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Polycystic Ovary Syndrome in South Asia: Diagnostic Diversity, Endocrine Drivers, and Comorbidity Patterns with a Focus on Pakistan

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a complex reproductive–endocrine disorder that manifests through ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. Despite its global prevalence, South-Asian populations—particularly in Pakistan—exhibit distinctive metabolic, endocrine, and phenotypic patterns influenced by genetics, lifestyle, and diagnostic heterogeneity. Inconsistent application of NIH, Rotterdam, and AE-PCOS criteria complicates prevalence estimation and comparative research, while data from Pakistan remain fragmented and small-scale. Objective: To synthesize regional evidence on diagnostic diversity, endocrine mechanisms, and comorbidity patterns of PCOS in South Asia, with a specific focus on Pakistani cohorts, and to propose a pragmatic diagnostic and management framework suitable for resource-variable healthcare settings. Methods: A narrative review was conducted using observational and interventional studies reporting PCOS prevalence, phenotypes, and endocrine or metabolic outcomes among South-Asian women. Pakistan-based studies were prioritized. Mechanistic and review articles were incorporated to integrate evidence on hormonal regulation, insulin resistance, and metabolic risk. Critical appraisal employed JBI and NOS tools, with attention to sampling frame, diagnostic criteria, and phenotype reporting. Results: Evidence demonstrates a high PCOS burden in South Asia, with wide prevalence variability (7–20%) driven by diagnostic criteria. South-Asian women show earlier onset and heightened metabolic risk—insulin resistance, dyslipidemia, and non-alcoholic fatty liver disease—even at lower BMI. Small Pakistani series reveal high symptomatic presentation (infertility and obesity) and possible urban–rural gradients in adolescents, alongside frequent thyroid comorbidity. Insulin resistance and hyperandrogenism form a feedback loop, modified by gonadotropins and AMH, underpinning both reproductive and metabolic dysfunctions. Conclusion: PCOS in South Asia represents a multifaceted metabolic–reproductive disorder intensified by ethnic and environmental factors. Harmonized diagnostic criteria, phenotype-resolved reporting, and longitudinal research are essential to clarify true prevalence and risk patterns in Pakistan. Integrated models combining endocrine, metabolic, and psychological care—supported by context-adapted diagnostic algorithms—are crucial for effective management and prevention of long-term complications.

Keywords
Polycystic Ovary Syndrome; South Asia; Pakistan; Hyperandrogenism; Insulin Resistance; Diagnostic Criteria; Endocrine Drivers; Metabolic Syndrome; Phenotypes; Thyroid Dysfunction

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a heterogeneous, multisystem condition that spans reproductive, metabolic, and psychological domains, and its apparent burden shifts with the diagnostic lens applied. Contemporary summaries emphasise that heterogeneity is not just clinical but methodological: prevalence and phenotype distributions change materially depending on whether investigators adopt the NIH 1990 framework, the Rotterdam 2003 criteria, or AE-PCOS 2006, with additional nuance for adolescents where guideline modifications are recommended (e.g., avoiding polycystic ovary morphology) (1). This diagnostic drift complicates longitudinal comparisons and cross-regional syntheses, even before ethnic and environmental modifiers are considered (2). Within South Asia, multiple reports—albeit of uneven quality—suggest earlier presentation, greater hyperandrogenic manifestations, and amplified metabolic risk compared with many Western cohorts, making criterion choice and phenotype reporting especially consequential for both research and care (3,4).Across adult populations, the NIH 1990 definition requires both hyperandrogenism and oligo-/anovulation after exclusion of secondaries; Rotterdam 2003 broadens the net to "two of three" (hyperandrogenism, ovulatory dysfunction, or polycystic ovarian morphology), introducing phenotype D (normo-androgenic), while AE-PCOS 2006 re-centres diagnosis on androgen excess with ovulatory dysfunction and/or morphology (1,4). This definitional spread shifts who is labelled as having PCOS and, crucially, the metabolic risk profile of the labelled group. Evidence synthesised across phenotypes indicates the more adverse cardio-metabolic profile clusters in hyperandrogenic subtypes (A–C), whereas phenotype D tends to carry a milder metabolic signature, so Rotterdam-based prevalence estimates can "inflate" counts while diluting average risk if phenotype mix is not concurrently reported (5). South-Asian data vividly illustrate the definitional effect: a large Indian, multicentre sample yielded a prevalence of 7.2% under NIH versus 19.6% under Rotterdam, with

