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Advances in Diabetes Management

A Case Compendium

Editors
Unnikrishnan AG
Sanjay Agarwal
Shailaja Kale
Mohan Magdum
R Kiwalkar
Suhas Erande
Anjali A Bhatt

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CHAPTER

2 Polycystic Ovarian Syndrome: A Metabolic Disease

Vaishali C Deshmukh

THE CASE

A 24-year-old girl presented to the polycystic ovary syndrome (PCOS) clinic with hirsutism, acne, irregular and scanty menses, hair loss and increased irritability. On examination, she was found to have central obesity, increased body mass index (BMI) of 29 kg/m^2, increased waist-to-hip ratio 1.05 and waist circumference of 102 cm. She had acanthosis nigricans on the nape of neck and hirsutism on chin and upper lip. Her blood pressure was 140/90 mm Hg and she had elevated postprandial (PP) blood glucose of 156 mg/dL and triglycerides of 210 mg/dL and a low level of high-density lipoprotein (HDL) cholesterol 35 mg/dL.

QUESTIONS

- What are the different diagnostic criteria for diagnosis of PCOS?
- What are the various comorbidities pathogenetically linked to PCOS ?
- What are the therapeutic options to improve her insulin sensitivity and hirsutism?

DISCUSSION

Polycystic ovary syndrome (PCOS) is a common endocrine disorder in reproductive-age women (one in five). The prevalence varies between 4% to -8% in Greece, Spain and the USA and varies up to 18% in community-based studies. The presence of irregular menses and polycystic ovaries was put forth first by Stein and Leventhal in 1935. Other symptoms include acne, male pattern balding (androgenic alopecia), hirsutism (excessive hair growth), infertility, central obesity, skin tags, and high androgen hormone levels. PCOS has significant reproductive, metabolic and psychological features. Collectively, these symptoms became known as "Stein-Leventhal syndrome". A polycystic ovary is defined as having 12 or more follicles (or cysts) within the 2–9 mm range under ultrasound (Table 1). Based on the Rotterdam criteria, a woman can be diagnosed with PCOS if she has two of the following three symptoms, viz.

1. A history of irregular menstrual cycles and anovulation, with onset at puberty.
2. The presence of chemically measurable hyperandrogenism, with documentation of high plasma levels of ovarian androgens, including total and free testosterone.

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Table 1: The different diagnostic criteria for polycystic ovary syndrome (PCOS)

National Institutes of Health criteria consensus statement	*European Society for Human Reproduction and Embryology/ American Society for Reproductive Medicine consensus statement*	*Androgen Excess Society position statement*
Oligo-ovulation and clinical and/or biochemical signs of hyperandrogenism, and exclusion of other etiologies	Two out of three of: oligo-ovulation and/or anovulation, clinical and/ or biochemical signs of hyperandrogenism, or polycystic ovaries, and exclusion of other etiologies	Hyperandrogenism (hirsutism and/or hyperandrogenemia), ovarian dysfunction (oligoanovulation and/or polycystic ovaries), and exclusion of other androgen excess-related disorders

3. The exclusion of other hormonal disorders with similar clinical features, including adult-onset congenital adrenal hyperplasia, hyperprolactinemia, adrenal or ovarian androgen-producing adenomas, hyperthecosis, and Cushing's syndrome.

PATHOGENESIS OF POLYCYSTIC OVARIAN SYNDROME

Following hypothesis have been put forth to describe pathophysiology of PCOS (Fig. 1):

- Hypothalamic-pituitary abnormalities that result in gonadotropin-releasing hormone and luteinizing hormone dysfunction.
- A primary enzymatic defect in ovarian or combined ovarian and adrenal steroidogenesis.
- A metabolic disorder characterized by insulin resistance in conjunction with compensatory hyperinsulinemia that exerts adverse effects on the hypothalamus, pituitary, ovaries, and possibly adrenal glands.

