

Cardiometabolic risk profile of women with Polyendocrine Metabolic Ovarian Syndrome in Cameroon: a cross-sectional study

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Abstract

Background

Polyendocrine metabolic ovarian syndrome (PMOS) carries a substantial cardiometabolic burden, but its distribution across composite lipid/glycemic indices, capture by conventional global cardiovascular risk scores, and relationship to PMOS phenotype and hyperandrogenism severity remain incompletely characterized, particularly in Sub-Saharan Africa. We examined the cardiometabolic risk profile of Cameroonian women with PMOS, its association with phenotype, and its relationship to hirsutism severity.

Methods

We conducted a descriptive analytical cross-sectional study with prospective data collection over six months (January–June 2026) in two referral hospitals in Yaoundé, enrolling 69 women aged 18–45 with PMOS diagnosed by Rotterdam (2003) criteria. Four cardiometabolic indices – triglyceride-glucose (TyG) index, atherogenic index of plasma (AIP), LDL/HDL-cholesterol ratio, and non-HDL cholesterol – were categorized using standard thresholds, and the Framingham 10-year cardiovascular risk score was calculated for comparison. Phenotype (A vs. non-A) and metabolic syndrome association was assessed by logistic regression. Hirsutism severity (modified Ferriman–Gallwey, mFG) was correlated with the four indices using Spearman's and partial correlation adjusting for BMI.

Results

A high-risk AIP (> 0.21) was present in 53.6% of participants and elevated non-HDL cholesterol (≥ 130 mg/dL) in 66.7%, while probable insulin resistance by TyG (> 8.5) occurred in 23.2% and an elevated LDL/HDL ratio (> 3.5) in 8.7%. The Framingham score classified 98.6% as mild risk ($< 10\%$), reflecting the cohort's young age rather than absent cardiometabolic burden. Metabolic syndrome was present in 43.5% (30/69); women with complete Rotterdam phenotype (A) had higher odds of metabolic syndrome than non-A phenotype (49.0% vs. 27.8%), though not significant (OR = 2.50, 95% CI 0.78–8.04, $p = 0.124$). Among 61 participants with recorded mFG scores, none of the four indices correlated significantly with hirsutism severity (Spearman ρ 0.10–0.23, all $p \geq 0.075$), remaining non-significant after BMI adjustment.

Conclusion

Cameroonian women with PMOS carry a substantial cardiometabolic burden poorly captured by a conventional, age-driven global risk score, trending toward greater severity in the complete (Phenotype A) presentation, though not significantly in this cohort. Hirsutism severity was not associated with this

burden. These findings support routine use of composite lipid/glycemic indices, rather than global age-based risk scores or hirsutism severity alone, applied uniformly across PMOS phenotypes.

Background

Polyendocrine metabolic ovarian syndrome (PMOS) previously known as polycystic ovary syndrome affects about one of every 8 women in the world[1]. The term PCOS is misleading, as it emphasizes ovarian cysts, a feature that is neither universal nor pathognomonic while overshadowing the syndrome's broader endocrine and metabolic dimensions. This mismatch between name and pathophysiology contributes to diagnostic delay, fragmented clinical management, and patient stigma, and further limits how the condition is framed in research and policy contexts. Beyond its reproductive manifestations with oligo-anovulation, hyperandrogenism, and infertility, PMOS carries a substantial and often under-recognized metabolic burden. Insulin resistance is now considered a central pathophysiological feature of the syndrome, present in the majority of affected women regardless of body weight, and drives downstream abnormalities spanning glucose homeostasis, lipid metabolism, blood pressure regulation, and body fat distribution[2, 3].

This metabolic burden is multidimensional: it manifests clinically, through hypertension, obesity, and cutaneous markers of insulin resistance such as acanthosis nigricans, and biochemically, through dyslipidemia [4, 5], dysglycemia, and composite indices such as the triglyceride-glucose (TyG) index and the atherogenic plasma index (AIP), both of which have emerged as accessible, low-cost surrogates for insulin resistance and atherogenic risk in settings where direct assays (HOMA-IR, fasting insulin) remain difficult to access [6–8].

Because androgen excess is hypothesized to contribute mechanistically to these metabolic derangements through effects on visceral adiposity, hepatic lipid handling, and insulin signalling, it has been suggested that the clinical severity of hyperandrogenism, most readily assessed at the bedside via the modified Ferriman–Gallwey (mFG) score, could itself serve as a simple clinical marker of cardiometabolic risk in PMOS, and prior work has specifically examined the association between the mFG score and metabolic syndrome [9]. Evidence for this relationship is, however, mixed: other studies find that androgen markers assessed clinically (via the mFG score) and biochemically (via lipid and androgen assays) segregate largely independently, with the mFG score showing no significant association with hyperandrogenism status despite triglycerides and androgens themselves being correlated [10]. Clarifying whether hirsutism severity tracks with cardiometabolic risk has direct clinical relevance in resource-limited settings, where it could either justify prioritizing metabolic screening in women with more severe hyperandrogenism or, if no such relationship exists, reinforce the need for universal screening regardless of androgenic phenotype.

In Cameroon, prior work has established the prevalence and phenotypic distribution of PMOS among women attending gynaecological clinics in Yaoundé [11], and has explored insulin sensitivity [12] in affected women. However, whether hirsutism severity tracks with cardiometabolic risk in this population

has not been examined, and a comprehensive description spanning the sociodemographic, clinical, and biochemical metabolic profile of this population, bringing together anthropometric, blood pressure, cutaneous, and biochemical findings in a single, integrated picture, has not previously been reported. This study aimed to characterize the cardiometabolic risk profile of Cameroonian women with PMOS using composite indices and a conventional global risk score, to test whether this risk is associated with PMOS phenotype, and to determine whether hirsutism severity itself tracks with cardiometabolic risk indices, as a basis for informing routine metabolic screening practices in this population.

