TAMIL NADU FEDERATION OF OBSTETRICIANS & GYNAECOLOGIST

eNewsletter

Volume 2

# Poly Cystic Ovarian Syndrome

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# President's Message

**Dear Comrades**

Warm greetings from me. Hope all of you and your family are keeping good health. I am very happy that we are coming out with the second Newsletter on PCOS. PCOS is the fascinating disorder for Gynaecologists, Reproductive Medicine specialist and Endocrinologists. This contains discussions on all aspects of PCOS and can be used as a ready reckoner in your library

Enjoy reading it and stay safe.
Jai Hind!!!

Dr. Anjalakshi Chandrasekar
Founder President TNFOG

# Secretary's Message

**Dear Comrades**

Incidence of PCOS, the ill-understood phenomenon has been on the rise due to over- indulgence in media, be it audio-visual or social through the smart phones, followed by junk food and zero-calorie carbonated drinks consumption, exercise-less sedentary lifestyle. PCOS symptoms may begin shortly after puberty, but can also develop during the later teen years and early adulthood. If left uncorrected it leads to adverse marital issues and in future to metabolic syndrome.

It is to address these issues we have none other than the authority on PCOS Dr. Duru Shah. On this happy occasion we are also releasing our second newsletter.

TNFOG, as you all know, came into being only this January, is conducting its First Midyear Conference later this month – certainly a bigger step for a toddler, made possible with all your support and encouragement. Thanks to one and all.

Planning an event in this covid period is a laborious effort but equally important is ensuring its success with maximum participation. It is all in your hands, dear members, Presidents and Secretaries of all constituent OG societies. Let us all work together towards this end!

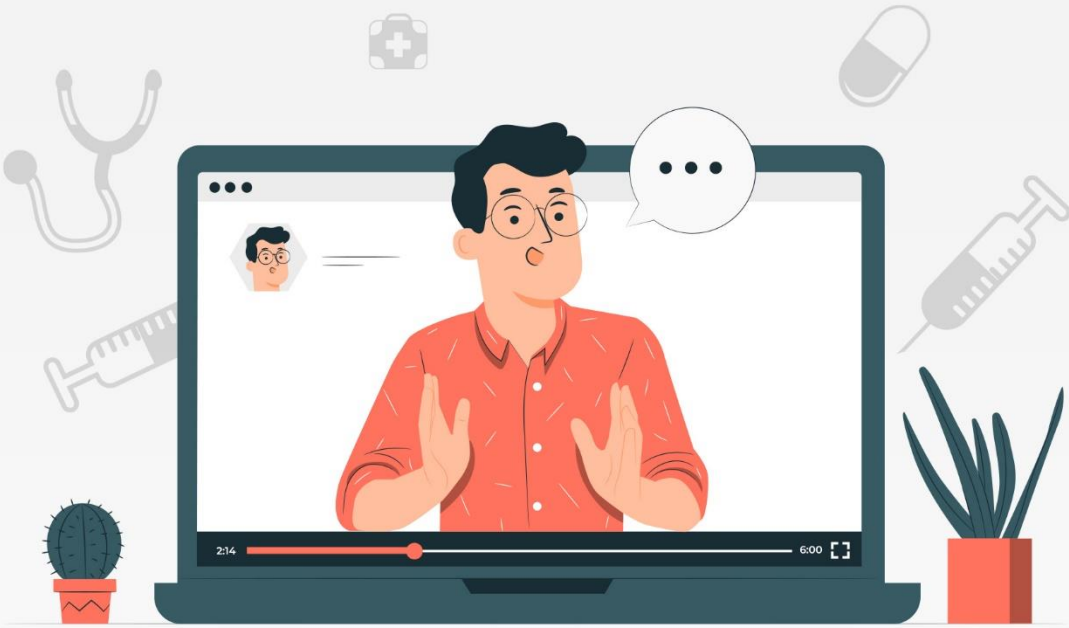
Wishing you a great scientific feast on PCOS!

Dr. S Sampathkumari
Founder Secretary - TNFOG

## TNFOG Plans to conduct TWO CME Program Every Month

**1. Marathon CME  2. Magalir Nalam**

# MARATHON CME??
# THIS POINTS TO TWO THINGS




### 1. The YUVA OGCIAN Competition

Yes, every month 2nd Friday CME will have a session with 2 YUVA speakers, Consultants less than 35 years.

The session will be judged by the same judges and at the end of the year,
First, Second and Third prize will be awarded to the best speakers at the Annual conference.

All societies gear up and suggest one YUVA speaker of your society.

**2. There is a question at the end of every session in the CME. The first Delegate who answers the question will be awarded a prize. This will continue in all the CMEs.**

**BE READY TO WIN THE PRIZE**

# TNFOG MARATHON
# CME ON "PCOS"

**DATE: 11.06.2021 (FRIDAY) | TIME: 4.30 – 7.00 PM**

## Scientific Programme

| DURATION | TOPIC | SPEAKERS |
|---|---|---|
| 04.30 — 05.00 pm | Inauguration | |
| | Welcome Address | Dr. Anjalakshi Chandrasekar |
| | Address by Chief Guest | Dr. Duru Shah |
| | Address by Guest of Honour | Dr. Rishma Dhillon Pai |

## Session I : Yuva Session

**Chairpersons : Dr. Nidhi Sharma & Dr. Vijayalakshmi Kandasamy**

| | | |
|---|---|---|
| 05.00 — 05.15 pm | Pathophysiology of PCOS | Dr. Prakruthi |
| 05.15 — 05.30 pm | Diagnosis of Pcos | Dr. Madhumitha |
| 05.30 — 05.35 pm | Q & A | Dr. Akila Ayyappan |

## Session II

**Chairperson: Dr. Maya Menon**

| | | |
|---|---|---|
| 05.35 — 06.00 pm | Update on PCOS | Dr. Aswath Kumar |
| | Q & A | Dr. Akila Ayyappan |

## Session III

**Moderators: Dr. T.K. Shaanthy Gunasingh & Dr. Rajapriya Ayyappan**

| | | |
|---|---|---|
| 06.00 — 07.00 Pm | PCOS Case Scenarios | Panelists<br>Dr. Shantha<br>Dr. Subashini<br>Dr. Sujatha<br>Dr. Vairamala<br>Dr. Prasanna<br>Dr. Emily Divya |
| | Q & A | Dr. Akila Ayyappan |
| 06.50 pm | Vote Of Thanks | Dr. Radha Senthilnathan |

**For all Registrants, Certificate will be provided**

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# TNFOG MARATHON
# CME ON "PCOS"

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**Dr. Anjalakshi Chandrasekar**
President, TNFOG

**Dr. S. Sampath Kumari**
Hony, Secretary, TNFOG

**Dr. Vijayalakshmi Gnanasekaran**
Treasurer, TNFOG

## CHIEF GUEST

**Dr. Duru Shah**

## GUEST OF HONOUR

**Dr.Rishma Dhillon Pai**

## CHAIRPERSONS

**Dr. Nidhi Sharma**

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Dr. Prakruthi

Dr. Madhumitha

Dr. Aswath Kumar

## MODERATORS

Dr. T.K. Shaanthy Gunasingh

Dr. Rajapriya Ayyappan

## PANELISTS

Dr. Asha Rao

Dr. Shantha Devi

Dr. Subashini

Dr. Sujatha

Dr. Vairamala

Dr. Prasanna

Dr. Emily Divya

# TNFOG MARATHON
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## CO-ORDINATOR

Dr. Akila Ayyappan

## VOTE OF THANKS

Dr. Radha Senthilnathan

**1**

**ICOG Credit Point Granted**

## Get Exiting Prize!

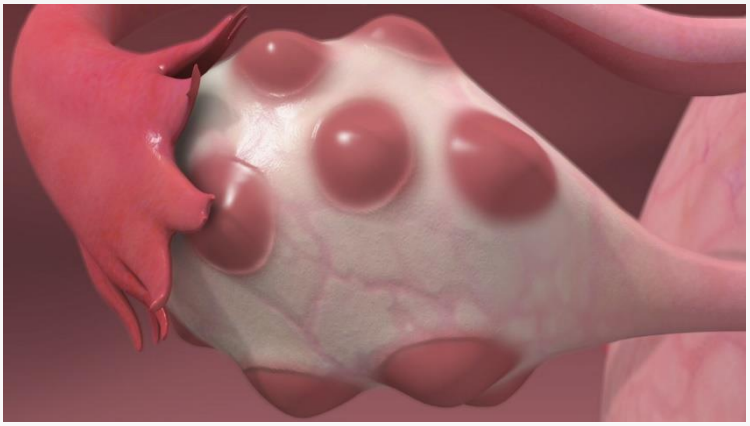
Be the ***FIRST*** to answer the 'Question' at the end of each session