Hyperandrogenism is seen in 60% to 80% of cases. Insulin resistance is a pathophysiological contributor in around 50% to 80% of obese women with PCOS; conversely, lean women have less severe hyperinsulinemia and insulin resistance. Insulin resistance contributes to not only the metabolic features but also to reproductive features through augmenting androgen production and increasing free androgens by reducing sex hormone-binding globulin (SHBG).

Schema of etiology and clinical features, including reproductive, metabolic and psychosocial features of polycystic ovary syndrome (PCOS).

CLINICAL FEATURES OF POLYCYSTIC OVARIAN SYNDROME

1. *Reproductive features*: Hyperandrogenism, hirsutism, ovulatory and menstrual dysfunction, infertility, complications in pregnancy, miscarriage, pregnancy-induced diabetes (gestational diabetes), pregnancy-induced hypertensive disorders, neonatal complications and increased endometrial hyperplasia (Figs 2 and 3).
2. *Metabolic features*: Insulin resistance, metabolic syndrome, dyslipidemia, high rates of premature impaired glucose tolerance (IGT), type 2 diabetes mellitus (T2DM) and increased cardiovascular risk factors.
3. *Psychological features*: Anxiety, depression, poor self-esteem, reduced quality of life, negative body image.

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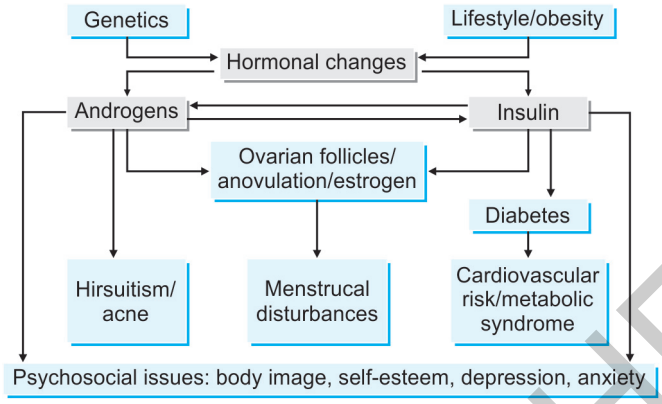

Figure 1 Clinicopathology of PCOS

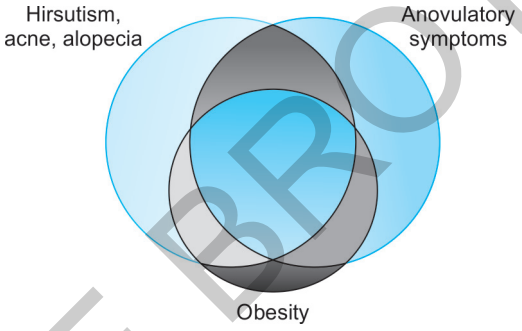

Figure 2 Polycystic ovary syndrome signs and symptoms overlap



Figure 3 Polycystic ovarian syndrome patient presenting with hirsutism, acne, obesity and acanthosis nigricans *(For color version, see Plate 1)*

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POLYCYSTIC OVARIAN SYNDROME AND INSULIN RESISTANCE SYNDROME

The prevalence of insulin resistance and compensatory hyperinsulinemia is increased in women with PCOS. Most reports indicate that at least 75% of women with PCOS fulfill the criteria for insulin resistance syndrome (IRS). Not all patients with insulin resistance have any or all of the abnormalities and clinical syndromes of the IRS, and these abnormalities and clinical syndromes can develop in the absence of insulin resistance. Nevertheless, the likelihood that a woman with PCOS will have insulin resistance or hyperinsulinemia is so substantial that all women with PCOS should undergo assessment for the adverse outcomes that constitute the IRS. Each component of the IRS in the subsequent list is a risk factor for cardiovascular disease (CVD), with greater risk for T2DM or CVD (or both) occurring in the presence of more abnormalities. The identifying features of IRS are as follows:

- Reduction of serum HDL cholesterol level to less than 50 mg/dL
- Increase in serum triglyceride level to greater than 150 mg/dL
- Hypertension, as defined by a blood pressure greater than 130/85 mm Hg
- Insulin resistance and increased tendency to have T2DM
- Fasting glucose level of 100 to 125 mg/dL
- Glucose level of 140 to 199 mg/dL, 120 minutes after challenge with 75 g of glucose.

