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REVIEW ARTICLE

Clinical manifestations of polycystic ovary syndrome

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Summary

Polycystic ovary syndrome, commonly abbreviated as PCOS, as the most common endocrine disorder in women of reproductive age, is a multifaceted disease characterized by various hormonal imbalances and a great degree of variation in its clinical presentation. This, coupled with its etiology and pathogenesis being incompletely understood, results in a broad disease spectrum that is challenging to accurately diagnose and manage. The primary clinical features which PCOS commonly manifests with include hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, though all three are not necessarily present in all PCOS patients.

Hyperandrogenism, manifesting as hirsutism, acne, and male-pattern alopecia, significantly affects both the physical and psychological wellbeing of these patients. Ovulatory dysfunction, presenting as irregular menstrual cycles due to oligo/anovulation, is an important element of PCOS's clinical presentation and leads to the infertility that some of these patients' experience. PCOS is commonly associated with insulin resistance and consequent hyperinsulinemia and metabolic disorders, seen in these patients. Subsequently, women affected with PCOS are at a greater risk of obesity, dyslipidemia, diabetes, and cardiovascular diseases, particularly later in life. The rate of mood disorders, namely depression and anxiety, is also increased in this population.

The complex nature of this syndrome makes difficulties in patient care, and its chronic nature emphasizes a proactive stance when it comes to treatment, but also a careful assessment of all the elements of the disease.

Keywords: PCOS, insulin resistance, hyperandrogenism, PCOM

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder occurring in women of the reproductive age (1). The syndrome was first described by Stein and Leventhal in 1935, who presented a series of cases that included seven female patients exhibiting enlargement of the ovaries and amenorrhea. The patients were treated with ovarian resection (2). The clinical presentation of the syndrome is heterogeneous, encompassing polycystic ovarian morphology, clinical and/or biochemical hyperandrogenism (HA), and ovulatory dysfunction accompanied with menstrual cycle irregularities and infertility (3).

The phenotypic expression of the syndrome is variable, and it changes throughout a woman's lifetime. This variation can occur because of aging or due to lifestyle changes. For example, reaching a healthy body weight can result in a "remission" of sorts (1). Due to this variability in phenotypic expression, over recent years PCOS has been defined as a syndrome instead of as a specific endocrine illness.

The consequences of this syndrome on the health of affected women persist throughout their lifespan and manifest in reproductive and psychological morbidity, cardiometabolic complications, an increased risk of malignancies, as well as diminished health-related quality of life (HRQoL) (3,4).

EPIDEMIOLOGY

A combination of considerable phenotypic variability, choice of patient population studied, as well as multiple different diagnostic criteria, all greatly influence the data on the prevalence of PCOS found in the literature. According to diagnostic criteria defined in 1990, the prevalence of PCOS among women of reproductive age is 5-9%, whereas, according to the Rotterdam criteria established in 2003, the prevalence ranges from 5.5 to 19.9% (5). Furthermore, according to the diagnostic criteria established in 2009, the prevalence ranges from10 to 15% (6).

In summary, the literature suggests the prevalence lies between 4 and 21% (5). It is important to note that in the general population, the incidence of PCOS increases proportionally with the rise in body mass index (BMI). The diagnosis of PCOS is most commonly made in the second or third decade of life, as it is at this time that the majority of patients begin to seek medical attention.

ETIOLOGY AND PATHOGENESIS

The etiology and pathogenesis of PCOS is incompletely understood. The initial clinical manifestations of PCOS appear in adolescence. It is currently thought to be a multifactorial disorder where endocrine, metabolic, genetic, epigenetic and environmental factors all play a role (7).

Namely, genetic loci linked to PCOS account for only 10% of its heritability. Furthermore, studies conducted in animal models point to a transgenerational inheritance pattern connected to intra-uterine epigenetic modulation that happens as a consequence of an aberrant intra-uterine milieu, specifically the presence of elevated androgen and Anti-Mullerian hormone (AMH) levels (8–10). Also, PCOS is more commonly found in the mothers and sisters of women who have been diagnosed with PCOS themselves (11). Apart from genetic factors, exogenous factors play a key role as well, most notably obesity, although obesity itself is oftentimes hereditary in nature. Obesity leads to changes in the functioning of the hypothalamus-pituitary-ovarian axis (12).

