Polycystic Ovarian Syndrome in Adult Indian Women: An Expanded Review of Prevalence, Pathophysiology, and Systemic Disease Risks

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Abstract

Objective. To synthesise 2003-2025 evidence on disease risks linked to PCOS, foregrounding Indian epidemiology while integrating global findings.

Design. Narrative review of PubMed, Scopus, and ICMR literature.

Results. Meta-analysis of 27 Indian studies shows pooled prevalence 11.3 % (Rotterdam) and 5.8 % (NIH) . Urban Maharashtra surveys reach 22–26 % . Insulin resistance underlies metabolic-syndrome prevalence 41–47 %, NAFLD 48–53 %, and a 10-year diabetes hazard ratio ≥5 . Adjusted Indian odds ratios: 1.6 for composite cardiovascular disease , 2.0–2.9 for gestational diabetes , 4–5 for endometrial carcinoma , and 2.3 for moderate-to-severe obstructive sleep apnoea .

Conclusion. PCOS in India is a chronic multisystem disorder demanding lifelong, integrated management across primary and specialist tiers.

Introduction

PCOS is the most common endocrine disorder of reproductive-aged women; Indian prevalence surpasses many Western estimates and manifests by the mid-twenties . Registry and hospital datasets confirm two- to four-fold elevations in metabolic syndrome, dyslipidaemia, NAFLD, major adverse cardiovascular events, gestational complications, endometrial carcinoma, mood disorders, and obstructive sleep apnoea, producing substantial but under-recognised public-health costs . The 2022 ICMR multidisciplinary-care model embeds routine metabolic screening and insulin-sensitising therapy within national non-communicable-disease frameworks .

Epidemiology in India

A 27-study meta-analysis reports national prevalence 11.3 % by Rotterdam and 5.8 % by NIH criteria . Urban college cohorts in Mumbai document 22.5 % prevalence; Pune adolescent screening shows 26.3 % . Rural Tamil Nadu surveys yield 3.7–9.3 %, illustrating wide geographic heterogeneity. Phenotype distribution skews to hyper-androgenic classes A/B in North India and normo-androgenic class C in South India, paralleling regional adiposity gradients. Median age at clinical diagnosis is 22–28 years, though biochemical insulin resistance and menstrual irregularity often arise by late adolescence.

Diagnostic Criteria and Phenotypic Classification

Rotterdam 2003 requires any two of oligo-/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology . Indian guidelines add compulsory exclusion of thyroid dysfunction and hyperprolactinaemia, adopt the Asian-Indian waist threshold (≥80 cm), and mandate a baseline 2-h OGTT even in lean phenotypes . Phenotype A (anovulation + hyperandrogenism + PCO) and B carry the greatest cardiometabolic load; phenotype D (ovulatory + PCO only) exhibits the mildest profile.

Pathophysiology

Post-receptor insulin-signalling defects coupled with β-cell dysfunction drive compensatory hyperinsulinaemia that enhances thecal androgen synthesis and suppresses hepatic SHBG . Heightened LH pulse frequency further accelerates androgen production . Adipokine imbalance, oxidative stress, and endothelial activation initiate early atherogenesis, hepatic steatosis, and pancreatic lipotoxicity, establishing a feed-forward loop of systemic injury .

Metabolic Risks

Metabolic Syndrome

Clinic-based Indian studies record metabolic-syndrome prevalence 41–47 % in PCOS versus 18–22 % in controls; central obesity, hyper-triglyceridaemia, and impaired glucose tolerance cluster by the second decade .

Type 2 Diabetes

A 10-year prospective Delhi cohort (n = 2 310) reported incidence 6.25 vs 1.49 per 1000 person-years and an adjusted hazard ratio 5.13 after controlling for BMI and socio-economic status . OGTT detects nearly half of dysglycaemia missed by fasting glucose, especially in lean women, justifying universal oral-glucose testing at diagnosis .

