

THE PCOS SOCIETY NEWSLETTER

# PANDORA

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Registered Address  
Kwality House, 1<sup>st</sup> Floor,  
August Kranti Marg, Kemp's Corner,  
Mumbai 400 026

Phone: 022 23802584, 022 23803965  
Fax: 022 23804839  
Email: [thepcossociety@gmail.com](mailto:thepcossociety@gmail.com)

## Executive Committee

**Dr. Duru Shah**, Founder President  
durushah@gmail.com

**Dr. Shashank Joshi**, Vice President  
shashank.sr@gmail.com

**Dr. Rekha Sheth**, Vice President  
docrsheth@gmail.com

**Dr. Piya Thakkar**, Honorary Secretary  
piyaballani@hotmail.com

**Dr. Sangeeta Agarwal**, Joint Honorary Secretary  
sangeetaagrawal@yahoo.com

**Dr. Uday Thanawala**, Honorary Treasurer  
udaythanawala@gmail.com

**Dr. Madhuri Patil**, Scientific Coordinator  
drmadhuripatil59@gmail.com

## Constitution Committee

**Krishnendu Gupta**, Chair  
krisim007@gmail.com

**Shashank Joshi**, Chair  
shashank.sr@gmail.com

## Newsletter Committee

**Anita Soni**, Chair  
anita.soni8@hotmail.com

**Toral Shinde**, Co-Chair  
toral.nilesh@gmail.com

## Research Committee

**Padma Rekha Jirge**, Chair  
rekha.jirge@gmail.com

**Ganpat Sawant**, Co-Chair  
dr.ganpatsawant@gmail.com

## CME Committee

**Sujata Kar**, Chair  
suju2463@gmail.com

**Kanthi Bansal**, Co-Chair  
kanthibansal@gmail.com

## Website Committee

**Nandini Rambabu**, Chair  
atg2016nandini@gmail.com

## Public Awareness Committee

**Nalini Mahajan**, Chair  
dr.nalinimahajan@gmail.com

**Sanjeev Khurd**, Co-Chair  
sanjeevkhurd@yahoo.com

**Sudha Tandon**, Co-Chair  
sudhantandon@gmail.com

**Sharda Maroju**, Co-Chair  
jgdsharda@gmail.com

**Gautam Khastgir**, Co-Chair  
birthindia@gmail.com

## Membership Committee

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ritujoshi01@rediffmail.com

## International Committee

**Shanti Shrinivasan**, Member  
drshantimani@yahoo.com

## Social Media Committee

**Bina Vasan**, Chair  
binavasan@gmail.com

**Altamash Shaikh**, Co-Chair  
ealtamash@gmail.com

# Welcoming....

## Our New Patrons



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**Dr. Padma Rekha Jirge**



**Dr. Sanjay Devidas Gandhi**



**Dr. Sonia Malik**



**Dr. Toral Shinde**



**Dr. Usha Mohan**

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Dr. Asha R. Paranjpe

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Dr. Suneera Jain

Dr. Uma Anil Khatri

Dr. Yashodhara Mhatre

Dr. Zareena A. Khalid

## Editorial

### Editorial Team



**Dr. Duru Shah**  
MD, FRCOG, FCPS, FICS, FICOG, FICMCH, DGO, DFP  
Director, Gynaecworld, The Center for Women's Fertility & Health, Mumbai  
**President, The PCOS Society (India)**  
**Chief Editor**



**Dr. Sabahat Rasool**  
MD, DNB, MNAMS, FMAS, MRCOG (UK)  
Ivan Donald Diplomate in OBGY Ultrasound, Croatia  
Fertility Consultant, Gynaecworld, Mumbai  
**Associate Editor**



**Dr. Anita Soni**  
MD, DNB, FCPS, DGO  
Consultant OBGYN, Hiranandani Hospital, Powai  
**Chair, The Newsletter Committee**



**Dr. Toral Shinde**  
DGO, Fellowship in ART  
**Co-Chair, The Newsletter Committee**



**Ms. Rochelle Lobo**  
**Administrative Assistant**

After being in existence for just over 2 years, the PCOS Society is still surviving! I was told that such a Society has no future, but I think the Society is doing great! The first year when we launched it, there were many formalities to complete, such as registering the society as a public trust, creating the logo, creating the website, starting a Bank Account and reaching out to all. In the second year the Annual Conference was a huge success and brought in many new members! And as the Society is growing, we have in place today an office secretariat, an Auditor, an accountant and various Committees of the Society, besides the Executive Committee in place We have published **2 Newsletters** and this is the third one reaching you. If you wish, please browse through the previous two issues on the following link: <http://www.pcosindia.org/newsletter.php>.

As a Society, we have initiated "**One Day Continuing Medical Education Programs**" with, the first one being held in October 2016, in Ahmedabad, which was extremely well organized by our member **Dr. Kanthi Bansal** (details on page 4). The next meeting is scheduled on 11<sup>th</sup> December 2016, organized by **Dr. Talukdar in Guwahati** (details on page 4). There are many forthcoming events and I urge all of you to participate in these very interesting meetings, which are multidisciplinary and extremely informative, with experts from the other disciplines of medicine and surgery addressing us. Such sessions become extremely enlightening and interesting because they are about the areas of medicine which we normally don't delve into!

The next **Annual Conference of the PCOS Society** of India will be held in **Bengaluru between 16<sup>th</sup> to 18<sup>th</sup>, June 2017**, organized by **Dr. Madhuri Patil**, one of our most well read experts in the field of Assisted Reproduction. Please do visit the Events page (page no. 4) to get details of all forthcoming events!

I am delighted to let you know that the PCOS Society has become a member of the **Federation of International Societies of Gynecologist Endocrinology (FISGE)** which conducts excellent conferences on Gynecological Endocrinology and Infertility every 2 years. The next meeting is between 7-10 March 2018, in Florence, where **the PCOS Society will have its own session**.

Various Committees have been formed and some have initiated work, whilst others have to still start! The various Committees have been listed on page 2, if any of you is enthusiastic and would like to participate in any of these Committees, please do write to the Chair of that Committee with a cc marked to me at [durushah@gmail.com](mailto:durushah@gmail.com) and to the secretariat at [thepcossociety@gmail.com](mailto:thepcossociety@gmail.com). We are looking for active interested volunteers who will make a difference to our Society, which is in its infancy and needs to be nurtured!

Many programs have been planned for the next financial year, do become our members, get involved and let's try to unravel the mystery of PCOS!

I am truly thankful to all those who have made this Newsletter possible – the New Chair and Co-chair of the Newsletter Committee, **Dr. Anita Soni, Dr. Sabahat Rasool**, who has put together the entire issue, Rochelle, who has coordinated between all, Alaka from Mohor, our designer, whom we harass quite a bit with all corrections and last but not the least, Ms. Farida Hussain and her team from USV who have given tremendous financial and logistic support to our 3 Newsletters, which we have published this year. Wishing you all a Merry Christmas and a Very Happy New Year!