The three most significant theories of the origin of PCOS are:

1. The theory of aberrant ovarian steroidogenesis
2. The theory of insulin resistance (IR)
3. The theory of hypothalamus-pituitary-adrenal axis dysregulation (13)

In both ovulatory and anovulatory PCOS patients, ovarian theca cells overproduce androgens resulting in increased growth of immature ovarian follicles and a polycystic morphology of the ovaries. This is also the underlying cause of elevated concentrations of AMH, whose levels correlate with the number of preantral and small antral follicles. AMH is produced in the granulosa cells of small preantral follicles, and its serum levels are significantly elevated in PCOS patients with anovulatory cycles. Elevated levels of AMH reduce the number of follicle stimulating hormone (FSH) receptors, thereby inhibiting the FSH-induced stimulation of ovarian follicle growth leading to the absence of dominant follicle selection. Furthermore, elevated AMH levels decrease the sensitivity of ovarian follicles to FSH and block the conversion of androgens into estrogens through inhibition of the aromatase enzyme, which contributes to the development of HA (13). Hypersecretion of luteinizing hormone (LH) also leads to premature luteinization of granulosa cells and androgen hypersecretion which is additionally stimulated by hyperinsulinemia (14).

The second theory arose due to the strong link between IR and PCOS. It has been shown that in women with PCOS there are elevations in basal insulin secretion and the insulin secretory response to glucose is inadequate (15). A large number of studies point to hyperinsulinemia as a significant factor that drives IR. The synergistic action of insulin and LH exert an influence on theca cell androgen hypersecretion (16). Androgen excess, most notably of testosterone, androstenedione and dehydroepiandrosterone sulfate (DHEAS) cause premature ovarian follicle atresia giving rise to multiple ovarian cysts and anovulatory cycles. Excessive androgen

production in the ovaries exacerbates obesity, especially visceral obesity, which worsens IR, leading to hyperinsulinemia. To come full circle, this hyperinsulinemia stimulates ovarian hypersecretion of androgens (15,16). This is further supported by the breadth of evidence supporting the use of insulin sensitizers in the treatment of PCOS. For example, drugs used commonly in the treatment of type 2 diabetes mellitus (DM2) such as metformin, the inositol stereoisomers myo-inositol and d-chiro-inositol, and more novel drugs such as pioglitazone, liraglutide and semaglutide, all either have indications or are being investigated for the treatment of PCOS, with the aim of boosting fertility and improving hormonal regulation (17–19).

The third theory points to a dysregulation on the level of the hypothalamus-pituitary-adrenal axis, with disordered secretion of gonadotropins causing elevated LH levels as well as normal or low levels of FSH (20). Anovulation is the consequence of the absence of the maturation of the dominant follicle. Because of anovulation, there is a reduction in progesterone, and progesterone surges become absent in the luteal phase of the cycle, preventing it from exerting its negative feedback effects at the level of the pituitary. The frequency of the pulsatile secretion of gonadotropin-releasing hormone (GnRH) is increased, causing an elevation in the frequency and amplitude of LH secretion (21). Elevated levels of LH stimulate ovarian androgen secretion while the relative lack of FSH lead to growth arrest of the dominant follicle and anovulation. This disruption in gonadotropin secretion, specifically the inverted FSH/LH ratio, is primarily found in PCOS patients of a healthy weight and is mostly normal in obese women (22). This disruption may in fact originate very early on in life, as exposure to higher levels of androgens during intrauterine development is thought to predispose towards PCOS (23).

Finally, several observational and interventional studies have demonstrated a possible role vitamin D deficiency plays in the metabolic, endocrine, as well as inflammatory aspects of PCOS. Supplementing with vitamin D might even prove to be a useful prevention strategy in those with a positive family history, though more research is needed (24).

