JURNAL SAINTIFIK
MULTI SCIENCE JOURNAL
PISSN 1693-668X
EISSN 2829-3975

Serum oxidative stress and vasoactive biomarkers in women with preeclampsia: a comparative cross-sectional study

Biomarker Stres Oksidatif Dan Vasoaktif Serum Pada Ibu Hamil Dengan Preeklampsia: Studi Potong Lintang Komparatif

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ARTICLE HISTORY
Received [19 April 2026]
Revised [30 April 2026]
Accepted [18 May 2026]

KEYWORDS
Preeclampsia; Oxidative Stress; Coenzyme Q10; Kallikrein–Kinin System; Renin–Angiotensin System

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ABSTRAK

Latar belakang: Preeklampsia berkaitan dengan stres oksidatif dan perubahan jalur vasoaktif. Penelitian ini membandingkan enam biomarker serum pada ibu hamil dengan preeklampsia dan ibu hamil normotensif di Irak. **Metode:** Studi potong lintang komparatif dilakukan pada Februari–April 2026 dengan melibatkan 40 ibu hamil dengan preeklampsia dan 20 kontrol ibu hamil sehat. Kadar koenzim Q10 (CoQ10), malondialdehida (MDA), renin, angiotensin-converting enzyme (ACE), kalikrein-1 (KLK1), dan bradikinin (BK) diukur menggunakan ELISA. Analisis meliputi perbandingan antarkelompok, korelasi Pearson, dan kurva receiver operating characteristic (ROC) secara eksploratif. **Hasil:** Rerata kadar MDA (5,97 ± 0,45 vs. 2,67 ± 0,35 nmol/mL), renin (2,35 ± 0,29 vs. 1,86 ± 0,54 pg/mL), ACE (1,08 ± 0,37 vs. 0,76 ± 0,16 U/L), dan KLK1 (1,44 ± 0,49 vs. 1,10 ± 0,36 pg/mL) lebih tinggi pada kelompok preeklampsia. Sebaliknya, CoQ10 (22,44 ± 20,56 vs. 39,43 ± 13,83 µg/mL) dan BK (0,72 ± 0,22 vs. 0,99 ± 0,27 ng/mL) lebih rendah. MDA menunjukkan AUC 1,00 pada sampel penelitian, sedangkan KLK1 berkorelasi positif dengan BK pada kelompok preeklampsia (r = 0,55). **Kesimpulan:** Profil biomarker stres oksidatif dan vasoaktif berbeda antara kedua kelompok; pemanfaatan diagnostiknya memerlukan validasi pada populasi yang lebih besar dan representatif.

ABSTRACT

Background: *Preeclampsia is associated with oxidative imbalance and altered vasoactive pathways. This study compared six serum biomarkers between women with preeclampsia and normotensive pregnant controls in Iraq.* ***Methods:*** *In a comparative cross-sectional study conducted from February to April 2026, 40 women with preeclampsia and 20 healthy pregnant controls were enrolled. Serum coenzyme Q10 (CoQ10), malondialdehyde (MDA), renin, angiotensin-converting enzyme (ACE), kallikrein-1 (KLK1), and bradykinin (BK) were measured using ELISA. Group comparisons, Pearson correlations, and exploratory receiver operating characteristic (ROC) analyses were performed.* ***Results:*** *Mean MDA (5.97 ± 0.45 vs. 2.67 ± 0.35 nmol/mL), renin (2.35 ± 0.29 vs. 1.86 ± 0.54 pg/mL), ACE (1.08 ± 0.37 vs. 0.76 ± 0.16 U/L), and KLK1 (1.44 ± 0.49 vs. 1.10 ± 0.36 pg/mL) were higher in the preeclampsia group. CoQ10 (22.44 ± 20.56 vs. 39.43 ± 13.83 µg/mL) and BK (0.72 ± 0.22 vs. 0.99 ± 0.27 ng/mL) were lower. MDA yielded an area under the ROC curve of 1.00 within the study sample, and KLK1 correlated positively with BK among cases (r = 0.55).* ***Conclusion:*** *Oxidative stress and vasoactive biomarker profiles differed between groups; their diagnostic relevance requires validation in larger, representative cohorts.*
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INTRODUCTION

Preeclampsia is a hypertensive disorder of pregnancy characterised by the new onset of hypertension after 20 weeks of gestation along with proteinuria and/or evidence of maternal organ or uteroplacental dysfunction. The presence of proteinuria is not essential in the presence of other established diagnostic features (Khan et al., 2022; Vera-Ponce et al., 2025; American College of Obstetricians and Gynaecologists [ACOG], 2020). It continues to be a major cause of maternal and perinatal morbidity.