Methods

Study type, population, and setting

We conducted a descriptive analytical cross-sectional study with prospective data collection over a six-month period, from January to June 2026, in two referral hospitals in Yaoundé: the Yaoundé Gynaecology, Obstetrics and Paediatrics Hospital and the Catholic Mother and Child Hospital Deo Gratias. Biochemical analyses were performed at the PRIMA SARL Laboratory. The study population consisted of women aged 18–45 years diagnosed with polyendocrine metabolic ovarian syndrome (PMOS) who consulted in the gynaecology and/or endocrinology departments during the study period.

Eligibility criteria and sampling

Of 102 women with PMOS identified during the study period, 95 had available contact information; 83 were successfully contacted, of whom 4 declined and 8 were unavailable. Of the 71 initially enrolled, 2 were excluded (pregnancy, $n = 1$; early postpartum, $n = 1$), leaving 69 women in the final analysis. Eligible participants met the Rotterdam (2003) diagnostic criteria for PMOS[13] and provided informed consent; pregnancy, breastfeeding, severe chronic illness, and cognitive impairment were exclusion criteria. This was an exhaustive sample of eligible PCOS patients at the centers of study over the study period.

Data collection and laboratory procedures

This study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee of the Université des Montagnes (Authorization No. 2026/062/UdM/PR/CEAQ) and the Institutional Ethics Committee for Human Health Research (CIERSH) of the Yaoundé Gynaeco-Obstetric and Pediatric Hospital (Authorization No. 369/CIERSH/DM/2026), with administrative authorizations from participating hospitals and laboratories. Administrative authorization to conduct the study at the Catholic Mother and Child Hospital Deo Gratias (Emana) was granted by Afrique Future, the humanitarian organization responsible for the hospital, on 26 January 2026. Each participant underwent a structured interview capturing sociodemographic characteristics, gynaeco-obstetric history, personal and family medical history, and lifestyle/toxic habits, followed by clinical examination including anthropometry (weight, height, waist circumference), 24-hour ambulatory

blood pressure monitoring, and assessment of acanthosis nigricans severity at the nape of the neck and axillae, with hirsutism assessed using the modified Ferriman–Gallwey (mFG) score[9].

Venous blood (10 mL) was drawn by antecubital venipuncture under aseptic conditions after a minimum 12-hour fast, and distributed into a dry tube (lipid profile) and a fluoride tube (fasting glucose). Samples were transported to the laboratory in a cold box within 2 hours of collection and centrifuged at 3,000 rpm for 5 minutes to separate serum. Fasting glucose, total cholesterol, and triglycerides were measured by enzymatic colorimetric methods (glucose oxidase-peroxidase for glucose, read at 500 nm; cholesterol esterase-oxidase-peroxidase for total cholesterol, read at 510 nm; lipase-based hydrolysis with a peroxidase-coupled endpoint for triglycerides, read at 500 nm), each run against a reagent blank and calibrator. HDL-cholesterol was measured after phosphotungstic acid-magnesium chloride precipitation of apolipoprotein-B-containing lipoproteins, with the same enzymatic method applied to the supernatant (read at 578 nm); LDL-cholesterol was calculated using the Friedewald formula (valid for triglycerides < 340 mg/dL). The TyG index was calculated as $\ln[\text{fasting triglycerides (mg/dL)} \times \text{fasting glucose (mg/dL)} / 2]$, and the atherogenic plasma index (AIP) as $\log_{10}[\text{triglycerides (mg/dL)} / \text{HDL-cholesterol (mg/dL)}]$ [14, 15]. Two further lipid-derived indices were calculated for the hirsutism-cardiometabolic risk analysis: the LDL/HDL-cholesterol ratio ($\text{LDL-cholesterol} \div \text{HDL-cholesterol}$) and non-HDL cholesterol ($\text{total cholesterol} - \text{HDL-cholesterol}$), both simple, assay-free markers of atherogenic risk requiring no additional laboratory measurement beyond the standard lipid panel.

Definitions and statistical analysis

Hypertension was defined using the final ambulatory blood pressure monitoring status (permanent, controlled, or masked hypertension classified as hypertensive; white-coat hypertension and normotension classified as non-hypertensive). Fasting glucose and lipid profile values were categorized per WHO thresholds (normoglycemia < 100 mg/dL; impaired fasting glucose 100–125 mg/dL; diabetes $\geq$ 126 mg/dL). Triglycerides were considered elevated at $\geq$ 150 mg/dL, HDL-cholesterol low at < 50 mg/dL, LDL-cholesterol elevated at $\geq$ 130 mg/dL, and total cholesterol elevated at $\geq$ 200 mg/dL.

PMOS phenotypes were classified according to the three Rotterdam (2003) diagnostic criteria recorded for each participant (clinical or biological hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology on ultrasound): Phenotype A = all three criteria present; Phenotype B = hyperandrogenism and ovulatory dysfunction without polycystic morphology; Phenotype C = hyperandrogenism and polycystic morphology without ovulatory dysfunction; Phenotype D = ovulatory dysfunction and polycystic morphology without hyperandrogenism (non-hyperandrogenic). For the metabolic syndrome analysis, Phenotype A was compared against the three non-A phenotypes combined; this dichotomization was adopted both because the individual B, C, and D subgroups were too small ($n = 5, 7, \text{ and } 6$, respectively) to support statistically meaningful separate comparisons, and because Phenotype A, representing the complete Rotterdam triad, is consistently identified in the literature as the most metabolically severe phenotype [18], making an A-versus-non-A grouping both analytically tractable and clinically interpretable.