Both obesity and physical inactivity are major factors that work synergistically with the inherent post-receptor defect and lead to insulin resistance in patients with PCOS. Not all women with insulin resistance have PCOS; this finding may be related to genetic differences in susceptibility of the ovary and pancreas. Obesity is often the trigger precipitating biochemical and clinical hyperandrogenism. Insulin resistance may be difficult to assess accurately in the absence of hyperinsulinemic-euglycemic clamp studies. Methods that measure only fasting glucose and insulin levels (including homeostasis model assessment and the quantitative insulin sensitivity check index) may be inaccurate in assessing insulin sensitivity in this group of women. Therefore, data are likely to underestimate the frequency of insulin resistance in patients with PCOS, who have a unique form of insulin resistance, and in those with mild insulin resistance and borderline normal fasting glucose and insulin levels. Because accurate assessment of insulin sensitivity is impossible in the clinical practice setting, it is prudent to regard all obese women as likely having insulin resistance and being at risk for the IRS and to assume that most non-obese women with PCOS to have the IRS as well.

POLYCYSTIC OVARIAN SYNDROME AND TYPE 2 DIABETES MELLITUS

The incidences of IGT and T2DM are significantly increased in women with PCOS. In a study from the University of Pittsburgh, T2DM was noted in 12.6% of women with PCOS (who had a mean age of 42 years) in comparison with 1.4% of matched control subjects. Studies with a 2-hour glucose tolerance test, 30–40% of patients with PCOS already have IGT or T2DM. Also seen in young teens with PCOS. Oligomenorrhea is a surrogate marker for probable PCOS and may predict a 2–2.5-fold increase in risk for T2DM, particularly in the presence of a family history of T2DM. In the Nurses' Health Study, more than 116,000 women 25–42 years old underwent follow-up for 8 years. A 2–2.5-fold increased incidence of T2DM was present in women with a history of oligomenorrhea (some

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of whom had severe cystic acne and hirsutism), in comparison with the women who had regular menses, although no physician-based diagnosis of PCOS was made. Obesity increased the frequency of diabetes in this population. Because approximately 80% of women with irregular menses have PCOS, these data are highly suggestive that PCOS is a strong risk factor for T2DM, independent of but exacerbated by the presence of obesity. Because the presence of T2DM abolishes the "gender gap" in coronary artery disease and because patients with concurrent diabetes and the IRS have the highest prevalence of coronary artery disease women with PCOS, IRS, and diabetes are likely to have not only an extremely high risk of coronary artery disease but also the microvascular consequences of uncontrolled diabetes. Prevention trials have shown that identification of IGT is important because intensive lifestyle modification, with or without pharmacologic intervention, will prevent the progression to overt T2DM and its consequent risks. Therefore, the American Association of Clinical Endocrinologists (AACE) and the American College of Endocrinology (ACE) have already included PCOS as an important risk factor for diabetes and have recommended screening for diabetes by age 30 years in all patients with PCOS.