Normal pregnancy requires trophoblast invasion and remodeling of the maternal spiral arteries to establish a low-resistance uteroplacental circulation. Defective vascular remodeling and placental hypoxia

JURNAL SAINTIFIK
MULTI SCIENCE JOURNAL
PISSN 1693-668X
EISSN 2829-3975

are implicated in the endothelial disturbance and oxidative injury associated with preeclampsia (Haram et al., 2020; Aouache et al., 2018; Colson et al., 2021). Placental oxidative stress and systemic endothelial dysfunction can adversely affect vascular adaptation and maternal organ function (Schoots et al., 2018; Boeldt & Bird, 2017).

Malondialdehyde (MDA), a product of lipid peroxidation, provides an indirect indication of oxidative damage, although it is not specific to preeclampsia. The relevance of oxidative stress biomarkers to pregnancy complications has been discussed in general reviews and disease-specific studies (Kıran et al., 2023; Liu et al., 2025). Systematic reviews also support investigation of their clinical relevance while highlighting heterogeneity between populations and assays (Freire et al., 2023; Afrose et al., 2022).

Coenzyme Q10 (CoQ10) contributes to mitochondrial electron transport and antioxidant defense. Reports have investigated its circulating concentration, redox biology, and potential role in preeclampsia (Roland et al., 2010; Teran et al., 2003; Teran et al., 2018; Deer et al., 2025). Broader studies address CoQ10 supplementation or its association with markers of inflammation and oxidative stress (Shaikh et al., 2021; Dabbaghi Varnousfaderani et al., 2023), while the review by Kudalkar (2025) discusses its systemic biochemical functions.

Vascular homeostasis during pregnancy is also influenced by the renin–angiotensin system (RAS) and the kallikrein–kinin system (KKS). These pathways participate in the regulation of vascular tone, inflammation, and permeability, but their circulating measurements cannot be assumed to represent activity throughout all tissues (Lumbers & Pringle, 2014; Bekassy et al., 2022). Research on ACE2 and placental RAS components, as well as studies of placental angiotensin metabolism, illustrates the complexity of these pathways in preeclampsia (Tamanna et al., 2020; Sun et al., 2021; Stettner-Kołodziejska et al., 2022). Investigations of kallikrein generation further suggest changes in the contact-system pathway (Godtfredsen et al., 2022), although this measure differs from serum kallikrein-1 (KLK1).

Evidence remains limited on the joint serum profile of oxidative stress markers and selected RAS and KKS components among Iraqi pregnant women. The present study therefore compared CoQ10, MDA, renin, ACE, KLK1, and bradykinin (BK) between women clinically classified with preeclampsia and normotensive pregnant controls. It also explored within-group biomarker correlations and their apparent ability to distinguish the two study groups. The study hypothesized that oxidative stress and vasoactive marker distributions would differ between groups; the correlation and ROC analyses were exploratory.

RESEARCH METHOD

A comparative cross-sectional study was conducted from February to April 2026 among 60 pregnant women attending private outpatient clinics in Iraq. The sample included 40 women diagnosed with preeclampsia and 20 normotensive pregnant controls. The preeclampsia group was identified through physician-recorded diagnoses after 24 weeks of gestation; ACOG Practice Bulletin No. 222 guided interpretation of the clinical diagnostic framework (ACOG, 2020). Women with preexisting chronic hypertension, diabetes mellitus, or chronic kidney disease were excluded. Age, body mass index (BMI), gestational age, blood pressure, and proteinuria were obtained from questionnaires and available clinical records.