Metabolic syndrome was defined using the 2009 Harmonized criteria (≥ 3 of the following 5: abdominal obesity [waist circumference ≥ 80 cm], elevated blood pressure [$\geq 130/85$ mmHg] or treated/known hypertension, elevated fasting glucose [≥ 100 mg/dL] or known diabetes, hypertriglyceridemia [≥ 150 mg/dL], and low HDL-cholesterol [< 50 mg/dL]). For descriptive purposes, each of the four cardiometabolic indices was additionally categorized: the TyG index as < 8.0 (insulin resistance unlikely), $8.0-8.5$ (moderate risk), or > 8.5 (probable insulin resistance); the AIP as ≤ 0.11 (low), $0.11-0.21$ (moderate), or > 0.21 (high) risk; the LDL/HDL-cholesterol ratio as < 2.5 (low), $2.5-3.5$ (moderate), or > 3.5 (high) risk; and non-HDL cholesterol as ≤ 100 mg/dL (low), $100-129$ mg/dL (moderate), or ≥ 130 mg/dL (high) risk. The association between PMOS phenotype (Phenotype A vs. non-A) and metabolic syndrome was assessed by univariate logistic regression, fitted by maximum likelihood (iteratively reweighted least squares).

As a global comparator, the Framingham General Cardiovascular Disease 10-year risk score was calculated for each participant using the lab-based, sex-specific algorithm of D'Agostino et al. (2008)[17], incorporating age, total cholesterol, HDL-cholesterol, systolic blood pressure, antihypertensive treatment status, current smoking, and diabetes status. Risk was categorized as mild ($< 10\%$), moderate (10 to $< 20\%$), or severe ($\geq 20\%$), consistent with conventional Framingham risk strata.

For the primary analysis, the continuous mFG score was correlated with each of the four cardiometabolic indices (TyG index, AIP, LDL/HDL ratio, non-HDL cholesterol) using Spearman's rank correlation coefficient (ρ), given the non-normal distribution of the mFG score (Shapiro–Wilk $p = 0.006$). Partial Spearman correlations adjusting for BMI were additionally computed to account for potential confounding by adiposity. A post-hoc power calculation determined the minimum correlation coefficient detectable with 80% power at the achieved sample size.

Statistical analyses were performed using SPSS version 26. Data are presented as frequencies and percentages for categorical variables and as mean $\pm$ standard deviation or median [interquartile range] for continuous variables, as appropriate to their distribution.

Patient and Public Involvement

Patients and members of the public were not involved in the design, conduct, reporting, or dissemination of this study. Their role was limited to participation as study subjects. Future research in Cameroon may benefit from involving patient advocacy groups and community representatives in study design and dissemination to enhance relevance and impact.

Results

Cardiometabolic risk profile of the cohort

Table 2 presents the distribution of each cardiometabolic index across its own specific, clinically defined risk categories in the full cohort (n=69). A high-risk atherogenic index of plasma (AIP >0.21) was the most prevalent biochemical abnormality, found in 53.6% of participants, and elevated non-HDL cholesterol (≥ 130 mg/dL) was present in 66.7%. Probable insulin resistance by the TyG index (>8.5) was found in 23.2% of the cohort, while an elevated LDL/HDL ratio (>3.5) was comparatively uncommon (8.7%). For a global comparator, the Framingham General Cardiovascular Disease 10-year risk score was also calculated for each participant. Nearly all participants fell in the mild risk category (<10%, 98.6%), reflecting the cohort's young age; only one participant (1.5%) had a severe 10-year risk ($\geq 20\%$), illustrating that this widely used, age-driven global risk formula does not capture the elevated cardiometabolic burden already detectable through the composite lipid/glycemic indices above. Metabolic syndrome, the most widely recognized composite cardiometabolic diagnosis, was present in 43.5% of the cohort (30/69)

Table 2. Categorical distribution of cardiometabolic risk indices in the study population (N=69)

Cardiometabolic index	Risk category	n	%
TyG index	<8.0 (insulin resistance unlikely)	21	30.4
	8.0–8.5 (moderate risk)	32	46.4
	>8.5 (probable insulin resistance)	16	23.2
AIP	≤ 0.11 (low risk)	21	30.4
	0.11–0.21 (moderate risk)	11	15.9
	>0.21 (high risk)	37	53.6
LDL/HDL ratio	<2.5 (low risk)	41	59.4
	2.5–3.5 (moderate risk)	22	31.9
	>3.5 (high risk)	6	8.7
Non-HDL cholesterol (mg/dL)	≤ 100 (low risk)	1	1.4
	100–129 (moderate risk)	22	31.9
	≥ 130 (high risk)	46	66.7
Framingham 10-year CVD risk* (n=69)	Mild (<10%)	68	98.6
	Moderate (10–<20%)	0	0.0
	Severe ($\geq 20\%$)	1	1.4
Metabolic syndrome	Present (≥ 3 of 5 criteria)	30	43.5
	Absent	39	56.5