PCOS PHENOTYPES

Although diagnostic criteria for PCOS aren't entirely standardized, four PCOS phenotypes have been defined based on clinical and hormonal characteristics:

1. Phenotype A: "Classic PCOS", characterized by clinical or biochemical HA, anovulation (ANOV) and polycystic ovarian morphology (PCOM)
2. Phenotype B: "Classic PCOS", characterized by HA and ANOV
3. Phenotype C: characterized by HA and PCOM (representing the ovulatory type of PCOS)
4. Phenotype D: characterized by ANOV and PCOM (representing the milder, non-hyperandrogenic PCOS phenotype) (25,26).

The classification of PCOS into phenotypes is important for epidemiological and clinical research on PCOS (27). In the majority of patients, after the fourth decade of life, the phenotypic expression becomes milder, which manifests as a decrease in the size of the ovaries, number of follicles, androgen levels, as well as greater menstrual cycle regularity (28). According to a meta-analysis performed in 2016, phenotype A is the most prevalent type (29). The prevalence of the different PCOS phenotypes is shown in **Figure 1** (30).

Women exhibiting the classic PCOS phenotypes, phenotypes A or B, have more pronounced menstrual cycle disturbances, hyperinsulinemia, and are more likely to have IR, metabolic syndrome, and a high BMI, including obesity. Values of AMH are also more commonly elevated in those women (26).

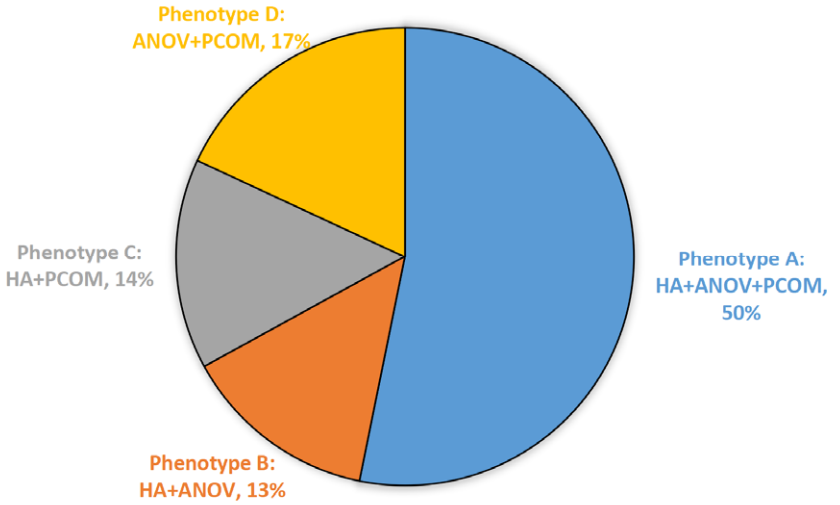

Figure 1. Prevalence of PCOS Phenotypes

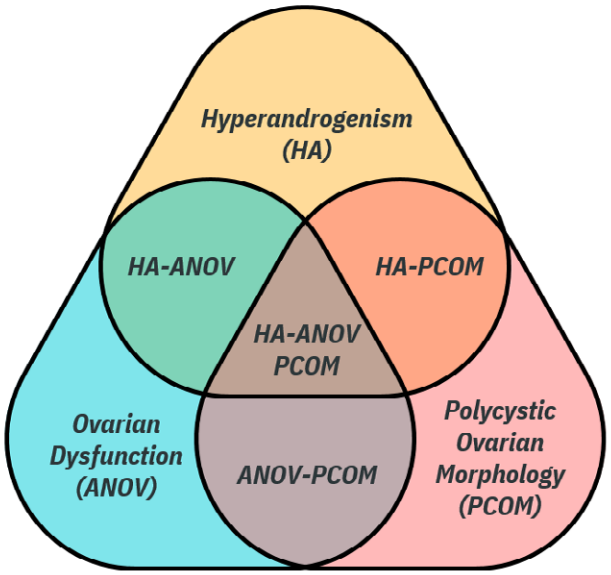

Figure 2. Clinical Characteristics and Phenotypes of PCOS

CLINICAL CHARACTERISTICS OF PCOS

The most important clinical characteristics of PCOS are HA with or without biochemical hyperandrogenism, ovarian dysfunction with or without menstrual cycle disturbances, and polycystic ovarian morphology, shown in **Figure 2**.

