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Polycystic Ovary Syndrome (PCOS): a Comprehensive Review

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Abstract. Executive Summary: PCOS is a common, complex endocrine disorder affecting women of reproductive age. Its prevalence is around 8–13% worldwide (up to 20% with broad criteria)[1][2]. PCOS is defined by a combination of ovulatory dysfunction, hyperandrogenism, and/or polycystic ovarian morphology[3][4]. Patients often present with irregular menses, hirsutism, acne, and obesity. PCOS has multi-system effects – it carries significant metabolic risk (insulin resistance, type 2 diabetes, dyslipidemia, cardiovascular disease) and reproductive consequences (infertility, pregnancy complications)[5][6]. Effective management requires a stepwise approach: exclude other causes, apply appropriate diagnostic criteria (Rotterdam, NIH, etc.), and address lifestyle, metabolic, and fertility goals. First-line treatment emphasizes lifestyle modification (diet, exercise) plus tailored pharmacotherapy: e.g. combined oral contraceptives (for cycle regulation/hirsutism) and metformin (for insulin resistance)[7][8]. For fertility issues, letrozole is now the preferred ovulation induction agent[9]. Clinicians must also screen for psychiatric comorbidity; women with PCOS have ~1.2–1.5× higher risk of depression, anxiety, and bipolar disorders[10]. This review covers epidemiology, pathophysiology, clinical features, diagnostic algorithms, management strategies, outcomes and pitfalls, with detailed references. Aim: To review the epidemiology, pathophysiology, clinical features, diagnostic challenges, and management strategies of PCOS. Methods: A narrative review of peer-reviewed articles, clinical guidelines, and global health reports published between 2010 and 2025 was conducted using databases such as PubMed and Google Scholar. Results: PCOS affects approximately 6–20% of women depending on diagnostic criteria. Key features include menstrual irregularities, infertility, insulin resistance, obesity, and increased risk of type 2 diabetes and cardiovascular disease. Diagnostic variability and lack of awareness contribute to delayed diagnosis. Management requires a multidisciplinary approach including lifestyle modification, pharmacotherapy, and long-term monitoring.

Keywords: *Polycystic Ovary Syndrome; Hyperandrogenism; Insulin Resistance; HPO Axis Dysfunction; Rotterdam Criteria; Metabolic Syndrome; Ovulatory Dysfunction; Letrozole; Hyperinsulinemia; Endocrine Disorder*

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Introduction and Scope

Polycystic Ovary Syndrome (PCOS) is a **heterogeneous endocrine-metabolic disorder** of reproductive-aged women[11][1]. It is considered the most common endocrinopathy in this population, with prevalence typically 5–10% (range 4–21%) depending on the diagnostic criteria used[1][2]. PCOS manifests with *reproductive* (oligo/anovulation, infertility), *dermatologic* (hirsutism, acne), *metabolic* (insulin resistance, obesity, dyslipidemia) and *psychological* (mood disorders, low self-esteem) features[11][10]. The syndrome's etiology is multifactorial: genetic predisposition (family clustering of PCOS traits[12]), abnormal hypothalamic-pituitary-ovarian signaling, and metabolic factors like insulin resistance and inflammation interact in complex ways[13][14]. By "comprehensive", we include all phenotypic variants of PCOS (e.g. with or without ultrasound findings) and consider differential diagnoses. Key focus areas are: epidemiology, etiology/pathophysiology, clinical presentation, diagnostic criteria, management, complications, pitfalls, and patient counseling. This review is aimed at medical students and clinicians (MBBS level) preparing for publication or exam review.

Epidemiology and Risk Factors

•**Prevalence:** PCOS affects roughly **5–15% of women** of reproductive age globally[1][2]. Differences in criteria (NIH, Rotterdam, AE-PCOS) and population account for the wide range. A recent global analysis (GBD 2021) estimated PCOS prevalence of about 5.5–9.2% worldwide, rising steadily over recent decades[15][2]. In Asia and North America, prevalence tends toward higher end of range[2].