ARTICLE 1

# DIAGNOSIS OF POLYCYSTIC OVARY SYNDROME

**Dr. T K Shaanthy Gunasingh**,
MD, DGO, FICOG
Former Director, IOG and ISO-KGH
Past President, OGSSI

**PCOS** is one of the most common endocrinopathy in women. Diagnosis is currently made by **Rotterdam Criteria** which is the most inclusive. Two out of three criteria are required to make the diagnosis.



a. Oligo and / or anovulation
b. Clinical and / or biochemical signs of hyperandrogenism
c. Polycystic ovaries by ultrasound

Oligo or anovulation present as irregular cycles. Irregular menstrual cycles are defined as:

- >1 or <3 years post menarche: <21 or >45 days
- >3 years post menarche to perimenopause: <21 or more than 35 days or <8 cycles per year
- >1 year post menarche: > 90 days for any one cycle
- Primary amenorrhoea by age 15 or > 3 years post thelarche

**Hyperandrogenism** may be diagnosed **clinically** in adults by acne, hirsutism or male pattern hair loss and in adolescents by severe acne and hirsutism or **biochemically** by elevated serum androgens. **Modified Ferriman and Gallwey Score** is used for assessing hirsutism depending on ethnicity. **Ludwig Visual Score** is preferred for assessing alopecia. **Total testosterone** using liquid chromatography – mass spectrometry should be measured. Free testosterone is the most sensitive test for hyperandrogenemia if done by equilibrium dialysis. Currently available free testosterone assays are unreliable. It can also be calculated from Sex Hormone Binding Globulin (SHBG). Abnormally low **SHBG** is a risk factor for increased biologically active testosterone and a more severe PCOS phenotype. **DHEAS** is not routinely measured. In severe hyperandrogenism, high levels of DHEAS indicate adrenocortical cancer. Role of serum **androstenedione** in evaluation of PCOS is unclear – important in certain populations like Icelandic women to document hyperandrogenemia.

**Ultrasound**: ≥ **20 follicles** in each ovary, and / or an **ovarian volume ≥ 10 ml** ensuring no corpora lutea, cysts or dominant follicle with a frequency bandwidth 8MHz should be documented. Transvaginal ultrasound is preferred if sexually active and if acceptable to the individual. Age based criteria defining PCOS have been proposed as ovarian volume and follicle number decrease with age. Ultrasound should not be used for the diagnosis of PCOS in adolescents with a gynaecological age of < 8 years due to the high incidence of multifollicular ovaries. However, pelvic ultrasonography is indicated if serum androgen levels or the degree of virilisation is concerning for an ovarian tumour. In postmenopausal women, presumptive diagnosis is based on a well-documented long-term history of oligomenorrhoea and hyperandogenism during reproductive age. PCO morphology on pelvic ultrasound provides additional support.

Morning basal serum **17 hydroxy progesterone** should be done in all women with possible PCOS to rule out non-classic congenital adrenal hyperplasia. Tests to rule out other causes of oligomenorrhoea should include serum **prolactin, TSH and basal FSH**. In general, it is not necessary to measure LH. AMH is currently not past of evaluation.

If women are **already taking pharmacological therapy** like COCP, metformin or spironolactone, serum androgens to be evaluated after stopping the drug for 4 to 6 weeks.

Once the diagnosis of PCOS is made, **cardio metabolic risk assessment** should include measurement of blood pressure, body mass index, fasting lipid profile and an oral glucose tolerance test.
No tests for insulin resistance.

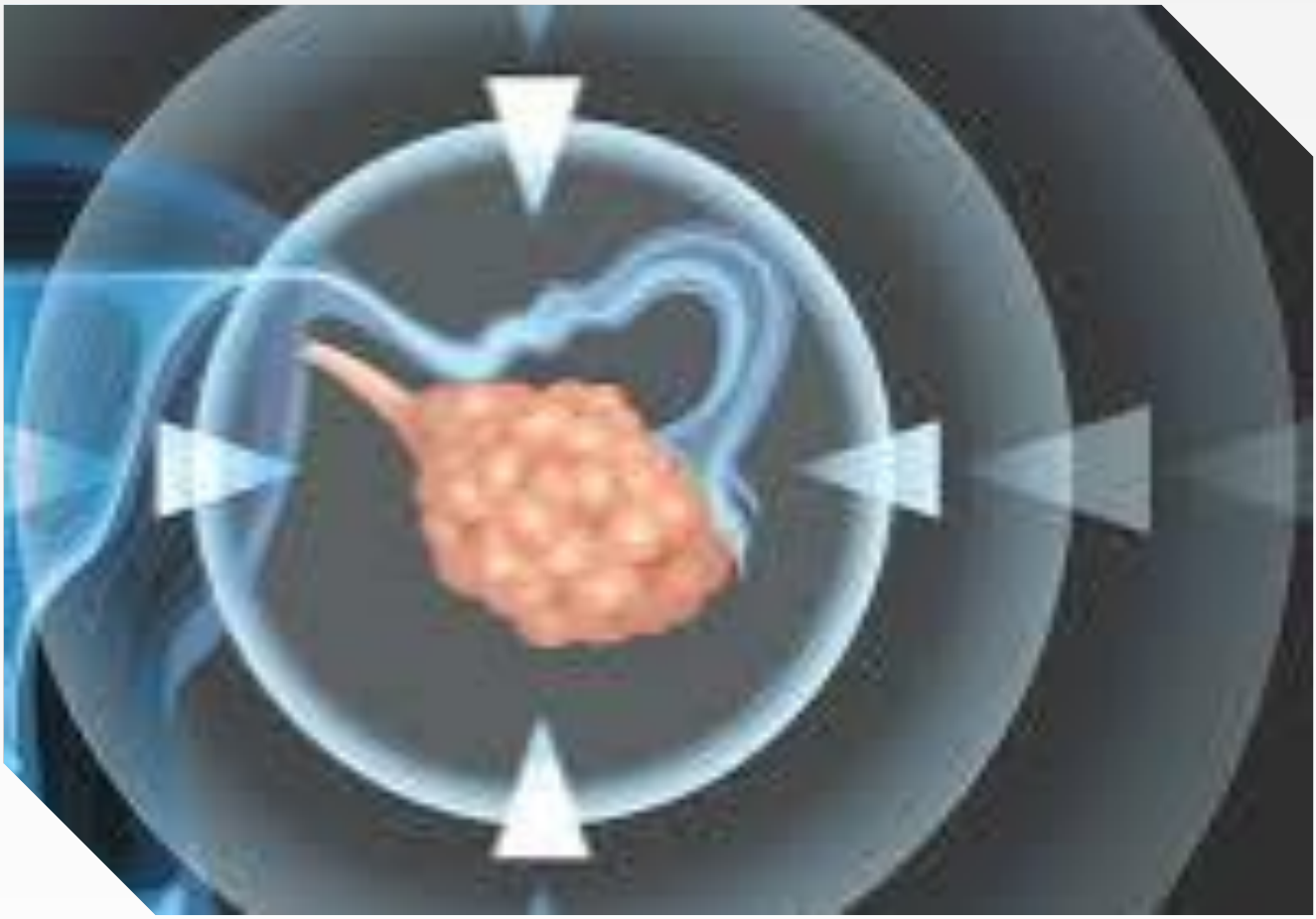
If symptoms and signs of **obstructive sleep apnea** are present (snoring, excessive daytime sleepiness, morning headache), sleep medicine specialist should be consulted.