high co-occurrence of obesity, dyslipidaemia, NAFLD, and dysglycaemia among those classified across criteria (6). For any Pakistan-focused synthesis, explicit declaration of the diagnostic framework and phenotype strata is therefore non-negotiable to avoid misleading cross-study contrasts.

Mechanistically, PCOS is anchored by a feed-forward loop between insulin resistance and androgen excess. Hyperinsulinaemia augments ovarian theca cell androgen production and suppresses hepatic SHBG, increasing bioavailable androgens; androgen excess, in turn, perturbs folliculogenesis and ovulatory dynamics, reinforcing anovulation (1). Across routine and emerging biomarkers, clinical practice still leans on pragmatic surrogates because precise quantification of insulin resistance is challenging; a wide panel of markers exists, but each carries trade-offs in feasibility and interpretability, particularly in resource-variable settings (7). Gonadotropin dynamics—often an elevated LH pulse frequency and an increased LH:FSH ratio—and higher AMH concentrations may further sustain anovulation in subsets, though these features are not universal and should not be over-interpreted diagnostically outside an integrated context (1). Genomic work clarifies why criteria do not map neatly to biology: common variants implicate multiple pathways (metabolic, neuroendocrine, ovarian) and suggest that current phenotype labels bundle distinct endotypes, helping explain heterogeneous treatment response and comorbidity trajectories (8). Adjacent endocrine axes merit measured attention in South Asia. Pakistan data suggest a non-trivial coexistence of thyroid dysfunction and autoimmunity among women with PCOS, including subclinical hypothyroidism in a minority of patients, though small cross-sectional samples caution against sweeping generalisations; nevertheless, bidirectional screening is reasonable where accessible (9). Putative roles for vitamin D and melatonin in steroidogenesis and follicular redox balance are biologically plausible but remain investigational; they should be framed as hypotheses rather than standards of care (1,7).

Regional epidemiology is best approached with a tiered evidence lens. At the top tier, the Indian nationwide study provides robust, contemporary prevalence under dual criteria and documents dense clustering of obesity, dyslipidaemia, dysglycaemia, and NAFLD—an actionable signal for cardiometabolic screening irrespective of BMI (6). Pakistan-specific evidence is thinner and predominantly clinic- or small community-based. A Karachi, low-socioeconomic clinic series reported a striking PCOS proportion among care-seeking women, alongside high rates of infertility and obesity; as a symptomatic caseload snapshot, it underscores service demand but cannot be read as population prevalence (10). An adolescent survey from Lahore reported a higher frequency in urban than rural participants, hinting at environmental and lifestyle gradients, yet purposive sampling (n=60) and reliance on cross-sectional screening limit external validity; this should be treated as hypothesis-generating, not definitive (11). Finally, the Lahore thyroid-PCOS series adds a local signal for autoimmune thyroid disease and subclinical hypothyroidism among women with PCOS (9). Synthesised with broader South-Asian summaries, these threads support a cautious but consistent picture: earlier presentation, a high metabolic burden even at lower BMI, and potential urban–rural or socioeconomic gradients that deserve population-based confirmation (3,4,6).