Five milliliters of venous blood were collected in plain plastic tubes without anticoagulant and allowed to clot at room temperature for approximately 10 minutes. Specimens were centrifuged at 3,000 rpm for 15 minutes, and serum was transferred to microcentrifuge tubes and stored at −20 °C. Samples were thawed once before analysis; a a protease inhibitor to reduce peptide degradation. Serum biomarkers were measured with SUNLONG Biotech (China) ELISA kits: CoQ10 (SL2187Hu), MDA (SL1135Hu), renin (SL1528Hu), ACE (SL0120Hu), KLK1 (SL1023Hu), and BK (SL0370Hu). Assays were performed according to the manufacturer's instructions. Urinary protein was assessed by dipstick and recorded as 1+, 2+, or 3+ in women with preeclampsia.

The principal variable used to group the women was the diagnosis of pre-eclampsia, as recorded by the physician. Biomarker measures were treated as continuous variables. All participants enrolled were included in the reported analyses, and the study reported no missing values for variables in the final analytical dataset. The study was performed according to the Declaration of Helsinki. A local ethics committee gave approval for the study (approval No. 2390; 31 January 2026) and consent was obtained from participants before samples were taken.

The data were analysed using SPSS version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical data as counts and percentages. Group comparisons were conducted using independent-samples t-tests. Welch's t-test was reported for between-group biomarker comparisons. 2 Pearson correlation coefficients (r) of pairwise associations of biomarkers in each group. Exploratory ROC analyses were used to estimate area under the curve (AUC), thresholds, sensitivity and specificity. All tests reported were two-sided, and $p < 0.05$ was

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PISSN 1693-668X
EISSN 2829-3975

considered statistically significant. These nominal p-values and ROC estimates should be interpreted with caution as multiple tests were conducted on a small sample without reporting for multiplicity adjustment or independent validation. This observational study was reported according to STROBE principles. Its exploratory discrimination analysis is presented in a descriptive fashion, rather than as a clinically validated diagnostic accuracy study .

RESULTS AND DISCUSSION

Participant Characteristics

Women with preeclampsia were slightly older than controls (29.5 ± 4.11 vs. 27.4 ± 3.01 years; p = 0.029). Systolic and diastolic blood pressure were higher in the preeclampsia group (both p < 0.001), whereas BMI and gestational age were not significantly different in the unadjusted comparisons. Among women with preeclampsia, dipstick proteinuria was 1+ in seven (17.5%), 2+ in eight (20.0%), and 3+ in 25 (62.5%) participants. Proteinuria was not recorded in the control group (Table 1).

Table 1. Demographic and clinical characteristics of women with preeclampsia and healthy pregnant controls.

Variable	Dipstick	Preeclampsia (n = 40)	Controls (n = 20)	p-value
Age (years)	—	29.5 ± 4.11	27.4 ± 3.01	0.029
BMI (kg/m²)	—	29.22 ± 1.33	28.6 ± 1.40	0.099
SBP (mmHg)	—	161.6 ± 20.8	111.0 ± 15.4	< 0.001
DBP (mmHg)	—	109.5 ± 16.0	72.4 ± 13.0	< 0.001
Gestational age (weeks)	—	26.55 ± 5.24	24.65 ± 7.99	0.343
Proteinuria (n, %)	1+	7 (17.5%)	0	—
	2+	8 (20.0%)	0	—
	3+	25 (62.5%)	0	—

Values are mean ± SD or n (%). BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure.

Serum Oxidative Stress and Vasoactive Biomarkers

The preeclampsia group had higher mean MDA, ACE, KLK1, and renin values than healthy pregnant controls. Conversely, mean CoQ10 and BK values were lower in the preeclampsia group. All six between-group comparisons reached nominal significance (Table 2). These findings describe cross-sectional group differences rather than changes over time.

Table 2. Serum oxidative stress and vasoactive biomarkers in women with preeclampsia and healthy pregnant controls.