TyG = triglyceride-glucose index; AIP = atherogenic index of plasma (Dobiášová’s classification). LDL/HDL ratio and non-HDL cholesterol categorized as described in Methods. *Framingham General Cardiovascular Disease 10-year risk score (D’Agostino et al., 2008), lab-based sex-specific algorithm incorporating age, total cholesterol, HDL-cholesterol, systolic blood pressure, antihypertensive treatment, smoking, and diabetes status[17]. Metabolic syndrome defined per the 2009 Harmonized criteria(≥ 3 of 5: abdominal obesity, elevated blood pressure or treatment, elevated fasting glucose or known diabetes, hypertriglyceridemia, low HDL-cholesterol)

PMOS phenotype and metabolic syndrome

Table 3 presents the univariate logistic regression of PMOS phenotype (Phenotype A vs. the three non-A phenotypes combined) predicting metabolic syndrome and, separately, each of the four individual cardiometabolic indices and metabolic syndrome (dichotomized at the high-risk threshold defined in Table 2). Women with Phenotype A had higher odds of metabolic syndrome than those with a non-A phenotype (49.0% [25/51] vs. 27.8% [5/18]), though this did not reach statistical significance (OR = 2.50, 95% CI 0.78–8.04, $p = 0.124$). In contrast, none of the four individual indices showed a meaningful association with Phenotype A (all OR 0.68–1.71, all $p \geq 0.53$), suggesting that this modest, non-significant trend toward higher metabolic syndrome in Phenotype A is not attributable to any single dominant component but instead reflects, at most, a diffuse aggregate effect across the composite outcome.

Table 3. Univariate logistic regression: PMOS phenotype (A vs. non-A), metabolic syndrome, and individual cardiometabolic risk indices

Outcome	OR	95% CI	p-value
Metabolic syndrome	2.50	0.78–8.04	0.124
TyG index >8.5 (probable IR)	1.71	0.43–6.87	0.533
AIP >0.21 (high risk)	0.90	0.31–2.65	1.000
LDL/HDL ratio >3.5 (high risk)	0.68	0.11–4.08	0.647
Non-HDL cholesterol ≥ 130 mg/dL (high risk)	1.00	0.32–3.13	1.000

OR = odds ratio; CI = confidence interval. Logistic regression fitted by maximum likelihood (iteratively reweighted least squares); Wald 95% CI and p-value reported. Non-A phenotypes (B, C, D combined) serve as the reference category throughout. High-risk thresholds for the individual indices are as defined in Table 2 (TyG index >8.5; AIP >0.21; LDL/HDL ratio >3.5; non-HDL cholesterol ≥ 130 mg/dL).

Because Phenotypes B and C, like Phenotype A, both require the presence of hyperandrogenism (only Phenotype D does not), the non-A comparison group above is not a clean hyperandrogenic vs. non-hyperandrogenic contrast: 12 of the 18 non-A women (Phenotypes B and C combined) are themselves hyperandrogenic, and only 6 (Phenotype D) are not. As a sensitivity analysis, we therefore repeated the

comparison grouping strictly by hyperandrogenism status: all hyperandrogenic phenotypes combined (A+B+C, n=63) versus the sole non-hyperandrogenic phenotype (D, n=6). Metabolic syndrome prevalence was 42.9% (27/63) in the hyperandrogenic group versus 50.0% (3/6) in the non-hyperandrogenic group (OR=0.75, 95% CI 0.14–4.01, p=1.000) – a direction opposite to what would be expected if hyperandrogenism itself were driving metabolic risk. This indicates that the null phenotype-metabolic syndrome association reported above is not attributable to hyperandrogenism being diluted across the non-A comparison group; isolating hyperandrogenism status directly did not reveal a hidden association. Given the very small size of Phenotype D (n=6), however, this sensitivity analysis should be interpreted as further evidence against, rather than definitive refutation of, a hyperandrogenism-metabolic syndrome link in this cohort.

Hirsutism severity and cardiometabolic risk indices

Hirsutism was reported by 88.4% of participants, and a modified Ferriman-Gallwey (mFG) score was recorded in 61 (mean 11.3±4.7, range 4-25); 35 (57.4%) fell in the mild range (mFG 8-15) and 13 (21.3%) in the moderate range (mFG 16-25), with no participant scoring in the severe range (>25).

Figure 1 presents the correlation between the continuous mFG score and each of the four cardiometabolic indices examined. None reached statistical significance: the TyG index showed the strongest, though still non-significant, association (Spearman $\rho=0.23$, $p=0.075$), while the AIP, the LDL/HDL ratio, and non-HDL cholesterol showed weak, non-significant correlations ($|\rho|$ 0.10-0.15, all $p\geq 0.25$). Adjusting for BMI did not materially change these estimates (partial ρ 0.07-0.21, all $p\geq 0.10$).

A post-hoc power calculation indicated that, at the achieved sample size (n=61), this analysis had 80% power to detect only correlations of $|\rho|\geq 0.35$; achieved power to detect the observed TyG index correlation ($\rho=0.23$) was approximately 43%. These findings should therefore be interpreted as showing no evidence of a moderate-or-larger association between hirsutism severity and cardiometabolic risk indices in this cohort, rather than definitive proof of no association.