HYPERANDROGENISM

Clinical or biochemical hyperandrogenism (hyperandrogenemia-elevated androgen levels) occurs among 5-10% of women of reproductive age. The most common hyperandrogenic disorder is PCOS, which occurs in 80-85% of women with HA (31).

There is considerable heterogeneity among women with HA, and the phenotype itself can vary over time. Clinical manifestations of HA can be observed even without biochemical confirmation of elevated androgens, which is thought to be due to increased sensitivity of local tissues to androgens, as well as ethnic differences in phenotypic expression (32,33). On the other hand, biochemical HA can exist in PCOS patients even without an obvious clinical manifestation, especially in Asian populations (33).

Hirsutism is the most common clinical manifestation of HA. Other manifestations include acne, seborrhea, and androgenic alopecia, commonly known as male-pattern baldness. Hirsutism is defined as excessive growth of terminal hair in women, specifically those found in androgen sensitive areas. It appears in 5-8% of women of reproductive age (34). It happens because of elevated androgen serum levels and/or increased sensitivity of the pilosebaceous unit to androgens. It occurs in 65-75% of women with PCOS and represents the most common clinical criterion for diagnosing HA (32). Hirsutism is assessed using the Ferriman-Gallwey score (35). Scoring is performed by grading terminal hair growth on a scale from 0 (absence of terminal hairs) to 4 (extensive terminal hair growth), assessed on a total of 11 specific body regions: the upper lip, chin, chest, upper and lower back, upper and lower abdomen, upper arms, forearms, groin, and legs (35). A modified score takes into account a total of nine body regions, excluding the legs and forearms, with a sum score of nine and above considered clinically evident hirsutism (34). When assessing HA based on hirsutism, it must be taken into account that the degree and distribution of HA do not necessarily correlate with levels of hyperandrogenemia (36).

Acne represents an inflammatory process in the pilosebaceous unit and occurs on the face, neck, back, and pectoral region. The incidence of acne in women with PCOS is between 15 and 25% and there are significant ethnic variations (37).

Androgenic alopecia represents the progressive loss of terminal hair on the scalp. Both androgens from the ovaries and those secreted by the adrenals are involved in its etiology. It occurs even in women with normal serum androgen levels and it is thought that genetic factors also play a role in its development (38).

Hyperandrogenemia exists in 60 to 80% of patients with PCOS (39). The androgens originate mostly from the ovaries, but there are often elevated concentrations of adrenal androgens as well. In most women with PCOS, elevated levels of circulating testosterone can be found. Changes in levels of albumin and sex hormone binding globulin (SHBG), both of which bind sex hormones, can influence testosterone levels. Hyperinsulinemia and obesity decrease the level of SHBG, and low levels serve as a marker of IR and androgen excess (40).

Biochemical HA is diagnosed by finding elevated concentrations of testosterone, androstenedione, 17OH progesterone, dehydroepiandrosterone (DHEA), DHEAS, an elevated serum Free Androgen Index (FAI), and decreased concentrations of SHBG (40).

OVULATORY DYSFUNCTION

Dysregulation of ovarian function in PCOS patients manifests with oligomenorrhea or amenorrhea as a consequence of oligo/anovulation. Rarely, patients develop polymenorrhea. A total of 75-85% of women with PCOS have irregular menstrual cycles (38). Irregularity of the menstrual cycle commonly manifests soon after menarche, or the menarche itself may occurs later in life. In some patients, the first menstruations are initially regular but later become irregular, often accompanied by an increase in body weight (41).

