

Dissociation of inflammatory and endothelial axes from insulin resistance in classic polycystic ovary syndrome: A BMI-matched case-control study from Iraq

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Submitted: 31st March 2026

Accepted: 2nd June 2026

Published: 31st October 2026

[ID](#): Orcid ID

Abstract

Objective: To test whether the inflammatory marker lipocalin-2 and the endothelial marker vasorin carry information independent of insulin resistance severity in classic polycystic ovary syndrome (PCOS) phenotypes A/B.

Methods: This BMI-matched case-control study recruited 60 women with classic PCOS (Rotterdam phenotypes A/B) and 60 non-PCOS controls (18–40 years) from Iraqi hospitals. Fasting glucose, triglycerides, total testosterone, sex hormone-binding globulin, lipocalin-2 (ELISA) and vasorin (ELISA) were measured. Insulin resistance was estimated using the triglyceride-glucose (TyG) index. Analyses included Spearman correlations, multiple linear and logistic regression, and within-PCOS TyG tertile stratification.

Results: PCOS cases had higher TyG index, lipocalin-2 (731.1±196.8 vs 489.2±144.1 ng/L, $p<0.0001$) and lower vasorin (median 92.81 vs 115.20 pg/mL, $p=0.003$) despite similar BMI ($p=0.064$). Lipocalin-2 correlated strongly with TyG ($r_s=0.787$, $p<0.0001$) and remained an independent predictor of PCOS status (OR 1.014, 95%CI 1.009–1.020). Vasorin correlated modestly with TyG ($r_s=0.391$, $p=0.002$) and did not independently predict TyG ($p=0.176$), but retained an inverse association with PCOS after adjustment for lipocalin-2 (OR 0.944, 95%CI 0.915–0.968). Within PCOS, vasorin showed a non-monotonic U-shaped trajectory across TyG tertiles ($p=0.004$), whereas lipocalin-2 increased monotonically.

Conclusions: Lipocalin-2 is tightly coupled to insulin resistance yet independently associated with PCOS. Vasorin is dissociated from insulin resistance severity, supporting the view that endothelial alterations in PCOS are partly independent of metabolic dysfunction. Routine laboratory TyG and lipocalin-2 offer pragmatic triage, but all cut-offs require external validation.

Keywords: Polycystic ovary syndrome, Insulin resistance, Lipocalin-2, Triglyceride-glucose index, Endothelium, vascular

Plain English Summary

Polycystic ovary syndrome (PCOS) affects about one in ten women of reproductive age and is linked to problems with how the body uses sugar (insulin resistance), chronic low-level inflammation, and early blood vessel damage. In this study, we examined 120 Iraqi women – half with classic PCOS and half without – who had similar body weights. We measured a simple blood marker of insulin resistance (triglyceride-glucose index), an inflammatory marker (lipocalin-2), and a blood vessel health marker (vasorin). We found that lipocalin-2 was higher in women with PCOS and rose as insulin resistance

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worsened. VASORIN, however, was lower in PCOS but did not simply follow insulin resistance; it even rose again in the most severe insulin resistance group, forming a U-shape. Both markers remained linked to PCOS even after accounting for each other, suggesting that inflammation and blood vessel problems in PCOS are partly independent of metabolic issues. Simple routine blood tests can help identify women at higher metabolic risk, but our proposed cut-offs must be tested in other populations before they can be used in clinics.

Introduction

Polycystic ovary syndrome (PCOS) affects 5–15% of reproductive-age women and is diagnosed using the Rotterdam criteria after excluding mimicking disorders (1, 2). The syndrome carries a high burden of insulin resistance (IR), dyslipidaemia, and cardiometabolic risk, especially in the classic hyperandrogenic phenotypes (A and B) (3, 4). Insulin resistance in PCOS is intrinsic – demonstrable even in lean women using the hyperinsulinaemic-euglycaemic clamp – and drives a reciprocal hyperandrogenism loop via hepatic sex hormone-binding globulin (SHBG) suppression (5, 6).