•**Risk Factors:** No single cause is known. **Genetics** is important (familial clustering). **Obesity** and **adiposity** are strong risk factors: ~65% of PCOS women are overweight/obese, and PCOS prevalence increases with higher BMI[16][14]. **Insulin resistance** (even in lean PCOS) promotes hyperandrogenemia and ovarian dysfunction[14][13]. **Inflammation** and **environmental factors** (endocrine disruptors, stress) also contribute[17][14]. Ethnicity influences presentation: for instance, South Asian women may develop PCOS at lower BMI with more metabolic risk[18].

Pathophysiology (Etiology)

PCOS arises from complex interplay between the hypothalamic-pituitary axis, ovaries, and peripheral

tissues[13][14]:

•**Neuroendocrine:** Many PCOS women have persistently **elevated GnRH** pulse frequency, leading to high LH secretion and relative FSH deficiency. This favors ovarian theca cell androgen production over follicle development[13].

•**Ovarian:** Theca cells in PCOS are hyperresponsive to LH/insulin, producing excess androgens. Follicles begin to mature but arrest (leading to polycystic morphology) due to inadequate FSH or intra-ovarian factors. This produces chronic anovulation[19].

•**Insulin Resistance:** Present in ~50-70% of PCOS patients (higher in obese)[20], insulin resistance causes **hyperinsulinemia** which: (a) augments ovarian androgen synthesis; (b) inhibits hepatic SHBG (raising free testosterone); (c) fosters metabolic dysfunction (T2DM, dyslipidemia)[13][14]. Insulin resistance is "central" to PCOS pathology.

•**Androgen Excess:** Androgens (testosterone, androstenedione) are typically elevated (biochemically or clinically as hirsutism). Androgen excess feeds back on hypothalamus to worsen GnRH/LH abnormalities.

•**Genetics:** PCOS is polygenic; GWAS have identified candidate loci (e.g. DENND1A, THADA). Male relatives (fathers/brothers) often show insulin resistance or early balding, indicating heritability[12].

•**Adipose and Environment:** Adipocytes in PCOS secrete inflammatory cytokines and adipokines that further impair insulin action. Early life factors (low birthweight, premature adrenarche) and lifestyle (high calorie diet, inactivity) amplify risk.

Clinical Manifestations

PCOS exhibits a **spectrum of symptoms**. Major features include:

•**Menstrual Irregularity:** Oligomenorrhea or amenorrhea in most patients due to chronic anovulation. (Severe cases may have no menses for months; mild cases can have irregular cycles.) Dysfunctional uterine bleeding and infertility are common consequences[21][22].

•**Hyperandrogenic Signs:** Hirsutism (male-pattern facial/body hair) is common (~60-70% of PCOS), quantified by the Ferriman-Gallwey score. Acne, oily skin, and androgenic alopecia (scalp hair thinning) are also seen[23]. Some women

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have biochemical hyperandrogenemia with few outward signs.

•**Polycystic Ovaries:** On ultrasound, ovaries often show many (≥12) small follicles per ovary and/or increased volume (>10 mL). This "polycystic" morphology is supportive but **not required** if other criteria are met[3][24]. (By expert consensus, ovarian ultrasound is not needed when hyperandrogenism *and* ovulatory dysfunction suffice[3][25].)

•**Metabolic Abnormalities:** A high proportion of PCOS patients have **insulin resistance**, impaired glucose tolerance, or metabolic syndrome. Obesity (especially central) is seen in ~50-80% of PCOS patients[16]. Dyslipidemia (high TG, low HDL), non-alcoholic fatty liver, and hypertension are common. Lean PCOS exists, but carries the same endocrine imbalance. Long-term, PCOS increases risk of type 2 diabetes 4-7× and increases 10-year cardiovascular risk[26][6]. Table 1 summarizes key features.