**Anxiety and depressive symptoms** should be routinely screened in all adolescents and women with PCOS using Patient Health Questionnaire (PHQ) – 9 for depression and the Generalized Anxiety Disorder 7 (GAD – 7) Anxiety Scale. If **eating disorders** are suspected, further assessment, referral and treatment should be offered.

No screening for Nonalcoholic Fatty Liver Disease.
PCOS is essentially a diagnosis of exclusion.

# References:


1. UpToDate on PCOS April 2021
2. ACOG PCOS Guideline 2019
3. ESHRE PCOS Guidelines 2018
4. International evidence-based guideline for the assessment and management of PCOS – Fertility and Sterility 2018 Teede *et al*.
5. Women's experiences of PCOS diagnosis, Gibson, Fam Pract, 2014

ARTICLE 2

# CAN WE PREVENT PCOS?

**Prof. Dr. Revathy Janakiram**
MD DGO MNAMS FICOG FICMCH,
Past Vice President FOGSI, 2016
Vice President TNFOG, 2021

**Being a metabolic disorder, PCOS may start in Utero. Prenatal origin, changes in etiology, changes in management of PCOS has led us to explore the possibility of prevention of PCOS.**

At puberty significant metabolic and endocrine changes occur in the female. This forms the background for physical & physiological transformation from a child to adolescent. There is a physiological hyperinsulinemia, which is necessary to counteract the lipolytic effect of Growth hormone. Insulin excess inhibits proteolysis, thereby helping growth & development. But when such physiological changes persist either as a result of Genetic or environmental cause, it can lead on to PCOS.

Apart from Genetic, the added factors for persistence of hyperinsulinemia are lifestyle, environment, diet, stress etc., which are modifiable. Genes load the Gun. But lifestyle pulls the trigger. The fetal developmental process is similar to creating a computer .The Genes provided by parents are the “Hardware”. Other factors in the maternal environment are the “Software” that causes the Hardware to behave in a certain way. The Software consists of “Epigenes” which modify the behaviour of Genes. Nutri genomics study show that even Gene expression can be modulated by Lifestyle changes.

- Deficiency of certain trace elements, are linked with ovulatory dysfunction, like
- Zinc deficiency – attenuates ovulation
- Manganese deficiency – delays ovulation
- Cobalt deficiency - ↓ fertility
- Magnesium & Chromium deficiency – provokes IR.

Thus trace elements play a vital role in pathogenesis of PCOS. Obviously prevention of such deficiencies may help in PCOS.

Endocrine disruptor chemicals: On Feb 2013 WHO released an updated report on role of EDC in human health. EDC are likely to affect all hormonal systems.

By avoiding exposure to EDC it may be possible to reduce PCOS symptoms.  Meat and dairy consumption in children may also reflect ingestion of environmental endocrine-disrupting chemicals (EDCs) that have accumulated in animal tissues.

# Normal ways to prevent PCOS complications are:

**1. Be strategic with calories:**
One study indicates that caloric intake timing can have a big impact on glucose, insulin and testosterone levels. Lowering insulin could potentially help with infertility issues. Women with PCOS who ate the majority of their daily calories at breakfast for 12 weeks significantly improved their insulin and glucose levels as well as decreased their testosterone levels by 50 percent, compared to women who consumed their largest meals at dinnertime. The effective diet consisted of a 980-calorie breakfast, a 640-calorie lunch, and a 190-calorie dinner.

**2. Decrease AGEs:**
Women with PCOS have been shown to have higher levels of advanced glycation end products (AGEs) in their blood. AGEs are compounds formed when glucose binds with proteins, and are believed to contribute to certain degenerative diseases and aging. One small study found that cutting down on dietary AGEs significantly reduced insulin levels in women with PCOS. Foods high in AGEs include animal-derived foods and processed foods. Applying high heat (grilling, searing, roasting) increases levels.

**3. Avoid deficiency of trace elements.**

**4. Omega 3, likely to reduce Androgens.**

**5. Get moving**.
Though no appropriate clinical trials are available on primary prevention of PCOS, research is going on to explore the possibilities. Early diagnosis and treatment help to prevent long term complications such as Infertility, Metabolic syndrome, Diabetes mellitus & Cardiac problems.

ARTICLE 3

# ADOLESCENT PCOS

**Dr. S. Sampathkumari**
MD, DGO, FICOG, FIME, FC Dia.,
Prof & HoD – OG, SMMCH & RI, Chennai
FOGSI Vice President Elect, 2022

**PCOS is not a disease but an ill-understood phenomenon defined in various forms over the years.** Prevalence of PCOS in India ranges from 3.7 to 22.5 per cent (A news report on 29-Nov-2019) and prevalence of PCOS in Indian adolescents is 9.13%. (J Pediatr Adolesc Gynecol, 2011 Aug;24(4):223-7). And, PCOS is common in women suffering from obesity. "Almost 80% of the PCOS patients are obese," said Dr. Duru Shah, adding that urban Indian women may be at a higher risk due to their poor lifestyle, eating habits and lack of physical activity. There is nearly a 40% chance that a female child may develop PCOS if the mother has it.

Polycystic ovarian syndrome, is a clinical and biochemical, multi factorial, heterogenous syndrome characterized by ovulatory dysfunction and hyperandrogenism.

Diagnosis and Management in adolescence is important to avoid the long-term sequelae such as metabolic syndrome.

The term 'adolescence' is derived from Latin 'adolescere'- meaning to grow, to mature and considered as transition from childhood to adulthood. It's the progression from appearance of secondary sexual characteristics to sexual and reproductive maturity is the marked feature.

**Adolescents are categorized by WHO as:**

- Early adolescence (10 -13 yrs): Spurt of growth of development of secondary sex,
- Middle adolescence (14-16 yrs): Separate identity from parents, new relationship to peer groups, with opposite sex and desire for experimentation
- Late adolescence (17-19 yrs): Distinct identity, well-formed opinion and ideas.

According to National Institute of Health Criteria, PCOS prevalence has been reported to be 8 to 13%, but the prevalence in adolescence has not been specifically studied as yet. In adolescent girls overweight & obesity amplifies the clinical implication of PCOS & increases the risk of metabolic function.