**Dr. Duru Shah**

Founder President, The PCOS Society, India  
Chief Editor

Email: [thepcossociety@gmail.com](mailto:thepcossociety@gmail.com)

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# Events & Update

Focused One Day Conference, Hyatt Regency, Ahmedabad

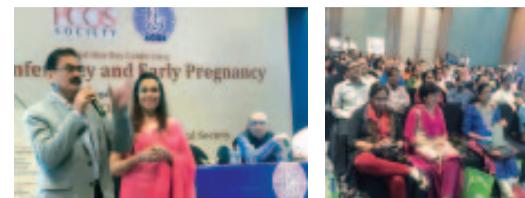
## PCOS – Infertility and Early Pregnancy

One day focused conference on "PCOS – Infertility and Early Pregnancy" was organized by The PCOS Society in collaboration with the Ahmedabad Obstetrics & Gynecological Society (AOGS) on Sunday 2<sup>nd</sup> October 2016 on the auspicious day of Gandhi Jayanti at Hyatt Regency, Ahmedabad, the chief convenor being Dr. Kanthi Bansal.

The topics were focused on the very complicated and vast subject of PCOS, as there is a huge increase in the incidence of PCOS, the impact of PCOS on infertility and early pregnancy, including diagnosis, management, and complications and giving the right guidance for patients.

The topics covered included latest views on diagnosis, clearing concepts in the complicated subject of insulin resistance, evidence based management of ovulation induction & fertility management, treating lean & obese PCOS patients, life style management & a panel on managing difficult cases.

The Conference was structured in four sessions which covered Overview, Evidence



Based Strategy for Ovulation Induction in PCOS, and Treating Infertility and Early Pregnancy in PCOS.

The lecture sessions were followed by a panel discussion on 'Difficult Cases Made Easy', moderated by Dr Prakash Bhatt & Dr. Kanthi Bansal.

The conference could not have been so unique without the support of Dr. Duru Shah, Founder President of The PCOS Society, Dr. Madhuri Patil, Scientific Coordinator, Dr. Geetendra Sharma, President, AOGS & Dr. Rajal Thaker Hon. Secretary, AOGS.

– **Dr. Kanthi Bansal**, Convenor of the Conference

# Coming Up



## Day 1 – 16<sup>th</sup> June 2017

### Workshop 1

#### Session 1: Ultrasound in PCOS

- Diagnosis of PCOS by USG – What are new criteria?
- Is ultrasound a good diagnostic method in adolescent PCOS?
- Should Serum AMH replace ultrasound PCO morphology as a diagnostic marker?
- Basic assessment of blood flow to the ovaries in PCOS. Does 3D power Doppler have any role?

#### Session 2: Monitoring a fertility cycle

- Ultrasound only protocols in the management of ovarian stimulation with gonadotropins in PCOS
- How important is it to monitor the endometrium during ovarian stimulation in PCOS
- Ultrasound assessment of ovarian hyperstimulation syndrome
- Monitoring post IVF in an PCOS women – What to look for

### Workshop 2: Lifestyle Modification in PCOS

#### Session 1

- Ethnic differences in obesity
- Role of Metformin and Inositol in weight reduction
- Does Alcohol and smoking have effect on the endocrine system in PCOS?

#### Session 2

- How lifestyle changes help in PCOS?

- Nutrition in PCOS and role of anti-inflammatory diet
- Role of exercise in PCOS (with demonstration)
- Benefits of yoga in PCOS (with demonstration of postures, which benefit PCOS)

## Day 2 – 17<sup>th</sup> June 2017

### Session 1

- Basics we need to Know
- Genetic aspect of prevalence and phenotype of PCOS
- Adipose tissue dysfunction in PCOS
- AMH in PCOS
- Role for measuring androgen and insulin levels in women with PCOS

### Session 2

- PCOS a spectrum of disorders
- Fetal and childhood origin of PCOS
- Challenges and controversies in treating adolescent PCOS
- Anxiety and mood disorders in PCOS

### Keynote Address

- PCOS – Time to move on
- Prevention the global epidemic of PCOS – Is it possible?

### Session 3

- Hirsutism and hyperandrogenism
- Quality of life in women with Hirsutism
- Treatment options for women with Acne and Hirsutism
- Does cosmetic Surgery have a role?

### Session 4

- Obesity and PCOS
- Obesity medications – How to select the right drug
- Bariatric surgery in PCOS – How effective is it?
- Sleep Apnea – How serious can it be?

### Session 5

- The Great Controversies

### Debate 1 – Should Metformin be continued in pregnancy (1<sup>st</sup>, 2<sup>nd</sup> and 3<sup>rd</sup> trimester?)

- Yes ■ NO ■ Remarks from Experts

### Debate 2 – PCOS is associated with higher pregnancy loss

- Yes ■ NO ■ Remarks from Experts

### Stump the experts – Interesting cases

- Menstrual dysfunction in PCOS women
- Insulin resistance and hyperandrogenemia

### General Body Meeting

- Poster Session

## Day 3 – 18<sup>th</sup> June 2017

### Session 6

- PCOS and Infertility
- Ovulation induction in non-ART cycles
- OI in patients undergoing ART – an evolving strategy
- Hormone dynamics following laparoscopic drilling

### Session 7

- PCOS and ART
- Does PCOS compromise the oocyte and embryo quality or the endometrium?
- Freeze all should be a strategy in all PCOS women undergoing IVF
- Does PGS followed by eSET have a role in women with PCOS

### Session 8

- PCOS and Pregnancy
- PCOS as a forerunner of Gestational Diabetes
- Ethnic differences in glucose measurement and control
- Obstetric and neonatal outcome in PCOS with GDM

### Session 9

- Long-term effect of PCOS
- PCOS and Cancer Risk
- Treatment of atypical endometrial hyperplasia
- Role of fatty liver in PCOS – NAFLD

### Valedictory

**One Day Conference in Guwahati**  
**December 11, 2017**  
**'PCOS Science to Practice'**  
 Organized by **Dr. R. Talukdar**  
**rktalukdar@gmail.com**

**2-Day conference in Dubai**  
**March 17-18, 2017**  
 organized by The PCOS Society  
 Scientific Programme to follow

# Obesity and Hormones in PCOS



**Dr. Anurag Lila**

Associate professor, Department of Endocrinology, Seth GSMC & KEM Hospital, Mumbai

## Introduction

Polycystic ovary syndrome (PCOS), a complex disorder with reproductive and metabolic implications, is one of the commonest endocrinopathies affecting approximately every fifth Indian woman of reproductive age group<sup>1</sup>. Its cardinal features oligo-anovulation, hyperandrogenism / hyperandrogenemia, and polycystic ovarian morphology (PCO), have been used in varying combinations over time to define diagnostic criterias. However exclusion of other masquerading endocrinopathies remains the backbone for establishing a diagnosis of PCOS.

Although many aspects of its patho-physiology remain to be elucidated, PCOS is known to involve a complex interplay of genetic and environmental factors. Obesity and insulin resistance are closely linked with PCOS. While weight gain definitely alters phenotypic expression of PCOS, it is debatable whether obesity per se can cause PCOS or vice-versa. In this article, underlying role of hormones in the patho-physiology, diagnosis, and management of both reproductive and metabolic aspects of PCOS are discussed.

