysia.

METHODOLOGY

This descriptive study aims to systematically collect current data and identify gaps among adults living with T1D in Malaysia. Data was extracted from electronic medical records of patients registered under the T1D clinic at Universiti Malaya Medical Centre (UMMC). All patients aged 18 and above in 2023 were included.

RESULTS

There was a total of 107 patients with T1D (mean age 42.0 ± 12.7, mean HbA1c 8.0 ± 1.6%, mean BMI 24.5 ± 4.1 kg/m^2). The majority were Chinese (52.3%), followed by Malays (24.3%) and Indians (23.4%). The median age at T1D diagnosis was 18.0 years (IQR: 14.0). Almost half (42.1%) presented with diabetic ketoacidosis (DKA) at diagnosis. One in four patients had diabetes-associated autoantibody tests done. Autoantibody positivity was in this order: GADA (22.4%), ICA (6.5%), IA2A (5.6%) and IAA (1.9%). Co-morbid autoimmune conditions were reported in 16.2%, of which thyroid disease (61.1%) was most common. In terms of treatment, the majority were on analogue insulin (89.7%) delivered using multiple daily injections (79.4%). Of the 22 (20.6%) patients using insulin pumps, 50% were using manual pumps, whereas the others were using either sensor-augmented pumps or advanced hybrid closed-loop systems. Most (81.3%) patients used self-monitoring of blood glucose (SMBG) whereas 23.4% employed continuous glucose monitoring (CGM) systems for glycemic surveillance. Incident hypoglycemia within the preceding three months was reported among 60.7%, the majority (95.4%) of which were mild, and none reported severe. The incidence of DKA within the preceding six months was 4.7%. Retinopathy (19.6%) was the most prevalent complication, followed by kidney disease (15%) and atherosclerotic cardiovascular disease (7.5%).

CONCLUSION

This data serves as a baseline for a registry of local T1D patients, whereby future longitudinal analyses may unveil patterns of disease outcomes to guide clinical care unique to our setting.

EP_A040

PRELIMINARY ASSESSMENT IN MANAGEMENT OF DIABETIC KETOACIDOSIS IN A TERTIARY CARE SETTING

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INTRODUCTION/BACKGROUND

Diabetic ketoacidosis (DKA) exacts a huge burden on the healthcare system despite numerous advancements in anti-diabetic therapies and updated clinical practice guidelines. The incidence of DKA for Type 1 and Type 2 DM is between 4.6 to 8 episodes per 1000 people.

METHODOLOGY

An assessment was made of the admissions of DKA into a tertiary centre in East Malaysia to evaluate the characteristics of these patients and identify potential management pitfalls. All patients admitted with a diagnosis of DKA and referred to the endocrine team in Hospital Tengku Ampuan Afzan, Kuantan between December 2023 to March 2024 were analysed. Data was collected for age, date of admission, HbA1c, total daily dose of insulin, DKA history and SGLT2 inhibitor use, amongst others.

RESULTS

Over four months, a total of 28 patients were assessed. There were 4 Type 1 and 24 Type 2 DM patients. Two type 1 DM patients were readmitted with DKA during the same period while 5 patients in total were admitted with DKA within the preceding 6 months. The mean age was 45 (± 17) years old and the mean HbA1c around their presentation was 11.2% (± 4.2%). Two patients were on SGLT2 inhibitors while 15 patients were on insulin with a mean total daily dose of 39 (± 17) units. Five patients were admitted to the ICU and the most common predisposing cause for DKA

was missed medications. The mean time taken to resolve the DKA was 931 (± 574) minutes.

CONCLUSION

Based on the results, the number of readmissions for DKA is worrying and the patients admitted also have high insulin doses, highlighting a possible consequence of over-insulinization. A longer period of evaluation is necessary to investigate the effect of SGLT2 inhibitors use on DKA admissions, as well as further focus on the causes of prolonged time for DKA resolution which may impact the length of hospitalization.

EP_A041

ONE-YEAR TREATMENT OUTCOMES WITH SUBCUTANEOUS SEMAGLUTIDE AT HOSPITAL QUEEN ELIZABETH II: A RETROSPECTIVE ANALYSIS

https://doi.org/10.15605/jafes.039.S1.052

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INTRODUCTION/BACKGROUND

Glucagon-like Peptide-1 receptor agonists (GLP-1 RAs) mimic endogenous GLP-1, improving glycemic control and promoting weight loss. Nevertheless, there is limited data available on the effect of semaglutide use among type 2 diabetes (T2D) patients undergoing insulin therapy, particularly those with high insulin requirements.

METHODOLOGY

We aimed to investigate the effects of the addition of subcutaneous semaglutide to a standard regimen of insulin on T2D patients, focusing on changes in HbA1c levels, body weight and total daily dose (TDD) of insulin. In this retrospective chart review, T2D patients who received once-weekly subcutaneous semaglutide with insulin were recruited from the Endocrine Unit of the Hospital Queen Elizabeth II (HQE II) from 2021 to 2023. Follow-up assessments occurred at 3-6 months and 9-12 months post-initiation, with the recording of key parameters such as HbA1c, weight, insulin TDD and adverse events.

RESULTS

Our study recruited a total of 35 patients and found that there were significant improvements across all parameters. HbA1c levels decreased from a mean of 8.9% at baseline to 7.7% at 9-12 months, representing a reduction of 1.2% (p <0.001). Weight decreased from a mean of 92.0 kg at baseline to 84.2 kg at 9-12 months, with a mean reduction of 7.7 kg (-8.4%) (95%CI: 4.9-10.6, p <0.001). Insulin TDD decreased from a median of 72u (40 - 114) at baseline to 48u (24 - 80) at 9-12 months (p <0.001). Six individuals experienced gastrointestinal side effects, with one discontinuing due to intolerable diarrhea. In the subgroup with insulin resistance, there were profound reductions in TDD of insulin used without compromising glycemic control.

CONCLUSION

The study confirmed the efficacy of once-weekly semaglutide in managing T2DM patients on insulin therapy, including those on basal-bolus and pre-mixed regimens. Further research is recommended to assess its effects on patients with high insulin requirements.

EP_A042

RISK OF KETOACIDOSIS WITH LUSEOGLIFLOZIN IN TYPE 2 DIABETES MELLITUS PATIENTS ON MODERATE DOSE INSULIN THERAPY: A RANDOMISED CONTROL TRIAL

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INTRODUCTION/BACKGROUND

Sodium-glucose cotransporter-2 (SGLT2) inhibitors, one of which is Luseogliflozin, are associated with a recognized risk of euglycemic diabetic ketoacidosis (DKA) particularly in patients on insulin therapy.

METHODOLOGY

This study aimed to assess the risk of ketoacidosis with Luseogliflozin in patients with type 2 diabetes mellitus (T2D) on moderate doses of insulin. This study involved patients who were attending the Endocrine Clinic, with stable disease and no recent acute events. The participants were randomized to either add-on Luseogliflozin to standard medical therapy or standard medical therapy only. Ketoacidosis was assessed using fasting blood and urine ketone pre- and post-intervention. The study duration was 12 weeks. Independent t-test was performed to assess changes in ketone levels. Pearson's Correlation was performed to determine the relationship between ketone levels with HbA1c and fasting blood glucose.

RESULTS
A total of 40 patients completed the study, with 20 patients receiving Luseogliflozin and the rest were on standard medical therapy. The mean age and HbA1c for patients were 53.6 ± 7.6 years and 9.1 ± 1.4%, respectively. There was a non-statistically significant increase in fasting blood ketones with the addition of Luseogliflozin compared to standard therapy (0.04 ± 0.12 vs 0.05 ± 0.15 mmol/L; p = 0.735). Similarly, there was a non-statistically significant increase in urine ketones (0.03 ± 0.3 vs 0.03 ± 0.1 mmol/L; p = 1.00). Correlation analysis demonstrated that the increased blood ketone levels were more likely to occur with higher HbA1c (r = 0.324; p = 0.04) and higher fasting blood glucose (r = 0.447; p = 0.004).

CONCLUSION
The addition of Luseogliflozin in T2D patients on moderate-dose insulin was not associated with a significant increase in fasting blood and urine ketone levels. However, those with higher HbA1c and FBS seemed to be more vulnerable to elevated blood ketone levels. Thus, this study suggests that Luseogliflozin is safe but should be used with caution in those with higher HbA1c and FBS.

EP_A043

RAMADAN FASTING AMONG TYPE 1 DIABETES MELLITUS PATIENTS IN A SINGLE TERTIARY CENTRE

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INTRODUCTION/BACKGROUND
Ramadan fasting among patients with Type 1 diabetes mellitus (T1DM) carries a higher risk given the nature of the disease and therapy used. Currently, Ramadan fasting practice among Muslim T1DM patients in our centre is not known.

METHODOLOGY
This is a questionnaire-based study done among Muslim patients attending the T1DM clinic in Hospital Putrajaya. All Muslim patients attending the clinic from January to April 2024 (before Eid) were given the questionnaire to fill out.

RESULTS
There were 56 respondents, 22 male and 34 female. The mean age was 30.2 years (± 8.04). The mean duration of illness was 13 years (± 8.39). The majority (79%) of the respondents received tertiary education. Most respondents (88%) have received advice from healthcare providers on Ramadan fasting. Four out of 5 intended to fast during Ramadan. Out of those who intended to fast, 3 quarters had high risk based on the DAR-IDR (Diabetes and Ramadan-International Diabetes Federation) risk calculator. Among all the respondents, 80% had high risk, 18.2% had moderate risk and only 1.8% had low risk. In comparison to the actual risk, only about one-third of total respondents perceived themselves as having high risk, half perceived themselves as moderate risk and the rest felt they had low risk. Forty percent of the respondents correctly estimated their risk of fasting. In terms of diabetes disease knowledge, our respondents had a mean score of 11.7 (± 2.29). Two-thirds of the respondents achieved high scores, 30.4% had average scores and only 3.6% had low scores.

CONCLUSION
Among Muslim T1DM patients in our centre, the majority received tertiary education and had been advised on Ramadan fasting in the past. Despite having high risk, most opted to fast. Therefore, Ramadan fasting education must emphasize measures to fast safely.

EP_A044

CLINICAL UTILITY OF KIDNEY FAILURE RISK EQUATION IN DIABETES AND CHRONIC KIDNEY DISEASE

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INTRODUCTION/BACKGROUND
Heterogeneity in disease course and prognosis makes managing CKD difficult. An accurate risk stratification algorithm is crucial to predict CKD progression to ESKD for individualized management. The Kidney Failure Risk Equation (KFRE), developed in 2011, is the most widely validated prediction model for 2- and 5-year ESKD progression risk across multiple underlying etiologies with potential for clinical utility.

METHODOLOGY
This study aims to investigate if KFRE risk scores differ significantly among individuals with or without diabetes. We conducted a retrospective study on adults with CKD (eGFR 15-59 ml/min/1.73 m^2) who attended our hospital outpatient follow-up from January to December 2022 with available data for calculation of 4-variable KFREs [age, sex, eGFR, urine albumin-creatinine ratio (uACR)]. Two-sample t-test and Mann-Whitney U test were performed to analyse the difference between the two groups.

RESULTS
Out of 10,391 adults with CKD, 1,823 that fulfilled the inclusion criteria were analysed, with a mean age of 70 years, 52% were male, mean eGFR of 45ml/min/1.73 m^2 and median uACR of 8.4 mg/mmol. Majority (84%) have diabetes with a mean HbA1c of 7.8%. Individuals with CKD and diabetes had lower eGFR, heavier albuminuria and had younger age than those without diabetes (p <0.001). These findings further translate to statistically significant higher KFRE risk scores for individuals with diabetes. For those with eGFR between 30-59 ml/min/1.73 m^2, 9.4% of individuals without diabetes and 14.8% of those with diabetes met the referral criteria for nephrology care when setting a KFRE score threshold of more than 5% over 5 years.

CONCLUSION
The lack of uACR monitoring resulted in a smaller sample size than anticipated. We advocate all healthcare professionals to monitor uACR and utilize the KFRE score in clinical practice when managing CKD or diabetes with eGFR between 15-60 ml/min/1.73 m^2 to guide referral to multi-disciplinary care and raise public awareness about the risk of ESKD.

EP_A045

THINGS MAY NOT ALWAYS BE AS THEY SEEM

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INTRODUCTION/BACKGROUND
Atypical diabetes and late-onset Type 1 Diabetes Mellitus are rare, affecting only 10% of patients with diabetes. Presence of diabetes-associated autoantibodies such as Anti-Islet Cell (ICA), Anti-Glutamic Acid Decarboxylase (GAD), Anti-Insulinoma Associated Antigen-2 (IA2) and Zinc Transporter 8 (ZnT8) point towards type 1 over type 2 diabetes mellitus.

CASE 1
A 55-year-old male with underlying autoimmune hypothyroidism and vitiligo was admitted for diabetic ketoacidosis (DKA) with the following laboratory findings: HbA1c 16.6%, random blood sugar (RBS) 43 mmol/L, serum ketone 6.7 mmol/L, pH 7.06 HCO3 5.1. He was lean with a body mass index (BMI) of 21 kg/m^2 and no family history of diabetes. He was discharged well with metformin, dipeptidyl peptidase-4 inhibitor (DPP-4i) and basal insulin. Follow-up after two weeks showed erratic glucose control. Results showed positive ICA: 224 IU/ml (>28 IU/ml) and GAD: >280 IU/ml (>17 IU/ml) but negative IA2: 3.611 (<28 IU/ml) and low C-peptide 146 pmol/L (<367 pmol/L). He was diagnosed with latent autoimmune diabetes of adults (LADA), with differentials being late-onset Type 1 Diabetes Mellitus and autoimmune polyglandular syndrome. Treatment was revised to basal-bolus insulin. HbA1c improved to 11.6% within one year.

CASE 2
A 33-year-old female, obese (BMI 28 kg/m^2), with features of insulin resistance and diabetic parents, was admitted for DKA. – Laboratory results were as follows: RBS 31 mmol/L, serum ketone 5.3 mmol/L, pH 7.23, HCO3 14. Baseline HbA1c was 17.1%. She was started on subcutaneous insulin isophane) 34 units, T, Metformin XR 2 g ON and T and Vildagliptin 50 mg BD (DPP-4i) and was discharged with these medications. Self-monitoring blood glucose after two weeks was unsatisfactory. Results revealed normal C-peptide of 470 pmol/L (367-1467 pmol/L), negative IA2: <2.5 IU/ml (<28 IU/ml), positive ICA: 157IU/ml (>28 IU/ml) and GAD: >280 IU/ml (>17 IU/ml). Maturity-onset diabetes of the young (MODY) was considered. Adding sulfonylureas resulted in suboptimal glycaemic control. HbA1c improved to 14.6% within one year after switching to premixed insulin.

CONCLUSION
Subclassifying diabetic patients with positive diabetes-associated autoantibodies necessitates a comprehensive approach, considering family history, phenotype and targeted genetic testing.

EP_A046

AN OVERVIEW OF ADMISSIONS FOR HYPERGLYCAEMIC CRISES IN HOSPITAL SULTANAH AMINAH JOHOR BAHRU, MALAYSIA

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INTRODUCTION/BACKGROUND
Hyperglycaemic crises are metabolic emergencies that encompass diabetic ketoacidosis (DKA) and hyperglycaemic hyperosmolar state (HHS). Both are associated with increased healthcare expenditure, morbidity and mortality.

METHODOLOGY
We describe the clinical and socioeconomic characteristics of patients admitted for hyperglycaemic crises from 1st June 2023 to 31st March 2024 in our hospital. An endocrine team reviewed all admissions for hyperglycaemic crises during the period mentioned above. Patients' demographic and clinical information were collected as part of routine comprehensive patient evaluation. All data were analysed using GraphPad Prism Version 9.5.0 software.

RESULTS
There were 132 admissions for hyperglycaemic crises (129 DKA and 3 HHS), involving 110 patients (mean age 41.3 years, SD = 17.6; 51.8% female; 57.3% Malay, 20.9% Indian, 19.1% Chinese; 71.3% completed secondary education; 23.6% active smokers). Six patients were readmitted for DKA and one for HHS within 90 days from their index admissions within this period. Fifty-three (55.2%) had a household income of RM 2500 and below. Two-thirds had type 2 diabetes mellitus, while 29.1% had type 1 diabetes. Fifteen patients (13.6%) had DKA as their first presentation of diabetes. Infection was the most common precipitant of hyperglycaemic crises, comprising 60% of cases. Among 95 patients who had pre-existing diabetes, 54.7% had their follow-up at primary care, while 24.2% received care at our endocrine clinic. Forty percent did not conduct self-monitoring of blood glucose at all. Moreover, at least two-thirds of patients with established diabetes fared poorly in sick day rules knowledge assessment.

CONCLUSION
More efforts are needed to reinforce diabetes self-management education and support (DSMES) services at all levels of care to reduce the healthcare burden of hyperglycaemic crises.

EP_A047

MEDICATION PRACTICES AND IMPACT ON GLYCAEMIC OUTCOMES AMONG FASTING MUSLIM TYPE 2 DIABETES MELLITUS IN PRIMARY CARE CLINICS DURING RAMADAN

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INTRODUCTION/BACKGROUND
Ramadan fasting has been shown to affect glycaemic outcomes among those with Type 2 Diabetes Mellitus (T2DM) due to adjustment of oral antidiabetic medications (OHA) and insulin.

METHODOLOGY
This study investigates how medication practices affect glycated haemoglobin (HbA1c) levels, hypoglycaemia, hyperglycaemia and hospitalization rates among fasting Muslim T2DM patients in Petaling district, Malaysia. A prospective observational study was conducted in seven government primary healthcare clinics in the Petaling district from March 14 to July 15, 2022. A questionnaire on medication types, practices and outcomes was administered to patients. Pregnant women were excluded. Chi-square and logistic regression were used to determine the association between medication practices and glycaemic outcomes.

RESULTS
A total of 260 participants completed the study. In this study, 96.5% of participants were taking OHA; 41.5% were taking both insulin and OHA. Despite being counseled by healthcare providers (HCPs), 8.4% of participants had self-adjusted their OHAs, and 23.1% self-modified their insulin dose during Ramadan. Among those who adjusted OHAs, 2.2% stopped taking the medication, 6.9% decreased the dose and none increased the dose. For insulin users, 2.6% increased the dose, 9.1% reduced the dose and none discontinued the insulin. Chi-square showed a significant effect of self-adjustment of medication during Ramadan with hypoglycaemia (P = 0.046), with no significant association between self-adjustment of medication with HbA1c level (P = 0.48), hospitalization rate (P = 0.693), or hyperglycaemia (P = 0.757). However, logistic regression

did not show any significant association between self-adjustment of medication and hypoglycaemia ($P = 0.085$).

CONCLUSION

HCPs need to be aware and do close follow-ups among T2DM patients to prevent self-adjustment of medications during Ramadan. Self-adjustment of medication during Ramadan has no significant impact on glycaemic outcomes. Further studies are needed to explore other factors like medication adherence and dietary and lifestyle changes that may affect such outcomes.

EP_A048

IMPACT OF ADVANCED CARBOHYDRATE COUNTING INTENSIVE PROGRAM IN TYPE 1 DIABETES THROUGH WHATSAPP-BASED MONITORING

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INTRODUCTION/BACKGROUND

Advanced carbohydrate counting is a meal planning method that allows better flexibility and glycaemic control. However, adopting and maintaining this nutritional therapy for long-term effectiveness can be highly challenging for individuals with type 1 diabetes (T1D).

METHODOLOGY

Using technology to facilitate patient access and offer real-time feedback, we aimed to assess the effectiveness of a multidisciplinary collaboration educational program known as the Advanced Carbohydrate Counting Intensive Program (ACCIP). This retrospective observational study involves T1D patients enrolled in ACCIP via a WhatsApp-based group monitoring from July 2020 to December 2022 in Hospital Queen Elizabeth II. The study included patients who could send food diary photographs and perform carbohydrate counting via WhatsApp Group for at least two meals a day for seven days. HbA1c measurements were recorded at initiation, 3-to-6 months and 9-to-12 months after the program ended.

RESULTS

The analysis included 62 patients with T1D who met the inclusion criteria. There were 22 (35.5%) men and 40 (64.5%) women. The median age of patients was 31.0 ± 10.2 years, with a median diabetes duration of 8.5 years ± 7.8 years and a median initial HbA1c of 9.2% ± 2.9. A total of 40 (64.5%) patients were able to maintain advanced carbohydrate counting (ACC) 12 months after the program ended. Overall, median HbA1c decreased significantly 3-to 6 months following the ACC intensive program (-1.1%, P <0.01). Reductions were maintained at 9-to-12 months but were not significant (-0.6%, $P = 0.086$). Subgroup analysis showed significant HbA1c reduction in patients who maintain ACC at 3 to 6 months (-1.5%, $P = 0.01$) and 9 to 12 months (-1.4%, $P = 0.02$).

CONCLUSION

Early and real-time intensive education in advanced carbohydrate counting via digital platforms may provide a long-term positive impact on glucose control. Larger clinical trials with structured programs are warranted to validate this positive impact.

EP_A049

CEREBELLAR ATAXIA PRESENTING WITH LATE-ONSET AUTOIMMUNE DIABETES MELLITUS: A CASE REPORT

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INTRODUCTION/BACKGROUND

Glutamic acid decarboxylase antibodies (GAD-Ab) are the predominant autoantibodies present in most adult-onset autoimmune diabetes cases. Furthermore, high levels of GAD-Ab have been associated with neurological syndromes such as stiff person syndrome, cerebellar ataxia, epilepsy, limbic encephalitis and other overlapping syndromes. We present a patient who exhibited symptoms of cerebellar ataxia and new-onset diabetes mellitus

CASE

A 38-year-old female with a medical history of endometrial polyps presented with a one-month history of progressive gait instability and vertigo resulting in difficulty ambulating. She had also experienced polydipsia and polyuria for two weeks. Neurological examination revealed staccato speech and gait ataxia with bilateral dysmetria and dysdiadochokinesia. No nystagmus or diplopia was observed. Power and tone were normal, with hyperreflexia in the left bicep and patella. Her glucose level at presentation was 28.9 mmol/L with no ketoacidosis. Cranial MRI was unremarkable, and CSF analysis showed lymphocytic

pleocytosis with elevated protein levels. CSF screening for infection, paraneoplastic antibodies and oligoclonal bands yielded negative results. Her CSF GAD-Ab was positive, and her serum GAD-Ab was markedly elevated (280 IU/L), as were her anti-islet cell antibodies (87.5 IU/mL). Her HbA1c was 10.9%, with evidence of proteinuria but no diabetic retinopathy. She was managed with intravenous (IV) immunoglobulin and methylprednisolone followed by oral steroids, with subsequent improvement in her ataxic gait. Her diabetes was managed with basal-bolus insulin.

CONCLUSION

Autoimmune cerebellar ataxia is a rare condition that can be associated with high levels of GAD-Ab and, frequently, autoimmune diabetes. As this condition may result in chronic disabling neurological impairment, prompt diagnosis to facilitate treatment is imperative.

EP_A050

UNDERPRESCRIPTION OF SGLT2i IN TYPE 2 DIABETES PATIENTS WITH CARDIORENAL DISEASE IN A PUBLICLY FUNDED TERTIARY CENTER IN MALAYSIA

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INTRODUCTION/BACKGROUND

Large-scale randomized controlled trials have proven that sodium-glucose cotransporter-2 inhibitors (SGLT2i) significantly reduce risks of atherosclerotic cardiovascular disease (ASCVD), heart failure (HF) and chronic kidney disease (CKD) in patients with type 2 diabetes (T2D). However, TARGET-T2D has shown that the use of guideline-directed medical therapy with SGLT2i is suboptimal even in Greater Kuala Lumpur, which has the highest mean household incomes in Malaysia.

METHODOLOGY

We hypothesize that the use is even lower in our centre. This cross-sectional clinical audit involves all patients aged 18 years and older with T2D who visited the general medicine clinic of Hospital Sultan Ismail from 31st March to 4th April 2024. Their electronic medical records were reviewed for the presence of ASCVD, HF and CKD, and SGLT2i prescriptions.

RESULTS

A total of 224 patients were included. After excluding those with eGFR <20 ml/min/1.73 m^2, 175 patients were identified. Among them, 116 (66.3%) have at least one cardiorenal disease. Fifty patients (28.6%) have ASCVD, 31 (17.7%) have HF and 92 (52.3%) have CKD. However, only 29 (25%) are on SGLT2i. Interestingly, it was higher than the 13.2% reported by TARGET-T2D for general medicine clinics in Greater Kuala Lumpur. Aside from 3 patients who received hospital-funded SGLT2i, most (89.7%) are self-funded.

Among the 87 patients with indications to start SGLT2i but are not on it, six were recommended to purchase the medication but could not afford it. The other 81 patients did not receive such advice. None had SGLT2i withheld due to urogenital tract infections or euglycemic diabetic ketoacidosis.

CONCLUSION

SGLT2i remains critically underused in T2D patients with cardiorenal disease. Increasing public funding for SGLT2i could help bridge the gap between evidence and clinical practice. Even if public funding is not sufficient, health professionals should advise patients with indications to start SGLT2i to self-purchase, considering its overwhelming clinical benefits.

EP_A051

ASSESSING THE IMPACT OF EXERCISE ON BLOOD SUGAR CONTROL: A STUDY AMONG HOSPITAL STAFF IN TELUK INTAN, MALAYSIA

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INTRODUCTION/BACKGROUND

With Malaysia experiencing a high prevalence of diabetes mellitus and obesity, emphasis is placed on promoting healthy lifestyle interventions such as dietary modifications and, notably, exercise. Exercise enhances insulin sensitivity, aids in managing blood glucose levels and promotes weight reduction.

METHODOLOGY

This research aims to determine the impact of exercise on glycemic control by investigating the effect of a 3-kilometer walk on glucose levels. The study was exclusive to hospital staff. Blood glucose levels were measured using a glucometer before and after the 3-km walk. The walk was conducted without breaks, and participants refrained from consuming food or drinks during the activity.

RESULTS
Ninety-six individuals participated in the exercise, with 64% classified as overweight or higher. Mean age was 38.9 years. All of the participants completed the 3 km walk in 30 minutes. Only 76 individuals consented to have their glucose levels checked before and after the exercise. Among the participants, 5.3% (n = 4) had pre-existing diabetes. The average sugar level before the 3 km walk was 6.12 mmol/L and decreased to 5.43 mmol/L after walking, indicating a mean reduction of 0.69 mmol/L. Gender and BMI had no significant impact on the difference in mean glucose levels. However, there was a notable difference in glucose levels among age groups, particularly in the older age group (51-60 years old), which showed a significant difference ($P = 0.038$) compared to the younger age group.

CONCLUSION
Walking provides a safe and accessible option for managing diabetes. Walking for 3 km can decrease sugar levels by up to 0.7 mmol/L within 30 mins. Additionally, it was observed that older patients experience a more pronounced reduction in glucose levels compared to younger individuals. Given that a significant proportion of patients with diabetes patients fall within the older age group, exercise is essential for better glycemic control.

EP_A052

OPTIMIZING MANAGEMENT FOR ADULT TYPE 1 DIABETES MELLITUS PATIENTS: AN ENDOCRINE CLINIC AUDIT

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INTRODUCTION/BACKGROUND
Type 1 Diabetes Mellitus (T1DM) imposes significant healthcare challenges due to its lifelong management requirements.

METHODOLOGY
This study aims to assess the demographics, treatment modalities, and glycemic control among T1DM patients attending the Endocrine Clinic at Hospital Teluk Intan.

RESULTS
A total of 24 adult T1DM patients were studied, with a mean age of 28 years at presentation and a mean age of 15.21 years at diagnosis. The majority were Malay, followed by Indian and Chinese. There were more females than males. Mean duration of follow-up was six years. The most prevalent comorbidities were retinopathy, mental illness and obesity. Only 12.5% of patients achieved target HbA1c levels (<6.5%). Basal-bolus human insulin was the most commonly prescribed regimen, followed by insulin analogues and a combination of both. Routine self-monitoring of blood glucose (SMBG) was infrequent, with only four patients adhering to it regularly. This may not accurately represent the entire nation, as some T1DM patients may be managed under private practices.

Several factors could contribute to suboptimal sugar control. First, socioeconomic challenges such as poverty may require individuals to work extensively, resulting in fewer food options besides cheap, high-glycemic index diets. Furthermore, insufficient understanding of the disease, possibly due to underlying mental health conditions, can impede individuals' comprehension of the ramifications of poorly controlled diabetes. Inadequate social support also plays a role; patients with strong familial support tend to achieve better glycemic control than those without. Another contributing factor is the absence of Diabetic Resource Centers (DRCs) in hospitals, depriving individuals of a place to seek assistance with insulin pen issues and other diabetes-related challenges.

CONCLUSION
This study highlighted the challenges in achieving optimal glycemic control among T1DM patients attending our clinic. Despite the availability of various insulin regimens, a significant proportion of patients have suboptimal HbA1c levels, emphasizing the need for treatment intensification, enhanced patient education, improved adherence to SMBG and individualized insulin titration. Addressing comorbidities and individualizing treatment regimens is crucial in enhancing the overall care of T1DM patients.

EP_A053

BALANCING HORMONAL CHAOS: A CASE REPORT ON TYPE 1 DIABETES MELLITUS AND LACTATION-INDUCED HYPOGLYCEMIA

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INTRODUCTION/BACKGROUND
Type 1 Diabetes Mellitus (T1DM) accounts for 0.6% of diagnosed diabetes cases in Malaysia, presenting unique challenges for young adults of childbearing age. Women with T1DM encounter numerous hurdles not only before and during pregnancy but also in the postpartum period. Here, we present a case study of a young patient with T1DM who experienced recurrent hypoglycaemia during lactation.

CASE
A 31-year-old mother of two was diagnosed with T1DM at the age of 15 and was managed with multiple daily insulin (MDI) injections. Prior to her second pregnancy, her HbA1c was 6.1%. During her second pregnancy, she was on prandial insulin aspart six units and glargine 18 units. The patient's insulin requirement was further reduced immediately postpartum. Despite these adjustments, she encountered frequent hypoglycaemic episodes, particularly during breastfeeding. During her clinic visits, she was advised to take small snacks before nursing and to use a continuous glucose monitor (CGM). However, at four months postpartum, she presented with facial nerve palsy and was admitted for transient ischemic attack (TIA) due to severe hypoglycaemia. She initiated a sensor-augmented insulin pump trial at 16 months postpartum, which resulted in the cessation of hypoglycaemic episodes and subsequently, better glycaemic control.

CONCLUSION
Several factors contribute to lactation-induced hypoglycaemia in women with T1DM, including hormonal and physiological changes. Increased energy demand for milk production, elevated oxytocin and prolactin levels that enhance insulin sensitivity, and unpredictable timing and duration of breastfeeding sessions can all exacerbate hypoglycaemia. This case highlights the challenges of managing T1DM during lactation, specifically the increased risk of hypoglycaemia due to the energy demands of breastfeeding. Personalized treatment plans, in collaboration with endocrinologists, and the expanded utilization of CGM and insulin pumps can significantly enhance glycaemic control and minimise the risk of hypoglycaemia in breastfeeding mothers with T1DM.

EP_A054

FAMILIAL HYPERTRIGLYCERIDEMIA MANIFESTING RECURRENT PANCREATITIS, ERUPTIVE XANTHOMAS, LIPEMIA RETINALIS IN A YOUNG FEMALE WITH TYPE 1 DIABETES MELLITUS AND PRIMARY OVARIAN FAILURE

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INTRODUCTION/BACKGROUND
Severe hypertriglyceridemia is one of the etiologies of pancreatitis and is associated with diabetes mellitus.

CASE
We present a case of a 22-year-old female with DM diagnosed at 17 years old with presentation of osmotic symptoms. On examination, she was normotensive with a BMI of 23 kg/m^2, no features of Cushing syndrome but with sexual immaturity (Tanner stage 1). Baseline laboratory showed HBAIC of 10%, elevated AST 132 U/L, ALT 138 U/L, triglyceride (TG) 16.62 mmol/L, LDL 0.3 mmol/L, HDL 0.8 mmol/L, macroalbuminuria, normal renal profile, thyroid function test and cortisol level. Serum FSH of 24.2 IU/L, LH of 8.02 IU/L and estrogen levels of 71.1 pmol/L confirmed primary ovarian failure and karyotyping excluded Turner's syndrome. Ultrasound of the abdomen showed a fatty liver, a small uterus and ovaries. Initial treatment included an oral hyperglycaemic agent, basal insulin, fenofibrate and statin.

On subsequent follow-up, diabetes control remains poor, with HbA1c persistently above 10%, requiring intensification with basal-bolus insulin. Fundoscopy showed bilateral lipemia retinalis but no retinopathy. She developed acute pancreatitis two years after diagnosis of DM, and imaging confirmed pancreatitis without calculi. Lipid levels were not available due to lipemic samples. She later noted xanthomas over her extremities and presented again with severe pancreatitis and uncontrolled diabetes. Markedly elevated triglyceride level at 59.73 mmol/L reduced to 5.49 mmol/L with continuous insulin infusion. Omega-3 oil tablets were added, and an outpatient review showed improved HbA1c levels from 12.3% to 10.4% and triglyceride of 5.36 mmol/L. DM autoantibodies confirmed autoimmune diabetes.

CONCLUSION

This patient illustrates a unique case of a likely familial hypertriglyceridemia co-existent with poorly controlled type 1 diabetes mellitus that presented with recurrent pancreatitis, eruptive xanthomas and lipemia retinalis, which can be controlled with appropriate treatment.

EP_A055

ASSESSING CLINICAL OUTCOMES OF SGLT2 INHIBITOR THERAPY IN ELDERLY HFrEF PATIENTS WITH AND WITHOUT DIABETES: A SINGLE-CENTRE STUDY

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INTRODUCTION/BACKGROUND

Heart failure with reduced ejection fraction (HFrEF) is a complex syndrome resulting in decreased ventricular function, leading to symptomatic left ventricle dysfunction and global cardiovascular morbidity and mortality. Type 2 Diabetes Mellitus (T2DM) escalates cardiovascular risk, necessitating tight glucose control. Sodium-glucose cotransporter 2 inhibitors (SGLT2i) promise to improve cardiovascular outcomes and diabetes therapy.

METHODOLOGY

This study aimed to assess the efficacy and safety profile of SGLT2i in elderly HFrEF patients, both with and without T2DM. In this retrospective observational study, we examined patients on SGLT2i aged 65 and older with an ejection fraction (EF) of ≤40% from our cardiology clinic. Patient medical records from 2018–2023 provided data for analysis, including demographics, comorbidities, changes in EF, New York Heart Association (NYHA) shifts, estimated glomerular filtration rate (eGFR) reduction, hospitalisation and mortality among patients with and without T2DM.

RESULTS

From 934 SGLT2 inhibitor-treated patients, our study focused on 167 elderly HFrEF patients, divided into T2DM (125 patients) and non-T2DM (42 patients). Both groups had similar demographics. Significantly, 80.6% of T2DM patients had hypertension, compared to 37.2% of non-T2DM patients (P <0.001). Both groups had improved EF (54% vs. 51.2%, P = 0.859). Guideline-Directed Medical Therapy (GDMT) showed a moderate association with observed outcomes, with no significant differences in EF or NYHA improvement between T2DM and non-T2DM patients (P = 0.859, P = 0.137, respectively). In T2DM patients, cardiovascular events, total hospitalisation, and mortality were greater but not statistically significant (P = 0.38, P = 0.128, and P = 0.113, respectively). Notably, patients without T2DM exhibited a more pronounced reduction in eGFR (P = 0.018).

CONCLUSION

SGLT2 inhibitors improved EF and NYHA classification in elderly patients with HFrEF, regardless of T2DM status. On the other hand, the presence of both T2DM and chronic kidney disease (CKD) emerged as significant risk factors associated with higher rates of hospitalisation and mortality.

EP_A056

METABOLIC BENEFITS OF ADDING SODIUM GLUCOSE CO-TRANSPORTER-2 INHIBITORS IN REAL-WORLD SETTINGS, A TERTIARY CENTRE EXPERIENCE

https://doi.org/10.15605/jafes.039.S1.067

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INTRODUCTION/BACKGROUND

Sodium-glucose co-transporter-2 inhibitors (SGLT2i) have revolutionized the landscape of type 2 diabetes (T2D). Ministry of Health facilities in Malaysia manage approximately 1.6 million individuals with diabetes. Due to the high risk for cardiovascular disease, SGLT2i are indicated for these patients.

METHODOLOGY

This study looks at metabolic benefits for subjects started on SGLT2 inhibitors in tertiary hospital settings. This retrospective cohort study included patients with T2D who started on SGLT2i (empagliflozin or dapagliflozin) from 2018 to 2024. Data on age, weight change, HbA1c and total daily dose of insulin (TDD) were obtained for one year from initiation of SGLT2i.

RESULTS

Total sample recruited was 100. Mean age was 57.2 years. Six subjects were on dapagliflozin, and 94 subjects were on empagliflozin. Mean baseline weight was 80.6 kg, HbA1c was 9.19% and insulin TDD was 45.46 units. At one year, mean weight reduction was 2.54 kg (95%CI [-3.556,-1.528]), P = <0.001. Mean HbA1c change was -0.02% (95%CI [-0.730, -0.695]), P = 0.961. Similarly, a slight reduction of insulin TDD by 2.6 units was observed at one year (95%CI [-6.51, -1.28], P = 0.184).

CONCLUSION

Significant weight reduction was observed at 1 year, similar to other studies. Most patients reported the greatest weight loss during the first three months, possibly due to the diuretic effect of SGLT2i. Though HbA1c did not show a significant reduction in our cohort, the insulin TDD was slightly lower at 12 months, which may translate to a long-term reduction in healthcare costs. Limitations include fewer patients on dapagliflozin as this medication was only recently available in our facilities. Future studies should include a follow-up period with data on cardiovascular and renal outcomes.

EP_A057

NEVER TOO OLD FOR AUTOIMMUNE DIABETES: A CASE REPORT OF LADA DIAGNOSED IN AN ELDERLY PATIENT

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INTRODUCTION/BACKGROUND

Latent autoimmune diabetes of adults (LADA) is characterized by slow, progressive immune-mediated destruction of pancreatic islet cells, accounting for 2-12% of diabetes in adults. It is diagnosed in individuals more than 30 years old with positive diabetes-autoantibody. Diagnosis can be challenging and sometimes delayed as these patients fit neither type 1 nor type 2 diabetes phenotypes.

CASE

We report a case of late diagnosis of LADA in a 70-year-old male who was presumed to have type 2 diabetes mellitus (T2DM) and initially presented with multiple episodes of diabetic ketoacidosis (DKA) four years ago.

A 70-year-old Chinese male was diagnosed with T2DM 4 years ago and was started on treatment with metformin, vildagliptin and premixed human insulin. Despite good compliance with treatment, HbA1c remained very high (10-12%). He did not have a history of DKA, had no family history of autoimmune disease, no previous COVID infection. He was lean with a BMI of 17 kg/m^2 and there were no features of insulin resistance.

He presented to the hospital with severe DKA (blood sugar 26.8 mmol/L, pH 7.003, HCO3 5 mmol, ketone 7 mmol/L), attributed to atypical pneumonia. He responded to antibiotics and insulin with dextrose infusion and was subsequently discharged well with oral antidiabetic medications and basal insulin. However, after seven days, he was admitted again for severe DKA and was given intravenous steroids for adrenal insufficiency (AI). Subsequent cosyntropin tests ruled out AI. Diabetes autoantibody was requested and came back positive for anti-GAD, anti-ICA and anti-IA2. Treatment was shifted to basal-bolus insulin, resulting in improved HbA1c and no recurrence of DKA.

CONCLUSION

Diagnosis of LADA can be challenging. However, features of insulinopaenia such as DKA and the absence of clinical features of insulin resistance should raise clinical suspicion regardless of the patient's age of presentation.

EP_A058

TIME TO DISCONTINUATION OF SGLT2 INHIBITORS AMONG ADULTS WITH TYPE 2 DIABETES AT UNIVERSITI MALAYA MEDICAL CENTRE

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INTRODUCTION

Sodium-glucose cotransporter-2 inhibitors (SGLT2i) have emerged as a new guideline-directed medical therapy (GDMT) for managing cardiovascular-kidney-metabolic (CKM) syndrome. Understanding the pattern of SGLT2i discontinuation can help prevent unwarranted discontinuation of this GDMT and simultaneously develop interventions to mitigate its possible adverse sequelae. We aimed to evaluate the time to discontinuation of SGLT2i based on patient-, clinical- and medication-related factors among adults with type 2 diabetes (T2D) at the Universiti Malaya Medical Centre, Kuala Lumpur, Malaysia.

METHODOLOGY

We conducted a retrospective cohort study involving adults aged 18 years and above with T2D who were initiated with SGLT2i between January 2016 and December 2021. We used the Kaplan-Meier curves with log-rank tests to estimate the median time to SGLT2i discontinuation.

RESULTS
A total of 602 adults with T2D were analysed. The overall median time to SGLT2i discontinuation was 40.5 months (95%CI [34.6, 54.0]). Adults with T2D who were on empagliflozin (vs. dapagliflozin; $P = 0.041$) and concomitant DPP4 inhibitors ($P = 0.028$) had significantly longer treatment persistence. Additionally, adults with baseline eGFR <60 ml/min/1.73 m^2 discontinued SGLT2i earlier than those with baseline eGFR ≥60 ml/min/1.73 m^2 ($p = 0.002$). The overall treatment persistence rates at 6 months, 1 year, 3 years and 5 years were 78.0%, 68.2%, 49.4% and 42.6%, respectively. The top 3 reasons for SGLT2i discontinuation were as follows: 1) high pill burden and nonadherence (15.8%); 2) a decline in eGFR and acute kidney injury (10.3%); and 3) financial constraints (8.4%).

CONCLUSION
This study provides valuable insights into the time to SGLT2i discontinuation in adults with T2D at an urban academic institution. As SGLT2i are the GDMT for CKM syndrome, the underlying factors behind unwarranted SGLT2i discontinuation should be explored to facilitate more personalized diabetes management to optimize health outcomes.

EP_A059

THE IMPACT OF LIFESTYLE MODIFICATION ON METABOLIC SYNDROME AMONG MOH STAFF

https://doi.org/10.15605/jafes.039.S1.070

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INTRODUCTION/BACKGROUND
Metabolic syndrome (MetS) represents a pressing global public health concern, marked by a constellation of metabolic irregularities such as elevated blood pressure, dyslipidaemia, elevated fasting blood glucose and central obesity, heightening the risk for type 2 diabetes mellitus and cardiovascular disease. Despite evidence endorsing lifestyle interventions, local data on their effectiveness in Malaysia are scarce.

METHODOLOGY
This study explores the impact of lifestyle modifications on MetS among Ministry of Health (MOH) staff at Hospital Shah Alam (HSAS) to guide policy-level interventions for improved public health outcomes. Using data from KOSPEN 2020 at HSAS, this cohort study focused on lifestyle modifications from July 2021 to July 2022, comprising four arms: the diet group, exercise group, exercise + diet group, and control group. Due to challenges with recruitment and adherence, the sample size was limited, and the follow-up period was abbreviated.

RESULTS
With 36 participants recruited (30.6% males, 69.4% females; mean age: 40.28 years), no significant differences in key parameters were noted at 3 and 6 months. However, during the 9-month reassessment, the diet group demonstrated a significant mean reduction in SBP ($P = 0.005$). On the other hand, the diet + exercise group exhibited decreased FBS compared to the diet ($P = 0.037$) and control groups ($P < 0.001$).

CONCLUSION
Despite constrained statistical significance likely attributed to high dropout rates and adherence issues, dietary control, exercise, or their combination indicate efficacy in managing MetS. Further methodically structured research is imperative to deepen our comprehension of these relationships.

EP_A060

EAST COAST ENDOCRINE TERTIARY CENTRE EXPERIENCE WITH GLUCAGON-LIKE PEPTIDE-1 (GLP-1) RECEPTOR AGONISTS

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INTRODUCTION/BACKGROUND
GLP1-RA is licensed for the treatment of Type 2 Diabetes Mellitus (DM) and weight reduction in obese patients. These agents have been proven effective without increased risk of hypoglycaemia and with significant weight reduction benefits.

METHODOLOGY
This retrospective cohort study aims to determine the clinical outcome of Type 2 DM and obese patients started on

GLP-1RA in Hospital Sultanah Nur Zahirah Terengganu, a tertiary centre in East Coast Malaysia. Clinical outcomes assessed were weight, BMI, total daily dose of insulin (TDD), HbA1c reduction and adverse events after six months of treatment initiation.

RESULTS

A total of 12 patients were eligible for this study, with a median age of 48 years (24.3). The majority were Malays (91.7%), with 1 Indian. There were equal numbers of males and females. Ten patients had diabetes, with four diagnosed for more than ten years. Nine (75%) were on insulin treatment prior to GLP1-RA initiation, with a median baseline TDD of 66 IU/day (74.0). Two patients were started on GLP1RA for obesity. Eight patients were on injectables, and the remaining were on oral GLP1RA. At baseline, the median weight, BMI and HbA1c were 123.3 kg (49), 48 kg/m^2 (18.9), and 8.5% (3.8), respectively. After six months, there were significant reductions in median weight and BMI, 111 kg (47.3) and 41.3 kg/m^2 (16.2) (P-value = 0.012, 0.018, respectively). A median weight reduction of 5% from baseline was observed. There was a reduction in median HbA1c to 7.7% (4.0), however, this was not statistically significant (P-value = 0.34). No change in TDD was observed (P-value = 0.85). Three patients (25%) experienced mild gastrointestinal symptoms, which did not require discontinuation of GLP1RA.

CONCLUSION

GLP1-RA is effective for weight loss even with a shorter treatment duration, while the effect on HbA1c and TDD reduction may require a longer treatment duration.

EP_A061

DECIPHERING THE PUZZLE: GLP-1 AGONIST-INDUCED ACUTE KIDNEY INJURY UNRELATED TO MEDICATION SIDE EFFECT

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INTRODUCTION/BACKGROUND

GLP-1 agonists are crucial treatments for type 2 diabetes and obesity due to their positive impact on glucose control and weight. However, concerns have arisen about their potential to cause pre-renal acute kidney injury (AKI), typically attributed to gastrointestinal side effects. This case presents a notable anomaly: AKI occurring without gastrointestinal symptoms, prompting further investigation into the mechanisms behind GLP-1 agonist-associated AKI.

CASE

A 35-year-old Malay male was seen with morbid obesity class II, young-onset hypertension and stable chronic kidney disease stage 3. He was started on semaglutide at 0.25 mg weekly, with plans to up titrate to 0.5 mg weekly. Upon initiation, renal function showed a concerning decline in eGFR from 54 to 35 mL/min/1.73 m^2. Despite ruling out gastrointestinal side effects or dehydration and obstructive uropathy through ultrasound, semaglutide was temporarily withheld. After a month without the medication, his eGFR improved to 53 mL/min/1.73 m^2. We then cautiously initiated Liraglutide at 0.6 mg OD which resulted in a weight reduction from 98 kg to 94 kg within a month. However, renal function deteriorated further, with eGFR dropping to 34 mL/min/1.73 m^2 and creatinine levels rising to 204 mmol/L. Understanding the importance of preserving renal function, we subsequently discontinued GLP-1 agonist therapy.

CONCLUSION

Despite the absence of gastrointestinal side effects, GLP-1 receptor agonists can still be associated with acute kidney injury. Hence, it is important to monitor renal profile regularly, especially when starting treatment with these medications.

EP_A062

INTERTWINING FATE OF PROLACTIN AND METABOLISM: THE OVERLOOKED CAUSAL EFFECT RELATIONSHIP

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INTRODUCTION/BACKGROUND

Hyperprolactinaemia has always been regarded as an excess of hormones resulting in metabolic abnormalities. However, recent studies have shown that prolactin has a protective value against metabolic disorders in obese individuals.[1] Increased circulating prolactin inhibits adipocyte hypertrophy, downregulates expression of inflammatory cytokines in visceral adipose tissue and alleviates insulin resistance.[1] However, chronically high prolactin influences orexigenic-anorexigenic hormones, resulting in hyperphagia, weight gain and obesity.[2] We present a patient with Metabolically-Healthy-Obesity [MHO] with hyperprolactinemia.

CASE
A 33-year-old male, presented with hypogonadism and infertility. His symptoms of hypogonadism preceded an increase in weight of 40 kg within two years. Blood pressure was 112/78 mmHg. He had sparse facial and axillary hair with Tanner stage 2. His latest weight was 164 kg (BMI 52 kg/m^2). He had no acanthosis nigricans. His serum prolactin was 5985 m IU/L. He had low fasting morning serum testosterone [1.11 nmol/L]. FSH and LH levels were both low [0.3 IU/L]. His metabolic parameters were all normal: HDL 1.1, LDL 2.7, Triglyceride 1.1 (mmol/L), HbA1c 5.3%, Fasting Blood Sugar 4.7 mmol/L. MRI showed pituitary microadenoma. Other anterior pituitary hormonal assays were normal. He was started on Cabergoline 0.5 mg once a week.

CONCLUSION
The relationship between hyperprolactinemia and obesity is complex. In our patient, the sudden weight gain coincided with symptoms of hypogonadism due to hyperprolactinaemia. Thus, hyperprolactinaemia may have contributed to his weight gain. He also has normal metabolic parameters despite being obese class 3, which could indicate the protective value of elevated prolactin levels. In treating obese patients with hyperprolactinemia, a prolactin level that is too high or too low is proven to be unfavourable.[3] Thus, treatment with cabergoline has been shown to help in reducing weight in this group of patients while keeping the serum prolactin at a slightly higher level to avoid diminishing the protective value of prolactin in obese patients.[3,4,5]

EP_A063

A CASE OF KETOGENIC DIET-INDUCED ELEVATED CHOLESTEROL

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INTRODUCTION/BACKGROUND
Weight management through exercise and dietary intervention has been shown to reduce cardiovascular risk. Here, we report a case of significant weight loss achieved through exercise and a ketogenic diet, resulting in a deranged lipid profile.