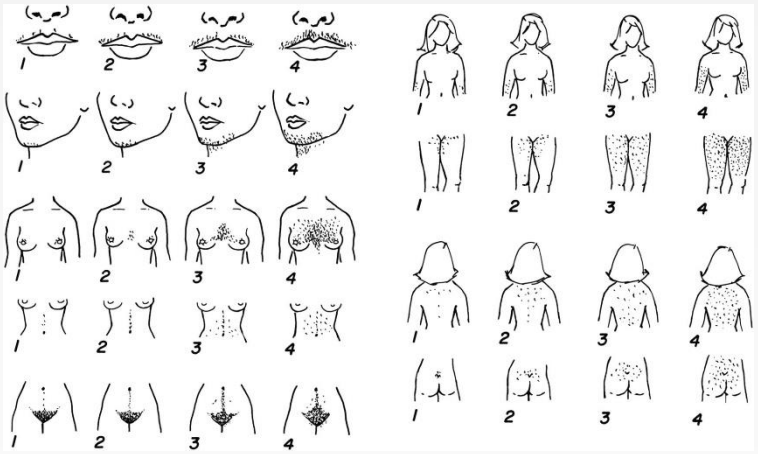
Adolescent girls with PCOS may present with abnormal menstrual periods, hirsutism, and/or acne. Menstrual irregularity is a common feature of PCOS and is often the earliest clinical manifestation in the adolescent. However, this menstrual pattern can be difficult to distinguish from anovulation associated with puberty because, the hypothalamic-pituitary ovarian axis matures progressively over a period of several years after menarche

# Mechanism of PCOS:

Hyperinsulinemia and insulin resistance:
Acts at 3 levels – HP axis, ovary & liver.
*On ovary*: Insulin acts synergistically with LH to stimulate thecal cells& ↑ levels of Androstenedione and testosterone

*On Liver*: Inhibition of hepatic syntheses of SHBG, Increased circulating concentrations of Sex steroid hormones

Other signs of PCOS are Hirsutism, Acne, Acrochordons, Acanthosis Nigricans, Alopecia & infertility.
Hirsutism grading depends on FERRIMAN - GALLWEY SCALE.



Score more than 6 indicates Hirsutism.

**PCOS investigations**
Rule out Pregnancy, thyroid -TSH, Fasting & 2-hr GTT, Prolactin, Total and free testosterone, DHEAS, 17-OH progesterone, FSH, LH, estradiol (in amenorrheic adolescents), Lipid profile and ultrasound if needed.
Measurements of total and/or free testosterone is the hormone to determine hyperandrogenemia.
Dehydroepiandrosterone sulfate (DHEAS) to screen for primary adrenal source of hyperandrogenemia.
Recommendations have been made about the usefulness of a morning 17-OHP level of >200 mg/dL as a screening tool for NC-CAH.

**Criteria for metabolic syndrome in adolescents are:**
1. Blood glucose > 100mg,
2. HDL-C <40mg,
3. Triglycerides >110mg,
4. Waist circumference >90$^{th}$ percentile for age & sex,
5. Blood pressure >90th percentile for age & sex

Presence of any 3 of above.

**Management of PCOS in adolescents**
Lifestyle modifications is the first-line management of adolescents with PCOS. Improved menstrual regularity, decreased cardiometabolic risks, and improved androgen excess can all be achieved with weight loss - 3 meals, 3 snacks & 30 minutes of exercise.

Menstrual irregularity corrected by progesterone alone or estrogen-progesterone combination.

The estrogen-progesterone combination suppresses the endogenous hypothalamic-pituitary-ovarian (HPO) axis and reduces ovarian androgen production. COC increase sex hormone binding globulin & thereby reduce androgen excess.

- *COC with antiandrogen*, Spironolactone can improve menstrual irregularities and hyper androgenimic features as hirsutism, acne & alopecia
- *OC Pills* can be given for 6 months to 1 year with life style changes.
- *Cosmetic hair removal,* Electrolysis and laser hair removal therapies done before treatment.
- *Metformin*, Used in cases of Insulin resistance cases & to certain extent reduce obesity also. Metformin can be combined with OC Pills also.
- *Orlistat* to reduce obesity used rarely.
- *Laparoscopic drilling* of PCOS not advised for adolescents.
- *Bariatric surgery* rarely advised for adolescents.
- Role of *Myoinositol* needs still more studies.

**Conclusion**
PCOS in adolescents needs to be diagnosed earlier with history & needed investigations. USG is not mandatory in adolescents. LIFE STYLE CHANGE, OCPILLS (in menstrual problems), Antiandrogen OCP (Hirsutism & acne), Metformin is preferred in insulin resistance cases. Depending on the case above mentioned drugs can be combined. Avoid complications by diagnosing it earlier.

## *Adolescent Health is Nation's Wealth*

ARTICLE 4

# LIFESTYLE MODIFICATIONS IN PCOS IN ADOLESCENT GIRLS

**Dr. Rajapriya Ayyappan**
MD DNB FRM FICOG FRCOG
Managing Director, Srinivas priya hospital & Om fertility center
Secretary, Indian Fertility Society, TamilNadu Chapter

**Polycystic ovary) syndrome (PCOS** is a common endocrine condition affecting up to 18 % of reproductive-aged women. It manifests with adverse reproductive (hyperandrogenism, menstrual dysfunction, infertility and pregnancy complications), metabolic (insulin resistance, dyslipidaemia, non-alcoholic fatty liver disease, endothelial dysfunction, early atherosclerosis and increased impaired glucose tolerance, type 2 diabetes mellitus and cardiovascular disease), and psychological (worsened quality of life and increased anxiety and depression).

The Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group addressed the PCOS' health implications. . About 30-40 % women with PCOS also have the metabolic syndrome, most do not. Obesity appears to play an independent role in all of the long term complications and may best predict both reproductive and metabolic dysfunction, as well as negatively affect the response to treatment.

It is important to catch them young, to instill lifestyle changes as early as adolescent stage of woman's life. However it is a challenge to diagnose polycystic ovaries in Adolescents who have functional ovarian cysts as normal sonographic findings by Transabdominal sonography. They may present with anovulatory and/or irregular periods occur in ~40% of girls until about 2-3 years following menarche with clinical evidence of androgen excess specially acne is common during puberty and resolves over time. High prevalence of obesity confounds diagnosis, recent lifestyle change in covid times has seen a BMI rise. BMI>24 is associated with oligomenorrhea.

Lifestyle modification is the first form of therapy, combining behavioral (reduction of psychosocial stressors), dietary, and exercise management. This non-invasive intervention causes 5 to 10% weight loss in obese girls with PCOS and brings positive outcomes. Reduced-energy diets (500–1000 kcal/day reduction) are effective options for weight loss over a period of 6 to 12 months. Dietary pattern should be nutritionally complete and appropriate for life stage and should aim for <30% of calories from fat, <10% of calories from saturated fat, with increased consumption of fiber, fibre, whole-grain breads and cereals, and fruit and vegetables. Women must eat sensibly to their appetite as small portions just to satisfy hunger, avoid eating food either because its tasty or is leftover. Structured exercise is an important component of a weight-loss regime; aim for >30 minutes per /day. Any physical activity which they enjoy like a game or dance or yoga and which burns calories must be selected and made as a routine. Individualization of the program, intensive follow-up and monitoring by a physician, and support from the physician, family, near and dear will improve compliance.

The covid pandemic has clearly devastated the lifestyle of our adolescent girls by closed down schools and colleges and gadget-based education. Women in reproductive age have to juggle multitasking, taking care of household chores and professional life of Online working from home. The food habits have changed in lockdown ,as availability becomes an issue, increased consumption of processed ready meals and of hotel food. The sleep cycle has got reversed with the new norm becoming late awakening at brunch time, Online classes, movies ,digital entertainment platform and the virtual world is quickly replacing the real! Trying to reset the clock on quality of sleep hours and keeping stress level low is key to LSM besides regular exercise and low calorie balanced diet.

Educate the adolescent as to how to manage her medical care independently, as it is lifelong disease. Review regularly, the adolescent's concerns about her physical, reproductive, sexual and mental health. Indeed, the management of the PCOS patient varies with stage of life and it's goal. The medical management of PCOS can be broken down into four components, three of which are "acute" issues (control of irregular menses, treatment of hirsutism and management of infertility) and one that is chronic, "metabolic syndrome". September is celebrated all over the world as PCOS awareness month. Care givers need to reach out to the affected young women that PCOS is a lifestyle disease with remissions and exacerbations depending on the persistence of life style modifications.

