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Original Research Article

EARLY-ONSET METABOLIC SYNDROME IN OBESE ADOLESCENT GIRLS WITH POLYCYSTIC MORPHOLOGY ON SONOGRAPHY (PMOS): A HOSPITAL-BASED COMPARATIVE CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Polycystic morphology on sonography (PMOS) is an increasingly recognised ultrasound finding in obese adolescent girls and represents an early hormonal-metabolic phenotype that may precede the full clinical expression of Polycystic Ovary Syndrome (PCOS). Obesity and PMOS share the common pathophysiological axis of hyperinsulinaemia and insulin resistance, both of which are pivotal drivers of early-onset metabolic syndrome (MetS) in the adolescent age group. The burden of MetS in PMOS-positive obese girls in India remains poorly characterised. The objective is to assess the prevalence of early-onset MetS and its individual components in obese adolescent girls with sonographically confirmed PMOS compared to obese girls without PMOS; to evaluate the degree of insulin resistance in both groups using HOMA-IR; and to identify independent predictors of MetS in this population.

Materials and Methods: A hospital-based comparative cross-sectional study was conducted at Shantiniketan Medical College and Hospital, Bolpur, West Bengal, from January 2025 to March 2026. Few patients were added from private clinic. Eighty obese girls aged 10–18 years with confirmed PMOS (Adams' criteria on pelvic ultrasonography) and 80 obese non-PMOS girls of matching age and BMI z-score formed the two study groups (n=160 total). MetS was defined by IDF 2006 paediatric criteria. HOMA-IR cutoff ≥2.5 was used for insulin resistance. Statistical analysis included Chi-square test, Student's t-test, and binary logistic regression.

Results: MetS prevalence was 40.0% in PMOS girls versus 17.5% in non-PMOS controls (χ^2=9.34, p=0.002). Insulin resistance (HOMA-IR ≥2.5) was present in 55.0% of PMOS cases versus 25.0% of controls (p<0.001). Mean HOMA-IR was significantly higher in PMOS girls (3.24 ± 1.12 vs 2.36 ± 0.86, p<0.001). PMOS status (OR=3.12, 95% CI: 1.52–6.39, p=0.002) and BMI z-score (OR=1.86, 95% CI: 1.23–2.81, p=0.003) were independent predictors of MetS.

Conclusion: PMOS in obese adolescent girls is an independent and significant predictor of early-onset MetS, concomitant insulin resistance, and cardiometabolic risk. Systematic metabolic screening including HOMA-IR assessment and early lifestyle intervention are essential for all PMOS-positive obese girls.

Keywords: Polycystic morphology on sonography; PMOS; PCOS; Metabolic syndrome; Insulin resistance; Adolescent obesity; HOMA-IR; Early-onset metabolic syndrome; Cardiometabolic risk; Paediatric endocrinology.