Women with PCOS may also have completely regular cycles, which doesn't exclude them from potential ovula-

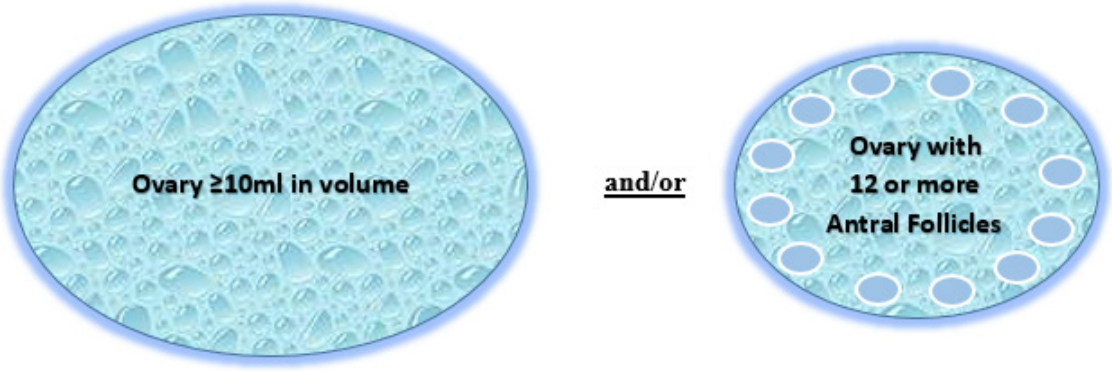

Figure 3. Diagnostic criteria for PCOM

tory dysfunction. Chronic anovulation may still be present, even in patients with completely regular menstrual cycles. It is estimated that PCOS can occur in 20-30% of women with regular menstrual periods, independently of their ovarian function (40).

Infertility in women with PCOS is most associated with chronic anovulation, but also with an increased propensity toward early spontaneous miscarriages. Additionally, PCOS related endometrial changes, lower oocyte quality, and obesity, may lead to a higher incidence of infertility in this population (41).

Recently conducted research shows IR is present in as many as 65-85% of women with PCOS and is more pronounced in patients who are also obese (38). IR represents a reduced ability of insulin to fulfill its metabolic activity consequently necessitating a higher level of secretion to exert its metabolic effect. Hyperinsulinemia develops secondarily, due to IR, and exists in 50 to 70% of women suffering from PCOS (42). Hyperinsulinemia negatively impacts follicular development. Elevated insulin levels can decrease the number of preovulatory follicles and cause anovulation (41).

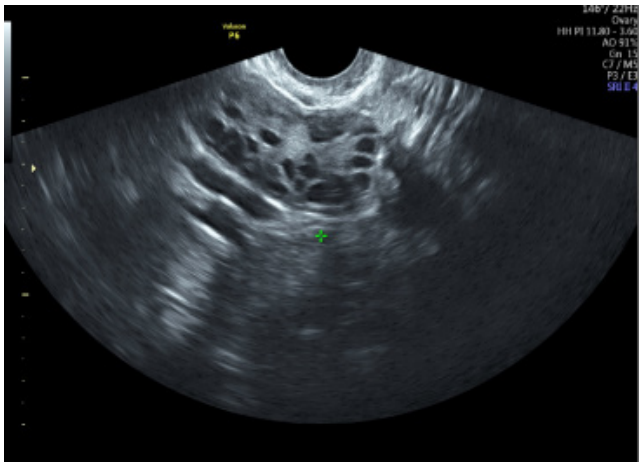

Figure 4. Ultrasonographic image showing PCOM

POLYCYSTIC OVARIAN MORPHOLOGY (PCOM)

In women with PCOS, the finding of enlarged ovaries is common, and the average volume of their ovaries is higher compared to women in the general population of the same age and body weight. Criteria used in defining PCOS includes the ovary/ovaries being larger than 10 ml and/or existence of ≥12 follicles 2-9 mm in diameter, as can be seen in **Figure 3** (43). Arguably, the condition for diagnosing PCOM is the presence of ≥25 antral follicles (44).

PCOM, although a common finding, is not crucial for the diagnosis of PCOS. PCOM is encountered in around 20% of healthy women with ovulatory cycles, during their reproductive years. The incidence in adolescents is even greater (45). During an ultrasound examination numerous peripherally arranged follicles surrounding the central stroma can be observed, shown in **Figure 4**.