Discussion

The cardiometabolic risk profile of this cohort illustrates a recurring limitation of conventional, age-driven global cardiovascular risk scores when applied to PMOS: despite a substantial burden of atherogenic dyslipidemia (high-risk AIP in 53.6%, elevated non-HDL cholesterol in 66.7%) and probable insulin resistance (TyG index > 8.5 in 23.2%), the Framingham 10-year risk score classified 98.6% of participants as mild risk. This divergence is expected given the cohort's young age, since age enters the Framingham algorithm as a dominant weighted term [17]. This aligns with broader recognition that traditional scores such as the Framingham score can underestimate cardiovascular risk in women generally, and specifically in women with PCOS, who carry risk factors not fully captured by these age-driven algorithms [21]. Practically, this carries a message: relying on a global, age-based score to triage cardiometabolic risk in young women with PMOS would systematically miss the burden already

detectable through composite lipid/glycemic indices, reinforcing the case for supplementing, rather than substituting for, such scores with PMOS-specific composite markers in this population [3].

Phenotype A, representing the complete Rotterdam triad, showed a near two-fold higher odds of metabolic syndrome than the non-A phenotypes combined (49.0% vs. 27.8%; OR = 2.50, 95% CI 0.78–8.04, $p = 0.124$), directionally consistent with Phenotype A being widely reported as the most metabolically severe PMOS phenotype [18]. This association did not reach statistical significance in this cohort, most plausibly reflecting limited statistical power given the modest combined size of the non-A subgroup ($n = 18$, pooling Phenotypes B, C, and D); a substantially larger sample would be needed to confirm or refute a true phenotype-specific difference in metabolic syndrome risk. A sensitivity analysis regrouping strictly by hyperandrogenism status (all hyperandrogenic phenotypes, A + B+C, versus the sole non-hyperandrogenic phenotype, D) found no evidence that this null result reflects dilution of a true Phenotype A effect within the non-A comparison group; if anything, the non-hyperandrogenic group trended toward higher, not lower, metabolic syndrome prevalence, though this comparison was severely underpowered (Phenotype D, $n = 6$). This contrasts with larger studies using biochemically confirmed hyperandrogenemia, which have found hyperandrogenic PCOS phenotypes to carry a significantly higher risk of insulin resistance than non-hyperandrogenic phenotypes [19], a discrepancy that may reflect this study's clinical, rather than biochemical, ascertainment of hyperandrogenism (see Limitations) combined with limited statistical power. Notably, when the four individual cardiometabolic indices were tested against Phenotype A separately, none showed a meaningful association (all OR 0.68–1.71, all $p \geq 0.53$), indicating that the modest phenotype-metabolic syndrome trend observed here is not driven by any single component but reflects, at most, a diffuse effect distributed across the composite outcome.

Clinical hirsutism severity, however, was not significantly associated with any of the four cardiometabolic risk indices examined (the TyG index, the atherogenic index of plasma, the LDL/HDL ratio, or non-HDL cholesterol) whether analyzed continuously or adjusted for BMI. This aligns with the view that clinical hyperandrogenism and cardiometabolic risk, while both core features of PMOS, may reflect at least partially distinct underlying pathophysiological processes rather than a single androgen-driven cascade. It is consistent with prior work finding no significant association between the mFG score and hyperandrogenism status despite triglycerides and androgen markers themselves being correlated [10], though it contrasts with other work that has specifically investigated an mFG-metabolic syndrome relationship [9], underscoring genuine heterogeneity in this relationship across populations. It is further consistent with work finding no significant difference in Ferriman-Gallwey score between insulin-resistant and non-insulin-resistant PCOS groups, despite significant differences in anthropometric and biochemical insulin-resistance markers between these same groups [20]. This dissociation between clinical and biochemical hyperandrogenism markers may explain why our findings diverge from studies using biochemically confirmed hyperandrogenemia (elevated free or total testosterone), which have more consistently reported an independent association between androgen excess and insulin resistance or metabolic syndrome, even after adjustment for age and BMI [19]; unlike these studies, hyperandrogenism in the present cohort was ascertained entirely on clinical grounds, which may attenuate any true biological association that would be detectable with biochemical androgen

measurement. Practically, this finding suggests that hirsutism severity should not be used as a bedside proxy to triage which women with PMOS warrant metabolic screening: the metabolic burden documented above appears to affect this population broadly, independent of the degree of clinical androgen excess.

The four indices examined here were chosen to span distinct pathways through which insulin resistance and dyslipidemia are thought to elevate cardiovascular risk in PMOS: the TyG index and AIP incorporate glycemic and triglyceride/HDL information, while the LDL/HDL ratio and non-HDL cholesterol capture atherogenic lipoprotein burden more broadly [6]. That none of these four indices tracked with hirsutism severity, individually or after adjustment for BMI, suggests this dissociation is not an artifact of any single, idiosyncratic marker: it held consistently across markers spanning glycemic, triglyceride-based, and cholesterol-based dimensions of cardiometabolic risk.

Hirsutism, a clinical marker of hyperandrogenism, was reported by the large majority of participants (88.4%), consistent with hyperandrogenism-driven diagnostic criteria in this predominantly phenotype-A cohort [16]. However, as detailed above, the severity of this hyperandrogenic expression did not track with the cardiometabolic risk indices examined in this cohort, suggesting that these two dimensions of PMOS that is, reproductive/androgenic and metabolic, while co-occurring, may not be mechanistically coupled in a dose-dependent manner in this population.

