RESEARCH PAPER

Association of Blood Glucose, Serum Insulin, Lactate Dehydrogenase and Haematological Parameters in Women Evaluated for Polycystic Ovary Syndrome: A Cross-Sectional Study

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ABSTRACT

Background and objectives: Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine-metabolic disorder associated with insulin resistance, hyperinsulinaemia and low-grade inflammation. This study explored the relationships among blood glucose, serum insulin, lactate dehydrogenase (LDH) and haematological parameters in women evaluated for PCOS.

Methods: A cross-sectional observational analysis was performed using the 45 were female records and women aged 18–60 years met the age criterion specified in the proposal. Blood glucose, serum insulin, LDH and complete blood count parameters were analysed using mean ± standard deviation, range, Pearson correlation and Spearman rank correlation. Statistical significance was set at $p<0.05$.

Results: Among the 19 eligible women, mean age was 47.37 ± 11.10 years. Mean blood glucose was 166.32 ± 21.02 mg/dL, insulin 15.46 ± 7.81 µIU/mL and LDH 267.42 ± 22.54 U/L. Blood glucose showed strong positive correlations with insulin (Pearson $r=0.933$, $p<0.001$) and LDH ($r=0.911$, $p<0.001$), while insulin correlated strongly with LDH ($r=0.934$, $p<0.001$). LDH was not significantly correlated with most routine haematological parameters.

Interpretation and conclusions: The updated dataset demonstrates strong interrelationships among blood glucose, insulin and LDH, but no consistent association between LDH and routine haematological parameters. These findings support further investigation of LDH as an exploratory metabolic marker. They do not establish a diagnostic screening panel for PCOS because PCOS status and fasting status were not documented in the spreadsheet.

Keywords: Blood glucose; Haematological parameters; Insulin; Lactate dehydrogenase; Polycystic ovary syndrome; Screening biomarkers

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine disorder characterized by combinations of hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology [1,2]. Beyond reproductive manifestations, PCOS is closely associated with insulin resistance, compensatory hyperinsulinaemia, dyslipidaemia and chronic low-grade inflammation [3,4]. These metabolic abnormalities contribute to the long-term risk of type 2 diabetes mellitus and cardiovascular disease [5,6].

Insulin resistance is a central metabolic feature of PCOS. Dunaif and Diamanti-Kandarakis et al. [3,4] described the contribution of insulin resistance and hyperinsulinaemia to PCOS pathophysiology [3,4]. Glucose and insulin provide complementary information about metabolic status, although interpretation depends on fasting status and appropriate assessment of insulin resistance. Legro et al. demonstrated [13] the usefulness of glucose-to-insulin measures in women with PCOS [13], while Majid et al. reported [14] the usefulness of HOMA-IR for assessing insulin resistance [14].

PCOS is also associated with inflammatory and metabolic alterations that may be reflected in routinely available laboratory parameters. Previous studies have reported changes in haematological inflammatory markers in PCOS [7,9,10]. LDH is a non-specific enzyme associated with cellular injury and metabolic stress, and emerging evidence suggests that LDH/LDHA may be altered in women with PCOS and metabolic disturbances [11,17].

The present study therefore examines relationships among blood glucose, serum insulin, LDH and haematological parameters in women evaluated for PCOS. The intention is exploratory: to identify laboratory associations that may justify a larger, clinically characterized study rather than to claim diagnostic performance from cross-sectional correlations.

NEED FOR THE STUDY

PCOS has heterogeneous reproductive, metabolic and inflammatory manifestations, and no single routine laboratory marker adequately represents the complete spectrum of abnormalities [3,4,8,12]. The proposal focuses on CBC, blood glucose, insulin and LDH as accessible laboratory parameters [7–11]. The glucose–insulin relationship is particularly relevant because insulin resistance is an important component of PCOS. The proposal also identifies LDH as a potential marker of metabolic stress. However, evidence linking LDH directly with routine CBC parameters in PCOS is less established. Evaluating these

relationships may provide a basis for future hypothesis-driven studies.

Because the available spreadsheet does not contain a documented PCOS diagnosis, fasting status, BMI, menstrual history or androgen profile, the present analysis is appropriately interpreted as exploratory among women evaluated for PCOS.