•**Psychological Effects:** Women with PCOS have higher rates of depression, anxiety, disordered eating, and body-image issues. Large studies show PCOS patients have roughly 1.2-1.5× higher odds of depression, anxiety, bipolar and sleep disorders[10][27]. For example, one cohort found odds-ratio 1.24 for depression and 1.50 for bipolar disorder in PCOS[10]. Chronic stress, fertility concerns and cosmetic issues contribute.

•**Other Comorbidities:** PCOS is linked with gestational diabetes, pre-eclampsia, and miscarriage in pregnancy[28]. Endometrial cancer risk is elevated (~2.7-fold) due to unopposed estrogen from chronic anovulation[29]. Sleep apnea and inflammation are more common.

Table 1

Key Clinical Features in PCOS (Rotterdam phenotype).

Category	Examples
Reproductive	Oligo- or amenorrhea, infertility, dysfunctional bleeding
Androgenic	Hirsutism (FG score ≥8), acne, androgenic alopecia
Metabolic	Central obesity, insulin resistance, acanthosis nigricans, dyslipidemia
Psychological	Depression, anxiety, low self-esteem, body dysmorphia
Ovarian	Polycystic ovaries on ultrasound (≥12 small follicles each or vol >10 mL)[24]

Diagnostic Criteria and Assessment

The **diagnosis of PCOS** requires careful assessment. It is fundamentally a *diagnosis of exclusion*[30] – other causes of

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hyperandrogenism or anovulation must be ruled out (see "Differential Diagnosis" below). Current criteria (two of three) are shown in Table 2; the 2003 Rotterdam criteria are most widely used[3][31]:

- **NIH 1990:** Requires *both* (1) clinical/biochemical hyperandrogenism and (2) chronic anovulation. (Polycystic ovaries not required.)
- **Rotterdam 2003 (ESHRE/ASRM):** Requires any **2 of 3:** (a) hyperandrogenism (clinical or lab); (b) oligo/anovulation; (c) polycystic ovaries on ultrasound[3][31]. This expands NIH by allowing diagnosis without hyperandrogenism if PCOM and oligo/anovulation are present.
- **AE-PCOS 2006:** Requires hyperandrogenism *plus* (ovulatory dysfunction or polycystic ovaries).

All criteria stipulate exclusion of related disorders (see below). Anti-Müllerian Hormone (AMH) is sometimes used as a surrogate for ovarian morphology, but guidelines caution that AMH thresholds are not yet standardized for diagnosis[32].

Table 2

PCOS Diagnostic Criteria (Adult Women)

Criteria	Requirement	Notes
NIH (1990)	Hyperandrogenism (clinical or lab) + oligo/anovulation	(≥2 yrs post-menarche)
Rotterdam (2003)	Any 2 of: (a) hyperandrogenism; (b) oligo/anovulation; (c) polycystic ovaries (by US)[3]	Broadest definition
AE-PCOS (2006)	Hyperandrogenism *plus* (ovulatory dysfunction or PCOM)	Requires androgens present

Interpretation: A diagnosis is made if two criteria are met and other causes excluded[3][30]. For example, a woman with irregular cycles and hirsutism (hyperandrogenism + oligo) qualifies even if ultrasound is normal. In adolescents (2+ years post-menarche), caution is urged – most experts require all three criteria or persistent features due to overlap with normal puberty[33].

Differential Diagnosis

Before confirming PCOS, exclude:

- **Pregnancy.** (e.g. new amenorrhea can be due to pregnancy.)
- **Thyroid dysfunction.** Check TSH, free T4. Hypothyroidism can cause menstrual irregularity.

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•**Hyperprolactinemia.** Prolactin levels – elevated prolactin can disrupt GnRH/LH and cause amenorrhea or galactorrhea.