## Pathophysiology of hormonal imbalance

Intrinsic steroidogenic dysfunction of theca cells leading to functional ovarian hyperandrogenism is hypothesized as a central factor in pathogenesis of PCOS<sup>2</sup>. At the level of ovary, hyperandrogenism stimulates the initiation of primordial follicles and increases the number of small antral follicles during early gonadotropin-independent stage. At the level of hypothalamus, hyperandrogenism dampens the negative feedback regulation of ovarian steroids on gonadotropin releasing hormone pulse generator, leading to abnormal gonadotropin secretion, in the form of relatively high LH and low FSH levels. LH excess, in turn, appears to amplify androgen production by the theca cells, while lower FSH levels inhibit further follicular growth<sup>3</sup>.

Additionally, insulin resistance influences the deranged endocrine milieu at multiple levels. Compensatory hyperinsulinism resulting from obesity-related and/or intrinsic insulin resistance, adds to ovarian as well as adrenal androgen secretions. Insulin functions as a co-gonadotropin through its cognate receptor to modulate ovarian steroidogenesis<sup>2</sup>. Metabolic derangements associated with insulin resistance are found to be associated more with a severe PCOS phenotype, i.e. one having a combination of hyperandrogenism and oligo-anovulation.



Obesity per se, is associated with suppressed levels of SHBG, leading to higher free androgen levels which prolong follicular phases without affecting ovulation, thus leading to longer menstrual cycles. Unless known, these features could mislead to a PCOS diagnosis.

## Diagnosis and Hormonal Investigations

### Hyperandrogenemia

Testosterone and androstenedione are the markers of ovarian hyperandrogenism, whereas dihydroepiandrosterone sulfate (DHEAS) represents adrenal androgen source. In clinical practice serum total testosterone (done in follicular phase of cycle) is commonly used. It should be noted that the routinely used total testosterone assays are relatively inaccurate at the lower levels (as observed in women)<sup>3</sup>. Hence, in this clinical scenario, serum total testosterone levels exhibit a significant overlap between normal women and those with PCOS. Serum free testosterone is the single most predictive test for hyperandrogenemia. It should be calculated using measurements of total testosterone and sex hormone-binding globulin. Routinely available free testosterone assays are inaccurate, hence measured free testosterone values should be interpreted cautiously. Dihydroepiandrosterone sulfate, androstenedione and other steroidogenic metabolites are currently used in research settings, and their utility in clinical practice is limited.

Before ascribing it to PCOS, it is important that clinicians should rule out other causes of hyperandrogenism, clinically important being non-classic congenital adrenal hyperplasia (NCCAH), Cushing's syndrome and androgen-producing tumors. Early follicular-phase, morning 17-hydroxyprogesterone levels < 200 ng/dL effectively rules out 21-hydroxylase deficiency, which is the most common cause of NCCAH<sup>4</sup>. Markedly elevated total

testosterone level >150 ng/dL are rarely encountered, but in such a case, possibility of adrenal or ovarian androgen secreting tumor should be considered<sup>3</sup>. Exclusion of Cushing's syndrome should be guided by clinical suspicion, and when needed, overnight dexamethasone suppression test with its good sensitivity and specificity should be the preferred test.

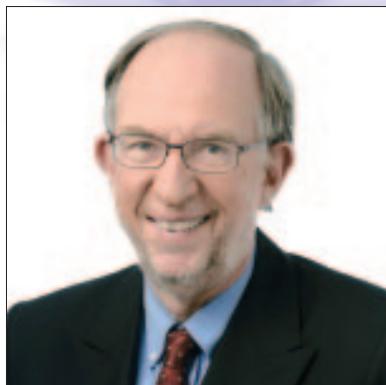
### Oligo / anovulation

Oligo-anovulation is recognized best by a detailed menstrual history. However, serum progesterone level (presumed mid-luteal phase of the cycle) lower than 3- 4 ng/mL, is highly suggestive of oligo/anovulatory cycles. Elevated serum AMH levels frequently found in women with PCOS are related to increased follicle numbers and granulosa cell hypersecretion.

Clinicians should diligently rule out other causes of ovulatory dysfunction before ascribing it to PCOS, clinically important causes being thyroid dysfunction, hyperprolactinemia, ovarian failure, and hypogonadotropic hypogonadism (e.g. functional hypothalamic amenorrhea)<sup>4</sup>. Thyroid function tests should be interpreted with caution in women taking oral contraceptive pills as they may cause a high total T4 and T3 values, due to elevation of thyroid binding globulins. Sample for serum prolactin estimation can be drawn at any time of the day, but excessive venipuncture stress should be avoided. A single (as against pooled) sample determination is usually sufficient to establish the diagnosis of hyperprolactinemia, but when in doubt, multiple samples can be repeated on a different day at 15- to 20-min intervals to account for possible prolactin pulsatility.<sup>5</sup> PCOS being a hyperestrogenic state can cause mild hyperprolactinemia. Patients with ovarian failure will have persistent elevations of LH and FSH levels, whereas those with hypogonadotropic hypogonadism exhibit inappropriately low LH and FSH with respect to estradiol.

*Continued on page 09*

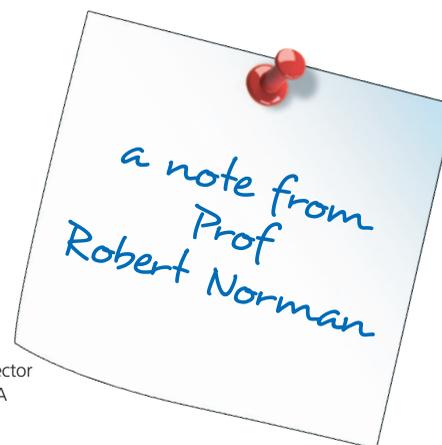
# International Guidelines for Polycystic Ovary Syndrome



## Professor Robert J. Norman

AO, BSc (Hons), MBChB(Hons), MD, FRANZCOG, FRCPA, FRCPath, FRCOG, CREI, FAHMS

- Professor of Reproductive and Periconceptual Medicine
- Robinson Research Institute, The University of Adelaide Medical Director
- Sub specialist in reproductive endocrinology and infertility Fertility SA



Two decades ago, different countries had different definitions for PCOS and there was very little discussion among nations about the best management, diagnosis and investigation of PCOS. There was a lot of division of opinion between Europe and North America.

This all started to change with the Rotterdam Consensus, in which people from different countries came together in The Netherlands to come up with a more universal definition of what encompasses PCOS. Twenty key experts from North America, Europe, Australia and Asia determined what we now know as ASRM ESHRE definition, or more briefly, the Rotterdam Criteria. Most countries in the World now accept this definition and it has been endorsed by the NIH in the USA. The major addition was that of ultrasound, which now is universal, as the machinery becomes more sophisticated and experience from operators is more extensive.

However, there are still many issues that need to be resolved and, in Australia, we developed the Australian PCOS Alliance to develop guidelines for PCOS in our country. We accepted the Rotterdam Criteria as the definition and came up with guidelines which were published based on the strongest possible evidence, done in the most expert fashion available. This was endorsed by the National Health and Medical Research Council of Australia, the prime research body in the country. Materials for translation were given to every doctor in the country, systematic reviews were published and the PCOS community engaged to get information across to everyone with PCOS. We think that there has been an improvement in diagnosis and that more patients are being identified and having investigations much earlier.