Limitations

This study has several limitations. Its cross-sectional design precludes causal inference. The sample size ($n = 69$ overall; $n = 61$ for the hirsutism-cardiometabolic correlation analysis) limited statistical power throughout: the hirsutism correlation analysis could detect only correlations of $|r| \geq 0.35$ with 80% power, so weaker associations between hirsutism severity and cardiometabolic risk cannot be excluded; similarly, the phenotype A vs. non-A comparison for metabolic syndrome was likely underpowered given the modest combined size of the non-A subgroup ($n = 18$). Null findings reported here should therefore be interpreted as inconclusive-for-modest-effects rather than definitive evidence of no association. The Framingham General Cardiovascular Disease risk score was developed and validated in a predominantly White North American population and has not been specifically validated in Sub-Saharan African women with PMOS; its use here is intended as an illustrative comparator to composite lipid/glycemic indices rather than a validated risk-stratification tool for this population. As a hospital-based sample drawn from two referral centers, findings may not generalize to women with PMOS who do not seek care at referral facilities.

A further limitation concerns the ascertainment of hyperandrogenism used to construct PMOS phenotype. Although the Rotterdam criteria permit either clinical or biochemical evidence of hyperandrogenism, no participant in this cohort had a recorded serum testosterone or free androgen index value; hyperandrogenism was therefore determined entirely on clinical grounds (hirsutism, acne, or androgenic alopecia). Moreover, 61 of the 63 women classified as hyperandrogenic were classified as

such specifically on the basis of hirsutism, meaning that PMOS phenotype (and Phenotype A status in particular) and the mFG score examined in the primary hirsutism-cardiometabolic analysis are not fully independent measurements but substantially overlapping operationalizations of the same clinical observation. This is a meaningful caveat given that clinical and biochemical hyperandrogenism do not always concord [9, 10]: phenotype assignment here reflects clinical rather than biochemically confirmed hyperandrogenism, which may have affected the precision of phenotype classification (particularly the distinction between Phenotypes C and D, which depends entirely on hyperandrogenism status) and should be considered when interpreting the phenotype-metabolic syndrome association reported above.

Conclusion

In this group of Cameroonian women with PMOS, cardiometabolic risk indicators showed a significant burden. Over half of the women had a high-risk atherogenic index of plasma, and two-thirds had elevated non-HDL cholesterol. These indicators were not reflected in a conventional, age-based global cardiovascular risk score, which classified almost the entire group as having mild risk. However, the burden was more pronounced in women with the complete (Phenotype A) PMOS phenotype. Despite this, the association between the phenotype and metabolic syndrome did not reach statistical significance in this sample. In contrast, clinical hirsutism severity was not associated with any of the four cardiometabolic indices examined, regardless of whether the indices were analyzed continuously or adjusted for BMI. Overall, these findings suggest that composite lipid/glycemic indices should be included in routine cardiometabolic screening for all women with PMOS in this setting, regardless of their PMOS phenotype or the extent of clinical androgen excess.

Abbreviations

AIP

Atherogenic Index of Plasma

BMI

Body Mass Index

CI

Confidence Interval

HDL/HDL-c

High-Density Lipoprotein Cholesterol

HOMA-IR

Homeostatic Model Assessment of Insulin Resistance

IR

Insulin Resistance

LDL/LDL-c

Low-Density Lipoprotein Cholesterol

mFG

modified Ferriman–Gallwey (score)

OR
Odds Ratio
OSAHS
Obstructive Sleep Apnea–Hypopnea Syndrome
PCOS
Polycystic Ovary Syndrome (legacy term; see PMOS)
PHQ-4
Patient Health Questionnaire-4
PMOS
Polyendocrine Metabolic Ovarian Syndrome (renamed from PCOS)
SD
Standard Deviation
SHBG
Sex Hormone-Binding Globulin
SPSS
Statistical Package for the Social Sciences
TyG
Triglyceride–Glucose (index)
WHO
World Health Organization

Declarations

Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee of the Université des Montagnes (Authorization No. 2026/062/UdM/PR/CEAQ, dated 14 January 2026) and the Institutional Ethics Committee for Human Health Research (CIERSH) of the Yaoundé Gynaeco-Obstetric and Pediatric Hospital (Authorization No. 369/CIERSH/DM/2026, dated 27 February 2026). Administrative authorization to conduct the study at the Catholic Mother and Child Hospital Deo Gratias (Emana) was granted by Afrique Future, the humanitarian organization responsible for the hospital, on 26 January 2026. All participants provided written informed consent prior to enrollment.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Conflict of interest

All authors declare that they have no conflicts of interest directly or indirectly related to this study.

What is known about this topic?

PMOS is associated with insulin resistance and an adverse metabolic profile globally, and both PMOS phenotype and clinical hyperandrogenism have been proposed as markers of this cardiometabolic risk. However, whether conventional global cardiovascular risk scores adequately capture this burden, whether PMOS phenotype tracks with metabolic syndrome, and whether hyperandrogenism severity itself correlates with cardiometabolic risk are all inconsistently reported across populations, and none have previously been examined together in a Sub-Saharan African PMOS population.

What does this study add?

This study shows that a substantial cardiometabolic burden in Cameroonian women with PMOS, reflected in composite lipid/glycemic indices, is missed by a conventional, age-driven global risk score; that this burden trends toward being more pronounced in the complete (Phenotype A) PMOS phenotype; and that it is not explained by hirsutism severity, whether examined continuously or BMI-adjusted. These findings support routine use of composite cardiometabolic indices, applied uniformly across PMOS phenotypes and irrespective of androgenic severity, rather than reliance on global risk scores or clinical hyperandrogenism alone, in resource-limited settings.