CASE
A 39-year-old female with underlying class 3 obesity and type 2 diabetes mellitus on metformin 500 mg BD was first seen in March 2022. Her weight was 153 kg and her BMI was 58.2 kg/m^2. Since then, she has diligently followed a strict ketogenic diet and exercised daily. She ate one meal a day, and her diet primarily consisted of meat, eggs, nuts, cheese and fresh cream. Over the past 18 months, she successfully lost 85 kg, and her BMI decreased to 25.6 kg/m^2. Notably, her body composition improved, and her diabetes went into remission. However, her lipid profile exhibited marked derangement.

Physical examination revealed no significant abnormalities, and it was noteworthy that both her parents had a history of dyslipidaemia. In response to the deranged lipid profile, the patient was advised to discontinue her current dietary plan. Instead, a more balanced diet emphasizing lower fat content, higher fibre intake and appropriate carbohydrate levels was recommended. Additionally, she was initiated on atorvastatin in October 2023 to address the lipid abnormalities. Regular monitoring and follow-up appointments were scheduled to track her progress.

CONCLUSION
While intermittent fasting and the ketogenic diet can undoubtedly lead to desired weight loss, patients should be counselled and carefully monitored by their physicians. This case illustrates that much is still unknown about the impact of these diets.

EP_A064

BODY COMPOSITION CHANGES 12 MONTHS POST-METABOLIC SURGERY IN MALAYSIAN ADULTS WITH OBESITY: THE DIFFERENCES IN DIABETIC STATUS

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INTRODUCTION
Body composition analysis following metabolic surgery is vital for clinical evaluation and monitoring treatment outcomes. In Malaysia, however, the evidence for these changes is limited. We aimed to explore the changes in body composition in patients with obesity after 12 months of metabolic surgery, according to their diabetes status.

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/m^2 were categorized into two groups: non-diabetes mellitus (non-DM) and diabetes mellitus (DM). Body composition components, including skeletal muscle mass (SMM), percentage body fat (PBF) and visceral fat area (VFA), were measured using a bioimpedance analyser (InBody S10). Statistical analysis was conducted using the SPSS software version 29.

RESULTS

This study involved 121 patients, most of whom were female (n = 74, 61.2%) and Malay (n = 82, 67.8%). The overall mean age was 39.02 (SD 7.8) years. At baseline, there was no significant difference in mean BMI between the non-DM and DM groups (P = 0.203). At six months, significant improvement was observed in weight, BMI, WC, PBF, VFA, and SMM in both groups (P <0.001) compared to baseline. These significant improvements in all parameters were maintained up to month 12 in both groups (P <0.05), except for SMM in patients in the non-DM group (P >0.999). Nevertheless, there is no significant difference in between-group comparison for all parameters throughout the study period (P >0.05).

CONCLUSION

Metabolic surgery has significantly improved body composition both in diabetic and non-diabetic individuals at six months, with benefits persisting at 12 months. Continuous monitoring for both groups is crucial for maintaining long-term benefits and optimizing outcomes.

EP_A065

AN OBSERVATIONAL ANALYSIS OF INSULINOMA FROM A TERTIARY CARE CENTRE

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INTRODUCTION/BACKGROUND

Insulinomas are the most common pancreatic neuroendocrine neoplasms. Diagnosis requires the demonstration of inappropriately high insulin and C-peptide levels after a prolonged fast, followed by tumour localization by radiological methods and endoscopic ultrasound with functional scans if suspicious for metastasis. Tumour removal by surgery or radiofrequency ablation (RFA) remains the mainstay of treatment.

METHODOLOGY

Data of patients from 2000 to 2023 diagnosed as functioning pancreatic NET in Hospital Putrajaya, Malaysia, were analysed retrospectively. This study aimed to evaluate the clinical features, preoperative laboratory results, imaging diagnosis, surgical treatments and pathologic findings of insulinomas in this centre.

RESULTS

Of 21 patients with functioning pancreatic NET, 19 patients were diagnosed with insulinoma with a male/female ratio of 0.6:1. Malignant insulinomas comprised 16.7% of patients. The median age of onset was 41 years. All patients presented with autonomic symptoms, more than 80% presented with neurological symptoms, and 38% had weight gain. Mean duration of symptoms prior to diagnosis was 2.3 years. All three diagnostic criteria of the functional European Neuroendocrine Tumour Society were met by 89% of patients, while 11% met two of the three criteria. The preoperative detection rates of CT, MRI and EUS were 67%, 40% and 78%, respectively. ASVS was diagnostic in 71% of patients. Regarding treatment modalities, 40% of patients underwent pancreatic enucleation, 40% had partial pancreatectomy and 6% had RFA. The mean tumour size was 2.3 cm. Ki-67 were all less than 20%, with 88% having Ki-67 G1 or were well-differentiated. On further follow-up, 10% of these patients developed other manifestations of MEN-1 syndrome.

CONCLUSION

Our observational analysis showed that insulinomas were diagnosed in young to middle-aged patients with female preponderance. Initial presentations were mainly neurological, autonomic and with weight gain. Most also fulfilled the ENET biochemical criteria. The tumours are mostly small in size and have a low proliferative index. Clinical and biochemical manifestations for malignant insulinomas do not significantly differ from benign ones.

EP_A066

WHEN INFERTILITY UNVEILS UNIFYING DIAGNOSIS: AN INSIGHT TO THE DISEASE

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INTRODUCTION/BACKGROUND
Infertility involves investigation for primary or secondary causes. The presence of hypergonadotropic hypogonadism signifies premature ovarian failure (POF). When POF is accompanied by other endocrinopathies, a suspicion of underlying autoimmunity is possible. Autoimmune Polyglandular Syndrome (APS) is one of the rarer polyendocrinopathies that have multiple subtypes and clinical manifestations, including POF. We present a patient with a five-year history of diabetes and Graves' disease (in remission) who came to the fertility clinic due to secondary amenorrhea.

CASE
A 36-year-old female with a background history of diabetes and Graves' disease since 2018 presented to the gynaecology team in 2021 with primary infertility. Her diabetes was diagnosed as she presented with osmotic symptoms and was then treated accordingly with a combination of insulin and oral glucose-lowering medicine by a primary care physician. The diagnosis of Graves' disease was made by the presence of typical thyrotoxic symptoms and a positive Anti-Thyroid Peroxidase Antibody (TPO). She was prescribed carbimazole and achieved remission after one year. With regard to primary infertility, she experienced oligomenorrhea, which then progressed to amenorrhea within two years. Her serial Follicle-Stimulating Hormone (FSH) and Luteinizing Hormone (LH) levels were noted to be in the menopausal range, while her progesterone was indicative of anovulation. She was then prepared for in-vitro fertilization (IVF). Suspicion of APS arose when she was admitted for diabetic ketoacidosis. The diabetes autoantibody panel was significantly positive – Anti-Islet Cells (ICA) and Anti-Glutamic Acid Decarboxylase (GAD); hence, a diagnosis of APS was made.

CONCLUSION
Manifestations of APS vary and, most of the time, will present multiple endocrine dysfunctions within a period of time. Diagnosis and management are clinically challenging. Therefore, it may be practical to screen for other autoimmune disorders in such patients periodically.

EP_A067

THE LIVER VERSUS BONE CONUNDRUM IN ISOLATED RAISED ALKALINE PHOSPHATASE LEVEL

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INTRODUCTION/BACKGROUND
Alkaline Phosphatase (ALP) is an enzyme that catalyses the hydrolysis of organic phosphate esters. It is concentrated in the liver, bone, kidney, intestinal mucosa and placenta. When ALP is elevated in isolation, isoenzyme studies using electrophoresis can confirm the source. Here, we describe a case of a patient with an incidental finding of elevated ALP.

CASE
A 30-year-old female with no known medical illness was referred to the endocrine clinic for an incidental finding of persistently elevated ALP detected during routine blood investigation. She did not report any fractures or myopathy. There were no symptoms or risk factors for liver or connective tissue disease.

Clinical examination was unremarkable, with no blue sclera, bony deformities or stigmata of chronic liver disease. Her growth was normal, achieving her mid-parental height of 150 cm. Her laboratory results showed isolated elevation of ALP 226 U/L (N: 45-129) with normal gamma-glutamyl transferase 11 U/L (N: <38), alanine transaminase 11 U/L (N: 10-49), aspartate transaminase 22 U/L (N: <34), phosphate 1.24 mmol/L (N: 0.78-1.65), calcium 2.4 mmol/L (N: 2.2-2.6), haemoglobin 136 g/L (N: 120-150), white blood cell 9.4 10^9/L (N: 4-10) and platelet 230 10^9/L (N: 150-400). Liver ultrasonography did not reveal any abnormalities.

Further investigation showed a mildly raised parathyroid hormone of 9.4 pmol/L (N: 1.96-8.49) with a low 25-OH Vitamin D level at 31 nmol/L (N: >50). She was started on cholecalciferol. Parathyroid hormone and Vitamin D became normal after treatment with cholecalciferol. Her bone scan and bone density were normal. We excluded Macro-ALP by performing a polyethylene glycol precipitation test. Subsequently, ALP Isoenzyme electrophoresis was done, confirming predominant ALP from the liver at 73% (N: 18-

85%), 14% from bone (N: 14-68%), 13% from the intestine (N: <18%). As the patient was asymptomatic and had normal liver function, the hepatologist decided to monitor her biochemically and clinically.

CONCLUSION
Patients with elevated ALP should be thoroughly investigated and examined to rule out the common treatable causes. In cases of isolated raised ALP, isoenzyme electrophoresis could identify the source with the highest accuracy.

EP_A068

LIPOPROTEIN CHAOS

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INTRODUCTION/BACKGROUND
Hypertriglyceridemia pertains to blood triglyceride values greater than 2.0 mmol/L. Familial combined hyperlipidaemia, residual dyslipidaemia in well-controlled Type 2 DM and familial hypoalphalipoproteinemia are common hereditary disorders associated with hypertriglyceridemia.

CASE A
A 30-year-old female with Type 2 DM, admitted for uncontrolled DM and hypertriglyceridemia, with the following laboratory results: TG of 82.9 mmol/L (0.0-1.7 mmol/L). T Cholesterol (TC) of 14.97 mmol/L (0.0-5.2 mmol/L), non-HDL 14.48 mmol/L, LDL unmeasurable (0.0-1.95 mmol/L). She was initiated on insulin infusion, with target glucose of 8-10 mmol/L, medium chain TG (MCT) Oil 5 mls TDS, Rosuvastatin 20 mg ON, T. Fenofibrate 145 mg OD, Omega-3 1500 mg TDS. TG level decreased from 82 mmol/L to 2.7 mmol/L within one week and remained low during follow-up.

CASE B
A 48-year-old male with no comorbidities presented with left-sided weakness and facial asymmetry. He was treated as a case of cerebrovascular accident. He was incidentally noted to have hypertriglyceridemia of 11.5 mmol/L. TCl and LDL of 17.6 mmol/L and 11.7 mmol/L, respectively. Insulin infusion was initiated with fenofibrate 145 mg OD, atorvastatin 80 mg ON and Omega 3 capsules- 1 g 3 times daily. Upon discharge, his TC level was 6.3 mmol/L with TG of 4.8 mmol/L and LDL of 3.4 mmol/L. TG levels were 3.75 mmol/L during his follow-up visit with the same treatment.

CASE C
A 30-year-old female was diagnosed with hypertriglyceridemia and type 2 diabetes mellitus in the young. On admission, TC was 8.63 mmol/L, TG >12.4 mmol/L, LDL (lipaemic sample), non-HDL 8.11 mmol/L. She was initiated with insulin infusion, T. Fenofibrate 145 mg OD and Omega-3 FFA 2 g TDS. She was co-managed with a dietitian for a low TG diet with the addition of MCT oil of 8 ml BD, reduced to 5 ml BD due to intolerance. TG levels remained >12.4 mmol. After 48 hours, oral niacin 500 mg OD was added. During follow-up, TG level reduced to 3.64 mmol/L, with TC of 3.54 mmol/L and LDL of 0.82 mmol/L.

CONCLUSION
Management of hypertriglyceridemia is somewhat debatable, with some familial cases requiring plasma exchange. However, in the 3 case reports presented above, management was successful with insulin infusion, omega 3 FFA, MCT oil and statins.

EP_A069

MEN2A

https://doi.org/10.15605/jafes.039.S1.080

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INTRODUCTION/BACKGROUND
Multiple endocrine neoplasia (MEN) 2A is a rare inherited syndrome with manifestations depending on the specific RET mutation. Classical MEN2A is characterised by medullary thyroid cancer (MTC), pheochromocytoma and primary parathyroid hyperplasia.

CASE
We report a case of a 43-year-old female whose initial presentation was left flank pain, with an ultrasound showing hydronephrosis. CT scan showed a bilateral adrenal mass (>4 cm) with central necrosis. Her serum CEA and 24-hour urine metanephrine were markedly elevated. She did not present with the classical triad of pheochromocytoma. She then underwent bilateral adrenalectomy with histopathologic examination confirming pheochromocytoma. Postoperatively, she was started on fludrocortisone and hydrocortisone. A neck ultrasound was done, revealing a TR5 thyroid nodule. FNAC was done, and results showed medullary thyroid carcinoma. She was scheduled for a total thyroidectomy.

CONCLUSION
Patients with MEN might present with atypical symptoms with no positive family history. The diagnosis of pheochromocytoma will lead the clinician to investigate further to rule out MEN2A. Although MTC is usually the first manifestation of MEN2A, our patient did not present with related symptoms. Definitive treatment

of pheochromocytoma should be performed prior to treatment of MTC. All patients with pheochromocytoma should be adequately prepared before surgery, including blood pressure control. Patients with pheochromocytoma and MTC should be given a high index of suspicion for the diagnosis of MEN2A. Screening may include CEA, 24-hour urine metanephrine and neck ultrasound. Adrenalectomy and lifetime replacement of adrenal hormones should be given. Ideally, genetic testing for RET mutation should be done.

EP_A070

CHRONIC HYPONATREMIA: RESET OSMOSTAT, CHALLENGES IN DIAGNOSIS

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INTRODUCTION/BACKGROUND
Hyponatremia remains one of the most common electrolyte imbalances encountered. Hyponatremia has many causes, and it requires systematic investigation. SIADH is one of the most common causes. Reset osmostat is a rare and poorly recognised cause of mild to moderate hyponatraemia, with a presentation similar to SIADH.

CASE
We present the case of a 69-year-old male with a history of cerebrovascular disease who had been diagnosed with SIADH after extensive workup for chronic hyponatremia. He had been on fluid restriction and oral salt for years but with little effect on his serum sodium. Given his stable mild hyponatraemia, he was investigated for possible reset osmostat. Patient underwent an oral water loading test in daycare. He was given 1000 ml (15 ml/kg) of water to drink within 30 minutes and then monitored for 4 hours. Serum and urine osmolality, serum electrolytes and urine output were obtained at baseline, then hourly for 4 hours. Urine volume was also measured hourly.

His baseline serum sodium was 125 mmol/L, which dropped to 122 mmol/L at 2 hours, then returned to baseline (125 mmol/L) at 4 hours. The serum osmolality was 266 mOsm/kg, which dropped to 259 mOsm/kg and returned to baseline (266 mOsm/kg) after 4 hours. The urine osmolality at baseline was 233 mOsm/kg and dropped to 123 mOsm/kg midway through the test. Urine volume was greater than 300 ml/hour throughout the test, and the patient excreted more than 1000 ml of urine in total.

CONCLUSION
The results showed that the patient successfully excreted the water load, diluted his urine, and maintained serum sodium levels at the end of 4 hours. Although there is no consensus guideline, the findings in this case would be consistent with reset osmostat. Patients with reset osmostat usually do not require treatment. It is worthwhile to consider this diagnosis in a small subset of patients with a prior diagnosis of SIADH.

EP_A071

SEVERE HYPERTRIGLYCERIDEMIA-INDUCED ACUTE PANCREATITIS COMPLICATED WITH PERIPANCREATIC COLLECTION IN PREGNANCY

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INTRODUCTION/BACKGROUND
Acute pancreatitis in pregnancy is rare and may result in severe complications and high mortality. Elevated oestrogen levels and insulin resistance during pregnancy may lead to raised triglyceride levels, which can precipitate acute pancreatitis. We report a case of severe hypertriglyceridemia-induced acute pancreatitis complicated with peripancreatic abscess in a pregnant patient.

CASE
A 35-year-old female, G3P0+2 at 28 weeks period of gestation (POG), with a strong family history of dyslipidaemia, was admitted due to acute onset of epigastric pain and vomiting associated with shortness of breath for two days. She was electively intubated due to worsening metabolic acidosis. Initial blood results revealed elevated serum amylase at 840 IU/ml. Abdominal CECT demonstrated a bulky pancreas with free fluid at the peripancreatic region, suggesting acute pancreatitis. Further workup revealed severe hypertriglyceridemia of >32.1 mmol/L, and her capillary blood sugar ranged from 9 to 11 mmol/L. She was kept Nil by mouth and was initiated on an intravenous insulin infusion with dextrose solution. She had preterm labour on day 2 of admission. She was started on a low-fat diet and fenofibrate postpartum. Her triglyceride level reduced significantly and finally normalized on day 6 of admission. She was discharged well until nine days after discharge; she presented with right flank pain and low-grade fever. CT of the abdomen revealed extensive multiloculated rim-enhancing peripancreatic collection suggestive of

pancreatic abscess. She proceeded with image-guided drainage and started on empirical antibiotic coverage. Her symptoms improved after six days of drainage, and she was discharged well.

CONCLUSION

Hypertriglyceridemia-induced pancreatitis in pregnancy is associated with high maternal and foetal morbidity and mortality. A multidisciplinary approach, including an obstetrician, endocrinologist, surgeon and radiologist, is needed to provide the best supportive care for the patient to reduce triglyceride levels rapidly to ensure good outcomes.

EP_A072

A COMPREHENSIVE 12-MONTH ANALYSIS: REAL-WORLD ASSESSMENT OF THE CARDIOVASCULAR RISK REDUCTION CLINIC (CRRC) AT HOSPITAL PULAU PINANG, MALAYSIA

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INTRODUCTION/BACKGROUND

Atherosclerotic Cardiovascular Disease (ASCVD) remains the primary driver of cardiovascular disease (CVD)-related mortality, responsible for a staggering 80% of CVD-related deaths. The consistent association of low-density lipoprotein cholesterol (LDL-C) with CVD and coronary heart disease (CHD) underscores the potential of reducing LDL-C levels in mitigating atherosclerotic plaque progression and lowering ASCVD incidence. This has elevated lipid-lowering therapies (LLT) as pivotal interventions to curtail ASCVD-related mortality and morbidity.

METHODOLOGY

This single-arm, observational study, enrolled patients aged 18 years and above with established ASCVD or ASCVD-risk equivalent conditions and LDL-C levels exceeding 1.8 mmol/L.

RESULTS

A cohort of 58 patients, with a mean age of 57.5 (SD 11.0), predominantly male (86%), were included. Almost all patients had prior ASCVD (98.6%), 67.1% had a history of cardiac surgery/intervention and 71.4% had multivessel disease. Hypertension was common (74.3%), followed by diabetes (35.7%). Only 4.3% had familial hypercholesterolemia. Most patients were Malay (41.4%) and Chinese (38.6%). Fifty-seven (81.4%) had follow-ups at month 6 and thirty-four patients (48.6%) followed up at month 12. Analysis of patients who returned for follow-up showed that compared to baseline, mean LDL-C reduced by 1.7 mmol/L (44.0%) at month 6 (P <0.0001) and 1.20 mmol/L (38.3%) at month 12 (P <0.0001). However, none of the patients attained LDL-C <1.8 mmol/L and <1.4 mmol/L at baseline, 52.6% and 44.1% achieved LDL-C <1.8 mmol/L at month 6 (P <0.0001) and month 12 (P = 0.0003), respectively. Similarly, 26.3% and 20.6% attained <1.4 mmol/L at month 6 (P = 0.0003) and month 12 (P = 0.023) post-CRRC, respectively.

CONCLUSION

This pilot study provides substantial real-world evidence supporting the effectiveness of the Cardiovascular Risk Reduction Clinic in reducing mean LDL-C levels within 12 months. The findings underscore the positive influence of physician-prescribed lipid-lowering therapy strategies and patient counselling on LDL-C management.

EP_A073

NON-ISLET CELL TUMOR HYPOGLYCAEMIA IN A FRAIL ELDERLY PATIENT: A CASE REPORT

https://doi.org/10.15605/jafes.039.S1.084

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INTRODUCTION/BACKGROUND

Non-islet cell tumor hypoglycemia (NICTH) is a rare paraneoplastic phenomenon, representing the second most common aetiology of spontaneous hypoglycemia in non-diabetic patients after insulinoma. It is seen in patients with extra-pancreatic tumors, usually of mesenchymal, vascular, or epithelial origin. The underlying mechanism involves tumor over secretion of incompletely processed insulin-like growth factor-2 (pro-IGF-2), leading to the activation of insulin receptors and thereby hypoinsulinaemic hypoglycaemia. We report an elderly female, who presented with recurrent spontaneous hypoglycaemia, following which subsequent workup confirmed the diagnosis of NICTH secondary to a left lung mitotic lesion.

CASE

An 86-year-old, non-diabetic female presented with early morning spontaneous hypoglycaemia of two years' duration. She was found by her family confused and was only able to regain full consciousness after taking sweet beverages or nutritional drinks. Her documented capillary blood glucose levels during these episodes confirmed that she had hypoglycemia. She denied weight change and altered bowel habits. She had a strong family history of malignancy, wherein five of her eight children had colorectal carcinoma. Supervised fasting test showed low C-peptide, undetectable serum insulin and low blood ketone when the concurrent blood glucose level was 1.1 mmol/L. Following glucagon challenge, her blood glucose rose to 3.3 mmol/L. Sulphonylurea screen was negative. Serum IGF-2 was not tested due to unavailability. A diagnosis of NICTH was made. CECT thorax revealed a large left lower lobe lung tumour, measuring 11.5 x 8.9 x 12.9 cm, which partially encased the left main bronchus and descending thoracic aorta. Due to her advanced age, she refused biopsy and surgery. Her hypoglycaemia was treated with raw cornstarch therapy before bed and at 3 am, as well as oral prednisolone 5 mg daily.

CONCLUSION

The possibility of NICTH should be considered in the evaluation of spontaneous hypoglycemia in an elderly non-diabetic patient. Glucocorticoids can be effective in ameliorating hypoglycemia in this setting when surgery is not feasible.

EP_A074

ENDOSCOPIC ULTRASOUND (EUS) AND EUS GUIDED RADIOFREQUENCY ABLATION (RFA) AS A DIAGNOSTIC AND THERAPEUTIC INNOVATION FOR INSULINOMA: A CASE REPORT

https://doi.org/10.15605/jafes.039.S1.085

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INTRODUCTION/BACKGROUND

Though rare in occurrence, insulinomas are the most common hormone-producing pancreatic neuroendocrine tumour (PNET) with a reported incidence of 4 cases per million per year. Surgical intervention has been the gold standard of treatment for insulinoma but an emerging minimally-invasive method – endoscopic ultrasound (EUS) guided radio-frequency ablation (RFA) is increasing in use in the current management of insulinomas.

We present a case where EUS and EUS-guided RFA played a pivotal role in localising the lesion and serving as a therapeutic approach.

CASE

A 55-year-old female with underlying primary hypothyroidism, bronchial asthma, and class III obesity presented with refractory hypoglycaemia. Biochemical workup confirmed hyperinsulinaemic hypoglycaemia (plasma glucose 1.6 mmol/L, plasma insulin 107 pmol/L and serum c-peptide 1106 pmol/L). Initial imaging with computed tomography (CT) scan failed to localise any pancreatic lesion. Subsequent EUS discovered a hypoechoic lesion at the pancreatic tail which was later histologically confirmed to be PNET. The patient was treated with subcutaneous octreotide and oral diaxozide, then underwent EUS-RFA rather than surgery due to high operative and anaesthetic risk. The procedure was uneventful however, she developed acute pancreatitis two weeks later. Following recovery from pancreatitis, she remained in euglycemic state for a few weeks before she developed a recurrence of hypoglycemia, albeit being less frequent and less severe (plasma glucose 1.9 mmol/L, plasma insulin 67.3 pmol/L and C peptide 1179 pmol/L). Repeat diagnostic and therapeutic EUS are planned for her.

CONCLUSION

EUS is a valuable tool as a diagnostic modality in localizing insulinoma. It can be an alternative therapeutic option to surgery, especially among high-risk patients.

EP_A075

A RARE CASE OF HYPOVOLEMIC HYPONATREMIA IN A PATIENT WITH EXCESSIVE BILIARY TRACT LOSS

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INTRODUCTION/BACKGROUND

Hyponatremia is the most common electrolyte abnormality observed among inpatients in the hospital setting. Severe hyponatremia (defined as serum sodium <125 mmol/L) is associated with significant morbidity and mortality. Hypovolemic hyponatremia occurs in the context of extracellular fluid depletion. Therefore, accurate diagnosis

and judicious fluid therapy are essential in managing patients with hypovolemic hyponatremia.

We report a case who developed severe hypovolemic hyponatremia following the insertion of a percutaneous transhepatic biliary drain for pancreatic head cancer.

CASE
A 66-year-old female with type-2 diabetes mellitus and hypertension was diagnosed with carcinoma of the head of the pancreas. The patient underwent percutaneous transhepatic biliary drainage (PTBD), which involved inserting a pigtail to facilitate biliary drainage. She presented with a 2-day history of altered behaviour and lethargy. Her Glasgow Coma Scale (GCS) was E4V3M5 and serum sodium level was 112 mmol/L. Prior to PTBD insertion, the patient had a baseline sodium level of 133 mmol/L. The patient's PTBD output ranged from 300 mL to 1400 mL daily. Furthermore, the patient's serum osmolarity was 252 mOsm/kg, her urine osmolarity was 331 mOsm/kg, and her urine sodium was 21 mmol/L, which indicated hypovolaemic hyponatremia. She was initially managed with isotonic fluid (NaCl 0.9%) replacement, and her serum sodium improved to 130 mmol/L. However, the patient's IVD regime was not adjusted to account for PTBD drain output, resulting in fluctuating serum sodium levels ranging from 107 to 119 mmol/L. The patient's bile fluid was sent for analysis, which revealed a sodium level of 119 mmol/L. She was restarted on IVD (NaCl 0.9%) for 4 pints to normalise her serum sodium level while waiting for surgery and oncology therapy.

CONCLUSION
Biliary tract loss is a rare cause of hypovolemic hyponatremia. It is important to recognize it in order to plan for appropriate fluid replacement.

EP_A076

POSTPRANDIAL HYPOGLYCEMIA: A RARE PRESENTATION OF AN INSULINOMA

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INTRODUCTION/BACKGROUND
Insulinomas typically present with fasting hypoglycemia, owing to the autonomous secretion of insulin from this neuroendocrine tumor. A presenting complaint of postprandial hypoglycemia in a patient with insulinoma is rare, with a slight male predominance.

CASE
A 66-year-old male with chronic kidney disease (stage G4A2) and interstitial lung disease presented to our centre with two episodes of post-prandial hypoglycaemia, resulting in loss of consciousness. The first occurred 2 hours after breakfast; the second, a month later, occurred 3 hours after breaking fast following a 14-hour fast during Ramadan. During the episode of postprandial hypoglycaemia, he fulfilled Whipple's triad with venous blood glucose 1.0 mmol/L, insulin 42.9 m IU/L and C-peptide 21.1 ng/mL. A 72-hour prolonged fast conducted and terminated at 42 hours revealed a nadir glucose of 2.5 mmol/L but low insulin of 2.0 m IU/L and elevated C-peptide 2.2 ng/mL. Peak ketone was 2.4 mmol/L, consistent with endogenous hyperinsulinism. A mixed meal test triggered hypoglycaemia of 2.3 mmol/L at 210 minutes, with insulin 25.5 m IU/L and C-peptide 17.2 ng/mL. Sulphonylurea screen and insulin autoantibodies were negative.

Both transabdominal and endoscopic ultrasounds revealed a 3.3 x 3.3 cm hyperechoic head of pancreas mass. A fine needle biopsy during EUS was done and immunohistochemistry of the specimen was positive for insulin and synaptophysin, consistent with insulinoma. The patient was started on diazoxide and put on a continuous glucose monitoring system. This also showed low glucose readings predominantly after meals. Unfortunately, he developed severe pneumonia and passed away prior to functional imaging and definitive surgery.

CONCLUSION
Kidney disease does elevate C-peptide levels, which may confound the evaluation of hypoglycemia.2 Clinicians should have a high index of suspicion for insulinoma, even in patients with a history of predominantly postprandial hypoglycaemia when confronted with high C-peptide levels in both fasting and postprandial states.

EP_A077

EFFECT OF RAMADAN FASTING ON NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD): A PROSPECTIVE OBSERVATIONAL STUDY

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INTRODUCTION

Ramadan fasting is known to have positive impacts on health. Its influence on individuals with non-alcoholic fatty liver disease (NAFLD), as measured by the liver stiffness measurement (LSM) remains uncertain. Therefore, this study aimed to determine the effect of Ramadan fasting on patients with NAFLD by assessing the LSM, anthropometric and biochemical parameters of fatty liver.

METHODOLOGY

A prospective observational study was conducted on 51 NAFLD patients, who were fasted during Ramadhan month. We analysed pre- and post-Ramadan liver stiffness measurement (LSM), and anthropometric and biochemical parameters of fatty liver.

RESULT

The results showed a statistically significant decrease in LSM after fasting (P <0.05), with a mean difference of 1.62 (95%CI: 1.34, 1.90). There were also statistically significant weight, waist circumference, and BMI reductions after the fasting period (P <0.05). The mean differences, along with their respective 95% confidence intervals, demonstrate a decrease in weight by 5.02 (3.90, 6.15) kg, waist circumference by 3.41 (1.99, 4.83) cm, and BMI by 2.14 (1.58, 2.70) kg/m^2. Based on biochemical parameters, there were statistically significant reductions in fasting blood sugar (FBS) and HbA1c (P-value <0.05), with a decrease of 0.55 mmol/L and 0.16%, respectively. Similarly, aspartate aminotransferase (AST), alanine aminotransferase (ALT), cholesterol, triglycerides (TG), and low-density lipoprotein (LDL) were also shown to have reductions post-fasting, with a mean difference (95%CI), 18.35 (14.88, 21.83), 21.27 (17.21, 25.34), 0.80 (0.58, 1.01), 0.27 (0.18, 0.36), and 0.82 (0.61, 1.02), respectively.

CONCLUSION

The study demonstrates that Ramadan fasting could improve LSM, anthropometric, and biochemical measurements among patients with NAFLD.

EP_A078

COMPARISON OF DENOSUMAB AND ALENDRONATE EFFICACY AND RELATED DIABETES RISK IN PATIENTS WITH OSTEOPOROSIS

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INTRODUCTION/BACKGROUND

Osteoporosis and diabetes mellitus are highly prevalent among postmenopausal women and the elderly in Malaysia. Recent studies have suggested blocking the RANK ligand may improve glucose metabolism and delay the development of diabetes.

METHODOLOGY

We compared the efficacy of denosumab with alendronate in treating osteoporosis and examined the potential reduction of the risk of diabetes in each treatment group.

RESULTS

This is a cross-sectional analysis of patients over 40 years old who received osteoporosis treatment with denosumab and alendronate for more than a year at Putrajaya Hospital. Independent t-test and Pearson's chi-square were used to examine associations between continuous and categorical variables.

A total of 182 patients were included (mean [SD] age, 71.2 [10] years; 170 [93%] female). The majority were of Malay descent (47%) followed by Chinese (32%) and Indian (20%). Hypertension was the most common comorbidity (63%) followed by dyslipidaemia (48%), diabetes (29%) and prediabetes (15%). Majority with previous osteoporosis fractures received denosumab (54%) versus alendronate (44%). The denosumab group showed significant improvements in bone strength (p <0.01) mainly at the lumbar spine compared to the alendronate group. Patients who developed diabetes were numerically lower in the denosumab group compared to the alendronate group, 53% vs 63%, respectively, with an OR of 0.688 (95%CI, 0.34 – 1.39).

CONCLUSION

These findings highlight significant improvements in BMD with the use of denosumab compared to alendronate, particularly at the lumbar spine. Additional prospective studies are needed to establish the role of denosumab in lowering diabetes risk in osteoporosis patients.

EP_A079

A RETROSPECTIVE STUDY ON PATIENTS UNDERGOING PARATHYROIDECTOMY FOR PRIMARY HYPERPARATHYROIDISM

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INTRODUCTION/BACKGROUND
Primary hyperparathyroidism (PHPT), characterized by hypercalcemia with elevated or inappropriately normal parathyroid hormone (PTH) level, has an estimated incidence of one to seven per 1000 adults. Parathyroidectomy remains the only definitive treatment and offers cure for this condition.

METHODOLOGY
A retrospective study was done to determine the demographic and clinical severity of patients undergoing parathyroidectomy in Hospital Raja Permaisuri Bainun (HRPB), and its surgical outcomes.

A retrospective study was done for all patients with PHPT who underwent parathyroidectomy in HRPB, from the year 2018-2023. Demographics, laboratory and radiologic investigations including levels of serum PTH, serum adjusted calcium, serum alkaline phosphatase, and post-operative complications were recorded from patients' admission notes and electronic medical records and were analysed using SPSS.

RESULTS
Twenty-five patients with a mean age of 58.8 ± 9.4 years were included in the study, of which, 72% were female. Of the total, 76% had a single parathyroid adenoma, 12% had parathyroid hyperplasia and 12% had parathyroid carcinoma. Pre-operative mean serum calcium was 2.86 ± 0.26 mmol/L, mean serum alkaline phosphatase was 104 ± 87 IU/L, while pre-operative median PTH level was 38.38 pmol/L (interquartile range = 68.63).

Parathyroid lesions were localized by ultrasound of the neck (87.5%) and by parathyroid scintigraphy (88.9%). Complications included osteoporosis (57.1%) and nephrolithiasis (52.6%). Sixty percent received calcium supplementation during the immediate postoperative period. Two patients developed post-operative hypocalcaemia, two had neck hematoma and one was complicated with recurrent laryngeal nerve palsy.

CONCLUSION
Half of our patients were diagnosed with PHPT-related complications prior to surgery, suggesting a need for better screening strategies. Parathyroidectomy offers high cure rates for primary hyperparathyroidism in the hands of experienced surgeons and should be recommended to patients meeting the criteria for surgery.

EP_A080

HYPOPARATHYROIDISM IN PREGNANCY AND LACTATION: BALANCING CALCIUM SWINGS

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INTRODUCTION/BACKGROUND
Management of hypoparathyroidism includes calcium and activated vitamin D supplementation. The dose often requires adjustment during pregnancy and lactation. The combination of increased calcium requirements and the dynamics of parathyroid hormone-related protein (PTHrP) secretion from placental and breast tissue may shift calcium balance to a variable extent.

CASE
A 32-year-old female with a history of hypoparathyroidism after undergoing total thyroidectomy for micropapillary thyroid cancer was followed up during her pregnancy. Her baseline treatment was calcitriol 1.0 mcg and calcium carbonate 1000 mg twice daily. There was a significant drop in serum calcium levels during early pregnancy that required admissions for correction of symptomatic hypocalcaemia. Her calcitriol dose tripled in the first trimester before being slowly reduced back to baseline in the mid-second trimester and stabilizing afterward. She had an uneventful delivery at 37 weeks and gave birth to a healthy newborn. She stopped taking calcitriol postpartum while fully breastfeeding for 4 months and was completely asymptomatic during that period with a documented calcium level of 2.14 mmol/L. However, within 2 weeks of cessation of breastfeeding, she presented with symptoms of hypocalcaemia and a corrected calcium level of 1.87 mmol/L. Treatment with calcitriol was hence reinstated.

CONCLUSION
PTHrP production by the placenta and lactating breast results in increased endogenous calcitriol levels. This subsequently enhances intestinal calcium absorption to meet heightened physiological calcium demands. However,

the balance between these two opposing physiologies varies between individuals. This is a rare case documenting a dramatic decline in the need for calcitriol in a patient with hypoparathyroidism during the postpartum and lactation period, followed by a sudden resurgence in calcitriol requirement occurring immediately upon cessation of breastfeeding.

EP_A081

DIFFERENT FACADES OF PTH-DEPENDENT HYPERCALCEMIA IN PREGNANCY

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INTRODUCTION/BACKGROUND
Hypercalcemia is a rare occurrence during pregnancy. This can present variably and pose unique challenges in management. The general diagnostic approach is similar to the non-pregnant population however, additional considerations must be taken regarding the modality of investigations and safe treatment options during pregnancy. We present 3 pregnant patients who had PTH-dependent hypercalcemia. We explore their clinical presentation, diagnostic evaluation, management, and outcomes. Through this case series, we aim to highlight different aspects of management for hypercalcemia during pregnancy.

CASE 1
A 32-year-old patient at 33 weeks period of gestation (POG) presented with acute pancreatitis and was found to have hypercalcemia 2.99 mmol/L and raised iPTH 16.64 pmol/L (reference range 1.59 - 7.24). Calcium levels showed a decreasing trend with hydration alone and the patient had an uneventful delivery at term. Postpartum calcium: creatinine clearance ratio (CCCR) of 0.02 confirmed primary hyperparathyroidism. Further evaluation was planned, however she defaulted on follow-up.

CASE 2
A 37-year-old patient at 15 weeks POG presented with renal impairment due to nephrolithiasis, with severe hypercalcemia 3.9 mmol/L and elevated iPTH 162.4 pmol/L. Ultrasonography of the neck showed a left lower pole parathyroid lesion measuring 1.9 x 2.3 x 2.4 cm. Hypercalcemia was refractory to hydration and required calcitonin, cinacalcet and pamidronate. Left-focused parathyroidectomy was performed at 17 weeks POG. Calcium levels normalized postoperatively. Histopathological examination confirmed parathyroid adenoma. Unfortunately, the patient opted for termination of pregnancy due to worsening renal function.

CASE 3
A 31-year-old patient was diagnosed with Familial Hypocalciuric Hypercalcemia (FHH), evidenced by mild hypercalcemia 2.8 mmol/L, elevated iPTH 8.2 pmol/L, CCCR <0.01, and normal Vitamin D levels. There was worsening hypercalcemia at 2.98 mmol/L during pregnancy which improved with hydration. The pregnancy then continued uneventfully.

CONCLUSION
Hypercalcemia is rare in pregnancy, but its treatment necessitates a delicate balancing act to ensure the safety of both mother and offspring. Treatment must be given in a timely manner, and reassurance has to be provided to patients with benign conditions such as FHH.

EP_A082

POSTMENOPAUSAL VITAMIN D SCREENING AND INITIATION OF TREATMENT

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INTRODUCTION/BACKGROUND
Menopause induces bone density loss due to oestrogen deficiency, predisposing women to osteoporosis and fractures. It is estimated that of the 200 million women affected globally, 50% are post-menopausal. Vitamin D deficiency further compounds bone healing. Recent meta-analyses show that over half the Malaysian population has inadequate levels of Vitamin D, underscoring the need for proactive measures in women's health screening. Initiating anti-resorptive medication during the early post-fracture period has in the past raised concerns about fracture healing, however, recent studies do not reflect this. The preponderance of available data suggests that anti-resorptives are safe to be initiated as early as 1-2 weeks post-fracture.

METHODOLOGY
We examined the awareness of screening for Vitamin D deficiency and the time to initiation of treatment within this demographic.

This is a retrospective study among women with postmenopausal osteoporotic fractures seen from the years 2022 to 2023 in Hospital Putrajaya, looking into screening for Vitamin D deficiency and the timing of initiation of definitive osteoporotic treatment.

RESULTS
Of the total of 101 patients screened from various departments, including endocrinology, rheumatology, orthopaedics and gynaecology, 20 patients (19.8%) with osteoporotic fractures were not screened for Vitamin D deficiency. Among the 81 screened patients, 54.3% were Vitamin D deficient, of which 2.4% were severely deficient. Furthermore, 77.2% of patients were found to have initiated osteoporosis treatment beyond two weeks after the fracture.

CONCLUSION
This study showed most patients were screened for Vitamin D deficiency, but its high prevalence should be considered. The study also shows that osteoporosis treatment was initiated beyond two weeks post fracture in majority of our patients.

EP_A083

SEVERE REFRACTORY HYPERCALCEMIA DUE TO ECTOPIC PARATHYROID LEADING TO MORTALITY

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INTRODUCTION/BACKGROUND
Hypercalcemia can manifest in a nonspecific manner, with vague symptoms which can be easily missed. Most cases of severe hypercalcemia are due to primary hyperparathyroidism or malignancy-related hypercalcemia, which is discernible by parathyroid hormone levels. We describe a case of severe refractory hypercalcemia attributed to ectopic parathyroid, which led to multiple morbidities and eventually mortality.

CASE
A 61-year-old male, with known hypertension and chronic kidney disease Stage III, presented with abdominal discomfort, loss of appetite, nausea and vomiting for 2 weeks duration. On examination, he was dehydrated, obese and hypertensive. Laboratory investigations showed markedly raised serum corrected calcium level of 5.17 mmol/L, low serum phosphate 0.63 mmol/L and iPTH of 35.39 pmol/L [NR 1.95-8.49]. Other investigations: Hb 15.6 g/dL, creatinine 303 umol/L, eGFR 18 ml/min/1.73 m^2 and urea 5.7 mmol/L. Tumour markers CA 19-9, CA 125, AFP and CEA were normal. Paraneoplastic markers were negative. Neck ultrasound did not reveal any parathyroid lesion however, computed topography of the neck-thorax-abdomen-pelvis, revealed a well-defined hypodense soft tissue lesion at the superior mediastinum, inferior to the left inferior thyroid border, measuring 2.1 x 2.6 x 3.6 cm which may represent an ectopic parathyroid gland. Severe refractory hypercalcemia was treated with vigorous intravenous saline hydration, subcutaneous calcitonin, intravenous bisphosphonates and subcutaneous denosumab. His admission was prolonged and complicated with septicaemia requiring intubation and intensive care. The patient passed away after three weeks of admission.

CONCLUSION
This case demonstrates that severe and refractory hypercalcemia attributed to an ectopic parathyroid lesion may present late due to vague initial symptoms. Admission due to severe hypercalcemia require multiple modalities of treatment, may be prolonged and carries a high risk of mortality before definitive treatment with parathyroidectomy.

EP_A084

ENDOMETRIOSIS-TRANSFORMED UTERINE CLEAR CELL CARCINOMA WITH ASYMPTOMATIC PTHrP MEDIATED HYPERCALCEMIA: A CASE REPORT

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INTRODUCTION/BACKGROUND
Hypercalcemia is a well-recognized complication of various solid tumours and hematologic malignancies. Clear cell carcinoma arising from the malignant transformation of endometriosis is a rare and typically aggressive cancer which occasionally presents only with hypercalcemia.

In this report, we describe a case of parathyroid hormone-related protein (PTHrP) hypercalcemia secondary to endometrial clear cell carcinoma including the results of biochemical laboratory tests and discuss treatment strategies with related literature reviews.

CASE
A 50-year-old female with endometriosis was incidentally found to have mild hypercalcemia during hospitalization for SAR COV (COVID-19) infection. Parathyroid hormone (iPTH) was suppressed, while PTHrP was significantly elevated at 30 pmol/L (<1.3 pmol/L). A comprehensive investigation for malignancy was done, which revealed no abnormalities except for the progressive enlargement of her underlying endometriosis. An extended hysterectomy was performed, and subsequent histological examination confirmed the presence of endometrial clear cell carcinoma. Post-surgery, her serum calcium level went back to normal levels.

CONCLUSION

This is a case of uterine clear cell carcinoma arising from endometriosis complicated with hypercalcemia and highlights that hypercalcemia may be the sole sign of disease transformation, despite the well-established aggressive nature of the disease. It is then crucial to perform a timely and thorough assessment, followed by appropriate management.

EP_A085

A GIANT PARATHYROID ADENOMA

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INTRODUCTION/BACKGROUND

Giant parathyroid adenomas, defined as adenomas weighing >3.5 gm, are rare, comprising a small fraction of all parathyroid adenomas. We describe a patient who presented with a giant parathyroid adenoma and markedly elevated parathyroid hormone.

CASE

A 57-year-old male, with a family history of adrenal Cushing's syndrome and hyperthyroidism, was incidentally diagnosed with primary hyperparathyroidism during admission for cerebral infarction, with hypercalcemia (3.2 mmol/L), elevated intact parathyroid hormone (iPTH) (140.2 pmol/L), and vitamin D deficiency (46 nmol/L). He was treated with saline diuresis, subcutaneous denosumab 60 mg, and subcutaneous calcitonin 200 U BD, but defaulted to further workup.

Nine months later, he returned with altered sensorium, hypercalcemia (3.43 mmol/L) and elevated iPTH (448.9 pmol/L), the same treatment was given as in the previous admission. Ultrasound of the parathyroid showed an interior hypoechoic lesion measuring 2.0 x 2.6 x 3.2 cm. Tc-99m Sestamibi scan suggested a left inferior parathyroid lesion without an ectopic tissue. DXA scan showed osteoporotic changes in the distal third radius and femoral neck. KUB Ultrasound showed no renal calculi. One month later he was admitted for hypercalcemia and acute kidney injury, treated with saline diuresis and subcutaneous denosumab 120 mg, and eventually underwent left inferior parathyroidectomy with intraoperative iPTH monitoring. From his highest pre-operative iPTH at 828 pmol/L, a reduction to 236.7 pmol/L was seen at 10-minutes post-incision. Intra-op findings showed a large left inferior parathyroid tumour, measuring 3.5 x 2.7 x 2.0 cm, weighing 14 gm. Histopathology was consistent with parathyroid adenoma. He was started on calcitriol and calcium carbonate post-operatively and did not develop hungry bone syndrome.

CONCLUSION

In giant parathyroid adenomas, a disproportionate rise in serum iPTH may result from factors like vitamin D deficiency, chronic iPTH elevation, or resistance to physiological effects of PTH. Distinguishing them from parathyroid carcinoma is challenging due to shared high iPTH and calcium levels, though studies showed that giant parathyroid adenomas may be asymptomatic. Histopathological examination is essential for diagnosis, warranting early surgical removal.

EP_A086

BONE HEALTH ASSESSMENT AMONG PROSTATE CANCER PATIENTS TREATED WITH ANDROGEN DEPRIVATION THERAPY IN A TERTIARY CENTRE IN MALAYSIA

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INTRODUCTION/BACKGROUND

Androgen deprivation therapy (ADT) is the cornerstone of treatment for castration-sensitive Prostate Cancer (PCa). However, the use of ADT can negatively impact bone health. This study aims to assess the prevalence of osteoporosis and osteopenia in men with PCa who have undergone ADT and identify any potential factors associated with osteoporosis among this population.

METHODOLOGY

This single-centre, cross-sectional study recruited 107 PCa patients treated with ADT at the Urology Unit, Hospital Sultanah Bahiyah from January 2020 to August 2023. Data collected included socio-demographics, comorbidities, treatment details and FRAX scores. Patients underwent Dual-energy X-ray absorptiometry (DEXA) scans and blood

investigations including renal and liver function, serum calcium, vitamin D, testosterone and oestradiol levels. Osteoporosis-associated factors were identified using logistic regression and adjusted with confounders.

RESULTS

Our patients had a mean age of 73.1 years old (SD 7.2), with 62.6% being Malay (n = 67) and a mean BMI of 24.96 (SD 4.31). Among PCa patients treated with ADT, the prevalence of osteoporosis was 57.9% (n = 62), osteopenia was present in 39.3% (n = 42), and only 2.8% (n = 3) had normal bone mineral density. The most vulnerable site was the 1/3 radius with the highest osteoporosis prevalence of 43% (n = 46), followed by femoral neck at 29% (n = 31), lumbar spine at 22.4% (n = 23), and total hip at 11.2% (n = 12). Several predictive factors of osteoporosis were identified, including the absence of calcitriol usage (Adjusted Odd Ratio (AOR) = 5.07, CI 1.04-24.75, $p = 0.04$), duration of ADT (AOR = 1.02, CI 1.0-1.04, $p = 0.03$), and ongoing ADT (AOR = 5.08, CI 1.169-22.09, $p = 0.03$). In contrast, a higher weight conferred a lower risk for osteoporosis (AOR = 0.957, CI 0.92-0.99, $p = 0.01$).

CONCLUSION

This study highlights the importance of screening for osteoporosis in men who are undergoing ADT for PCa. Peripheral BMD is an effective tool to assess their bone health. Various risk factors, such as being underweight, not receiving calcitriol, continuous ADT, and longer treatment duration, can increase the likelihood of osteoporosis.

EP_A087

A RARE CASE OF RECURRENT PARATHYROID CARCINOMA

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INTRODUCTION/BACKGROUND

One of the rare causes of primary hyperparathyroidism is parathyroid cancer. It is usually diagnosed post-operatively with histopathology. Surgery is always definitive in parathyroid carcinoma, but there have been reported recurrences of parathyroid cancer up to 20 years after a successful surgery. We report a case of a 61-year-old male who had bilateral inferior parathyroid carcinoma, surgically cured in 2020, but had a recurrence of parathyroid carcinoma after 3 years.

CASE

A 54-year-old male presented with headaches and dizziness and was suspected of having had a stroke. Further investigations revealed that the patient had primary hyperparathyroidism, with a corrected calcium level of 3.56 mmol/L (normal range 2.1-2.55), a phosphate level of 0.93 mmol/L (normal range 0.74–1.52), and an iPTH level of 148.28 pmol/L (normal range 1.59–7.24). Thyroid ultrasound detected a bilateral enlarged inferior parathyroid gland measuring 1.5 x 1.3 cm and 1.6 x 1.3 cm, and a SESTAMIBI scan confirmed the presence of parathyroid hormone hypersecretion. He underwent a successful bilateral inferior parathyroidectomy, and a histopathological examination revealed parathyroid carcinoma. He remained normocalcaemic, but his iPTH levels increased with time, from 12.05 pmol/L to 30.23 pmol/L. A subsequent ultrasound of the thyroid showed a tiny extra-thyroidal lesion adjacent to the left internal jugular vein, and a repeat SESTAMIBI scan revealed concordant parathyroid hypersecretion over the left superior thyroid gland. However, a neck CT scan failed to locate the lesion. Parathyroid carcinoma is commonly related to Multiple Endocrine Neoplasia (MEN Syndrome) and familial isolated hyperparathyroidism. 4D CT, MRI, and hybrids of SESTAMIBI and CT/MRI enable more precise localization of ectopic disease glands.