Biomarker	Preeclampsia (n = 40)	Controls (n = 20)	Difference (95% CI)	p-value
CoQ10 (µg/mL)	22.44 ± 20.56	39.43 ± 13.83	−16.99 (−25.99 to −7.99)	0.0004
MDA (nmol/mL)	5.97 ± 0.45	2.67 ± 0.35	3.30 (3.09 to 3.51)	< 0.001
ACE (U/L)	1.08 ± 0.37	0.76 ± 0.16	0.32 (0.18 to 0.46)	0.00002
BK (ng/mL)	0.72 ± 0.22	0.99 ± 0.27	−0.27 (−0.41 to −0.13)	0.00049
KLK1 (pg/mL)	1.44 ± 0.49	1.10 ± 0.36	0.34 (0.12 to 0.56)	0.004
Renin (pg/mL)	2.35 ± 0.29	1.86 ± 0.54	0.49 (0.22 to 0.76)	0.00085

Values are mean ± SD. Differences are calculated as preeclampsia minus controls. The original analysis reports 95% confidence intervals and Welch's t-tests.

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PISSN 1693-668X
EISSN 2829-3975

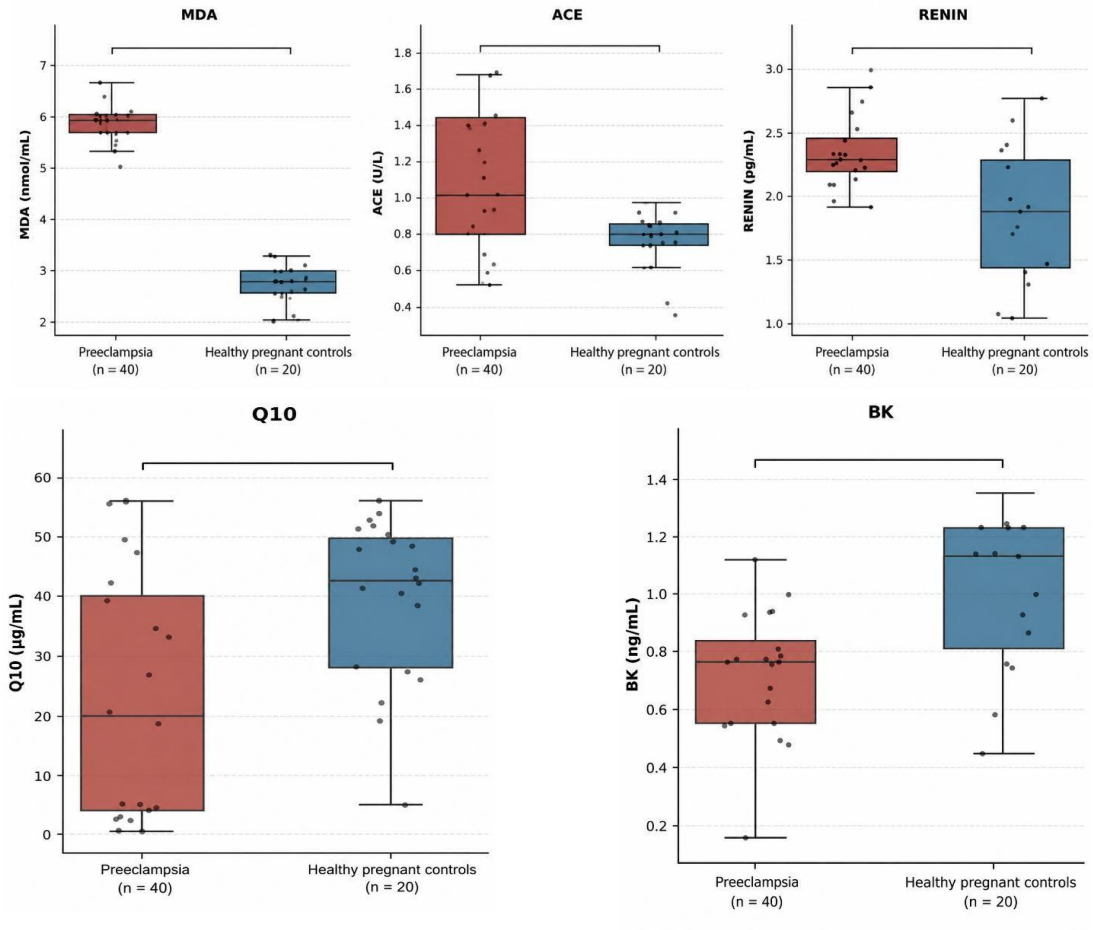


Figure 1. Distribution of selected serum oxidative stress and vasoactive biomarkers in women with preeclampsia and healthy pregnant controls.