POLYCYSTIC OVARIAN SYNDROME AND DYSLIPIDEMIA

Women with PCOS are frequently found to have atherogenic lipid abnormalities that may reflect underlying insulin resistance, as well as the effects of genetics, ethnicity, obesity, and lifestyle factors. Low HDL and high triglyceride levels are found in both obese and nonobese PCOS. A ratio of triglycerides to HDL of greater than 3.0 appears to predict insulin resistance effectively. Obesity exacerbates the elevated triglyceride levels in women with PCOS. Atherogenic modifications of low-density lipoprotein (LDL) cholesterol toward smaller, more dense particles have been demonstrated in women with PCOS as against control subjects. In a study in Central Pennsylvania of 153 obese or overweight (BMI >27 kg/m^2) and 42 non-obese non-Hispanic white women with PCOS having a mean age of 28 years, lipids were compared with those in control subjects of the same ethnicity and matched for BMI and waist-to-hip ratio. The women with PCOS had higher LDL levels than did the control subjects, who are independent of obesity. A large-scale epidemiologic study of 244 women with PCOS demonstrated elevated levels of LDL and reduced HDL cholesterol levels in the women with PCOS younger than age 45 years in comparison with matched control subjects. This early prolonged exposure to hyperlipidemia is a significant cardiovascular risk factor in women with PCOS in comparison with their peers who had normal menstruation.

ENDOTHELIAL DYSFUNCTION AND POLYCYSTIC OVARIAN SYNDROME

Women with PCOS have impaired endothelial function due to altered insulin regulation of endothelial nitric oxide synthesis, which leads to impaired nitric oxide dependent vasodilatation. The arterial consequences of metabolic dysregulation lead to reduced vascular compliance of large vessels as well as reduced vasodilatation. An abnormal vasodilatory response, endothelial dysfunction and decreased arterial compliance correlate with long-term risk for CVD. Increased endothelin-1 (ET-1) levels, a marker for vasculopathy in patients with insulin resistance, have been noted in patients with PCOS. Plasma ET-1 levels were increased five-fold in obese and nonobese subjects with PCOS over those in control subjects. These increased ET-1 levels may be an early sign of abnormal vascular

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reactivity induced by this vasoconstrictor polypeptide. Administration of 1,700 mg of metformin daily for 6 months reduced ET-1 and testosterone levels; this result suggests that insulin resistance and hyperandrogenemia potentially contribute to endothelial injury.

HYPERTENSION AND POLYCYSTIC OVARIAN SYNDROME

As patients with PCOS age, they have a higher incidence of hypertension than do matched control subjects. Increased systolic blood pressure has been noted in patients with PCOS. When blood pressure was monitored for 24 hours, women with PCOS had higher daytime systolic blood pressure, even after adjustment for BMI, insulin sensitivity, and body fat distribution, in comparison with that in matched control subjects.

PROINFLAMMATORY AND ATHEROGENIC MARKERS IN POLYCYSTIC OVARIAN SYNDROME

In comparison with control subjects, patients with PCOS have decreased fibrinolytic activity, higher levels of plasminogen activator inhibitor-1, and increased C-reactive protein (CRP) levels, all of which are markers for inflammation and correlate well with an increased risk for CVD in epidemiologic studies. Elevated CRP levels have been found in both obese and nonobese women with PCOS. Treatment directed toward reduction of cardiovascular risk (smoking cessation, diet, aspirin, statins, and possibly metformin and thiazolidinediones) should probably be more aggressive in those women with PCOS who have increased CRP levels.

As compared to controls:

- PCOS subjects have a four-fold to seven-fold higher risk of myocardial infarction
- A seven-fold increased prevalence of T2DM (15% versus 2.3% in control subjects)
- A three-fold increased incidence of treated hypertension
- A significantly higher waist-to-hip ratio
- A significant increase in plasma insulin levels and decrease in SHBG levels thus increasing risk for cardiovascular and metabolic disease
- Imaging studies in women with PCOS have uniformly identified a higher prevalence of anatomic and functional abnormalities indicative of existing underlying CVD or dysfunction in comparison with findings in age-matched control subjects.

INVESTIGATIONS IN POLYCYSTIC OVARIAN SYNDROME

Studies should be performed early in the morning, with the patient in a fasting state and, in women with regular menses, sometime between day 5 and day 9 of the menstrual cycle.