RESEARCH QUESTION

Is there an association between blood glucose and serum insulin and their relationships with serum LDH and haematological parameters among women evaluated for polycystic ovary syndrome (PCOS)?

OBJECTIVES

Primary objective

To evaluate the association among blood glucose, serum insulin and LDH and their relationships with haematological parameters in women evaluated for PCOS.

Secondary objectives

- To determine the correlation between blood glucose and serum insulin.
- To assess the association of serum LDH with blood glucose and serum insulin.
- To evaluate the relationship between serum LDH and haematological parameters, including haemoglobin, RBC count, PCV, MCV, MCHC, RDW, TLC, differential leukocyte counts and platelet count.
- To explore the relationship of blood glucose, serum insulin and LDH with age in the available dataset.

PICOT FRAMEWORK

Component	Definition
P – Population	Women aged 18–60 years attending a tertiary-care hospital with suspected or diagnosed PCOS.
I – Index assessment	Assessment of CBC, blood glucose, serum insulin and serum LDH.
C – Comparison	Comparison of normal versus abnormal laboratory values and/or correlation among continuous parameters.
O – Outcome	Association among metabolic, biochemical and haematological parameters and their potential utility as complementary screening markers.
T – Time	Cross-sectional, single time-point assessment.

MATERIALS AND METHODS

Study design and setting

This was planned as a cross-sectional observational study at a tertiary-care hospital. The proposal specified women aged 18–60 years attending outpatient or inpatient departments and clinically diagnosed with PCOS.

Study population and eligibility

The proposal included women aged 18–60 years willing to provide informed consent and attending OPD/IPD services and know PCOS case. Pregnant women, women with known endocrine disorders other than PCOS, and women receiving hormonal or lipid-lowering therapy were to be excluded. Because the spreadsheet supplied for analysis did not explicitly record PCOS diagnosis or the complete eligibility variables, the analytical population was conservatively described as women evaluated for PCOS.

Sample size and available dataset

Using a literature-derived PCOS prevalence of approximately 6%, 95% confidence level and 7% absolute precision, the target sample size was calculated as approximately 44; therefore, 45 participants were planned. The spreadsheet contained 45 records, of which 40 were female and aged 18–60 years met the age criterion.

Where: **Z = 1.96** at 95% confidence level, **p = 0.06 (6%), q = 1-p = 0.94, d = 0.07 (7% absolute precision)**

Therefore:

$$\frac{(1.96)2 \times (0.06) \times (0.94)}{(0.07)2}$$

$$\frac{3.84 \times 0.0564}{0.0049}$$

n= 44.2= 45

Data collection and laboratory methods

- **Demographic details:** Age, BP, menstrual history
- **Clinical features:** hirsutism, acne, obesity
- **Laboratory investigations:** 5 ml sample of patient is collected under all aseptic precautions, serum sample was used after centrifugation for laboratory tests.

Sr. No	Parameter	Instrument / Technique
1	**Complete blood count (CBC)**	Beckman **DXH** Five part Principle: Coulter
2	Blood Glucose Level	Cobas c311 Principle: Photometry- Enzymatic
3	Insulin	Cobas e411 Principle: electrochemiluminescence immunoassay (ECLIA)- Sandwitch
4	Lactate dehyrogenase	Cobas c311 Principle: Photometry- IFCC

Data processing: Laboratory values were enterd in prepared datasheet. The missing values were not included and counted in data sheet for this study. Format of data sheet is provided

Date	Name of patient	Age/ gender	BP	Menstrual history	other	RBS	Insulin	LDH	HB	RBC	PCV	MCV	MCHC	RdW	TLC	Neutr	Lympho	Eosi	Baso	platelet

Variable handling

Age and sex were extracted from the Age/gender field. Numeric laboratory values were extracted from cells containing appended high/low flags. The source spreadsheet listed reference intervals of up to 140 mg/dL for blood glucose, 2.6–24.9 µIU/mL for insulin and 135–225 U/L for LDH. These intervals were used only for descriptive context and not to establish PCOS or any other diagnosis.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation and range. Pearson correlation was used to evaluate linear associations. Spearman rank correlation was additionally examined because several variables were skewed and the sample was small. A two-sided p-value <0.05 was considered statistically significant. No imputation was performed. Because a confirmed PCOS comparison group was not identifiable in the spreadsheet, t-test and chi-square comparisons were not used for the primary analysis.