Dyslipidaemia

Mean LDL-C increases by 25–30 mg dL^{-1} and HDL-C falls by 7–10 mg dL^{-1} independent of BMI; atherogenic indices (apoB/apoA1 ratio, small dense LDL fraction) are significantly elevated, accelerating subclinical atherosclerosis .

Cardiovascular Disease

Meta-analysis of 12 Indian cohorts (≈58 000 women) yields an odds ratio of 1.63 for myocardial infarction or stroke after adjusting for age, BMI, and smoking . Surrogate measures show carotid intima-media thickness +0.15 mm and flow-mediated dilation −30 % by age 25, confirming early endothelial injury. Masked hypertension and left-ventricular diastolic dysfunction are also prevalent, reinforcing annual cardiometabolic screening .

Hepatic Injury

Ultrasound and transient-elastography clinics in Pune and Hyderabad document NAFLD prevalence 48–53 % in PCOS versus 23–26 % in non-PCOS controls; lean phenotype still shows 31 % steatosis, implicating androgen-mediated hepatic lipotoxicity . Alanine-aminotransferase correlates strongly with HOMA-IR, not BMI, indicating intrinsic insulin-signalling defects as the dominant driver .

Oncologic Risk

Persistent unopposed oestrogen triples to quintuples lifetime risk of endometrial carcinoma; pooled risk ratio ≈4.1 . Indian cancer registries attribute ≈12 % of endometrial cancers to antecedent PCOS, with mean malignancy age 47 years—approximately a decade earlier than population averages.

Reproductive and Pregnancy Complications

Indian obstetric cohorts reveal odds ratios 2.0–2.9 for gestational diabetes , 1.3–1.6 for hypertensive disorders, 1.8 for pre-term birth, and 1.5 for early miscarriage. Hyperinsulinaemia and androgen excess impair spiral-artery remodelling, explaining early-onset pre-eclampsia. Neonates show increased large-for-gestational-age incidence and transient neonatal hypoglycaemia.

Psychological and Sleep Disorders

Hospital surveys report depression prevalence 31–66 % and anxiety 57 % among Indian PCOS patients . A 2024 meta-analysis records a pooled odds ratio 2.3 for moderate-to-severe obstructive sleep apnoea; androgen-mediated pharyngeal-muscle hypertrophy and visceral adiposity are contributory . Untreated OSA worsens insulin resistance and heightens cardiovascular risk.

Management in the Indian Context

Screening Algorithm

At diagnosis and annually: fasting glucose, 2-h OGTT, HbA1c, lipid profile, liver enzymes, blood pressure, BMI, waist circumference, PHQ-9, GAD-7, and Berlin OSA questionnaire; repeat every six months if any parameter is abnormal .

Lifestyle

A daily 500-kcal energy deficit focused on millets and low-glycaemic staples, with ≥150 min week^{-1} moderate aerobic activity, is first-line. A 12-week yoga module (āsana, prāṇāyāma, meditation) significantly reduces HOMA-IR, total testosterone, and ovarian volume while improving cycle regularity .

Pharmacotherapy

Metformin 1.5–2 g day^{-1} remains first choice for dysglycaemia and weight control; GLP-1 receptor agonists suit BMI ≥27 kg m^{-2} or inadequate weight loss. Combined oral contraceptives regulate cycles and mitigate androgenic symptoms; cyclic medroxyprogesterone or an LNG-IUS prevents endometrial hyperplasia in chronic anovulation .

Service-Delivery Innovations

ICMR-NIRRCH one-stop PCOS clinics integrate endocrinology, dermatology, nutrition, psychiatry, and reproductive medicine, reducing fragmented care and improving adherence . Rural outreach leverages accredited social-health activists for menstrual-irregularity screening, with tele-endocrinology linkage fulfilling national non-communicable-disease targets.

Conclusion

PCOS in Indian women initiates a cascade of metabolic, cardiovascular, hepatic, oncologic, reproductive, psychological, and sleep disorders that begin early and persist lifelong. Strict nationwide enforcement of modified Rotterdam diagnosis, Indian-specific anthropometric thresholds, and integrated, lifelong multidisciplinary care is essential to blunt this systemic burden.

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