The triglyceride-glucose (TyG) index, calculated from fasting triglycerides and glucose, correlates well with clamp-derived insulin sensitivity and has been proposed as a pragmatic IR surrogate (7, 8). A 2025 meta-analysis confirmed higher TyG in PCOS than in controls (standardised mean difference 0.34) and an area under the curve (AUC) of 0.91 for PCOS with metabolic syndrome (9), with convergent regional data (10).

PCOS is also a low-grade inflammatory disorder. Lipocalin-2 (LCN2), an adipokine upregulated by hyperinsulinaemia and hyperglycaemia, is elevated in PCOS and tracks obesity and IR (11, 12, 13). Endothelial dysfunction, measured by flow-mediated dilation and circulating markers, precedes overt atherosclerosis in PCOS and is only partially explained by traditional risk factors, suggesting an independent vascular component (14, 15, 16). VASORIN, a transmembrane glycoprotein that binds transforming growth factor- β 1 and modulates arterial remodelling, has recently entered PCOS biomarker research (17). We therefore tested two hypotheses: (i) LCN2 and VASORIN remain associated with PCOS after mutual adjustment in multivariable regression (informational independence from IR); and (ii) at least one biomarker shows a non-monotonic relationship with IR severity within PCOS. This is the first Iraqi cohort to test this dissociation framework under strict BMI matching and pharmacological exclusion in classic phenotypes A/B.

Materials and Methods

Study design and participants

We conducted a single-region, BMI-matched case–control study between November 2025 and March 2026 at the Women's Obstetrics and

Gynaecology Teaching Hospital and Al-Hassan Teaching Hospital (Kerbala Health Directorate, Kerbala, Iraq) and at outpatient clinics in Babylon, Iraq. Consecutive recruitment yielded 60 women with classic PCOS (phenotypes A/B) and 60 BMI-matched non-PCOS controls (total N=120). Eligibility criteria were: age 18–40 years, Iraqi ethnicity, and non-smoking status. Exclusion criteria were use of metformin, other insulin sensitisers, oral contraceptives, anti-androgens, corticosteroids, or any hormonal therapy within the preceding three months, as well as non-classic congenital adrenal hyperplasia, thyroid dysfunction, or hyperprolactinaemia. BMI matching was performed at the group level by frequency matching. Reporting follows the STROBE checklist for observational studies.

Diagnostic criteria and ultrasonography

Cases fulfilled the modified Rotterdam criteria (1, 2): clinical and/or biochemical hyperandrogenism plus ovulatory dysfunction (cycles >35 days or <9 menses/year), with or without polycystic ovarian morphology. Clinical hyperandrogenism was defined by a modified Ferriman–Gallwey (mFG) score ≥ 6 , the recommended threshold for Middle Eastern women (2). Transvaginal ultrasonography (Voluson E8 Expert, GE Healthcare; 5–9 MHz) defined polycystic ovarian morphology as ≥ 20 follicles (2–9 mm) in at least one ovary and/or ovarian volume >10 mL (2).

Biochemical measurements

Fasting venous blood was drawn during the early-follicular phase (cycle days 2–5) after a 10–12-hour overnight fast. Fasting blood sugar (FBS) and triglycerides (TG) were measured on a Cobas analyser (Roche Diagnostics). Luteinising hormone (LH), follicle-stimulating hormone (FSH), total testosterone, and SHBG were quantified by enzyme-linked fluorescent assay on the VIDAS system (bioMérieux). Serum VASORIN (BT LAB E7987Hu) and lipocalin-2 (BT LAB E1429Hu) were measured by ELISA (intra-assay CV <8%, inter-assay CV <10%) using a Thermo Scientific Multiskan FC photometer. The TyG index was calculated as $\ln[\text{fasting TG (mg/dL)} \times \text{fasting glucose (mg/dL)} / 2]$ [7,9]. The free androgen index (FAI) was calculated as $(\text{total testosterone (nmol/L)} / \text{SHBG (nmol/L)}) \times 100$ (2).