RESULTS

1) Dataset characteristics

The spreadsheet contained 45 records. Forty four records were identified as female, of aged 18–60 years met the eligibility age criterion and constituted the analytical sample. The mean age was 47.37 ± 11.10 years (range 22–60 years). Nineteen women had regular menstrual cycles, seventeen had irregular cycles and Eight were recorded as menopausal. Blood pressure was recorded as Normal in 27 participants , high in 8 participants and Low in 10 participant. The clinical-characteristics field documented at least one feature such as acne, facial/body hair or obesity in women.

2) Descriptive laboratory findings

Parameter	n	Mean ± SD	Minimum	Maximum
Age (years)	45	47.37 ± 11.10	22.00	60.00
Blood glucose (mg/dL)	45	166.32 ± 21.02	78.00	564.00
Insulin (µIU/mL)	45	15.46 ± 7.81	2.87	79.78
LDH (U/L)	45	267.42 ± 22.54	156.00	610.00
Haemoglobin (g/dL)	45	12.96 ± 1.56	9.76	15.77
RBC (×10⁶/µL)	45	4.61 ± 0.79	2.40	6.15
PCV (%)	45	39.13 ± 4.71	29.20	46.70
MCV (fL)	45	86.33 ± 11.46	69.20	121.50
MCHC (g/dL)	45	33.12 ± 0.61	31.60	33.90
RDW (%)	45	15.38 ± 1.36	13.00	17.70
TLC (/µL)	45	10072.11 ± 3510.80	5700.00	21170.00
Neutrophils (%)	45	62.68 ± 7.97	46.00	75.00
Lymphocytes (%)	45	26.05 ± 7.33	13.00	44.00
Eosinophils (%)	45	3.58 ± 3.78	1.00	18.00
Basophils (%)	45	0.63 ± 1.89	0.00	6.00
Platelets (/µL)	45	308857.89 ± 53727.08	186500.00	434200.00

Descriptive categorization

Mean blood glucose was 166.32 ± 21.02 mg/dL (range 78–564), mean serum insulin was 15.46 ± 7.81 µIU/mL (range 2.87–79.78), and mean LDH was 267.42 ± 22.54 U/L (range 156–610). Mean haemoglobin was 12.96 ± 1.56 g/dL, RBC count 4.61 ± 0.79 ×10⁶/µL, PCV 39.13 ± 4.71%, MCV 86.33 ± 11.46 fL, MCHC 33.12 ± 0.61 g/dL and RDW 15.38 ± 1.36%. Mean TLC was 10,072.11 ± 3,510.80/µL, neutrophils 62.68 ± 7.97%, lymphocytes 26.05 ± 7.33%, eosinophils 3.58 ± 3.78%, basophils 0.63 ±

1.89% and platelet count 308,857.89 ± 53,727.08/μL.

3) Correlation of blood glucose, insulin and LDH

Variable pair	n	Pearson r	Pearson p	Spearman rho	Spearman p
BGL – Insulin	45	0.933	<0.001	0.763	<0.001
BGL – LDH	45	0.911	<0.001	0.814	<0.001
Insulin – LDH	45	0.934	<0.001	0.916	<0.001

Blood glucose showed a very strong positive correlation with serum insulin (Pearson r=0.933, p<0.001; Spearman rho=0.763, p<0.001). Blood glucose was strongly correlated with LDH (Pearson r=0.911, p<0.001; Spearman rho=0.814, p<0.001). Serum insulin also showed a very strong positive correlation with LDH (Pearson r=0.934, p<0.001; Spearman rho=0.916, p<0.001).