As a result of these national guidelines we were granted a National Centre of Research Excellence in

PCOS for five years, and this has been headed up by Professor Helena Teede from Melbourne, and myself.

We have ten Chief Investigators and a large array of Assistant Investigators, many of them being young researchers and clinicians. We have several aims:

**1.** Improving the diagnostic criteria. Although we all agree on anovulatory cycles, increased testosterone and polycystic ovaries on ultrasound, the criteria for this are changing and controversial. We are seeking to discover what does constitute an abnormal menstrual cycle, to work out what is the best way to measure testosterone, and also to come up with criteria for good ultrasound which is very patchy across the country. We are also investigating the role of AMH in substituting for ultrasound.

**2.** Life course of PCOS. We have several cohorts where we are able to follow patients with PCOS to see what happens to them, and particularly in terms of their metabolic and emotional outcomes. We are hoping that this will give us a much better idea as to which patients are at risk of future disease.

**3.** International Guidelines for PCOS. The Australian group, together with ESHRE and ASRM, have put in a substantial amount of money to develop international guidelines for PCOS and we have endorsement from FIGO, ASPIRE, IFFS and the WHO to develop the database and systematic reviews that will inform evidence-based guidelines. We are hoping that we will complete this in under eighteen months, and that every country will then be able to use the data to come up with guidelines that are specific for their country. Almost every significant researcher and key opinion leader in PCOS is represented on one of the many committees. The International Committee is headed by myself and Bart Fauser and your Indian representative is Dr. Duru Shah, Dr. Jaideep Malhotra is also on one of the committees.

The Guideline Development Groups will follow clear principals on grading the evidence and will be supported by systematic reviews produced by experts in the field who are all experienced with the Cochrane and other evidence-based guideline methodologies. Once this evidence is available, recommendations will be put forward to the International bodies that are participating, and each country can then make up its mind about what recommendations to make in their health system. For instance, in some countries metformin may not be allowed and if the guidelines suggests that meformin is suitable for treatment for PCOS, the country can ignore that. In other countries Letrozole is not permitted, and for others, the cost of laparoscopic diathermy may be prohibitive. The aim is to have a central evidence base which is upgraded regularly, which allows each national body to access and make recommendations to their health service.

The concept of evidence-based guidelines is common in many branches of medicine, but has not been well applied in aspects of gynaecology. Our obstetric colleagues are much better at it than gynaecologists and I hope that the PCOS community will lead the way.

I would encourage Indian delegates to consider coming to the Androgen Excess PCOS meeting in Lorne, Melbourne, or alternatively to come to the Masterclass in PCOS in Hanoi where you will be able to interface very closely with some of the world experts in this area.

The time has come for us to practice medicine that is based on evidence, rather than eminence and given the outstanding experience and large number of patients seen by Indian specialists in PCOS, we are looking forward to your contributions to the guidelines, to the Androgen Excess PCOS meeting and to the Masterclass in Hanoi.

# Polycystic Ovaries – Beyond Menopause Climacteric. 2014 Apr, 17(2):109-15.

**Dr. Duru Shah**

MD, FRCOG, FCPS, FICS, FICOG, DGO, DFP, FICMCH

**Dr. Shefali Bansal**

MD

## Introduction

PCOS is the most common endocrine disorder in women, affecting upto 10% of women in the reproductive age group. It is characterised by chronic oligo or anovulation, hyperandrogenism, hyperinsulinemia, dyslipidemia, and metabolic syndrome. With various studies on PCOS, many different phenotypes of PCOS are identified apart from the classical phenotype.

## Pathophysiology of PCOS

The basic pathophysiologic defect in PCOS is unknown. Studies have shown that familial and genetic factors may cause predisposition to PCOS. Hyperinsulinemia causes or exacerbates hyperandrogenemia. Increased insulin levels lead to increased androgen production from the ovarian theca cells. By suppressing hepatic production of sex hormone binding globulin (SHBG), insulin also increases unbound levels of testosterone. Conversely, androgens may indirectly contribute to insulin resistance through facilitating free fatty acid release from abdominal fat. Insulin resistance in PCOS is also associated with abdominal obesity, elevated free fatty acid levels, increased androgens and anovulation.

The clinical expression of PCOS is varied and includes oligo – ovulation or anovulation, hyperandrogenism (clinical or biochemical) and polycystic ovaries.

## Insulin resistance

Insulin resistance is a prominent feature of PCOS and occurs in approximately 60-80% of women with PCOS and in 95% of obese women with PCOS. The impaired glucose tolerance is seen more often in PCOS women with a classic phenotype and is a predictor of Type 2 diabetes mellitus later in life and increased risk of GDM. Presence of obesity and family history of type 2 diabetes can further contribute to the risk. However, a systematic review and meta analysis by Moran et al concluded that PCOS women have an elevated prevalence of impaired glucose tolerance, type 2 diabetes mellitus and metabolic syndrome in both BMI matched as well as non BMI matched studies.

## Obesity

Obesity is present in 30-70% of PCOS women. These women are more likely to have abdominal or visceral adiposity which is associated with greater insulin resistance. This may exacerbate the reproductive and metabolic abnormalities in PCOS. However such a risk may vary in different patients exhibiting different phenotypes.

## Dyslipidemia

Women with PCOS have deranged lipid profile with higher triglyceride levels, low density lipoprotein (LDL), non-high density lipoprotein (HDL) cholesterol and lower HDL compared with women who do not have PCOS. A systematic review by Wild et al concluded that women with PCOS have higher LDL cholesterol, and non HDL cholesterol regardless of BMI. Altered levels of triglycerides, HDL, LDL, and non-HDL (reflecting altered ApoB/ApoA metabolism)

are prevalent in women with PCOS and are more severe in hyperandrogenic women .

## Metabolic syndrome and cardiovascular risk in PCOS

Metabolic syndrome is defined as elevated blood pressure (BP $\geq$  130/85 mm Hg), increased waist circumference ( $>88$  cm in non Asian,  $>80$  cm in Asian and East/South Asian women), elevated fasting glucose ( $\geq 100$ mg/dl), reduced HDL-C ( $<50$  mg/dl in women) and elevated triglyceride levels ( $>150$ mg/dl) levels. Studies have shown increased cardiovascular and metabolic risk in hyperandrogenic women with classic features of PCOS. A long term follow up study by Muriam H et al on prevalence of metabolic syndrome in women with previous diagnosis of PCOS found an increased prevalence of metabolic syndrome in the PCOS group (23%) compared with the control group (8%). Presence of abdominal obesity increases the prevalence of metabolic syndrome in PCOS women. Dyslipidemia, IGT, and type 2 diabetes mellitus are classic risk indicators of atherosclerosis and CVD, and are more prevalent in women with PCOS, even when weight matched with normal control women. The metabolic syndrome is associated increased risk of cardiovascular disease. PCOS women with obesity, cigarette smoking, dyslipidemia, hypertension, impaired glucose tolerance are at risk, whereas those with metabolic syndrome and/or type 2 diabetes mellitus are at higher risk of cardiovascular CVD. Insulin-resistant women with PCOS also have vascular dysfunction, which is associated with total and abdominal adiposity. Various biochemical inflammatory and thrombotic markers of CVD risk are seen in women with PCOS. A systematic review and meta analysis by De Groot PC et al concluded that there is a 2 fold risk of arterial disease (coronary heart disease and stroke) in patients with PCOS than without PCOS. The risk of atherosclerosis is associated with obesity and insulin resistance, which are related to PCOS. However, a study by Soares GM, et al showed that young women with PCOS have changes in vascular elasticity even in the absence of classical risk factors for CVD, such as hypertension and obesity.