INTRODUCTION

Childhood and adolescent obesity has emerged as one of the most pressing global public health challenges of the twenty-first century, with prevalence rates rising steeply across both developed and developing nations, including India, where the twin epidemics of undernutrition and overnutrition now coexist across socioeconomic strata.[1,2] Obesity in the paediatric and adolescent age group is not merely an excess of adipose tissue; it constitutes a complex metabolic derangement characterised by insulin resistance, atherogenic dyslipidaemia, chronic low-grade inflammation, and dysregulation of the hypothalamic-pituitary-gonadal (HPG) axis — perturbations that, in obese girls, create a particularly unfavourable hormonal milieu predisposing to the development of polycystic ovary syndrome (PCOS) and its precursor spectrum.[3,4] Polycystic morphology on sonography (PMOS) — defined as the presence of ≥12 follicles measuring 2–9 mm in diameter in at least one ovary and/or an ovarian volume >10 mL on pelvic ultrasonography, in the absence of a dominant follicle or corpus luteum,[5] — represents an early and highly prevalent sonographic phenotype in obese adolescent girls that may or may not fulfil the complete Rotterdam criteria for PCOS, which additionally require clinical or biochemical hyperandrogenism and/or oligo-anovulation.[6] Crucially, PMOS in the context of adolescent obesity is not a benign incidental finding; it represents a convergence of gonadotrophin dysregulation, hyperinsulinemia-driven thecal androgen hypersecretion, and anovulatory follicular arrest — a metabolic and endocrine phenotype that carries significant cardiometabolic risk independent of obesity alone.[7,8] Metabolic syndrome in the paediatric and adolescent population, defined using IDF 2006 age-stratified criteria, encompasses central obesity, atherogenic dyslipidaemia (elevated triglycerides, reduced HDL cholesterol), elevated blood pressure, and impaired fasting glucose — a constellation that predicts adult-onset type 2 diabetes mellitus, polycystic ovary syndrome, hypertension, and cardiovascular disease with high fidelity when present during adolescence.[9,10] Studies in adult PCOS populations have consistently documented MetS prevalence rates of 33–46%,[11,12] and impaired glucose tolerance and insulin resistance have been identified as the earliest and most prevalent metabolic abnormalities, frequently preceding the composite MetS diagnostic threshold — a temporal precedence that implicates hyperinsulinemia as the primary driver rather than a downstream consequence of PCOS.[13,14] However, data on early-onset MetS specifically in adolescent girls with PMOS — a phenotypically distinct and earlier-spectrum entity than fully expressed PCOS — remain sparse, particularly from Indian cohorts where the obesity-PMOS-MetS axis may manifest differently owing to the South Asian metabolic phenotype characterised by greater visceral adiposity at lower absolute BMI, more pronounced insulin resistance, and earlier onset of atherogenic dyslipidaemia.[2,15] The clinical imperative for studying this population is substantial: if PMOS independently confers MetS risk beyond that attributable to obesity alone, then routine pelvic ultrasonography in obese adolescent girls should prompt a structured metabolic work-up — including HOMA-IR-based insulin resistance assessment — rather than being dismissed as an isolated ultrasound observation pending the development of full PCOS. Timely identification of MetS and insulin resistance in adolescent girls with PMOS would enable early lifestyle and, where indicated, pharmacological intervention during a developmentally critical window when metabolic trajectories may be substantially modified.[16] Against this background, the present study was designed with the following specific objectives: (i) to determine the prevalence of MetS and its individual IDF components in obese girls with PMOS compared to obese non-PMOS controls; (ii) to compare the degree of insulin resistance (HOMA-IR) between groups; (iii) to assess differences in anthropometric and biochemical metabolic parameters; and (iv) to identify independent predictors of MetS in obese adolescent girls using binary logistic regression, thereby testing the hypothesis that PMOS independently confers a significantly elevated MetS risk beyond that explained by obesity alone.[17-20]

MATERIALS AND METHODS

Study design and setting: A hospital-based comparative cross-sectional study was conducted in the Department of Dermatology and in collaboration with the Departments of Paediatrics and Radiology at Shantiniketan Medical College and Hospital (SMCH), Bolpur, West Bengal, India, from January 2023 to June 2024. Ethical clearance was obtained from the Institutional Ethics Committee (IEC/SMCH/2022/48), and written informed assent from participants and consent from parents/guardians were obtained prior to enrolment. The study was conducted in accordance with the Declaration of Helsinki.[18]. Participants: Eighty obese adolescent girls aged 10–18 years with sonographically confirmed PMOS (PMOS group) and 80 obese girls of matching age and BMI z-score without PMOS (non-PMOS control group) were enrolled, yielding a total sample of 160 participants. Inclusion criteria (PMOS group): female sex; age 10–18 years; obesity defined as BMI ≥95th percentile for age and sex per Cole et al. reference standards,[1] pelvic ultrasonography demonstrating PMOS by Adams criteria (≥12 follicles 2–9 mm diameter in one or both ovaries and/or ovarian volume >10 mL); willingness to participate with guardian consent. Inclusion criteria (control group): same age range, BMI z-score within ±0.5 SD of the PMOS group mean, absence of PMOS on pelvic USG. Exclusion criteria (both