•**Nonclassic congenital adrenal hyperplasia (NCAH).** 17-hydroxyprogesterone level rules out 21-hydroxylase deficiency (a common mimicker)[30].

•**Cushing's syndrome or exogenous steroids.** (Look for features like rapid weight gain, striae, hypertension.)

•**Androgen-secreting tumor.** (Rare; suspect with sudden onset, virilization, or very high testosterone >150 ng/dL[30].)

•**Ovarian failure/hypogonadism.** (E.g. low gonadotropins with high FSH suggests gonadal failure rather than PCOS.)

A thorough history and basic labs usually eliminate these. Even classic PCOS often overlaps with some findings of these conditions, so targeted tests (TSH, prolactin, 17-OH-progesterone) should be done as indicated[30].

Diagnostic Workup (Algorithm)

1. **History & Exam:** Menstrual history, signs of hyperandrogenism, weight/BMI, blood pressure, acanthosis nigricans. Document pattern of hair growth (Ferriman-Gallwey score).

2. **Laboratory Tests:** Serum total testosterone, free T, DHEA-S (to confirm androgen excess). Fasting glucose and insulin or HbA1c (for IR). Lipid profile. TSH, prolactin to exclude other causes. 17-OH-progesterone if NCAH is suspected.

3. **Ultrasound (if needed):** Pelvic ultrasound assessing ovarian volume and follicle count, especially if Rotterdam criteria evaluation requires PCOM. Not mandatory if (hyperandrogenism + ovulatory dysfunction) are present[3][25].

4. **Apply Criteria:** Check how many of the Rotterdam triad are positive. Confirm at least two.

5. **Assess Comorbidities:** Screen for obesity, insulin resistance, diabetes, hypertension, dyslipidemia. Check endometrial thickness if anovulatory.