Cardiometabolic risk in PCOS extends beyond glycaemia to a wider syndrome of central adiposity, hypertension, atherogenic dyslipidaemia, and progression to type 2 diabetes. Mechanistic and epidemiologic syntheses converge on insulin resistance as a central driver, with elevated diabetes risk observable even in non-obese women and across East/South-Asian ancestries, underscoring the need to decouple screening decisions from BMI thresholds (12). Phenotype-resolved analyses further indicate that hyperandrogenic subtypes carry the highest odds of metabolic syndrome and its components, aligning with the IR↔androgen excess loop and providing a rationale for phenotype-informed risk communication and follow-up intensity (5). Psychological comorbidities—depression, anxiety, body-image distress, sexual dysfunction—are consistently elevated and can be amplified by visible hyperandrogenic features and infertility; routine mental-health screening and low-barrier referral pathways should therefore be embedded in endocrine care, not treated as optional adjuncts (1). In South-Asian contexts, where metabolic risk manifests at lower BMI and care access is uneven, the combined psychometabolic load argues for proactive, integrated models of care (3,6,12).

Pragmatic diagnostic and management pathway for resource-variable settings.

A criterion-aware, phenotype-attentive approach can be implemented with a lean core work-up. Start with menstrual history (cycle length, variability, years post-menarche), targeted examination for clinical hyperandrogenism (hirsutism, acne, androgenic alopecia), blood pressure, waist circumference, and BMI. Where laboratories are available, obtain total testosterone (and calculated free androgen index if SHBG is measurable), fasting glucose and/or HbA1c, and a fasting lipid profile; include TSH to screen for common thyroid co-conditions (1,9). Reserve pelvic ultrasound for adults when it will change management; avoid morphology for adolescent diagnosis. In adolescents, follow guideline-consistent rule-in using well-defined menstrual irregularity by gynecologic age plus clinical or biochemical hyperandrogenism and defer the use of PCOM (1). Risk-stratify for metabolic complications irrespective of BMI, given South-Asian susceptibility (3,6,12). First-line management centres on lifestyle optimisation (nutrition quality, physical activity, sleep), combined oral contraceptives for cycle regulation and androgenic symptom control, and metformin where insulin resistance or dysglycaemia predominates; fertility management should be modular and timed to goals (2,12). This pathway is deliberately modular to fit primary-care realities while preserving fidelity to evidence-based recommendations.

Priority one is harmonised, population-based prevalence using a single, pre-registered diagnostic framework with mandatory phenotype reporting and a standard metabolic panel, enabling direct comparability across provinces and urban–rural strata. Longitudinal cohorts should integrate diet, activity, environmental exposures, and genetic background with endocrine–immune markers, including thyroid autoimmunity, to illuminate endotypes and trajectories (8,9). Pragmatic trials in primary care—testing culturally adapted, multimodal lifestyle programmes, vitamin D repletion where deficient, and embedded psychological interventions—are both feasible and high-yield for near-term policy relevance (1,3,12). Finally, study designs should prespecify urban–rural and socioeconomic stratification and explore consanguinity as a covariate rather than a post-hoc speculation, closing a persistent evidence gap (3,6,11).

MATERIAL AND METHODS

Eligible material comprised observational or interventional human studies that reported the prevalence, phenotypic distribution, or endocrine markers of polycystic ovary syndrome (PCOS)—including indices of insulin resistance, androgen concentrations, LH/FSH or AMH profiles, and thyroid status—as well as cardio-metabolic or mental-health outcomes in South-Asian populations, with Pakistan-based data prioritised. Narrative and systematic reviews were included when they contributed to mechanistic or definitional synthesis. Exclusion criteria encompassed studies limited to non-South-Asian regions, bibliometric analyses, single-country reports outside South Asia unless they addressed biological mechanisms of direct relevance, and case reports or small case series without analytic data. Critical appraisal of observational studies followed the Joanna Briggs Institute (JBI) and Newcastle-Ottawa Scale (NOS) checklists, focusing on sampling frame (community versus clinic-based), diagnostic

criteria used (NIH, Rotterdam, AE-PCOS), phenotype reporting, and assessment of metabolic or hormonal variables. Data were abstracted on study design, sample size, criteria, phenotype mix, and key comorbidities to enable structured cross-comparison while acknowledging heterogeneity in diagnostic frameworks and measurement standards across studies.