Author Contributions

Conception: CON, SRSN, HMTN

Design: CON, HMTN, MAJ

Data collection: MAJ, PT, HMTN

Biomedical analyses: CN, DCTL

Statistical analyses: HMTN, SFMM

Data interpretation: HMTN, SFMM, NNAN

Supervision: CON, SRSN, CCNN

Validation of results: SFMM, NNAN, CCNN

Manuscript drafting: HMTN

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References

1. Teede HJ, Khomami MB, Morman R, Laven JSE, Joham AE, Costello MF, Patil M, Rees DA, Berry L, Cree MG, et al. Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process. *Lancet*. 2026;407(10545):2329–39. 10.1016/S0140-6736(26)00717-8.
2. Zhao H, Zhang J, Cheng X, Nie X, He B. Insulin resistance in polycystic ovary syndrome across various tissues: an updated review of pathogenesis, evaluation, and treatment. *J Ovarian Res*. 2023;16:9. 10.1186/s13048-022-01091-0.
3. Pililis S, Lampsas S, Kountouri A, Pliouta L, Korakas E, Livadas S, Thymis J, Peppas M, Kalantaridou S, Oikonomou E, et al. The Cardiometabolic Risk in Women with Polycystic Ovarian Syndrome (PCOS): From Pathophysiology to Diagnosis and Treatment. *Medicina*. 2024;60(10):1656. 10.3390/medicina60101656.
4. de Ávila MAP, Borges LP, Paez MS, Bruno RV, Nardi AE, de Pessôa ACM, de Palmeira E. [Acanthosis nigricans: metabolic interrelations inherent to the polycystic ovary syndrome]. *Revista Brasileira De Ginecol E Obstet*. 2014;36(9):410–5. 10.1590/so100-720320140005078.
5. Rojas J, Chávez M, Olivar L, Rojas M, Morillo J, Mejías J, Calvo M, Bermúdez V. Polycystic Ovary Syndrome, Insulin Resistance, and Obesity: Navigating the Pathophysiologic Labyrinth. *Int J Reproductive Med*. 2014;2014:719050. 10.1155/2014/719050.
6. Liu Q, Xie Y, Qu L, Zhang M, Mo Z. Dyslipidemia involvement in the development of polycystic ovary syndrome. *Taiwan J Obstet Gynecol*. 2019;58(4):447–53. 10.1016/j.tjog.2019.05.003.
7. Guan M, Feng J, Zhu M, Wang Y, Shi B, Lu F, Yu J, Gao Z, Yu H, Wu X. Triglyceride glucose index - body mass index predicts insulin resistance, metabolic syndrome and associates with impaired ovulation in Chinese women with polycystic ovary syndrome. *Front Endocrinol*. 2025;16. 10.3389/fendo.2025.1653636.
8. Rhaïem TB, Owczarek AJ, Wójtowicz M, Ciura D, Madej P, Chudek J, Olszanecka-Glinianowicz M. The Cut-Off Value for the Triglyceride–Glucose Index (TyG) Discriminating Insulin Resistance Based on the SHBG Level and HOMA-IR Values in Women with Polycystic Ovary Syndrome. *Life*. 2025;15(5):691. 10.3390/life15050691.
9. Aswini R, Jayapalan S. Modified Ferriman–Gallwey Score in Hirsutism and its Association with Metabolic Syndrome. *Int J Trichology*. 2017;9(1):7–13. 10.4103/ijt.ijt_93_16.

10. Hestiantoro A, Karimah PD, Shadrina A, Wiweko B, Muharam R, Astuti BPK. Triglycerides, independent of Ferriman Gallwey Score, is a main determinant of free testosterone index in PCOS. *F1000Research*. 2019;8:94. 10.12688/f1000research.16815.1.
11. Kemfang JDN, Omgba JYO, Ngo EU, Chebu CAN, Fouogue JT, Kingsley O, Eugène S. Prevalence and phenotypic features of polycystic ovary syndrome among patients attending gynaecology clinic in two referral hospitals in Yaoundé, Cameroon. *Int J Reprod Contracept Obstet Gynecol*. 2022;11(2):304. 10.18203/2320-1770.ijrcog20220152.
12. Dohbit JS, Sobngwi E, Kemfang JD, Foumane P, Tochie JN, Elong FA, Bate B, Mboudou ET. Insulin Sensitivity and Gynaecological Features of Infertile Cameroonian Females with Polycystic Ovary Syndrome: A Cross-Sectional Study. *Open J Obstet Gynecol*. 2017;7(13):1247–54. 10.4236/ojog.2017.713127.
13. The Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod*. 2004;19(1):41–7. 10.1093/humrep/deh098.
14. Araújo SP, Juvanhol LL, Bressan J, Hermsdorff HHM. Triglyceride glucose index: A new biomarker in predicting cardiovascular risk. *Prev Med Rep*. 2022;29:101941. 10.1016/j.pmedr.2022.101941.
15. Niroumand S, Khajedaluee M, Khadem-Rezaian M, Abrishami M, Juya M, Khodaei G, Dadgarmoghaddam M. Atherogenic Index of Plasma (AIP): A marker of cardiovascular disease. *Med J Islamic Repub Iran*. 2015;29:240.
16. Spritzer PM, Marchesan LB, Santos BR, Figuera TM, Hirsutism. Normal Androgens and Diagnosis of PCOS. *Diagnostics*. 2022;12(8):1922. 10.3390/diagnostics12081922.
17. D'Agostino RB, Sr, Vasan RS, Pencina MJ, Wolf PA, Cobain M, Massaro JM, Kannel WB. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation*. 2008;117(6):743–53. 10.1161/CIRCULATIONAHA.107.699579.
18. Mishra N, Mishra P, Kapoor V, Kishun J, Kumar A, Singh U. Lipid variations in different polycystic ovary syndrome phenotypes: A systematic review & meta-analysis. *Indian J Med Res*. 2025;161(5):491–501. 10.25259/IJMR_1455_2024.
19. Borzan V, Lerchbaum E, Missbrenner C, Heijboer AC, Goschnik M, Trummer C, Theiler-Schwetz V, Haudum C, Gumpold R, Schweighofer N, Obermayer-Pietsch B. Risk of Insulin Resistance and Metabolic Syndrome in Women with Hyperandrogenemia: A Comparison between PCOS Phenotypes and Beyond. *J Clin Med*. 2021;10(4):829. 10.3390/jcm10040829.
20. Alviggi C, Conforti A, De Rosa P, Strina I, Palomba S, Vallone R, Gizzo S, Borrelli R, Andersen CY, De Placido G, Guerriero S. The Distribution of Stroma and Antral Follicles Differs between Insulin-Resistance and Hyperandrogenism-Related Polycystic Ovarian Syndrome. *Front Endocrinol (Lausanne)*. 2017;8:117. 10.3389/fendo.2017.00117.
21. Isiadinso I, Wenger NK. Do We Need a Different Approach to Assess Cardiovascular Risk in Women? *US Cardiol Rev*. 2017;11(1):5–9. 10.15420/usc.2016:8:2.