CONCLUSION

It is important to have lifetime surveillance for parathyroid carcinoma survivors, as the recurrence rate is high despite surgery, with a mean survival of 6-7 years.

EP_A088

25 HYDROXY-VITAMIN-D LEVEL INVESTIGATION AND MANAGEMENT: CLINICAL AUDIT IN A TERTIARY HOSPITAL IN CENTRAL PAHANG, MALAYSIA

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INTRODUCTION/BACKGROUND

The prevalence of vitamin D deficiency in Malaysia in different populations has been quoted to be between 20 to 90%. Adequate vitamin D in food sources, sun exposure, or supplementation are preventative measures for vitamin D deficiency. Vitamin D level screening is limited by resources in government hospitals and targeted screening in high-risk patients are performed. This study was conducted to ascertain the practice of 25-hydroxyvitamin D screening and management of vitamin D deficiency.

METHODOLOGY
All patients with 25-hydroxyvitaminD levels done at the Hospital Sultan Haji Ahmad Shah, Temerloh in 2023 were included. Patient demographic data, clinical profile, 25-hydroxyvitamin D and vitamin D deficiency management were assessed through electronic medical records.

RESULTS
A total of 126 samples of 25-hydroxyvitamin D were done in 2023 for 100 patients. Majority were paediatric patients (65%) and the rest were adult patients (35%). Mean 25-hydroxyvitamin D levels for adult and paediatric patients were 44.5 nmol/L and 99.8 nmol/L respectively. Most of the investigations were for screening of 25-hydroxyvitamin D status (68.6%) while 21.4% were for monitoring of 25-hydroxyvitamin D levels for patients who are already undergoing vitamin D treatment.

Common indications for 25-hydroxyvitamin D in paediatric patients included renal disease (24.6%), prematurity (16.9%), hypocalcaemia (9.2%) and high ALP (7.6%). In adult patients, common indications included renal disease (20%), hypocalcaemia (11.4%), hypercalcemia (7.6%) and osteoporosis (7.6%).

Vitamin D deficiency was present in 43.1% (n = 28) of paediatric patients and 54.3% (n = 19) of adult patients. Among paediatric patients with vitamin D deficiency, 67.9% (n = 19) were treated with inactivated vitamin D while 25% (n = 7) did not receive any treatment. Among adult patients with vitamin D deficiency, 15.7% (n = 3) were treated with inactivated vitamin D and 42.1% (n = 8) did not receive any treatment.

CONCLUSION
There was a huge discrepancy in the number of 25-hydroxyvitamin D samples sent in adult and paediatric patients, which may indicate lower awareness of vitamin D screening among adult patients. The high proportion of adult patients with vitamin D deficiency who are not optimally managed with vitamin D supplementation reflect the need to standardize and monitor vitamin D treatment in the hospital.

EP_A089

TRANSIENT OSTEOPOROSIS SECONDARY TO TENOFOVIR

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INTRODUCTION/BACKGROUND
Tenofovir is the first-line antiviral therapy for chronic hepatitis B, however, long-term use may induce osteoporosis. This is a case of a patient who developed transient osteoporosis after chronic use of tenofovir.

CASE
A 57-year-old male with chronic Hepatitis B on tenofovir, presented with a 2-year history of progressive limb weakness, myalgia, and weight loss of 6 kg. He sustained a low impact fracture of his left ankle 2014 and incidentally noted a right pelvic fracture through an MRI of the pelvis in 2017. His blood parameters were normal including serum calcium, phosphate, vitamin D and parathyroid hormone levels. First BMD examination in 2018 showed severe osteoporosis with a T-score of – 4.9 and -4.7 for the distal one-third of the left forearm and spine respectively. Serial BMD examination one year later, showed the persistence of severe osteoporosis with a T-score of -3.4, -3.3 at the hip and spine respectively. Moreover, there was a worsening of T-score to -6.0 at the distal one-third of the forearm. He was then initiated on oral bisphosphonate. Additional proximal myopathy workup including FDG-PET scan, CECT Thorax, abdomen and pelvis and muscle biopsy were all normal. EMG showed diffuse neurogenic with secondary myogenic changes, suggestive of a metabolic aetiology. Thus, tenofovir was switched to entecavir and lamivudine after all other metabolic causes were ruled out. Osteoporosis treatment with oral bisphosphonate and vitamin D supplements was continued. The latest BMD examination in 2024 showed a markedly improved T-score and resolution of his osteoporosis.

CONCLUSION
Tenofovir may lead to osteoporosis development through directly altering osteoclasts and/or osteoblasts activity. Furthermore, literature showed it can also affect the proximal renal tubules and vitamin D metabolism. Hence, close monitoring of tenofovir plasma concentrations coupled with renal and bone function is essential. Early detection, diagnosis, and treatment of osteoporosis induced by these drugs should be emphasized.

EP_A090

INVISIBLE PARATHYROID ADENOMA WITH REFRACTORY HYPERCALCEMIA IN PREGNANCY

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INTRODUCTION/BACKGROUND
Hypercalcemia in pregnancy is an uncommon event that can have serious consequences on both the mother and foetus. We describe a patient with refractory parathyroid hormone-dependent hypercalcemia in pregnancy.

CASE
A 39-year-old female presented with asymptomatic moderate hypercalcemia (corrected calcium: 3.35 mmol/L and iPTH: 24.1 pmol/L) during the third trimester of her first pregnancy in 2021, requiring subcutaneous calcitonin before elective Caesarean section. Post-delivery, her serum calcium level remained between 2.7-2.9 mmol/L, with PTH level of 10.6 pmol/L. She underwent several imaging investigations including neck ultrasound, sestamibi parathyroid scan and computed tomography of the neck but all failed to localize a parathyroid lesion. The urine calcium creatinine ratio was inconclusive.

She conceived again in 2023 while still being investigated for primary hyperparathyroidism. During this second pregnancy, she had recurrent admissions for asymptomatic hypercalcemia, with the highest calcium level of 3.3 mmol/L, iPTH 14.8 pmol/L and PTH-related peptide <0.4 pmol/L. Neck ultrasound showed only bilateral thyroid nodules with no lesions suggestive of parathyroid adenoma. Ultrasound of the kidneys showed bilateral nephrocalcinosis. She refused to undergo exploration parathyroidectomy. Due to refractory hypercalcemia ranging from 2.9-3.3 mmol/L despite intravenous hyperhydration, she was started on cinacalcet until delivery. The delivery via Caesarean section was uneventful, and both patient and baby were well. Four days after delivery, she underwent sestamibi and CT neck that showed a small hyperfunctioning parathyroid adenoma inferior to the right thyroid lobe. Interestingly, her calcium levels decreased to 2.8 mmol/L post-delivery. She was then referred to the surgical team for parathyroidectomy.

CONCLUSION
Investigation and management of PTH-dependent hypercalcemia in pregnancy is challenging due to limited options in imaging modalities and medical interventions.

EP_A091

A CHALLENGING CASE OF PERSISTENT HYPERCALCEMIA POST TOTAL PARATHYROIDECTOMY IN A DIALYSIS-DEPENDENT PATIENT

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INTRODUCTION/BACKGROUND
Tertiary hyperparathyroidism is a frequently encountered complication of advanced chronic kidney disease, characterized by an increase in parathyroid hormone (PTH) levels due to disturbances in calcium, phosphate, and vitamin D homeostasis. In most cases, total parathyroidectomy results in the resolution of hypercalcemia. However, primary hyperparathyroidism occurring in the context of tertiary hyperparathyroidism is a rare occurrence. We present a challenging case of persistent hypercalcemia in a dialysis-dependent patient following total parathyroidectomy.

CASE
A 41-year-old female was diagnosed with dialysis-dependent end-stage kidney disease at the age of 27 due to hypertension in the young. In 2014, she was diagnosed with tertiary hyperparathyroidism and underwent two staged operations for total parathyroidectomy in 2014 and 2020. Despite the procedure, she experienced persistent hypercalcemia, with serum calcium levels reaching 2.8-2.86 mmol/L (2.1-2.55) and phosphate levels of 1.49- 2.18 mmol/L (0.74-1.52). Her serum iPTH was 71.4 pmol/L (1.6-6.9) and 25 (OH) D was 30 nmol/L. A DEXA scan showed severe osteoporosis, with a T-score of -3.4 and Z-score of -2.6 in the lumbar spine, and a T-score of -4.6 and Z-score of -4.2 in the 1/3 radius.

Sestamibi imaging performed in December 2023 demonstrated the presence of sestamibi- avid ectopic parathyroid tissue within the inferior pole of the left thyroid lobe and superior mediastinum, measuring 1.2 x 1.1 cm at the level T5 vertebra. Correlating with features of post-total parathyroidectomy for tertiary hyperparathyroidism, this may represent an ectopic parathyroid adenoma. While waiting for definitive surgical management, the patient was administered subcutaneous Denosumab 60 mg every 6 months with calcium and vitamin D supplementation.

CONCLUSION
The diagnosis and management of primary hyperparathyroidism in patients with advanced chronic kidney disease presents a distinct set of challenges. In the absence of

a clearly identifiable parathyroid adenoma, it is imperative to employ a combination of imaging techniques to identify any possible ectopic focus, which yields the maximum benefit. Following localization, surgical resection continues to be the preferred mode of treatment for achieving a permanent cure.

EP_A092

COMPLEX SCENARIO OF MEN 1 WITH ECTOPIC PARATHYROID GLAND: A CASE REPORT

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INTRODUCTION/BACKGROUND
In the context of Multiple Endocrine Neoplasm 1 (MEN1), primary hyperparathyroidism (PHPT) is the most prevalent form of endocrinopathy and is often the earliest endocrine manifestation among patients. It represents 2–4% of all forms of PHPT. Ectopic parathyroid adenomas (EPTA) account for a significant proportion, approximately 22% of PHPT cases. To mitigate the adverse effects of PHPT in MEN1 patients, the optimal course of treatment is parathyroidectomy. We present a complex case of MEN1 that involves an ectopic parathyroid gland.

CASE
A 54-year-old female presented with symptomatic hypercalcemia with a serum calcium of 3.63 mmol/L (2.1-2.55) along with multiple duodenal ulcers and a Hb of 8.7g/dL (12-15) in 2008. Clinical diagnosis of Multiple Endocrine Neoplasia 1 was made, as validated by primary hyperparathyroidism, microprolactinoma and non-functioning pancreatic neuroendocrine tumour grade 1. She underwent total parathyroidectomy in July 2008 with a right inferior auto-transplantation into the sternocleidomastoid muscle. Histopathological analysis confirmed parathyroid hyperplasia in all 4 glands. Ten years later, she exhibited an increasing trend of serum calcium 2.57-2.63 mmol/L and iPTH (7.05->12.89->14.4 pmol/L) (1.58-6). Neck ultrasonography revealed a well-defined elongated hypoechoic structure within the right sternocleidomastoid muscle measuring 0.2 x 0.4 x 0.9 cm (AP x W x CC). Parathyroid scintigraphy Tc99M Sestamibi with SPECT-CT demonstrated the presence of an ectopic parathyroid adenoma measuring 0.7 x 0.9 cm at the right upper paratracheal/suprasternal region. Subsequently, she underwent exploratory parathyroidectomy with the removal of the right auto-transplant parathyroid gland and right thymus. Histopathological analysis was consistent with parathyroid hyperplasia and ectopic parathyroid accordingly. Postoperatively she remained hypercalcaemic 2.7 mmol/L with non-suppressible iPTH 27.46 pmol/L. Levels of 25-OH (D) were insufficient at 40.72 nmol/L. Further localization studies were contemplated. However, 4D CT assessment was not done due to her deteriorating renal function. She was given oral cholecalciferol 1000 IU daily and cabergoline 1 mg daily.

CONCLUSION
Despite significant progress in imaging technologies and surgical techniques, the management of EPTA remains a challenging task in clinical practice. Specialized multidisciplinary input is crucial in managing such cases.

EP_A093

A MORE SINISTER CAUSE OF LOWER BACK PAIN IN THE THIRD TRIMESTER: A CASE REPORT OF PREGNANCY AND LACTATION-ASSOCIATED OSTEOPOROSIS

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INTRODUCTION/BACKGROUND
Pregnancy and lactation-associated osteoporosis (PLO) is a rare but painful condition that tends to occur during the third trimester or postpartum period, with an incidence of 0.4 cases/100,000 women and 70% of those affected are primiparous. The main symptom is severe lower back pain as this condition often causes vertebral fractures which can be multiple.

CASE
We present a case of a 28-year-old female with an underlying right coronary artery fistula and endometriosis. She is para 1 and delivered her child in December 2022. During this pregnancy, she had a history of severe back pain during the third trimester. There was no history of falls or any neurological deficits. She gave a history of coccyx fracture following a fall eight years ago but recovered uneventfully. She breastfed her baby for five months post-partum and her back pain persisted during this period which prompted further investigations for her. She did not consume any steroids and there were no signs and symptoms to suggest Cushing's Syndrome.

Initial biochemical results were all within normal range: calcium 2.41 mmol/L, phosphate 1.0 mmol/L, ALP 84 U/L, TSH 1.83 m IU/L, FT4 14.8 pmol/L and i-PTH 4.8 pmol/L. Overnight dexamethasone suppression test was appropriately suppressed (15 nmol/L). Her vitamin D level was insufficient at 51 nmol/L. Radiography confirmed T10 vertebral compression fracture and her DEXA scan revealed that she is osteoporotic with spine Z-score -3.2, and femur Z-score -3.5.

She was managed with oral cholecalciferol 1000 units daily and calcium carbonate 500 mg twice daily as she preferred to not use any anti-resorptive agent. Her back pain improved after cessation of lactation and with analgesics.

CONCLUSION

PLO is a rare condition and should be suspected in pregnant women who complain of back pain to ensure early diagnosis and intervention. Secondary causes of osteoporosis need to be ruled out as well.

EP_A094

SEVERE HYPERCALCEMIA UNMASKS A CAMOUFLAGED PARATHYROID CARCINOMA

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INTRODUCTION/BACKGROUND

Parathyroid carcinoma is an exceedingly rare endocrine malignancy and an uncommon cause of primary hyperparathyroidism. We report a case of severe hyperparathyroidism which required urgent surgery and unveiled a parathyroid carcinoma.

CASE

A 31-year-old Malay female, who initially presented with severe epigastric pain and vomiting, was treated for acute pancreatitis in the surgical ward. There was no history of alcohol consumption. Biochemistry results showed acute renal impairment, hypercalcemia (corrected Ca2+ 4.06 mmol/L), hypophosphatemia (PO43- 0.38 mmol/L), elevated serum amylase (370 U/L) and urine diastase (663 U/L). However, no sonographic evidence of cholelithiasis was found. She was referred to the medical team for further investigations which revealed primary hyperparathyroidism with vitamin D deficiency (iPTH 705 pg/ml, 25 (OH) vitamin D 25 nmol/L, alkaline phosphatase 543 U/L). Clinically, no neck mass was observed but the neck detected a TIRADS 5 nodule in the right thyroid. Given the discordant clinical and sonographic findings, Tc99m Sestamibi SPECT/CT was arranged but she defaulted. Subsequently, she was admitted for severe hypercalcemia manifesting as severe bone pain and requiring saline-forced diuresis and intravenous zolendronic acid. Tc99m Sestamibi SPECT/CT scan demonstrated a parathyroid adenoma measuring 2.2 x 2.6 x 2.9 cm posterior to the right thyroid gland. Further imaging work-up showed bilateral medullary nephrocalcinosis and osteopenia. She underwent urgent right hemithyroidectomy, right superior parathyroidectomy and central neck dissection successfully. Postoperatively she developed hungry bone syndrome which required intravenous calcium infusion together with high dose activated vitamin D and calcium supplements. Histopathological report confirmed the diagnosis of parathyroid carcinoma with lymphovascular and capsular invasion. Postoperatively she was scheduled for PET-CT scan but she defaulted the follow-up. She is currently pregnant.

CONCLUSION

Parathyroid carcinoma is an indolent but progressive disease. Surgery is the mainstay of treatment. Early detection with attempts to remove local recurrence and distant metastasis can provide good short- and long-term control.

EP_A095

SEVERE HUNGRY BONE SYNDROME, COULD WE HAVE PROGNOSTICATED IT BETTER? A CASE SERIES

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INTRODUCTION/BACKGROUND

Hungry bone syndrome (HBS) is characterized by serum calcium less than 2.1 mmol/L and/or prolonged hypocalcaemia more than four days following parathyroidectomy or thyroidectomy. Here, we report two cases of HBS following parathyroidectomy for primary hyperparathyroidism (PHPT).

CASE 1

A 17-year-old female was diagnosed with PHPT when she presented with bilateral femoral neck and humeral fractures. Her serum calcium was 3.69 (2.2-2.65 mmol/L),

serum phosphate 0.92 (0.81- 1.45 mmol/L), and serum intact parathyroid hormone (iPTH) 1187.2 (14.9-56.9 pg/ml). There was deficient 25-hydroxy vitamin D at 18.9 (<25 nmol/L), ALP was elevated at 1413 (47-162 U/L) and renal function was normal. Ultrasound (US) of the parathyroid showed a lobulated hypoechoic lesion at the posterior right thyroid, measuring 1.6 x 2.3 x 3.3 cm. Preoperatively she received IV bisphosphonates and subcutaneous calcitonin. Haemodialysis was initiated prior to surgery for hypercalcaemic crises. Postoperatively, she required calcium gluconate infusion for 2 weeks.

CASE 2

A 53-year-old female had an incidental finding of hypercalcemia, which was subsequently confirmed to be PHPT. Serum calcium range was 2.92-3.02 mmol/L and phosphate 0.6-0.72 mmol/L. Serum iPTH 456.9 pg/ml, 25-Hydroxy vitamin D 58.97 (25-75 nmol/L), ALP 372 U/L, and renal profile was normal. Parathyroid ultrasound showed a left extrathyroidal lesion measuring 0.6 x 0.7 x 0.8 cm, concordant with her sestamibi scan. DEXA scan showed osteoporosis. Preoperatively, she received IV bisphosphonate and cholecalciferol. She was discharged on day 3 with serum calcium of 2.05 mmol/L. However, she was re-admitted a day later for HBS (calcium 1.67 mmol/L) requiring calcium gluconate infusion for 3 days.

RESULTS

These cases illustrated severe HBS following parathyroidectomy. HBS may ensue in patients with elevated PTH and ALP, radiological evidence of bone diseases, large volume or weight of parathyroid gland, higher preoperative calcium, and vitamin D deficiency.

CONCLUSION

Rigorous proactive measures and timely management of HBS may avert the detrimental effects of hypocalcaemia. Identifying the likelihood that patients may develop HBS before surgery is of utmost importance for successful management.

EP_A096

SPONTANEOUS RESOLUTION OF PRIMARY HYPERPARATHYROIDISM WITH PARATHYROID APOPLEXY

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INTRODUCTION/BACKGROUND

Primary hyperparathyroidism (PHPT) is characterized by autonomous parathyroid hormone (PTH) secretion from one or more of the 4 parathyroid glands. Most cases are due to a single parathyroid adenoma with parathyroidectomy offering a potential cure, however, spontaneous resolution of PHPT does occur following parathyroid apoplexy but has been sparsely reported in the literature.

CASE

We present a 27-year-old Malay male with no known comorbidities, who first presented in May 2023 with symptomatic hypercalcemia [Corrected Calcium 4.26 mmol/L (N 2.2-2.6); phosphate of 1.67 mmol/L (N 0.87-1.45); PTH of 159.7 pmol/L (N 1.6-6.9)] and was diagnosed with PHPT secondary to left inferior parathyroid adenoma. He was also diagnosed with polycystic kidney disease considering a positive family history, renal impairment, and the presence of renal and liver cysts on imaging. With saline diuresis and a dose of 30 mg intravenous pamidronate, his calcium level improved to 3.19 mmol/L. Unfortunately, he was subsequently lost to follow-up.

He presented 4 months later with acute pain and increased neck swelling associated with numbness over the extremities and perioral region. Chvostek's sign was positive with a palpable anterior neck swelling measuring 2 x 2 cm. There was no palpable cervical lymphadenopathy. Biochemical analysis revealed severe hypocalcaemia [Corrected Calcium 1.69 mmol/L (N 2.2-2.6); Phosphate 0.51 mmol/L (N 0.87-1.45)] with a marked reduction in PTH level to 39 pmol/L (N 1.6-6.9). Intravenous calcium, oral calcium carbonate and calcitriol were simultaneously initiated. Repeated 99 mTc sestamibi imaging showed negative uptake, confirming the diagnosis of parathyroid apoplexy. He was maintained on calcium carbonate 1 g twice daily and calcitriol 0.5 mcg twice daily with his latest corrected calcium level at 2.24 mmol/L.

CONCLUSION
Parathyroid apoplexy resulting in resolution of PHPT is uncommon and the best management approach (surgery or conservative) remains uncertain. As recurrence have been reported, long term monitoring is essential for patients managed conservatively.

EP_A097

A CASE OF PRIMARY HYPERPARATHYROIDISM COEXISTING WITH MONOCLONAL GAMMOPATHY OF UNDETERMINED SIGNIFICANCE (MGUS)

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INTRODUCTION/BACKGROUND
Hypercalcemia is a relatively common clinical problem in hospitalized patients. Primary hyperparathyroidism and plasma cell dyscrasias such as multiple myeloma and monoclonal gammopathy of undetermined significance (MGUS) are known to be the most common causes of hypercalcemia. Although the occurrence of these disorders in one patient has been reported previously, it is still believed to be a rare phenomenon. We report a case of hypercalcemia, resulting from coexistent primary hyperparathyroidism and MGUS.

CASE
A 45-year-old female with no previous medical illness was admitted for symptomatic hypercalcemia. A review of symptoms during admission was significant for constipation, nausea, anorexia, polyuria and thirst. Initial blood investigations showed hypercalcemia (corrected calcium 4.43 mmol/L), renal impairment (creatinine 154 mmol/L) and anaemia (haemoglobin 9 g/dL). Hypercalcemia was managed with intravenous hydration, bisphosphonates and furosemide. Subsequent tests include elevated intact parathyroid hormone (820.4 pg/ml) and an elevated 24-hour urinary calcium/creatinine clearance ratio of 0.07, suggestive of primary hyperparathyroidism. This was further supported by the finding of a right parathyroid adenoma on neck ultrasound.

Due to an abnormal albumin/globulin ratio of 0.89, serum protein electrophoresis was also done which revealed IgG lambda paraproteinemia. The skeletal survey and bone scan were normal. Bone marrow and trephine biopsy showed the presence of clonal plasma cells at less than 10 percent, which confirmed the diagnosis of MGUS. The patient is currently under multidisciplinary care in endocrinology and haematology subspecialties. Sestamibi parathyroid scan has been arranged for preoperative localization. She is also being monitored closely for progression to multiple myeloma.

CONCLUSION
This case gives significant insights into potential concomitant causes of hypercalcemia. A high index of suspicion and a systematic approach to performing relevant screening tests are essential, as earlier diagnosis leads to improved clinical outcomes.

EP_A098

REVEALING THE HIDDEN MASK: A CASE ON PRIMARY HYPERPARATHYROIDISM MIMICKING PREGNANCY SYMPTOMS

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INTRODUCTION/BACKGROUND
Parathyroid adenoma in pregnancy is uncommon, posing diagnostic and treatment challenges. We report a case of primary hyperparathyroidism (PHPT) due to a parathyroid adenoma successfully treated with surgery during pregnancy.

CASE
A 20-year-old female, gravida 2 para 0+1, presented with vomiting since the fourth week of pregnancy. She also reported experiencing left shoulder pain, abdominal discomfort, polyuria, nocturia, and constipation. Initially diagnosed with hyperemesis gravidarum, further investigation revealed hypercalcemia with a serum calcium level of 3.66 mmol/L, phosphate level of 0.7 mmol/L, and markedly elevated intact parathyroid hormone (iPTH) at 15.53 pmol/L (normal value: 1.6-6.9 pmol/L). Despite attempts to lower calcium levels through hydration and diuresis with furosemide, her serum calcium remained elevated at 3.14 mmol/L. She received six doses of subcutaneous calcitonin due to persistent hypercalcemia, resulting in a reduction of calcium to 2.83 mmol/L. Ultrasonography of the neck detected a left superior parathyroid adenoma. At 25 weeks of gestation, she underwent a successful left superior parathyroidectomy with intraoperative parathyroid hormone monitoring, resulting in positive outcomes for both mother and baby.

We described a case of PHPT secondary to a parathyroid adenoma presenting with typical pregnancy symptoms. A high index of suspicion warrants screening for serum calcium levels in hyperemesis gravidarum if symptoms persist beyond the first trimester or are severe, and if symptoms suggestive of hypercalcemia are present. Early detection is crucial for the timely management and improvement of maternal and foetal outcomes. Maternal complications can be as high as 67% including nephrolithiasis, pancreatitis, hyperemesis gravidarum, muscle weakness, confusion, hypercalcaemic crisis, and can also lead to miscarriages and pre-eclampsia.

CONCLUSION

Recognizing primary hyperparathyroidism can be challenging as symptoms may overlap with typical pregnancy. Surgery is the sole curative measure for primary hyperparathyroidism, well-tolerated during pregnancy with minimal adverse effects.

EP_A099

THE MISSING PIECE OF ADULT HYPOPHOSPHATEMIC RICKETS PUZZLE: A CASE REPORT OF SUSPECTED X-LINKED HYPOPHOSPHATEMIA (XLH) WITH RECURRENT DENTAL ABSCESS

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INTRODUCTION/BACKGROUND

X-linked Hypophosphatemia (XLH) is associated with primary musculoskeletal complications and can present with recurrent dental-related complications.

CASE

We present a case of poorly treated XLH who not only presented with typical bone deformities but also with overlooked dental complications. A 24-year-old female presented to us with short stature, severe bowing of the legs with leg pain, frontal bossing, and bilateral genu varus with recurrent dental-related problems requiring multiple visits to the dentist, causing distress as she is losing her teeth. She had undergone an osteotomy four times on her left femur. Family history was insignificant. Assessment by the dentist reveals multiple cyst and abscess formations at both the upper and lower jaws and malocclusion.

She has normal calcium (2.18 mmol/L), low phosphate (0.47 mmol/L) with low Renal-Tubular-Reabsorption-of-Phosphate (TMP/GFR) [0.67 mmol/L] and vitamin D deficiency (35 nmol/L). No evidence of other renal electrolytes or acid-base loss was noted. Her intact PTH and ALP were normal. Serum calcium, phosphate, and vitamin D levels improved with Sandoz phosphate 500 mg given twice daily, alphacalcidol 1 mg once daily, and calcium carbonate 500 mg twice daily. Her latest serum calcium was 2.39 mmol/L, serum phosphate increased to 0.71 mmol/L, vitamin D level likewise improved to 88.9 nmol/L, and iPTH was normal 27.7 pg/ml (14.9–56.9). Ultrasound of the kidneys did not show any medullary nephrocalcinosis. No confirmatory genetic tests to look at PHEX mutation gene were done due to financial constraints.

CONCLUSION

Adult XLH can present with only dental-related issues and are often overlooked. It can lead to premature tooth loss, resulting in adverse practical, cosmetic, and social sequelae. Hence, dental-related complaints should always be addressed and treated. Studies have shown that dental issues are milder among people who underwent conventional therapy compared to those who did not receive continuous treatment. Supplementation with phosphorus and a vitamin D analogue enhances the mineralization of dentin and decreases the frequency of dental abscesses.

EP_A100

FAILED LOCALIZATION IN PRIMARY HYPERPARATHYROIDISM DUE TO POLYGLANDULAR DISEASE

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INTRODUCTION/BACKGROUND

Primary hyperparathyroidism (PHPT) is characterized by hypercalcemia driven by excess secretion of parathyroid hormone (PTH). While solitary hyperfunctioning parathyroid adenomas account for up to 90% of cases, localizing hyperfunctioning glands in multiglandular disease (MGD) is more challenging.

CASE

A 46-year-old female presented with chronic vomiting and significant weight loss, leading to a diagnosis of primary hyperparathyroidism with secondary osteoporosis and severe vitamin D deficiency. She had five admissions over 10 months for severe hypercalcemia, (3.6- 4.4 mmol/L) requiring intravenous bisphosphonates.

She underwent multiple imaging studies for parathyroid adenoma localization, including parathyroid ultrasound and subsequent sestamibi scan, which showed no evidence of hyperfunctioning parathyroid tissue. A computed tomography scan using a parathyroid protocol did not demonstrate any parathyroid adenoma. After multiple hypercalcaemic crises requiring IV bisphosphonates, oral cinacalcet 50 mg twice daily was initiated to control her hypercalcemia. However, her calcium levels remained elevated, leading to the decision to do bilateral neck exploration (BNE) due to failed multimodal localization studies. Calcitonin (total dosing of 300 units) was administered preoperatively to optimize calcium levels. Intraoperatively, the right superior and left inferior parathyroid glands were removed, preserving only the right inferior parathyroid gland. The left superior parathyroid gland was not visualized. Intraoperative iPTH was not available in our setting. Histopathological examination revealed a right superior parathyroid adenoma and left inferior gland hyperplasia. Postoperatively, she transiently required calcium infusion and was discharged with oral calcium and vitamin D supplementation. Preoperatively, serum intact PTH was 167 pg/mL (NR 14.9-56.9), which decreased to 16.8 pg/mL one month postoperatively, indicating successful removal of the target adenoma.

CONCLUSION

In cases of failed localization in PHPT, recognizing MGD is crucial. BNE may yield higher cure rates compared to minimally invasive parathyroidectomy, which require two concordant imaging studies. Preoperative calcium optimization is essential for minimizing intraoperative complications and the risk of postoperative hungry bone syndrome.

EP_A101

CLINICAL AUDIT ON BONE MINERAL DENSITY (BMD) CONDUCTED IN SERDANG HOSPITAL IN THE YEAR 2023

https://doi.org/10.15605/jafes.039.S1.112

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INTRODUCTION/BACKGROUND

BMD measurement using dual-energy x-ray absorptiometry (DXA) remains the gold standard for the diagnosis of osteoporosis. This non-invasive, radiographic test is a convenient tool that guides physicians when to initiate osteoporotic therapy, especially among the elderly population without fragility fracture.

METHODOLOGY

We conducted a cross-sectional audit on all the BMDs conducted in the year 2023 at HSIS, to study the bio-demographics of the patients, severity of BMDs and adequacy of treatment initiated. In this clinical audit, all BMDs were extracted from the electronic medical record (EMR). Medications and bio-demographics of the patients were extracted from the EMR to calculate the FRAX score.

RESULTS

A total of 473 BMD reports were interpreted. The mean age of the patients was 65.5 (13.4) years while the mean BMI was 25.3 (5.4) kg/m^2. About 89% of patients were female and 11% were male. According to ethnicity, the majority were Chinese (47.4%), followed by Malay (34.7%) and Indian (17.3%). A total of 168 (35.5%) patients who had BMD had a fragility fracture but only 76.2% were treated with anti-osteoporotic agents. Majority of the requests for BMD were from the Orthopaedics Department (57.7%) followed by Rheumatology Unit (23.5%) and Endocrinology Unit (3.6%). About 64.1% (n=303) of patients qualify for anti-osteoporotic treatment due to a fragility fracture, a T score ≤-2.5 or osteopenia with FRAX score warranting treatment. However, only 198 (65.3%) were started on anti-osteoporotic treatment. In 165 patients with severe osteoporosis (T score ≤-3.0), only 127 (77%) were started on treatment, and only 2 of them (1.2%) were started on anabolic agents.

CONCLUSION

Lack of screening, awareness and inadequate funding for anti-osteoporotic treatment were the main contributing factors for delay in treatment or initiation of less potent agents among patients with osteoporosis.

EP_A102

MANAGING A YOUNG MALE WITH PRIMARY HYPERPARATHYROIDISM PRESENTING WITH SEVERE HYPERCALCAEMIA IN A DISTRICT HOSPITAL: A CASE REPORT

https://doi.org/10.15605/jafes.039.S1.113

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INTRODUCTION/BACKGROUND
Primary hyperparathyroidism (PHPT) is characterized by hypercalcemia with an inappropriately elevated intact parathyroid hormone level (iPTH). Clinical presentation may range from asymptomatic to severe symptomatic hypercalcemia with complications such as osteoporosis, fracture and renal stones.

CASE
A 31-year-old male was admitted following a motor vehicular accident where he sustained a fracture over the midshaft of the left ulnar and radial bones. On further history, he had experienced right hip and bilateral knee pain in the past 3 months prior to the accident. During this admission, his serum calcium was elevated at 4 mmol/L with a low phosphate level at 0.65 mmol/L. His PTH level was markedly elevated (858 pg/ml). Renal profile, vitamin D level and liver function test were normal. X-ray of bilateral knee joint and hip was suggestive of generalized osteopenia. He required several admissions for severe refractory hypercalcemia managed with bisphosphonate and hyperhydration. Ultrasound of the parathyroid revealed a homogenous hypoechoic lesion at the right posterior thyroid lobe measuring 0.9 x 2.1 x 2.8 cm. Preoperatively, he was given hyperhydration along with furosemide and cinacalcet to achieve a target calcium level of 3 mmol/L. He successfully underwent a right inferior parathyroidectomy. Histopathological examination revealed a right parathyroid adenoma. Post-operatively, the patient required calcium infusion and was able to be discharged with a calcium supplement and active vitamin D.

Solitary parathyroid adenoma accounts for the majority of primary hyperparathyroidism followed by multiple gland hyperplasia and less commonly, parathyroid carcinoma. The definitive treatment is parathyroidectomy.

Our patient fulfilled the criteria for surgery and successfully underwent total parathyroidectomy at our district hospital by a visiting endocrine surgeon. Intra-operative PTH monitoring was not done as it was not available at our centre. Genetic testing should be considered in this case because PHPT was diagnosed below the age of 40.

CONCLUSION
Primary hyperparathyroidism presenting with severe refractory hypercalcemia warrants immediate treatment in order to prevent complications from hypercalcemia. In addition to hyperhydration and bisphosphonate, cinacalcet is another option to reduce calcium levels in PHPT.

EP_A103

RARE PRESENTATION OF OSTEOPOROSIS IN PREGNANCY: A CASE REPORT AND LITERATURE REVIEW

https://doi.org/10.15605/jafes.039.S1.114

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INTRODUCTION/BACKGROUND
Osteoporosis in pregnancy is a rare condition, with a reported occurrence of only 1.2% among individuals aged 20 to 40 years. Despite its infrequency, the condition carries significant potential for disability and adverse outcomes.

CASE
We present a 29-year-old female at 33 weeks of gestation, with a background history of type 2 diabetes mellitus with retinopathy. She, came in with a 5-week history of right hip pain. Subsequently, she fell from a standing position rendering her immobilized. A pelvic x-ray was performed, revealing a right femoral neck fracture. The patient had no history of steroid use. Initial investigations including thyroid function test, calcium and phosphate levels were within normal limits. However, serum parathyroid hormone was low at 0.95 pmol/L (normal range: 1.59-7.24) and vitamin D was low at 35 nmol/L (sufficiency >75).

Following a Caesarean section, she underwent screw fixation of the neck of the right femur and was advised to refrain from weight-bearing on the affected limb for 3 weeks. Histopathological examination of the bone fragment revealed scanty bony trabeculae without atypical cells. A dual X-ray absorptiometry (DXA) scan revealed a normal bone mineral density (BMD) of the spine with Z-score of 0.1. The total left hip BMD was 0.673 g/cm^3 with a Z-score of -1.7 and T score of -2.0.

The fracture was attributed to a combination of factors, including vitamin D deficiency, hormonal changes during pregnancy and diabetes. The patient was started on calcium

and vitamin D supplements. However, subsequent follow-ups indicated non-union of the fracture with early avascular necrosis, necessitating referral to the arthroplasty team for further management.

CONCLUSION

This case underscores the importance of considering osteoporosis during pregnancy, especially among patients with comorbidities such as diabetes mellitus. Early recognition and adequate supplementations are paramount to mitigate complication of fracture.

EP_A104

THE DOUBLE-EDGED SWORD - SEVERE HYPOPHOSPHATEMIA POST INTRAVENOUS BISPHOSPHANATE FOR SEVERE REFRACTORY HYPERTHYROID-INDUCED HYPERCALCEMIA: A CASE REPORT

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INTRODUCTION/BACKGROUND

Severe hypophosphatemia, defined as serum phosphate levels of <1 mg/dl (0.32 mmol/L), after intravenous bisphosphonate is a rare occurrence. It can lead to rhabdomyolysis, respiratory failure, convulsions and arrhythmias if not detected and treated early. Few reported cases of bisphosphonate-induced hypophosphatemia are related to malignancy-induced hypercalcemia or osteoporosis treatment. We present a case of severe hypophosphatemia post intravenous zoledronic acid for hyperthyroid-induced refractory severe hypercalcemia.

CASE

A 62-year-old female presented with thyrotoxicosis (weight loss, palpitation, heat intolerance) for 5 months. On physical examination, she had hand tremors, proximal myopathy and tachycardia with multinodular goitre. Other systemic examinations were normal.

She was biochemically hyperthyroid (TSH: <0.01 m IU/L, FT: 467 pmol/L). Serum PTH was suppressed (0.59 mmol/L). Initial corrected calcium was 3.5 mmol/L with phosphate of 0.72 mmol/L. Intravenous zoledronic acid 4 mg was administered as she did not respond to hyperhydration. After 48-hours, repeat serum phosphate was 0.22 mmol/L with low calculated renal-tubular-reabsorption-of-phosphate (TMP/GFR) [0.41 mmol/L] indicating renal phosphate wasting. Repeat serum PTH level was normal (4.35 mmol/L), corrected calcium was 2.01 mmol/L with low 25-OH vitamin D level (<7.5 nmol/L). A CT TAP done showed no evidence of malignancy. All her tumour markers were negative. Biopsy of thyroid nodules were negative for malignancy.

CONCLUSION

Based on previous case reports, bisphosphonate-induced hypophosphatemia is postulated to be a result of secondary hyperparathyroidism (drug-induced) causing severe hypophosphatemia through renal-phosphate wasting. One of the risk factors that can precipitate this is vitamin D deficiency. In our case, the slightly elevated PTH level post bisphosphonate coupled with reduced TMP/GFR level support the diagnosis of bisphosphonate-induced severe hypophosphatemia. Removal of the offending drug is the mainstay of treatment in drug-induced hypophosphatemia. Asymptomatic, mild to moderate hypophosphatemia is being treated with oral phosphate whereas severe symptomatic hypophosphatemia is being given intravenous phosphate. It may also be prevented with vitamin D and calcium supplements.

EP_A105

OSTEOPOROSIS TREATMENT WITH BISPHOSPHONATE THERAPY: A CLINICAL AUDIT

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INTRODUCTION/BACKGROUND

Osteoporosis is a progressive and debilitating bone disease with compromised bone strength leading to fragility fractures. Bisphosphonate is the predominant treatment for osteoporosis with an overall positive risk-benefit ratio. Pre-treatment counselling and standardized practice in prescribing bisphosphonate is important to ensure safety during treatment.

METHODOLOGY

This clinical audit was conducted to assess the practice of bisphosphonate prescription and pre-treatment counselling and assessment. All patients with osteoporosis and on active bisphosphonate treatment in Hospital Sultan Haji Ahmad, Temerloh, Central Pahang from August 2023 to January 2024 were included in the audit. Electronic medical records were assessed for demographic data, pre-treatment screening, DEXA scan investigations, pre-treatment counselling documentation, dental screening and secondary osteoporosis screening.

RESULTS
A total of 130 patients (86% females) were on active bisphosphonate treatment. Of these, 48 (36.9%) patients were from 70-79 years of age. Majority of the treatment was initiated by orthopaedic surgeons (63.8%) and endocrinologists (15.4%). Fragility fracture was the most common indication for bisphosphonate therapy in 56.9%. The most prevalent risk factors for osteoporosis were postmenopausal (80.7%), followed by prolonged steroid use (18.5%) and other endocrine disorders (11.5%). Only 35.3% (n=48) had bone mineral densitometry done prior to initiation of treatment. Less than 10% of patients had documented fracture risk assessment with FRAX. About 40% of patients had no baseline renal function prior to initiation of treatment. Referral for dental screening was not documented in 48.5% of patients. There was also a lack of counselling and documentation prior to the initiation of treatment. Majority of patients (86.9%) received vitamin D and calcium supplementation with bisphosphonate therapy.

CONCLUSION
A standardized osteoporosis pre-treatment checklist is required to ensure good and safe practice of treatment. Awareness and appropriate counselling among patients with osteoporosis on bisphosphonate treatment needs to be improved.

EP_A106

CASE REPORTS OF PRIMARY HYPERPARATHYROIDISM IN PREGNANCY

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Primary hyperparathyroidism (PHPT) during pregnancy is uncommon. Early detection is crucial due to its association with increased maternal and foetal morbidity and mortality. Diagnosis is challenging and requires high clinical suspicion due to nonspecific presentation and the overlap of symptoms of hypercalcemia with those of pregnancy. Furthermore, serum calcium is not routinely tested antenatally. The interpretation of serum calcium and parathyroid hormone levels differs significantly from that in nonpregnant patients due to physiological changes during pregnancy. Preoperative localisation and treatment options are limited due to uncertainties regarding safety in pregnancy. We present 2 cases of PHPT who underwent parathyroidectomy during pregnancy.

We retrospectively reviewed PHPT cases in Hospital Pulau Pinang from 2020 to 2023. Patients were identified from the laboratory database and clinical details were obtained from their medical records.

CASE
Two patients, with mean age of 34 years, were diagnosed with PHPT pre-pregnancy. The first patient was diagnosed with PHPT during routine blood testing for chronic myeloid leukaemia follow-up. She had left inferior parathyroidectomy and yet her post-operative serum calcium was persistently elevated. Repeated Tc99m sestamibi showed 2 foci of increased tracer uptake. During scheduled clinic visit, she informed us of her pregnancy. Exploratory parathyroidectomy was scheduled. The second patient was diagnosed with PHPT when she was admitted for acute pancreatitis. She was found to be pregnant when she was re-admitted for another episode of acute pancreatitis. Emergency parathyroidectomy was arranged due to persistent hypercalcemia despite on rehydration. Postoperatively, both were discharged with normalization of serum calcium level. However, the first patient had complete miscarriage in the second trimester; the second patient developed preeclampsia and delivered a preterm baby at 34 weeks.

CONCLUSION
Early parathyroidectomy in PHPT patients diagnosed at child-bearing age helps to prevent complications during pregnancy.

EP_A107

PITUITARY GLAND METASTASIS OF BREAST CANCER PRESENTING AS DIABETES INSIPIDUS

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INTRODUCTION/BACKGROUND
Metastasis to the pituitary gland is extremely rare and represents only 1% of pituitary tumours. The most frequently reported malignancies that metastasize to the pituitary gland are lung, renal and breast cancers.

CASE
A 49-year-old female with advanced left breast carcinoma with bone metastasis presented with a week's history of worsening back pain and bilateral lower limb weakness. On examination, vital signs were stable and neurological examination showed bilateral lower limb motor neuron lesions with muscle strength of 3/5 and loss of sensation

up to T4. During admission, she developed polyuria with hypernatremia. Laboratory examinations showed sodium of 150 mmol/L (NR: 136-145), serum osmolality of 318 mOsm/kg (NR: 275-295), urine osmolality of 142 mOsm/kg (NR: 275-295). Renal function, serum calcium, potassium and glucose were all normal. Anterior pituitary hormone panel showed panhypopituitarism with TSH: 0.24 m IU/L (NR: 0.27-4.2), FT4: 11.7 pmol/L (NR: 12-22), FSH: 7.35 IU/L (NR: 25.8-134) and LH: 5.92 IU/L (NR: 7.7-58.3). FSH and LH were low despite the patient being post-menopausal. The cortisol axis was not assessed as the patient was on dexamethasone. Cranial and pituitary MRI revealed parietal and occipital skull lesions, small right temporal and frontal brain lesions, and thickened pituitary stalk measuring 0.5 cm with a non-enhancing posterior pituitary lesion suggestive of metastasis.

Central diabetes insipidus occurs in less than 1% of patients with primary pituitary adenoma, while it is the presenting symptom in 70–80% of patients with pituitary metastasis. The radiological diagnosis is based on MRI which can highlight an iso-intense or hypo-intense mass on T1-weighted images with a high-intensity signal on T2-weighted images, a homogeneous enhancement with gadolinium and loss of the high signal pituitary signal intensity on T1-weighted images.

CONCLUSION

The prognosis for metastases to the pituitary gland is poor due to the presence of advanced neoplastic disease. Posterior pituitary lesions are rare, therefore being a red flag for metastasis.

EP_A108

NEUROSARCOIDOSIS PRESENTING WITH CENTRAL DIABETES INSIPIDUS AND SECONDARY AMENORRHEA

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INTRODUCTION/BACKGROUND

Sarcoidosis is a multisystem granulomatous inflammatory disorder with common involvement of the lungs, lymph nodes and heart. Neurosarcoidosis, especially hypothalamic-pituitary involvement, is a rare phenomenon (<10% of sarcoidosis patients). Recognizing this holds significance in guiding investigations and early commencement of treatment.

CASE

A 32-year-old nulliparous female with no prior medical illness, presented to the ophthalmology clinic with 6 months history of painless red eye, without cough or constitutional symptoms. She was managed as a case of left eye anterior uveitis. Further assessment to exclude tuberculosis revealed perihilar lobulated opacities on CXR, negative serial sputum AFB and positive tuberculin skin test (Mantoux) of 16mm. CT of the thorax showed multiple lung nodules with lymphadenopathies involving cervical, mediastinal, hilar and axillary regions. Bronchoscopy was negative for TB PCR and MTB culture had no growth. She was treated for latent PTB for 3 months. Further history revealed secondary amenorrhea and polyuria with polydipsia. Pituitary workup confirmed hypogonadotrophic hypogonadism [serum FSH: 2.9 IU/L, LH: 1.13 IU/L, estradiol: 52.8 pmol/L]. After an overnight water fasting, serum osmolality increased to 301 mOsm/kg, with hypernatremia (serum Na: 151 mmol/L) and markedly diluted urine (urine osmolality: 45 mOsm/kg). These were all suggestive of central diabetes insipidus. Other pituitary hormones were normal [serum cortisol: 573.5 nmol/L, TSH: 1.916 m IU/L and prolactin: 331.1 mU/L]. Pituitary MRI revealed an absence of a posterior pituitary bright spot, without a pituitary lesion. Since TB has been ruled out, multi-systemic sarcoidosis was considered. Serum angiotensin-converting enzyme was elevated at 72.80 U/L (NR 8-52). A respiratory consult concluded a stage 2 pulmonary sarcoidosis. She was started on oral desmopressin 60 mcg BID and hormone replacement therapy. Oral prednisolone at a dose of 0.5 mg/kg/day was initiated for sarcoidosis treatment.

CONCLUSION

Neurosarcoidosis is often diagnosed late due to a low index of suspicion. It should be included in the differential diagnosis when patient presented with hypothalamic-pituitary disorder as it is associated with higher morbidity. Corticosteroids and simultaneous hormonal therapy remain the mainstay of treatment.

EP_A109

OBSTACLES IN MANAGING GIANT PROLACTINOMA: A SUDDEN RESURGENCE WITH NEW ONSET SEIZURE IN GIANT PROLACTINOMA

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INTRODUCTION/BACKGROUND

Prolactinoma is the most common type of secretory pituitary adenoma, caused by oversecretion of prolactin (PRL). Giant prolactinomas are uncommon, accounting for only 2-3% of all prolactinomas and are more common in males. Management of giant prolactinomas is also more challenging as these patients require a higher dose of dopamine agonist (DA) and are at risk of developing aggressive prolactinomas or carcinoma.

CASE

This is a case of a 29-year-old male who was diagnosed with giant prolactinoma at the age of 24, with extension to both cavernous sinuses (knops 4), causing 3rd ventricle compression. He was treated medically with an increasing dose of DA for the last 5 years. There was an improvement in both prolactin level and tumour size with cabergoline 1.5 mg daily (total weekly dose 10.5 mg) until the current presentation when he had a sudden increase in prolactin levels accompanied by new onset seizures.

Assay interference was excluded after 3 samples of serum prolactin levels sent to different platforms revealed almost similar results. Macroprolactinemia was also excluded after the PEG test came back negative. The cranial MRI also revealed a minimal increase in tumour size without any indication of tumour aggressiveness. Upon further inquiry, he admitted being less compliant with his cabergoline dose for the past year, with no apparent reason. He was reminded to strictly adhere to the prescribed dosage of DA, and the follow-up MRI of the pituitary was planned after 6 months. Indications for adjunctive therapy (i.e., transsphenoidal surgery for tumour enlargement despite compliance with medication) were also explained to the patient.

CONCLUSION

This case highlighted the challenges in managing giant prolactinomas, the differential diagnoses of biochemical relapse with new-onset seizure and the importance of medication adherence. We also highlight the indication for surgery in macroprolactinoma.

EP_A110

TRIPHASIC PHASE OF CENTRAL DIABETES INSIPIDUS (DI) POST TRANSPHENOIDAL SURGERY: A NORTHERN REGION GOVERNMENT HOSPITAL EXPERIENCE

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INTRODUCTION/BACKGROUND

The transsphenoidal pituitary surgery approach is a potential cause of central diabetes insipidus (DI) due to its impact on the pituitary stalk. Triphasic DI is a rare manifestation of central DI. In the initial phase, patients experience polyuria secondary to axonal shock or injury of vasopressin-secreting neurons in the hypothalamus. In the second phase, there is transient inappropriate antidiuretic hormone secretion (SIADH) due to vasopressin leakage from the damaged posterior pituitary tissues. The third phase ensues if >80% of vasopressin-secreting neurons are destroyed.