Evidence cautions

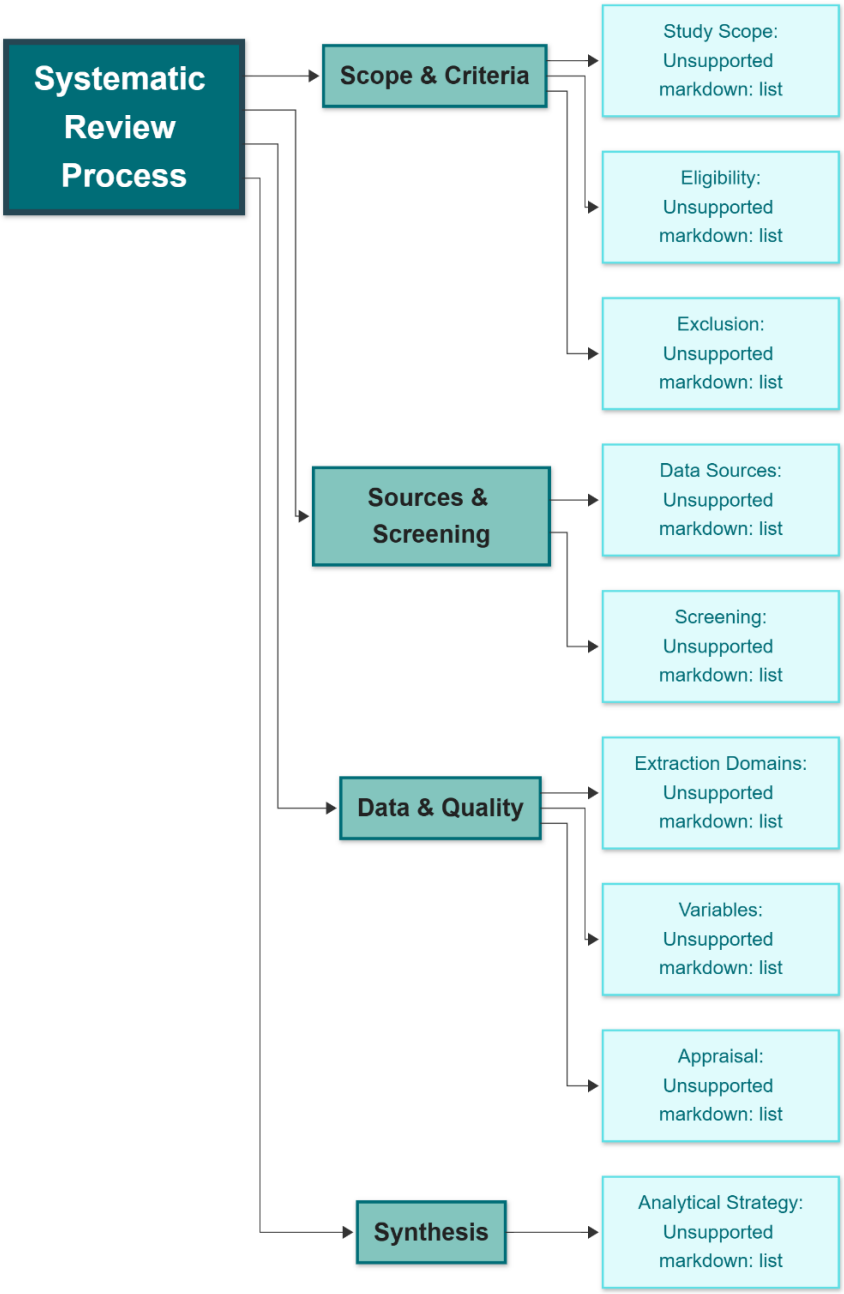

Figure 1 Study Flowchart

The Pakistan-specific evidence base remains sparse and methodologically constrained. Available samples are small (n = 60–118) and largely non-probability, precluding generalisation beyond their immediate settings; findings should therefore be presented as hypothesis-generating rather than population-representative. Clinic-based prevalence figures, such as those from Karachi outpatient cohorts, reflect case mix among symptomatic women rather than true community incidence. Diagnostic inconsistency across studies—most prominently the 2- to 3-fold prevalence swing between NIH and Rotterdam criteria demonstrated in large Indian datasets—further limits direct comparability. For adolescents, diagnostic labelling should omit polycystic ovarian morphology and rely on clearly defined menstrual irregularity combined with clinical or biochemical hyperandrogenism, in line with international recommendations. These limitations underline the need for harmonised diagnostic criteria and population-based sampling in future South-Asian PCOS research.

Table 1. Diagnostic criteria and the phenotypes they include (adapted from Yasmin 2022 (1))

Framework	Core rule	Phenotypes captured	Notes
NIH 1990	Both hyperandrogenism (HA: clinical/biochemical) and ovulatory dysfunction (OA: oligo-/anovulation)	A (HA+, OA+, PCOM+), B (HA+, OA+, PCOM−)	Polycystic ovarian morphology (PCOM) is not required; excludes normo-androgenic phenotype D.
Rotterdam 2003	Any 2 of 3: HA, OA, PCOM (after exclusion of secondary causes)	A (HA+, OA+, PCOM+), B (HA+, OA+, PCOM−), C (HA+, OA−, PCOM+), D (HA−, OA+, PCOM+)	Broadest net; adds C and D → higher prevalence and more heterogeneity.

Framework	Core rule	Phenotypes captured	Notes