In these patients, the growth of antral follicles concludes when the follicle is smaller than 10 mm and the emergence of dominant follicles does not occur. Follicular arrest is a consequence of HA in the ovary, as well as LH and insulin-mediated stimulation of follicular cells. When considering the diagnosis of PCOS, it is essential to bear in mind that polycystic ovarian morphology (PCOM) can be observed through transvaginal ultrasound in healthy women without PCOS, even more frequently in adolescent girls. Even with this in mind, it should be noted that the prevalence of PCOM in women with PCOS is approximately 80% (38).

MANIFESTATIONS OF PCOS AT DIFFERENT STAGES OF LIFE

During adolescence and the reproductive period, the clinical picture of PCOS is dominated by reproductive morbidity. Notable features include menstrual cycle disturbances, infertility, hirsutism, and acne. Obesity and DM2 can be found, but is not an obligatory component of the clinical picture (46). In the majority of cases, obesity is of the visceral type, which significantly influences the reproductive

and cardiometabolic morbidity of these patients (47).

Patients' fertility associated morbidity during the reproductive period is often due to the increased frequency of spontaneous miscarriages, occurrence of ovarian hyperstimulation syndrome during infertility treatment, gestational diabetes, and preeclampsia during pregnancy, often seen in this patient population (26). Premature delivery, before the 32nd week of gestation, are more common among women with PCOS. Newborns are also more frequently macrosomic, with a lower Apgar score, specifically less than 7 at 5 minutes of life, and are also at an increased risk of meconium aspiration syndrome (48).

Dyslipidemia, which manifests as elevated triglyceride levels, and decreased levels of high-density lipoprotein (HDL), happens as a consequence of IR and is found in 70% of patients with PCOS. Interestingly, levels of low-density lipoprotein (LDL) are also often decreased in these patients (28).

In general, PCOS patients have a lower HRQoL in all domains, but most often in those domains are affected by the clinical manifestations of the disorder. The HRQoL is especially negatively impacted by obesity, hirsutism, androgenic alopecia, acne, menstrual irregularities, and infertility (48). Furthermore, symptoms of depression, anxiety, as well as low self-esteem are frequent findings in this population (49). According to the literature, PCOS influences the daily life of more than 65% of patients affected by it, while fully half (50%) report that PCOS affects how they interact with their environment (40). Lower scores on HRQoL assessment scales significantly correlate with the severity IR (48).

With age and the gradual onset of menopause, the clinical presentation of PCOS changes. The regularity of the menstrual cycles improves, whereas the size of the ovaries, number of follicles, and serum androgen levels all decrease. The prevalence of classic PCOS phenotypes, phenotypes A and B, decreases in the 4th decade of life (28). However, according to current literature, there is no data available on the connection between the milder phenotypic expressions of PCOS in the peri- and post-menopause, and their long-term health outcomes. In later life, individuals with PCOS more commonly suffer from cardiovascular and cerebrovascular disease, pronounced obesity, metabolic syndrome, DM2, obstructive sleep apnea, endometrial hyperplasia and carcinoma, as well as anxiety and depression (28,48). The relative risk of developing cardiovascular disease is the highest among those with phenotype A and B, in whom it is estimated to be 1.3%. The risk of cardiovascular morbidity in these women persists throughout their lives and increases over time (48,50). The risk of venous thromboembolism in women with PCOS who have been treated with oral contraceptives is twice as high as in the general population (51).

Hyperinsulinemia and IR in later life can give rise to DM2 and cardiovascular disease. Menopause, in and of itself relates to IR and a propensity towards glucose intolerance and the development of DM2, which is then exacerbated by the higher incidence of this disease spectrum in women with PCOS. High insulin levels are also responsible for the development of obesity in these patients. These women are also at a higher risk of hypertension, metabolic syndrome, and obstructive sleep apnea (28,52).

Furthermore, as a consequence of their exposure to a large number of cumulative risk factors, specifically obesity, hyperinsulinemia, DM2 and menstrual irregularities coupled with chronic anovulation, PCOS patients have a higher risk of endometrial hyperplasia and endometrial cancer later in life (28). According to literature data, the risk of developing endometrial cancer is four times higher in these women compared to the general population (28). Although there is no definitive data on PCOS patients having a higher incidence of ovarian or breast cancer, chronic oligo/anovulation and the consequent prolonged exposure to estrogen theoretically increases the risk for the development of other estrogen-dependent tumors (28).