ARTICLE 5

# POLYCYSTIC OVARIAN SYNDROME IN INFERTILITY

**Dr Asha R. Rao**
MD DGO FICOG,
Director - RAO HOSPITAL, Coimbatore
Chairperson, Endometriosis Committee, FOGSI 2020-22

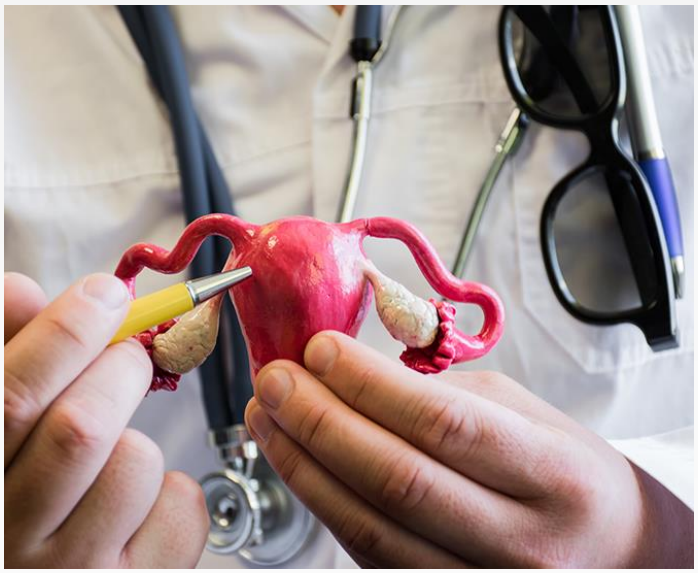
**Polycystic ovarian syndrome (PCOS)** is an endocrine and reproductive disorder which is one of the commonest but treatable cause of infertility in women.

**Prevalence:** 5% to 13% of women in reproductive age have PCOS.
The prevalence of infertility in women with PCOS varies between 70 and 80%

**Pathology**: The most common cause for infertility in women with PCOS is anovulation which is primarily due to hyperandrogenism and/or insulin resistance
PCOS accounts for 80% of anovulatory infertility.

**Evaluation in PCOS:** According to the American Society for Reproductive Medicine, the evaluation of infertility in women with PCOS should start after six months of attempting pregnancy without success if the couple has regular sexual intercourse without using contraceptive methods.

**80% are obese**, **25% has dysglycemia and 65% has IR at the time of diagnosis of PCOS**. Hence all women with pcos should be screened for impaired sugars.



To optimize the efficacy of the treatment of infertile women with PCOS, semen analysis (spermogram) is mandatory before deciding on treatment.

However, tubal patency evaluation may not be necessary prior to initiating ovulation induction treatment. Notably, if a patient is resistant to this drug and/or requires the use of gonadotropins and/or presents with other causes of infertility, a tubal patency evaluation becomes mandatory prior to initiating the therapeutic treatment of infertility

# Management:

**Non pharmacological management**:
Lifestyle modification in the form of calorie restriction, low carb diet and modest exercise is considered the first-line treatment for the management of infertile anovulatory PCOS women. It is recommended not only to overweight ones but also the ones with normal BMI as central adiposity and increased visceral fat is found in 2/3 of Lean PCOS.

It helps in reducing the hepato visceral fat ,10% weight loss reduces 30% visceral fat and hence improved insulin sensitivity, increased SHBG levels which may help in Spontaneous ovulation and pregnancy.

**British Fertility Society Recommendation:**
- No OI if BMI>35,
- In young women <37 years – WAIT TILL BMI < 30 before OI
- Counselling regarding the **importance of weight reduction** should be done in all PCOS women in terms of
    - Improved response to OI drugs
    - Decreased time to Pregnancy,
    - Decreased risk of Miscarriage
    - Pregnancy complications
    - Epigenetic influence on future generations
    - Pre-marital counselling is worthwhile in young woman with PCOS for better fertility outcomes.

**Pharmacological measures**
**First line: Ovulation induction with oral ovulogens**
Goal of Ovulation induction should be using the lowest effective dose of medication to achieve mono-follicular development.

Letrozole is recommended as the first line ovulation induction agent as it is proven to be superior to Clomiphene citrate in many RCTs. Letrozole favours monofollicular ovulation and does not have anti-estrogenic effect on the endometrium. CPR with Letrozole is 61% vs 40%, LBR 49% vs35% and Time to pregnancy 4 months Vs 6 months when compared to CC, Systematic review and network meta-analysis, (Wang *et al*, BMJ 2017)

Maximum recommended dose for Letrozole is 5 mg and CC is 100mg for the maximum of 6 cycles. (Cochrane review Franik *et al* ,2018)

15% of PCOS could be resistant to oral ovulogens, one may resort to second line measures.

Addition of metformin to letrozole / CC may be beneficial in achieving slightly higher rates of ovulation in women who are resistant to ovulation induction. Current recommendation is to start metformin for metabolic indications and not for all.

**Second line measures:**
1) Ovulation induction with **Gonadotropins:**

Chronic low dose protocol is preferred as gonadotropin stimulation carries a high risk of higher order pregnancies and OHSS

Urinary gonadotropins are found to be equally efficacious and safe as compared to recombinant ones. Choice of gonadotropins can be decided based on the patient affordability

2) **LOD** is reserved only in a very small subset of patients who cannot afford frequent hospital visits for monitoring, having financial restrictions for IVF

LOD is associated with the risk of adhesion formation and the risk of losing the ovarian reserve and more than 50% of women anyway need Gonadotropins for ovulation induction even after LOD as the cumulative pregnancy rate is 40% at 6 months of LOD

**Third line measure: IVF**
Though the ovarian reserve and cumulative pregnancy rates are good, one has many challenges with IVF treatment in PCOS patients like

- Propensity for an exaggerated ovarian response
- Difficulties in titrating gonadotropin dose
- Increased risk of cycle cancellation
- Increased incidence of early and late OHSS
- Increased risk of spontaneous pregnancy loss

Measures to be followed to prevent OHSS prior to, during stimulation and post oocyte retrieval are:

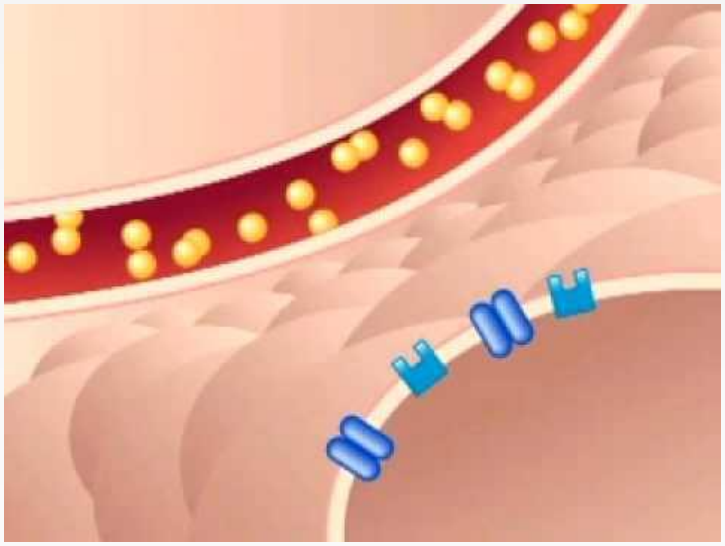
- Antagonist protocol should be the protocol of choice
- Careful titration of the Gonadotropin dosage
- GnRH agonist for triggering final oocyte maturation
- Cryopreservation of all embryos (segmentation of IVF),
- Agents to promote luteolysis and reduce VEGF levels - (GnRh antagonists, Cabergolin, androgenic progesterones )etc  may be done in women with extremely high response .

ARTICLE 6

# PCOS & GDM

**Dr. Anjalakshi Chandrasekar**
MD, DGO, PhD,
Prof & HOD, OG, Madha Medical College,
Past President, OGSSI,
Founder President, TNFOG

PCOS and GDM – are two edges of a knife.