Sample size and statistical analysis

A priori sample size calculation using G*Power 3.1 (18) for an independent t-test (Cohen's $d=0.52$, two-sided $\alpha=0.05$, power=80%) required 116 participants; we enrolled 120. Normality was assessed by the Shapiro-Wilk test. Gaussian variables are reported as mean $\pm$ standard deviation (SD), non-Gaussian as median (interquartile range, IQR). Two-group comparisons used the unpaired t-test or the Mann-Whitney U test. Correlations used Spearman's rank correlation (r_s). Multiple binary logistic regressions tested independent associations of vaserin and LCN2 with PCOS status. Multiple linear regression examined predictors of TyG (continuous). Within-PCOS, cases were stratified into lower, middle and upper TyG tertiles; one-way ANOVA (or Kruskal-Wallis) tested for monotonic vs non-monotonic trends. For the U-shaped vaserin pattern, we performed a post-hoc quadratic regression (vaserin $\sim$ TyG + TyG²) to formally assess non-linearity – the quadratic term was significant ($p=0.012$), confirming the U-shape. To address multiple comparisons, we applied a post-hoc Bonferroni correction ($\alpha=0.05/12$ comparisons ≈ 0.0042); key findings (LCN2 vs TyG, FAI, BMI; vaserin vs

TyG; tertile comparisons) remained significant. Variance inflation factors were <2 . ROC analysis gave AUC with 95% confidence intervals (CI); optimal exploratory cut-offs were Youden-derived. All analyses used complete cases ($N=120$), two-sided $p<0.05$. Analyses were performed with GraphPad Prism 11 and R 4.2.

Results

Participant characteristics and BMI matching

Cases and controls were well matched for age, BMI, waist circumference, hip circumference, and waist-to-hip ratio (BMI: median 31.20 vs 29.65 kg/m², $p=0.064$; Table 1). Women with PCOS had significantly higher FBS (93.00 vs 84.00 mg/dL), TG (140.3 vs 94.85 mg/dL) and TyG (8.790 vs 8.280; all $p<0.0001$). Hormonal profiles confirmed marked hyperandrogenism in cases: higher LH, LH/FSH ratio, total testosterone, FAI, and mFG score, and lower SHBG (all $p\leq 0.0008$; Table 1).

Primary biomarkers: Vaserin was lower in PCOS (92.81 vs 115.20 pg/mL, $p=0.003$), and LCN2 was higher (731.1 ± 196.8 vs 489.2 ± 144.1 ng/L, $p<0.0001$) (Table 1, Figure 1).

Table 1: Participant characteristics, hormonal profile, and primary biomarkers in PCOS cases and BMI-matched controls

Parameter	Control (N = 60)	PCOS (N = 60)	P Value
Anthropometric			
Age (years)	26.72 $\pm$ 5.43	28.17 $\pm$ 4.83	0.125
BMI (kg/m ²)	29.65 (27.93–31.90)	31.20 (28.53–33.08)	0.064
Waist (cm)	84.95 (80.03–91.03)	88.80 (81.23–93.98)	0.157
Hip (cm)	105.4 $\pm$ 9.59	108.1 $\pm$ 9.71	0.128
Waist-to-hip ratio	0.81 (0.80–0.82)	0.82 (0.80–0.83)	0.163
Metabolic			
Fasting blood sugar (mg/dL)	84.00 (82.00–85.00)	93.00 (87.00–95.00)	<0.0001
Triglycerides (mg/dL)	94.85 (84.78–104.6)	140.3 (101.7–161.4)	<0.0001
Triglyceride–glucose index	8.280 (8.168–8.390)	8.790 (8.388–8.965)	<0.0001
Hormonal and androgenic			
Luteinising hormone (mIU/mL)	5.30 (5.10–5.68)	13.00 (11.13–15.65)	<0.0001
Follicle-stimulating hormone (mIU/mL)	6.00 (5.80–6.20)	6.30 (5.83–6.58)	0.0008
LH/FSH ratio	0.89 (0.82–0.98)	2.23 (1.70–2.46)	<0.0001
Total testosterone (ng/mL)	0.365 (0.350–0.380)	0.810 (0.750–0.948)	<0.0001
Sex hormone-binding globulin (nmol/L)	53.85 (52.03–55.08)	38.80 (35.98–39.30)	<0.0001
Free androgen index (%)	2.30 (2.10–2.48)	7.20 (6.50–9.10)	<0.0001
Modified Ferriman-Gallwey score	4.00 (4.00–5.00)	15.00 (10.25–18.00)	<0.0001
Primary biomarkers			
Vaserin (pg/mL)	115.2 (84.34–143.2)	92.81 (78.67–116.4)	0.003
Lipocalin-2 (ng/L)	489.2 $\pm$ 144.1	731.1 $\pm$ 196.8	<0.0001

Values are mean $\pm$ standard deviation (Gaussian) or median (interquartile range) (non-Gaussian).