- Several determinations of total and free testosterone levels or a free androgen index help to assess the status of androgens
- Serum levels of luteinizing hormone and follicle-stimulating hormone can be determined. An increased ratio of luteinizing hormone to follicle-stimulating hormone greater than 2 is found in 60–70% of women with PCOS and is more likely to occur in non-obese than in obese women.
- Measurement of serum SHBG may reveal decreased levels in patients with PCOS

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- Measurements of serum prolactin, dehydroepiandrosterone sulfate, and 17-hydroxyprogesterone helps in diagnosing cause of PCOS
- A lipid profile can be obtained, including serum HDL and LDL cholesterol and triglycerides
- Plasma insulin may be measured, however, one should remember that methodologic inconsistencies occur and that insulin levels are not necessary for the diagnosis of the IRS
- The AAC and ACE recommend screening for diabetes by age 30 years in all patients with PCOS, including obese and nonobese women. The risk for diabetes is further heightened by a family history of diabetes, a personal history of gestational diabetes, and obesity, sedentary behavior and ethnicity

  Determination of a blood glucose level after challenge with a 75 g load of glucose may be performed. Because T2DM evolves over time, women with PCOS who initially test negative for diabetes should be periodically reassessed throughout their lifetime
- Pelvic ultrasonography, although nonspecific, affords a pretreatment view of the ovaries. The effect of treatment of PCOS can be monitored, in part, by noting ovarian size, follicle number, endometrial lining, and possible development of benign ovarian dermoids or other neoplasms (incidence of approximately 5–10%). The finding of morphologic evidence of polycystic ovaries on pelvic ultrasonography in 23% of apparently normal women limits its specificity in the diagnosis of PCOS.

TREATMENT FOR POLYCYSTIC OVARIAN SYNDROME

Presently, high androgen levels (hyperandrogenemia) and improving insulin sensitivity have largely become the clinical focus of PCOS treatment. Short-term management focuses around treatment of infertility, anovulation, obesity, hirsutism, acne. Long-term management revolves around decreasing cardiovascular risk, diabetes, dyslipidemia, hypertension and prevention of endometrial cancer. Lifestyle modification, metformin and weight loss therapies and antiandrogen therapies try to target the pathophysiologic abnormalities in PCOS. Moreover, PCOS need to be treated as a chronic disease and provides an excellent opportunity for prevention of chronic lifestyle diseases in future (Fig. 4).

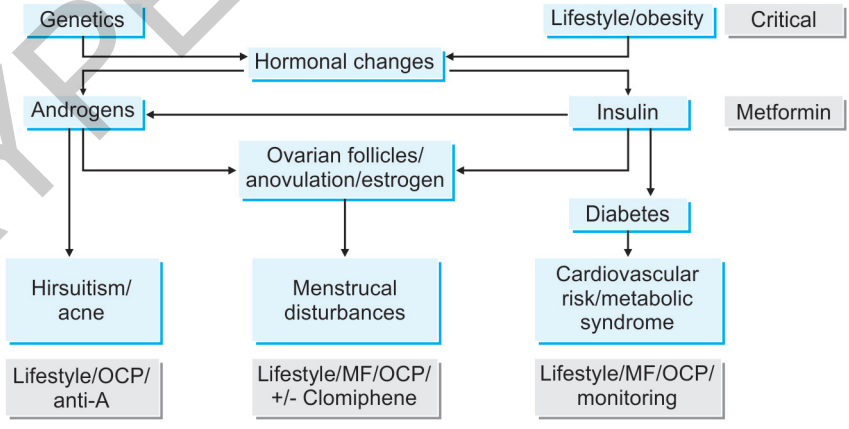

Figure 4 Summary of a targeted approach to therapy in polycystic ovary syndrome
Abbreviations: Anti-A, antiandrogens; MF, metformin; OCP, oral contraceptive pill.

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ISBN 978-93-5152-903-3

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