## Pathophysiology of Menopause – Weight gain and Metabolic changes at Menopause

Since menopause occurs along with ageing, occurrence of metabolic disorders may be influenced by chronological ageing as well as changes due to menopausal transition. Menopausal transition itself is associated with several hormonal and metabolic changes.

## Weight gain

Weight gain is an important finding in women after menopause. A substantial increase in waist circumference and fat mass has been shown in longitudinal data from postmenopausal women. The changes during menopause are associated with an increase in total body fat and an increase in abdominal fat. Studies have tried to determine whether weight gain in mid life is due to age or due to hormonal changes that occur in menopause. Many studies have now shown that this weight gain may be due to age, rather than menopause itself. However the increased fat distribution in the

abdomen leading to abdominal obesity, has been attributed to the hormonal changes across the perimenopause.

## Metabolic syndrome

A review of metabolic syndrome after menopause concluded that there is an increased prevalence of Metabolic syndrome in postmenopausal women which is driven by weight gain and obesity after menopause. The metabolic role of Leptin has been well established in many studies. Hyperleptinemia has been related to increased body weight and development of metabolic syndrome. Similarly, a positive correlation between resistin and metabolic syndrome has been demonstrated in many studies. On the other hand, adiponectin has been positively correlated with plasma HDL cholesterol and has inverse relation with triglyceride levels. Post-menopausal obese women with metabolic syndrome have a significant increase in leptin levels. A systematic review done by Touli et al to compare adiponectin levels in women with PCOS with healthy controls revealed that adiponectin levels are lower in women with PCOS compared with non-PCOS controls. The authors also suggested that the lower adiponectin levels may be related to insulin resistance. Similarly a study done to evaluate the role of adiponectin and resistin in PCOS women showed that the serum adiponectin levels and the ratio of resistin to adiponectin levels are reduced in obese women with PCOS. Proatherogenic changes in lipid and apolipoprotein profiles seem to be specifically related to ovarian ageing. Mathews et al did a prospective study to examine the changes in lipid profile within one year of the final menstrual period(FMP). They found a substantial increase in total cholesterol, LDL cholesterol and Apo B within one year interval before and after the FMP. Hence, the authors concluded that women experience a unique increase in lipids at the time of FMP. Menopausal transition is also associated with a rise in blood pressure. Blood pressure is typically lower in premenopausal women than in men. However, after menopause the prevalence of hypertension in women is higher than in men. It has been difficult to define whether it is a direct effect of menopause or result of co existence of other risk factors like ageing and obesity. The SWAN study followed women longitudinally over 9 years and showed that both diastolic and systolic BP rise in a linear fashion along with advancing age, irrespective of date of FMP (final menstrual period). A longitudinal study on 150 middle aged women showed that longitudinal changes in diastolic BP were independent of the menopausal transition. Yanes LL et al suggested that post menopausal hypertension could be multifactorial. Changes in estrogen / androgen ratio favouring an increase in androgen, activation of rennin-angiotensin and endothelin systems, increased vasoconstrictor eicosanoids, anxiety and depression may be important in the pathogenesis of postmenopausal hypertension.

Kim C et al studied the association of the menopausal status and the risk of diabetes among women with glucose intolerance. They concluded that among women with glucose intolerance, natural menopause did not increase the risk of diabetes. They further demonstrated that diabetes risk may be linked with



other factors like chronological ageing, rather than changes in menopausal status per se. The incidence of metabolic syndrome increases substantially during perimenopause and after menopause. A 9 year longitudinal study of 949 participants to study the prevalence of metabolic syndrome during menopausal transition found that, the prevalence of metabolic syndrome increases during the menopausal transition. Menopausal transition has also been associated with increased cardiovascular risk factors such as downregulation of the NO-c-GMP pathway resulting in endothelial dysfunction and other inflammatory markers.

### PCOS and Menopause

The classic phenotype of PCOS ameliorates with ageing. Hence, the PCOS phenotype after menopause is uncertain and difficult to define. The various criteria for diagnosis of polycystic ovarian syndrome may not be helpful after menopause and a specific phenotype for PCOS after menopause is poorly understood. Some studies have relied on a prior history of irregular menses, the presence of polycystic ovaries, or current hyperandrogenemia or hirsutism to diagnose PCOS in menopausal women. Other studies have expanded these criteria to include an elevated waist circumference or evidence of biochemical insulin resistance.

### Hyperandrogenism

Studies have shown that in normal women androgen secretion decreases with age. This decrease of circulating androgens in women with PCOS during their late years could reduce their cardiovascular risk. However, a case control, cross sectional study to compare 20 postmenopausal women with PCOS with 20 age and BMI matched controls showed that, postmenopausal PCOS women are exposed to higher adrenal and ovarian androgen levels than non PCOS women and hyperandrogenism in PCOS women persists after the menopause. Another cross sectional, university based hospital study by Puurunen et al found that the impaired glucose tolerance, enhanced ovarian androgen secretion and chronic inflammation observed in premenopausal women with PCOS persists after menopausal transition. Studies have also shown that the hyperandrogenism seen in PCOS women is both of adrenal as well as ovarian origin. Recently, results from National Institute of Health published by Shaw LJ et al showed a significant association between postmenopausal androgen levels and cardiovascular events. Therefore, androgens may play an important role on cardiovascular outcome and the vascular events may be influenced by the presence of excess androgen after menopause. To summarize, hyperandrogenemia may increase the risk of cardiovascular events in postmenopausal women with PCOS.

### Insulin resistance

Long term studies have shown that PCOS is a risk factor for Type 2 diabetes mellitus. PCOS women continue to manifest the metabolic alterations like insulin resistance after menopause, which makes them more susceptible to Type 2 diabetes mellitus. Hyperinsulinemia has been seen in postmenopausal women with PCOS. Similarly Cobin RH et al in their review on PCOS and insulin resistance concluded that PCOS women continue to manifest the metabolic alterations inherent in the insulin resistance syndrome after menopause, rendering them more susceptible to Type 2 diabetes and cardiovascular events. Shaw

et al compared 104 postmenopausal women with clinical features of PCOS with 286 post menopausal women without PCOS. They concluded that women with PCOS were often more diabetic than women without PCOS. In addition, women with clinical features of PCOS were more often insulin resistant.