DIAGNOSIS

While PCOS represents the most common endocrinopathy of the reproductive period in women, variations in the phenotypic expression of the syndrome significantly complicate the diagnosis. Since 1990, several professional associations have put forth diagnostic criteria for the diagnosis of PCOS, with the aim of coming to a diagnostic standardization, and which have been collated in **Table 2**.

Initially, the US National Health Institute (NIH) defined diagnostic criteria for PCOS in 1990 as the following: clinical and/or biochemical HA with chronic anovulation, following the exclusion of other potential

Table 1. Clinical manifestations of PCOS throughout a woman's lifespan

Adolescence	Premenopause	Postmenopause
Insulin Resistance	Insulin resistance and DM2	Cardiovascular Morbidity
Obesity	Obesity	Obstructive Sleep Apnea
Hyperandrogenism	Hyperandrogenism	Endometrial Carcinoma
Oligoanovulation	Hypertension	Depression and Anxiety
	Dyslipidemia	
	Metabolic Syndrome	
	Obstructive Sleep Apnea	
	Depression and Anxiety	

Table 2. Criteria for establishing a PCOS diagnosis.

	1990 US NIH Criteria			
	2006 AE-PCOS Criteria			
	2003 Rotterdam Criteria			
	Phenotype A	Phenotype B	Phenotype C	Phenotype D
Hyperandrogenism and/or Hyperandrogenemia	✓	✓	✓	X
Ovulatory Dysfunction	✓	✓	X	✓
PCOM	✓	X	✓	✓

AE-PCOS Androgen Excess and PCOS Society

causes of HA and anovulation (53). The drawback of this definition is that it only recognizes the classic PCOS phenotypes.

In 2003, the European Society for Human Reproduction and Embryology (ESHRE) and the American Society for Reproductive Medicine (ASRM) established the "Rotterdam criteria". These expanded on the previously mentioned NIH criteria, but they also incorporated PCOM as a diagnostic criterion. Thus, according to the Rotterdam criteria, the conditions required for diagnosing PCOS are the presence of at least two out of the following three diagnostic signs: clinical and/or biochemical HA, chronic anovulation, and PCOM, with the prior exclusion of other pathological conditions with these symptoms (43).

The Androgen Excess Society (AE), together with the PCOS Society (PCOSS), defined a set of common diagnostic criteria for PCOS in 2009, emphasizing HA as one of the criteria for establishing the diagnosis of PCOS (48). No matter the diagnostic criteria, a prerequisite for the diagnosis is the exclusion of other conditions which may mimic the presentation of PCOS (54).

DIFFERENTIAL DIAGNOSIS

The diagnosis of PCOS is established by the exclusion of other causes of anovulation, such as thyroid diseases and hyperprolactinemia, and of other causes of HA, such as congenital adrenal hyperplasia, Cushing syndrome, severe IR syndromes, idiopathic hirsutism, androgen secreting tumors of the ovaries and adrenal glands (15,45,55).

TARGETING TREATMENT BASED ON A PHENOTYPIC APPROACH

The metabolic phenotype of PCOS, characterized by insulin resistance and an increased risk of type 2 diabetes, necessitates interventions focused on improving insulin sensitivity. Lifestyle modification, including diet and exercise, remains the cornerstone of treatment. Weight loss has been shown to improve metabolic parameters, and even a modest reduction in weight can significantly enhance insulin sensitivity and reduce hyperandrogenism (56). Pharmacological interventions, such as metformin, are also effective in improving insulin sensitivity and can be used particularly in women who do not respond adequately to lifestyle changes (57). Additionally, thiazolidinediones (TZDs) have shown promise in improving insulin sensitivity and menstrual regularity, although their use is limited due to adverse effects (58). In cases of severe obesity, bariatric surgery might be beneficial as it can significantly reduce weight and resolve metabolic disorders (39).