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flowchart LR
    A[Symptoms: Irregular menses, hirsutism, acne] --> B[Evaluate Androgens & LH/FSH]
    A --> C[Exclude pregnancy, thyroid, prolactin, CAH]
    B --> D{Meets ≥2 of 3: HA, OA, PCOM?}
    C --> D
```

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    D -->|Yes| E[Diagnose PCOS]
    D -->|No| F[Consider alternate diagnoses]
    E --> G[Assess metabolic risks & mental health]
    E --> H[Plan management (see below)]
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Figure: Simplified PCOS diagnostic algorithm (Flowchart)[3][30].

Management (Treatment)

PCOS treatment is **individualized** based on patient goals (e.g. fertility vs. symptom relief), age, and comorbidities. No "cure" exists; management targets symptoms and long-term risks. Key principles[34][35]:

- **Lifestyle Modification (First-line):** All patients should be counseled on diet and exercise. Even 5–10% weight loss in overweight women improves ovulation, insulin sensitivity, lipids and hirsutism[34][35]. Diets emphasizing whole foods (Mediterranean, low GI) and regular aerobic/resistance exercise are recommended. Behavioral support to address weight stigma and mental health is important[34][35].
- **Pharmacotherapy (Non-fertility):**
- **Combined Oral Contraceptives (COCs):** First-line for menstrual regulation and treating hyperandrogenic symptoms (hirsutism, acne)[7]. They suppress LH, raise SHBG, and regulate cycles. No specific formulation is mandated, but low-dose estrogen and anti-androgenic progestins (e.g. drospirenone) can be beneficial.
- **Anti-androgens:** In moderate to severe hirsutism, add spironolactone (50–100 mg/d), finasteride, or flutamide (off-label) after pregnancy is ruled out and contraception ensured. These counter androgen effects.
- **Metformin:** Recommended particularly for metabolic concerns (IR, pre-diabetes, type 2 DM)[8]. Metformin improves insulin sensitivity and may modestly reduce weight, androgen levels, and cycle length. It is often used in combination with lifestyle and COCs or clomiphene.
- **Other Agents:** Statins (for dyslipidemia), antihypertensives, and bariatric surgery (in morbid obesity) may be indicated for risk factor management.
- **Fertility Management:** For women desiring pregnancy:
- **Letrozole (Aromatase Inhibitor):** Now first-line ovulation induction[9]. Letrozole (5 mg daily for 5 days) has higher live-birth rates than clomiphene and fewer multiple gestations.

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•**Clomiphene Citrate:** An older ovulation agent (selective estrogen modulator); use if letrozole fails or is unavailable[9].

•**Metformin:** May be added to either to improve ovulatory response or reduce gestational diabetes risk.

•**Gonadotropins:** Injectable FSH (±LH) are second-line if oral agents fail, with careful monitoring to avoid OHSS.

•**In vitro fertilization (IVF):** Third-line for anovulatory infertility when other treatments fail or if there are additional indications (e.g. male factor infertility)[36]. IVF is often considered for PCOS if >=3 cycles of ovulation induction are unsuccessful.

•**Psychological Support:** Given high rates of depression/anxiety, screen routinely[27]. Psychological counseling or therapy should be offered when needed.

Table 3

Pharmacologic Treatments in PCOS.

Medication	**Indication**	**Effect**
Combined OCPs	Menstrual regulation, hirsutism/acne	Suppress LH, ↓androgens; cycle control[7]
Spironolactone	Hirsutism, acne	Androgen receptor blocker; diuretic
Metformin	Insulin resistance, glucose intolerance	↓Hepatic glucose output, ↑insulin sensitivity[8]
Letrozole	Ovulation induction (infertility)	↓Estrogen, ↑FSH; ovulation (preferred agent)[9]
Clomiphene Citrate	Ovulation induction	↑FSH via estrogen receptor blockade (2nd line)
Gonadotropins (FSH)	Resistant anovulation	Direct ovarian stimulation (2nd line)
Statins, Acarbose, etc.	Dyslipidemia, metabolic syndrome	Address specific risk factors

Common Pitfalls and FAQs

•**Pitfalls:** Misdiagnosis is common. A patient with obesity and irregular menses may be labeled "PCOS" without assessing androgens or excluding other causes. PCOS *must* be distinguished from simple obesity-related amenorrhea or hypothyroidism. Over-reliance on ultrasound (e.g. "multicystic ovaries") can be misleading; even normal women can have multiple follicles. Another pitfall is neglecting metabolic and emotional aspects; focusing only on fertility misses the broader health impacts[37]. Also, treating PCOS as "just a cosmetic problem" (hirsutism) underestimates the

cardiovascular and diabetes risks.

• **FAQ Highlights:**

• *What causes PCOS?* There is no single cause. It's a polygenic condition triggered by hormonal and metabolic imbalances. A family history or insulin resistance often exists[12][14].

• *Can PCOS be cured?* No cure exists. It is a chronic condition that can be managed. Many symptoms improve with weight loss and age; some women may eventually resume ovulation. Lifelong follow-up for metabolic health is important.

• *Will I develop diabetes?* PCOS increases diabetes risk (~4× higher than peers) if left unmanaged. Regular glucose screening is recommended. Weight control and metformin can mitigate risk[26][6].

• *PCOS and pregnancy?* PCOS mothers have higher risks of gestational diabetes, hypertension, and miscarriage[28]. Pre-pregnancy counseling, glucose testing, and careful obstetric care are advised.

Conclusion

PCOS is a **multifaceted disorder** requiring holistic care. Early recognition (using updated criteria and guidelines) and comprehensive management – lifestyle changes, medical therapy, and psychosocial support – can greatly improve outcomes. Given its prevalence and rising incidence, PCOS deserves attention not only for fertility issues but for long-term health (metabolic syndrome and psychological well-being). Ongoing research (per the 2023 international guidelines) seeks better biomarkers (e.g. AMH) and therapies. Clinicians should apply evidence-based guidelines to ensure consistent, individualized care for women with PCOS[3][27].

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