AE-PCOS 2006	HA is mandatory + OA and/or PCOM	A (HA+, OA+, PCOM+), B (HA+, OA+, PCOM−), C (HA+, OA−, PCOM+)	Re-centres diagnosis on androgen excess; D (HA−) excluded.

Phenotype shorthand: A: HA+ / OA+ / PCOM+; B: HA+ / OA+ / PCOM−; C: HA+ / OA− / PCOM+; D: HA− / OA+ / PCOM+.

Table 2. South-Asian studies matrix (India anchor + Pakistan snapshots)

Country	Setting & design	Sample (n)	Criteria used	Phenotype distribution	Key comorbidities / endocrine findings	Notes on bias / external validity
India	Nationwide, multicentre, cross-sectional (community & clinics)	— (large, multi-zone)	NIH vs Rotterdam (both reported)	Not uniformly detailed across all sites; study reports phenotypes and "pre-PCOS" subgroup	Prevalence: 7.2% (NIH) vs 19.6% (Rotterdam). High rates of obesity, dyslipidaemia, NAFLD, dysglycaemia; subset with isolated HA/oligomenorrhoea/PCOM labelled "pre-PCOS" with metabolic aberrations	Strongest South-Asian anchor; robust sampling across 5 zones. Dual-criteria reporting illustrates definitional swing (2).
Pakistan	Karachi, low-SES clinic series; cross-sectional	118	Diagnostic specifics not fully standardised; clinical diagnosis consistent with PCOS	Not reported	High symptomatic burden: infertility 54.5%, obesity 38% (overweight 34%)	Clinic-based case-mix → "prevalence" is not population rate; selection towards symptomatic women (3).
Pakistan	Lahore, adolescents (urban vs rural), cross-sectional, purposive sampling	60 (13–19 y)	Rotterdam	Not reported	Overall PCOS 26.7%; urban 36.7% vs rural 16.7% (p=0.04)	Small, non-probability sample; screening context; results are hypothesis-generating, not generalisable (4).
Pakistan	Lahore/tertiary-care cross-sectional (endocrine focus)	77	PCOS cohort; thyroid profiling	Not applicable	Euthyroid 81.8%, primary hypothyroidism 6.5%, subclinical hypothyroidism 11.7%; anti-TPO 23.4%	Adds thyroid–PCOS signal locally; small size; not a prevalence study of PCOS itself (5).