### Dyslipidemia

A long term prospective study by Johanna Schmidt et al showed that PCOS women had higher prevalence of hypertension and higher triglyceride levels than controls. Similarly, another long term follow up study by Miriam H et al discovered that the metabolic syndrome occurred more often in patients with PCOS than in controls and did not depend on phenotypic presentation at diagnosis or the persistence of PCOS at follow up. Jones H et al did a cross sectional case control study comparing 29 PCOS women with 22 healthy controls. PCOS patients with hyperandrogenism had significantly higher liver fat versus PCOS women with normal androgen levels and versus controls. Similarly Gutierrez et al have shown that NAFLD (Non alcoholic fatty liver disease) is more prevalent in postmenopausal and women with PCOS than those in premenopausal women. Therefore to summarize, insulin resistance and dyslipidemia are more common in postmenopausal women with PCOS.

### CVD Risk markers

There is sparse data on the CVD risk in postmenopausal women with a history of PCOS. Whilst some studies have shown an increased CVD disease risk in women with PCOS, others have not. See Table 1

Cibula et al did a study to determine the prevalence of NIDDM, hypertension and Coronary artery disease

in perimenopausal women with history of PCOS. They did not find any difference in mean concentration of lipids and fasting glucose. However, PCOS women with markedly expressed clinical symptoms of PCOS had a higher prevalence of NIDDM and coronary artery disease. Similarly, a study was done by Solomon CG et al to assess the risk of CVD in women with history of irregular menstrual cycle. The women with history of irregular cycles had an increased risk of CHD even after adjusting for BMI and other confounders. A non significant increase in overall risk of stroke was also seen in women with very irregular cycles. Therefore, the authors concluded that menstrual irregularities (which can be considered as a predictor of a PCOS phenotype) may predispose to increased risk of CVD. In a substudy of the Women's Ischemia Evaluation Study (WISE) done to evaluate the risk of cardiovascular events in the postmenopausal women with clinical features of PCOS, it was shown that postmenopausal women with premenopausal menstrual irregularity and present hyperandrogenemia (PCOS like women) have a larger number of cardiovascular events than other postmenopausal women. The authors found an increased incidence of diabetes, obesity, metabolic syndrome and angiographic coronary artery disease in PCOS women compared with non PCOS women. When the cumulative 5 year cardiovascular event free survival was analysed, it was found to be 78.9% in PCOS women and higher in women without PCOS (88.7%). Krentz et al did a cross-sectional study of 713 postmenopausal women (mean age: 73.8 years), and identified a putative PCOS phenotype.

*Continued on page 10*

**Table 1: Shows various studies on the cardiovascular diseases in women with PCOS.**

Authors	Study design	No. Of PCOS pts	Mean age(yrs)	Conclusion
Cibula et al	Cross sectional	28	52+-5	PCOS women with markedly expressed clinical symptoms of PCOS had a higher prevalence of NIDDM and coronary artery disease
Solomon et al	prospective cohort study	82,439 subjects studied	ages (20-35 yr) 14 year follow up	Increased risk of CHD in women with irregular cycles, nonsignificant increased risk of stroke
Shaw et al 2008	Prospective	104	post-menopausal	Increased incidence of diabetes, obesity, metabolic syndrome and angiographic coronary artery disease in PCOS women compared with non PCOS women
Krentz et al 2007	Cross sectional	64	73.8+/-7.9 years	positive association between CVD and a number of features of PCOS in women with intact ovaries and no diabetes.
Wild et al 2000	Retrospective study	678	<75 at follow up	No difference in cardiovascular morbidity and mortality compared with age-matched women. Cerebrovascular events were more frequent in PCOS women compared with controls.
Wild S Pierpoint et al		319	Long term follow Up study	Cardiovascular mortality in PCOS women not higher than national rate
Schmidt et al 2011	Prospective	35 PCOS women and 120 age matched controls	61-79 years	High prevalence of Hypertension and high triglyceride levels but no increased risk of cardiovascular events like stroke, MI. diabetes, cancer

Continued from page 05

## Obesity and Hormones in PCOS

### Insulin resistance and Hyperinsulinemia

Although insulin resistance represents an important parameter, its laboratory assessment by measurement of fasting insulin levels, is not routinely recommended, as these measurements are imprecise with limited clinical usefulness. There is no simple validated clinical test for insulin sensitivity<sup>6</sup>. Though euglycemic hyperinsulinemic clamp studies and frequent-sampling, intravenous glucose tolerance test (with minimal model analysis) are "gold standard" methods for measuring insulin sensitivity and secretion, they are too cumbersome to be clinically feasible. Methods like homeostasis model of assessment for insulin resistance and oral glucose stimulated insulin sensitivity (eg, the Matsuda model) are less sensitive<sup>6, 8</sup>.

Rather than quantifying insulin resistance, it is clinically more relevant to assess the metabolic derangements resulting from insulin resistance by simple tests like glycated hemoglobin levels, 2-hour oral glucose-tolerance test, and fasting lipid levels.

Rare syndromes of primary insulin resistance such as Type A syndrome (Rabson-Mendenhall syndrome, Donohue syndrome, or leprechaunism) or type B syndrome (insulin receptor autoantibodies) manifest as profound hyperinsulinemia, acanthosis nigricans, and severe hyperandrogenism. Similarly rare cases of familial lipodystrophy are associated with extreme insulin resistance and hyperandrogenism. These rare conditions may also be misdiagnosed as PCOS.

### Management: Restoring hormonal harmony

Management of a woman with PCOS should be based on her specific concerns at different periods of time.

### Hyperandrogenism

Oral contraceptive pills and antiandrogens are commonly used for hirsutism management. Progestin component of OCPs suppresses LH secretion and hence the ovarian androgen production, while the estrogen component increases hepatic production of sex hormone-binding globulin, thus decreasing androgen bioavailability. Although no particular preparation of OCPs is found to be superior for clinical hyperandrogenism, commonly used preparations include monophasic combined OCP containing progestins with low androgenic potential (e.g. 35 µg ethinyl estradiol plus 0.25 mg norgestimate)<sup>3,7</sup>.

Among the antiandrogens, spironolactone is most commonly used. It is an androgen-receptor antagonist, and inhibits several enzymes in androgen biosynthesis pathway. Its anti-mineralocorticoid action can cause mild hyperkalemia.

Finasteride is 5α-reductase (type 1 enzyme) inhibitor which prevents conversion of testosterone to dihydrotestosterone in the pilo-sebaceous unit. Flutamide, an androgen receptor blocker is a potent antiandrogen, but its use is limited because of potentially severe hepatotoxicity. Antiandrogen usage should be avoided peri-conception, as they may disrupt androgen-dependent processes (e.g., formation of external genitalia) in a male fetus<sup>8</sup>.

### Oligo-anovulation and infertility

Clomiphene citrate is the most widely used antiestrogen for ovulation induction in women with polycystic ovary syndrome. It is proposed to occupy estrogen receptors in the hypothalamus and pituitary, thereby blocking the negative feedback action of estradiol. Consequently, serum FSH concentrations rise by around 50 percent, resulting in the stimulation of follicle growth and follicular estradiol production. Aromatase inhibitors are another class of potent ovulation inducers. Role of Metformin (an insulin sensitizer) for improving ovulation is largely equivocal.<sup>9</sup> However, one school of thought suggests that in women with PCOS, for whom pregnancy is

a more distant goal (after > 6 months time), initial treatment with metformin, combined with diet and exercise, is an option to induce ovulation. An advantage of achieving pregnancy with metformin as against clomiphene in this situation may be a decrease in risk of multiparity<sup>4</sup>. Other modalities of treatment for difficult cases include gonadotropin based therapy and surgical ovarian drilling.