Managing hyperandrogenism in PCOS involves the use of medications that reduce androgen production or block androgen receptors. Combined oral contraceptives (COCs) are the first-line treatment for hyperandrogenism, effectively reducing hirsutism and acne by suppressing ovarian androgen production (59). Antiandrogens, such as spironolactone, flutamide, and cyproterone acetate, can be added to COCs for additional reduction in hirsutism (60). These medications act by blocking androgen receptors or inhibiting androgen production. In cases where COCs and antiandrogens are contraindicated or not tolerated, alternative treatments like finasteride, an inhibitor of 5-alpha-reductase, can be considered. Combining these treatments with insulin sensitizers like metformin may enhance metabolic benefits. In some cases, cosmetic procedures such as laser therapy may be employed to manage hirsutism (61).

The reproductive phenotype of PCOS is managed with the aim of inducing ovulation and achieving pregnancy. Clomiphene citrate (CC) remains the first-line pharmacological treatment for ovulation induction, with a success rate of approximately 80% for inducing ovulation and a 50% live birth rate (62). For women who are resistant to CC, letrozole, an aromatase inhibitor, has been shown to be more effective in inducing ovulation (63). Gonadotropins are used as a second-line treatment in women who do not respond to oral agents, but they require careful monitoring to reduce the risk of ovarian hyperstimulation syndrome (OHSS) (64). For women with PCOS undergoing assisted reproductive techniques (ART), lifestyle modifications

to achieve weight loss are recommended before starting treatment to improve outcomes (65).

CONCLUSION

PCOS can result in numerous health disorders in women and may present with a wide spectrum of symptoms and complications. These often appear at menarche, last throughout the reproductive period of life and continue into menopause. The continuous and persistent treatment of this syndrome is of major importance in those affected, not only for the sake of the reproductive health and fertility of these women, but also for their overall health and HRQoL improvement.

Author contributions

Conceptualization: RS and DM; Data acquisition: JZ, MOP, LR, and LN; Data interpretation and analysis: all authors; Preparing the draft of the manuscript: RS, JZ and LN; Revising and editing of the manuscript RS, DM, JBM and SO. Approval of the submitted manuscript version: all authors.

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KLINIČKE MANIFESTACIJE SINDROMA POLICISTIČNIH JAJNIKA

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Sažetak

Sindrom policističnih jajnika, odnosno PCOS, kao najčešće endokrino oboljenje kod žena u reproduktivnom periodu predstavlja kompleksno oboljenje koje se karakteriše različitim hormonskim poremećajima i brojnim varijacijama u kliničkoj slici. Dijagnoza i lečenje PCOS-a otežani su izrazito varijabilnom kliničkom slikom i nedovoljno poznatom etiologijom i patogenezom ovog poremećaja. Primarni klinički znaci PCOS-a obuhvataju hiperandrogenizam, ovulatornu disfunkciju i policističnu morfologiju jajnika, iako sva tri ne moraju biti prisutna kod svih pacijentkinja sa PCOS.

Hiperandrogenizam, koji se manifestuje kao hirzutizam, akne ili androgena alopecija, značajno utiče kako na fizičko, tako i na psihičko stanje ovih pacijentkinja. Ovulatorna disfunkcija, koja se manifestuje iregularnim menstruacionim ciklusima kao posledica oligo/anovulacije, predstavlja značajan element kliničke prezentacije PCOS-a i uzrokuje infertilitet koji se sreće kod pojedinih pacjentkinja. Takođe, insulinska rezistencija koja je karakteristična za PCOS dovodi do hiperinsulinemije i metaboličkih poremećaja koji se sreću kod ovih pacijentkinja. Posledično, žene sa PCOS imaju povećan rizik od gojaznosti, displipidemije, dijabetesa, i kardiovaskularnih bolesti, naročito u kasnijoj životnoj dobi. Učestalost poremećaja raspoloženja, pre svega depresije i anksioznosti takođe je značajno povećana u ovoj populaciji.

Kompleksnost sindroma otežava brigu o pacijentkinjama, a njegova hronična priroda ističe potrebu za proaktivnim pristupom lečenju i pažljivom procenom svih elemenata oboljenja.

Ključne reči: PCOS, insulinska rezistencija, hiperandrogenizam, PCOM

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