groups): known or newly diagnosed type 1 or type 2 diabetes mellitus; established hypothyroidism or other endocrinopathy (Cushing's syndrome, congenital adrenal hyperplasia) on clinical/biochemical assessment; prior or current use of oral contraceptive pills, metformin, glucocorticoids, or other medications affecting metabolic parameters; pregnancy; severe systemic illness; incomplete data; and refusal to participate.[3,6,20] Sample size was calculated based on an estimated MetS prevalence of 33% in PCOS (Apridonidze et al,[11]) vs 15% in obese non-PCOS controls, at 80% power and 5% significance, requiring a minimum of 72 per group; 80 per group were enrolled for robustness.

Methods: Clinical and anthropometric assessment: A structured pro forma recorded clinical history (menstrual history, family history of PCOS or type 2 DM, dietary habits, physical activity levels) and full clinical examination including Tanner staging for pubertal assessment. Anthropometric measurements — body weight, height, waist circumference (WC) — were taken per WHO standards.[19] BMI z-score was calculated using WHO-recommended reference software. Waist circumference was measured at the mid-point between the lower costal margin and the iliac crest. Blood pressure was measured after 5 minutes rest in the sitting position, using a paediatric-appropriate cuff; two readings were averaged. Pubertal stage was recorded per Tanner criteria.[3] Pelvic ultrasonography: All participants underwent pelvic ultrasonography (transabdominal approach for pre-pubertal and early pubertal girls; transvaginal approach only in sexually active older adolescents with consent) performed on a Mindray DC-70 Pro ultrasound machine by a single experienced radiologist, blinded to metabolic status. PMOS was defined by Adams criteria: ≥12 follicles of 2–9 mm diameter in one or both ovaries and/or ovarian volume >10 mL, in the absence of a dominant follicle, corpus luteum, or luteinised cyst.[5] Laboratory investigations: After 12-hour overnight fast, venous blood samples were collected for: fasting plasma glucose (FPG) by glucose oxidase-peroxidase (GOD-POD) method; serum triglycerides (TG) by enzymatic colorimetric method; total cholesterol (TC); HDL-C by direct precipitation method; fasting serum insulin by chemiluminescence immunoassay (CLIA); and thyroid-stimulating hormone (TSH) by electrochemiluminescence immunoassay (ECLIA) to screen for and exclude hypothyroidism at enrolment. All assays were performed in the NABL-accredited Central Laboratory, SMCH, on the day of sample collection to ensure pre-analytical integrity. Insulin resistance was quantified by HOMA-IR = [Fasting insulin (μU/mL) × Fasting glucose (mmol/L)] / 22.5, as originally described by Matthews et al,[21] a cutoff ≥2.5 indicated insulin resistance.[13] MetS diagnosis: MetS was defined by IDF 2006 paediatric criteria: central obesity (WC ≥90th percentile for age and sex, or adult thresholds if ≥16 years: ≥80 cm in females) plus any two of: TG ≥150 mg/dL; HDL-C <40 mg/dL; SBP ≥130 or DBP ≥85 mmHg; FPG ≥100 mg/dL.[9]

Statistical analysis: Data were analysed using IBM SPSS Statistics version 25.0. Continuous variables are expressed as mean ± SD; categorical variables as frequencies (%). Between-group continuous comparisons were by Student's independent t-test; categorical comparisons by Chi-square or Fisher's exact test. Binary logistic regression identified independent MetS predictors, expressed as OR with 95% CI. $p<0.05$ was considered statistically significant.[10,20]