For women not desiring fertility, oligomenorrhea, can be effectively treated and endometrial protection can be provided by either combined contraceptive pills, or continuous /cyclic progestin-only treatment<sup>3</sup>.

### Insulin resistance and Hyperinsulinism

Weight loss (and weight gain prevention) is likely an important element to reducing the ongoing severity of PCOS phenotypic expression. Dietary modification is an important part of any weight-loss program, with most studies suggesting that exercise alone is inadequate to improve symptoms relating to PCOS phenotype. Though insulin sensitizer metformin is not a weight loss drug, it is a reasonable adjunct to diet and exercise for obese women with PCOS. The additional parameters like glycemia and lipids should be periodically screened for and addressed as needed<sup>3</sup>.

Conventionally, therapy for PCOS has been centered on treatment of hirsutism and restoration of ovulation. However, over past 30 years, the increasing recognition of role of insulin resistance and its metabolic consequences in PCOS, phenotypic expression and management has led to the concept that PCOS is a misleading term. It may restrain the care-givers' perspective and impede effective communication with public and research funders. Moreover, the polycystic ovarian morphology, which the name signifies, is neither necessary nor sufficient to diagnose the syndrome. So in the recent 76<sup>th</sup> Scientific sessions of American Diabetes Association (June 2016), a case was made for change in nomenclature from PCOS to "Metabolic Reproductive Syndrome"<sup>10</sup>.

To conclude, PCOS represents a heterogeneous disorder with complex hormonal interactions resulting in a syndrome with wider metabolic ramifications beyond reproductive implications. It requires judicious use of tests to establish the diagnosis, followed by patient centric placement of management strategies. Lastly, importance of providing multidisciplinary care to address comprehensive needs of an individual patient as well as her at risk family members cannot be over-emphasized.

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The analysis was stratified by the diabetic status. The authors found a positive association between CVD and a number of features of PCOS in women with intact ovaries and no diabetes.

Therefore this study concluded that PCOS increases the risk of atherosclerotic CVD after menopause. Wild S Pierpoint did a retrospective cohort study to compare the cardiovascular mortality and morbidity in women previously diagnosed with PCOS and age matched controls. The authors found that cerebrovascular events were more frequent in PCOS women compared with controls. Whether PCOS increases the cardiovascular risk has been evaluated by many studies. Pierpoint et al studied 786 PCOS women and did a long term follow up of these women to test the cardiovascular mortality. The authors found that PCOS women do not have higher cardiovascular mortality than the national rate. Similarly, Schmidt et al did a long term prospective study on 35 PCOS women and 120 age matched controls. The authors found a higher prevalence of hypertension and high triglyceride levels in women with PCOS. However MI, stroke, diabetes and mortality prevalence was similar in the two cohorts with similar BMI. A meta-analysis of CVD risk markers in women with PCOS, revealed significantly elevated CRP, homocysteine, PAI-1 activity, VEGF, asymmetric dimethyl arginine(ADMA), advanced glycation end products (AGE's) and Lipoprotein a levels compared with controls, concluding that women with PCOS have increased serum concentrations of CVD risk markers compared with controls. Likewise, a systematic review and meta analysis on circulating markers of oxidative stress and PCOS also revealed that circulating markers of oxidative stress are abnormal in women with PCOS.

However, whether this apparent risk is translated into increased incidence of CVD in later life is uncertain. Therefore, some studies have shown that postmenopausal women with PCOS may have increased rates of obesity, diabetes, and cardiovascular events, whilst others have shown that these markers of cardiovascular risk may not cause an increased rate of mortality. Due to a lack of clarity regarding the actual risk of cardiovascular disease in postmenopausal women with PCOS, more long term studies are required.

**PCOS and Cancers**

A systematic review and meta analysis to assess the risk of gynecological cancers in PCOS suggested that women with PCOS are more likely to develop endometrial cancer (OR 2.7) and ovarian cancer(OR 2.52) but not breast cancer. Similarly, Zeina et al evaluated the association between endometrial cancer and PCOS. They concluded women that women with PCOS are 3 times more likely to develop endometrial carcinoma compared with women without PCOS. A Consensus on women's health aspects of PCOS (2012) revealed that most cancers have been found to be well differentiated and have a good prognosis. Also present data does not show increased risk of ovarian or breast cancer in women with PCOS. There is insufficient evidence to evaluate any association of PCOS with vaginal, vulvar, or cervical cancer.

**General Health of postmenopausal PCOS women**

A long term study on body composition, bone mineral density and fractures in late postmenopausal women with PCOS, concluded that PCOS with persistently higher FAI had similar muscle mass, BMD

and fracture incidence as controls. Since women with PCOS have multiple risk factors for CVD and may be at increased risk for premature CVD events. It seems prudent to inform the health risks to women in middle age or menopause, even though the exact long term risk of cardiovascular morbidity and mortality are still uncertain. The life style modification measures may be continued well into later in life in these women.

**Conclusion**

Women with PCOS have increased risk of dyslipidemia, insulin resistance, obesity, abnormal glucose metabolism and hyperandrogenemia. Long term studies have shown that metabolic syndrome occurs more often in women with PCOS.

The change in the hormonal milieu at menopause is associated with an increase in total body fat and an increase in abdominal fat. This weight excess at midlife is itself associated with an increased risk of cardiovascular and metabolic disease which may be pronounced by presence of PCOS phenotype. PCOS women continue to manifest the metabolic alterations like insulin resistance and hyperandrogenemia after menopause which makes them more susceptible to Type 2 Diabetes Mellitus. There is uncertainty as to whether PCOS per se increases cardiovascular mortality, however atherosclerotic CVD in postmenopausal women is associated with several PCOS-like features. However, whether PCOS causes an increased cardiovascular disease risk later in life is still uncertain. This dilemma needs to be solved since young PCOS women may be required to undergo expensive investigations and screening for cardiovascular disease whereas their true disease risk is still unknown.