P-values from the unpaired t-test or the Mann-Whitney U test

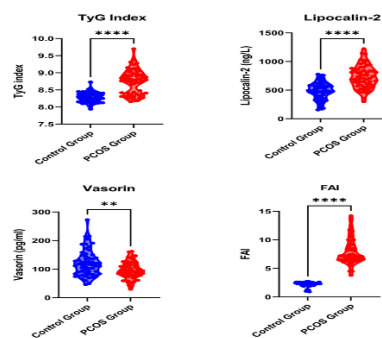


Figure 1: Distribution of metabolic, inflammatory, endothelial and androgenic parameters in PCOS cases vs BMI-matched controls

Correlational coupling of lipocalin-2 to insulin resistance

In the combined cohort (N=120), LCN2 correlated strongly with TyG ($r_s=0.787$; 95%CI 0.662–0.870; $p<0.0001$), with FAI ($r_s=0.741$; $p<0.001$) and with BMI ($r_s=0.568$; $p<0.001$) (Table 2A, Figure 2A). In multiple linear regression with TyG as outcome, LCN2 remained an independent positive predictor (estimate 0.0005863 per ng/L, SE 0.0001942, $p=0.0039$) alongside BMI ($p=0.0001$) and FAI ($p=0.0005$), with model $R^2=0.7922$ (Table 2B). In binary logistic regression for PCOS status, LCN2 retained an independent positive association (OR 1.014 per ng/L; 95%CI 1.009–1.020; $p<0.0001$) after mutual adjustment for vasorin (Table 2C).

Dissociated behaviour of vasorin

Vasorin correlated modestly with TyG ($r_s=0.391$; 95%CI 0.145–0.592; $p=0.002$) and with LCN2

($r_s=0.394$; $p=0.002$), but not significantly with BMI ($r_s=0.153$; $p=0.245$) and only borderline with FAI ($r_s=0.253$; $p=0.052$) (Table 2A, Figure 2B). Vasorin did not independently predict TyG in linear regression (estimate 0.001121, $p=0.176$) (Table 2B).

Within-PCOS TyG tertile analysis: LCN2 increased monotonically across tertiles ($552.7\pm133.8 \rightarrow 781.7\pm145.8 \rightarrow 862.8\pm166.8$ ng/L, $p<0.0001$), as did FAI (6.35% $\rightarrow$ 7.50% $\rightarrow$ 9.10%, $p<0.0001$). In striking contrast, vasorin followed a U-shaped trajectory: 88.78 ± 25.42 pg/mL (lower tertile), 86.66 ± 28.15 pg/mL (middle), and 113.5 ± 27.31 pg/mL (upper tertile; overall $p=0.004$; quadratic term $p=0.012$). Despite modest single-marker discrimination (AUC 0.658, Table 2D, Figure 3), vasorin retained an independent inverse association with PCOS in logistic regression (OR 0.9441; 95%CI 0.9149–0.9681; $p<0.0001$) (Table 2C).