RESULTS

A total of 160 girls were analysed — 80 PMOS and 80 non-PMOS obese controls. Baseline demographic and anthropometric characteristics are presented in [Table 1]. The two groups were well matched for age (mean 14.2 ± 2.4 vs 13.9 ± 2.6 years, p=0.426), Tanner stage distribution (p=0.614), and BMI z-score (2.38 ± 0.42 vs 2.34 ± 0.39, p=0.512). Despite the matching, PMOS girls demonstrated significantly higher mean waist circumference (88.4 ± 8.6 vs 82.1 ± 7.9 cm, p<0.001), fasting plasma glucose (98.6 ± 14.8 vs 89.4 ± 11.2 mg/dL, p<0.001), serum triglycerides (168.4 ± 46.2 vs 138.8 ± 39.4 mg/dL, p<0.001), and HOMA-IR (3.24 ± 1.12 vs 2.36 ± 0.86, p<0.001), along with significantly lower mean HDL-C (44.8 ± 7.4 vs 50.2 ± 8.1 mg/dL, p<0.001) compared to non-PMOS controls. These differences were independent of matched BMI z-score and age, confirming the metabolic impact of PMOS as an entity distinct from obesity alone.[11,20]

Table 1: Baseline demographic and metabolic characteristics of PMOS and non-PMOS obese adolescent girls

Parameter	PMOS Group (n=80)	Non-PMOS Group (n=80)	p-value
Age, years (mean ± SD)	14.2 ± 2.4	13.9 ± 2.6	0.426
BMI z-score (mean ± SD)	2.38 ± 0.42	2.34 ± 0.39	0.512
Tanner Stage ≥3, n (%)	52 (65.0%)	50 (62.5%)	0.735
Waist Circumference, cm (mean ± SD)	88.4 ± 8.6	82.1 ± 7.9	<0.001*
FPG, mg/dL (mean ± SD)	98.6 ± 14.8	89.4 ± 11.2	<0.001*
Serum TG, mg/dL (mean ± SD)	168.4 ± 46.2	138.8 ± 39.4	<0.001*
HDL-C, mg/dL (mean ± SD)	44.8 ± 7.4	50.2 ± 8.1	<0.001*
SBP, mmHg (mean ± SD)	126.2 ± 12.8	118.4 ± 11.6	<0.001*
Fasting Insulin, μU/mL (mean ± SD)	18.4 ± 6.8	12.6 ± 5.2	<0.001*
HOMA-IR (mean ± SD)	3.24 ± 1.12	2.36 ± 0.86	<0.001*

*Statistically significant (p<0.05). SD = standard deviation; FPG = fasting plasma glucose; TG = triglycerides; HDL-C = high-density lipoprotein cholesterol; SBP = systolic blood pressure; HOMA-IR = Homeostatic Model Assessment for Insulin Resistance.

The prevalence of MetS and individual IDF component abnormalities are presented in Table 2. MetS was present in 32 of 80 PMOS girls (40.0%) compared to 14 of 80 non-PMOS controls (17.5%), a statistically highly significant difference (χ^2=9.90, p=0.002). Insulin resistance (HOMA-IR ≥2.5) was the most prevalent metabolic abnormality in PMOS girls, present in 55.0% of cases versus 25.0% of controls (p<0.001) — notably exceeding even the composite MetS prevalence, implying that IR is an earlier-emerging cardiometabolic marker than the composite MetS criterion in this population. Low HDL-C (50.0% vs 25.0%, p=0.001), hypertriglyceridaemia (45.0% vs 22.5%, p=0.002), elevated blood pressure (42.5% vs 20.0%, p=0.002), elevated FPG (37.5% vs 15.0%, p=0.002), and central obesity (60.0% vs 38.8%, p=0.006) all showed significantly higher prevalence in PMOS girls.[11-13]

Table 2: Prevalence of metabolic syndrome, insulin resistance, and IDF components in PMOS versus non-PMOS obese girls