Within-Group Correlations

In the preeclampsia group, KLK1 and BK were positively correlated (r = 0.55; p = 0.00024), while CoQ10 and MDA also showed a positive correlation (r = 0.44; p = 0.0045). ACE and renin exhibited a weaker positive association (r = 0.32; p = 0.0441). Other pairwise coefficients were smaller or were not statistically significant at the nominal threshold (Table 3). Figure 2 illustrates the positive KLK1–BK association in participants with preeclampsia.

Table 3. Pearson correlations among serum biomarkers in women with preeclampsia (n = 40).

biomarkers	CoQ10	MDA	ACE	BK	KLK1	Renin
CoQ10	1.00	0.44 p = 0.0045	0.04 p = 0.806	-0.21 p = 0.193	-0.16 p = 0.3240	0.09 p = 0.5807
MDA	0.44 p = 0.0045	1.00	0.10 p = 0.5392	-0.10 p = 0.5393	0.16 p = 0.3240	-0.28 p = 0.0801
ACE	0.04 p = 0.806	0.10 p = 0.5392	1.00	-0.14 p = 0.3889	-0.10 p = 0.5392	0.32 p = 0.0441
BK	-0.21 p = 0.193	-0.10 p = 0.5393	-0.14 p = 0.3889	1.00	0.55 p = 0.00024	-0.04 p = 0.8064
KLK1	-0.16 p = 0.3240	0.16 p = 0.3240	-0.10 p = 0.5392	0.55 p = 0.00024	1.00	-0.01 p = 0.9511
Renin	0.09 p = 0.5807	-0.28 p = 0.0801	0.32 p = 0.0441	-0.04 p = 0.8064	-0.01 p = 0.9511	1.00

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Cells show Pearson r and the corresponding nominal p-value. CoQ10: coenzyme Q10; MDA: malondialdehyde; ACE: angiotensin-converting enzyme; BK: bradykinin; KLK1: kallikrein-1.

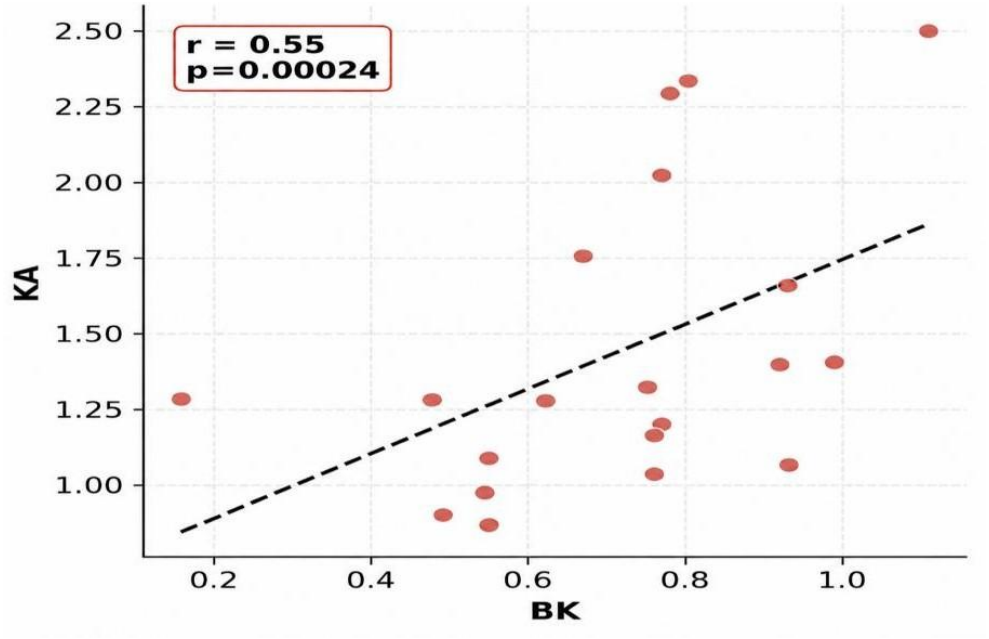


Figure 2. Positive correlation between serum bradykinin and kallikrein-1 in women with preeclampsia.