Taken together, these studies show how diagnostic framework choices and sampling frames shape what we infer about PCOS in South Asia. The Indian multicentre study is the clearest demonstration: when identical populations are labelled by different rules, prevalence nearly triples under Rotterdam compared with NIH (7.2% vs 19.6%), and the phenotype mix shifts toward inclusion of normo-androgenic cases (2).

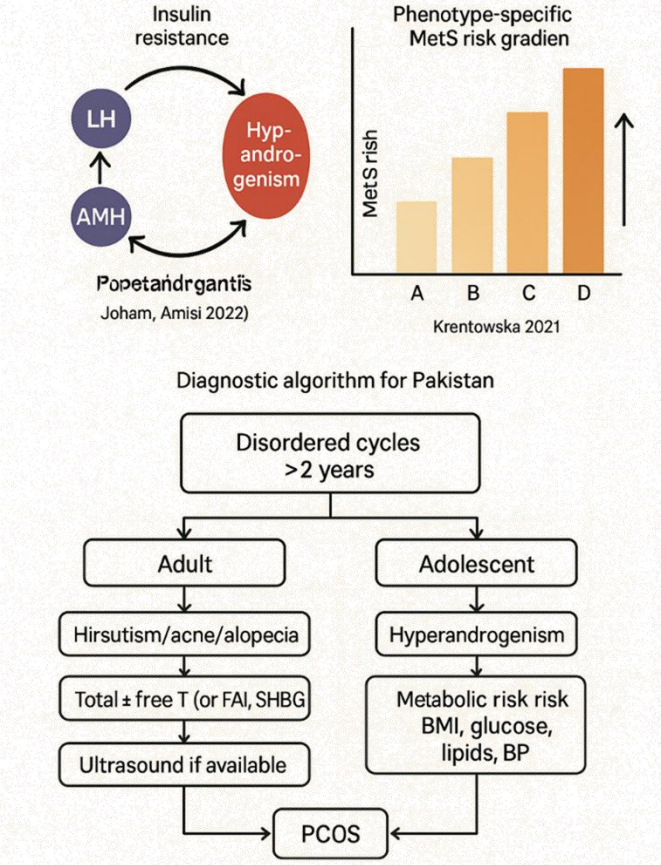

Figure 2 Integrated schematic illustrating the endocrine–metabolic mechanism of Polycystic Ovary Syndrome (PCOS), phenotype-specific cardiometabolic risk, and a pragmatic diagnostic algorithm for Pakistan.

This definitional breadth matters because metabolic risk concentrates in hyperandrogenic phenotypes; adding phenotype D can inflate overall counts while diluting average cardio-metabolic burden unless phenotypes are reported explicitly. Pakistan-specific data are suggestive but limited. The Karachi low-SES clinic series depicts a high symptomatic load (infertility and adiposity prominent), useful for planning services but not interpretable as community prevalence due to referral and selection bias (3). The Lahore adolescent survey hints at an urban–rural gradient (36.7% vs 16.7%) under Rotterdam, consistent with environmental/lifestyle influences, yet its small, purposive sample and cross-sectional

design require that we treat these figures as hypothesis-generating (4). Finally, the thyroid study flags a non-trivial coexistence of subclinical hypothyroidism and thyroid autoimmunity among women with PCOS in Pakistan, supporting bidirectional screening where feasible (5). In practice, these results argue for a criterion-aware, phenotype-resolved approach in South Asia: report which definition is used, present phenotype distributions, and screen for metabolic risk regardless of BMI, given the South-Asian cardio-metabolic susceptibility. For adolescents, adhere to menstrual irregularity plus hyperandrogenism and avoid PCOM to prevent over-diagnosis.