CASE

A 44-year-old Malay female presented with sudden slurring of speech, numbness of the right upper limb and headaches. Her cranial CT and MRI showed pituitary macroadenoma (1.1 x 1.5 x 1.1 cm). Pituitary hormone profile was normal. Ten months later, she developed bitemporal hemianopsia with a repeated cranial MRI showing unchanged size of the sellar lesion. Eventually, she underwent transsphenoidal surgery and tumour excision for pituitary macroadenoma.

About 8 hours post-op, she developed polyuria with urine output of 400 ml/hour, sodium level: 145 mmol/L, urine sodium: <20 mmol/L, serum osmolality: 299 mmol/L, urine osmolality: 110 mmol/L which was consistent with central DI. Subcutaneous desmopressin was given on days 1 to 3 post-op.

On day 7 post-op, she developed SIADH as evidenced by serum osmolality of 241 mmol/L, urine osmolality of 527 mmol/L and urine sodium of 124 mmol/L.

Upon entering day 11, she had another episode of polyuria (200 ml/hour). Laboratory tests revealed serum sodium: 126 mmol/L, serum osmolality: 269 mmol/L, urine osmolality: 111 mmol/L and urine sodium: 111 mmol/L which were suggestive of central DI in the triphasic phase. Hence, subcutaneous desmopressin was resumed. Sodium levels normalized and she was discharged home clinically well on sublingual desmopressin.

CONCLUSION
Although triphasic phase of central DI is relatively rare, it is important to identify the phase, as the treatment differs depending on the phase.

EP_A111

UNRAVELING THE ENIGMA: TRIMETHOPRIM-SULFAMETHOXAZOLE-INDUCED SIADH

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INTRODUCTION/BACKGROUND
Syndrome of inappropriate antidiuretic hormone secretion (SIADH) presents a complex clinical scenario characterized by the aberrant secretion of antidiuretic hormone (ADH), leading to hyponatremia, water retention and potential neurological manifestations. Trimethoprim-sulfamethoxazole (TMP-SMX, Bactrim) is a potential cause of medication-induced SIADH.

CASE
A 21-year-old female came in with left gluteal abscess and newly diagnosed diabetes mellitus complicated by diabetic ketoacidosis (DKA). Following treatment for DKA and abscess incision and drainage, which grew Staphylococcus argenteus, she received intravenous cloxacillin for 7 days. Antibiotics were then shifted to oral TMP-SMX as she was deemed fit for discharge. After 3 days on TMP-SMX, she developed severe hyponatremia with a sodium level of 114 mmol/L, despite having baseline sodium levels ranging between 135-143 mmol/L. Despite hydration with 4 L of NaCl per day, her serum sodium levels continued to decline, reaching a nadir of 108 mmol/L. She was then referred to the medical team for further management.

Urine sodium and osmolarity were elevated at 95 mEq/L and 316 mOsm/L, respectively, with a low serum osmolality at 262 mOsm/L. Morning cortisol level and thyroid function tests were within normal level and she was euvolemic. A diagnosis of medication TMP-SMX-induced SIADH was made. She was started on fluid restriction of less than 1 L per day. Serum sodium levels gradually improved to 130 mmol/L, with stable electrolytes, and renal function and she was discharged well.

CONCLUSION
TMP-SMX is a potential cause of medication-induced SIADH. Additionally, trimethoprim (TMP) shares structural similarities with amiloride and functions on the identical epithelial sodium channels (eNAC) in the distal nephron, leading to natriuresis and hyponatremia. Prompt identification of the cause of hyponatremia (diuresis vs SIADH) is crucial in averting severe complications linked with hyponatremia.

EP_A112

WHEN TWO DIABETES MET: HYPERGLYCAEMIC EMERGENCY OR VASOPRESSIN DISORDER?

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INTRODUCTION/BACKGROUND
Central diabetes insipidus (CDI) is caused by decreased secretion of or resistance to ADH. The clinical and laboratory findings may be similar to the hyperosmolar hyperglycaemic state (HHS). We reviewed case notes, investigation results, imaging and treatment options based on literature review.

CASE
A 30-year-old female with history of gestational diabetes mellitus presented with 3-day history of vomiting and fever. She also had polyuria, polydipsia and fatigue for the past 2 years. Her blood sugar level was 24.9 mmol/L, serum osmolarity was 346 mOsm/L and serum sodium was 162 mmol/L, with no acidosis or ketosis. The patient was diagnosed with HHS and received appropriate treatment. However, she continued to experience polyuria. Further investigation revealed weight gain, irregular menstrual cycles and recent absence of menstruation. Subsequent investigations revealed features of diabetes insipidus (DI) (serum sodium: 160 mmol/L, serum osmolality: 350 mOsm/kg, urine osmolality: 114 mOsm/kg). Following the administration of desmopressin, the urine osmolality increased to 505 mOsm/kg. Additional tests conducted showed normal prolactin, cortisol and thyroid function, but low IGF-1 and hypogonadotropic hypogonadism. The patient was started on regular sublingual desmopressin and her symptoms improved. She is currently awaiting an MRI of the pituitary gland.

The patient's initial presentation resulted in treatment for HHS. However, subsequent investigation uncovered the presence of CDI, which has been obscured by the diabetes mellitus. In younger patients who present with CDI, hypophysitis is typically the cause, reported to occur in up to 50% of patients. \Treatment decisions should be guided by clinical evaluation and imaging, as patients with pituitary dysfunction but no mass effect and likely lymphocytic hypophysitis may be managed with medical therapy and close monitoring.

CONCLUSION

Clinicians should have a high index of suspicion of CDI among patients manifesting with possible HHS who do not improve despite adequate control of hyperglycemia.

EP_A113

LOCAL EXPERIENCE WITH TARGETED RADIONUCLIDE THERAPY IN MALIGNANT PHEOCHROMOCYTOMA AND PARAGANGLIOMA

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INTRODUCTION/BACKGROUND

Targeted radionuclide therapy (TRT) is a promising therapeutic option for patients with malignant pheochromocytoma and paraganglioma (PPGL) but it is not widely available locally.

METHODOLOGY

We conducted a retrospective cohort study of patients with malignant PPGL from 2000 to 2023.

RESULTS

We report the experience of TRT among 15 patients with malignant PPGL (26% pheochromocytoma, 60% paraganglioma, 13% combined) under follow-up in a tertiary endocrine referral centre from 2000 to 2023. There was equal gender distribution with a median of 41 years at diagnosis. They had elevated 24-hr urine normetanephrine (100%), 3-methoxytyramine (53.3%) or urine metanephrine (27%). A total of 11 patients had multiple operations with residual primary and metastatic tumours, 2 had recurrence after initial complete resection and another 2 had unresectable primary tumour. The choice of TRT was based on avidity in functional imaging and consensus from multidisciplinary meetings. Ten patients had peptide receptor radionuclide therapy (PRRT) and 5 patients had iodine 131-meto-iodo-benzyl-guanidine (MIBG). A 177Lu-DOTATATE was used for PRRT with a mean dose of 201.23 mCi (7.47GBq/cycle). There was a reduction in both urine normetanephrine (93%) and requirements for antihypertensive medications (80%) after TRT. Using Response Evaluation Criteria in Solid Tumours (RECIST), disease control rate was 40% after 4 cycles of PRRT (n = 4) or MIBG (n = 2). Among patients with disease progression, a subsequent plan was additional TRT cycles up to a total of 6 cycles (n=3), chemotherapy (n=2), or watchful waiting (n = 1). One patient with SDHB mutation, who had multimodal therapies including multiple surgeries, chemoembolization, PRRT and chemotherapy with temozolamide, succumbed to her progressive disease 20 years after diagnosis. With regards to toxicities using Common Terminology Criteria for Adverse Events (CTCAE), there were grade I hypotension post PRRT (n = 1), grade I leucopenia (n = 1) and grade I-II renal impairment (n = 3).

CONCLUSION

TRT is well tolerated and worthy of extensive research to explore full potential in the treatment of advanced or non-resectable PPGL.

EP_A114

HYPERNATREMIC DEHYDRATION AND ACUTE MASSIVE PULMONARY EMBOLISM: COINCIDENCE OR TRUE RISK FACTOR

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INTRODUCTION/BACKGROUND

Diabetes Insipidus (DI) is a common complication after extensive transcranial surgery for craniopharyngioma. DI mainly causes impairment of the sense of thirst and vasopressin (AVP) secretion. This puts the patient at risk of severe dehydration and hypernatremia. Venous thromboembolism is one of the potentially fatal complications which can occur due to severe dehydration and hypernatremia.

CASE

We report a 22-year-old Malay male, college student, with underlying panhypopituitarism and chronic diabetes insipidus post-removal of craniopharyngioma at the age of 8 years with acute massive bilateral pulmonary embolism (PE). He presented to our Emergency Department with acute onset of shortness of breath and chest pain for a day. Although feeling unwell, he was still able to ambulate and attend school.

One day PTA, due to poor oral intake, he did not take his replacement therapy tablets: hydrocortisone, L- thyroxine and desmopressin. Upon arrival, he was noted to be hypotensive with a blood pressure of 70/50 mmHg and a heart rate of 110 bpm. His O_2 saturation at room air was 80%. He appeared to be dehydrated with dry tongue. His GCS on arrival was E3V5M6. The abnormalities of his blood investigations were urea of 6.4 mmol/L, creatinine of 292 umol/L and sodium of 150 mmol/L. ECG showed sinus tachycardia with features of acute right ventricular strain pattern with S1Q3T3. Bedside echocardiogram showed features of acute PE with a dilated right ventricle and the presence of McConnell's' sign. A CTPA showed evidence of bilateral main pulmonary artery saddle embolism with RV thrombus. He was then referred to the National Heart Institute (IJN) for EKOS and catheter-guided thrombolysis where he was successfully treated.

CONCLUSION

The case illustrates the importance of severe hypernatremia and dehydration as predisposing factors for venous thromboembolism.

EP_A115

CENTRAL SEROUS CHORIORETINOPATHY (CSCR): AN UNCOMMON MANIFESTATION OF CUSHING'S SYNDROME

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INTRODUCTION/BACKGROUND

Central serous chorioretinopathy (CSCR) has been identified as a rare clinical presentation linked to elevated cortisol levels, both in overt Cushing's syndrome and in subclinical cases of hypercortisolism.

We report a case of Cushing's syndrome with uncommon presentation.

CASE

A 42- year-old female with pre-existing diabetes mellitus, hypertension and class III obesity came to the ophthalmology clinic for blurring of vision. Upon presentation, her blood pressure was 198/100 mm Hg and her blood glucose was 20 mmol/L. She was therefore admitted due to hypertensive emergency and uncontrolled diabetes mellitus. As the patient exhibited stigmata of Cushing's syndrome, further investigations revealed unsuppressed serum cortisol level after an overnight low dose (1mg) dexamethasone suppression test (ODST), elevated 24- hour urinary cortisol 1912 nmol/24hours, elevated plasma adrenocorticotrophic hormone (ACTH): 14.8 pmol/L, elevated serum dehydroepiandrosterone sulphate (DHEAS): >27 umol/L and elevated serum testosterone: 5.59 nmol/L. Eye assessment with fundoscopy and optical coherence tomography was suggestive of CSCR. Magnetic resonance imaging (MRI) revealed a left lateral pituitary microadenoma. She was treated with steroid-lowering therapy and scheduled for eye laser treatment by a retina surgeon.

CONCLUSION

When CSCR is diagnosed, it is important to consider a work-up for Cushing's syndrome due to the association between high cortisol levels and CSCR. Laser therapy is one of the treatment options for CSCR while addressing the underlying cause.

EP_A116

POSTERIOR STALK INTERRUPTION SYNDROME: A PECULIAR PRESENTATION OF AN UNCOMMON DISEASE

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INTRODUCTION/BACKGROUND

Posterior stalk interruption syndrome (PSIS) is a rare anatomical congenital anomaly that is characterised by a radiological triad of a thin or interrupted pituitary stalk, an absent or ectopic posterior lobe and anterior lobe hypoplasia or aplasia. Patients typically manifest with anterior pituitary hormone deficiencies at varying ages of presentation ranging from infancy to early adulthood.

CASE

We present a 15-year-old female who was initially referred for thrombocytopenia and hepatosplenomegaly. Further evaluation revealed that she also had short stature and primary amenorrhea. Antenatal history was unremarkable with no reported obstetrics complications. Clinical examination is consistent with Tanner Stage 1 with a height measuring below the third centile for her age.

Initial work-up with peripheral blood, iron studies and haemoglobin analysis confirmed true thrombocytopenia and ruled out haemoglobinopathies. Screening of the anterior pituitary hormone profile revealed that she had hypothyroidism, hypocortisolism, growth hormone deficiency, low gonadotropins levels with normal prolactin. MRI demonstrated a small anterior pituitary measuring 0.4 cm, with an ectopic posterior lobe and the infundibulum was not visualised. Her presentation, complemented with the biochemical and radiological findings confirmed a diagnosis of PSIS. She was started on hormonal replacement therapy.

CONCLUSION

PSIS occurs in about 0.5/100,000 births. The pathogenesis is largely unknown, though genetic factors and obstetric trauma are considered potential contributors. Common presentation includes short stature and delayed puberty. A combination of clinical assessment and biochemical tests is required to form a suspicion with MRI as the confirmatory test, differentiating PSIS from other pituitary pathologies. Treatment involves replacing the deficient hormones.

EP_A117

A RARE CASE OF KALLMAN SYNDROME IN A FEMALE

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INTRODUCTION/BACKGROUND

Kallman Syndrome, a rare genetic disorder referring to hypogonadotropic hypogonadism, is associated with anosmia or hyposmia. It is due to abnormal migration of gonadotrophin-releasing hormone-producing neurons. The reported incidence is 1 in 8000 in men and is 5 times rarer in women. We reviewed case notes, investigation results, and imaging studies and discussed treatment options based on literature review and treatment availability.

CASE

A 19-year-old female was referred to endocrinology for primary amenorrhoea with pituitary microadenomas. The patient was born to non-consanguineous parents. She is the second child with 3 healthy siblings. The patient cannot smell since childhood. On physical examination, Tanner's staging of the breasts and pubic hair were 3 and 1, respectively, with no axillary hair. Perineal examination revealed a not well-formed labia majora with well-formed labia minora. Urethra and vaginal orifice were seen. She had bilaterally small fingers. Ultrasound of the abdomen showed a small uterus at 2.1 x 1.1 cm with no ovaries seen. MRI of the brain showed bilateral pituitary microadenomas measuring 5 x 4 x 3 mm and 5 x 4 x 2 mm on the right and left side of the anterior pituitary lobe, respectively. Unfortunately, the olfactory bulb was not assessed. Hormonal assays identified a hypogonadotropic hypogonadism profile with total serum testosterone <0.24 nmol/L (NR: 0.29-1.21 nmol/L), serum oestrogen <43.3 pmol/L (NR: 59.1-874.6 pmol/L), serum luteinizing hormone 0.11 IU/L (NR: 1.0-52.5 IU/L), serum follicular stimulating hormone 1.02 IU/L (NR: 2.2-10.1 IU/L). Serum prolactin was normal at 79.23 uIU/mL. The patient was started on oestrogen pills and started to have fullness and tenderness in her breasts. She was referred to the Genetic Clinic for genetic studies.

CONCLUSION

Patients presenting with primary amenorrhoea and anosmia should prompt suspicion of Kallman Syndrome. Laboratory and radiological evaluation may be helpful as genetics confirmation will take time. Early detection and initiation of hormonal treatment will enable the progression of the secondary sexual characteristics. However, achieving fertility will still be a challenge depending on the availability of gonadotrophins or pulsatile GnRH therapy.

EP_A118

AN AGGRESSIVE CATECHOLAMINE-SECRETING GLOMUS PARAGANGLIOMA: A CASE REPORT

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INTRODUCTION/BACKGROUND

Head and neck paragangliomas (HNPGLs) are commonly detected at the carotid artery bifurcation and carotid body but may arise in the middle ear. HNPGL presents as slow-growing, painless neck mass. Majority are non-functional with approximately 5% being biochemically active. HNPGLs are generally locally invasive, and destructive and up to 19% may be malignant. Management for this type of paraganglioma is difficult and requires a multidisciplinary approach. We present a patient with a huge and locally aggressive functioning left glomus-jugulotympanicum-paraganglioma with significant management challenges.

CASE

A 33-year-old male presented with left ear pulsatile tinnitus which was treated as left otitis media. Despite the persistent symptoms and progressive hearing

impairment in the left ear, an investigation for suspicion of glomus tympanicum commenced 6 months later. He had repeated ER visits for the next year due to the persistence of symptoms and uncontrolled hypertension. However, he was only referred to the endocrine clinic for young hypertension investigation 20 months from the initial presentation with significant paroxysms of palpitation, headaches and elevated BP. Diagnosis of catecholamine-secreting glomus-jugulotympanicum-paraganglioma was confirmed with elevated urinary metanephrine and huge soft tissue mass in the left jugular fossa with local bony erosion and intracranial extension on MRI.

His BP control was labile and required multiple oral-antihypertensives including phenoxybenzamine. Multi-disciplinary team management prepared him for definitive surgical intervention. He underwent tumour embolization prior to the actual surgery. Pre-operative management was extremely challenging which required CCU admission for BP stabilization. Intra-operative period was surprisingly uneventful, but he developed multiple cranial nerve palsies postoperatively. A second operation was required due to infection and enlarging tumor with compression. Paroxysm of symptoms improved after second surgery but he still had significant residual tumor. MIBG-therapy was planned but management was delayed due to the COVID-19 pandemic and treatment funding.

CONCLUSION

Awareness of functional paraganglioma presentation is imperative to avoid late detection. HNPGLs that are both aggressive and functional pose extreme difficulty in achieving disease remission.

EP_A119

ADIPSIC CENTRAL DIABETES INSIPIDUS IN A PATIENT WITH SUPRASELLAR GERMINOMA

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INTRODUCTION/BACKGROUND

Adipsic central diabetes insipidus (CDI) is a rare and life-threatening disease which is difficult to manage as the patient experiences loss of thirst sensation compounded by fluid loss and hypernatremia. We present a case of a patient with suprasellar germinoma with panhypopituitarism who presented with adipsic CDI.

CASE

A 20-year-old male presented with generalized body lethargy for 1 month and polyuria for 1 year. He did not complain of polydipsia. Blood tests revealed severe hypernatremia with serum sodium (Na) of 160 mmol/L, serum osmolality of 300 mOsm/kg and urine osmolality of 130 mOsm/kg. Other electrolytes and blood glucose were normal. MRI showed a large sellar/suprasellar mass with periventricular subependymal spread causing acute obstructive hydrocephalus. Hormonal panel showed panhypopituitarism. He was started on sublingual desmopressin, L-thyroxine, hydrocortisone and intramuscular testosterone. During his confinement, he denied polydipsia despite intermittent polyuria. Strict intake and output monitoring were instituted with hourly urine output and regular renal profile monitoring. Despite initial normalisation of Na levels, he developed 2 episodes of hypernatremia when he had breakthrough polyuria. Intravenous fluids were given intermittently to balance his output. The patient and caregiver were constantly reminded to take adequate oral fluid. He then underwent transcranial biopsy. Histopathology examination showed a diagnosis of central nervous system germinoma. A referral to the Oncology team for chemotherapy was made.

Adipsic CDI has been reported to account for about 10% of CDI cases. Patients with adipsic CDI have higher prevalence of complications such as hypernatremia, renal insufficiency and venous thrombosis. Apart from desmopressin, crucial management steps include regular monitoring to ensure adequate fluids and desmopressin replacement.

CONCLUSION

This case highlights the difficulties in managing adipsic CDI and the need for constant and regular monitoring to prevent life-threatening hypernatremia.

EP_A120

PRECISION MANAGEMENT IN A CASE OF PHEOCHROMOCYTOMA CATALYSING EARLY DETECTION OF MEDULLARY THYROID CARCINOMA

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INTRODUCTION/BACKGROUND

Approximately 35% of pheochromocytoma carry a germline mutation, highlighting the importance of genetic screening in early detection and follow-up of patients with hereditary syndromes. We report a case of a young female in whom individualized pheochromocytoma management expedited the diagnosis of medullary thyroid cancer.

CASE

A 21-year-old female student presented to the intensive care unit with malignant hypertension complaining of severe headache and abdominal pain. Computed tomography (CT) of the abdomen revealed a heterogenous mass measuring 5.9 x 6.3 x 6.2 cm arising from the left adrenal gland. Plasma free normetanephrines were significantly elevated at 53 nmol/L (normal range: <0.9 nmol/L). She was prepared preoperatively with phenoxybenzamine and propranolol, and successfully underwent open left adrenalectomy. Intraoperative findings revealed a well encapsulated adrenal tumour measuring 7 x 6 cm, weighing 122 grams, confirmed as pheochromocytoma. Genetic testing revealed RET proto-oncogene missense mutation (Cys634Trp). Screening for medullary thyroid carcinoma (MTC) and primary hyperparathyroidism were carried out in order to exclude other possible coexisting disorders in MEN2A syndrome. Serum calcium and parathyroid hormone were normal. Neck ultrasound revealed TIRAD 4 nodules on the upper pole of the right and left thyroid lobe with the largest measuring 1.1 cm and 1.3 cm, respectively. Lymph nodes were not enlarged. Serum calcitonin was elevated at 111 ng/L (normal range: ≤7.6 ng/L). Fine needle aspiration of bilateral thyroid nodules revealed papillary thyroid carcinoma (Bethesda V). She subsequently underwent total thyroidectomy. However, pathological examination of the tumours disclosed low grade multifocal MTC, staining positive for calcitonin, chromogranin A, synaptophysin and CD56. There was no evidence of extrathyroidal tumour extension or lymph node metastasis.

The patient was started on replacement L-thyroxine post-operatively.

CONCLUSION

Early diagnosis of multifocal MTC had been made in this young female due to the benefit of germline mutation. This has reduced the likelihood of unfavorable clinical outcomes associated with MEN2 syndrome.

EP_A121

HARMONISATION OF MACROPROLACTIN REPORTING

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INTRODUCTION/BACKGROUND

The post-analytical phase is the last phase of the total testing process and it is an important step since it maximises the quality and effectiveness of laboratory information. Macroprolactin is a well-known analytical problem in laboratory diagnostics. There is no agreement on when to screen for macroprolactin in patients with hyper-prolactinaemia nor is there consensus on the reporting, terminology and reference intervals for macroprolactin.

METHODOLOGY

To create a consensus document that standardises the reporting of prolactin results after precipitation with polyethylene glycol (PEG) to minimise errors in result interpretation.

As a part of the joint initiative by the Chapter of Chemical Pathology and Metabolic Medicine, College of Pathologists, Academy of Medicine Malaysia and the Malaysian Association of Clinical Biochemists to harmonise the laboratory reporting of macroprolactin in Malaysia, an audit was conducted to obtain feedback from endocrino-logists from public, private and academic institutions on macroprolactin reporting. The questionnaire included the level of prolactin that was considered as hyperprolactinaemia, its cut-off value in males and female and whether screening for macroprolactin should be clinician-centred or laboratory-centred. It also addressed post-PEG precipitation, whether the report should specify the presence of macroprolactin and the preferred reporting format.

RESULTS
Responses were obtained from 33 endocrinologists. The majority (81%) used reference limits given rather than a fixed cut-off of 700 m IU/L and 70% used a sex-specific range. More than half (69%) favoured that the laboratory should screen for macroprolactin in all samples with high prolactin. On report format, 61.5% preferred the use of post-PEG monomeric reference ranges and 100% required the laboratory to indicate the presence of macroprolactin.

CONCLUSION
The laboratory working group will consider the responses from endocrinologists in preparing the consensus recommendations on reporting macroprolactin and the reference intervals.

EP_A122

UNVEILING PITUITARY PARADOX: OCTREOTIDE LONG-ACTING RELEASE (LAR) INDUCED APOPLEXY IN POST-OPERATIVE RESIDUAL MACROADENOMA

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INTRODUCTION/BACKGROUND
The development of a somatostatin analogue (SSA) has revolutionized the treatment of acromegaly. Octreotide LAR is a long-acting release formulation of SSA, often used as an alternative when surgery is not possible in patients with acromegaly or as an adjuvant therapy in post-operative patients with residual pituitary adenoma. Pituitary apoplexy, characterized by infarction or bleeding of the pituitary gland, is a rare condition.

CASE
An 18-year-old male with a clinical and biochemical diagnosis of acromegaly underwent transsphenoidal surgery in November 2021 with incomplete tumour resection, complicated by pituitary apoplexy after administration of octreotide LAR.

Post-operatively, he had residual pituitary adenoma with optic chiasm compression and persistent elevations of insulin-like growth factor 1 (IGF-1) (1.7x > upper limit of normal) and growth hormone (GH) level (10x > normal limit). After extensive discussion, the patient was started on medical treatment, cabergoline 0.5 mg twice weekly, but failed to achieve biochemical control despite continued use for 9 months. Subsequently, octreotide LAR 30 mg monthly was started, aiming to achieve better biochemical control and shrink the tumour size while awaiting stereotactic radiosurgery.

Unfortunately, 6 weeks after the first injection of octreotide LAR, he developed a sudden severe headache and visual disturbance, presenting clinically with bitemporal hemianopia, subsequently diagnosed with pituitary apoplexy on cranial MRI. Octreotide LAR was discontinued. Patient underwent repeated transsphenoidal surgery, which was uncomplicated, albeit with pre-existing central hypothyroidism and hypocortisolism.

CONCLUSION
Pituitary apoplexy is one of the rare complications of SSA. However, clinicians need to maintain a high level of suspicion for this complication if the patient presents with sudden headache with or without neuro-ophthalmic signs after receiving SSA, given its significant morbidity and potential fatality.

EP_A123

A RARE CASE OF ECTOPIC GIANT PROLACTINOMA MIMICKING CLIVAL CHORDOMA

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INTRODUCTION/BACKGROUND
Ectopic pituitary adenomas are extremely rare tumours which develop outside the sella turcica. The most common locations are the sphenoid sinus, clivus, suprasellar space, nasopharynx and cavernous sinus. Due to their rarity, these tumours are frequently misdiagnosed as other skull lesions such as chordoma, chondrosarcoma, meningioma or astrocytoma. We present a case of ectopic giant prolactinoma mimicking as clival chordoma.

CASE
A 38-year-old nulliparous female presented with oligomenorrhoea since the age of 20 and secondary amenorrhea at the age of 38 associated with galactorrhoea. Otherwise, she denied headache, symptoms of increased ICP or blurring of vision. There were no other symptoms to suggest pituitary hyper- or hypofunction. Her initial hormonal work-up showed hyperprolactinemia (>8000 ng/mL) with hypogonadotropic hypogonadism. MRI of the brain was signed out as clival chordoma measuring 2.3 x 3.6 x 4.1 cm displacing the pituitary gland and

infundibulum. She underwent a biopsy of the lesion and the HPE confirmed pituitary lactotroph adenoma. She was started on cabergoline and her prolactin levels reduced significantly.

CONCLUSION

Ectopic pituitary adenomas occurring in the clivus are very rare. The main differential diagnosis to be considered is chordoma. Other than radiological imaging, additional endocrinological workup may be useful as well to establish the diagnosis. Biopsy of the lesion should be performed whenever the diagnosis is in doubt, as medical treatment with cabergoline often yields favourable outcomes in cases of ectopic prolactinoma which leads to a reduction in tumour size and prolactin level.

EP_A124

BATTLING THE UNCOMMON: A CASE REPORT ON ECTOPIC ACTH SYNDROME FROM PANCREATIC NEUROENDOCRINE TUMOUR WITH LIVER METASTASES

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INTRODUCTION/BACKGROUND

Pancreatic neuroendocrine tumours (pNETs) are rare malignancies originating from the islet cells of the pancreas, comprising only 1%-2% of pancreatic tumours. Among functional pNETs, insulinoma and gastrinoma are the most common, while ACTH-secreting tumours are very rare. Ectopic ACTH syndrome (EAS) caused by pNETs is particularly aggressive, often presenting with metastatic disease, primarily to the liver.

CASE

We present a case of a 25-year-old male who presented acutely with upper gastrointestinal bleeding. Clinical examination revealed features consistent with Cushing's syndrome, and further investigations identified an ACTH-secreting neuroendocrine pancreatic tumour with liver metastasis. The disease was complicated by extensive inferior vena cava thrombosis and concurrent thrombocytopenia, necessitating the insertion of an IVC filter. The patient underwent distal pancreatectomy, splenectomy and wedge resection of the stomach. Hypercortisolism was controlled with steroidogenesis inhibitors, including metyrapone, ketoconazole and the newer agent, osilodrostat. Post-operatively, he underwent a Ga-68 Dotatate scan which showed evidence of somatostatin-receptor avid metastatic disease in the liver and he was started on a somatostatin analogue.

pNETs typically localized in the head and body of the pancreas with an average size of 4.6 cm (2.5–7 cm) and the source of ectopic ACTH secretion may remain hidden for several years. In our case, the pancreatic lesion measured 11 cm which was larger than the average size. Despite the challenges posed by the tumour's size and complications such as IVC thrombosis, the multidisciplinary approach involving endocrinologists, surgeons and radiologists allowed for effective management. Surgical intervention was complemented by medical therapies such as metyrapone, ketoconazole, lanreotide and eventually osilodrostat to control hypercortisolism. Osilodrostat demonstrated good efficacy in reducing cortisol levels in our patient.

CONCLUSION

This case highlights the aggressive nature of EAS-pNETs and the challenges in the management, particularly when presenting with advanced metastatic disease. The use of osilodrostat represents a promising advancement in the management of EAS.

EP_A125

CUSHING'S DISEASE AND THROMBOSIS: A CLINICAL PERSPECTIVE

https://doi.org/10.15605/jafes.039.S1.136

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INTRODUCTION/BACKGROUND

Cushing's disease is the most common cause of endogenous Cushing's syndrome, which is caused by an adrenocorticotropin (ACTH) -secreting pituitary tumour. It poses a myriad of complications due to the state of excess cortisol levels, including thrombosis. It is interesting that thrombotic risk due to the hypercoagulable state in Cushing's disease is higher after pituitary surgery when cortisol levels are diminished.

We present a case of a female with recurrent Cushing's disease who developed extensive thrombotic complications post-successful transsphenoidal surgery (TSS) to highlight the importance of anticoagulation therapy in mitigating the risk of thrombosis.

CASE
A 31-year-old female with underlying diabetes mellitus, hypertension and a history of previous treatment for Cushing's disease presented with symptoms of weight gain, hirsutism and purplish striae for 2 years. MRI showed a pituitary adenoma measuring 2.8x4.4x3.3 mm. A 24-hour urinary cortisol and overnight dexamethasone suppression tests were not suppressed. She underwent endoscopic TSS. Post-surgery, her cortisol levels reduced from 419 to 57.3 nmol. Subsequently, she was found to have a saddle pulmonary embolism and extensive right lower limb deep vein thrombosis requiring pulmonary thrombectomy. Post-procedure, she was started on anticoagulants.

Hypercoagulation in Cushing's disease is due to the increase in clotting factors II, V, IX, and VIII, fast-acting plasminogen activator inhibitors and the decrease of tissue-type plasminogen. The stress post-surgery causes an abnormal Von Willebrand Factor pattern production leading to platelet aggregation and the drop in cortisol levels will trigger an inflammatory response that initiates the coagulation cascade. The elevated thrombotic risk will decrease after 3 months to a year later as the glucocorticoid effect takes time to wean off, hence requiring anticoagulation.

CONCLUSION
Recognition of thrombosis post-surgery for Cushing's disease is vital to prevent mortality and morbidity. An individualized strategy based on the degree of thrombosis is therefore essential in the management.

EP_A126

HYPONATRAEMIA SECONDARY TO SIADH: COULD IT BE METHAMPHETAMINE USE?

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INTRODUCTION/BACKGROUND
Hyponatraemia can be a result of SIADH. Patients with methamphetamine use frequently present with hyponatraemia, possibly secondary to SIADH, among other complications.

We describe a patient with a history of methamphetamine use presenting with persistent hyponatraemia secondary to SIADH.

CASE
A previously well, 50-year-old male with a history of methamphetamine usage (last intake 30 years ago), presented with generalized body aches and weakness, vomiting, reduced oral intake and constipation for 1 week. On examination, his GCS was full, blood pressure was 120/72 mm Hg, heart rate was 72 and afebrile. Other systemic examinations were unremarkable. Blood parameters showed hyponatraemia with hypokalaemia (sodium 121 mmol/L, potassium 3 mmol/L). Despite 4 days of intravenous drip hydration and oral sodium chloride, his clinical condition and sodium levels did not show any improvement. His lowest sodium was 110 mmol/L, and hyponatraemic workup was consistent with SIADH (serum osmolality: 238 mOsm/kg, urine osmolality: 819 mOsm/kg and urine sodium: 187 mmol/L). Morning cortisol, thyroid function test, renal profile, Synacthen test, ACTH and tumour markers were normal. Patient was diagnosed with symptomatic hyponatraemia secondary to SIADH due to methamphetamine. Subsequently, he was started on intravenous hypertonic saline for 2 days coupled with fluid restriction of 500 mL/day. In view of imperceptible improvement of clinical symptoms and sodium level (115 mmol/L), fludrocortisone 0.1 mg tablet bid was then added. After more than 1 week of treatment, the peak serum sodium level achieved was 120 mmol/L.

Literature showed that amphetamines can be associated with serotonin-mediated hyponatraemia. This can happen as a result of SIADH or excessive water intake from hyperpyrexia following drug ingestion.

CONCLUSION
This case illustrates possibility of hyponatraemia secondary to SIADH which could be a result of methamphetamine use. Absence of urine toxicology test upon patient's presentation causes difficulty to confirm this diagnosis, and the possible duration of effect of methamphetamine-induced SIADH is yet unknown. Nevertheless, a history of recreational drug consumption should be included in the clinical evaluation of unexplained hyponatraemia.

EP_A127

RARE CASE OF PINEAL GERMINOMA WITH ARGININE VASOPRESSIN DEFICIENCY

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INTRODUCTION/BACKGROUND
Pineal tumour is an uncommon type of brain tumour in Malaysia, with low incidence rate of 2.1% based on a demographic study done in 1 of the states in Peninsular Malaysia. On the other hand, germinoma is a common cause of intracranial tumor leading to arginine vasopressin deficiency (AVP-D). In a literature review, 82% of 95 patients were diagnosed with AVP-D.

CASE
We report a case of a 22-year-old Malay male, who was diagnosed with pineal gland tumour complicated with obstructive hydrocephalus. He underwent endoscopic biopsy of pineal region tumor and right ventriculo-peritoneal shunt insertion in January 2024.

Two days after his surgical intervention, he developed an episode of polyuria with urine output more 5 ml/kg/hr for consecutive hours in the ward without any accompanying thirst. Investigations showed high serum osmolarity of 294 mOsm/kg (NR: 271-286 mOsm/kg) with inappropriately low urine osmolarity of 161 mOsm/kg (NR: 300-900 mOsm/kg).

He was diagnosed with AVP-D and responded well to intravenous desmopression (DDAVP), a vasopressin synthetic analogue, with reduction in urine volume and passing more concentrated urine. Over the next few days, he started to develop more episodes of polyuria, requiring regular dosing of desmopressin.

The tissue histopathology of the pineal gland revealed germ cell tumour consistent with germinoma.

CONCLUSION
AVP-D is a known complication of intracranial germ cell tumour. Our case report shows that AVP-D may also happen as a consequence of neurosurgical intervention. Careful clinical and biochemical postoperative monitoring remains essential to correctly diagnose and manage AVP-D following neurosurgery intervention.

EP_A128

WORSENING OF EXISTING CRANIAL DIABETES INSIPIDUS SYMPTOMS AFTER COMMENCEMENT OF HYDROCORTISONE

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INTRODUCTION/BACKGROUND
Central diabetes insipidus (DI) is a condition that causes persistent thirst and frequent urination. It can develop when a pituitary tumour compresses on the posterior pituitary gland and impairs the gland's ability to secrete vasopressin hormone.

CASE
We report a case of a 22-year-old male who presented with a 5-month history of delayed responses, progressive visual loss and lethargy. He also complained of excessive thirst, frequent urination and increased urine volume which was diluted. Clinically, he was dehydrated. Ophthalmological assessment revealed monocular blindness. Computed tomography of the brain reported a suprasellar mass with obstructive hydrocephalus. Anterior pituitary hormone profile revealed panhypopituitarism and laboratory results confirmed the presence of DI. He was managed with hydrocortisone tablets and BD dosing of desmopressin. Unfortunately, he developed episodes of worsening polyuria, and his sodium increased from 143 to 150 mmol 24 hours following commencement of hydrocortisone. His urine output improved following an increment of desmopressin to TDS dosing and his serum sodium normalized.

Anterior pituitary hormone profiles showed panhypopituitarism with stalk effect; TSH: 1.192 m IU/L, FT4: 3.9 pmol/L, LH: 0.35 IU/L, FSH: 0.61 IU/L, testosterone: <0.35 nmol/l, ACTH: <0.22 pmol/L, morning cortisol: 108 nmol/L and serum prolactin: 2042 m IU/L. His urine sodium: 47 mmol/L, serum osmolality: 345 mosm/kg, and urine osmolality: 277 mOsm/kg. Pituitary MRI showed a well-defined lobulated suprasellar cystic lesion measuring 2.4 x 2.3 x 1.7 cm, causing compression to the floor of the 3rd ventricle which leads to obstructive hydrocephalus. There was a focally enhancing lesion at the pineal gland. He subsequently underwent a right ventriculoperitoneal shunt and supraorbital craniotomy and biopsy. HPE was reported as germinoma. He is currently stable on hydrocortisone, thyroxine and sublingual desmopressin replacement.

CONCLUSION
Glucocorticoid replacement may precipitate diabetes insipidus in the setting of adrenal insufficiency. Low cortisol levels will stimulate the release of antidiuretic hormone and increase water reabsorption in the kidney. This effect can be inhibited by exogenous steroids which may then rapidly unmask or worsen central DI.

EP_A129

SPONTANEOUS RHINORRHOEA SECONDARY TO NORMAL-PRESSURE PARTIAL EMPTY SELLA SYNDROME

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INTRODUCTION/BACKGROUND
Empty sella syndrome is a disorder where the sella turcica is partially or completely filled with cerebrospinal fluid (CSF), resulting in compression and displacement of the pituitary gland. Empty sella is frequently coupled with increased intracranial pressure which causes spontaneous CSF leaks. Some people may experience normal-pressure CSF leaks, which can be due to idiopathic or congenital abnormalities. Both high-pressure and normal-pressure CSF leaks are commonly seen in middle-aged obese women.

CASE
A 43-year-old obese female with a BMI of 34.9 kg/m^2 presented with a two-day history of headache and clear nasal discharge persisting for a month. She reported no weakness or visual disturbances. Upon examination, her blood pressure measured 142/70 mmHg, her Glasgow Coma Scale was intact, and her neurological assessment revealed no abnormalities. The ophthalmological evaluation showed no signs of papilledema. Subsequently, a lumbar puncture was performed, indicating a normal opening pressure of 12 cm H_2O. Brain MRI revealed a partially empty sella, while CT imaging of the paranasal sinuses aimed to identify the cause of the CSF leak, was suggestive of cribriform plate dehiscence. Evaluation of anterior pituitary hormones yielded results within normal ranges. The case was then referred to a neurosurgical team for further evaluation and potential surgical intervention.

The occurrence of a spontaneous cCSF leak frequently coincides with the radiographic discovery of an empty sella. Its clinical manifestation can vary, encompassing symptoms such as headaches, rhinorrhoea, visual impairments, and hormonal imbalances. Assessing pituitary hormone levels is crucial, with hormonal supplementation warranted when deficiencies are identified. Surgical intervention is essential in managing CSF leaks to mitigate potential complications like meningitis and brain abscesses.

CONCLUSION
A CSF leak is important to be recognised at the initial presentation, as surgical intervention is required to reduce the complication of infections.

EP_A130

IMMUNOLOGICAL CROSSFIRE: ENDOCRINOPATHIES IN THE AGE OF IMMUNE CHECKPOINT INHIBITORS

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INTRODUCTION/BACKGROUND
Pembrolizumab is a PD1 receptor inhibitor which is a type of immune checkpoint inhibitor (ICI) for cancer therapy. Immune-related adverse events (IRAE) are more commonly encountered with increased use.

CASE
A 52-year-old male with recurrent renal cell carcinoma developed endocrinopathies following pembrolizumab. After failure of first-line treatment, he received the first dose of pembrolizumab in November 2022.

Six weeks later, he was symptomatic of hyperthyroidism with corresponding biochemistry of TSH <0.01 m IU/L (reference interval 0.55 – 4.8), free T4 of 28.8 pmol/L (reference interval 11.5 – 22.7) and free T3 of 9.7 pmol/L (reference interval 3.5 – 5.5). Following treatment with low dose and tapering carbimazole for eight weeks, he quickly became hypothyroid. Biochemistry showed TSH of 87.3 m IU/L, fT4 of 2.9 pmol/L and fT3 of 1.3 pmol/L. Anti-thyroid peroxidase and anti-thyroglobulin antibodies were negative. Carbimazole was discontinued and levothyroxine 50 mcg daily were commenced. Pembrolizumab was continued.

Four months later, he presented with lethargy and postural symptoms. A random serum cortisol level was undetectable at <14 nmol/L (reference interval 145 – 619) and ACTH level was inappropriately normal at 6 pg/ml (reference interval 0 – 46). He denied any polyuria, polydipsia, or

symptoms of mass effect. His gonadal function, prolactin and electrolytes were normal. There was marked clinical improvement following hydrocortisone replacement. MRI of the pituitary gland was completely normal. A diagnosis of ICI-induced hypophysitis and thyroiditis was made. He completed the nine cycles of pembrolizumab as initially planned with good cancer response. He remains well on levothyroxine and hydrocortisone replacement.

CONCLUSION

This case illustrates the typical sequelae of ICI-induced endocrinopathy of thyroiditis that occurred earlier and hypophysitis later. All patients in ICI should be monitored at close intervals for hormonal dysfunction and replaced as required.

EP_A131

CUSHING'S DISEASE PRESENTING YEARS AFTER PITUITARY APOPLEXY: A CASE REPORT

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INTRODUCTION/BACKGROUND

Pituitary apoplexy is a life-threatening condition resulting from haemorrhage or necrosis of a pituitary tumour. After an apoplectic event, recurrence of pituitary adenoma from the remaining pituitary tissue is still possible. We report an interesting case who had cushingoid features that manifested several years after her initial episode of pituitary apoplexy.

CASE

A 29-year-old female initially presented with a sudden onset of headache and diplopia. Clinically, she had left cavernous sinus syndrome. A cranial MRI showed a normal-sized pituitary gland with an internal haemorrhage suggestive of apoplexy and no filling defect within the cavernous sinus. She had hypocortisolism post pituitary apoplexy and needed hydrocortisone replacement. Her subsequent yearly MRI surveillance showed a normal-sized pituitary gland with no residual haemorrhage. Three years later, she exhibited typical full-blown cushingoid features of the moon face, facial plethora, proximal myopathy, hirsutism and purplish striae over the abdomen. Hydrocortisone replacement was withheld as repeated morning cortisol was 1002 nmol/L (145.4- 619.4) raising the possibility of endogenous hypercortisolism.

On further evaluation, she had an increased 24-hour urine-free cortisol, and abnormal serum cortisol during both overnight dexamethasone suppression test and low-dose dexamethasone suppression test. Laboratory findings strongly supported ACTH-dependent Cushing's syndrome whereby plasma ACTH was elevated at 20.58 pmol/L (1.6-13.9). However, her brain MRI did not show any evidence of a pituitary adenoma, hence underwent inferior petrosal sinus sampling to further evaluate the likely possibility of Cushing's disease.

CONCLUSION

This case demonstrates the possibility of recurrence Cushing's disease years after an episode of pituitary apoplexy, whereby the residual ACTH-producing adenoma might have grown slowly to form the second tumour. Therefore, long term monitoring is crucial for patients with pituitary apoplexy as they may develop tumour recurrence as well as hormonal deficiencies or excesses over time.

EP_A132

A CURIOUS CASE OF PITUITARY STALK THICKENING – LYMPHOCYTIC INFUNDIBULONEUROHYPOPHYSITIS

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INTRODUCTION/BACKGROUND

Hypophysitis is a rare inflammatory disorder that affects the pituitary gland and infundibulum. Lymphocytic infundibuloneurohypophysitis (LINH), one of the causes of primary hypophysitis, is a rare autoimmune inflammatory process that selectively affects the neurohypophysis and infundibulum, typically presenting with arginine vasopressin deficiency (AVP-D). Magnetic resonance imaging (MRI) with contrast demonstrates thickening of the pituitary stalk, enlargement of the neurohypophysis, or both with homogeneous enhancement. The inflammatory process in LINH can be self-limited and regression can be seen radiologically during follow-up.

CASE

A 22-year-old male presented with sudden onset polyuria and polydipsia in 2016, with clinical and laboratory findings consistent with AVP-D. Brain MRI demonstrated enlargement of the pituitary stalk, measuring 6 mm, and absence of the posterior pituitary bright spot. Other anterior pituitary hormones were normal, except for mildly raised prolactin levels. Investigations for secondary aetiologies were not significant. He was given a trial of glucocorticoid treatment. Serial brain MRI showed a reduction of the

pituitary stalk, but AVP-D persisted, and he was diagnosed with LINH.

The evaluation of a patient with thickening of the pituitary stalk involves assessing the function of both the anterior and posterior pituitary glands and identifying the underlying cause. LINH is characterized by lymphocytic infiltration, leading to the eventual destruction of the pituitary tissue accompanied by varying degrees of pituitary dysfunction. Histopathology remains the gold standard for diagnosis, and definite diagnosis can only be established via pituitary stalk biopsy. Due to the wide range of possible aetiologies, caution and close monitoring are strongly recommended for the treatment of presumed cases lacking histopathologic confirmation.

CONCLUSION

There are no evidence-based guidelines on the management of LINH due to its rarity. The glucocorticoid response rate has been variable. An individual approach is warranted. A conservative medical approach is often used as LINH is often self-limiting, especially when symptoms of mass effect are absent.

EP_A133

SECONDARY HYPERTHYROIDISM PRESENTING WITH MASSIVE STROKE

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INTRODUCTION/BACKGROUND

Hyperthyroidism secondary to pituitary adenoma is rare (TSHoma). It comprises 1-3% of all pituitary adenomas. TSHoma itself does not directly cause strokes; however, the associated hyperthyroidism predisposes patients to cardiovascular complications, including atrial fibrillation and hypertension, thereby increasing the risk of stroke.

CASE

A 54-year-old Malay female complained of sudden-onset right-sided body weakness, aphasia, and confusion. Clinical examination revealed rapid atrial fibrillation (AF) and neurological deficits consistent with a left middle cerebral artery (MCA) territory infarct. CT scan of the brain showed a sellar mass. Initial investigation revealed elevated FT4 levels, with nonsuppressed TSH, prompting further diagnostic workup to confirm secondary hyperthyroidism. Her remaining pituitary functions were normal. Transthoracic echocardiography showed normal ventricular size, mild MR, AR, and moderate TR; however, there was no left ventricular thrombus. Pituitary MRI later showed a 1.9 x 1.9 x 3.2 cm pituitary macroadenoma with suprasellar extension. AF was subsequently controlled with a beta blocker and direct oral anticoagulants (DOACs) were initiated. Hyperthyroidism was managed with octreotide LAR and biochemical euthyroidism was achieved, and the patient showed neurological improvement.

CONCLUSION

TSHoma-associated with stroke is rarely reported. Hyperthyroidism-induced cardiovascular complications are well-documented, including the risk of stroke. Treatment strategies for TSHoma aim to control hyperthyroidism and alleviate associated complications. While surgical resection is the definitive treatment, medical therapy with somatostatin analogues may be considered, as demonstrated in this case.

The management of TSHoma is challenging due to its association with hyperthyroidism-induced cardiovascular complications, and the risk of stroke. Recognizing TSHoma and early intervention may prevent its cardiovascular complications.

EP_A134

CONFRONTING THE GIANT: A CASE REPORT ON THE MANAGEMENT OF RESISTANT PROLACTINOMA

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INTRODUCTION/BACKGROUND

Giant prolactinoma (tumor size more than 4 cm) is extremely rare, even more so in adolescents. Although generally benign, giant prolactinomas are locally aggressive, extending out of the pituitary fossa causing compression to the surrounding structures. While the mainstay of treatment for prolactinoma is medical therapy, lesions of such a large calibre may exhibit resistance to dopamine agonists.

We report a case of a 14-year-old female with a giant prolactinoma complicated with obstructive hydrocephalus which was resistant to cabergoline.

CASE

A 14-year-old female presented with a 4-month history of worsening peripheral vision loss. Neuroimaging revealed a large lobulated solid cystic sellar mass with suprasellar extension measuring 4.3 x 3.7 x 4.1 cm with optic chiasm

compression and obstructive hydrocephalus. Further workup revealed markedly elevated prolactin levels at 36,872 m IU/L suggestive of a giant prolactinoma.

She was started with cabergoline 0.25 mg twice per week which was gradually titrated to 0.5 mg five times per week. However, the prolactin levels only reduced slightly to 31,365 mIUs/L after 2 months of therapy. A craniotomy resection of the tumour was done given medical treatment failure and the mass effect of the lesion. Post-operation histopathological examination confirmed a prolactinoma. Unfortunately, she developed a large cerebral infarct post-operatively and eventually succumbed due to aspiration pneumonia.

CONCLUSION

Giant prolactinoma is a rare condition which is potentially treatable with dopamine agonists; however, cystic macroprolactinomas tend to be resistant to medical therapy. In such patients, surgical resection is often needed. However, a transcranial tumour resection posts higher risks and potential complications. Young patients with giant prolactinoma often require genetic testing for aryl hydrocarbon receptor-interacting protein (AIP) mutation and multiple endocrine neoplasia 1 (MEN1) mutation.