Figures

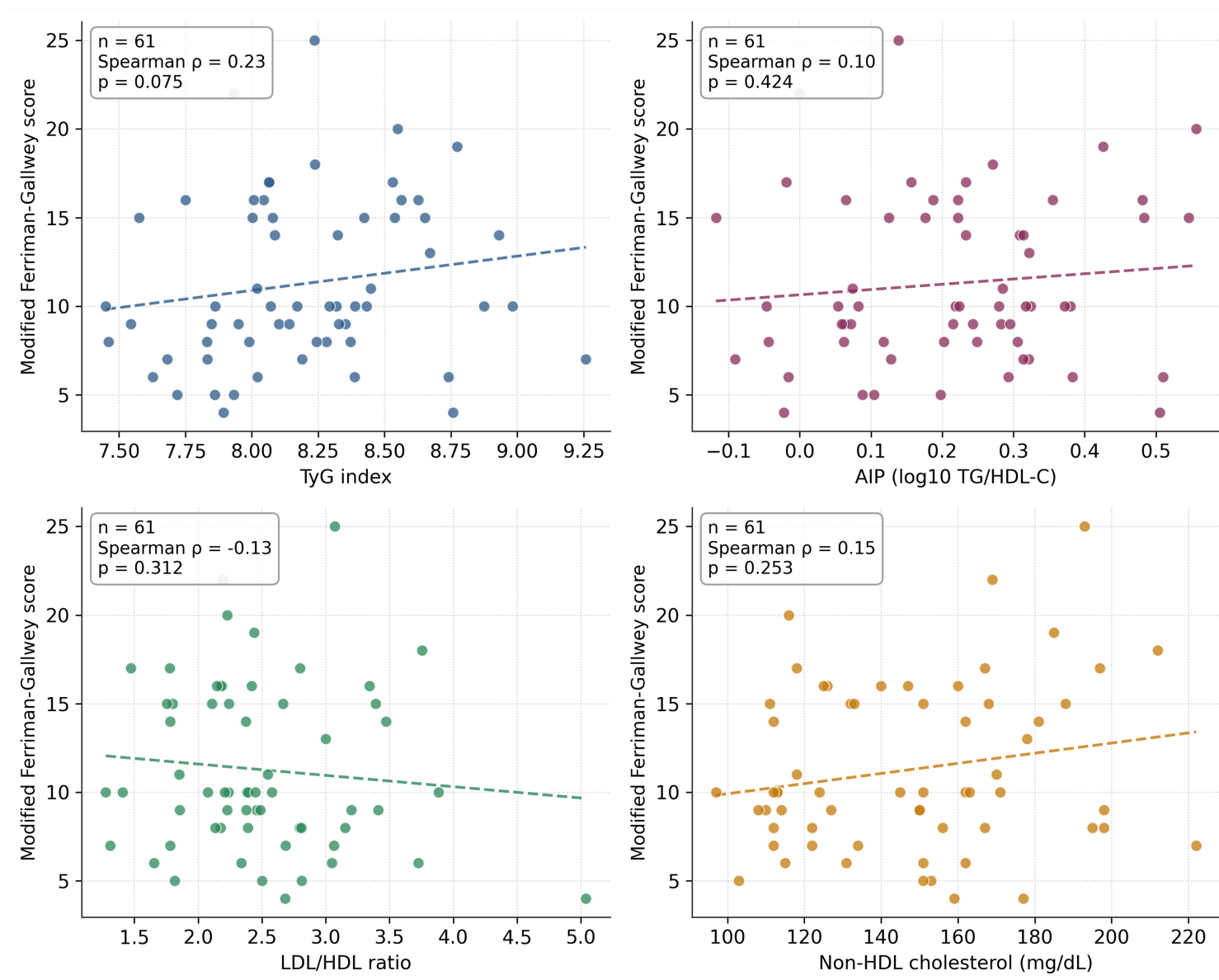


Figure 1

Scatter plots of the modified Ferriman-Gallwey score against each of the four cardiometabolic risk indices, with least-squares trend lines and Spearman correlation statistics (n=61).

Supplementary Files

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