**PCOS Quiz**

1. Which of the following conditions mimic PCOS?

- a. CAH
- b. Ovarian hyperthecosis
- c. Cushing syndrome
- d. all of above

2. Surgical approach to anovulatory PCOS patients includes

- a. Unipolar coagulation current at 3-6 different sites
- b. Coagulation current at the depth of 4-10 mm
- c. Helps in activate intra ovarian growth factor
- d. All of above

3. Which among these are the risks associated with PCOS

- a. Premature pubarche
- b. Preterm delivery
- c. Low birth weight
- d. all of above

4. Which of the following happen in PCOS

- a. Serum androgens are produced by ovary

b. Androstenedione is produced by ovary

c. Testosterone is produced by ovary

d. All of above

5. After agonist stimulation, which of these hormones are increased in PCOS

- a. 17-hydroxyprogesterone
- b. androstenedione
- c. androgens
- d. all of above

6. Which hormone is responsible for significantly amplifying steroid production in PCOS granulosa cells

- a. FSH
- b. LH
- c. Insulin growth factors
- d. All of above

7. Gene responsible for hirsutism in PCOS

- a. CYP11A1
- b. CYP222B
- c. B- cells

d. D19S884

8. Potential marker for insulin resistance is

- a. Acanthosis nigricans
- b. Hyperpigmented skin
- c. lichen planus
- d. none of the above

9. Clinical phenotype of PCOS is because of

- a. Intrauterine androgen exposure
- b. hyperandrogenesis by foetal ovary
- c. 21-hydroxylase deficiency
- d. all of above

10. Using the NCEP III criteria, the prevalence of Metabolic Syndrome in PCOS patients is

- a. 45%
- b. 20%
- c. 10%
- d. 5%

**Answers for PCOS QUIZ**

1. D	2. D	3. D	4. D	5. D	6. C	7. A	8. A	9. D	10. A
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## Diet for PCOS



**Shilpa Joshi**  
RD, Mumbai Diet and Health Centre,  
Bandra

Polycystic ovary syndrome (PCOS) is a complex endocrine disorder which is not entirely understood. Most patients with PCOS are obese and insulin resistant. Obesity, particularly abdominal obesity, is mediated by the development of insulin resistance and is closely linked to the development of this condition and its clinical features, particularly menstrual irregularities and increased androgen levels. Life style modifications focusing predominantly on diet and exercise behaviour are considered the preferred first line treatment for PCOS management. Several studies have shown that weight loss of 5-10% of weight in PCOS patient via energy restriction can reduce circulating insulin levels and hyperandrogenism.

### Traditional Indian Diets

Traditionally Indian diets are high in carbohydrate, moderate in fat and protein. These diets evolved as a part of agrarian culture which also involves a lot of physical activity. With modernization, the traditional diets have continued and some western dietary snacks/meals have been added. Due to this, diets have become hyper caloric. To add to the problem, modernization has led to sedentary lifestyle which actually demands decreased caloric intake. All these factors have led to increase in prevalence of obesity and hence PCOS in women.

### Energy Recommendation

The recommended energy should be adequate to maintain ideal weight and health in adults. If body weight and physical activity of an individual are known, it is easy to calculate the extra needs of energy for a particular situation. The energy requirements are suggested based on type of activity profile (sedentary, moderate, and heavy), age, gender, and physiological status of an individual.

### Carbohydrates

The daily carbohydrate intake should be approximately 50-60% of the total calorie intake. The primary source of complex carbohydrates in the diet should be cereals (whole wheat, brown rice, etc.), millets, pearl millet [bajra], finger millet [ragi], great millet [jowar]), pulses, red gram [tur dal], green gram [sabutmoong], etc., and legumes, soya, horse gram [kulthi]). Complex carbohydrates should be preferred over refined carbohydrates and its products (e.g., whole grain Roti over white [maida] bread, biscuits, toast or khari). Low glycemic index (GI) foods like oats with bran, unpolished rice, parboiled rice, whole pulses, beans, and legumes (whole pulses like moong, matki, chawli, masur) should be preferred. High GI foods (refined flour, root vegetables such as

yam/sooran/shakarkand, potato, colocasia/arbi) should be consumed in moderation. Simple carbohydrates like sugar, jiggery, honey, fruit juices (even without added sugar), and sabudana should be avoided. Individuals should be encouraged to consume about 4 servings of vegetables and 2 servings of fruits per day.

### Fibre



Dietary fibre is that part of food that is not digested by the gut and is considered as unavailable carbohydrate. It is not a single entity, but consists of a wide range of complex carbohydrates. Fibre is present in vegetables, fruits and legumes. Fenugreek seed is effective in controlling blood sugar and serum lipids than the insoluble fibre present in cereals and millets. Long-term consumption of fibre also improves glucose tolerance.

### Protein



Recent Indian studies like STARCH study have shown that Indian diets are very poor in protein. The so-called non-vegetarians in India do not consume non-veg more than 3 times a week. Plant based proteins like legumes, sprouts and dhal are consumed in very small quantity as are milk and milk products.

The adult recommended dietary intake (RDI) of 0.8 g protein/kg body weight meets metabolic and nutritional needs. Though current ADA recommendations allow 10 to 20% of energy intake as protein, a more conservative recommendation of 10 to 15% of total energy needs as protein is also in place.

Proteins of high biologic value should be given consideration, though protein should be included from both animal and vegetable sources. In short, every meal should contain one serving of protein in form of dal, sprouts, pulses, curd, milk, eggs or non vegetarian foods.

### Fats

Dietary fats (lipids) are important components of human diet, providing energy and essential fatty

acids, linoleic acid [LA] and a-linolenic acid [ALA] and serving as a transport for fat-soluble vitamins like A, D, E, and K. Fats improve texture and palatability of foods and have an important role in inducing satiety.

There is a growing consensus that diets should include only modest amounts of saturated fat (ghee, butter, coconut oil) but could include moderate levels of monounsaturates (olive oil, ground nut oil, rice bran oil). The World Health Organization (WHO) recommendation for the general population is 3 to 7% of energy from polyunsaturates. High intakes of polyunsaturates have been suggested to be potentially damaging, relating to increased production of lipid peroxides. Polyunsaturated fats are present in sunflower oil, kardi oil and safflower.

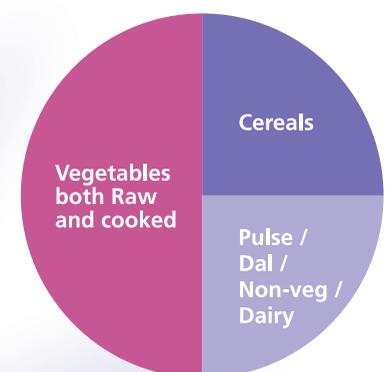
### Meal Replacers

Meal replacers (e.g. liquid formulas) are a popular weight loss diet, but their short-term use does not substitute for a long-term healthy eating pattern, which must be followed for a lifetime to achieve and maintain a healthy weight. Obese individuals typically underestimate their calorie intake by 40% to 50% when consuming a diet of conventional foods because of difficulty in estimating portion sizes, macronutrient composition, calorie content and in remembering all foods consumed. Meal replacements seem to decrease these difficulties and simplify food choices.

### Dietary Fibre Supplements

These are fibre based drinks/foods, give a feeling of fullness and satiety when consumed with hypo caloric diets, leading to weight loss. These also show other effects like reduction in cholesterol, triglycerides, uric acid and blood pressure.

### Other tips for eating



Other tips for eating smart are using a smaller-than-normal plate, and avoiding second helpings. Ideal way to fill a plate is shown in the figure below.

### Conclusion

Diet of PCOS is not very different from a normal ideal balanced diet. Current prevailing dietary scenario in India is high carbohydrate and high fats compounded by low physical activity. The pointers which can be given to patients are inclusion of proteins, fibres in form of vegetables and fruits and minimising portion sizes.

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