Parameter	PMOS n=80 (%)	Non-PMOS n=80 (%)	χ^2	p-value
Metabolic Syndrome (IDF 2006)	32 (40.0%)	14 (17.5%)	9.90	0.002*
Insulin Resistance (HOMA-IR ≥2.5)	44 (55.0%)	20 (25.0%)	15.00	<0.001*
Central Obesity (WC ≥90th centile)	48 (60.0%)	31 (38.8%)	7.25	0.006*
Low HDL Cholesterol	40 (50.0%)	20 (25.0%)	10.67	0.001*
Hypertriglyceridaemia	36 (45.0%)	18 (22.5%)	9.08	0.002*
Elevated Blood Pressure	34 (42.5%)	16 (20.0%)	9.42	0.002*
Elevated Fasting Plasma Glucose	30 (37.5%)	12 (15.0%)	10.46	0.001*

*Statistically significant (p<0.05). IDF = International Diabetes Federation; HOMA-IR = Homeostatic Model Assessment for Insulin Resistance; WC = waist circumference; HDL = high-density lipoprotein.

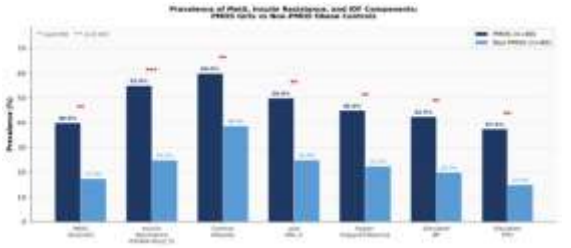

Figure 1: Prevalence of metabolic syndrome, insulin resistance, and IDF components in PMOS versus non-PMOS obese adolescent girls.

Comparison of mean HOMA-IR values between PMOS and non-PMOS girls by age subgroup [Table 3] revealed consistently and significantly elevated HOMA-IR values in PMOS girls across all age categories (10–12 years, 13–15 years, and 16–18 years), with the most pronounced absolute difference in the 13–15 year age group (3.42 ± 1.18 vs 2.28 ± 0.92, p<0.001). This pattern suggests that the greatest metabolic divergence between PMOS and non-PMOS girls occurs during mid-adolescence, a hormonally dynamic period characterised by physiological transient insulin resistance of puberty (Tanner stages 2–3), which may be further exacerbated by PMOS-associated hyperinsulinaemia.[14,16]

Table 3: Mean HOMA-IR comparison between PMOS and non-PMOS girls by age subgroup

Age Subgroup	PMOS HOMA-IR (mean ± SD)	Non-PMOS HOMA-IR (mean ± SD)	t-value	p-value
10–12 years (n=40)	2.96 ± 0.98	2.18 ± 0.76	4.38	<0.001*
13–15 years (n=66)	3.42 ± 1.18	2.28 ± 0.92	5.32	<0.001*
16–18 years (n=54)	3.18 ± 1.08	2.52 ± 0.88	3.14	0.002*
Overall (n=160)	3.24 ± 1.12	2.36 ± 0.86	5.46	<0.001*

*Statistically significant (p<0.05). HOMA-IR = Homeostatic Model Assessment for Insulin Resistance; SD = standard deviation.

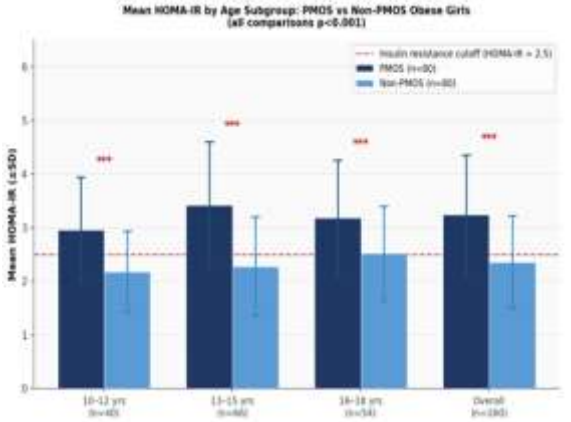

Figure 2: Mean HOMA-IR comparison between PMOS and non-PMOS obese girls stratified by age subgroup (error bars = ±1 SD; all comparisons p<0.001).