**PCOS** is a common endocrine disorder seen in women of reproductive age characterised by Hyper androgenism, Hyper-insulinaemia, insulin resistance and Hyperestrogenism

**Insulin Resistance and Hyper-insulinism:**
**Insulin Resistance:**
Though insulin resistance is not included in the definition of PCOS, many studies documented that both lean and obese PCOS have insulin resistance (IR). This IR has associated with various disorders including IGT, T2DM, HT, Dyslipidaemia and Heart Disease, together known as Syndrome X or Dysmetabolic Syndrome. This IR plays a central role in the pathogenesis of PCOS.

IR is defined as a state in which greater than normal insulin are required to produce a normal response. Because of peripheral insulin resistance there is increased pancreatic secretion of insulin resulting in Hyperinsulinaemia. The insulin resistance in PCOS is due to post binding defect of insulin receptor. There is also Beta cell dysfunction in PCOS

**Hyperinsulinemia:**
It plays a role in the pathogenesis of Hyperandrogenism. Hyperinsulinemia stimulate P450c, 17alpha activity and is the key in the biosynthesis of ovarian androgens (17 OH progesterone, Androstenedione and Testosterone). In PCOS women the P450c, 17 alpha activity is increased. Amelioration of IR would return the activity of the enzyme towards normal. Further insulin indirectly stimulate ovarian androgen production by augmenting LH stimulated ovarian androgen biosynthesis

**Gestational Diabetes Mellitus:**
GDM is defined as varying degree of glucose intolerance which starts during pregnancy. It is associated with increased fetal and maternal morbidity as well as long-term complications in the mother and offspring. GDM & PCOS are the most common endocrine disorder in the reproductive age group, the prevalence of GDM varies from 9 to 25% and PCOS varies from 5 to15% depending upon the study population and criteria used for diagnosis. Both disorders are associated with IR, overwt/obesity and also genetic factors play a significant role. GDM & PCOS can be early signs of increased risk of manifest diseases related to insulin resistance such as T2DM and both disorders are connected with cardiovascular risk factors such as metabolic syndrome, HT and dyslipidaemia.

Several studies have shown that PCOS increases the risk for GDM. Mikole *et al* found that PCOS independently increases the risk of GDM and overwt was the strongest predictor of GDM in women with PCOS. The risk factors for GDM in PCOS are overwt/obesity, advanced maternal age, family history DM and personal history of dysglycaemia, own preterm birth, Ethinicity (south Asian/Indian) other predictors are FBG, non-high density lipoprotein and SHBG. The interesting observations in various studies are 7% increase in SGA in GDM+PCOS newborn, preterm birth is increased and the newborns are slightly shorter(1to1.5cm) The two conditions are interlinked and together increases the incidence of preeclampsia and pregnancy induced hypertension and neonatal hypoglycaemia. Many studies have observed an increased prevalence of the signs and symptoms of PCOS in women with previous history of GDM. Lean women with PCOS have a higher incidence of GDM & PIH than her controls. Maternal PCOS and GDM were also associated with 3 fold increased ODDs of neonatal hypoglycaemia. It is still uncertain to what extent coexisting GDM and PCOS affect pregnancy outcome.

**Risk of T2DM following GDM in PCOS women:**
The hallmark of T2DM is IR and Hyperinsulinaemia, hence both PCOS & GDM are risk factors for T2DM. The prevalence of T2DM in women with PCOS is five to10 times higher than those without PCOS. It is estimated that 10 to 50% of women with GDM + PCOS develop diabetes during the 5 yr interval following delivery. The predictors of both GDM and T2DM are obesity, F/H of T2DM, Waist hip ratio, pre. h/o GDM, ethinic background, HT, cigarette smoking, macrosomia and advanced maternal age

**Role of insulin sensitizer in PCOS and GDM:**
Metformin is an effective insulin sensitizer for treating T2DM and for preventing T2DM in high-risk population. In GDM use of metformin is associated with a lower risk of hypoglycaemia and potentially a lower wt. gain. It may be preferable to insulin for maternal health if it controls hyperglycaemia sufficiently, however metformin may slightly increase the risk of premature birth. but long-term outcome of offspring has not been evaluated. Thus pts. treated with metformin should be informed that the drug does cross the placenta and while no adverse effects on the fetus have been demonstrated, long term studies are lacking. Glueck *et al*, found that metformin during pregnancy in non-diabetic women with PCOS decreased preconception weight and resulted in less weight gain during pregnancy, decreased IR and the risk of developing GDM.

Metformin may improve ovulatory function in pts with PCOS. However metformin is not recommended as a first line drug for induction of ovulation. Studies showed that continuing metformin therapy throughout pregnancy in women with PCOS can improve pregnancy outcome by decreasing spontaneous abortion rates and preventing GDM with its inherent comorbidity and mortality rate. Whether metformin could reduce the occurrence of GDM in PCOS is still controversial. Recent meta-analysis showed that metformin had no significant effect on GDM in women with PCOS.

**Conclusion:**
Nationwide population based study has demonstrated that pregnant women with h/o PCOS have more than 2 fold increased probability of GDM compared with women without PCOS. Medication for PCOS or pre pregnancy use of OHA does not reduce the risk of GDM. Both IR and obesity increase the risk of dysglycaemia including GDM and T2DM. In a study on GDM risk factors in Iranian infertile women with PCOS, the results are the incidence of GDM were 44.4% in PCOS and 29.9% in non PCOS. The important and significant predictors for development of GDM in PCOS women are menstrual irregularity, serum TGL>150 mg% and pregestational metformin use. Obstetrician should be more aware of the increased risk of subsequent GDM in women with h/o PCOS. When at risk women become pregnant they should follow a stricter diet and adhere to wt. gain control to avoid obstetric complication due to GDM.

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ARTICLE 7

# LAPAROSCOPIC OVARIAN DRILLING

**Dr.Mala Raj**
Laparoscopic and Fertility Specialist,
Aesthetic Gynaecologist,
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**Introduction**

The most widely accepted surgical treatment for PCOS is Laparoscopic Ovarian Drilling (LOD). Setji et al stated that because of high sensitivity of polycystic ovaries to gonadotropin stimulation, it was plagued by an unacceptable rate of multiple pregnancies and OHSS. An alternative to medical approach is this surgical treatment. Cleemann et al stated that laparoscopic ovarian drilling can dissolve infertility within 4 – 6 months in 50-60% couples. Laparoscopic ovarian Drilling will shorten the time to pregnancy, reduce multiple pregnancy and OHSS and enable diagnosis of those women with anatomic infertility who can achieve pregnancy only with IVF treatment.

**Indications for Laparoscopic Ovarian Drilling**

Choosing the right patient for laparoscopic ovarian drilling is mandatory. In spite of all these advantages, laparoscopic ovarian drilling is not considered superior to Clomiphene Citrate, neither as a first line therapy for ovulation induction nor for Clomiphene Citrate failure or prior to In-vitro fertilization. A recent Cochrane systemic review of 9 RCTs and 16 trials concluded that there was no evidence of a significant difference in rates of clinical pregnancy (39.7% vs. 40.5%) or live birth (34% vs. 38%) in women with CC resistant PCOS undergoing laparoscopic ovarian drilling compared to other medical treatment. This implies that laparoscopic ovarian drilling is a valid, but not the sole option for CC resistant PCOS. Also laparoscopic ovarian drilling should not be offered for non fertility indications like amelioration of acne or hirsutism or regularization of menstrual cycle.