EP_A135

CYCLICAL CUSHING'S DISEASE WITH DISSEMINATED TUBERCULOSIS

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INTRODUCTION/BACKGROUND

Corticotroph adenomas account for the majority of cases of cyclical Cushing's Syndrome (CS). We described a middle-aged female with cyclical Cushing's Disease (CD) complicated with disseminated tuberculosis (TB) infections involving lymph nodes, the brain and the colon.

CASE

A 30-year-old Malay female presented with classical CS symptoms: weight gain of 10 kg, acne eruption, abdominal striae, proximal myopathy, and skin bruising. Her random 4 pm serum cortisol was 1003 nmol/l and 24-hour urine cortisol levels were elevated 3 times above the upper limit. However, a month later, her overnight dexamethasone test (ODST) was suppressed at 38.4 nmol/l with a serum ACTH of 1.8 pmol/L, and her symptoms had resolved. Three months later, she had a recurrence with an unsuppressed low-dose dexamethasone test and elevated 24-hour urine cortisol. She was diagnosed with cyclical CD following high serum ACTH level 20.5 pmol/L (1.6–13.9 pmol/L). Further testing was planned, but she was found to be pregnant. Her disease remained quiescent throughout her pregnancy. Postpartum, her CS symptoms and hypercortisolaemia recurred along with hypokalaemia and prediabetes, though her blood pressure was normal.

Post-partum, she underwent total thyroidectomy and left central lymph node dissection for a suspicious thyroid nodule with thyrotoxicosis. Histopathology revealed left micropapillary thyroid carcinoma with chronic granulomatous changes in the lymph nodes, consistent with TB. Pituitary MRI revealed a pituitary microadenoma measuring 0.7 cm and a tuberculoma in the cerebellum. TB meningitis was confirmed after an MTB GeneXpert test was performed on her cerebrospinal (CSF) fluid and yielded a positive result. CT scan of the abdomen and pelvis showed features suggestive of TB in the gut, and a colonoscopy revealed multiple transmural ulcers with positive MTB PCR results. Anti-TB therapy was initiated, and a multidisciplinary meeting recommended pituitary surgery after the intensive phase of anti-TB therapy.

CONCLUSION

This case illustrates the complexities in managing CD which may be cyclical and further complicated with severe opportunistic infections.

EP_A136

UNVEILING SIADH: A CASE REPORT OF HYPONATREMIA SECONDARY TO ABIRATERONE THERAPY

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INTRODUCTION/BACKGROUND

Hyponatremia is an uncommon complication of abiraterone treatment with an incidence rate of 0.4%-5%. Syndrome of inappropriate antidiuretic hormone secretion (SIADH) as a complication of abiraterone therapy is even rarer, and to our knowledge, this is the first-ever report on abiraterone-induced SIADH in a patient with metastatic hormone-sensitive prostate cancer (mHSPC).

CASE

An 83-year-old Chinese male with newly diagnosed mHSPC presented acutely confused and lethargic with symptomatic hyponatremia (serum sodium of 126 mmol/L) following two weeks of oral abiraterone 1 g daily. The trial of intravenous fluids appeared to worsen hyponatremia, whereby serum sodium dropped further to 117 mmol/L. His vital signs were otherwise stable, with serum potassium, urea, creatinine, glucose, thyroid function and other electrolytes within normal range. His urine sodium was 138 mmol/L, urine osmolality was 259 mOsm/kg, serum osmolality was 259 mOsm/kg, and early morning serum cortisol was 396 nmol/L. Contrast-enhanced CT scan of the brain ruled out intracranial lesions or brain metastasis. Diagnosis of SIADH was made and the patient was put on fluid restriction. Within 4 days, his serum sodium improved to 125 mmol/L. Abiraterone was withheld and was subsequently discontinued.

CONCLUSION

Abiraterone is a selective, irreversible androgen biosynthesis inhibitor used for the treatment of metastatic prostate cancer and it has shown to improve the rate of overall survival. The prevalence of SIADH among patients receiving abiraterone therapy is not well established and the exact mechanism by which abiraterone induces SIADH remains elusive. It is hypothesized that abiraterone's inhibition of CYP17A1, an enzyme crucial for androgen synthesis, may lead to dysregulation of ADH release, subsequently giving rise to SIADH.

Further research is needed to fully understand this relationship. Prompt recognition and management of abiraterone-induced SIADH are crucial to prevent associated complications.

EP_A137

THE MALADIES OF CUSHING SYNDROME

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INTRODUCTION/BACKGROUND

Ectopic ACTH secretion (EAS) occurs in 1.6-6% of cases of small-cell lung carcinoma (SCLC). Diagnosis can be challenging due to a wide variety of clinical manifestations.

CASE

A 58-year-old male smoker presented with acute respiratory distress. He had a 2-month history of worsening dyspnoea, generalized oedema and reduced effort tolerance. At presentation, he was tachypnoeic with an oxygen saturation of 90%, blood pressure of 187/94 mm Hg and heart rate of 103 beats/minute. Examination revealed bilateral lung crepitations with generalized pitting oedema. A CXR showed multiple ill-defined lung masses with pulmonary infiltrates. Laboratory results revealed hypokalaemia (potassium 2.4 mmol/L) with metabolic alkalosis (pH 7.58, HCO 46.9). A CT TAP was consistent with lung malignancy with lymphangitis, carcinomatosis and bone and liver metastases. He was oxygen-dependent and had persistent hyperglycaemia (HbA1c 7.7%) and hypertension with hypokalaemia requiring >3 antihypertensive agents. Despite not being cushingoid-looking and with no hyperpigmentation, ectopic CS was suspected and confirmed with grossly elevated cortisol (>1740 nmol/L) and ACTH level 57.1 pmol/L (1.6- 13.9), non-suppressible by high dose dexamethasone suppression test (cortisol >1750 nmol/L, ACTH 57.9 pmol/L). Lung biopsy confirmed SCLC. Spironolactone and ketoconazole were started, with improvement in BP and metabolic parameters. Cortisol level reduced to 352 nmol/L within 3 weeks of treatment. He was planned for palliative chemotherapy but desaturated further. Therapeutic anticoagulant, IV piperacillin-tazobactam and Pneumocystis pneumonia (PCP) treatment with clindamycin and primaquine were added. PCR for PCP returned as positive later. Due to his poor ECOG score, family opted for conservative treatment and he succumbed later on palliative care.

CONCLUSION

Presence of profound hypokalaemia, hypertension with oedema and new-onset diabetes with lung malignancy should alert the clinicians to possible EAS. The intense hypercortisolism in EAS requires prompt treatment to reduce hypercortisolism and targeted therapy for associated co-morbidities. Early diagnosis is important as EAS confers poorer prognosis to SCLC and is associated with more extensive disease and reduced response to first-line treatment.

EP_A138

XANTHOMATOUS HYPOPHYSITIS PRESENTING WITH PROGRESSIVE HYPOPITUITARISM AND PITUITARY APOPLEXY

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INTRODUCTION/BACKGROUND
Primary hypophysitis is a rare condition characterised by isolated inflammation of the pituitary gland and infundibulum. Among the histopathological subtypes, xanthomatous hypophysitis is rare.

CASE
We describe a 28-year-old female with xanthomatous hypophysitis (XH), its clinical course over 4 years, as well as the transsphenoidal surgery outcome.

She first presented 4 years ago with intermittent headaches and amenorrhea for four months. Investigations showed that she had central hypogonadism (FSH 0.9 IU/L, LH <0.1 IU/L, estradiol 77 pmol/L) and hyperprolactinemia (prolactin 2195 m IU/L). She continued to have central hypogonadism despite prolactin normalisation and was subsequently started on an oral contraceptive pill. A pituitary MRI showed a normal pituitary gland and no stalk lesion. One year later, she developed arginine vasopressin (AVP) deficiency, leading to desmopressin treatment. She was diagnosed with autoimmune hypophysitis, was started on prednisolone and 50 mg of azathioprine daily. However, she developed transaminitis after two months of treatment and it was withheld.

Two and a half years later, she developed central hypothyroidism and central adrenal insufficiency and was started on hydrocortisone and levothyroxine replacement. She was admitted to the ward two months later for pituitary apoplexy, and a repeat MRI showed an enlarging sellar cystic mass with mass effect and increasing T1-weighted hyperintensity within. The Humphrey visual field examination was normal. She underwent transsphenoidal hypophysectomy which revealed a sellar mass with a thickened capsule containing cheesy material. Histopathological findings were consistent with xanthomatous hypophysitis. Three months after surgery, she still had panhypopituitarism, and a repeat MRI revealed a pituitary gland within the sella with no evidence of recurrent disease.

CONCLUSION
Surgery is the treatment of choice for patients with XH who present with pituitary apoplexy. Close follow-up and monitoring of hormonal recovery and disease remission are essential to observe the clinical progression of this rare disease.

EP_A139

FERTILITY IN CONGENITAL ADRENAL HYPERPLASIA: A CASE REPORT

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INTRODUCTION/BACKGROUND
Congenital adrenal hyperplasia (CAH), an autosomal recessive disorder affecting cortisol biosynthesis enzymes, results in virilization in affected females. Fertility challenges are often faced by females with CAH. We present two women with CAH who achieved successful spontaneous pregnancy and their pregnancy outcomes.

CASE 1
Ms. NAS, a 28-year-old Malay female, was diagnosed with 21-hydroxylase deficiency (21-OHD) at birth (presentation: ambiguous genitalia) and underwent corrective surgery at one year of age. She attained menarche at the age of 12 before having secondary amenorrhea for one year at 13 years old. She had normal menstrual cycles while on hydrocortisone 10 mg BD. Pre-pregnancy investigations were as follows FSH 4.04 IU/L, LH 2.22 IU/L, testosterone 2.22 nmol/L, dehydroepiandrosterone Sulphate (DHEA-S) 1.040 umol/L (NR 2.68-9.23). She safely delivered her baby via spontaneous vaginal delivery.

CASE 2
Ms. NN, a 22-year-old female, was diagnosed with CAH at 1 month of life presenting with a salt-losing crisis. She developed precocious puberty at 9 years old due to poor compliance to treatment. She was on triptorelin (Decapeptyl) sc for 2 years until the age of 12. She successfully conceived at the age of 21. Pre-pregnancy, she was treated with T hydrocortisone 5 mg/5 mg/7.5 mg TDS and T fludrocortisone 0.1 mg OD. Her hormonal level preconception were as follows: testosterone <0.1 nmol/L, 17 hydroxyprogesterone (17 OHP) 71.9 nmol/L (elevated), DHEA-S 0.349 umol/L (suppressed). Her pregnancy was uneventful, and successfully delivered her baby surgically after having transverse lie at term.

The differences between the 2 cases (presentation of CAH, the need for genital reconstructive surgery, the nature of puberty) did not impair the fertility potential of these 2 patients during adulthood.

CONCLUSION
Management of CAH during pregnancy is important to ensure successful term delivery with no added pregnancy complications like gestational diabetes or hypertension.

EP_A140

VIRILISING OVARIAN TUMOUR: A TERTIARY CENTRE EXPERIENCE

https://doi.org/10.15605/jafes.039.S1.151

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INTRODUCTION/BACKGROUND
To determine the clinical features, a spectrum of imaging characteristics and histopathologic findings of virilizing tumours of the ovary.

CASE
A 69-year-old female presented with a 1-year duration of increasing hair growth over both arms and androgenic and male pattern facial hirsutism which needed facial hair trimming once a week.
She attained menarche and menopause at the age of 12 and 55 respectively. She was diagnosed with hypertension and Graves' disease at 30 years of age. She was clinically obese with a BMI of 30 kg/m^2, blood pressure 149/84 mmHg, deepening of voice and hirsutism score of 16. There was no clitoromegaly, no proximal myopathy, no cutaneous bruising or abdominal striae. The total testosterone level was >52.05 nmol/L (NR: 0.1-1.6) with normal ACTH, cortisol and DHEA-S. An abdominal CT scan showed a left ovarian mass measuring 7.3 x 7.4 x 5.4 cm with solid components and signs of peritoneal carcinomatosis; however, no calcification within the mass was noted. She underwent primary debulking total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAHBSO), omentectomy and pelvic node dissection.

Histopathological examination demonstrated steroid cell tumour of the ovary. Post-operatively, testosterone levels reduced to normal and showed improvement of alopecia and hirsutism on follow-up 6 months later.

CONCLUSION
In post-menopausal women the appearance of signs of virilization and high testosterone levels should be investigated systematically for underlying malignancy and to determine if the high testosterone levels are of an adrenal or ovarian origin. Failure of recognition will lead to a poor prognosis. In this patient, hematological investigations ruled out an adrenal cause however radiological imaging identified an ovarian etiology.

EP_A141

WHAT TESTOSTERONE CONCENTRATIONS SHOULD WE EXPECT IN HEALTHY MEN?

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INTRODUCTION/BACKGROUND
Older men generally have lower testosterone concentrations compared with younger to middle-aged men. In aging men, lower testosterone concentrations are associated with poorer health outcomes. Understanding the relationship between testosterone and men's health status has implications for defining appropriate testosterone reference ranges to apply in clinical practice.

METHODOLOGY
Studies defining reference ranges for testosterone in selected groups of healthy men and meta-analyses of prospective cohort studies which analysed associations of testosterone with key health outcomes in community-dwelling men were identified. These studies used mass spectrometry to assay testosterone concentrations.

RESULTS
In 124 men aged 21-35 years with normal reproductive function, 95% confidence limits for testosterone were 10.4-30.1 nmol/L. In 1185 non-obese men aged 19-39 years, the range was 9.2-31.8 nmol/L. In 394 healthy men aged 70-89 years, the range was 6.4-25.7 nmol/L. Individual participant data (IPD) meta-analyses of 21,074 men from nine studies showed lower testosterone concentrations in men older than 70 years (with higher luteinising hormone), and in those with higher BMI, or with diabetes or cancer. Extension of these IPD meta-analyses showed non-linear associations of testosterone with all-cause and cardiovascular mortality risk. Men with low testosterone concentrations (<7.4 nmol/L) had higher all-cause mortality.

CONCLUSION
Reference ranges for testosterone in healthy younger to middle-aged men are higher than for healthy older men.

Leydig cell impairment is apparent in men older than 70 years. Age >70 years, BMI, and presence of ill-health need to be considered when interpreting testosterone results. Testosterone concentrations expected in healthy men can also be defined by thresholds below which risks of poorer health outcomes, such as mortality, increase.

EP_A142

A RARE CASE OF CONGENITAL ANORCHIA PRESENTED AS GYNAECOMASTIA IN ADULTHOOD

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INTRODUCTION/BACKGROUND
Congenital anorchia is a rare condition characterised by the absence of testes in a 46,XY individual with a male phenotype. The incidence appears to be 1:20,000 males. The lack of testosterone production will lead to issues with puberty, bone health and fertility.

CASE
A 28-year-old male with no known medical illness presented to us at the age of 21 with absence of secondary sexual characteristics and gynaecomastia since entering pubertal period. A thorough physical examination revealed a male with a height of 171 cm with a BMI of 28.7 kg/m^2 with Tanner 1 pubic hair, and absence of axillary hair and moustache. His male sexual organs were prepubertal and his scrotum was empty. His parents noticed the empty scrotum since his neonatal period but did not seek further medical attention. An MRI was done revealing a micropenis without visualised testes in the abdomen or pelvis. Further hormonal panels showed primary hypogonadism with a very low testosterone level of 0.62 nmol/L (Normal range: 8.6-29). Thyroid function and prolactin were normal. Chromosomal analysis revealed a 46, XY karyotype. He was then started with intramuscular testosterone injection at 22-years-old.

CONCLUSION
The most common cause of congenital primary hypogonadism is sex chromosome aneuploidy, present in Turner syndrome and Klinefelter syndrome. Studies have shown that about 4.5 percent out of 6000 cryptorchid children are anorchid, and 14 percent of them have absence of bilateral testes. A hypothesis of vascular occlusion in early foetal development leading to atrophy of functional testes was made. It is supported by findings of a fibrotic node at the end of vasa differentia in anorchid patients. Congenital anorchia is rarely seen among the male population. It is congenital and presents late to clinical setting if missed during childbirth. Testosterone replacement is essential for secondary sexual characteristics and bone health.

EP_A143

HURTHLE CELL THYROID CARCINOMA (HTC): A RARE INCIDENCE OF BRAIN METASTASIS

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INTRODUCTION/BACKGROUND
Oncocytic or Hurthle cell thyroid carcinoma is a rare type of carcinoma which occurs in 5% of the population with known thyroid carcinoma. Metastasis to the brain is even rarer with 3% of follicular subtypes reported.

CASE
A 60-year-old female presented with left-sided hemiparesis and slurred speech. She exhibited full consciousness. A cranial CT showed a right frontal lobe intra-axial lesion causing obstructive hydrocephalus. Subsequent MR revealed an enhancing hypointense lesion at the frontoparietal lobe which is suggestive of a glioma. She was referred to the neurosurgical outpatient clinic; however, she experienced a seizure episode and ended up in the emergency department. She was subjected to emergency right craniectomy and tumour excision. Histopathological examination of brain tissue revealed a metastatic carcinoma consistent with a primary thyroid origin. Surveillance CT post-operatively revealed a right thyroid lobe lesion. A biopsy of the right thyroid nodule was performed during the tracheostomy procedure. Histopathological findings were consistent with HTC. A delayed thyroid ultrasound revealed a TIRADS 4 hypoechoic lesion in the right lower pole (1.7 x 2.1 cm). Otherwise, she was clinically and biochemically euthyroid. She underwent whole-brain radiotherapy and was scheduled for total thyroidectomy.

CONCLUSION

This patient was primarily investigated for glioma, but the histopathology report changed the course of investigation and treatment. Histologically, the oncocytic cell is a follicular "derived" thyroid cell which exhibits abundant granular eosinophilic cytoplasm and is positive for TTF1 and thyroglobulin immunostain. Clinical presentation varies from capsular to vascular and/or distant lymph node invasion, and metastatic spread. In this case, we describe the challenges encountered in diagnosing HTC. Brain metastasis of HTC is rare.

The unique presentation as a primary brain tumour with no thyroid nodule or neck swelling delayed the diagnosis. The prognosis of such cases is worse in a high-grade and poorly differentiated disease.

EP_A144

IODINE-131 RESISTANCE IN A CASE OF TOXIC ADENOMA REQUIRING MULTIPLE COURSES OF RAI-131

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INTRODUCTION/BACKGROUND

Hyperthyroidism is a state of hyperactive thyroid gland secreting excessive thyroid hormone causing a constellation of symptoms to multiple organs and systems. Hyperthyroidism can be caused by an autoimmune condition (Graves' disease), inflammation of the thyroid (thyroiditis), or due to functioning thyroid nodules (hot nodule or toxic multinodular goitre).

We report a case of toxic adenoma, who received Iodine-131 four times with a cumulative dose of 69 mCi; however, persistent hyperthyroidism required additional treatment with ATD. Subsequently, she underwent left hemithyroidectomy.

CASE

A 29-year-old female was referred to the nuclear department for radioactive iodine-131 (RAI-131) therapy. She received her first RAI-131 with 15 mCi in September 2020. Due to persistent hyperthyroidism, she received another RAI-131 with 15 mCi in April 2021. Her third RAI-131 with 21 mCi done in January 2022 and fourth RAI-131 with 18 mCi was done in June 2022 due to persistent hyperthyroidism requiring ATD. She had Tc-99 m pertechnetate thyroid uptake scan done with scan findings suggestive of toxic multinodular goitre in left thyroid lobe. She was planned for another RAI-131, however she refused.

Left hemithyroidectomy done in September 2023 with HPE reported as nodular hyperplasia with dominant nodule and cystic degeneration. She developed transient hypothyroidism after surgery requiring levothyroxine and subsequently euthyroidism without any medication.

CONCLUSION

RAI-131 is relatively safe and easy to administer making it the treatment of choice for many causes of hyperthyroidism. Around 10% of patients would require subsequent dose of RAI-131. Failure of RAI-131 for treatment of hyperthyroid is rare, mainly due to inadequate preparation. Some patients have delayed response to RAI-131, up to years after iodine treatment.

EP_A145

NAVIGATING THE CHALLENGES OF UNCONTROLLED THYROTOXICOSIS

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INTRODUCTION/BACKGROUND

Thyrotoxicosis is a condition characterized by the excessive production of thyroid hormones. Commonly presented as Graves' disease, other aetiology includes toxic multinodular goitre or subacute thyroiditis. Therapeutic approaches depend on the aetiology which includes anti-thyroid medications, radioactive iodine, or surgical intervention. We highlight 2 cases with different aetiologies of thyrotoxicosis that remained uncontrolled despite medical therapy and necessitated surgical intervention.

CASE 1

23-year-old female with diffuse goitre that was progressively increasing in size since the age of 15. She presented with classic thyrotoxicosis symptoms. She was confirmed to have Graves' disease and was treated with carbimazole therapy. However, she remained uncontrolled after 2 years despite high dose carbimazole therapy (90 mg/day), lithium (600 mg/day), prednisolone (20 mg/day) and cholestyramine. She finally relented to surgical intervention as her definitive treatment. Her perioperative optimization was equally challenging and the addition of Lugol's iodine a week prior to surgery brought her free T4 levels below 20 pmol/L. Through multidisciplinary collaboration between endocrinologists, surgeons and anaesthetists, she had a successful total thyroidectomy.

CASE 2
A 58-year-old female presented with a 7-year history of toxic multinodular goitre before her referral to our centre. She had been receiving fluctuating doses of carbimazole and her TFT remained uncontrolled. She also had retrosternal thyroid extension with mass effect. Her TFT remained uncontrolled despite carbimazole 25 mg/day, and coupled with compressive symptoms, navigated us towards definitive surgical intervention. Timely Lugol's iodine treatment optimized her TFT preoperatively and she successfully underwent total thyroidectomy.

CONCLUSION
Both cases highlighted the difficulty in managing thyrotoxicosis and surgical intervention was the best definitive treatment. Perioperative preparation is often challenging requiring multimodal approach to lower free T4 to acceptable levels prior to definitive thyroidectomy.

EP_A146

GAZE BEYOND THE BOUNDARIES: TRANSCENDING THE HURDLES IN MANAGING GRAVES OPHTHALMOPATHY IN A DISTRICT HOSPITAL SETTING

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INTRODUCTION/BACKGROUND
Graves ophthalmopathy (GO) is an autoimmune condition characterized by inflammation and tissue expansion within the orbit. The pathogenesis of GO involves complex interactions between autoantibodies, inflammatory mediators, and orbital fibroblasts. GO affects both the soft tissues and the extraocular muscles, leading to a range of ocular manifestations, including proptosis, eyelid retraction, diplopia, and in severe cases, vision loss.

CASE
An 18-year-old female, a passive smoker with no significant medical history, presented to a district hospital with sudden onset of exophthalmos, eye discomfort, and double vision persisting for three weeks. She has a strong family history of thyroid disease. Physical examination revealed the patient to be in a state of thyroid storm and a clinical activity score (CAS) of 3. The ophthalmologic evaluation revealed bilateral proptosis, conjunctival injection, and restricted extraocular movements consistent with GO. Laboratory investigations confirmed hyperthyroidism. Orbital CT demonstrated enlargement of the extraocular muscles and expansion of orbital fat, further supporting the diagnosis of GO. The patient was promptly initiated on treatment for thyroid storm, including antithyroid medications and supportive care to stabilize thyroid function. Systemic corticosteroids and lubricating eye drops were administered to alleviate ocular symptoms. With aggressive medical management, the patient's thyroid storm resolved, and ocular symptoms showed significant improvement throughout treatment.

CONCLUSION
This case highlights the importance of considering GO in the differential diagnosis of patients presenting with exophthalmos and ocular symptoms, especially when complicated by thyroid storm. Early recognition and collaborative management are essential for optimizing outcomes in such cases.

EP_A147

OUTCOME OF THYROID STORM CASES IN 2023 AT HOSPITAL TELUK INTAN, MALAYSIA

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INTRODUCTION/BACKGROUND
Thyroid storm, also known as thyrotoxic crisis, is an acute, life-threatening condition induced by the excessive release of thyroid hormones in individuals with thyrotoxicosis that present with systemic involvement. Thyroid storm mortality is estimated to be 8 to 25% despite modern advancements in its treatment and supportive measures. It is now an uncommon condition because of earlier diagnosis and treatment of thyrotoxicosis. Thyroid storm is commonly associated with Graves' disease, but it may occur in patients with toxic nodular goitre or any other cause of thyrotoxicosis. Thyroid storm may be precipitated by a number of factors including intercurrent illness, especially infections.

METHODOLOGY
This is a retrospective audit which included all patients who were admitted for thyroid storm from January 2023 to December 2023. The data was collected from clinical notes and electronic medical records.

RESULTS
A total of 23 subjects were included in this audit, which predominantly were female at 87%. Median age of the study population is 40 with the youngest subject aged 13 and the oldest was aged 64. The mean Burch and Wartofsky score was 47. For identifiable causes of storm, 13 out 23 subjects

(56%) were due to infection, mostly pneumonia, followed by 22% (5 subjects) due to undiagnosed hyperthyroidism and 17% (4 subjects) were due to defaulted treatment. There were two mortalities (8.7%). Both mortalities required intubation and presented with pulmonary oedema on arrival, and had Burch and Wartofsky scores of 50 and 60 respectively. Mortality rate for thyroid storm in 2023 was 8.7%.

CONCLUSION

Death from thyroid storm is not as common as in the past owing to its prompt recognition and aggressive treatment in an intensive care unit, but mortality is still approximately 10-25%. Early detection and understanding of hyperthyroidism symptoms among the public are critically important. Analysing the outcomes of thyroid storms at Hospital Teluk Intan highlights the need for increased public education to prevent future deaths caused by thyroid storms.

EP_A148

SOMEBODY CALL 9-1-1: HYPOTHYROIDISM MIMICKING WELLENS SYNDROME

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INTRODUCTION/BACKGROUND

Wellens syndrome is characterized by a distinctive pattern of electrocardiographic (ECG) pattern, specifically deep symmetrical inverted T-waves or biphasic T-waves in leads V2-V3. This condition is highly indicative of critical stenosis in the left anterior descending artery (LAD) which poses a significant risk of mortality, hence its nickname "widow maker."

CASE

A 38-year-old police officer was under Endocrine clinic follow-up for Graves' disease. He underwent radioactive iodine (RAI) treatment in June 2022 and subsequently did not require thyroxine replacement for 1 year as he was clinically and biochemically euthyroid. During a clinic review in August 2023, he reported symptoms of cold intolerance, weight gain and reduced effort tolerance which hindered his ability to do his daily work. Blood investigation showed a free T4 of 7 pmol/L, and TSH was 13.6 m IU/L. The patient was diagnosed with overt hypothyroidism and was given thyroxine replacement.

Electrocardiogram (ECG) done revealed deep symmetrical T-wave inversions in leads V2-V5, indicative of Wellens Type B pattern, which carries a 97% specificity for LAD occlusion. The patient was referred to cardiology service and underwent r an early coronary angiogram, which surprisingly revealed normal coronaries. Additionally, he did not exhibit any features of common Wellen mimics such as pulmonary embolism, pancreatitis, heart failure or acute stroke. He denied consuming alcohol or using any illegal stimulant substances. Blood analysis done revealed normal electrolytes. Patient was initiated on treatment with 12.5 mcg of L-Thyroxine tablets daily, with the dose topped up every 4 to 6 weeks. Upon subsequent clinic reviews, patient's initial symptoms have resolved, enabling him to resume his job without any difficulties.

CONCLUSION

This case highlights overt hypothyroidism as a mimicker of Wellens Syndrome. After promptly excluding critical coronary artery disease, it is imperative to evaluate and treat other potential causes of Wellens Syndrome or its mimics.

EP_A149

AUTOIMMUNE/INFLAMMATORY SYNDROME INDUCED BY ADJUVANTS (ASIA): POST-VACCINATION SUBACUTE THYROIDITIS

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INTRODUCTION/BACKGROUND

Autoimmune/inflammatory syndrome induced by adjuvants (ASIA), also known as Shoenfeld's syndrome, encompasses a spectrum of autoimmune conditions and responses triggered by exposure to substances with adjuvant activity such as vaccines.

CASE

A healthy 43-year-old male with no known medical illness or family history of thyroid disorder developed painful thyroiditis after receiving his influenza vaccination. He undergoes regular health checkups, which have consistently shown normal results, including previous thyroid function tests (TFT). Patient received his influenza vaccine (Vaxigrip tetra) and developed left sided neck pain and severe thyrotoxicosis symptoms after 1 week. Blood investigation done showed free T4 25 pmol/L, TSH <0.01 m IU/L and a raised CRP. Physical examination revealed a tender diffuse

goitre and fine tremors. Ultrasonography of the neck was done with the impression of subacute thyroiditis. These findings fulfilled the Japanese Thyroid Association (JTA) criteria for subacute thyroiditis. A Tc-99m Pertechnetate also confirmed the findings of thyroiditis. The patient was commenced on oral prednisolone 25 mg daily (0.5 kg/BW/day) with a tapering regimen over 2 weeks. He also received oral celecoxib 200 mg daily for 5 days and oral propranolol 20 mg daily. He gradually improved upon the 2-week clinic review and all medications were discontinued. Upon re-evaluation at 3 months, thyroid function tests normalized, and the thyroid ultrasound displayed the resolution of thyroiditis characteristics, accompanied by amelioration of all symptoms.

CONCLUSION

Subacute thyroiditis is an inflammatory thyroid condition characterized distinctly by painful enlargement of the thyroid. Transient hyperthyroidism is a hallmark of subacute thyroiditis where the inflamed thyroid gland releases unregulated excessive thyroid hormone into the bloodstream, leading to thyrotoxicosis symptoms. In this case, we treated our patient with a short-term 2-week combination of steroids + NSAIDs which showed non-inferior efficacy to the traditional long-term steroids (4-8 weeks).

EP_A150

HUMAN CHORIONIC GONADOTROPHIN (HCG) AND HYPERTHYROIDISM: RARE BUT PARALLEL CAUSE

https://doi.org/10.15605/jafes.039.S1.161

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INTRODUCTION/BACKGROUND

Choriocarcinoma is a hCG-producing malignancy, with the beta subunit being structurally similar to TSH, allowing it to bind to TSH receptors on thyroid follicular cells and at high levels to elicit biochemical hyperthyroidism.

CASE

We report a case of choriocarcinoma-induced hyperthyroidism in a 29-year-old female. She was initially admitted for breathing difficulties and was found to have a posterior mediastinal mass, which was later histologically confirmed to be choriocarcinoma with B-HCG levels of 466,511 that peaked to 825,316. Thyroid functions measured shows TSH of 32.07, T4 of 0.009 with negative thyroid antibodies (Anti Thyroid Peroxidase <9, Anti Thyroglobulin 11.9). Ultrasonography showed bilateral thyroid nodules, ACTR TR3 (1.5 cm), however, staging CT did not pick up an overt goitre. Patient was started on carbimazole and planned for chemotherapy with thyroid function monitoring.

CONCLUSION

TSH and Beta-HCG are highly homologous and can cross-link to produce elevated thyroid hormone levels. The prevalence of hyperthyroidism in choriocarcinoma is not known; however, prolonged exposure to high HCG levels is required for it to occur. It is generally accepted that 25,000 IU/L of HCG is equivalent to 1 mU/L of TSH activity, with suggestions to measure thyroid function in patients with HCG >50,000 IU/L. Patients with symptomatic hyperthyroidism are treated with antithyroid drugs and the primary choriocarcinoma is treated with chemotherapy. Reduction or normalisation of the beta hCG levels quickly induces euthyroidism. Beta-HCG-induced hyperthyroidism is rare; however, with high levels found in choriocarcinoma, suspicion of concurrent hyperthyroidism should be raised. Patients with HCG-secreting tumours should be evaluated for hyperthyroidism and may benefit from treatment until the underlying cause is treated.

EP_A151

DOUBLE WHAMMY: CIRCULATORY COLLAPSE AND LIVER DYSFUNCTION IN THYROID STORM

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INTRODUCTION/BACKGROUND

Liver dysfunction is not an uncommon association in patients presenting with thyroid storm and could limit the treatment armamentarium. Circulatory collapse precipitated by the use of long-acting non-cardioselective beta-blockers in certain groups of patients can complicate the course of the disease.

CASE

We report three cases of thyroid storm with circulatory collapse and ischemic hepatitis complicating the use of beta-blockers and thionamides. All were females in their 40's. Two presented with rapid atrial fibrillation (ventricular rate 158- 196 per minute) and biventricular failure, and one with acute pulmonary oedema. All developed hypotension required inotropic support; two after beta-blocker and one after intubation. Their free T4 was 53.6 pmol/L to 74.3 pmol/L, Burch-Wartofsky scores were 60-95. All received ventilatory support and were treated for

sepsis. They received thionamides, glucocorticoid, Lugol's iodine, antiarrhythmic and one received cardioversion. All developed ischemic hepatitis with transaminases increased from the initial 2-7 x to 10-80 x upper limit of normal, and two had coagulopathy. Thionamide dose was reduced in two and withheld temporarily in one. Cholestyramine was added as an adjunct for all. All responded to therapy. Two were discharged with carbimazole and beta-blocker. Unfortunately, one succumbed despite initial improvement due to hospital-acquired infection.

CONCLUSION

Although beta blockers play an important role in the management of thyroid storm, caution should be exercised due to its potential life-threatening side effect especially in the presence of clinical or subclinical thyro-cardiac disease. Lugol's iodine and cholestyramine are useful adjuncts in the presence of severe liver dysfunction when choices of antithyroid drugs are limited.

EP_A152

A CHALLENGE IN MANAGING THYROID STORM WITH CONCURRENT PERFORATED GASTRIC ULCER PATIENT

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INTRODUCTION/BACKGROUND

Thyrotoxic crisis can be fatal if not treated promptly. Individuals with severe thyrotoxicosis who lack a functional gastrointestinal system present an uncommon but significant therapeutic challenge with a high fatality rate. We describe a case of Graves' disease in a thyroid storm with a concurrent perforated gastric ulcer.

CASE

A 36-year-old male presented to the emergency department with acute abdominal pain and vomiting. He also had heat intolerance and significant weight loss. He was borderline hypotensive, tachycardic with a regular pulse, and had generalized abdominal guarding. Chest radiograph showed air under the diaphragm. Thyroid function test (TFT) confirmed thyrotoxicosis with Thyroid Stimulating Hormone (TSH) <0.008 m IU/L and free T4 (FT4) 64.32 pmol/L. He was scheduled for an emergency laparotomy for a perforated gastric ulcer, thus contraindicated to taking anything by mouth. We gave him 200 mg of intravenous hydrocortisone. We did not administer an antithyroid drug because our centre did not have any intravenous or per-rectal antithyroid drug preparation. Post-operatively, his condition deteriorated, and he developed rapid atrial fibrillation which required inotropic support and synchronised cardioversion. After he was permitted to sip fluids for medication, he was started on oral propylthiouracil (PTU) and Lugol's iodine in addition to regular intravenous hydrocortisone. Following this, his general condition and thyrotoxic status improved. Upon discharge, he received oral carbimazole and propranolol.

In the 2-month follow-up, he was clinically euthyroid. His anti-thyroglobulin receptor antibody level was elevated, supporting the Graves' disease diagnosis. His TFT improved, with TSH <0.008 m IU/L and FT4 17.40 pmol/L. We further titrated down his oral carbimazole until the next appointment.

CONCLUSION

Managing a thyroid storm with concurrent perforated gastric ulcer is challenging due to limited antithyroid options other than oral medication and high mortality rates. The attending physician should collaborate with the surgical team to determine the optimal timing for oral antithyroid medication to manage the thyrotoxic crisis.

EP_A153

PITFALLS IN THE DIAGNOSIS OF AMIODARONE-INDUCED THYROTOXICOSIS: A CASE SERIES

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INTRODUCTION/BACKGROUND

One of the lesser known but unique mechanisms of action of amiodarone is its ability to inhibit the 5'deiodinase enzyme that converts T4 into the active T3. Inadvertently, this results in an elevated T4 and a lower T3 which suppresses pituitary TSH, giving the impression of a classic thyrotoxicosis. Only when this peripheral effect is overcome by the explosive release of T4 and T3 that they begin to manifest as amiodarone-induced thyrotoxicosis (AIT).

CASE
We reviewed the last 11 patients who were treated as presumed AIT in our institution who had an elevated fT4 >22 pmol/L and suppressed TSH <0.27 m IU/L at the time of diagnosis. All of them were treated with carbimazole whilst one was started with dexamethasone. However, when the heart rate, symptoms and signs of thyrotoxicosis as well as fT3 levels were analysed, 8 out of the 11 patients were deemed to have demonstrated harmless peripheral or physiological effects of amiodarone which did not require any active intervention. Only one out of the 8 patients had a heart rate above 70/minute at the time of diagnosis notwithstanding the fact they were on low doses of beta-blockers (2.5-5 mg of bisoprolol). Three out of the 8 patients had low fT3 whilst the remaining 5 had normal levels of fT3. Out of the 3 who had true AIT, two were treated as type 2 AIT and started on prednisolone whilst the third was treated as type 1 AIT and managed with carbimazole.

CONCLUSION
One of the pitfalls in managing AIT is the failure to recognise the peripheral effect of amiodarone which produces high T4 and suppressed TSH. Only by analysing the T3 level whilst assessing the heart rate and symptomatology will we be able to discern this phenomenon from that of AIT.

EP_A154

A UNIQUE ENCOUNTER OF PAPILLARY THYROID CANCER AND HODGKIN LYMPHOMA IN TANDEM

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INTRODUCTION/BACKGROUND
Papillary thyroid carcinoma (PTC) comprises the largest proportion of differentiated thyroid carcinoma cases. A notably uncommon scenario involves the simultaneous manifestation of PTC and Hodgkin's lymphoma as distinct primary malignancies.

CASE
A 35-year-old female with no history of radiation exposure presented with painless neck swelling for one year. Thyroid ultrasonography revealed 2 hypoechoic lesions over the left lobe measuring 1.8 x 1.6 cm and 0.7 x 0.9 cm (TIRADS 4) and the right lobe measuring 0.4 x 0.4 cm (TIRADS 1) with multiple prominent cervical lymph nodes. Her thyroid profile was normal. Cervical lymph node biopsy was performed and result suggestive of metastatic papillary thyroid carcinoma. She subsequently underwent total thyroidectomy with modified radical neck dissection. Histopathological examination confirmed multifocal (>5 foci) classical variant papillary thyroid carcinoma, with the largest nodule measuring 17 mm, demonstrating lymphovascular involvement and regional nodal metastasis. According to American Thyroid Association (ATA) guidelines, this case was stratified as high recurrence risk. An unexpected diagnosis of nodular sclerosis classical Hodgkin lymphoma was made during lymph node dissection. Bone marrow assessment ruled out lymphomatous involvement and her computed tomography neck, thorax, abdomen, and pelvis revealed extensive bilateral supraclavicular mediastinal and abdominal lymphadenopathy. Therefore, stage 3 Hodgkin lymphoma was diagnosed. After six cycles of escalated BEACOPP chemotherapy for Hodgkin lymphoma, her positron emission tomography (PET) scan revealed no active lymphoma and resolved thyroid bed issues. She is on TSH suppression with 100 mcg of Levothyroxine daily and plans for radio-ablation therapy. Thyroid function, calcium, and parathyroid hormone levels are all normal.

CONCLUSION
This case highlights the rarity of synchronous papillary thyroid carcinoma and Hodgkin's lymphoma. Thorough investigations are crucial to confirm both pathologies and prioritizing treatment becomes essential. Meta-analysis shows that delaying radio-ablative iodine treatment does not impact long-term overall survival in differentiated thyroid cancer. Therefore, lymphoma treatment takes precedence in this case.

EP_A155

DELAYED-ONSET AMIODARONE-INDUCED THYROTOXICOSIS

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INTRODUCTION/BACKGROUND
Amiodarone is a commonly used antiarrhythmic drug for treatment of refractory tachyarrhythmias. However, its use can lead to development of amiodarone-induced thyrotoxicosis (AIT). AIT is classified into type 1, a form of iodine-induced hyperthyroidism, and type 2, which is a

drug-induced destructive thyroiditis. AIT can be difficult to treat because of the long half-life of amiodarone. We describe a patient who developed AIT 15 months following cessation of amiodarone.

CASE

A 58-year-old male presented with sudden onset palpitations and hand tremors. He had history of ventricular tachycardia, for which he had undergone Implantable Cardiac Device (ICD) insertion. He had been on amiodarone for two years, from September 2021 to February 2023, following which the medication was discontinued. At the time of admission, amiodarone had been stopped for 15 months. On examination, he was tachycardic with heart rate of 150/min. The electrocardiogram (ECG) showed sinus tachycardia with spontaneous ICD shock. Thyroid function test indicated TSH <0.08 m IU/L (0.35-4.95), FT3 11.8 pmol/L (2.9-4.9), and FT4 >64 m IU/L (9.01-19.05). T4/T3 ratio was >4. Considering the patient's history of amiodarone use, a diagnosis of AIT was established. The patient was prescribed carbimazole 30 mg daily and fT4 remained >64 m IU/L after 1 week. Prednisolone 40 mg daily was added to treat mixed AIT 1 and 2. Thyroid ultrasonography revealed heterogeneous echogenicity in both thyroid lobes, with no focal lesion. Doppler study demonstrated a normal pattern of vascularity. The thyroid peroxidase antibody was 185 IU/L (<35) and the patient is currently awaiting thyroid scintigraphy.

CONCLUSION

The differentiation between type 1 and type 2 AIT can be challenging and indistinct, and some patients exhibit mixed forms of AIT. In such cases, combination therapies are often employed. It is critical to emphasize the importance of maintaining a high index of suspicion for AIT, irrespective of treatment duration or the time elapsed after discontinuation of amiodarone, due to the prolonged half-life of the drug.

EP_A156

METASTATIC MEDULLARY THYROID CARCINOMA DESPITE PROPHYLACTIC TOTAL THYROIDECTOMY IN MULTIPLE ENDOCRINE NEOPLASIA (MEN) 2A

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INTRODUCTION/BACKGROUND

Medullary thyroid carcinoma (MTC) is a rare neuro-endocrine tumour originating from parafollicular cells. Seventy five percent of MTC occur in a sporadic form while 25% are hereditary and associated with Multiple Endocrine Neoplasia (MEN) type 2. As the age of onset of MTC and penetrance of MTC in MEN2 varies by subtype, prophylactic thyroidectomy is recommended for patients with the highest risk of pathogenic variants. We describe a patient with MEN2a with metastatic MTC 10 years following prophylactic thyroidectomy.

CASE

A 29-year-old female was diagnosed with MEN2a at the age of 15 years. The diagnosis was made through genetic screening after her mother was diagnosed with the same condition with RET proto-oncogene mutation. She underwent prophylactic thyroidectomy at 16 years old. Her tumour markers, calcitonin and carcinoembryonic antigen (CEA) were within the normal ranges. She had no loco-regional recurrence during serial follow-up. However, her tumour markers were noted to increase 10 years after surgery. CEA doubling time was 19 months indicating progressive disease. Markedly elevated serum calcitonin at 783 pg/ml (<7.6) indicated high tumour burden and likely metastases. Neck ultrasonography did not reveal any disease recurrence. Serial CT scan of the abdomen demonstrated multiple liver lesions suspicious of metastases. Ga-68 DOTATATE and F-18 FDG PET-CT scan showed multiple non-somatostatins nor FDG-avid liver lesions. There were FDG-avid cervical lymph nodes suspicious of nodal metastases. Histopathologic analysis of the liver biopsy specimen confirmed metastatic MTC. She is planning for a multi-targeted tyrosine kinase inhibitor as systemic therapy for metastatic disease.

CONCLUSION

Management of MTC in the context of MEN2a is challenging and advances in molecular diagnosis and risk stratification systems have led to better individualized treatment and follow-up strategies. Prophylactic thyroidectomy as early

as 6 months for the highest risk and before 5 years old for other high-risk codon mutations is crucial in reducing the risk of micro-metastases.

EP_A157

SIGHT-THREATENING ACTIVE GRAVES' OPHTHALMOPATHY WITH NEWLY DIAGNOSED HEPATITIS B

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INTRODUCTION/BACKGROUND

Graves' Ophthalmopathy (GO) is an orbital autoimmune disease that is the most frequent extrathyroidal expression of Graves' disease. Full-blown disease is associated with disfiguring features (exophthalmos, stare), inflammatory signs and symptoms, ocular dysfunction (diplopia), and rarely, visual loss due to compressive Dysthyroid Optic Neuropathy (DON). The prevalence of GO in Malaysia was 34.7%.

CASE

A 66-year-old Orang Asli female, active smoker with underlying type 2 diabetes, hypertension and dyslipidaemia presented to us in March 2024 with complaints of eyesore and redness for 3 months. In addition, she had photophobia, insomnia, and intermittent headache for 1 month. She had exophthalmos and DON with a clinical activity score (CAS) of 6. Her thyroid stimulating hormone (TSH) level was 40 IU/L. She was started on oral carbimazole 30 mg once a day. In view of sight-threatening DON, she was commenced on high-dose intravenous methylprednisolone 1 gm daily for 3 days, subsequent tapering dose of 500 mg weekly for 6 weeks, and then 250 mg weekly for another 6 weeks. However, her Hepatitis B surface antigen (HBsAg) was reactive and she had mild transaminitis which were relative contraindications for corticosteroid pulse therapy. She was co-managed with the Gastroenterology team and started on oral tenofovir 300 mg once daily. Post IV methylprednisolone, her eye signs and vision had clinical improvement. Her liver function tests remained stable.

CONCLUSION

This case highlights the challenges in managing severe sight-threatening active Graves' ophthalmopathy in a patient with newly diagnosed hepatitis B. Prompt treatment is crucial to prevent further deterioration of her eye condition.

EP_A158

A LETHAL CASE OF SEVERE CARBIMAZOLE-INDUCED AGRANULOCYTOSIS

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INTRODUCTION/BACKGROUND

Agranulocytosis is a severe complication of carbimazole, the primary drug for treating hyperthyroidism. It is rare with an incidence rate of 0.3–0.6% and mortality rate of 21.5%. Onset may develop within 7 days of initiation of anti-thyroid drug therapy. This case report highlights the deleterious effect of carbimazole-induced agranulocytosis in an elderly female.

CASE

A 70-year-old female with newly diagnosed hyperthyroidism (baseline TSH: 0.002 m IU/L, free T4: 58 pmol/L) was initiated on carbimazole 30 mg once daily at a health clinic. After approximately one month on carbimazole, she developed fever, sore throat, and multiple oral ulcers. On examination, she exhibited a spiking temperature of 39.4°C, injected throat, multiple oral ulcers over the hard palate, tongue, and lower lip, and a diffuse goitre. She had leucopoenia with total white blood cell count of 1.0, with immeasurable absolute neutrophil count (ANC) and no blast cells. Repeat TSH was 0.003 m IU/L and fT4 was 39.55 pmol/L. Chest radiograph showed consolidation over the right lower lung zone. Initial treatment included intravenous piperacillin-tazobactam, subcutaneous granulocyte colony-stimulating factor (G-CSF) 300 mcg daily, cholestyramine, Lugol's iodine, and propranolol. Due to the deterioration in her clinical condition, we promptly escalated her antimicrobials to meropenem, micafungin and increased her G-CSF dosing to 300 mcg two times a day. Her ANC remained at 0.01-0.02 (10^9/L). Despite treatment escalation, she succumbed to severe sepsis after 8 days of admission.

CONCLUSION

The primary treatment for carbimazole-induced agranulocytosis involves discontinuing the offending drug and administering intravenous broad-spectrum antibiotics. G-CSF may be used to expedite haematological recovery. Clinical vigilance is crucial when initiating carbimazole, especially in high-risk patients such as the elderly and those receiving high doses initially, by conducting early repeat blood investigations. This approach enables early intervention to mitigate adverse outcomes and ensure a favourable prognosis.

EP_A159

SHIFTING SPECTACLE OF THYROID ANTIBODIES: A UNIQUE PRESENTATION

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INTRODUCTION/BACKGROUND
Graves' disease typically manifests with hyperthyroidism. However, the switch between TSH receptor-stimulating antibodies (TSAb) and TSH receptor-blocking antibodies (TSB Ab) is uncommon. We discuss three patients with Graves' disease who switched between hypothyroidism and hyperthyroidism throughout the course of their disease.

CASE
Case A involves a 44-year-old female with initial diagnosis of hyperthyroidism. She was treated with carbimazole for two years and remained euthyroid for a few years without medication. Four years following the diagnosis, she developed overt hypothyroidism requiring levothyroxine. She remained hypothyroid for nine years until her TSH levels trended towards the lower end, hence, she was restarted on carbimazole. She had elevated TRAb [2.0 IU/L, N: <1.75] and anti-TPO [890 IU/ml, N:<9] at screening. Her neck ultrasound showed a goitre with a solitary left thyroid nodule.

Case B involves a 68-year-old male diagnosed with hyperthyroidism [FT4: 140 pmol/L, TSH:0.008 m IU/L]. Twelve months following diagnosis, he developed overt hypothyroidism while on low dose carbimazole and eventually required levothyroxine. Thyroid antibodies were elevated [TRAb 37 IU/L and anti-TPO 534 IU/ml]. His neck ultrasound revealed a small thyroid nodule with benign features.

Case C is a 43-year-old female who presented with overt hypothyroidism [Ft4 9 pmol/L,TSH 95.75 m IU/L] and was treated with levothyroxine. Initial antibodies were elevated [anti-TPO 235 IU/ml, anti-Tg 104.8 IU/ml]. Three years following diagnosis, her TSH levels trended towards the lower ranges and eventually showed overt hyperthyroidism [FT4 28, TSH <0.008]. She was commenced on oral carbimazole. Repeat antibodies were elevated [anti-Tg 30 IU/ml, anti-TPO 218.25 IU/ml, TRAb 23.8 IU/L]. Her neck ultrasound showed multiple subcentimetre thyroid nodules.