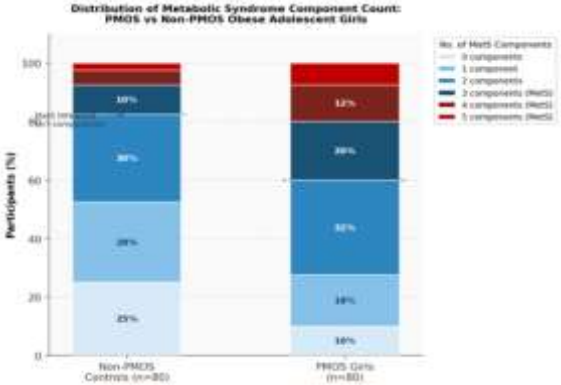

Figure 3: Distribution of metabolic syndrome component count in PMOS versus non-PMOS obese adolescent girls; dashed line marks MetS threshold (≥3 components).

Binary logistic regression analysis (Table 4), adjusting for age and BMI z-score, identified PMOS status (OR=3.12, 95% CI: 1.52–6.39, p=0.002) and BMI z-score (OR=1.86, 95% CI: 1.23–2.81, p=0.003) as the only statistically significant independent predictors of MetS in this obese adolescent cohort. Age and Tanner stage were not significant independent predictors after adjustment (p=0.384 and p=0.276 respectively). The Hosmer-Lemeshow test confirmed good model fit (χ^2=6.92, p=0.547). Nagelkerke R^2=0.386. The OR of 3.12 for PMOS status signifies that obese girls with PMOS carry more than three times the adjusted risk of MetS compared to equally obese non-PMOS peers, establishing PMOS as a clinically significant, independent cardiometabolic risk phenotype in this age group.[11,12,20]

Table 4: Binary logistic regression — Independent predictors of metabolic syndrome in obese adolescent girls (n=160)

Predictor Variable	B coefficient	Odds Ratio (OR)	95% CI	p-value
PMOS Status (vs non-PMOS)	1.139	3.12	1.52–6.39	0.002*
BMI z-score (per unit increase)	0.620	1.86	1.23–2.81	0.003*
Age (per year)	0.104	1.11	0.87–1.41	0.384
Tanner Stage (≥3 vs <3)	0.284	1.33	0.80–2.20	0.276

*Statistically significant (p<0.05). OR = odds ratio; CI = confidence interval; BMI = body mass index; PMOS = polycystic morphology on sonography. Hosmer-Lemeshow χ^2=6.92, p=0.547; Nagelkerke R^2=0.386.

DISCUSSION

The present study provides robust, statistically compelling evidence that PMOS in obese adolescent girls is an independent and clinically significant predictor of early-onset metabolic syndrome and insulin resistance — findings with important implications for paediatric endocrinology, adolescent gynaecology, preventive cardiology, and primary care practice in India and the broader South Asian region.

The MetS prevalence of 40.0% in PMOS girls in the present study is notable given that the comparison group was equally obese, suggesting that PMOS confers a metabolic risk increment beyond that attributable to obesity alone. This is consistent with — and in fact somewhat higher than — findings from adult PCOS literature: Apridonidze et al,[11] documented MetS in 33.4% of women with fully expressed PCOS using modified NCEP-ATP III criteria; Azziz et al,[12] similarly reported a 33.4% MetS prevalence in an unselected PCOS population. The higher prevalence in our PMOS cohort — despite the phenotypically less severe entity — is likely explained by the adolescent setting, where physiological pubertal insulin resistance,[14] operates synergistically with PMOS-driven hyperinsulinaemia to produce a compounded metabolic insult. The South Asian metabolic phenotype — characterised by greater visceral adiposity, elevated visceral-to-subcutaneous fat ratio, and more pronounced insulin resistance at equivalent BMI levels — further amplifies this risk, as documented by Misra and Khurana,[2] in their comprehensive analysis of metabolic phenotyping in developing country populations. The IDF 2006 paediatric criteria,[9] appropriately capture this by applying age- and sex-specific waist circumference percentile thresholds rather than fixed values, which is particularly relevant in the 10–15 year age subgroup.