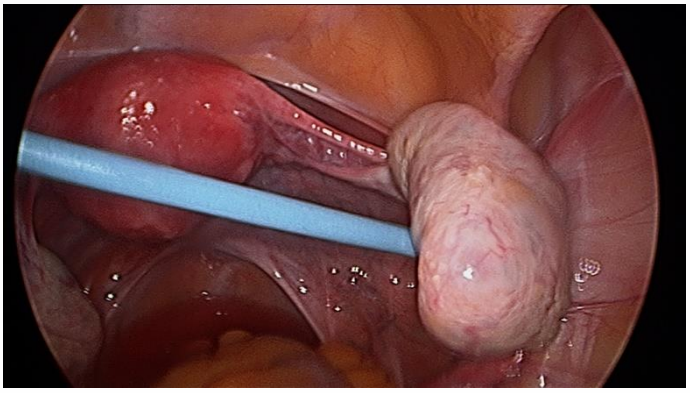
1. CC resistant patient
2. High LH with altered LH:FSH ratio
3. Married < 3years
4. BMI < 30 kg/m2
5. AMH > 8.3 ng/ml
6. Other causes of infertility should be ruled out.
7. LH > 10 IU/ml or LH /FSH ratio > 2
8. Free androgen index ≥ 4
9. Those needing laparoscopic assessment of the pelvis or who live too far away from the hospital for the intensive monitoring that is required during gonadotropin therapy.

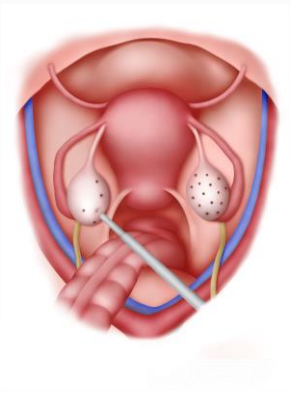
**Advantages of Laparoscopic Ovarian Drilling**

With ovarian drilling, there is decrease in pregnancy loss. In subsequent ovulation induction, ovaries become more responsive with less ovulation induction drugs or gonadotropins injections. Cohort studies report ovulatory rates of 70-90% and pregnancy rates of 40-70%. The response is influenced by the body weight. It has been found that compared to gonadotropin treatment, with laparoscopic ovarian drilling, pregnancy rates were though comparable, the abortion rate was 6-7%, with laparoscopic ovarian drilling but 26-28% with gonadotropin only treatment. The miscarriage is due to high LH levels, which is reduced with laparoscopic ovarian drilling. Even after 6-12 ovulatory cycles if pregnancy has not occurred, then it is better to offer ART.

**Techniques of Laparoscopic Ovarian Drilling**

Laparoscopy is performed via 3 portal of entry under general anaesthesia. The primary port is 10 mm or 5mm intra-umbilically and the 2 secondary ports are placed 1.5cm above the Antero Superior Iliac Spine. The entire pelvis is visualized and any pathology if present is documented. The drilling is done on the right ovary by stabilizing the right ovary with non-tooth grasper and the ovarian stroma is drilled with monopolar drilling needle. The number of punctures is empirically chosen depending on the ovarian size. The rule of 4 is employed to drill the ovary, with 4 punctures per ovary, each of 4 seconds at 40 watts, delivering 640J of energy per ovary. (Lowest effective dose recommended). The clinical response is dose dependent, with higher ovulation and pregnancies observed by increasing dose of thermal energy up to 600J/ovary, irrespective of ovarian volume. But, adjusting thermal dose based on ovarian volume (60J/cc) has better reproductive outcomes with similar post-operative adhesion rates than fixed dose of 600J/ovary. Drilling should not be done within 8-10mm of ovarian hilum. Also care is taken to stay away from the fallopian tube to prevent iatrogenic tubal block. The same is repeated on the left ovary. If unilateral drilling is decided then, better to drill the right ovary. Always thorough irrigation and suction should be done after drilling to cool the ovary. Hemostasis should be checked before completing the procedure and the other ovary also is drilled in the same manner. Pneumoperitoneum let out and port site closed.





**Mechanism of Action of Laparoscopic Ovarian Drilling**
Laparoscopic ovarian drilling causes destruction of ovarian follicles and a part of the ovarian stroma, inducing a reduction of serum androgens and inhibin levels which results in an increase of FSH and 3 restores the ovulation function. It also increases ovarian blood flow, allowing a high delivery of gonadotropins and post-surgical local growth factors like Insulin like growth factor 1 in response to thermal injury which further potentiates the action of FSH on folliculogenesis. It also improves insulin sensitivity.

**Complications of Laparoscopic Ovarian Drilling**
The two main adverse effects of laparoscopic ovarian drilling are periovarian adhesions and reduced ovarian function. The rate of periovarian adhesions varies from 19-43% and with greater damage to the ovaries the risk becomes higher. The risk of Premature Ovarian Failure (POF) is also dependent on the number of punctures made on the ovary (>4-6). Adhesions after laparoscopic ovarian drilling are mainly due to bleeding from the ovarian surface or premature contact between the ovary and the bowel after cauterization. Adhesion risk was more after Laser, probably due to lesser thermal penetration (2- 4mm) by the cone shaped lesions of laser drilling compared to cylindrical shaped lesions of 8mm with monopolar coagulation. Adhesion prevention strategies like liberal peritoneal lavage, applications of adhesion barriers like interceed and performance of Adhesiolysis at early second look laparoscopy are not effective in preventing de novo adhesions or in improving pregnancy rates. Ovary should be raised before application of energy and saline washed after the procedure to decrease the temperature thereby reducing the risk of injury. Premature ovarian failure occur if the ovarian blood supply in damaged inadvertently or if large number of punctures are made, causing excessive destruction of ovarian follicular pool or production of anti-ovarian antibodies. In fact, changes in ovarian reserve markers are interpreted as normalization of ovarian function rather than a reduction of ovarian reserve.

**Conclusion**
Laparoscopic ovarian drilling is currently recommended as a safe, efficacy and cost effective alternative to gonadotropin for anovulatory CC resistant infertile women. The main advantage of laparoscopic ovarian drilling is extended period of efficacy that lead to more than one pregnancy in certain cases and less incidence of multiple pregnancy and OHSS. However, normal body mass index, and less than 3 years of infertility and AFC less than 50 and age less than 35 years seems to produce a good outcome.

ARTICLE 8

# PCOS & MENOPAUSE

**Dr. T. Ramani Devi,**
**MD., DGO., FICS., FICOG.,**
Vice President FOGSI 2020
Director Ramakrishna Medical Centre LLP
Director Janani Fertility Centre, Trichy

Polycystic Ovarian Syndrome (PCOS) is the most common endocrine disorder of reproductive age group women. Prevalence ranges between 4 – 21%. Any 2 out of 3 of the Rotterdam criteria is enough to say that the patient has PCOS. There are different phenotypic variations in PCOS. PCOS is a life-long condition that is associated with metabolic and non-metabolic health issues which extends beyond the reproductive age group. Incidence of anovulatory infertility is quite high in PCOS. Beyond menopause following health issues can happen, like obesity, glucose intolerance, diabetic mellitus, hypertension, dyslipidemia, metabolic syndrome, obstructive sleep apnoea, endometrial cancer, depression and anxiety. With aging and nearing menopause, cycles get regularized because of decreasing ovarian volume, follicular number and serum androgen level. We do not know whether this improvement of menstrual cycle is associated with favourable long term health issues.