CONCLUSION
Graves' disease is characterized by the presence of TRAb, which can exhibit either TSAb or TSB Ab activity. Treatment with anti-thyroid drugs (ATD) such as carbimazole may further trigger the switch to hypothyroidism. Therefore, close monitoring and follow-up are crucial for these patients.

EP_A160

A RARE CASE OF THYROTOXICOSIS PRESENTING AS HYPERBILIRUBINAEMIA

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INTRODUCTION/BACKGROUND
Hyperthyroidism affects multiple body systems, including the nervous, cardiovascular, gastrointestinal, and hepatobiliary systems. Presentation of severe cholestatic jaundice in thyrotoxicosis, although uncommon, has been described in literature.

CASE
A 28-year-old Malay male presented with 1-week history of painless jaundice, associated with tea-coloured urine and diarrhoea. He also had a significant weight loss of 12 kg over the past 10 months. Physical examination showed an underweight young male, deeply jaundiced, with fine tremors. He was normotensive and not tachycardic. He did not have a goitre, thyroid eye disease or pretibial myxoedema. He had no stigmata of chronic liver disease. Blood investigation showed transaminitis with conjugated hyperbilirubinemia, with ALT 174 IU/L, AST 112 IU/L, total bilirubin 357 µmol/L, and predominant direct bilirubin (252 µmol/L). Autoimmune, infectious, and primary hepatobiliary disorders were ruled out. Thyroid function test was taken on day 16 of admission, which showed suppressed TSH <0.01 m IU/L, and elevated free T4 at 77 pmol/L. He was started on carbimazole, prednisolone and cholestyramine. carbimazole was withheld after 1 week of treatment in view of worsening hyperbilirubinemia and transaminitis. Subsequently, he received radioactive iodine therapy after 3 weeks of treatment. He had clinical and biochemical improvement after the radioactive iodine therapy. He eventually progressed into a hypothyroid state. His bilirubin levels subsequently normalized.

CONCLUSION
Severe jaundice is a rare consequence of hyperthyroidism and can be due to various pathologies. A thorough investigation should be done to look for contributing

factors and hence, be treated accordingly. Treatment of hyperthyroid patients with liver abnormalities is rather challenging as antithyroid drugs have been associated with liver injury. Various case reports showed remission of hyperbilirubinemia after radioactive iodine therapy. Therefore, radioactive iodine therapy should be offered as early as possible for patients with severe hyperbilirubinemia that is likely due to hyperthyroidism.

EP_A161

BIZARRE THYROID FUNCTION TEST IN A PATIENT WITH MULTINODULAR GOITRE: A CASE REPORT

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INTRODUCTION/BACKGROUND
Multinodular goitres may be accompanied by various thyroid function abnormalities. Assessment of thyroid status and correct interpretation of thyroid function tests (TFTs) is important to ascertain the aetiology.

CASE
A 68-year-old female was referred for re-evaluation of abnormal TFT associated with a huge multinodular goitre. She was seen 3 years ago by a private practitioner due to progressive goitre enlargement since age 30 years. TFTs then showed markedly reduced fT4 at 1.3-3.6 pmol/L (12-22), with normal TSH at 0.3-0.65 μIU/mL (0.27-4.2). She was started on L-thyroxine 100 ug daily based on these results, but she was only taking it intermittently. At the time of evaluation, there was no sign or symptom of hypothyroidism, but she complained of weight loss and irritability. On examination, she had a huge goitre with no lymphadenopathy. TFTs done showed low fT4, 8.6 pmol/L and TSH <0.005 μIU/mL. Central hypothyroidism was ruled out by a paucity of signs of hypothyroidism with no accompanying hypopituitarism. Due to the persistent and markedly suppressed TSH (<0.005) but fT4 at a low normal limit, fT3 was assessed and was found to be elevated at 11-16.7 pmol/L (3.1-6.8). L-thyroxine was stopped.

Three months later, a repeat TFT off L-thyroxine still showed a very low fT4 at 1.94 pmol/L, but normal fT3 (5.02 nmol/L) and TSH (0.291 μIU/mL). SHBG was normal at 52.4 nmol/L (16.8- 125.2) supporting euthyroidism. She remained well and euthyroid on subsequent follow-up with similar TFT but refused FNAC or surgical intervention for her goitre.

CONCLUSION
Low fT4 with normal TSH points towards central hypothyroidism but in patients with goitre and clinically euthyroid, disorders like iodine deficiency and thyroid dyshormonogenesis need to be considered. A T3 measurement should be done. A high T3/T4 ratio may be found in rarer entities such as resistance to thyroid hormone α and has also been reported in follicular thyroid cancer due to increased thyroidal deiodinase activity.

EP_A162

TUBERCULOUS MENINGOENCEPHALITIS MASKING MYXOEDEMA COMA

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INTRODUCTION/BACKGROUND
Myxoedema coma is a rare but potentially lethal complication of extreme hypothyroidism. Despite its low incidence, the mortality rate may reach 60%.

CASE
A 40-year-old male presented with shortness of breath, vomiting, frontal headache and abnormal behaviour for 2 days. He also suffered from fever, chesty cough, and chronic back pain for 2 weeks. He was confused, had unequal pupils, loss of lateral 1/3rd of his eyebrows, and reduced breath sounds bilaterally. Cranial CT scan demonstrated obstructive hydrocephalus necessitating external ventricular drainage. Pus aspirated from a right exudative pleural effusion yielded an ADA value of 68.78U/L. An MRI showed intracranial hyperintense lesions and L3/L4 spondylitis. Diagnosed with disseminated TB, anti-TB treatment with tapering doses of dexamethasone was commenced. He needed tracheostomy for prolonged intubation and had poor GCS recovery. On day 28 of hospitalization, he developed hypotension with a BP of 70/50 mm Hg, warranting noradrenaline infusion.

In retrospect, he had been bradycardic (heart rate ranged 30-55 bpm), hypothermic with a temperature of 35.7°C, and had recurrent hypoglycaemic episodes 7 days prior. Blood gas demonstrated CO_2 retention. Echocardiography did not exhibit pericardial effusion. His TSH level was >48.8 m IU/L, T4 level <3.2 pmol/L, and morning cortisol 163 nmol/L. He was administered IV Hydrocortisone 100 mg including IV Thyroxine 200mcg slow bolus. IV Thyroxine was then reduced to 100 mcg OD for 2 days and subsequently switched to an oral maintenance dose of 100 mcg OD. His

heart rate along with his temperature normalized. He was eventually weaned off inotropic support. Repeat TFTs after 6 days showed T4 of 11.1 pmol/L and TSH of 9 m IU/L.

CONCLUSION

In this case, the presence of TB meningoencephalitis obscured the diagnosis of severe hypothyroidism, resulting in treatment delay. In cases with high clinical suspicion of myxoedema coma, stress doses of hydrocortisone and thyroxine replacement are vital even prior to laboratory confirmation to enhance survival.

EP_A163

CLINICAL AUDIT ON REFLEX-FREE T3 TESTING AT HOSPITAL PUTRAJAYA, MALAYSIA

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INTRODUCTION

Reflex-free T3 (fT3) testing has long been used to optimize the use of laboratory tests in Hospital Putrajaya. It involves the automatic addition of fT3 reflexively when the TSH level is below the normal range and free T4 (fT4) is within normal limits. Excessive reflex testing can lead to an added economic burden. The objective of this audit was to determine the usefulness of reflex fT3 testing using different TSH cut-offs.

METHODOLOGY

Previously, fT3 was performed automatically when TSH was below the normal limits (<0.38 m IU/L) with normal fT4 (7.9 to 14.4 pmol/L). A new workflow was implemented in March where reflex fT3 was only done when TSH is <0.1 m IU/L with normal fT4. This reflex testing is only applied to adults above 18 years old. Patients who underwent reflex fT3 testing three weeks before (Group 1) and after (Group 2) implementation of the new workflow were identified. Patients who would have had reflex fT3 testing with the old workflow but not in the new workflow (TSH 0.1 - 0.37 m IU/L with normal fT4) were also identified (Group 3). Data on patient characteristics were retrospectively collected and analysed.

RESULT

There were 105 patients in Group 1, 66 in Group 2 and 41 in Group 3. The new TSH cut-off of <0.1 resulted in a 38% reduction in reflex fT3 testing. Only 9 (4.25%) out of the 212 patients in the 3 groups had clinical necessity for fT3 testing. The fT3 result changed management in only 6 cases. The other 3 cases were planned for follow-up with repeat tests as clinically euthyroid.

CONCLUSION

Reflex fT3 testing was unnecessary in a large number of cases. The usefulness of reflex fT3 testing in this cohort was very low. Hence, reflex fT3 testing is being discontinued at our centre. Further evaluation is needed to determine strategies that can optimise the ordering of fT3 tests.

EP_A164

WHEN THIONAMIDES ARE CONTRAINDICATED: OUTCOME OF CHOLESTYRAMINE THERAPY IN HYPERTHYROID PATIENTS: A SINGLE TERTIARY CENTRE EXPERIENCE

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INTRODUCTION

Cholestyramine, a bile acid sequestrant, binds to thyroid hormones in the intestine and enhances their clearance. Thionamides are the mainstay in the treatment of hyperthyroidism, however, this may not be an option in the presence of profound hepatitis and agranulocytosis. Here, we aim to assess the efficacy and tolerability of cholestyramine therapy in patients with hyperthyroidism where thionamides are contraindicated.

METHODOLOGY

A one-year retrospective review of patients with hyperthyroidism who were treated with cholestyramine was performed from April 2023 to April 2024.

RESULT

A total of 10 patient medical records (8 females and 2 males) were reviewed. The mean age was 51.7 years old and the median duration of hyperthyroidism was 7.5 years. Graves' disease was the underlying aetiology in 7 cases, and the rest was a toxic multinodular goitre. Six of our patients already had atrial fibrillation, with four of them

having cardiomyopathy. The reason for conversion from carbimazole to cholestyramine was transaminitis for one patient, and the remaining was due to neutropenia and thrombocytopenia. Seven patients (70%) received Lugol's iodine for not more than 10 days, relying on its Wolff-Chaikoff effect. One patient received prednisolone as an adjunct therapy for hyperthyroidism. The total daily dose of cholestyramine commenced was 12 g given in TDS dosing for a median duration of 1.4 months. Median FT4 level pre- and post-cholestyramine therapy were 50.2 pmol/L and 25.5 pmol/L respectively (NR 7.86-14.41), $p = 0.028$. The median TSH level was <0.005 m IU/L. We were able to rechallenge six patients (60%) with carbimazole as they showed an improvement in their laboratory parameters. Only two patients underwent subsequent definitive therapy with RAI and thyroidectomy. None of our patients developed any adverse side effects from cholestyramine.

CONCLUSION

Our experience demonstrated that in selected cases, cholestyramine may be used as an effective and well-tolerated therapy when first-line options are contraindicated.

EP_A165

THYROTOXIC CARDIOMYOPATHY COMPLICATED BY FULMINANT HEPATIC FAILURE: A CASE REPORT

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INTRODUCTION/BACKGROUND

Thyrotoxic cardiomyopathy with cardiac failure can lead to liver congestion and ischaemic hepatitis. Fulminant hepatic failure secondary to thyrotoxic cardiomyopathy is rare.

CASE

We report a 45-year-old woman with strong family history of hyperthyroidism. She presented with palpitations and cardiac failure symptoms for a month. Electrocardiograph showed atrial fibrillation. Echocardiogram revealed a preserved ejection fraction (55%), mid-septal wall hypokinesia, severe mitral and tricuspid regurgitation, with pulmonary hypertension. She had an elevated free T4 (fT4) level of 16.4 pmol/L (7.86-14.41 pmol/L) and free T3 (fT3) level of 7.6 pmol/L (3.10-6.80 pmol/L). TSH receptor antibody was elevated 13.7 IU/L (<1.75 IU/L) consistent with Graves' Disease. She was treated for thyroid storm and initiated on an anticoagulant. She was discharged with carbimazole 30 mg OD and bisoprolol 2.5 mg OD.

After 10 days, she returned with worsening cardiac failure, high-grade fever and jaundice. Upon admission, the fT4 level was 12 pmol/L. Her liver transaminases were normal except for hyperbilirubinemia secondary to liver congestion. Subsequently, transaminases showed rapid progression of liver failure with peak aspartate aminotransferase (AST) of more than 10,000 U/L, total bilirubin of 481.3 umol/L (5.0-21.0 umol/L), and severe coagulopathy. She required mechanical ventilation due to hepatic encephalopathy. Ultrasonography of the hepatobiliary system showed cholelithiasis with acute cholecystitis. Budd-Chiari Syndrome was ruled out since the hepatic veins were patent. Viral hepatitis was likewise ruled out. She was managed with N-acetylcysteine, diuretics, and second-line anti-thyroid treatment (cholestyramine, hydrocortisone, and Lugol's solution). Her sepsis responded to intravenous meropenem. She was not suitable for liver transplantation due to multi-organ failure after consulting the hepatology team.

CONCLUSION

A comprehensive approach involving cardiac evaluation with echocardiogram, assessment of liver dysfunction, and consideration of autoimmune causes of liver failure is crucial in the management of patients with thyrotoxicosis and liver failure. Liver transplant is an option in the management of thyrotoxicosis with fulminant liver failure.

EP_A166

T3 THYROTOXICOSIS IN A PATIENT WITH METASTATIC FOLLICULAR THYROID CARCINOMA

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INTRODUCTION/BACKGROUND

Differentiated thyroid cancers are usually associated with normal thyroid function. Rarely, thyrotoxicosis can develop due to functioning metastatic thyroid carcinoma. We present a case of a male with metastatic follicular thyroid cancer associated with T3 thyrotoxicosis.

CASE

A 57-year-old male with underlying multinodular goitre presented with rapidly enlarging neck swelling, heat intolerance, loose stools, weight loss, and left shoulder pain over three months' duration. He exhibited a huge left goitre with right tracheal deviation. Laboratory tests revealed

normal FT4, elevated FT3, and suppressed TSH. Thyroid ultrasound and contrast computed tomography (CT) scan unveiled a large left hemithyroid mass with retrosternal extension and contralateral tracheal displacement. Metastatic lesions were observed in the lungs, pleura, left scapula, cervical, and mediastinal lymph nodes. Needle aspiration of the thyroid mass showed a follicular nodule, while biopsy of the left scapula confirmed metastatic follicular thyroid carcinoma. The patient underwent total thyroidectomy with left modified radical neck dissection. Histopathologic examination revealed widely invasive follicular thyroid carcinoma, with areas of transformation to anaplastic thyroid carcinoma.

CONCLUSION
The coexistence of T3 thyrotoxicosis and thyroid cancer, particularly the follicular subtype, is uncommon and warrants careful consideration in clinical practice.

EP_A167

PROPRANOLOL-INDUCED CARDIAC DECOMPENSATION IN THYROID STORM

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INTRODUCTION/BACKGROUND
Propranolol is one of the preferred beta-blocking agents used in thyroid storm. It is highly lipid-soluble and effective in reducing T3 concentration up to 30% if given in high doses. However, only a few cases reported on the side effects of this drug, especially life- threatening complications in thyroid storm.

We reported 4 cases of propranolol-induced circulatory collapse in patients with thyro-cardiac disease who presented with thyroid storm between 2022- 2024.

CASE 1
A 28-year-old male diagnosed with Graves' disease developed thyroid storm with cardiac decompensation post-wound debridement. He received carbimazole 30 mg and propranolol 40 mg prior to surgery. The propranolol was withheld following the unfortunate event and he recovered after 3 days.

CASE 2
A 32-year-old female with Graves' disease presented with acute heart failure and tachyarrhythmia. She was initially normotensive on arrival; however, she developed circulatory collapse after receiving propranolol 40 mg. She was managed in the ICU before succumbing to her death due to severe cardiac decompensation.

CASE 3
A pregnant female at 34 weeks AOG presented with an impending thyroid storm and premature uterine contraction. She was normotensive and tachycardic on presentation. The condition was complicated by cardiogenic shock and acute heart failure right after propranolol 40 mg administration. She was placed on mechanical ventilation but had an intrauterine foetal loss.

CASE 4
A 43-year-old female presented with thyroid storm and unstable atrial fibrillation. She was intubated and received synchronized cardioversion at 150 J together with antithyroid and glucocorticoid drugs. Her condition worsened after she was given oral propranolol 20 mg and she eventually succumbed due to cardiac decompensation.

CONCLUSION
Long-acting beta-blockers should be used with caution in thyroid storms with pre-existing thyro-cardiac disease as they can potentially impede the compensatory mechanism and consequently cause hemodynamic instability.

EP_A168

A CHALLENGING CASE OF GRAVES' DISEASE WITH MYELODYSPLASTIC SYNDROME

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INTRODUCTION/BACKGROUND
Graves' disease is an autoimmune condition where antibodies are produced against the thyrotropin (TSH) receptors on the thyroid gland. The condition can be associated with haematologic manifestations.

CASE
A 44-year-old male with underlying Graves' disease, Schizophrenia, Chronic Hepatitis B and Myelodysplastic Syndrome presented with a week's history of loose stools and vomiting. On examination, blood pressure was 115/78 mmHg and heart rate was 97 bpm. He had pallor, tremors, sweaty palms, and a small goitre. Thyroid function tests were: TSH <0.001 m IU/L (0.27-4.2), T4 21.9 pmol/L (12.0-22.0), T3 2.65 pmol/L (3.1-6.8). His complete blood count was: Hb 11.7 g/dl (13-17), WBC 3.52 x 10^9/L (4-10), ANC 1.68 x 10^9/L (2.0-7.0), Platelets 69 x 10^9/L (150-410).

He was started on a thionamide with close monitoring of blood counts. However, the thionamide was withheld in view of his reducing absolute neutrophil count. He was then treated with steroids, lithium and cholestyramine with no improvement in his thyroid function tests.

Hence, he was eventually given radioactive iodine. Graves' disease with myelodysplastic syndrome proves to be challenging for endocrinologists to treat. The probable underlying pathophysiology is that high blood levels of thyroid hormones can be toxic to bone marrow cells leading to an increase in functional activity of reticuloendothelial cells, causing insufficient hematopoietic cells. In one study, free T3 and T4 were noted to be higher with lower TSH in patients with myelodysplastic syndrome. In view of the difficulty of treating hyperthyroidism with anti-thyroid drugs, our patient was treated with radioiodine ablation.

CONCLUSION

In conclusion, managing Graves' disease in individuals with myelodysplastic syndrome requires detailed evaluation and monitoring.

EP_A169

MANAGING THYROTOXIC ATRIAL FIBRILLATION IN A BIOCHEMICALLY EUTHYROID PATIENT

https://doi.org/10.15605/jafes.039.S1.180

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INTRODUCTION/BACKGROUND

Hyperthyroidism induces cardiovascular changes like increased heart rate and atrial automaticity, leading to conditions such as atrial fibrillation and heart failure, contributing to higher mortality rates. Despite achieving euthyroidism with treatment, cardiovascular manifestations may persist, necessitating further investigation into factors associated with persistent atrial fibrillation to guide appropriate anticoagulation therapy.

CASE 1

A 66-year-old Malay male with high blood pressure, dyslipidaemia, and thyrotoxic atrial fibrillation (TAF) due to Graves' disease of 5 years duration. He had two failed radioactive iodine treatments and thyroid surgery. He had periodic palpitations, dyspnoea, and left chest pain. His ECG revealed rapid atrial fibrillation. He has uncontrolled elevated blood pressure. The thyroid function tests were normal (T4 = 14.21, TSH = 4.78). He was eventually referred to the cardiology team who recommended cardiac ablation.

CASE 2

A 34-year-old female with Graves' disease and atrial fibrillation (AF) despite taking bisoprolol, went to the emergency department due to frequent palpitations and dizziness. She did not have chest pain. Her ECG showed atrial fibrillation. She had normal thyroid function tests (T4 = 15.21, TSH = 3.56) with elevated troponin levels. She was treated for symptomatic AF. She was subsequently referred to cardiology for cardiac ablation.

CONCLUSION

Thyroid hormones affect cardiovascular function, predisposing hyperthyroid individuals to atrial fibrillation even after achieving euthyroidism. The thromboembolic risk in TAF is reduced by oral anticoagulants. Treatment for TAF involves antithyroid medications to restore euthyroidism together with rate and rhythm regulation. Wong et al., found an unexpected relationship between decreased free thyroxine levels and chronic atrial fibrillation. TAF has a high thromboembolic risk even after euthyroidism, requiring anticoagulants and ongoing monitoring to prevent recurrence. Sometimes ablation is recommended, especially for persistent AF. In conclusion, hyperthyroidism-related AF therapy requires collaboration between endocrine and cardiovascular specialists. Prompt diagnosis and personalised treatment can improve the prognosis and reduce complications.

EP_A170

A RARE CASE OF FUNCTIONAL METASTATIC FOLLICULAR THYROID CARCINOMA WITH EGGSHELL CALCIFICATION

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INTRODUCTION/BACKGROUND

Only a few cases of follicular thyroid carcinoma (FTC) with eggshell (or rim-like peripheral) calcification have been reported. Here, we report a rare case of functional metastatic FTC with eggshell calcification.

CASE

A 57-year-old female presented with progressive neck enlargement, dysphagia, and weight loss of 10 kg over 2 months. She also had a hoarse voice. On examination, she appeared thyrotoxic. She had a palpable 3 x 4 cm mass over the left neck, which was hard in consistency and immobile. Biochemically, she was hyperthyroid with suppressed TSH and high free T4 of 67.9 pmol/L (7.9-14.4). Her chest radiograph showed an eggshell calcification over the neck

region with right tracheal deviation. Her neck ultrasound showed a thyroid nodule (ACR TI-RADS 5). CT scan revealed a left thyroid nodule (3.3 x 3.2 x 3.7 cm) with peripheral coarse calcifications. Mass effects were seen on the adjacent vessels, trachea, and oesophagus. Left vocal palsy was likewise noted. There was a destructive lytic soft tissue lesion seen at the manubrium of the sternum with multiple suspicious lung nodules. FNAB of the thyroid and sternal lesions demonstrated Bethesda II follicular lesions. She underwent thyroid surgical resection with histopathology-confirmed widely invasive FTC. Postoperatively, she remained thyrotoxic for which radioactive iodine therapy was given subsequently.

CONCLUSION

Functional thyroid carcinoma (TC) is rare with the FTC subtype being more prevalent (especially the metastatic disease) and having a less favourable prognosis. FNAB cannot distinguish FTC from benign follicular neoplasm, hence, histologic evaluation of the thyroid specimen is required. About 40-60% of patients with eggshell calcification within the thyroid gland were reported to be malignant and commonly in papillary TC. Sonographic features of a peripheral halo with discontinuity of the calcification are predictive of malignancy. Thyroid carcinoma should be considered in patients with aggressive symptoms and presence of eggshell calcification on radiograph.

EP_A171

THE CONUNDRUM OF BEING CONFRONTED WITH A DIRE THYROTOXICOSIS ON THE MORNING OF CORONARY ARTERY BYPASS GRAFT SURGERY (CABG)

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INTRODUCTION/BACKGROUND

This case illustrates the management of an acute medical condition with complex comorbidities, highlighted by a patient with acute pulmonary oedema post-NSTEMI and severe preoperative thyrotoxicosis, undergoing urgent CABG.

CASE

A 52-year-old female with a history of myocardial infarction presented with an acute pulmonary oedema following an episode of NSTEMI. An urgent coronary angiogram revealed a thrombosed stent in the left anterior descending and left circumflex arteries. She was deemed a high-risk patient; hence, an urgent CABG was planned. However, on the morning of the planned surgery, she had tachycardia of 130/minute. Thyroid function tests showed elevated fT4 of 99.2 pmol/L (normal range: 12-22) and suppressed TSH <0.01 m IU/L (normal range: 0.4-4.5).

At the insistence of the cardiothoracic surgeon who was concerned about an impending cardiogenic shock, the endocrinologist reluctantly agreed to allow the surgery to proceed with the following provisions: 1. Immediate loading with 10 drops of Lugol's iodine, 100 mg IV hydrocortisone, 10 mg carbimazole, and 4 grams of cholestyramine 2. Heart rate was to be lowered with 80 mg of propranolol repeated every half an hour till the heart rate went to <100/min. 3. Surgery was to be delayed for a further 4 hours to allow for the anti-thyroid regimen to take effect whilst controlling the heart rate. Throughout surgery, the heart rate was maintained at 100/minute. Despite the risk of hemodynamic instability, the surgery was uneventful. The patient was kept in the ICU and eventually extubated 2. All the anti-thyroid regimens were continued diligently except for hydrocortisone which was stopped on POD 3. On POD 5, she developed an episode of atrial fibrillation which was promptly terminated with synchronised cardioversion. Notwithstanding the concern of a life-threatening thyroid storm, her recovery was seemingly uneventful. On POD 9, her fT4 had steadily come down to 16.4 pmol/L and she was promptly discharged home with a maintenance dose of 10 mg carbimazole.

CONCLUSION

The successful outcome in this high-risk patient, achieved through a multidisciplinary approach, underscores the potential benefits and ongoing debate regarding the optimal strategy for such complex clinical scenarios.

EP_A172

STORMY SEAS: MANAGING THYROID STORM TREATMENT-RELATED COMPLICATION WITH BETA-BLOCKER TOXICITY

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INTRODUCTION/BACKGROUND
Thyroid storm is a life-threatening condition involving multiple organ systems due to thyrotoxicosis. The standard treatment often includes the preferred option of the beta blocker (BB) propranolol. However, usage of BB in thyroid storm management was linked to cardiogenic collapse due to its toxicity. We present a case of thyroid storm who was treated with a beta-blocker and developed toxicity.

CASE
A 65-year-old female presented with shortness of breath, palpitations, fever, and diarrhoea. In the Emergency Department, she was fully conscious but agitated. She had tachycardia with signs of heart failure. Urgent TFT showed suppressed TSH <0.01 m IU/L and elevated T4 level of 44.12 pmol/L. Her Burch and Wartofsky Score was 70. Thus, diagnosis of thyroid storm with thyrotoxic cardiomyopathy was made. She was started on propylthiouracil, Lugol's Iodine, steroids, Propranolol 40 mg QID.

After 8 hours of treatment, she became drowsy, developed junctional bradycardia and hypotension. Appropriate resuscitation with IV Atropine and inotropes was started. Excluding other causes of hypotension with bradycardia, we considered beta-blocker toxicity. Subcutaneous glucagon was initiated. Within one day, inotropes were weaned off.

Thyroid storm can lead to lethal complications. The presentation ranges from thermoregulatory, neurologic, gastro-hepatic, cardiac dysfunctions to circulatory collapse and shock. The treatment includes BB, anti-thyroid drugs, and potassium Iodide or Lugols Iodine, along with hydrocortisone. Second-line options may include lithium, dialysis, or plasmapheresis.

Beta blockers work by reducing hyperadrenergic states and blocking the peripheral conversion of T4 to T3. They can have adverse effects such as peripheral coldness, syncope, bradycardia, hypotension, circulatory collapse, and even cardiac arrest. Glucagon is the first-line antidote for BB toxicity.

CONCLUSION
The use of beta blockers in treating thyroid storm requires close monitoring due to the risk of devastating cardiogenic collapse.

EP_A173

NODULAR PRETIBIAL MYXEDEMA FOLLOWING TREATMENT OF GRAVES' DISEASE

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INTRODUCTION/BACKGROUND
Pretibial myxoedema (PM) is an uncommon manifestation of Graves' disease (GD). Typically, thyroid dermopathy may present as non-pitting oedema with indurated skin giving a 'peau d'orange' appearance. Less commonly, patients may present with other variants such as plaques, nodules and elephantiasic type lesions.

We report a case of biopsy-proven PM in a patient with GD.

CASE
An 80-year-old woman presented with multiple painless nodules over both shins with gradual increase in size over 6 months. She had a history of hypertension and difficult-to-manage GD, complicated by thyroid storm, atrial fibrillation, and heart failure. Her thyroid function fluctuated from hypo- to hyperthyroidism within weeks. She refused radioactive iodine ablation and was subsequently controlled with a block and replace regimen. She did not have any constitutional symptoms, preceding trauma, or insect bite. Clinically, she was euthyroid and did not have any active thyroid eye disease.

On examination, there were multiple ill-defined, firm, non-tender, flesh-coloured nodules over both shins. Her FT4 was 18.12 pmol/L (12.0 - 22.00) and TSH <0.005 m IU/L (027 – 4.20). A punch biopsy revealed fragmented collagen fibres with conspicuous mucin deposits over the reticular dermis and subcutaneous layer, consistent with PM. She was started on potent topical corticosteroids with marked improvement in her skin lesions.

CONCLUSION
Thyroid dermopathy can present in an atypical manner, hence, physicians should be aware of this. Treatment of hyperthyroidism may not have any significant effect on the cutaneous lesions. PM may occur even after successful control of the disease.

EP_A174

NEUROLOGIC MANIFESTATION AND PERICARDIAL EFFUSION UNVEILING AN AUTOIMMUNE HYPOTHYROIDISM

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INTRODUCTION/BACKGROUND
Autoimmune hypothyroidism is an antibody-mediated chronic inflammatory process. Thyroid destruction may be intermittent. Given its chronic and progressive nature, the diagnosis is often challenging since the exhibited signs and symptoms are often subtle and non-specific. We report a middle-aged male with bilateral upper and lower limb weakness and pericardial effusion. Investigation led to a diagnosis of autoimmune hypothyroidism.

CASE
A 64-year-old male presented with a two-week history of lethargy, poor appetite, and lower limb swelling. Initially, he was treated for pneumonia and cardiac failure due to chest radiography showing obscured cardio-phrenic angle. Further history revealed he had inability to walk for one year due to bilateral lower limb weakness. He had proximal muscle weakness of all four limbs. Sensation and reflexes were preserved in the upper limbs but absent in the lower limbs. His nerve conduction study and electromyography revealed myopathic changes involving all four extremities with absent neurosensory responses in both lower extremities. His cranial CT scan showed bifronto-temporal subdural effusion while his echocardiography exhibited pericardial effusion with cardiomegaly. His thyroid function tests revealed profound hypothyroidism (TSH>100, fT4 <1). Together with the presence of markedly raised anti-TPO antibodies, he was diagnosed to have autoimmune primary hypothyroidism. He also had normocytic normochromic anaemia and hypercholesterolemia consistent with severe hypothyroidism. He was eventually started on oral L-thyroxine (1.6 mcg/kg/day).

CONCLUSION
This case report highlights the potential for severe neuromuscular and cardiovascular consequences due to untreated chronic autoimmune hypothyroidism. Thyroid dysfunction is a consideration in a patient with neurologic manifestation. Early diagnosis and prompt treatment of hypothyroidism can potentially avert long-term hypothyroid sequelae.

EP_A175

THYROID FUNCTION ABNORMALITIES: CONNECTING THE DOTS BETWEEN GENETICS AND CLINICAL PRESENTATION

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INTRODUCTION/BACKGROUND
Subclinical hyperthyroidism presents with low or undetectable levels of thyroid stimulating hormone (TSH), alongside normal levels of free thyroid hormones (fT4 and fT3). While certain individuals may not show any symptoms, others may experience hyperthyroid symptoms like palpitations, weight loss, and heat intolerance. Early recognition and prompt appropriate management are crucial to prevent potential complications, including atrial fibrillation, osteoporosis, and progression to overt hyperthyroidism.

CASE
A 59-year-old female was referred to our endocrine clinic due to abnormal thyroid function tests (TFTs) revealing subclinical hyperthyroidism. She was asymptomatic.

Her thyroid function tests 5 years ago showed similar results, however, she had not received proper consultation or treatment during that time.

She has no significant medical history. She has been in menopause since 50 years old with regular menses before that. She had five pregnancies, four of which were preterm, with her eldest child having cerebral palsy and her fourth child deceased due to prematurity. Two of her children have hyperthyroidism. One son is on carbimazole while one daughter has subclinical hyperthyroidism.

A repeat thyroid function test still showed suppressed TSH level of 0.04 m IU/L and normal FT4 level of 13.2 pmol/L and free triiodothyronine (fT3) level of 4.3 pmol/L. Molecular studies showed polymorphism of exon 3 of the TSHR gene from the son.

CONCLUSION
In conclusion, this case report emphasized the need for thorough evaluation and appropriate management of abnormal thyroid function tests, particularly in the presence of familial clustering. Early recognition and treatment can prevent potential complications and improve patient outcomes. Additionally, the potential role of genetic factors, such as polymorphisms in exon 3 of the TSHR gene, should be considered in cases of familial clustering of thyroid disorders. Genetic testing and clinical correlation may be necessary for a comprehensive assessment and management of thyroid disorders associated with genetic polymorphisms.

EP_A176

MASSIVE PERICARDIAL EFFUSION AS A PRIMARY MANIFESTATION OF HYPOTHYROIDISM

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INTRODUCTION/BACKGROUND
Hypothyroidism is an endocrine disorder with multiorgan involvement and various complications. Mild pericardial effusion is a common cardiovascular complication but massive pericardial effusion with cardiac tamponade as initial presentation of hypothyroidism is rare.

CASE
We report a 70-year-old female with a history of hyperthyroidism who was treated with radioiodine ablation more than 20 years ago. She defaulted follow-up and hence was not on L-thyroxine. She presented with progressive exertional dyspnoea and hypothyroid symptoms (weight gain, fatigue, cold intolerance) for a month. On examination, she had coarse dry skin, periorbital oedema, and bradycardia. She was normotensive. Her heart sounds were not muffled. Biochemically she was in overt hypothyroidism, TSH 16.825 m IU/L (0.35-4.94), T4 <5.41 pmol/L (9.01-19.05). She also had hyponatremia with a sodium level of 118-125 mmol/L and hyperlipidaemia. She had cardiomegaly on a chest x-ray. Her electrocardiogram showed normal voltage complexes with no electrical alternans. Her echocardiography showed massive pericardial effusion (3.1 cm) with a collapsible right atrium. She had normal ventricular function. Pericardiocentesis was performed and 150 cc straw-coloured fluid was aspirated. The pericardial fluid was exudative. Cultures were negative for bacteria and acid-fast bacilli. There were no malignant cells. She was treated with L-thyroxine 75 mcg daily. TFTs repeated six weeks later were already normal with TSH of 2.521 m IU/L (0.35-4.94) and T4 of 12.76 pmol/L (9.01-19.05). Repeat echocardiography showed resolution of the pericardial effusion. Clinically, she remained asymptomatic.

CONCLUSION
Although massive pericardial effusion is an uncommon initial presentation of hypothyroidism, it can occur in long-standing untreated cases. Pericardial effusion can resolve with adequate thyroid hormone replacement therapy.

EP_A177

VANISHING THYROID NODULES: SUBACUTE THYROIDITIS MIMICKING SUSPICIOUS THYROID NODULES IN A PATIENT ON TYROSINE KINASE INHIBITOR

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INTRODUCTION/BACKGROUND
Dasatinib is a tyrosine kinase inhibitor (TKI) used as a second-line treatment for chronic myeloid leukaemia. Thyroid dysfunction is rare with dasatinib. We report a patient with chronic myeloid leukaemia on Dasatinib who developed subacute thyroiditis mimicking a suspicious thyroid nodular disease.

CASE
A 57-year-old female was started on dasatinib in June 2021. She presented with a one-month history of fever, palpitations, heat intolerance, and neck swelling in April 2023. Her thyroid function tests (TFTs) showed elevated free-T4 30.9 pmol/ and suppressed thyroid stimulating hormone (TSH), <0.008 m IU/L, hence, carbimazole 20 mg daily was initiated. Thyroid ultrasound revealed hypoechoic solid nodules at both upper poles, measuring 1.7 x 2.1 x 4.7 cm and 1.7 x 2.0 x 3.4 cm, respectively. Both nodules had TIRADS scores of 5. Another hypoechoic solid nodule with a TIRADS score of 4 was also found at the right mid-pole. However, during the scheduled ultrasound-guided fine needle biopsy two months later, the repeat ultrasound no longer showed any thyroid nodule. TSH-receptor antibody was negative. Her thyroid function normalised and her carbimazole dose was tapered off after 2 months of treatment. Repeat neck ultrasound six months later demonstrated a normal thyroid gland. The subsequent serial TFTs remained normal. Dasatinib was continued throughout this period.

TKI-induced thyroid abnormality usually appears within the first 6 months but can still manifest after the first year of treatment. Ultrasound descriptions of subacute thyroiditis

include diffuse heterogeneity, focal hypoechogenicity, decreased vascularity, as well as nodular lesions which can be mistaken for malignancy.

CONCLUSION
TFT measurement prior to TKI initiation is recommended, with repeat tests every 6 weeks for the first 6 months, every 3–6 months for a year, then biennial screening beyond the first 18 months of therapy. Recognition of sonographic patterns of subacute thyroiditis is important to avoid unnecessary procedures or increased patient anxiety.

EP_A178

THERAPEUTIC PLASMA EXCHANGE IN THREE SCENARIOS COMPLICATING HYPERTHYROIDISM: A RETROSPECTIVE CASE SERIES

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INTRODUCTION/BACKGROUND
Therapeutic plasma exchange (TPE) represents a viable option for managing thyroid storms when conventional therapies prove inadequate. Despite its utility, the precise indications for TPE have not been well established. Herein, we present our experience with three cases, elucidating treatment responses through changes in free T4 levels, which ultimately facilitated rapid clinical improvement. We describe the clinical presentations and laboratory profiles of three young patients (aged 17-27 years) admitted to Hospital Kajang for hyperthyroidism.

CASE 1
A 17-year-old female, presented with a severe thyroid storm complicated by hepatic encephalopathy and cardiomyopathy requiring mechanical ventilation. On day 3, TPE was initiated along with conventional therapy, which resulted in a 78% reduction in free T4 levels by day 4, with subsequent recovery by day 6.

CASE 2
A 27-year-old female with carbimazole-induced agranulocytosis and had an inadequate response to second-line antithyroid drugs, underwent four cycles of TPE as preoperative optimization for total thyroidectomy, achieving a 43% reduction in free T4 levels within 5 days, facilitating a successful surgical outcome.

CASE 3
An 18-year-old male, following a trivial fall resulting in a left femoral neck fracture, developed a severe thyroid storm. The urgency for joint surgery prompted four cycles of plasmapheresis, culminating in a 54% reduction in free T4 levels within 3 days, allowing for successful surgery by day 8.

All patients were discharged well without complications.

CONCLUSION
The action of TPE results primarily from plasma removal of cytokines, circulating autoantibodies, thyroid hormones, and their bound proteins. Our cases underscore the potential efficacy of plasmapheresis in hyperthyroidism management. They exemplify its effectiveness in diverse scenarios: managing severe, complicated thyroid storm; bridging to total thyroidectomy in carbimazole-induced agranulocytosis and failing conventional therapy; and urgently ameliorating thyroid storm before a joint-preserving procedure for a femoral neck fracture.

EP_A179

GRAVES' DISEASE PRESENTING WITH SUPERIOR MESENTERIC ARTERY SYNDROME

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INTRODUCTION/BACKGROUND
Superior Mesenteric Artery (SMA) syndrome is a rare manifestation of small bowel obstruction caused by compression of the third portion of the duodenum between the SMA and aorta. It is associated with extreme weight loss due to malnutrition/malabsorption, hypermetabolism or cachexia-causing conditions such as malignancy.

We report a case of SMA syndrome due to acute weight loss secondary to undiagnosed Graves' disease.

CASE
A 63-year-old female with a medical history of schizophrenia in remission, presented to the emergency department with a two-week history of persistent postprandial vomiting and upper abdominal pain. She had a history of unintentional weight loss of approximately 11 kg over 3 months.

On examination, she appeared cachectic. She had a blood pressure of 137/72 mmHg and a heart rate of 120 bpm. Thyroid function tests showed severe hyperthyroidism with TSH <0.01 m IU/L and FT4 100 pmol/L. She had elevated TSH receptor antibodies of 32.7 IU/L. Her abdominal CT revealed a grossly distended stomach filled with oral contrast and significant narrowing at the D4 level of the duodenum. She was diagnosed with SMA syndrome secondary to Graves' disease. Hence, she was treated with nasogastric intubation for gastric decompression, total parenteral nutrition, antiemetic, PTU per rectal, Lugol's iodine and intravenous propranolol to control her thyrotoxicosis. Despite conservative treatment and normalisation of FT4 level, the patient had persistent symptoms hence she underwent exploratory laparotomy and duodenal kocherisation. Postoperatively, her symptoms improved. She was able to resume a normal diet and continued to gain weight appropriately.

CONCLUSION

This case highlights the importance of considering SMA syndrome in patients with Graves' disease presenting with gastrointestinal symptoms and rapid weight loss. Prompt treatment of thyrotoxicosis alongside nutritional optimization and duodenal obstruction relief by conservative or surgical management is equally crucial.

EP_A180

NAVIGATING THE CONUNDRUM: ACUTE LIVER FAILURE IN HYPERTHYROIDISM AND THE TREATMENT DILEMMA

https://doi.org/10.15605/jafes.039.S1.191

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INTRODUCTION/BACKGROUND

Hyperthyroidism is a complex endocrine disorder associated with various systemic manifestations. Liver dysfunction in hyperthyroidism is a relatively rare but potentially serious complication. We present a case of a patient with hyperthyroidism who initially received inadequate treatment and subsequently developed acute liver failure. The causative role of hyperthyroidism itself versus antithyroid medication-induced liver injury remains elusive, posing a therapeutic challenge. A comprehensive review of the patient's medical records, laboratory findings, imaging studies, and clinical progress was undertaken. Additionally, relevant literature concerning liver dysfunction associated with hyperthyroidism and drug-induced liver injury was explored.

CASE

Two months after initiating carbimazole therapy, a 42-year-old male with a history of hyperthyroidism presented with jaundice. Subsequent liver function tests indicated significant conjugated hyperbilirubinemia, accompanied by abnormalities in prothrombin time, development of hepatorenal syndrome, and encephalopathy. Imaging studies detected no structural abnormalities. Despite thorough evaluation, the exact cause of his liver failure remained elusive, posing challenges in distinguishing between exacerbation of hyperthyroidism and carbimazole-induced hepatotoxicity. Close monitoring ensued, with consideration given to liver transplant if necessary. Discontinuation of carbimazole and initiation of Lugol's iodine and cholestyramine led to clinical improvement. Radioactive iodine therapy was planned as the definitive treatment.

CONCLUSION

While acute liver failure in Graves' disease is rare, its management poses significant hurdles. Despite cholestasis and liver dysfunction, meticulous methimazole administration can effectively control hyperthyroidism with careful monitoring. However, when the cause of liver injury remains elusive—whether from the disease itself or its treatment—crafting an appropriate management plan becomes particularly complex. A different treatment approach may be necessary to achieve euthyroid state, often necessitating definitive therapy in such cases.

EP_A181

BATTLE OF AUTO-IMMUNITIES: GRAVES' DISEASE AND RHEUMATOID ARTHRITIS: A BIDIRECTIONAL CAUSAL EFFECT

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INTRODUCTION/BACKGROUND

Graves' disease is an autoimmune disorder characterized by hyperthyroidism secondary to circulating thyroid autoantibodies. Co-existence with other autoimmune diseases such as vitiligo, chronic autoimmune gastritis and rheumatoid arthritis (RA) have been reported. We report a patient who developed RA more than 10 years following her diagnosis of Graves' disease.

CASE
A 37-year-old female with Graves' disease diagnosed at age 22 years presented with a 2 to 3-week history of multiple joint pains and morning stiffness involving both knees, ankles, elbows, and metacarpophalangeal joints (MCPJs). She did not have any preceding trauma, fever, genitourinary or gastrointestinal infections. There were no sicca symptoms, rashes, oral ulcers or constitutional symptoms. Examination revealed right knee and multiple MCPJ synovitis. She had a small diffuse goitre and no exophthalmos. Inflammatory markers were elevated with an ESR of 70 mm/H and CRP of 21.6 mg/L. She had mild hypochromic microcytic anaemia (Hb11.8 g/dL) and lymphopenia (1.3 103/uL). Rheumatoid factor and anti-Ro60 were both positive. Uric acid (277 umol/L) and FT4 (21.07 pmol/L) levels were normal. Knee joint aspirate culture and AFB were negative. Joint x-rays were unremarkable. Thyroid ultrasound showed enlarged thyroid lobes with coarse echotexture and increased vascularity. A rheumatology consult was obtained, and she was diagnosed with RA. She commenced on methotrexate and oral steroids which resulted in marked improvement in her joint condition. Her fT4 remained stable on low dose carbimazole.

CONCLUSION
Graves' disease is associated with an increased risk of RA and vice versa. Hence, it is important to screen patients with inflammatory joint pain for RA to ensure prompt management and prevent long-term joint damage and other complications.

EP_A182

A CASE OF MASSIVE PERICARDIAL EFFUSION IN SUBCLINICAL HYPOTHYROIDISM

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INTRODUCTION/BACKGROUND
Hypothyroidism is associated with multiorgan involvement and various complications. Pericardial effusion is a rare complication of hypothyroidism. However, if left untreated, it may progress to critical, life-threatening conditions such as cardiac tamponade and hemodynamic instability. Early identification of the diagnosis, with effective management of pericardial effusion in hypothyroidism, is essential.

CASE
A 67-year-old female with hypothyroidism since 2016 presented with worsening exertional dyspnoea, bilateral lower limb swelling, and fatigue. She had a background history of hypertension and bronchial asthma. She had elevated jugular venous pressure, but no muffled heart sounds. Her ECG showed small-voltage QRS complexes, and chest X-ray revealed cardiomegaly with pulmonary congestion. Her echocardiography showed a large pericardial effusion with a collapse of the right ventricle. An urgent pericardiocentesis was performed, and her symptoms improved after draining 500 cc of pericardial fluid. TFT showed elevated TSH (83.42 m IU/L) with normal free T4 (13.5 pmol/L). She had markedly elevated anti-thyroid peroxidase (>600 IU/mL) and anti-thyroglobulin (>4000 IU/mL). Her pericardial fluid investigations were unremarkable. The patient has been taking her levothyroxine inconsistently with her meals. Her levothyroxine dose was increased from 100 mcg to 150 mcg daily. She showed improvement by the third day of hospitalisation. She was discharged and advised to adhere to the levothyroxine. Her subsequent TFTs normalised with normal echocardiography during the follow-up visit.

Hypothyroidism causes protein-rich pericardial effusion due to increased membrane permeability, increased albumin distribution volume, and diminished lymphatic drainage, which happens gradually over time.

CONCLUSION
Pericardial effusion in hypothyroidism is an infrequent entity. It is more frequent in long-standing clinical hypothyroidism than subclinical hypothyroidism. An early cardiac assessment, adequate thyroid replacement therapy, and medication adherence can help mitigate the risk of pericardial effusion or cardiac tamponade.

EP_A183

SEVERE HYPOTHYROIDISM IN CHRONIC HEPATITIS C INFECTION: A QUANDARY OF AUTOIMMUNITY VERSUS ANTIVIRAL THERAPY

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INTRODUCTION/BACKGROUND
Chronic Hepatitis C virus infection (HCV) may have extrahepatic manifestations, mainly related to autoimmune and malignant disorders. Autoimmune thyroid disease (AITD) may present in up to 10% of cases as influenced by the presence of autoantibodies as well as direct virus invasion that promotes T-cell mediated cytotoxicity.

Besides host factor, interferon previously used as a standard treatment in HCV, is known to cause thyroid dysfunction either by direct inhibitory effect on the thyroid gland or immune activation particularly in those with genetic predisposition to autoimmune disease

Direct-acting Antivirals (DAA) targeting specific non-structural proteins of the virus, hinders viral replication. Since it was introduced, there are few studies demonstrating the effect of DAA on thyroid dysfunction.

We report a case of new-onset hypothyroidism in a patient with HCV soon after commencement of DAA.

CASE

A 53-year-old male, former intravenous drug user with Chronic Hepatitis C Child Pugh A, was treated with DAAs (sofosbuvir and daclatasvir) and ribavirin based on viral load and genotyping. He denied preceding hypothyroid symptoms or family history of thyroid disorder. Two months into treatment, he complained of facial puffiness, weight gain and was eventually admitted for heart failure. Biochemical investigations revealed overt hypothyroidism with FT4 <3.2 pmol/L (7.86 - 14.41) and TSH >300 uIU/mL (0.38 - 5.33) with positive thyroid peroxidase antibody. Levothyroxine was started subsequently, however due to poor compliance, he showed poor clinical and biochemical response in HCV viral suppression and thyroid disorder.

CONCLUSION

Hypothyroidism related to HCV infection is a relatively uncommon association but an important one to diagnose, nonetheless. The condition can be part of the extrahepatic viral manifestation or may be treatment related. Supported by previous study that demonstrated similar effect of DAA on the thyroid gland, further larger RCTs are needed to substantiate this association.

EP_A184

CHALLENGES IN THE DIAGNOSIS AND DIFFERENTIATION OF THYROID HORMONE RESISTANCE FROM TSHOMA

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INTRODUCTION

An elevated fT4 with non-suppressed TSH levels may present as a diagnostic challenge resulting in inappropriate treatment. Although rare, resistance to thyroid hormone (RTH) and TSHomas can present in this manner and diagnosis is important to guide management.

CASE

A 43-year-old male was referred from a district hospital for evaluation of atypical chest pain. He was noted to have an elevated fT4 with normal TSH for the past four years and had received carbimazole previously. He reported symptoms of hyperthyroidism including intermittent palpitations, tremor and anxiety. No goitre was noted clinically. Both his mother and maternal aunt had undergone thyroid surgery. After stopping treatment, he had elevated fT4 (28.31 and 19.39 pmol/L) and normal TSH (1.55 and 1.619 m IU/L) performed on two different platforms. Sex hormone binding globulin (22.6 nmol/L), alpha-subunit (0.22 IU/L) and neck ultrasound were normal. Pituitary MRI showed an ill-defined hypo-enhancing nodule measuring 2.0 mm x 2.2 mm x 1.9 mm. Other pituitary hormones were unremarkable. He went on to have a thyrotropin stimulation test which showed an exaggerated TSH response with an 11-fold increase at 20 minutes, supporting the diagnosis of RTH. Genetic testing was not performed due to resource limitations. Subsequently, he was managed symptomatically with beta blockers.