The finding that insulin resistance (HOMA-IR ≥2.5) was present in 55.0% of PMOS girls — a prevalence exceeding that of composite MetS (40.0%) by 15 percentage points — is of considerable clinical significance. This temporal hierarchy, in which IR precedes and drives composite MetS, was mechanistically proposed by Reaven,[13] in his foundational description of syndrome X, and is supported by Moran et al,[8] who documented that impaired glucose tolerance and IR frequently predate composite MetS in PCOS-spectrum populations. The mechanistic pathway from hyperinsulinaemia to PMOS in obese adolescents operates through multiple axes: elevated insulin levels directly stimulate ovarian thecal cell androgen synthesis, upregulate LH pulsatility, suppress sex hormone-binding globulin (SHBG), and promote stromal hypertrophy — collectively impairing follicular maturation and producing the characteristic polycystic ovarian morphology even in girls who do not yet meet full Rotterdam criteria for PCOS.[3,6] Carmina and Lobo,[15] specifically recommended routine HOMA-IR measurement in at-risk adolescent female populations, noting that it is a more sensitive and earlier-emerging marker of cardiometabolic risk than the composite MetS threshold in this age group — a recommendation strongly endorsed by the present findings. The consistently and significantly elevated HOMA-IR in PMOS girls across all three age subgroups (10–12, 13–15, 16–18 years) establishes that PMOS-associated insulin resistance is not a developmental artefact confined to the peak physiological IR window of Tanner stages 2–3 (Reaven), but rather persists across the full adolescent age spectrum in this population.

The independent OR of 3.12 for PMOS status as a predictor of MetS — after adjusting for age and BMI z-score — is the most clinically important quantitative finding of the present study. This magnitude of association is consistent with findings by Ehrmann,[4] in adult PCOS populations, and with Bremer's,[16] advocacy for early paediatric PCOS-spectrum screening. The fact that age and Tanner stage did not achieve independent significance after adjustment confirms that PMOS status and BMI z-

score, rather than pubertal progression per se, are the primary metabolic risk determinants — a finding that mandates immediate metabolic work-up on PMOS detection, rather than watchful waiting for PCOS evolution.

Strengths and Limitations: Strengths: The matched design (age and BMI z-score) isolates the independent contribution of PMOS to MetS risk beyond obesity. Ultrasonography was performed by a single blinded radiologist using a standardised protocol, minimising operator variability. Age-appropriate IDF paediatric criteria were applied, including WC percentile thresholds for younger adolescents. NABL-accredited laboratory processing on the day of collection assured assay integrity. TSH was measured at enrolment to exclude hypothyroidism, a key confounder of insulin resistance and ovarian dysfunction. Limitations: The cross-sectional design precludes causal inference. The single-centre setting limits demographic generalisability. Sex hormone assays (total testosterone, LH/FSH ratio, DHEA-S, SHBG) were not performed, precluding full hormonal phenotyping and formal PCOS classification per Rotterdam criteria. Oral glucose tolerance testing (OGTT) was not conducted; OGTT would have identified impaired glucose tolerance earlier and more sensitively than FPG alone, potentially increasing the documented metabolic burden further. Dietary intake and physical activity were not objectively quantified by validated instruments. The HOMA-IR cutoff of ≥2.5, while widely used, was originally validated in adult populations; paediatric-specific cutoffs remain debated in the literature. Multi-centre prospective studies with full hormonal profiling, OGTT, and longitudinal follow-up are needed to confirm these findings and evaluate whether early metabolic intervention modifies the cardiometabolic trajectory in PMOS-positive obese girls.[18,19]