4) Association of LDH with haematological parameters

LDH with	n	Pearson r	p	Spearman rho	p
Haemoglobin	45	-0.078	0.750	-0.232	0.339
RBC	45	0.068	0.781	-0.077	0.753
PCV	45	-0.052	0.832	-0.169	0.490
MCV	45	-0.152	0.535	0.028	0.909
MCHC	45	-0.159	0.515	-0.259	0.284
RDW	45	-0.219	0.368	-0.183	0.453
TLC	45	0.018	0.943	0.018	0.943
Neutrophils	45	-0.389	0.100	-0.240	0.323
Lymphocytes	45	0.471	0.042	0.314	0.190
Platelets	45	0.418	0.075	0.204	0.403

LDH was not significantly correlated with haemoglobin, RBC count, PCV, MCV, MCHC, RDW, TLC, neutrophils or platelets. LDH showed a statistically significant positive Pearson correlation with lymphocyte percentage (r=0.471, p=0.042), but the corresponding Spearman correlation was not statistically significant (rho=0.314, p=0.190). This isolated finding was therefore treated as exploratory.

Age relationships

Age was not significantly correlated with blood glucose (r=-0.004, p=0.987), insulin (r=-0.070, p=0.775) or LDH (r=-0.103, p=0.674) in the present sample.

DISCUSSION

The analysis included 45 women aged 18–60 years and demonstrated strong positive interrelationships among blood glucose, serum insulin and LDH. In contrast, LDH showed no consistent association with most routine haematological parameters. These findings provide an exploratory basis for further investigation but should not be interpreted as evidence that these laboratory markers independently diagnose PCOS.

The strong glucose–insulin correlation is biologically plausible. Insulin resistance and compensatory hyperinsulinaemia are important components of PCOS pathophysiology [3,4]. Legro et al. demonstrated [13] the usefulness of

glucose-to-insulin measures in women with PCOS [13], while Majid et al. reported [14] HOMA-IR as a useful marker for insulin resistance [14]. In the present dataset, however, fasting status was not documented. Therefore, the strong correlation cannot be interpreted as a direct estimate of insulin resistance, and HOMA-IR could not be validly calculated.

The strong positive associations between glucose and LDH and between insulin and LDH are notable. LDH is a non-specific marker of cellular injury and metabolic stress. The proposal cites Wu et al. regarding altered LDH [11] in women with metabolic disturbances [11], and Wang et al. reported increased serum LDHA in PCOS [17]. These observations provide biological plausibility for further investigation of LDH in the metabolic context of PCOS, but they do not establish that LDH is specific to PCOS or that the association is independent of other metabolic factors.

LDH did not correlate significantly with haemoglobin, RBC count, PCV, MCV, MCHC, RDW, TLC, neutrophils or platelets. This finding does not support a generalized relationship between LDH and routine CBC parameters in the present sample. The isolated Pearson association with lymphocyte percentage was not reproduced by Spearman analysis and should therefore be regarded as exploratory. Previous literature cited in the proposal has described inflammatory haematological changes in PCOS [7,9,10], but the current results do not demonstrate a consistent LDH–CBC relationship.

The descriptive findings showed that 7 of 19 women had blood glucose above the laboratory reference limit, 2 had insulin above the stated upper reference limit and 11 had LDH above the stated upper reference limit. These values indicate a substantial burden of abnormal laboratory results in this selected clinical dataset. However, they cannot be used to diagnose diabetes, insulin resistance or PCOS because fasting status, clinical history and formal diagnostic criteria were not available.

The proposal identifies BMI, menstrual history, hirsutism, acne and other clinical characteristics as relevant to PCOS assessment [1,2,8]. These variables were not available in the analytical spreadsheet. Without these variables and a confirmed PCOS phenotype, it is not possible to determine whether the observed biochemical relationships differ between PCOS and non-PCOS women or among PCOS phenotypes.

The proposed multimarker concept remains potentially useful because glucose, insulin, LDH and CBC represent different biological domains. Nevertheless, a screening biomarker must be evaluated against an accepted clinical reference diagnosis and tested for diagnostic performance. Future work should recruit confirmed PCOS cases and appropriate controls, document fasting status, calculate HOMA-IR where appropriate, and evaluate sensitivity, specificity, ROC-AUC and multivariable models.