CONCLUSION

When managing discordant thyroid function tests, a high index of suspicion and proper clinical assessment, including laboratory and imaging studies, are needed to ensure precise diagnosis and avoid potentially harmful or unnecessary treatment such as radioactive iodine, anti-thyroid medication or pituitary surgery. Small non-functioning pituitary adenomas are not uncommon. Abnormal imaging needs to be correlated carefully.

EP_A185

UNVEILING PRETIBIAL MYXEDEMA: A CASE REPORT OF GRAVES' DISEASE

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INTRODUCTION

Pretibial myxedema (PTM), a rare manifestation of Graves' disease, holds importance as it constitutes a component of the classical triad associated with the condition. Historically observed in up to 5% of Graves' disease patients, the incidence of pretibial myxedema has notably decreased, likely attributed to advancements in early diagnosis and prompt initiation of antithyroid therapy.

We present a rare case of biopsy-proven pretibial myxedema in Graves' disease. We reviewed case notes, investigation results, imaging studies and discussed prevalence based on published reports.

CASE

A 39-year-old Chinese male presented with significant weight loss, neck swelling and bilateral lower limb nodular skin lesions. Clinical examination revealed diffuse goiter and bilateral anterior shin swelling. Thyroid imaging showed features consistent with thyroiditis, while bilateral anterior shin lesions indicated pretibial myxedema. Biochemical analysis revealed elevated thyroid function tests and positive thyroid-stimulating hormone antibody levels (>40 IU/L). A skin biopsy confirmed dermal mucinosis consistent with myxedema. Antithyroid medications were initiated. The patient expressed willingness to undergo radioactive iodine treatment if remission is not achieved.

CONCLUSION

Global reported cases of PTM are scarce. In China, a retrospective study revealed a prevalence of 1.6% within thyroid disorders, notably 1.7% in thyrotoxicosis and 0.36% in other thyroid conditions. In Malaysia, reported cases of PTM are minimal. PTM typically coexists with ophthalmopathy, mainly affecting the pretibial region. Pathologically, it results from glycosaminoglycan accumulation triggered by circulating thyrotropin-receptor antibodies, akin to thyroid ophthalmopathy.

In summary, PTM is a rare autoimmune manifestation of Graves' disease, commonly associated with ophthalmopathy and localized to the pretibial region. Clinical diagnosis is typically straightforward, often obviating the need for biopsy, particularly when Graves' disease is active.

EP_A186

CARBIMAZOLE-INDUCED AGRANULOCYTOSIS WITH CONCURRENT SCRUB TYPHUS

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INTRODUCTION

While carbimazole is an effective treatment for hyperthyroidism, it carries a risk of agranulocytosis. Concurrently, rickettsial infections like scrub typhus can worsen neutropenia. We reviewed case notes, investigation results, imaging studies and treatment options based on a literature review.

CASE

A 55-year-old male farmer with hyperthyroidism on high-dose carbimazole treatment sustained a machete injury to his left middle finger. Upon presentation, he had fever, normal thyroid function, stable hemodynamics, severe neutropenia (total white count 0.4 × 10^9/L, absolute neutrophil count 0.02 × 103/μL) and typhus eschars. He was treated with doxycycline, piperacillin-tazobactam and subcutaneous granulocyte-colony stimulating factor (G-CSF). Abnormal thyroid function (FT4 46 pmol/L and TSH <0.01 m IU/L) and elevated C-reactive protein (234 mg/L) were also observed. Carbimazole was discontinued and replaced with oral cholestyramine and lithium. Positive serologic findings confirmed scrub typhus. With targeted treatment and G-CSF support, the patient's condition improved, as evidenced by normalized blood counts. Radioactive iodine therapy was contemplated once thyroid function was controlled.

CONCLUSION

Carbimazole carries the risk of severe adverse effects, including agranulocytosis. This risk may be compounded with a concurrent rickettsial infection, which can also cause neutropenia. Diagnosis relies on clinical suspicion and profound neutropenia, requiring thorough evaluation including serological tests and PCR to differentiate between agranulocytosis-related and rickettsial infections. Immediate discontinuation of carbimazole and replacement with alternative antithyroid drugs is necessary, often supplemented with broad-spectrum antibiotics and G-CSF to prevent overwhelming infection risks. Tailored antibiotic therapy should also be administered for the rickettsial infection. Prompt recognition and intervention are crucial, particularly in endemic areas. Early diagnosis and aggressive management can help mitigate morbidity and mortality. Educating patients on symptom recognition remains the most effective preventive measure.

EP_A187

"SWINGING HEART" IN A SEVERELY HYPOTHYROID PATIENT: A CASE REPORT

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INTRODUCTION

Hypothyroidism is a disorder with multiorgan involvement that may lead to various complications. Pericardial effusion is commonly seen in cases of severe hypothyroidism, which may deteriorate into life-threatening cardiac tamponade. Early diagnosis and management of pericardial effusion in hypothyroidism is crucial.

CASE
A 45-year-old female was brought to the emergency department with breathlessness and pleuritic chest pain. She had a background history of diffuse large B-cell lymphoma of the thyroid gland stage 2Bx. She had achieved complete remission for the past year following thyroidectomy and a full course of chemotherapy in December 2022. Thyroxine replacement was only given for a month post-debulking thyroid surgery. She had hypotension (79/56 mm Hg), tachycardia (129 bpm) and muffled heart sounds. Chest X-ray showed globular enlargement of the cardiac silhouette with ''water bottle" configuration and right pleural effusion. Echocardiogram demonstrated early diastolic right ventricular collapse with a large pericardial effusion. The heart was seen swinging within the effusion, suggestive of cardiac tamponade. Urgent pericardiocentesis drained 200 mL of exudative serous fluid. There was no growth on pericardial fluid culture, and cytology was negative for malignant cells. Tests showed severe hypothyroidism (TSH >51.6 m IU/L and fT4 <3.2 pmol/L). Intravenous levothyroxine 50 mcg was given for two days. This was converted to thyroxine 100 mcg orally daily, then increased to 200 mcg daily based on serial thyroid function tests. She was discharged well after eight days of hospitalization. Her latest tests showed improved TSH (24.07 m IU/L) and fT4 (14.03 pmol/L).

CONCLUSION
A high index of suspicion is important for timely diagnosis of cardiac tamponade due to severe hypothyroidism, followed by prompt intervention. While it is a treatable cause of cardiogenic shock, it may be fatal if left unrecognized.

EP_A188

AUTOIMMUNE HEMOLYTIC ANAEMIA: A RARE MANIFESTATION OF GRAVES' DISEASE

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INTRODUCTION
Anemia is an atypical manifestation of Graves' Disease (GD). Autoimmune hemolytic anaemia (AIHA) is one of the rarest anemias seen in GD.

CASE
A 55-year-old female presented with failure symptoms and palpitations, with no other hyperthyroid symptoms. She was tachycardic (130 to 140 bpm) with an irregularly irregular heart rhythm. She had pallor, jaundice, thyromegaly without bruit, fine crepitations on both lung fields and pedal edema. She had no murmur or hepatosplenomegaly. Chest radiograph showed congested lungs. Initial blood investigations showed normochromic normocytic anaemia (hemoglobin 6.7 g/dL), and normal WBC and platelet count. Hemolytic workup showed elevated indirect bilirubin (63 µmol/L), positive direct Coombs test, high reticulocyte count (10.8%), and RBC agglutination with few spherocytes on full blood picture. LDH was normal. She had elevated fT4 (58.5 pmol/L), low TSH (<0.005 m IU/L) and high levels of antithyroid peroxidase antibodies (84 IU/mL). She was not in thyroid storm. She was treated with carbimazole 30 mg OD and prednisolone 30 mg OD with respective tapering doses. She was well during our clinic review two weeks later. Hemoglobin (10.4 g/dL) and indirect bilirubin (28 µmol/L) improved without blood transfusion.

CONCLUSION
The presentations of Graves' disease may vary. While pernicious and iron deficiency are the common causes of anemia in GD, AIHA is rare. It is crucial to screen for thyroid disease in AIHA. The scarcity of pure AIHA manifestation in GD may potentially delay the diagnosis and lead to poor patient outcomes. AIHA in GD responds well to antithyroid and steroid.

EP_A189

HAEMOPTYSIS AND HIDDEN THREATS: UNRAVELLING FOLLICULAR THYROID CANCER IN PREGNANCY

https://doi.org/10.15605/jafes.039.S1.200

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INTRODUCTION
Haemoptysis in pregnancy is a rare but serious complication that demands prompt investigation and intervention. While pulmonary embolism is often considered, it is essential to explore other potential underlying pathologies such as lung cancer, bronchiectasis or infectious causes. In rare cases, metastatic differentiated thyroid cancer may present with haemoptysis as the primary symptom.

CASE
We describe a distinctive case involving a 30-year-old female in her 34th week of twin pregnancy. She had a history of left partial thyroidectomy four years prior for a large thyroid nodule. Histopathology showed papillary-like nuclear features in favour of adenomatous hyperplasia. Haemoptysis began at 20 weeks of pregnancy

and increased significantly in amount at 34 weeks. There were no constitutional symptoms. On examination, she was not tachypnoeic, with SpO_2 98% under room air, BP 133/70 mmHg and HR 108 bpm. She appeared clinically euthyroid, with unremarkable physical findings. Initial blood tests including full blood counts, renal profile and liver function tests were all within normal ranges. Thyroid function tests indicated normal TSH (3.699 m IU/L) and fT4 (13.27 pmol/L). However, D-dimer levels were elevated (3.04 mg/L). Tuberculosis screening with sputum AFB was negative. CT imaging showed multiple bilateral scattered enhancing lung nodules, predominantly in the lower lobes, with possible haemorrhagic nodules in the left lower lobe. She was scheduled for elective caesarean section at 36 weeks with combined care from the respiratory, obstetric and anaesthesia teams.

CT-guided biopsy of the lung lesion at two weeks post-delivery confirmed metastatic follicular thyroid carcinoma.

CONCLUSION

This case highlighted the slow and insidious nature of differentiated thyroid cancer with lung metastases which unfortunately were presented during pregnancy. Thorough evaluation in pregnant patients presenting with haemoptysis are of utmost importance.

EP_A190

DIFFERENTIATED THYROID CANCER WITH POSITIVE ANTI-THYROGLOBULIN ANTIBODY AND FINDINGS OF DISEASE EVALUATION ON FDG PET-CT SCAN

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INTRODUCTION

Fluorodeoxyglucose positron emission tomography-computerised tomography (FDG PET-CT) imaging has been advocated in differentiated thyroid cancer (DTC) cases with negative radioiodine scan but elevated serum thyroglobulin. Although there is limited available data, FDG PET-CT is also used to assess patients with progressively increasing anti-thyroglobulin antibodies (anti-TG). We aimed to determine characteristics of anti-TG positive DTC patients referred for FDG PET-CT and their association with abnormal imaging findings.

METHODOLOGY

We performed a cross-sectional retrospective study of all DTC patients with positive anti-TG who were managed with radioiodine therapy in our institution and referred for FDG PET-CT. Those who defaulted on the PET-CT appointment and clinic follow up with incomplete documentation were excluded. Baseline nodal disease and metastasis were determined following the first radioiodine therapy.

RESULT

Majority were females (70.8%). Mean values for age and cancer duration were 51.29 and 7.13 years respectively. Papillary thyroid carcinoma was predominant (95.8%). Majority had baseline nodal involvement (87.5%); a quarter had detectable distant metastasis. Most patients underwent less than five sessions of radioiodine therapy (83.3%). Majority had negative radioiodine scan prior to PET-CT (87.5%). Cases of positive residual radioiodine-avid disease showed lower mean values of anti-TG compared to those with negative radioiodine scan (1423 vs. 4671 IU/mL, p <0.05). FDG-avid malignant disease was observed in 62.5%; three patients were considered to have mixed disease following the PET-CT assessment. Female gender and those with baseline nodal involvement were found to be significantly associated with FDG-avid disease (p <0.05).

CONCLUSION

FDG PET-CT has an important role in evaluating DTC patients with positive anti-TG. Those with negative radioiodine scan had higher mean values of anti-TG. Females and patients with baseline nodal involvement were associated with FDG-avid disease.

EP_A191

CLINICAL CHARACTERISTICS AND DEFINITIVE TREATMENT FOLLOWING THYROID STORM: 10-YEAR EXPERIENCE

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INTRODUCTION

The prevalence of thyroid storm is 0.2 per 100,000 people per year with mortality rates varying from 11% to 25%. Given the rarity of occurrence and the high mortality rates associated with thyroid storm, it is imperative to understand the definitive therapy pattern following such an event.

METHODOLOGY

We conducted a retrospective review of the medical records of patients diagnosed with thyroid storm at Hospital Putrajaya between September 2013 and December 2023. Demographic data, comorbidities, definitive treatments, and patient outcomes were collected and analysed.

RESULT

We included 30 patients with thyroid storm. Most of the subjects were Malay and female. Mean age at diagnosis of thyroid storm was 42 years. Thyroid storm occurred after a mean duration of 57 months following diagnosis of thyrotoxicosis. Seven patients (23.3%) presented with thyroid storm as the initial clinical manifestation of thyrotoxicosis. Most were triggered by respiratory tract infections, followed by gastrointestinal and urinary tract infections. Three patients (10%) died due to multiorgan failure. Thirteen subjects (43.3%) received definitive treatment: twelve (40%) received radioactive iodine and one underwent thyroidectomy. Eight (26.7%) defaulted on follow-up. Three patients (10%) were on medical therapy and undecided on definitive treatment. The mean duration between thyroid storm and definitive treatment was about 5 months.

CONCLUSION

In our cohort, majority of patients with thyroid storm had similar clinical characteristics to those in previous studies. The mortality rate was slightly lower than in previous studies. Less than half of the patients received definitive treatment. Therefore, measures should be taken to improve our post-thyroid storm management.

Basic Science E-Poster

EP_B001

GLUCOCORTICOID WITHDRAWAL SYNDROME MASQUERADES AS UNEXPLAINED PERSISTENT FEVER

https://doi.org/10.15605/jafes.039.S1.203

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INTRODUCTION/BACKGROUND
With the availability of over-the-counter medicine, individuals have turned to the convenient means of receiving their supply of medicine instead of thorough physician consultation. Steroids are one of the over-the-counter medicines that are often used inappropriately to manage chronic pain. When steroids are used at supraphysiological doses for a prolonged period, it suppresses the hypothalamic-pituitary-adrenal axis. In this review, we present a case of unexplained persistent fever which responds miraculously to steroids.

CASE
A 77-year-old male with underlying type 2 diabetes mellitus, hypertension, dyslipidemia, chronic obstructive pulmonary disease and degenerative spine disease presented with a 3-day history of diarrhea, abdominal pain, poor appetite and altered mental status. Skin examination shows skin thinning and multiple bruises. X-rays, imaging and CT scan were unremarkable. One week after hospitalization, he developed a fever, which was attributed to thrombophlebitis of the left forearm. Multiple antibiotics were infused: IV Ceftriaxone, IV Piperacillin / Tazobactam, IV Meropenem, IV Vancomycin and T Doxycycline. There is no positive culture, aside from the urine culture growing *Enterococcus faecalis*, susceptible to Vancomycin. Still, the fever persisted until the trial of IV hydrocortisone 100 mg TDS for the possibility of fever caused by glucocorticoid withdrawal syndrome. After 1 full day of IV hydrocortisone, the fever resolved.

CONCLUSION
Unexplained fever remains a diagnostic and therapeutic challenge. A full workup of fever does not reveal the source of infection. Despite multiple antibiotics, fever does not settle until introduction of glucocorticoid. With suggestive history of chronic pain, skin thinning and remarkable response to glucocorticoid, glucocorticoid withdrawal syndrome is one the causes of unexplained persistent fever that we should all not miss.

EP_B002

SIRT1-SRSF10 PATHWAY PROMOTES BROWN-LIKE FEATURES OF WHITE ADIPOCYTES

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INTRODUCTION/BACKGROUND
Energy expenditure predominantly occurs in brown adipose tissue (BAT), therefore promoting BAT-like features or inducing the browning of white adipose tissue (WAT) can be an attractive approach to reduce obesity. Studies have found the interplay between splicing factor SRSF10 and Sirt1-mediated pathway during adipocyte differentiation and lipid metabolism in obese liver. Therefore, we aimed to investigate whether activation of Sirt1-SRSF10 can lessen lipid accumulation in white adipocytes and induce the expression of typical genes of BAT *in vitro*.

CASE
3T3-L1 cells were differentiated into mature adipocytes for 10 days. The cells were also treated with Sirt1 activator, Sirt1 Inhibitor or Rosiglitazone throughout the adipogenic differentiation period and gene expression was analysed by real-time polymerase chain reaction.

Upregulation of Sirt1 was directly proportional to the level of SRSF10 in differentiated adipocytes resulting in lesser intracellular lipid accumulation. Expectedly, attenuation of Sirt1 activity enhanced lipid production in the cells. Lipin1, one of SRSF10-affected splicing events implicated in adipogenesis was further investigated and its variant *Lipin1a* was found significantly increased as compared to *Lipin1b*. Finally, the expression of 'browning' genes such as *PGC1a* and *Cidea* were upregulated in Sirt1-activated adipocytes.

CONCLUSION
Overall, Sirt1 affects important splicing events via SRSF10 during adipocyte differentiation hence preventing excessive lipid accumulation in vitro. It also promotes the browning of white adipose tissue, indicating that the Sirt1-SRSF10 pathway can be a potential drug target to reduce obesity.

EP_B003

IMPACT OF PHARMACIST-LED DIABETES EDUCATION ON PATIENTS' ACHIEVEMENT ACCORDING TO INDIVIDUALIZED DIABETES TARGET BASED ON PATIENT CHARACTERISTICS

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INTRODUCTION/BACKGROUND

The latest evidence showed setting a general HbA1c target for all patients is outdated. No published study in Malaysia investigates the patients' achievement based on individualized diabetes targets. The study aims to focus on the impact of pharmacist-led diabetes education on patient achievement based on individualized HbA1c targets.

CASE

This retrospective cross-sectional study involved 306 patients who completed the Diabetes Medication Therapy Adherence Clinic (DMTAC) in 2020. They were conveniently sampled across 13 health clinics in Kota Tinggi district. Gestational diabetes mellitus patients and those under treatments that may affect the HbA1c level were excluded. Target HbA1c level was stratified according to the patient's characteristics. Data obtained from electronic databases and medical reports were analysed using SPSS version 28.0.0. Descriptive analyses were utilized to compare differences in achievement between individualized and general HbA1c targets. Chi-square test of independence analysed the association between the HbA1c target group and age group with pre-stratified target achievement.

More patients were able to achieve individualized HbA1c targets compared to the general HbA1c targets (25.5% vs 13.1%, respectively). A significant association was found between the HbA1c target group and target achievement (p <0.001) with the most achievable individualized HbA1c target group being 7.1-8% (43.9%). Age group and target achievement were found to be significantly associated when using individualized HbA1c targets (p <0.001), but not when using a general HbA1c target (p = 0.189).

CONCLUSION

An individualized HbA1c target gives better achievement compared to the general HbA1c target across diabetic patients under the DMTAC service. This suggests integrating individualized HbA1c targets in diabetes management.

Paediatrics Oral Presentation

OP_P001

TRENDS OF CENTRAL PRECOCIOUS PUBERTY AMONG CHILDREN FROM 2004 TO 2024

https://doi.org/10.15605/jafes.039.S1.206

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INTRODUCTION
Temporal trends worldwide demonstrate evidence of an earlier onset and progression of puberty. This study aims to describe the trends in precocious puberty among children in Hospital Putrajaya between 2004 to 2024.

METHODOLOGY
Data retrieved from the electronic database were reviewed. All patients diagnosed to have precocious puberty (i.e., onset of puberty before age 8 years for girls and 9 years for boys) in the Department of Paediatric Endocrinology from January 2004 until April 2024 were included. These patients were stratified according to the diagnosis of idiopathic central precocious puberty (CPP) and normal variant puberty [i.e., premature thelarche (PT) or premature adrenarche (PA)].

RESULT
A total of 89 children were registered with a diagnosis of CPP, PT and PA. They were predominantly girls (96.6%), with median (interquartile) age at diagnosis of 7 years (6;10) for boys and 7 years (2;9) for girls. Majority were Malay (60.7%), followed by Chinese (28.1%), Indian (9%) and Nigerian (2.2%). Majority of the cases were idiopathic CPP (91%), with a median (interquartile) LH:FSH ratio of 1.4 (0.16;7.23). The MRI findings were a mix of normal (35.3%), pituitary microadenoma (34.1%) and pineal gland cyst (1.12%). There was a general increase in the number of cases of CPP over time, from 25.8% between 2013 to 2018, to 68.5% between 2019 to 2024. Nearly half of the cohort had a body mass index (BMI) of overweight and obese (41.3%), with median (interquartile) bone age of 4 years (2;7).

CONCLUSION
This study demonstrated an increase in the number of patients with central precocious puberty over time. We also demonstrated a possible association with increased BMI and earlier onset of puberty in girls.

OP_P002

LIFESTYLE INTERVENTION IMPROVES CARDIOMETABOLIC PROFILES AMONG CHILDREN WITH OBESITY

https://doi.org/10.15605/jafes.039.S1.207

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INTRODUCTION

There has recently been a surge of interest in the metabolic phenotype among children with obesity characterized by the absence of associated cardiometabolic risk factors (CRFs), known as metabolically healthy obesity (MHO), as opposed to those with metabolically unhealthy obesity (MUO). This study investigated the effect of lifestyle intervention on CRFs among children with MHO and MUO.

METHODOLOGY

A total of 102 school-age children with obesity age 8 to 16 years completed a 16-week school-based lifestyle modification intervention program, MyBFF@school phase I. The intervention consisted of physical activity, healthy eating promotion, and psychological empowerment. MHO and MUO statuses were defined based on the 2018 consensus-based criteria.

RESULT

After the intervention, the CRFs of the children with MUO improved with significant decreases in systolic ($p<0.001$) and diastolic ($p = 0.01$) blood pressure and a significant increase in high-density lipoprotein cholesterol ($p = 0.005$), while the CRFs of the children with MHO had a significant decrease in uric acid ($p = 0.04$). Additionally, 51.6% of the children with MHO transitioned to the MUO, while 26.8% of the children with MUO crossed over to the MHO at the end of the intervention. Furthermore, the odds of having high systolic blood pressure among children with MUO were 59% lower at week 16 compared to baseline [OR 0.41, 95%CI (0.18, 0.92)] ($p = 0.03$).

CONCLUSION

Our findings demonstrated that CRFs improved more prominently among children with MUO following the intervention. More important, our findings indicate that MHO in children is transient, hence, strategies to protect children against MUO are warranted.

OP_P003

HORMONAL AND METABOLIC OUTCOMES OF PAEDIATRIC CONGENITAL ADRENAL HYPERPLASIA

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INTRODUCTION

Individuals with congenital adrenal hyperplasia are exposed to hyperandrogenism in utero and need a lifelong replacement of supraphysiological corticosteroids and mineralocorticoids. Hence, they are at risk for complications from both the disease and treatment.

METHODOLOGY

Sixty-eight paediatric patients with CAH were included in a retrospective cross-sectional study. We analysed the clinical and biochemical profiles at the initial presentation and the latest visit. Information was extracted from a digital database.

RESULT

Most patients were female (51%), Malay (82%), and diagnosed with salt-wasting CAH (77.9%). Of these, 76.3% received the diagnosis after the first week of life, including 10.3% of which were diagnosed after the first year of life. Central precocious puberty was seen in 20.5% at a mean age of 6.1 ± 1.79 years. Testicular adrenal rest tumor was detected in 10.3% at a mean age 9.8 ± 4.6 years. Overweight and obesity was seen in 36.7%. Hypertension, seen in 5.8%, was detected at mean age of 5.5 ± 2.1 years; while dyslipidemia in 4.4% was diagnosed at a mean age of 10 ± 4 years. None had diabetes. Eight females underwent surgical feminizing surgeries at a mean age of 6.1 ± 3.4 years. Both genders exhibited a short final height (FH) at completion of growth. The mean FH and final height standard deviation score (FHSDS) were 155.6 ± 4.4 cm and -2.53 ± 0.71 in males, and 142.0 ± 6.9 cm and -4.64 ± 2.98 in females, respectively. Mean bone age advancement was +2.4 ± 1.6 years in males and +1.2 ± 0.8 years in females.

CONCLUSION

Most of the patients had a late diagnosis, hence a high index of suspicion for the diagnosis is crucial during initial evaluation. Close monitoring is important while ensuring compliance to the therapy is important as metabolic and hormonal complications start at pre-pubertal age.

OP_P004

DIAGNOSTIC ACCURACY OF ARM SPAN-TO-HEIGHT RATIO IN DIFFERENTIATING PHYSIOLOGICAL AND PATHOLOGICAL SHORT STATURE

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INTRODUCTION
Physiological causes account for the most common etiology of short stature (SS). However, it may be the first manifestation of an underlying pathological condition. Thus, careful evaluation and identification of the underlying etiologies is crucial for early prevention and treatment. In order not to miss significant pathological causes or overtreatment of physiological causes of SS, this study aimed to describe the proportion of short stature (SS) based on its etiology in Paediatrics Clinic Hospital Universiti Sains Malaysia and determine the accuracy of arm span-to-height (AS/H) ratio in differentiating physiological and pathological SS.

METHODOLOGY
A cross-sectional retrospective study was conducted and a total of 106 patients were analyzed for their demographics, clinical characteristics, aetiology, bone age and auxological data. AS/H ratio was evaluated for its accuracy using the receiver operating characteristic (ROC) curve and its sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were calculated.

RESULT
In decreasing order of frequency, the causes of SS were endocrine disorders (28.3%), physiological (22.6%), syndromes (18.9%), chronic systemic illness (13.2%), low birth weight (10.4%) and disproportionate group (6.6%). The sensitivity and specificity of theAS/H ratio as an auxological tool to differentiate physiological from pathological causes of SS were 93% and 40%, respectively, with an AUC of 0.65 (p-value 0.05), at 95%CI (0.54, 0.77).

CONCLUSION
Endocrine disorders accounted for the most common cause of SS among 106 patients referred to our institution. The AS/H ratio has 93% sensitivity and 40% specificity in differentiating physiological from pathological causes of SS.

OP_P005

DIAGNOSIS AND MANAGEMENT OF CHILDHOOD OBESITY: A SURVEY OF DOCTORS WORKING IN PRIMARY CARE

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INTRODUCTION
Childhood obesity has increased steadily over the years. Primary health care doctors are in the best position to tackle this issue. This study aims to identify the factors affecting the ability to correctly define and manage childhood overweight or obesity and explore the usage of obesity clinical practice guidelines (CPG) in their practice.

METHODOLOGY
A cross-sectional study was carried out on a total of 246 primary health care doctors in Klang Valley region. A questionnaire was used to determine the prevalence of doctors who can correctly identify and manage childhood overweight or obesity and explore the use of the CPG in their practice.

RESULT
Forty percent of primary health care doctors answered the definition of childhood overweight correctly. Eighty percent prescribed correct intervention. Majority performed the measurement and screened the complications of childhood obesity. Government clinic doctors were more likely to correctly define [p <0.001, OR 2.52, 95%CI (1.49, 4.27)], diagnose [p = 0.001, OR 3.03, 95%CI (1.52, 6.03)] and manage [p = 0.005, OR 0.38, 95%CI (0.19, 0.76)] childhood obesity. Usage of CPG in 49% of the doctors increased the likelihood of diagnosing [p = 0.035, OR 2.05, 95%CI (1.04, 3.96)] and managing [p = 0.006, OR 2.65, 95%CI (1.30, 5.41)] childhood obesity.

CONCLUSION
Although majority of the primary health care doctors correctly diagnosed and managed childhood obesity, there are fewer private compared to government clinics. An approach focusing on creating more awareness in that sector would increase the effectiveness of primary health care doctors as they are the first line in the management of childhood obesity. The CPG would be a useful tool in the endeavour.

Paediatrics Best Poster Presentation

PP_P001

CYSTIC FIBROSIS-RELATED DIABETES: A SMALL COHORT IN MALAYSIA

https://doi.org/10.15605/jafes.039.S1.211

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INTRODUCTION/BACKGOUND
Cystic fibrosis (CF) has a high prevalence in Europe and America but is very rare in Malaysia. Here we report a small cohort of patients with cystic fibrosis-related diabetes (CFRD) from University Malaya Medical Centre (UMMC), a main tertiary centre in Malaysia. We searched through the registry of patients diagnosed with CFRD in our centre from 2019 to 2024. We compared their demographic, clinical and management data related to CF and CFRD.

CASES
There were four patients in our registry. Three of them were diagnosed with CF requiring pancreatic enzyme supplements and had positive sweat tests, but only one had a confirmed genetic test. The fourth patient was diagnosed with non-CF bronchiectasis and pancreatic insufficiency, given negative sweat tests and genetic tests. All of them developed complications related to CF.

The patients developed CFRD about 9 to 17 years after diagnosis of pancreatic insufficiency, mostly triggered by steroid medication. They had hyperglycemia, high HbA1c, hyperosmolar symptoms and poor body mass index (BMI) at diagnosis. All had normal C-peptide; none had developed ketoacidosis. The third patient was diagnosed with CFRD from an oral glucose tolerance test (OGTT). She had poor weight gain but no hyperosmolar symptoms.

All except the third patient were started on basal-bolus insulin during diagnosis of CFRD, and subsequently transitioned to mixed insulin or only bolus insulin. The third patient opted for a trial of metformin. We faced challenges with various insulin regimes. Most of them had high insulin requirements with a high-calorie diet and steroid treatment. However, they were also prone to hypoglycemia at fasting.

CONCLUSION
CFRD is a unique condition. Even though insulin is the mainstay of treatment, the best regime is not easy to determine, given the fluctuating nature of the disease, multiple comorbidities, and psychosocial issues. The physician should always explore the condition with sympathy, empathy and understanding.

PP_P002

REPORT FROM THE PAEDIATRIC DIABETES MELLITUS REGISTRY (MYHDW PRIS): A SINGLE CENTRE EXPERIENCE ON TYPE 1 DIABETES MELLITUS (T1DM)

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INTRODUCTION
The Malaysian Health Data Warehouse (MyHDW) is a source of comprehensive healthcare data structured for query and analysis purposes. Data generated in granular forms are collected via Sistem Maklumat Rawatan Pesakit (SMRP). Data for a specific disease is further collected by the Patient Registry Information system (PRIS). There is dependency between the two systems to minimize duplication of data entry and to maintain data integrity. The distribution of the data variables between SMRP and PRIS is harmonised.

METHODOLOGY
We analysed the data variables generated from PRIS which included the summary of characteristics of 167 patients with diabetes mellitus notified in 2005 to 2022. We present the preliminary findings here for all diabetes patients aged 19 and below in a single centre.

RESULT
Our findings are in keeping with the technical report of Diabetes in Children and Adolescents Registry 2005 to 2022, where 70% or more of patients had T1DM, presented predominantly at age 5 to 14 years, with 66% having DKA at presentation. Upon diagnosis, 100% T1DM patients received insulin, 16% of which were on insulin analogues. All T1DM patients practiced SMBG. However, only 8% do ketone testing during sick days/severe hyperglycemia. Out of 117 T1DM patients, 83% were on active follow-up. Only 15% were able to achieve HbA1c below 7.5%.

CONCLUSION
We were able to achieve one of the WHO Global Targets for Diabetes from the 75th World Health Assembly, wherein 100% of persons with T1DM have access to affordable insulin treatment and SMBG. However, more efforts need to be done to improve ketone testing and HbA1c levels. As global incidence of diabetes mellitus continues to increase, nationwide participation is crucial to monitor diabetes care and outcomes to guide future healthcare planning for the country.

PP_P003

THYROID NODULES IN CHILDREN: OUTCOME AND THERAPY

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INTRODUCTION
Thyroid nodules occur less frequently in children compared to adults. The estimated prevalence of solid thyroid nodules is approximately 1% to 1.7% of the paediatric population, with the incidence increasing with age. This study aimed to describe the prevalence and outcomes of paediatric patients with thyroid nodule.

METHODOLOGY
Children and adolescents diagnosed with thyroid nodules from 2012 to 2024 at the University Malaya Medical Centre were included in this retrospective review.

RESULT
Twelve patients were referred and diagnosed with thyroid nodule (s). Most were female (91.7%), with a mean age at diagnosis of 12.97 ± 1.97 years. Multinodular goitre was seen in 58.3%. The prevalence of thyroid nodules among paediatric patients with thyroid disease including congenital and acquired cases in our centre was 2%. Seven (58.3%) were Chinese, 4 (33.3%) Malay, and 1 (8.3%) Indian. Seven (58.3%) patients presented with goitre, while 4 (33.3%) were referred post-total thyroidectomy and 1 (8.3%) with symptoms of hyperthyroidism (tremors and palpitations). Eight (66.7%) patients were euthyroid and 3 (25%) were hypothyroid post-total thyroidectomy. Fine needle aspiration cytology was done in 10 (83.3%) patients. Four (33.3%) were confirmed malignant. Of the 4 patients with papillary thyroid cancer, 3 (75%) patients were initially diagnosed with a solitary thyroid nodule. Their mean age at presentation was 12.83 ± 1.66 years, and all underwent total thyroidectomy. One (8.3%) patient developed hypercalcaemic seizure after thyroidectomy. None developed vocal cord paralysis.

CONCLUSION
The prevalence of thyroid nodules in our centre is similar to other studies, with multinodular goitre being most diagnosed. The majority of adolescents with papillary thyroid cancer presented with solitary thyroid nodules. Total thyroidectomy in papillary thyroid cancer is safe with minimal complications.

PP_P004

THE PREVALENCE OF HYPOPHOSPHATEMIA AND ITS ASSOCIATED RISK FACTORS IN DIABETIC KETOACIDOSIS PATIENTS

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INTRODUCTION
DKA is a serious complication of DM. It causes multiple electrolyte imbalances. Hypophosphatemia is one of the unrecognized complications. The study of DKA with hypophosphatemia is scarce as moderate hypophosphatemia is harmless to patients, however, severe hypophosphatemia causes significant complications. We aimed to study the prevalence of hypophosphatemia and its associated risk factors among DKA pediatric patients.

METHODOLOGY
We recruited 65 patients aged 7 months to 18 years old, admitted to HUSM for DKA. We studied their socio-demographics, past medical history, summary of current hospital admission, and physical examination, and analyzed their biochemical data. DKA was diagnosed based on the criteria by ISPAD. Multiple logistic regression models were used to examine the association between variables and DKA.

RESULT
The prevalence of hypophosphatemia in DKA was highest on day 1 of admission with 70.8% with a mean age of 11 years old on presentation. Multiple logistic regression analysis showed plasma bicarbonate at day 3 [adjusted odds ratio (OR) 1.2, with p-value of 0.027] and baseline hemoglobin [adjusted OR 0.62, with p-value 0.009] were the significant factors of hypophosphatemia associated with DKA pediatric patients.

CONCLUSION
The prevalence of hypophosphatemia in DKA pediatric patients admitted to our center was highest on day 1 of admission. Risk factors associated with hypophosphatemia were family history of DM, DKA severity, heart rate, duration and percentage of fluid correction, urine ketone, blood gas pH on admission, and plasma bicarbonate on day 3, baseline hemoglobin, serum calcium, and albumin. h However, plasma bicarbonate on day 3 and baseline Hb were the only significant risk factors of hypophosphatemia in DKA patients.

PP_P005

THE CHALLENGE OF METABOLIC CONTROL IN CONGENITAL GENERALISED LIPODYSTROPHY TYPE 2 (BERARDINELLI-SEIP SYNDROME)

https://doi.org/10.15605/jafes.039.S1.215

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INTRODUCTION

Berardinelli-Seip congenital lipodystrophy (BSCL) is a heterogeneous autosomal recessive disorder characterised by an almost total lack of adipose tissue in the body, associated with the progressive development of metabolic complications. There are four different subtypes (I to IV) resulting from mutations in AGPAT2, BSCL2, CAV1 and PTRF genes, respectively. We investigated the characteristics of BSCL2 variants in Sarawak patients.

METHODOLOGY

The clinical features and laboratory indices were obtained by medical interview and medical records review.

RESULTS

Patient 1 was a 19-year-old female diagnosed with lipodystrophy at 5 months, developed dyslipidaemia at 2 years and type 2 diabetes mellitus (T2DM) at 7 years. Her diabetes was difficult to control with metformin and insulin, as evidenced by progressive worsening of HbA1c from 7.4% to 12.1%.

Patient 2 was a 13-year-old male, the younger brother of patient 1. He had dyslipidaemia and T2DM detected at 7 years. Glycaemic control was suboptimal with metformin and insulin, with HbA1c ranging from 9.8% to 14.7%. Subcutaneous premixed human insulin was the initial insulin of choice due to its ease of administration. Inevitably, both progressed to diabetic nephropathy and fatty liver. Recent efforts to intensify glycaemic control with basal-bolus insulin regimen were deemed promising.

Patient 3 was a 7-year-old female who had T2DM at three years. Dyslipidaemia was apparent at one year of age.

Patient 4 was a 3-year-old female, the sibling of patient 3. She had not developed any metabolic complications and was under close surveillance.

Parental consanguinity was identified. All four patients resided in the same village. All of them exhibited a novel homozygous duplication mutation of c.567_573+1dupGAACTCGG.p. in intron 5.

CONCLUSION

The onset of metabolic complications occurs early in the first decade of life in BSCL. Optimal metabolic control is challenging in this condition.

Paediatrics E-Poster

EP_P001

A CHILD WITH AN AGGRESSIVE FUNCTIONAL ADRENOCORTICAL CARCINOMA

https://doi.org/10.15605/jafes.039.S1.216

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INTRODUCTION
Adrenocortical carcinomas, among the rarest and most aggressive paediatric endocrine tumours, manifest with diverse symptoms like virilization, Cushing's syndrome, or both.

CASE
We present a case of functional adrenocortical carcinoma in a female aged 6 years and 7 months, who initially presented with hypertensive encephalopathy and hypokalaemic hypochloremic metabolic alkalosis, which resolved with symptomatic treatments. Ten months later, she presented with frank Cushing's syndrome, refractory hypertension, generalised virilization, extensive skin fungal infection and severe backache. Breast cancer was diagnosed in her maternal aunt. Hormonal tests showed non-ACTH dependent hypercortisolism and marked elevation of androgens. Computed tomography revealed a large left suprarenal mass, with multi-focal liver lesions and lung nodules suggestive of distant metastasis, left renal vein thrombosis and multiple osteoporotic vertebral fractures.
A clinical diagnosis of stage 4 functional adrenocortical carcinoma was made. While complete surgical removal of the tumour is the gold standard, it was not feasible immediately due to the substantial size of the tumour and presence of distant metastases. Neo-adjuvant chemotherapy was started. Mitotane and ketoconazole were introduced concomitantly to control hypercortisolism, with initial success. Hydrocortisone replacement was needed for a short period when there was a rapid decline in cortisol levels following chemotherapy. Unfortunately, with poor commitment from the family, the disease advanced rapidly with worsening lung and liver metastases. Following a family conference, the parents opted for palliative treatment with mitotane monotherapy, and the child was transferred to the district hospital for comfort care.

CONCLUSION
Medical treatment is useful in controlling the symptoms of severe hypercortisolism. Steroid replacement may be needed with the use of adrenolytic agent. Adrenocortical carcinoma is aggressive and a high index of suspicion is needed for early diagnosis.

EP_P002

CONGENITAL HYPERINSULINISM SECONDARY TO ABCC8 MUTATION: A CASE STUDY

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INTRODUCTION
Congenital hyperinsulinism (CHI) results in persistent hypoglycaemia beyond infancy. Mutations in the ABCC8 and KCNJ11 genes are the most common aetiologies of congenital hyperinsulinism that leads to inappropriate insulin secretion irrespective of hypoglycaemia.

CASE
A 3-month-old male was referred to the clinic for persistent hypoglycaemia. He was born with a birth weight of 4480 g by elective caesarean section for macrosomia. His mother had an uneventful antenatal period and had a normal OGTT during the pregnancy.

Post-delivery, he was initially well until when he developed hypoglycaemia at 28 hours of life. He was transferred to the NICU from the postnatal ward. Hypoglycaemia was persistent requiring high glucose delivery rate up to 10 mg/kg/min. Glucagon infusion was started and was difficult to wean. Hypoglycaemic hyperinsulinaemia was confirmed at day 21 of life, with concomitant results of insulin 3.4 m IU/L and random blood glucose 1.8 mmol/L. Diazoxide was started. Glucagon infusion was stopped at day 26 of life, and the baby was discharged.

He was born from a non-consanguineous marriage. He was the second child in the family. His parents and older sister were well and healthy. There was no known history of neonatal hypoglycaemia nor early-onset diabetes in his family.

At 2 years, the patient still required diazoxide with episodes of hypoglycaemia when feeding was delayed. He was referred to the genetic team. Further investigation revealed compound heterozygous mutations at the ABCC8 gene (likely autosomal recessive type) which were c.2992C>T (path); similar to his mother, and another mutation c.4607C>T (VUS). The father did not have any ABCC8 mutations.

CONCLUSION

Clinical suspicion of CHI should be highly considered in macrosomic babies with persistent hypoglycaemia in the absence of maternal diabetes. Expedited genetic study should be considered to assist clinical management.

EP_P003

EXTREME SPECTRUM OF DYSGLYCAEMIA IN TWO SISTERS WITH CDKN1C MUTATION

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INTRODUCTION/BACKGROUND

CDKN1C mutation is mainly associated with Beckwith-Wiedemann syndrome (BWS), an overgrowth disorder, and IMAGe syndrome, an undergrowth disorder. In both conditions, hypoglycaemia can be part of the presenting features. Defect in this gene has not been directly linked with diabetes; however, evidence supports the hypothesis that loss of CDKN1C function leads to increased beta cell proliferation and causes hypoglycaemia. Some hypotheses also suggest that overactivity of CDKN1C gene results in the opposite phenotype: decreased proliferation of beta cells leading to reduced insulin production and onset of diabetes.

CASE

We report two cases of Malay siblings who presented with dysglycaemia of opposite ends of the spectrum. Both siblings were not dysmorphic with normal BMI. The elder sister, now 21 years, presented at the age of 5 years with hyperglycaemic symptoms and was treated as type 1 diabetes. Her diabetes autoantibodies were negative. She has been on insulin with an average HbA1c of 8%.

The younger sister presented at 16 years with frequent postprandial hypoglycaemia episodes associated with recurrent cramps and muscle weakness. Investigations showed hyperinsulinaemic hypoglycaemia with concurrent hypokalaemia. PET scan and MRI were negative for insulinoma. Oesophagogastroduodenoscopy did not find any suspicious gastrointestinal lesions. Munchausen by proxy was excluded. Her symptoms improved with oral diazoxide but not fully resolved. She is dependent on potassium supplements. Genetic testing on both sisters revealed same mutation at the CDKN1C gene, reported as variant of uncertain significance (VUS).

CONCLUSION

In our patients, CDKN1C mutation manifested with polar opposites of dysglycaemia. The molecular function of the gene in glucose homeostasis is yet to be defined.

EP_P004

UNVEILING THE MYSTERIES: GENETIC PERSPECTIVE ON PRIMARY ADRENAL INSUFFICIENCY

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INTRODUCTION

Primary adrenal insufficiency (PAI) in childhood is rare and potentially life-threatening. The most common cause is congenital adrenal hyperplasia (CAH) resulting from 21-hydroxylase deficiency. Advancements in molecular genetics have revealed more genetic mutations causing PAI, which helps in explaining associated clinical features and prognosis. Clinical data and genetic tests were reviewed for two patients who presented with PAI.

CASE 1

LA presented with hypotonia and global developmental delay at the age of 1 year, with normal brain MRI/MRA and inborn error of metabolism (IEM) workup. She defaulted follow-ups until she presented again with generalized hyperpigmentation to dermatology at 3 years. Workup showed ACTH >278 pmol/L, normal 17-hydroxyprogesterone (17-OHP), and no rise in cortisol (<1.8 nmol/L) on ACTH stimulation test. Adrenal ultrasound was normal. Genetic

testing was positive for NGLY-1 gene mutation, which is associated with global developmental delay, movement disorders, seizures, liver disease and alacrimia.

CASE 2

MI presented with being "easily tired" and hyperpigmentation since the age of 6 years. The endocrine team was consulted due to low cortisol. Investigations revealed ACTH >278 pmol/L, normal 17-OHP and flat response following ACTH stimulation test. Adrenal CT was normal. Genetic studies came back positive for ABCD1 mutation, a condition of adrenomyeloneuropathy, associated with progressive lower limb weakness and spasticity in the third or fourth decade of life.

With hydrocortisone replacement and fludrocortisone therapy, LA and MI improved noticeably by decreasing skin hyperpigmentation.

CONCLUSION

Non-specific presentations of PAI and the rising numbers of genetic aetiologies discovered warrant genetic testing in affected individuals. This will facilitate prompt diagnosis based on clinical features and prognostication. It provides opportunities for tailored patient management, family counselling and heightened surveillance of possible co-morbidities.

EP_P005

CO-OCCURRENCE OF OCULAR MYASTHENIA GRAVIS, TYPE 1 DIABETES MELLITUS AND GRAVES' THYROTOXICOSIS IN A YOUNG CHILD

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INTRODUCTION

Ocular myasthenia gravis (OMG), type 1 diabetes mellitus (T1DM) and Graves' thyrotoxicosis (GT) are autoimmune conditions in childhood. However, co-occurrence and sequential onset of these diagnoses is uncommon. It could signify a spectrum of polyglandular autoimmune syndrome type 2 with polygenic inheritance.

CASE

A 6-year-2-month-old female presented with progressive drooping of both eyelids for the past two months. Chest CT showed normal thymus, and the anti-acetylcholine receptor was positive (4.89 nmo/L) [reference value (RV) <0.25 nmol/L]. The diagnosis of ocular myasthenia gravis was ascertained. She responded well to pyridostigmine.

Nonetheless, she presented again at 8 years and 11 months old with polyuria, polydipsia and nocturia for three weeks, and significant weight loss. She had severe DKA requiring intensive care. Biochemical markers were consistent with T1DM: low C-peptide (57 pmol/L), low insulin (4.3 pmol/L), positive anti-ICA (45.61 IU/mL) (RV <28 IU/mL) and anti-GAD (98.18 IU/mL) (RV <17 IU/mL), while anti-IA2 was low (<2.5 IU/mL) (RV <28 IU/mL).

While her initial thyroid function was normal, thyroid auto-antibody screening was positive for anti-TPO (222 IU/mL) (RV <35 IU/mL). Following multiple daily insulin injections, her glycaemic control and weight gradually improved. Ten months later, at 9 years and 10 months old, her HbA1c worsened, and her mother reported a sudden increase in insulin needs with weight loss. She manifested symptoms of hyperthyroidism and was found to have tachycardia, tremors and diffuse goitre. She did not have Graves' ophthalmopathy; bilateral ptosis remained the same. Anti-TSH receptor antibodies were significantly positive (26.30 IU/L) (RV <1.75 IU/L).

CONCLUSION

OMG in young children is rarely associated with T1DM. Screening for diabetes auto-antibodies should be considered. In T1DM children, GT should be taken into account when there is unexplained weight loss or deterioration in glycaemic control.

EP_P006

AN UNUSUAL CASE OF MASSIVE NEONATAL GOITRE

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INTRODUCTION

Congenital hypothyroidism occurs in one out of 3000 live births in Malaysia. Over 95% of the cases have no clinical manifestations at birth. In this peculiar case, we present a patient with massive neonatal goitre with congenital hypothyroidism.

CASE

An 8-month-old male was diagnosed prenatally to have a neck mass on a detailed scan at 37 weeks. It was reported to be highly vascularised with possible goitre. Maternal biochemical markers showed euthyroid status, but neck ultrasound revealed multinodular goitre. There were no suggestive risk factors for iodine deficiency. Because of the possibility of airway compression, caesarean section was recommended and he was delivered via ex-utero

intrapartum treatment (EXIT procedure) at 38 weeks and 4 days. The infant was intubated and ventilated for respiratory distress. Newborn examination showed an anterior neck mass measuring 2 x 2 cm from midline to the left, with otherwise unremarkable systemic examination. Postnatal computed tomography of the neck revealed massive goitre causing airway compression and oesophageal narrowing from the oropharynx until the thoracic inlet. Laboratory studies supported the diagnosis of congenital hypothyroidism (TSH 37.41 μIU/mL) with possible thyroid dyshormogenesis. Treatment was initiated with oral levothyroxine 50 mcg daily. Serial ultrasound imaging showed a gradual reduction with resolved mass effect and airway compression.

CONCLUSION

Prompt diagnosis and meticulous thyroid replacement therapy led to significant regression of goitre to a more functional size. Rational intervals of clinical and biochemical evaluation are crucial to ensure optimum growth and neurodevelopmental outcomes.

EP_P007

INFANTILE HYPOCALCAEMIC SEIZURE AND VITAMIN D DEFICIENCY

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INTRODUCTION/BACKGROUND

Growing evidence suggests that vitamin D is essential for maternal and child health in many aspects. Nevertheless, a severe manifestation of vitamin D deficiency in the form of hypocalcaemic seizures continue to occur among Malaysian infants.

METHODOLOGY

A descriptive cross-sectional study was performed in the Paediatric Endocrinology Unit, Hospital Putrajaya. Records of all infants with hypercalcaemic seizures managed by our unit between January 2015 until April 2024 were retrieved from the electronic database system. Causes of hypercalcaemic seizure among this group of patients were identified. Further clinical, biochemical and hormonal results to assess the calcium-vitamin D-PTH axis were analysed.

CASE

A total of 24 patients were treated for hypercalcaemic seizures during the study period. Sixteen patients were male. Majority (75%) of the patients had hypercalcaemic seizures secondary to vitamin D deficiency, while 25% had hypoparathyroidism.