CONCLUSION

The present hospital-based comparative cross-sectional study provides robust and clinically actionable evidence that polycystic morphology on sonography (PMOS) in obese adolescent girls is an independent and statistically significant predictor of early-onset metabolic syndrome, conferring more than three times the adjusted risk of MetS compared to equally obese girls without PMOS — a magnitude of association that firmly establishes PMOS as a clinically significant cardiometabolic risk phenotype in its own right, beyond its sonographic definition and independent of the degree of obesity. The 40.0% MetS prevalence in PMOS girls, the 55.0% prevalence of insulin resistance, and the significantly elevated HOMA-IR values across all age subgroups collectively delineate a population of obese adolescent girls with PMOS as a high-risk metabolic subgroup requiring proactive clinical identification and structured metabolic intervention. The finding that insulin resistance, as measured by HOMA-IR ≥2.5, exceeds composite MetS prevalence in PMOS girls by a clinically meaningful margin reinforces the conclusion that HOMA-IR is a more sensitive, earlier-emerging cardiometabolic marker than the composite metabolic syndrome criterion in this age group, and provides strong empirical support for the routine inclusion of fasting insulin measurement and HOMA-IR calculation in the metabolic work-up of all obese girls with sonographically confirmed PMOS. The logistic regression data further confirm that BMI z-score, as an independent predictor alongside PMOS status, underscores the synergistic — rather than merely additive — relationship between adiposity and PMOS in driving early MetS, a relationship that may be particularly pronounced in the South Asian population owing to the well-documented propensity for greater visceral adiposity, earlier insulin resistance, and more aggressive atherogenic dyslipidaemia at equivalent BMI z-scores compared to Western counterparts. In light of these findings, the following practical recommendations are advanced for immediate implementation in clinical and public health practice. All obese adolescent girls undergoing pelvic ultrasonography for any indication should be formally assessed for PMOS using standardised Adams criteria, and those with confirmed PMOS should be referred immediately for a comprehensive metabolic assessment comprising HOMA-IR, fasting lipid profile (total cholesterol, TG, HDL-C, LDL-C), fasting plasma glucose, blood pressure, and waist circumference measurement — regardless of whether they fulfil full Rotterdam criteria for PCOS, as PMOS itself confers independent MetS risk. Intensive, structured lifestyle modification — encompassing individually tailored dietary caloric restriction, aerobic and resistance physical activity programmes, and behavioural counselling — should be initiated without delay in all PMOS-positive obese girls with documented insulin resistance or any MetS component, given the well-established reversibility of early metabolic abnormalities in adolescence with timely intervention. Paediatric endocrinologists, gynaecologists, and paediatricians in tertiary and secondary care institutions should establish formal PMOS-MetS screening protocols, particularly in high-risk populations in urban and semi-urban India where adolescent obesity prevalence is rising rapidly. Multidisciplinary care teams — bringing together paediatricians, endocrinologists, gynaecologists, dietitians, and clinical psychologists — should be constituted in tertiary care centres to provide integrated, holistic management for obese adolescent girls with PMOS and metabolic comorbidities, addressing both the somatic and the psychosocial dimensions of this complex condition. Finally, public health programmes should leverage school-health platforms, adolescent-health clinics, and primary care networks for early identification of obese adolescent girls at risk for PMOS and associated metabolic syndrome, ensuring that screening and preventive intervention penetrate beyond the

specialist centre into the community where the burden of adolescent obesity is greatest. In summary, the present study compellingly positions PMOS in obese adolescent girls at the intersection of endocrine dysregulation, insulin resistance, and early-onset cardiometabolic vulnerability, mandating its recognition as a pivotal target for early metabolic risk stratification, preventive intervention, and integrated paediatric-endocrinological-gynaecological care.

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