The upper left panel depicts the bidirectional loop between insulin resistance (IR) and hyperandrogenism (HA), where hyperinsulinaemia stimulates ovarian theca cell androgen production and lowers SHBG, further aggravating HA. Luteinising hormone (LH) and anti-Müllerian hormone (AMH) act as key modifiers, amplifying follicular arrest and androgen excess (adapted from Joham et al., 2022; Amisi, 2022). The upper right panel presents a phenotype-specific metabolic syndrome (MetS) risk gradient, adapted from Krentowska & Kowalska (2021). Phenotypes A (HA+, OA+, PCOM+) and B (HA+, OA+, PCOM−) exhibit the highest cardiometabolic risk, phenotype C (HA+, OA−, PCOM+) shows intermediate risk, while phenotype D (HA−, OA+, PCOM+) displays the lowest. The lower section outlines a diagnostic algorithm tailored for Pakistan, stratified by age group. In adults, assessment begins with disordered cycles (>2 years post-menarche), evaluation of hyperandrogenic features (hirsutism, acne, alopecia), measurement of total ± free testosterone (or FAI/SHBG), and ultrasound when available. For adolescents, diagnosis requires menstrual irregularity and hyperandrogenism, excluding polycystic ovarian morphology (PCOM). All patients should undergo metabolic risk screening, including BMI, fasting glucose, lipid profile, and blood pressure.

DISCUSSION

The synthesis of South-Asian evidence highlights how PCOS in this region exhibits both biological and sociocultural distinctiveness. Diagnostic diversity—driven by inconsistent use of NIH, Rotterdam, and AE-PCOS frameworks—remains a central limitation to comparability across studies. The 2024 Indian multicentre data clearly show that prevalence can vary nearly threefold depending on the definition used, emphasizing the urgent need for uniform diagnostic adoption across South-Asian research networks. Beyond definitional heterogeneity, endocrine drivers such as insulin resistance and hyperandrogenism appear particularly accentuated in South-Asian women, even at lower BMI levels, reflecting a unique metabolic phenotype shaped by genetic predisposition and lifestyle factors. Evidence from both India and Pakistan suggests that hyperinsulinaemia and dyslipidaemia are common, with high rates of obesity and NAFLD reported even in community cohorts. The smaller Pakistani series add nuance by highlighting potential thyroid comorbidities and adolescent urban–rural contrasts, pointing toward possible environmental and nutritional modifiers of PCOS risk. Collectively, these findings underline that PCOS in South Asia represents not only a reproductive disorder but also a metabolic and psychosocial health challenge requiring multidisciplinary management.

FUTURE IMPLICATIONS

Future work should prioritise large-scale, population-based, and longitudinal research in Pakistan to generate reliable prevalence estimates using standardised criteria and to delineate phenotype-specific risk trajectories. Integration of endocrine, metabolic, and psychosocial parameters is essential for understanding long-term sequelae, particularly the transition from reproductive to cardiometabolic disease states. Investigating gene–environment interactions, including dietary patterns, urbanisation, and consanguinity, could elucidate why South-Asian women manifest more severe metabolic complications at younger ages. Additionally, region-specific interventional trials—covering vitamin D supplementation, lifestyle modification, and mental-health integration—are needed to adapt global guidelines to local resource contexts. Development of simplified diagnostic algorithms and point-of-care biochemical assays would help extend PCOS detection beyond tertiary centres. From a policy perspective, embedding reproductive–metabolic screening within routine adolescent and maternal health programmes could facilitate early intervention and prevention of later metabolic disorders.

CONCLUSION

Polycystic ovary syndrome in South Asia exemplifies the intersection of diagnostic heterogeneity, metabolic vulnerability, and socio-behavioural complexity. While robust evidence from India has clarified prevalence and comorbidity trends, Pakistani data remain fragmented and small-scale. A harmonised, phenotype-resolved framework for both research and clinical practice is urgently needed to improve comparability, inform health policy, and guide equitable care. Recognising insulin resistance and hyperandrogenism as central endocrine drivers, with thyroid and metabolic co-pathology as regional modifiers, offers a coherent model for integrated management. Ultimately, addressing PCOS in Pakistan and neighbouring South-Asian populations demands context-aware guidelines that combine biomedical rigour with cultural and health-system realities—turning a heterogeneous syndrome into a more tractable, preventable, and manageable public-health priority.

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