Among the group of hypercalcaemic seizures secondary to vitamin D deficiency, the median age of presentation was 8 weeks. Their mean corrected calcium, phosphorus, magnesium and ALP on presentation were 1.4 mmol/L, 2.35 mmol/L, 0.73 mmol/L and 690 U/L respectively. Mean iPTH and vitamin D levels of the patients were 19 pmol/L and 16.4 nmol/L, respectively. Maternal vitamin D levels were available for 7 mothers, showing a low mean value of 28.7 nmol/L.

All patients with hypoparathyroidism in this study had concomitant vitamin D deficiency or insufficiency. The median age of presentation was 3.5 weeks. Their mean corrected calcium, phosphorus, magnesium and ALP upon presentation were 1.64 mmol/L, 2.80 mmol/L, 0.64 mmol/L and 384 U/L, respectively. Mean iPTH and vitamin D levels were 2.3 pmol/L and 41.7 nmol/L, respectively.

CONCLUSION

Vitamin D deficiency or insufficiency was present in all patients in our study population. Vitamin D deficiency remains the predominant cause of hypocalcaemic seizure. Thus, vitamin D supplementation for all pregnant women should be encouraged as part of routine care. All infants during the first year of life should be encouraged to receive an oral vitamin D supplementation.

EP_P008

MALIGNANT GONADAL TUMOUR IN TRISOMY 21 WITH COMPLETE SEX REVERSAL

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INTRODUCTION/BACKGROUND

Trisomy 21 is a chromosomal disorder with a high incidence worldwide. It is associated with characteristic physical features, delay in development and some congenital organ defects. However, disorder of sex development (DSD) is not usually seen in patients with Down Syndrome.

CASE

A 14-year-old phenotypically female with Down syndrome presented with a two-month history of progressive abdominal swelling, constipation and weight loss. At birth, typical Down Syndrome facial features were present and the female gender was assigned. The karyotype result of 86 cells showed 47XY, +21 with +SRY gene via FISH study.

Pelvic ultrasound at 2 months showed the presence of a uterus and 2 ovaries. A β-hCG stimulation test performed exhibited a suboptimal response: serum testosterone increased to 1.2 nmol/L from a baseline of 0.7 nmol/L. The patient subsequently failed to turn up after a planned diagnostic laparoscopy was cancelled.

At the current presentation, the patient was short and thin. Female pubertal changes were present. The abdomen was distended with a firm rounded palpable mass measuring 20 x 15 cm. There were no findings of clitoromegaly nor palpable gonads.

Primary gonadal failure was evident from high serum gonadotropins and disproportionately low levels of oestrogen and testosterone. Abdomen CT showed a highly vascularised mass arising from the anterior abdomen with multiple septations of mixed cystic and solid components with calcifications; the uterus and two ovaries were seen. Laparotomy revealed a huge mass measuring 15 cm x 16 cm x 6 cm and two gonad-like structures with bridging Müllerian structures and abnormal-looking lymph nodes. Histopathology revealed dysgerminoma and gonadoblastoma of the huge mass and the gonads and metastatic changes in the lymph nodes. A PET scan showed metastasis to the right lung. The patient underwent chemotherapy subsequently.

CONCLUSION

DSD in Down Syndrome with Y chromosome is at high risk of gonadal tumour. Prevention and early detection are possible with the continuation of surveillance and meticulous assessments.

EP_P009

CO-INCIDENTAL FINDING OF SUPRATENTORIAL EPENDYMOMA IN PATIENT WITH GRAVES' DISEASE

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INTRODUCTION

Ependymomas are rare primary tumours of the cranial nervous system in children, which can be classified into supratentorial, infratentorial and spinal cord. They may present with neurological deficits or an increase in intracranial pressure symptoms based on their anatomical sites. Their symptoms may overlap with Graves' disease. It is postulated that the occurrence of Graves' disease might be due to alterations in the immunological response involving the hypothalamus-pituitary-thyroid axis that results in the formation of TSH antibodies.

CASE

A 10-year-old male who was previously well presented with lethargy, loss of appetite, recurrent vomiting and loss of weight for the past month. Clinically, he had bilateral exophthalmos, no ophthalmoplegia or lid lag, and a pulse rate of 120 beats/min.

His thyroid function test showed overt hyperthyroidism (TSH 0.60 m IU/L, T4 24.70 pmol/L). However, his thyrotropin receptor antibodies are still pending. Thyroid ultrasound was consistent with thyroiditis. He was started on thyroid storm treatment and his condition improved.

He presented again with reduced consciousness with a Glasgow coma scale of 10/15, unequal pupil and hyperreflexia of the left limbs. Urgent brain CT brain revealed a right cerebrum intra-axial tumour. He underwent tumour excision. Histopathology revealed a supratentorial ependymoma.

Post-operative Cranial MRI showed tumour size reduction measuring from 7.9 x 6.4 x 9.1 cm to a residual of 2.7 x 2.5 x 2.7 cm over the right parietal lobe. Carbimazole was continued and his clinical course was monitored.

CONCLUSION

This is a rare case of a co-incidental finding of supratentorial ependymoma with Graves' disease.

EP_P010

NEONATAL GOITER WITH AIRWAY AND OESOPHAGEAL COMPRESSION WHICH IMPROVED WITH L-THYROXINE AND A CONSERVATIVE APPROACH

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INTRODUCTION/BACKGROUND

Congenital goitre occurs rarely in neonates and it becomes an emergency when it causes significant airway compromise in newborns transitioning from foetal to postnatal life. neonatal goitre is closely related to thyroid status and could be either a transient or permanent genetic condition.

CASE

Foetal ultrasound in 2nd trimester detected an anterior neck mass with increased vascularity. At 38 weeks gestation,

a baby girl was delivered with a birth weight of 3.37 kg, length of 51 cm and OFC of 33 cm. She was intubated with ETT size 3.0 and ventilated in NICU. Examination showed a diffuse neck swelling measuring 3 x 3 cm. Ultrasound and CT scan showed an enlarged right thyroid lobe 3.5 x 2.7 x 4.2 cm (AP x W x CC) and a left thyroid lobe 3.1 x 2.8 x 4.3 cm (AP x W x CC) with no cystic component or calcification. Thyroid lobes extended up to the angle of the mandible and inferiorly until thoracic inlet with the airway and esophagus almost circumferentially encased and airway patency only maintained by ETT. Cord TSH 25.12 m IU/L, fT4 10.0 pmol/L, while Day 5 TSH 37.41 m IU/L, 6.47 pmol/L. ATPO, ATG & TSH receptor antibodies were negative. Her thyroglobulin level was low at 0.6 ng/ml (intact thyroid, 3.5-77.0). Her mother also reported onset of goitre following 1st trimester. She complained of tiredness and started taking Himalaya salt. Urine iodine results for patient and mother results were 322.7 ug/L and 221.3 ug/L, respectively (250 - 499, sufficient for pregnant mother). She was treated with L-thyroxine at day 5 of life at 50 mcg daily (15 mcg/kg) with serial ultrasound neck and flexible endoscopic assessment. She was successfully extubated by day 26 and discharged after 1 month. On follow-up, her goitre remained small with normal development and hearing. Due to cost constraint, genetic test was not pursued.

CONCLUSION

Neonatal goitre with hypothyroidism may result from maternal ingestion of antithyroid drugs or goitrogens, transplacental transfer of antithyroid antibodies, or thyroid dyshormonogenesis. Low thyroglobulin level with raised TSH and low fT4 suggest thyroglobulin synthetic defect.

EP_P011

A BOY WITH UNTREATED PANHYPOPITUITARISM: CASE REPORT

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INTRODUCTION/BACKGROUND

Hypopituitarism in childhood is a complex disorder with diverse clinical presentation which can either be congenital or acquired. Hormonal deficits can evolve over time leading to a significant impact on a child's growth.

We describe a case of untreated panhypopituitarism presenting as an adrenal crisis managed in a district hospital.

CASE

A 13-year-old Indonesian male with a background of panhypopituitarism post mature teratoma resection in January 2020 presented an adrenal crisis after he defaulted treatment for 2 years. On presentation, he had hypoglycaemia with shock requiring extensive fluid resuscitation and double inotropic support. Examination revealed weight and height below 3rd centile, pre-pubertal with thin eyebrows and depressed tendon reflexes. Height velocity was 4.2 cm/year for past 2 years. His random cortisol was extremely low (<1.5 nmol/L) with hypothyroidism. Intravenous stress dose hydrocortisone was initiated and his hemodynamic status improved over time. Oral thyroxine supplement was restarted and he required regular dose of sublingual desmopressin for diabetes insipidus.

CONCLUSION

Untreated panhypopituitarism has been reported in adult as late as 45 years old with significant impairment in cardiac function, growth and regression of sexual characteristic. With appropriate hormonal replacement, growth can be optimized and lifetime expectancy can be improved without long term sequelae.

EP_P012

DELAYED PRESENTATION OF PITUITARY TUMOUR WITH HYPOPITUITARISM

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INTRODUCTION/BACKGROUND

Hypopituitarism is a relatively uncommon disorder in the paediatric population, and its prevalence in children is not yet well established. This condition can be caused by any disease that affects the pituitary gland, stalk or hypothalamus. We describe a patient who presented late with short stature and hypopituitarism.

CASE

A 16-year-old male presented with short stature and delayed puberty. He denied any headaches or visual problems. He was born full-term with a birth weight of 3.2 kg. Developmental milestones were normal. At presentation, his height was 139 cm and his weight was 35 kg (both below 3rd percentile). Midparental height was 167.5 cm. His Tanner stage was 1 and testicular volume was 2 ml bilaterally. His visual field assessment was unremarkable. Hormonal analysis demonstrated

hypopituitarism as validated by the following: LH <0.12 IU/L (1.8-11.8 IU/L) FSH 0.8 IU/L (3.03-8.08 IU/L) fasting morning testosterone 0.32 nmol/l (0.69- 26.16 nmol/l), IGF-1 38.7 ng/ml (226-903 ng/ml), morning serum cortisol 158 nmol/l (102-558 nmol/l) with inappropriately normal ACTH 4.37 pmol/L (1.6-13.9 pmol/L) fT4 7.74 pmol/L (11.4-17.6) TSH 2.08 m IU/L (0.47-3.41) prolactin 671.28 m IU/L (72.6-407.4). Synacten test revealed inadequate response with peak cortisol 184 nmol/l at 60 minutes. His bone age was delayed between 11 - 13 years. Magnetic resonance imaging of the pituitary gland revealed the presence of an enhancing lesion at the suprasellar region, at the centre of the optic chiasm abutting the proximal part measuring 1.0 x 1.2 x 1.1 cm (AP x W x CC). Differential diagnosis includes craniopharyngioma or pilocytic astrocytoma. He was replaced with glucocorticoid and levothyroxine while awaiting a parental decision regarding tumour excision.

CONCLUSION

Hypopituitarism can present in neonates, infants, children, and adolescents with multifactorial aetiologies. Timely diagnosis of this condition is crucial for effective intervention and management of affected children. The key to successful management of hypopituitarism lies in a high index of suspicion, coupled with increased awareness and appropriate hormone replacement therapy. Access to facilities for surgical intervention is essential for the survival and good prognosis of affected children.

EP_P013

INCREASING TRENDS OF CENTRAL PRECOCIOUS PUBERTY AMONG CHILDREN IN HOSPITAL PUTRAJAYA, 2004 TO 2024: A DESCRIPTIVE STUDY

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INTRODUCTION/BACKGROUND

Temporal trends worldwide demonstrate evidence of an earlier onset and progression of puberty worldwide. This study aims to describe the trends in Precocious Puberty among children in Hospital Putrajaya between 2004 to 2024. Data retrieved from the electronic database were reviewed. All patients diagnosed to have precocious puberty (i.e., onset of puberty before age 8 years for girls and 9 years for boys) in the Department of Paediatric Endocrinology of Hospital Putrajaya from January 2004 until April 2024 were included (n = 89). These patients were stratified according to the diagnosis; children diagnosed with Idiopathic Central Precocious Puberty (CPP) and normal variant puberty (i.e., Premature thelarche (PT) or Premature Adrenarche (PA).

CASE

Overall, a total of 89 children (86 (96.6%) girls; median [interquartile] age at diagnosis for boys, 7 [6;10] years; for girls, 7 [2;9] years) were registered with a diagnosis of CPP, PT, PA. Majority were Malay, 54 (60.7%), 25 (28.1%) were Chinese, 8 (9%) were Indian and 2 (2.2%) were Nigerians. Majority of the cases were idiopathic CPP, 81 (91%); with a median [interquartile] LH:FSH ratio of 1.4 [0.16;7.23]. The MRI findings show normal findings in 30 (35.3%), pituitary microadenoma in 29 (34.1%), and pineal gland cysts in 1 (1.12%). There was a general increase in the number of cases of CPP over time; between 2013 to 2018; 23 (25.8%), and a greater rise between 2019 to 2024; 61 (68.5%). Nearly half of the cohort had a body mass index (BMI) of overweight or obese 40 (41.3%); with median [interquartile] bone age, 4 [2;7].

CONCLUSION

This study demonstrated an increase in the number of patients with central precocious puberty over 20 years. We also demonstrated a possible association with an increased BMI and earlier onset of puberty in girls.

EP_P014

OBESITY IN TEMPLE SYNDROME

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INTRODUCTION/BACKGROUND

Temple syndrome is a rare imprinting disorder caused by a maternal uniparental disomy of chromosome, paternal deletion of 14q32 or isolated methylation defect of the MEG3-DMR. Review of the electronic medical records with salient clinical and investigations recorded.

CASE

MKA is an 8 years and 5 months old male who presented with central hypotonia with poor sucking at birth. He was delivered term at 2.62 kg. Antenatally, the mother had oligohydramnios. During clinic follow-up, MKA remained well but he remained obese with a BMI of more than 97th centile. He looked dysmorphic with plagiocephaly, narrow bifrontal diameter, almond-shaped eyes, downturned mouth, thin upper lip, thick earlobes, small hands and feet, left single palmar crease, pes planus and genu valgus.

Neurological examination revealed central hypotonia. He had failure to thrive during infancy where he was on orogastric feeding for 1 month, achieved brief normalisation at 1 year old, followed by hyperphagia and rapid weight gain since the age of 2 years old. He is a product of a non-consanguineous marriage. He also has isolated central hypothyroidism with left cerebral hemiatrophy.

Chromosomal study showed 46XY karyotype. A series of imaging including abdominal ultrasound, KUB, pelvis, cranium, echocardiogram and brain MRI - was normal. He was suspected to have Prader Willi Syndrome, but methylation test was normal. Genetics team co-managed and treated him for Temple Syndrome with maternal uniparental disomy 14 with Prader Willi Syndrome like phenotype.

CONCLUSION

Temple syndrome is largely manifested by physical defects necessitating obligatory supportive therapies early in the life of affected individuals. The syndrome has shown that a subset of patients develop obesity. BMIs increased along the reference curves in most patients. Endocrine anomalies are prevalent, with truncal obesity developing as early as 4 to 6 years.

EP_P015

IT'S MODY! NOT TYPE 2 DIABETES

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INTRODUCTION/BACKGROUND

MODY (maturity-onset diabetes of the young) is a rare form of diabetes which represents a clinically heterogeneous group of autosomal-dominant disorders caused by mutation in genes involved in beta cell development and insulin secretion. The classic phenotype of MODY includes nonketotic noninsulin-dependent diabetes with diagnosis before age 25 years and an affected parent. However, there is clinical overlap between MODY, type 1, and type 2 diabetes resulting in frequent misdiagnosis of MODY. Data collection was done by reviewing the electronic medical records of the selected patient and salient points were noted.

CASE

TSE, presented at the age of 9 years old with hyperglycaemia detected on routine monitoring. On presentation, she was asymptomatic and her investigations revealed nonketotic hyperglycaemia (RBS 10.5 mmol/L). Clinically there was no evidence of acanthosis nigricans and her BMI was within normal range. Further investigations revealed HbA1c of 8.6% with an abnormal OGTT (FBS: 8.9 mmol/L, 2 hours post: 20.97 mmol/L). Her pancreatic autoantibodies came back as all negative and had a high c-peptide level of 357.9 pmol/L. She was treated initially with oral metformin. On further history, the mother had early-onset diabetes and there was a strong family history of early-onset diabetes on the maternal side. Genetic testing by whole exome sequencing revealed a heterozygous variant in HNF1B. She was further investigated and renal function, lipid profile and ultrasound of the abdomen and kidneys were normal. She was subsequently transitioned to insulin therapy.

CONCLUSION

HNF1B has a wide phenotypic spectrum, and affected individuals may present with isolated renal disease, isolated diabetes, or both. This case highlights the importance of the precision medicine approach in MODY. Molecular genetic testing can identify the subtypes and has profound implications on diabetes treatment and prediction of future development of co-morbidities, allowing early preventive or supportive treatment.

EP_P016

HYPOPHOSPHATASIA, RARE BONE MINERALISATION DISORDER THAT MIMICS OSTEOGENESIS IMPERFECTA AT BIRTH

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INTRODUCTION/BACKGROUND

Hypophosphatasia is a genetic disorder characterized by impaired mineralization of bones and teeth that results in fractures and deformities. It affects less than 1:100,000 live births with different clinical spectrums. It is caused by loss-of-function mutations of ALPL gene that encodes tissue nonspecific alkaline phosphatase (TNSALP), leading to low activity of this enzyme that usually mediates the breakdown of inorganic pyrophosphate that blocks mineralization. We reported an infant who was initially admitted to NICU at birth.

CASE

Antenatally, the patient was detected with possible osteogenesis imperfecta and unilateral lung hypoplasia by a detailed foetal scan at 32 weeks gestation. His mother had GDM and underwent LSCS at 37 weeks 5 days with a weight of 2.42 kg. He was born with generalized bowing deformities of both upper and lower limbs, a broad

forehead, wide wide-spaced nipple, and no blue sclera. His respiratory assessment was normal. His skeletal survey reported generalised osteopenia with bowed limbs and plastic fracture over the right femur consistent with osteogenesis imperfecta. He had normal serum calcium, persistently low ALP (18-25 IU/L), iPTH: 69.3 pg/ml (14.9-56.9) and 25 (OH) vit D3: 51.70 nmol/L (insufficient). There was no family history of bone diseases and parents were not consanguineous. His genetic results showed heterozygous, autosomal recessive, likely pathogenic, ALPL (NM_000478.6), Exon 2, c.29T>C, p. (Ile10Thr), and ALPL (NM_000478.6), Exon 9, c.991G>A, p. (Val331Met). He continued to improve without treatment and was last reviewed at 5 months old, with appropriate development and no fracture.

CONCLUSION

There are very few cases of hypophosphatasia reported locally. It is important to highlight the differential as compared to osteogenesis imperfecta. Hypophosphatasia could present differently, with our case reflecting the benign perinatal type, whereas others would require enzyme replacement, or asfotase-alfa as the first medical treatment for perinatal, infantile and juvenile onset of the disease.

EP_P017

CONGENITAL HYPOTHYROIDISM WITH DIFFERING PHENOTYPE IN TWO SIBLINGS WITH THE SAME DUOX2 MUTATION

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INTRODUCTION/BACKGROUND

Congenital hypothyroidism (CH) is caused by thyroid gland structural or functional defects. Thyroid dysgenesis is typically sporadic but dyshormogenesis is frequently inherited as autosomal recessive. Dual oxidase 2 (DUOX2) gene mutation causes abnormal iodide organification in thyroid hormone synthesis.

We report 2 siblings with CH sharing the same mutation in DUOX2, however only one had permanent CH.

CASE

A 14-year-old female had CH detected at birth by high cord TSH (>100 m IU/L), and thyroid function test (TFT) on day 3 of life (TSH >100 m IU/L, free T4 8.57 pmol/L). Thyroid replacement was started at a dose of 8 mcg/kg/day. Ultrasound at age 3-year-old showed a hypoplastic thyroid gland. She had permanent CH as TFT was deranged when thyroxine was withheld at age 3. Her younger brother, now aged 9 years, had CH diagnosed at age 1 month when he presented with prolonged jaundice (TSH 56.4 m IU/L, free T4 8.89 pmol/L). His cord TSH was normal (7.18 m IU/L). Thyroxine replacement was started at a dose of 7 mcg/kg/day. Ultrasound at age 3 showed a normal-sized thyroid gland. Thyroxine was stopped at age 3, and subsequent TFT remained normal, indicating transient CH. Both had no goiter, nor comorbidity. No other family members had a thyroid disorder. Parents were non-consanguineous. Whole exome sequencing (3Billion, South Korea) revealed both siblings had the same pathogenic heterozygous DUOX2 mutation (c.3329G>A). It was inconclusive as the second abnormal allele was not detected.

CONCLUSION

The same DUOX2 mutation can have different phenotypes within the same family. Genetic testing has a role in evaluating CH etiology, especially when at least two family members are affected. Newborn siblings of a child with CH need timely monitoring of TFT if cord TSH is normal. Other genetic methods are needed to detect the second variant of DUOX2 in these siblings.

EP_P018

BONE HEALTH SURVEILLANCE AMONG AT-RISK CHILDREN AND ADOLESCENTS IN KUCHING, SARAWAK, MALAYSIA

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INTRODUCTION/BACKGROUND

Chronic health conditions impose poor bone health due to underlying inflammatory conditions, reduced weight-bearing activity and pubertal delay.

METHODS

This prospective study was conducted as the pilot project for bone health surveillance among at-risk children and adolescents followed up by a multidisciplinary team from Paediatrics Department, Sarawak General Hospital from January to February 2024. A bone health screening questionnaire was administered, followed by a physical examination, and biochemical and radiological investigation.

CASE

A total of sixteen subjects (6 males, 10 females) with a mean age of 10.7 ± 2.74 years were recruited. Four subjects had

primary bone disorders, while another 8 subjects (50.0%) had exposure to glucocorticoids for the management of various condition including systemic lupus erythematous (2 subjects, 12.5%), juvenile dermatomyositis (2 subjects, 12.5%), juvenile idiopathic arthritis (1 subjects, 6.3%), ANCA vasculitis (1 subject, 6.3%), autoimmune hepatitis (1 subject, 6.3%) and acute lymphoblastic leukaemia (1 subject, 6.3%). Only half of the subjects elicited adequate dairy consumption and took vitamin D supplements in the form of cholecalciferol or alfacalcidol. Sedentary lifestyle was observed in two thirds of the subjects. The physical stigmata of bone fragility disorders were present in 4 patients, and they had genetic confirmation of osteogenesis imperfecta. Five subjects (31.3%) had fracture of long bones, as well as osteoporosis. Bone-active therapy with bisphosphonate had commenced in three patients. Serum 25-hydroxy vitamin D and parathyroid level were examined in 5 subjects and 2 subjects were detected to have vitamin D deficiency. Four subjects (25.0%) displayed vertebral fractures. Overall, the mean areal bone mineral density Z- scores were -2.78 ± 1.74 for hip, -1.87 ± 1.71 for lumbar spines and -3.07 ± 2.16 for total body less head.

CONCLUSION

Bone health screening among the children and adolescents vulnerable to osteoporosis should be imparted as the standard of care.

EP_P019

"A GIRL, WITHOUT UTERUS OR VAGINA:" A CASE REPORT OF MAYER-ROKITANSKY-KUSTER-HAUSER SYNDROME

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INTRODUCTION/BACKGROUND

Mayer-Rokitansky-Kuster-Hauser (MRKH) syndrome is a rare congenital disorder that affects female reproductive organs and is often only diagnosed during adolescence or early adulthood. It is estimated to affect at least 1 out of 4500 women and it remains poorly investigated and underreported.

CASE

A 4-year-old child, raised as a girl was referred to the clinic for atypical genitalia. She was born premature at 28 weeks of gestation with a birth weight of 1.22 kg (between 50th to 90th centile). Her parents were not consanguineous and there was no family history of disorder of sexual development. On examination, there was no hyperpigmentation of the genitalia. There was clitoromegaly, with the size of 26 x 12 mm, urethral meatus was seen at the base of the phallus, with no vaginal opening. The labioscrotal folds were not rugated. There were palpable masses at the bilateral inguinal region suggestive of gonads.

Chromosomal study done revealed 46, XX. Baseline hormonal workup including 17-OH progesterone and cortisol were normal. Testosterone was not detectable. Pelvic MRI pelvis was suspicious of MRKH syndrome, as the only visualized Mullerian structures present were rudimentary uterus and bilateral ovaries. Both cervix and upper vagina were not visualized. Apart from that, there were bilateral cystic lesions seen at the inguinal region most likely consistent with canal of Nuck cyst. Patient has also been referred to both genetic and surgical team for further management.

CONCLUSION

MRKH syndrome is usually diagnosed later in life. Early radiological imaging aids earlier diagnosis. This condition requires multidisciplinary management that can help both the patient and their family to cope with this uncommon condition, including the psychological and physiological consequences.

EP_P020

MALE OR FEMALE? A CASE REPORT OF 17-BETA HYDROXYSTEROID DEHYDROGENASE 3 (17β-HSD3) DEFICIENCY

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INTRODUCTION/BACKGROUND

17β-HSD3 deficiency is a rare autosomal recessive cause of 46-XY Disorder of Sexual Development (DSD), occurring in approximately 1 in every 147,000 newborns. 17-β-HSD3 is encoded by the HSD17B3 gene (chromosome 9q22.32) and expressed in the testis where it converts the inactive steroid androstenedione to the active androgen testosterone. We report a case of DSD who presented with hoarseness of voice and primary amenorrhea.

CASE

A case of a 22-year-old who was raised as female presenting with primary amenorrhea at the age of 16, preceded by gradual hoarseness of voice since 12. Height was 158 cm. Pubertal assessment revealed Tanner stage 2 breast development, stage 3 axillary and stage 4 pubic hair distribution. Physical examination was notable of facial hair, male body habitus, a phallus measuring 4 cm in length and 2 cm in girth, bilaterally palpable inguinal mass and empty rugated labioscrotal folds. Laboratory parameters revealed low to normal testosterone, raised FSH, LH and mildly elevated estradiol levels, pelvic MRI revealed bilateral gonads seen in the inguinal region with a small corpus cavernosa, corpus spongiosum, seminal vesicles, prostate and no uterus or ovary The child and her parents received psychological counselling and she desired to maintain a female gender identity. Genetic studies by whole exome sequencing revealed homozygous mutation of HSD17B3 gene.

CONCLUSION

46-XY DSD due to 17β-HSD3 deficiency is a rare disorder that may prove a diagnostic conundrum in situations where specific endocrine panels and genetic testing are not readily available. In patients where the diagnosis is delayed there may be difficulty in gender assignment.

EP_P021

VITAMIN D DEFICIENCIES: A PILOT STUDY IN OVERWEIGHT AND OBESE CHILDREN AND ADOLESCENTS

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INTRODUCTION

In the post COVID era, the prevalence of overweight and obesity in children and adolescents is increasing, leading to multiple obesity-related comorbidities. Despite Malaysia being a tropical country, the occurrence of vitamin D deficiency has been increasing. This study aimed to determine the incidence of vitamin D deficiencies in normal and obese/overweight groups.

METHODOLOGY

A total of 43 participants aged 7 to 17 years old were recruited from the Hospital Al-Sultan Abdullah, UiTM. During the hospital visit, anthropometric measurements and a questionnaire on dietary intake, sunlight exposure and average family income were collected. Total serum vitamin D levels were taken and analysed using the electrochemiluminescence assay.

RESULT

The mean age of the participants was 9.30 ± 1.89 years old. A total of 48.8% were overweight or obese. The overweight/obese group had a significantly higher body mass index (BMI) (29.01 ± 11.23 kg/m^2 vs 14.95 ± 1.77 kg/m^2, p <0.01), weight (54.73 ± 22.7 kg vs 24.23 ± 7.24 kg, p <0.01) and waist circumference (82.65 ± 23.27 cm vs 55.66 ± 5.10 cm,

p <0.01) compared to the normal group. Male participants had significantly higher BMI (23.8 ± 8.27 kg/m^2 vs 19.92 ± 12.31 kg/m^2, p <0.05) and weight (47.14 ± 25.71 kg vs 31.47 ± 16.23 kg, p <0.05 compared to female. A total of 60.5% of the participants had serum 25 (OH) D levels ≤50 nmol/L. There were no significant differences in 25 (OH) D levels between both groups. Additionally, daily exposure to sunlight for more than 2 hours was associated with higher serum vitamin D levels among children and adolescents (p <0.05). Furthermore, a high household income was associated with a high risk of weight gain (p <0.01) and waist circumference (p <0.01) among children and adolescents.

CONCLUSION

There was a high incidence of vitamin D deficiencies in children and adolescents with no association between BMI and serum 25 (OH) D levels.

EP_P022

SEVERE STUNTING: A RARE CASE OF ACROMESOMELIC DYSPLASIA WITH CONCOMITANT GHD

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INTRODUCTION/BACKGROUND

Acromesomelic dysplasia, type Maroteaux (AMDM) is a rare skeletal dysplasia, characterized by severe disproportionate short stature, primarily affecting the middle and distal segments of the limbs.

CASE

An 11-year-old female presented with extremely short stature. She was born with a birth weight of 2.6 kg and has been noticed to be short since 2 years old. Her parents were not consanguineous but had a strong family history of short stature. Father's height was 154 cm (SDS -3.16) and mother's height was 148 cm (SDS -2.36). The girl has normal intelligence and an unremarkable medical history. She was prepubertal and markedly short with a height of 108.5 cm (SDS -5.03) and a weight of 23.3 kg (SDS - 2.74). Her height was 3 SD below her mid-parental height (MPH SDS-2.3). She had a disproportionate body proportion with an upper segment to lower body segment ratio of 1.25 and shortened middle and distal segments of the limbs. Skeletal survey showed shortening of the radius, ulna, tibia, and fibula as well as short and broad metacarpals and phalanges, with cone-shaped epiphyses. She had an insulin tolerance test that showed peak growth hormone of 8.4 ng/ml and peak cortisol of 586 nmol/L (normal), which suggested isolated severe growth hormone deficiency. Bone age was indeterminate due to abnormal epiphyses. Whole exome sequencing identified compound heterozygous pathogenic variants in NPR2 which is associated with autosomal recessive AMDM. We did not proceed with the 2nd GH test since the genetic results were confirmative.

CONCLUSION

We described a child with AMDM and severe growth hormone deficiency. Average adult height is estimated to be less than 120 cm. No published data showed that ADMD is related to GHD. Literature reviews showed that 3 AMDM children who received high-dose growth hormone treatment (0.05- 0.1 mg/kg/day) had positive effects on height improvements.

EP_P023

GROWING TOO FAST: A CASE OF TESTOTOXICOSIS

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INTRODUCTION/BACKGROUND

Testotoxicosis is a rare cause of gonadotropin-independent precocious puberty in males due to an activating mutation in the luteinizing hormone (LH)) /choriogonadotropin receptor (LHCGR) gene. This disorder usually presents at 2-4 years old with virilization, advanced bone age, and increased serum testosterone levels above adult ranges, despite low LH and FSH levels.

CASE

A 3-year 2-month-old male presented with complaints of acne, pubic hair, phallic growth, height accelerations, and behavioural issues. His parents were non-consanguineous and had no family history of precocious puberty. On physical examination, his height was 106cm (+2.36 SDS) and weight was 16 kg (+0.77 SDS). He had a muscular body build, a deep voice, and acne. He had Tanner stage 2 pubic hair, stretched penile length was 6 cm (>2 SDS), and both testicular volumes were 5 mls. He did not have café au lait spots. Bone age was 7 years old. Testosterone level was high at 17 nmol/L and gonadotropin-releasing hormone (GnRH) stimulation test revealed a prepubertal response (peak LH 1.44 u/L and FSH 3.02 u/L). Thyroid function tests (FT4: 13.77 pmol/L; TSH: 1.71 m IU/L) and adrenal hormone levels (DHEAS: 0.46 umol/L; 17-OHP: 3.42

nmol/L; cortisol: 400 nmol/L) were within normal ranges. Synacthen test excluded virilising congenital adrenal hyperplasia. Low serum beta-hCG (1.2 mIU/ml) excluded beta-hCG-secreting tumour. Testicular ultrasound showed no sonographic evidence of testicular lesion. Whole exome sequencing identified a heterozygous pathogenic variant c.169A>G (p.Asp564Gly) in LHCGR gene which supports the diagnosis of testotoxicosis.

The child was started on aromatase inhibitor, Anastrozole 1mg daily, and anti-androgen, spironolactone 2 mg/kg BD. At 6 months of treatment, there was a halt in pubertal progression with reduced height velocity from 9 cm/year to 6 cm/year.

CONCLUSION

There was no consensus on the management of this rare condition. Without intervention, the patient will have rapid progressive skeletal maturation and virilization which will result in compromised adult height and psychosocial distress.

EP_P024

EARLY BISPHOSPHONATE TREATMENT IN AN INFANT WITH COL1A1 OSTEOGENESIS IMPERFECTA

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INTRODUCTION/BACKGROUND

Bisphosphonate therapy is the mainstay treatment of patients with OI. It helps to increase bone mass, decrease fracture rate, improve growth and muscle strength as well as improve mobility. Initial studies were performed among older children and adolescents; however, recently, early treatment in infants with moderate-to-severe OI has been shown to be safe. The optimal age of starting is controversial, especially less than 6 months as there is a need to balance the benefits of therapy with the safety of treatment.

CASE

We report an 11-month-old male whose prenatal scan revealed suspicion of skeletal dysplasia. Parents are non-consanguineous and with no family history of frequent fractures or genetic disorders.

He was born term via EMLSCS for intrauterine growth restriction with highly resistant Doppler. The birth history was uneventful. He has low-set ears, macrocephalic with widened anterior fontanelle, triangular facies, and grey sclera, his hips were in a flexed and abducted position with bowed bilateral lower limbs.

The child had bilateral thigh swelling with deformity at birth. Radiological evaluation showed a bilateral femur fracture. He sustained a bilateral humerus fracture at day 12 of life, a left radius fracture at 2 months old and a right humerus fracture at 3 months old. Whole Exome Sequencing test revealed a pathogenic variant of COL1A1 gene.

The child was started on pamidronate at the age of 5 months old with a dose of 0.1mg/kg then the dose was increased to 0.25 mg/kg, and was given 3 consecutive days, monthly then every 2 months. The pamidronate dose was further increased to 0.5 mg/kg for 3 days, given 3 monthly. He tolerated treatment well and no adverse effects were noted. He has had no new fractures since treatment started.

CONCLUSION

OI is a complex disorder and involves multidisciplinary management. Early and appropriate treatment could help increase bone density and prevent recurrent fractures.

EP_P025

DILATED CARDIOMYOPATHY IN A CHILD WITH GRAVES' DISEASE

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INTRODUCTION/BACKGROUND

Individuals diagnosed with Grave's disease typically exhibit symptoms of hyperthyroidism, including chest pain, palpitations, and weight loss. Uncommonly, patients may also develop cardiomyopathy, a complication that is extremely serious and potentially life-threatening. Its occurrence is higher among middle-aged and elderly patients with pre-existing heart conditions. Although rare in children, we must acknowledge this complication because of its high mortality and morbidity rates.

CASE

The report details a case of an 11-year-old female with Graves' disease, thyroid storm, and cardiomyopathy. She had palpitations for almost 2 years, followed by recurrent syncopal attacks for 6 months. Her 'unexplained' syncopal attacks were only provided reassurance when she sought medical attention. Upon her first endocrine review, she was in a hyperthyroid state with bilateral exophthalmos, diffuse goitre with signs of heart failure. The initial thyroid function test showed significantly high FT4 levels of 85.6

pmol/L and suppressed TSH. Her TSH Receptor antibody showed elevated levels, and the initial CXR revealed cardiomegaly. She was started with oral carbimazole 30 mg daily, oral propranolol 20 mg 4 times a day, Lugol's iodine 4 drops 4 times a day, and intravenous hydrocortisone 50 mg q 6 hourly. Two anti-failure medications were used to treat her heart failure. Her symptoms improved, and she was discharged with oral carbimazole and oral propranolol.

CONCLUSION

It is important to plan an early definitive therapy in this case to prevent future cardiac decompensation during relapse. Medical practitioners need to be aware of the rare presentations of Graves' disease to avoid delayed diagnosis and treatment.

EP_P026

MATERNAL PREGNANCY LUTEOMA: A RARE CAUSE OF VIRILISATION IN A FEMALE NEWBORN AND MOTHER

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INTRODUCTION/BACKGROUND

Virilisation of a female newborn is commonly attributed to congenital adrenal hyperplasia but there are rarer causes that can be maternal in origin. Luteomas, a rare, benign androgen-producing ovarian tumour arising during pregnancy can result in both maternal and fetal virilization.

CASE

We describe a case of a newborn with ambiguous genitalia. This baby was born at 36 weeks via caesarean section for poor progress, weighing 2.8 kilograms at birth. Examination at birth revealed a prominent clitorophallic structure, fused labioscrotal folds but no palpable gonads. Otherwise, on general examination, there were no dysmorphic features or hyperpigmentation and serum electrolytes were normal with no hypoglycaemic episodes. On further assessment, 17 Hydroxyprogesterone (17-OHP) level was not elevated; karyotyping and radiological findings were consistent with a female gender. In hindsight, the mother recollected having signs of virilization, i.e., acneiform eruption on her upper chest and back, hirsutism, and deepening voice since the second trimester. Bilateral unhealthy, friable ovarian tumours were revealed intra-operatively which ruptured on handling. As the nature of the tumours was suspicious of malignancy, bilateral oophorectomy was done. Maternal beta human chorionic gonadotrophin (b-HCG) and alpha-fetoprotein (AFP) levels were elevated. The histopathological examination of the ovarian mass confirmed the diagnosis of pregnancy luteoma.

CONCLUSION

This case attests to the fact that rare causes of virilisation in a female baby cannot be overlooked. We thus need to be vigilant and have a high index of suspicion of maternal pregnancy luteomas as a possible cause of virilisation in a female baby.

EP_P027

PAEDIATRIC GRAVES' DISEASE AND DEFINITIVE TREATMENT

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INTRODUCTION

Paediatric Graves' disease (GD) is managed by antithyroid drugs (ATD), radioactive iodine (RAI) or thyroid surgery. This study aimed to describe the characteristics and outcomes of paediatric patients who received definitive therapy.

METHODOLOGY

Children and adolescents diagnosed with GD from 2012 to 2024 at the University Malaya Medical Centre were included in this retrospective review.

RESULT

A total of 37 patients were referred and diagnosed with GD; majority (78.4%) were female. Definitive therapy was given to 48%: 5 (35%) had total thyroidectomy and 9 (64%) received RAI. They had an average of four relapses during the disease. On average, the patients received ATD for 4.37 ± 2.28 years prior to the definitive treatment. The main factor in determining the choice of treatment was the size of the goitre. The mean goitre size for the RAI group was 21.68 ± 7.9 g, compared to 76.7 ± 22.88 g for the thyroidectomy group. Mean age in the RAI group was 15.53 ± 1.23 years. The youngest patient was 8 years old. Mean RAI dose was 9.3 ± 0.66 mCi. Six patients achieved hypothyroidism within 2.17 ± 2.44 months, while 1 patient achieved hypothyroidism 8 months post-RAI. Three had relapses post-RAI. Two patients required a second RAI one year later and achieved hypothyroidism within 2 to 4 weeks. Those who required a second RAI were given lower RAI doses initially (mean 5.6 ± 2.2 mCi). The mean age of patients who underwent total

thyroidectomy was 17.08 ± 0.78 years. Two out of 5 patients developed hypocalcaemia postoperatively. None had vocal cord paralysis.

CONCLUSION
From this cohort, the onset of hypothyroidism post-RAI was varied. Lower doses of initial RAI were seen in patients who had relapsed and required a second treatment. Total thyroidectomy in children and adolescents is safe with minimal complications under the care of a high-volume surgeon.

EP_P028

GRAVES' DISEASE WITH OSCILLATING THYROID FUNCTION

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INTRODUCTION/BACKGROUND
Oscillating thyroid status between hyper-and hypothyroidism in Graves' disease is a rare phenomenon. The change in the thyroid status is likely attributed to the simultaneous presence of both thyroid-stimulating autoantibodies (TSAbs) and TSH-blocking autoantibodies (TBAbs) in an individual.

CASE
We report the case history of a 14-year-old female who presented with goitre, palpitations and weight loss without eye signs. Thyroid function test revealed suppressed thyroid stimulating hormone and mildly raised free thyroxine. Antithyroid globulin antibodies and antithyroid peroxidase were both strongly positive. She was initially diagnosed as Hashimoto thyroiditis in hashitoxicosis and started on carbimazole. Four months later, the patient progressed into hypothyroid state requiring thyroxine therapy for 2 years. She however became hyperthyroid again in the past 1 year. Further evaluation revealed raised TSH receptor antibodies (TRAb) and hyperfunctioning thyroid gland on Tc99m thyroid radioisotope study, leading to a revision of diagnosis to Graves' Disease.

CONCLUSION
This case demonstrates Graves' disease with alternating thyroid status poses a challenge to the patient's diagnosis and management. Measurement of TRAb together with TSAbs and TBAbs would be helpful for diagnosis and to objectively explains the alternating thyroid function. Management includes close monitoring of thyroid function and possibly definitive therapy of radioactive iodine ablation or surgery in selected cases.

EP_P029

POLYOSTOTIC FIBROUS DYSPLASIA: RESPONSE TO ZOLENDRONIC ACID

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INTRODUCTION/BACKGROUND
Fibrous Dysplasia is a rare developmental bone disorder in which fibro-osseous tissue replaces normal bone tissue. It can manifest either monostotic or polyostotic associated with McCune-Albright syndrome. Bisphosphonates such as pamidronate and alendronate have been used to improve bone mineral density due to antiresorptive properties. However, the literature on the use of zoledronate is limited.

CASE
A 10-year-old female presented with a fracture of the right midshaft of the femur following a trivial fall. She had a history of precocious puberty and limping gait since the age of four years old. On examination, she was tall for her age and there was thoracolumbar scoliosis with huge cafe au lait patches at her lower back. Biochemically, she had elevated alkaline phosphatase level and low serum vitamin D. Skeletal survey revealed multiple patchy areas of lucency with irregular margins in the long bones, skull and pelvis. Bone mineral density was low suggestive of osteoporosis. Her fracture of the right midshaft of the femur was due to polyostotic fibrous dysplasia with underlying McCune-Albright syndrome. As bisphosphonate is required in fibrous dysplasia, she was treated with multiple doses of intravenous zoledronate starting at 0.0125 mg per kg which she tolerated well and then increased to 0.025 mg per kg. Her response was good, evidenced by reduced alkaline phosphatase level and improved bone mineral density. Her fracture healed with no complications or incidence of new fracture.

CONCLUSION
The administration of intravenous zoledronate enhances bone mineral density and demonstrates improvements in bone biomarkers. It was well tolerated and should be used in McCune-Albright syndrome with fibrous dysplasia of the bone.

EP_P030

ARM SPAN MINUS HEIGHT AND ITS RELATION TO FINAL HEIGHT IN PATIENTS WITH CONGENITAL ADRENAL HYPERPLASIA

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INTRODUCTION

This study aimed to analyse the sensitivity and specificity of arm span minus height (AS-Ht) in predicting final height outcomes in paediatric patients with Congenital Adrenal Hyperplasia (CAH) at Hospital Universiti Sains Malaysia, Kelantan. The management of CAH with glucocorticoid replacement is challenging. Undersuppression of sex hormones leads to precocious puberty and short final height while over-suppression with glucocorticoid leads to obesity and impaired growth too. Therefore, we need clinical tools to help clinicians to predict final height. Good control CAH will have near normal AS-Ht. Poor control CAH will have a high value of AS-Ht which indicates short linear height.

METHODOLOGY

A cross-sectional study that recruited 31 CAH patients and 78 control group participants. We analysed patients' demographics, clinical characteristics, and auxological parameters (arm span, arm span height, midparental height). The sensitivity and specificity of AS-Ht in predicting final height were calculated.

RESULT

AS-Ht was significantly higher in the CAH group; (3.8 ± 3.9) compared to the control group; (1.0 ± 2.0, $p<0.001$). The Sensitivity and specificity of AS-Ht for predicting final height were 60.9% and 62.5%, respectively with AUC of 0.622, 95%CI (0.38,0.86). 74.2% of CAH patients had good predicted final height while 25.8% had poor final height outcome.

EP_P031

LATE PRESENTATION OF ADVANCED CENTRAL PRECOCIOUS PUBERTY

https://doi.org/10.15605/jafes.039.S1.246

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INTRODUCTION/BACKGROUND

Precocious puberty is characterized by the emergence of secondary sexual traits in females before the age of eight, and in males before the age of nine. Late presentation in a setting of precocious puberty inadvertently resulted in short final adult height. We describe a case of advanced central precocious puberty diagnosed at a later stage.

CASE

A 9-year-old female visited Klinik Kesihatan with symptomatic anaemia. Later, at 10 years and 4 months old, she visited the combined reproductive endocrine clinic. Her mother was unsure of her exact height changes, but she weighed 42 kg (>97th centile) and experienced thelarche at 6-7 years old. She attained pubarche at 8 years old and menarche at 8 years and 6 months old. Her bone age was advanced >3.4 SD with an estimated bone age of 14 years and a chronological age of 10 years and 8 months. Her height was 147 cm (3rd centile).

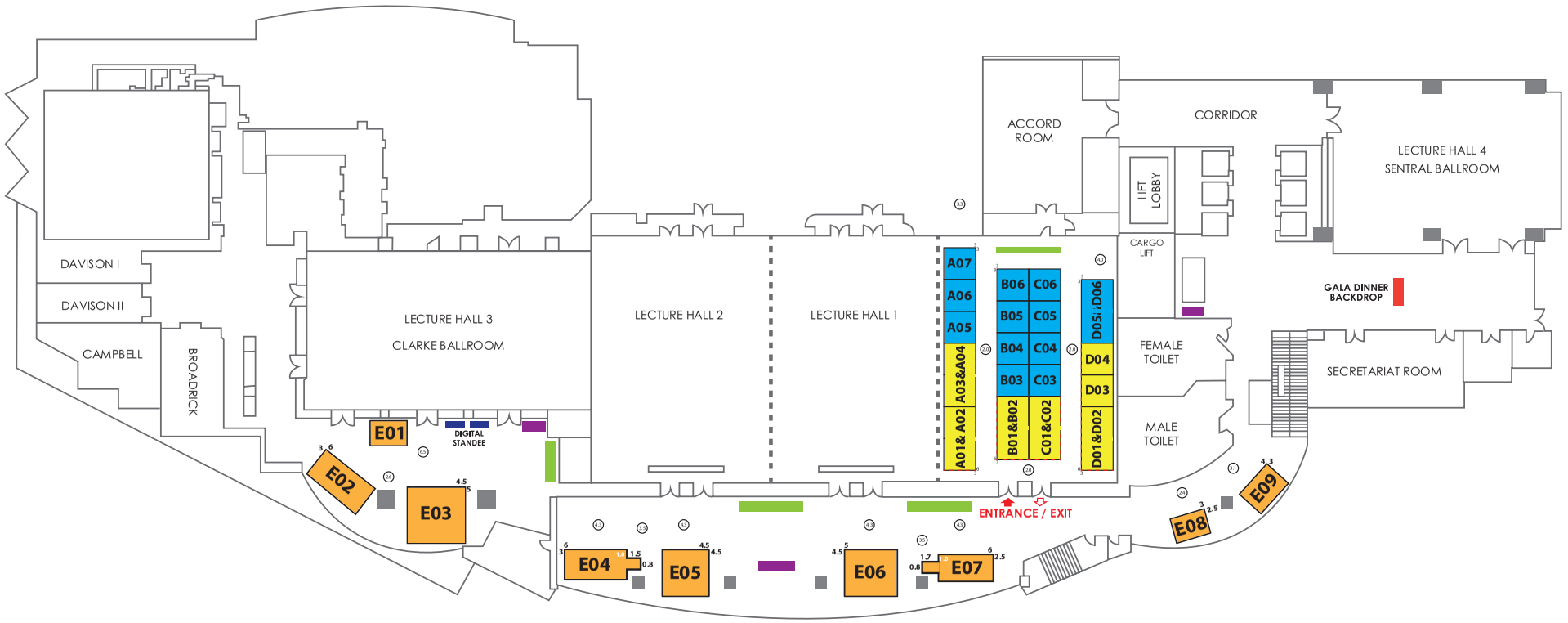
Hormonal profiles revealed E2 82 pg/mL, LH 10.34 IU/L, FSH 4.7 IU/L, Prolactin 573.7 m IU/L, TSH 1.08 m IU/L (0.35-4.94), T4 11.83 pmol/L (9-19.05), DHEAS 3.2 ug/dL (0.92-7.6), Basal 17-OHP 12.64 nmol/L (1.2-11.4). Synacthen 17-OHP test ruled out nonclassical congenital adrenal hyperplasia with peak 17-OHP 21.8 nmol/L at 60 minutes.

Transabdominal sonography revealed a uterus 5.8 cm x 3.8 cm with an endometrial thickness 11.55 mm. No obvious ovarian/adnexal mass and no obvious adrenal mass. Elevated LH level >0.3 IU/L indicates central precocious puberty and obviates the need for further GnRH stimulation test. IM Decapepty (Triptorelin) 3.75 mg monthly was administered to arrest her puberty aiming to reduce psychosocial stress due to early menarche and to improve final adult height.

CONCLUSION

Menarche is a late manifestation of puberty. In cases where a young girl presents with menses, likely, the optimal timing for pubertal blocker administration has already been missed. Therefore, early detection and management of precocious puberty is imperative.

VENUE LAYOUT



PREMIER

E01	CDiC
E02	ABBOTT
E03	Boehringer Ingelhiem
E04	Novo Nordisk
E05	Novo Nordisk
E06	Zuelig Pharma
E07	Zuelig Pharma
E08	Taisho Hoepharma
E09	Astra Zeneca

EXECUTIVE

A01&A02	Merck
A03&A04	P&G
B01&B02	Servier
C01&C02	DKSH
D01&D02	Amgen
D03	Nano Medic
D04	Ethos

STANDARD

A05	Medtronic	C03	Hovid
A06	MetaMed	C04	Kyowa Kirin
A07	Healthcon	C05	SunPharma
B03	Nestle	C06	Hyphens
B04	Brego	D05& D06	Duopharma
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