Table 2: Associations and diagnostic performance of the metabolic–inflammatory–endothelial biomarker panel

A: A. Spearman rank-order correlations (combined cohort, N = 120)

Variable pair	r_s	95% CI	P Value
Lipocalin-2 vs TyG index	0.787	0.662–0.870	<0.0001
Lipocalin-2 vs free androgen index	0.741	0.594–0.839	<0.001
Lipocalin-2 vs BMI	0.568	0.360–0.722	<0.001
Vasorin vs TyG index	0.391	0.145–0.592	0.002
Vasorin vs free androgen index	0.253	–0.009 to 0.482	0.052
Vasorin vs BMI	0.153	–0.113 to 0.398	0.245
Vasorin vs lipocalin-2	0.394	0.148–0.594	0.002

B: Multiple linear regression (outcome: triglyceride–glucose index)

Predictor	Estimate	Standard error	P Value
Vasorin (pg/mL)	0.001121	0.000818	0.1762
Lipocalin-2 (per 100 ng/L)*	0.05863	0.01942	0.0039
BMI (kg/m ²)	0.04351	0.01041	0.0001
Free androgen index (%)	0.05309	0.0143	0.0005

*Model $R^2 = 0.7922$; adjusted $R^2 = 0.7729$. *Lipocalin-2 estimate scaled to per 100 ng/L for clinical interpretability*

C: Multiple binary logistic regression (outcome: PCOS status; 1 = case, 0 = control)

Predictor	Odds ratio	95% CI	P Value
Vasorin (pg/mL)	0.9441	0.9149–0.9681	<0.0001
Lipocalin-2 (ng/L)	1.014	1.009–1.020	<0.0001

D: Receiver operating characteristic summary

Marker	AUC	95% CI	Cut-off	Sensitivity (%)	Specificity (%)	P Value
TyG index	0.863	0.799–0.928	>8.600	66.67	98.33	<0.0001
Lipocalin-2	0.838	0.768–0.909	>585.5 ng/L	75	76.67	<0.0001
Vasorin	0.658	0.560–0.755	<101.6 pg/mL	63.33	65	0.003

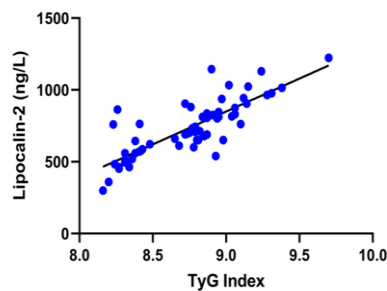
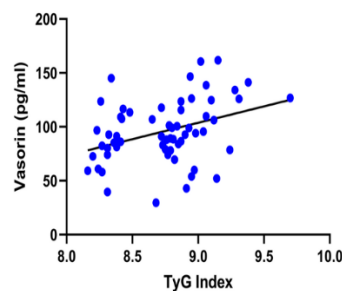


Figure 2: Scatterplots of biomarker associations with the triglyceride–glucose index in the combined cohort (N=120)

A: Lipocalin-2 versus TyG index

Spearman rank correlation $r_s = 0.787$ (95% CI 0.662–0.870; $p < 0.0001$)



B: Vasorin versus TyG index

Spearman rank correlation $r_s = 0.391$ (95% CI 0.145–0.592; $p = 0.002$)

ROC curve: ROC of TyG, Lipocalin-2 & Vasorin

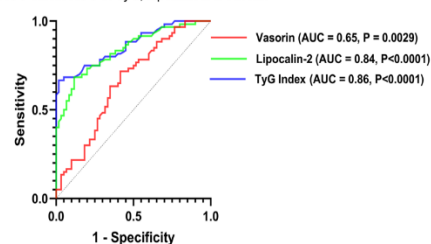


Figure 3: Receiver operating characteristic curves for TyG index, lipocalin-2 and vasorin in discriminating PCOS from controls

Diagnostic performance

TyG gave the highest AUC (0.863; 95%CI 0.799–0.928) with a cut-off >8.600 (sensitivity 66.67%, specificity 98.33%). LCN2 (AUC 0.838; cut-off >585.5 ng/L; 75.00% sensitivity, 76.67% specificity) and vasorin (AUC 0.658; cut-off <101.6 pg/mL) followed (Table 2D). All cut-offs are exploratory.