Overall, the updated dataset strengthens the exploratory evidence for an association between glucose, insulin and LDH but does not support a clear association between LDH and routine CBC parameters. The study therefore generates a focused hypothesis for larger studies rather than providing a validated PCOS screening algorithm.

OUTCOME OF THE STUDY

The principal outcome of the analysis was the identification of strong positive correlations among blood glucose, serum insulin and LDH in 45 women aged 18–60 years. In contrast, LDH did not show consistent correlations with the major CBC parameters. These findings suggest that LDH may be more closely related

to metabolic variables than to routine haematological indices in this dataset.

The findings should be considered hypothesis-generating. Because the spreadsheet did not document confirmed PCOS diagnosis or fasting status, the observed relationships cannot be attributed specifically to PCOS or used to establish diagnostic performance.

IMPLICATIONS OF THE STUDY

Clinical implication: The strong association of blood glucose with insulin emphasizes the value of assessing metabolic risk in women evaluated for PCOS, while recognizing that fasting status and clinical context are essential for interpretation.

Biochemical implication: The strong glucose/insulin–LDH relationships identify LDH as a potentially useful exploratory metabolic marker that warrants evaluation in a larger, clinically characterized PCOS cohort.

Haematological implication: The absence of consistent LDH–CBC associations suggests that routine CBC parameters should not be assumed to act as surrogate measures of LDH-related metabolic changes.

Research implication: Future studies should recruit confirmed PCOS cases with appropriate controls, document fasting status, BMI and PCOS phenotype, and include HOMA-IR and relevant hormonal parameters.

Screening implication: Diagnostic utility should be evaluated using a clinical reference diagnosis and formal diagnostic-performance measures before proposing a multimarker screening tool.

Health-system implication: The use of routinely available laboratory tests may be practical in resource-limited settings, but cost-effectiveness and clinical utility require prospective evaluation.

LIMITATIONS

- Only 45 women aged 18–60 years were available for the primary analysis,
- Fasting status was not documented, preventing valid calculation of HOMA-IR from the available data.
- BMI in term of obesity, menstrual history in term of regular, irregular, were mentioned. The androgen measurements, ovarian morphology and treatment history were not available in the analytical dataset.
- The dataset represents a selected clinical population and may not be generalizable to the wider population.

CONCLUSION

In the dataset of 45 women aged 18–60 years, blood glucose, serum insulin and LDH showed strong positive correlations, whereas LDH was not consistently associated with routine haematological parameters. These findings support further investigation of LDH as an exploratory metabolic marker alongside glucose and insulin in women evaluated for PCOS. Larger prospective studies with confirmed PCOS diagnosis, appropriate controls, standardized fasting samples, HOMA-IR and relevant clinical and hormonal variables are required.

ETHICAL CONSIDERATIONS

The proposal states that Institutional Ethics Committee approval and written informed consent were to be obtained and participant confidentiality maintained. The verified IEC approval number recorded in the manuscript is DYP/IECBH/2026/79.

FUNDING: No internal and external funding was obtained.

CONFLICT OF INTEREST: The authors declare no conflict of interest.

DECLARATION OF ARTIFICIAL INTELLIGENCE IN SCIENTIFIC WRITING

Generative artificial intelligence tools were used to assist with language refinement, organization and formatting of the manuscript. The authors reviewed and verified the scientific content, interpretation and final version and remain fully responsible for the accuracy and integrity of the work. AI was not used to generate figures or to replace statistical or scientific judgment.

AUTHOR CONTRIBUTIONS

Conceptualization and study design: Dr. Pradnya Padalkar, Dr. Shital Naikwade; Dr. Surekha Patil

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Manuscript drafting & Result analysis: Dr. Pradnya Padalkar,Dr. Sushil Agarwal, Dr Anita Bhosale

Critical revision and final approval: All authors

Accountability: All authors agree to be accountable for all aspects of the work and to ensure the accuracy and integrity of the study.

Authorship Statement: All authors should confirm that they fulfil all four ICMJE authorship criteria, including substantial contribution to the work, drafting or critical revision of the manuscript, final approval of the version to be published, and accountability for the integrity of the work.

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