In healthy pregnant controls, CoQ10 and BK were inversely correlated (r = −0.57; p = 0.0086), as shown in Table 4 and Figure 3. No other pairwise correlations reached the nominal p < 0.05 threshold within the control group. These analyses were exploratory and did not establish causal relationships between biomarkers.

Table 4. Pearson correlations among serum biomarkers in healthy pregnant controls (n = 20).

Biomarker	CoQ10	MDA	ACE	BK	KLK1	Renin
CoQ10	1.00	-0.32 p = 0.1690	0.02 p = 0.9333	-0.57 p = 0.0086	0.13 p = 0.5848	0.19 p = 0.4223
MDA	-0.32 p = 0.1690	1.00	0.11 p = 0.6443	0.34 p = 0.1424	-0.26 p = 0.2682	0.20 p = 0.3978
ACE	0.02 p = 0.9333	0.11 p = 0.6443	1.00	-0.06 p = 0.8016	0.43 p = 0.0584	0.24 p = 0.3081
BK	-0.57 p = 0.0086	0.34 p = 0.1424	-0.06 p = 0.8016	1.00	-0.30 p = 0.1987	-0.40 p = 0.0805
KLK1	0.13 p = 0.5848	-0.26 p = 0.2682	0.43 p = 0.0584	-0.30 p = 0.1987	1.00	0.30 p = 0.1987
Renin	0.19 p = 0.4223	0.20 p = 0.3978	0.24 p = 0.3081	-0.40 p = 0.0805	0.30 p = 0.1987	1.00

Cells show Pearson r and the corresponding nominal p-value. Values have not been adjusted for multiple testing.

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EISSN 2829-3975

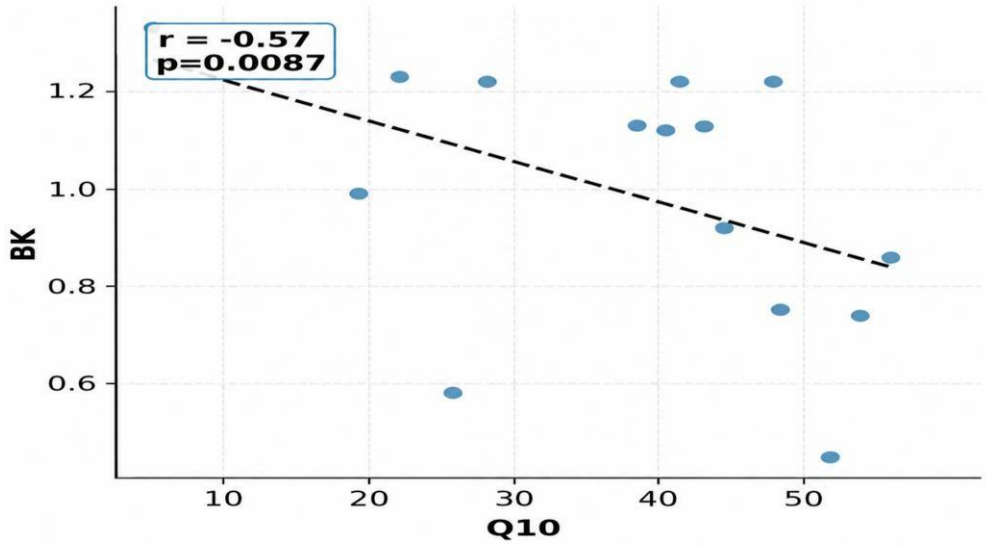


Figure 3. Negative correlation between serum bradykinin and coenzyme Q10 in healthy pregnant controls.

Exploratory ROC Analysis

ROC analysis evaluated the apparent discrimination between the two study groups for each marker. MDA showed perfect separation within this sample (AUC = 1.000; reported sensitivity and specificity = 100% at the selected cutoff of 5.07 nmol/mL). The AUC values for BK, renin, ACE, CoQ10, and KLK1 were 0.777, 0.760, 0.747, 0.746, and 0.680, respectively (Table 5).