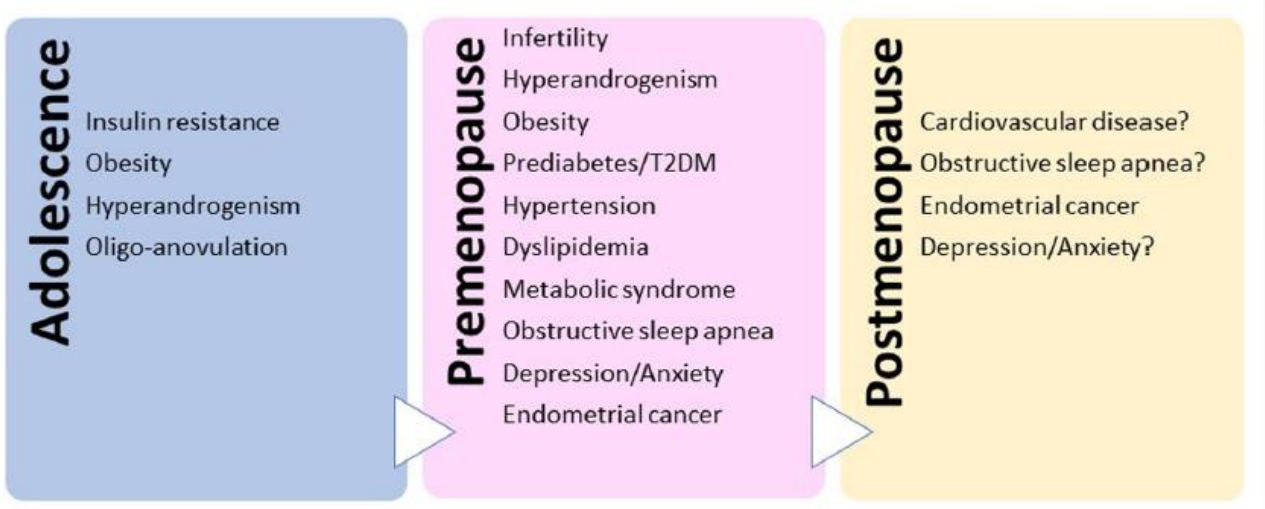



Fig. 1. PCOS and health outcomes across the life course. T2DM, type 2 diabetes mellitus.

**Obesity**
Obesity is of visceral origin which affects both reproductive and cardio metabolic issues. Increase in BMI and waist circumference which is seen during post menopause due to changes in hormonal mileu is aggravated in PCOS. Rotterdam study also proved the same.

**Glucose intolerance**
Women with PCOS had 3 times higher risk of developing Type 2 diabetes. Menopause is associated with increased incidence of insulin resistance and glucose intolerance. Few studies show, that prevalence of Type 2 diabetes reduces in post-menopausal women with PCOS. Though Rotterdam study showed higher incidence of Type 2 diabetes mellitus in post-menopausal women, it was not adjusted for BMI and WHR. As on date, available data are limited.

**Hypertension**
Recent meta-analysis of 30 studies showed higher prevalence of hypertension in PCOS patients. As per the available data the increased risk of hypertension during reproductive age group settles down with aging and becomes similar to healthy women after menopause. According to Rotterdam study dyslipidemia was higher is post-menopausal women with PCOS but we need more data.

**Metabolic syndrome**
Insulin resistance plays a major role in metabolic syndrome which include abdominal obesity, hypertension, low HDL – C, elevated triglycerides and hyper-glycemia. Menopause increases the incidence of metabolic syndrome. We have only limited data to show that metabolic syndrome is increased in post-menopausal women with PCOS.

**Cardio vascular disease**
Recent meta-analysis shows higher risk of developing cardio vascular disease among reproductive age patients with PCOS, whereas it was not statistically different in menopausal women with PCOS when compared to healthy controls. Data from recent studies show PCOS may not confer additional risk of cardio vascular disease in aging women.

**Obstructive Sleep Apnoea (OSA)**
There is increased risk of obstructive sleep apnoea in PCOS women of reproductive age group. Increasing age and obesity are associated with higher risk of OSA in women with PCOS during the post-menopausal period. Insulin resistance, hyper-androgenemia and low levels of estrogen and progesterone among PCOS patients play a role in development of OSA.

**Cancer**
Unopposed estrogenic action in women with PCOS increases the risk of oestrogen dependent tumours like endometrial, ovarian and breast cancer. Therefore, we need to be more vigilant among women with PCOS marching towards menopause. Obesity and type 2 diabetes increases the risk of endometrial carcinoma by 4-fold. Similarly, the risk of malignancy may be attenuated following menopause. There is no significant elevation of cancer breast and ovary in women with PCOS as per recent Danish Cohort study. There is increased risk of depression and anxiety among PCOS patients due to obesity, infertility and hyperandrogenism. This vulnerability persists during the menopausal period. However, no clear cut data are available.

**Conclusion**
PCOS is a life-long endocrine metabolic and reproductive disorder affecting many women. Current evidence says women of PCOS in reproductive age group have higher risk of developing cardio vascular disease, endometrial cancer and OSA. These cardio metabolic health issue does not appear to deteriorate after menopause, but there is higher risk of obstructive sleep apnoea, anxiety and depression. So we need to have long term cohort study for evaluation of complications in the post-menopausal women.

# UPCOMING EVENT
# TNFOG MID-YEAR E CONFERENCE

**19th & 20h June, 2021**

## INTERNATIONAL FACULTY

**KEYNOTE SPEAKER**
**Prof. Sir. Sabaratnam Arulkumaran**
Topic - 'Intrapartum Care bundle to improve clinical outcome'

**KEYNOTE SPEAKER**
**Dr. Maruf Siddique**
Topic - Selection of cases for IUI & Stimulation protocols

## TNFOG ORATION

**Topic - 'Obstet-Tricks" then to now**

20 June, Sunday
(11.00 am to 12.00 noon)

**Dr. Kurian Joseph**

## NATIONAL FACULTY KEYNOTE SPEAKERS

Dr. Hrishikesh D.Pai

Dr. Hema Divakar

Dr. Kamini A Rao

Mid-year E Conference

# TAMILNADU FEDERATION OF OBSTETRICIANS AND GYNAECOLOGIST - TNFOG

**19th & 20h June, 2021**

## HIGHLIGHTS

- 3 Keynote Addresses
- Panels
- Lectures
- TNFOG Oration
- Debates
- Free Papers & Posters
- Quiz on Labour Management

## KEY NOTE ADDRESSES

**Sir Sabarathnam Arulkumaran**

Intrapartum Care Bundle to improve clinical outcome

19th June
Saturday
(5 pm to 5.30 pm)

**Dr. Hema Divakar**

Adolescent PCOS

20th June
Sunday
(1.30 pm to 2.00 pm)

**Dr. Hrishikesh D.Pai**

ART - Present, Past & Future

20th June
Sunday
(2 pm to 2.30 pm)

# Mid-year E Conference

# TAMILNADU FEDERATION OF OBSTETRICIANS AND GYNAECOLOGIST - TNFOG

**19th & 20h June, 2021**

# Workshops
# 19th June, Saturday


## Mid-year E Conference

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## ONLINE QUIZ ON LABOUR MANAGEMENT

- Final Quiz on 20th June Sunday from 03:45 - 04:45 pm
- Preliminary qualifying rounds on 13th June Sunday.

**Interested & Enthusiastic Postgraduates of Medical Colleges & DNB Institutes can enroll for the Quiz.**

**To Enroll furnishing your Name, Institution & Mobile number (Through Whatsapp)**

Kindly attach a Copy of ID Card issued by your Institution.

## MORE DETAILS CAN BE GOT FROM THE QUIZ MASTERS


**Dr T V Chitra**
**Mobile: 9443353191 E mail: chitrbhat63@gmail.com**

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Mid-year E Conference

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# E PAPER & E POSTER PRESENTATION

## On any topic in Obstetricians & Gynaecologists
**18th June, Friday**

## Last Date for Abstract Submission
**16th June, Wednesday**

**ABSTRACT**

## Guidelines - Free paper / Poster

- Abstract should not exceed 300 words.
- Abstract must contain Objectives, Methodology, Results & Conclusion.
- For case reports - Introduction, Case presentation, Discussion & Conclusion.
- Registration for conference is a prerequisite for presentation.
- Authors are requested to adhere to the time limit.
- Last date for submission of abstract is 16th June, 2021.
- A copy of abstract must be sent by e-mail to tnfogoffice@gmail.com
- The organising committee reserves the right for accepting / rejecting any paper / poster without assigning any reason.
- The organising committee of "TNFOG" reserves the right to publish all papers presented at the conference.

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Mid-year E Conference

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