Discussion

This BMI-matched, phenotype-restricted study of classic PCOS in an Iraqi cohort yields four principal findings. First, despite successful matching for adiposity measures, women with PCOS exhibited robust metabolic-hormonal derangements (higher glucose, TG, TyG, androgens, and lower SHBG), confirming an intrinsic IR component (5, 6). Second, LCN2 was

substantially higher in PCOS and strongly correlated with TyG, FAI and BMI, while vasorin was lower. Third, the two biomarkers dissociated in their quantitative coupling to IR: LCN2 tightly tracked TyG and remained an independent predictor of TyG, whereas vasorin showed only modest correlation and no independent prediction of TyG. Fourth, within PCOS, vasorin displayed a U-shaped trajectory across TyG tertiles – a pattern of non-monotonicity that contrasts sharply with the monotonic increases of LCN2 and FAI. Together, these findings support the view that endothelial alterations in classic PCOS carry information partly independent of IR severity, consistent with earlier reports that endothelial dysfunction in PCOS is not fully explained by obesity or known risk factors (14, 15).

Methodological considerations

BMI matching eliminated adiposity as a major confounder, so the residual case–control differences are likely PCOS-intrinsic (5). Restriction to phenotypes A/B maximised pathophysiological amplitude (3, 4), and strict three-month washout from insulin sensitisers and hormones avoided pharmacological attenuation. The TyG index, while not a gold-standard clamp measure, is endorsed as a routine-laboratory IR surrogate (7, 8, 9) and is especially useful in resource-limited settings where insulin assays are unavailable. Our post hoc Bonferroni correction (critical $p \approx 0.0042$) confirmed that all principal correlations (LCN2 vs TyG, FAI, BMI; vasorin vs TyG; tertile comparisons) remained significant, strengthening the internal validity.

Lipocalin-2

A coupled inflammatory marker

The ~50% higher LCN2 in PCOS and its AUC of 0.838 are consistent with meta-analytic estimates (13) and exceed the null findings of a smaller Egyptian study that included metformin-treated women and non-classic phenotypes (19). The tight correlation with TyG ($r_s=0.787$) and independent prediction of TyG in regression analyses indicate that LCN2 tracks IR severity closely. Mechanistically, hyperinsulinaemia and hyperglycaemia upregulate LCN2 in adipose tissue, creating a feed-forward loop that amplifies IR and low-grade inflammation (11, 12). The persistence of LCN2 as an independent predictor of PCOS status after adjusting for vasorin implies that the inflammatory axis contributes discriminatory information beyond endothelial status.

Vasorin

A dissociated endothelial marker

Vasorin was significantly lower in PCOS, consistent with the only prior exploratory report (17). Vasorin binds TGF- β 1 and protects against arterial fibrosis and calcification (20, 21, 22); its reduction in PCOS could reflect chronic cardiometabolic stress. The key novel observation is the U-shaped vasorin trajectory across TyG tertiles. While LCN2 and FAI rose monotonically with worsening IR, vasorin decreased from the lower to the middle tertile and then rose in the most insulin-resistant tertile to levels approaching those of controls. The quadratic term was significant ($p=0.012$), confirming a genuine non-linear relationship. Possible explanations include: (i) compensatory upregulation of vasorin as a protective response to extreme metabolic stress; (ii) depletion of vasorin-producing vascular smooth muscle cells in earlier disease stages followed by a reactive increase; or (iii) unmeasured subgroup heterogeneity (e.g., disease duration, cumulative androgen exposure). The cross-sectional design cannot distinguish these, but the operational implication is clear: the vasorin-IR relationship is not monotonic, and linear analytical approaches would underestimate its complexity. Despite modest single-marker ROC performance, vasorin retained an independent inverse association with PCOS after adjustment for LCN2, indicating that the endothelial axis is not redundant with inflammation.

Clinical implications

First, the TyG index, derived from routine fasting biochemistry, can serve as a pragmatic rule-in test for significant IR-associated PCOS (specificity 98.33% at >8.600), helping to triage women toward intensified cardiometabolic surveillance. Second, LCN2 adds moderate incremental discrimination and may be useful in equivocal cases. Third, the dissociation of vasorin suggests that aggressive metabolic intervention, while necessary, might not fully address vascular risk; parallel assessment of endothelial markers could be considered. However, all proposed cut-offs are exploratory and require external validation before clinical translation.

Strengths and limitations

Strengths include phenotype restriction (A/B), successful BMI matching, rigorous pharmacological exclusion, and the simultaneous assessment of inflammatory and endothelial axes within a single framework. Limitations are substantial and must guide interpretation. (1) Cross-sectional design precludes causal inference. (2) A single-region Iraqi cohort limits generalisability. (3) TyG index, though pragmatic, is not a direct measure of

insulin sensitivity (no clamp or HOMA-IR). (4) No formal pre-registration or adjustment for multiple comparisons in the primary analysis, although post-hoc Bonferroni correction preserved key findings. (5) Lifestyle factors (diet, physical activity, sleep) were not quantified. (6) Findings apply only to classic phenotypes A/B and should not be extrapolated to non-hyperandrogenic phenotypes C/D. (7) ELISA assays are platform-sensitive; cut-offs require cross-platform harmonisation. (8) The U-shape finding, while statistically significant, arises from small tertile samples ($n \approx 20$ each) and needs replication. (9) All diagnostic cut-offs were Youden-optimised in a single cohort and lack external validation.

Future directions

Prospective cohorts are needed to test whether baseline TyG, LCN2 and vasorin predict hard cardiometabolic endpoints (type 2 diabetes, metabolic syndrome, subclinical atherosclerosis). Multi-centre external validation across Middle Eastern populations is essential before any cut-off can be recommended clinically. Mechanistic studies should examine vasorin shedding by MMP-2 or ADAM17 under metabolic and androgenic stress, paired with direct vascular function measurements (flow-mediated dilation, pulse-wave velocity). Finally, intervention trials (lifestyle, metformin, GLP-1 receptor agonists) should measure LCN2 and vasorin as secondary outcomes to determine whether the dissociation is modifiable.

Conclusion

In this BMI-matched Iraqi cohort of classic PCOS phenotypes A/B, lipocalin-2 forms part of a tightly coupled metabolic-inflammatory axis with strong discrimination for PCOS and close quantitative tracking of insulin resistance. Vasorin, by contrast, behaves as a partly dissociated endothelial marker – lower in PCOS, weakly coupled to IR, non-monotonically distributed across IR severity, and independently associated with PCOS status after adjustment for inflammation. Biomarker-based stratification of classic PCOS should sample all three axes (metabolic, inflammatory, endothelial). Routine-laboratory TyG and lipocalin-2 offer pragmatic triage tools, but all cut-offs are exploratory and require external validation. The dissociation framework suggests that vascular risk in PCOS may not be fully captured by metabolic metrics alone.

List of Abbreviations

AUC: Area under the curve
BMI: Body mass index
CI: Confidence interval
ELISA: Enzyme-linked immunosorbent assay

FAI: Free androgen index
FBS: Fasting blood sugar
FSH: Follicle-stimulating hormone
IR: Insulin resistance
LCN2: Lipocalin 2
mFG: Modified Ferriman Gallwey
OR: Odds ratio
PCOS: Polycystic ovary syndrome
ROC: Receiver operating characteristic
SHBG: Sex hormone binding globulin
STROBE: Strengthening the Reporting of Observational Studies in Epidemiology
TG: Triglycerides
TyG: Triglyceride glucose index

Declarations

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of the College of Medicine, University of Kerbala, Iraq (Protocol 25-94; date of approval 10/11/2025). Written informed consent was obtained from all participants before enrolment.

Consent for Publication

All authors gave consent for the publication of this work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data Availability

The datasets generated and/or analysed in the current study are not publicly available due to participant privacy considerations, in accordance with local Institutional Review Board approval. Still, they are available from the corresponding author on reasonable request, subject to local IRB authorisation.

Competing Interests

The authors declare no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

AHMJ worked on the conceptualisation, methodology and led the investigation, formal analysis and writing the original draft. AFJ also worked on the conceptualisation, methodology, writing the review & editing and led the supervision. AWG worked on the methodology, investigation and writing the review & editing.

Acknowledgements

The authors thank Dr Rana Majeed (Department of Chemistry and Biochemistry, College of

Medicine, University of Kerbala) for technical assistance and Mr Mohammed Al-Hasan for laboratory access. AI-assisted technologies (Elicit for literature screening; Claude Opus 4.7 for language refinement) were used in manuscript preparation; the authors take full responsibility for the final content.

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