Table 5. Exploratory receiver operating characteristic analysis for individual serum biomarkers.

Biomarker	AUC (95% CI)	p-value	Cutoff	Sensitivity (%)	Specificity (%)
MDA	1.000 (1.000–1.000)	< 0.001	5.07	100	100
BK	0.777 (0.623–0.930)	< 0.001	1.11	100	53
Renin	0.760 (0.602–0.918)	0.001	2.09	90	67
ACE	0.747 (0.585–0.909)	0.003	0.99	55	100
CoQ10	0.746 (0.593–0.900)	0.002	20.50	55	90
KLK1	0.680 (0.503–0.857)	0.047	1.04	85	53

Cutoffs are reported as provided in the original analysis; their selection method was not documented. Units are given in Table 2. These are in-sample exploratory estimates.

Interpretation and Comparison With Previous Studies

The principal finding is a distinct cross-sectional pattern of oxidative stress and vasoactive biomarkers in women with preeclampsia compared with normotensive pregnant controls. Their higher blood pressures are consistent with the clinical phenotype of preeclampsia. The groups also differed in mean age, so age-related influences cannot be excluded. Existing research has examined maternal age, obesity, placental pathology, and clinical management as important aspects of preeclampsia (Sheen et al., 2020; Durst et al., 2016; Weiner et al., 2018; Nirupama et al., 2021). Because the present comparisons were unadjusted, none of the measured group differences should be interpreted as independent of these or other unmeasured clinical factors.

The increased MDA together with the decreased CoQ10 in the preeclampsia group is consistent with an imbalance between lipid peroxidation and antioxidant defences . This pattern is consistent with reports of decreased circulating CoQ10 in preeclampsia (Teran et al., 2003; Teran et al., 2018) and general evidence linking oxidative stress markers to the condition (Aouache et al., 2018; Freire et al., 2023; Afrose et al., 2022). However, serum MDA is not specific for disease and total CoQ10 does not directly reflect its oxidized:reduced

redox ratio. Serum biomarker differences provide a rationale for further biological investigation, but do not confirm a particular pathogenic mechanism or the efficacy of CoQ10 supplementation (Shaikh et al., 2021). Higher serum renin and ACE values reflect variation in selected components of the RAAS. However, they do not themselves show activation of the entire RAS or increased angiotensin II signalling, which was not measured. Regulation of RAS in pregnancy involves both circulating and local tissue compartments (Lumbers & Pringle, 2014). Moreover, the studies on ACE2, placental RAS markers and placental angiotensin metabolism use different biological matrices and assays (Tamanna et al., 2020; Sun et al., 2021; Stettner-Kołodziejska et al., 2022) and their results cannot be directly compared to serum ACE measurements.

Women with preeclampsia also exhibited higher KLK1 and lower BK than controls, and within the affected group, the two biomarkers were positively correlated. This apparent difference in the average values of the groups does not necessarily contradict a positive correlation within each group: between-group comparison and within-group association are different statistical relationships. Although studies of kallikrein generation and contact activation (Godtfredsen et al., 2022) provide a relevant context, circulating KLK1 concentration and kallikrein-generating capacity are not interchangeable measures. Crosstalk between RAS, KKS and complement pathways is biologically plausible (Bekassy et al., 2022), but causation cannot be inferred from these serum data.

The inverse correlation between CoQ10 and BK observed only in controls should be considered as a hypothesis-generating finding. Exploratory analysis of multiple marker pairs increases the chance of nominally significant associations. The relatively small control group further limits precision. The distribution of proteinuria is descriptive rather than a classification of severity: dipstick results are semiquantitative and should not independently define the severity of preeclampsia (Okamoto et al., 2022).

MDA achieved an AUC of 1.000, but this is an estimate for the sampled cases and healthy controls only. Apparent performance may be inflated by chosen thresholds, possible differences in the spectrum and lack of external validation. Other markers show their utility alone or in combination as well, but their AUCs are lower. Future studies should incorporate prospective recruitment, standardised assays, prespecified thresholds, clinically relevant comparator groups, and validation cohorts before diagnostic use is entertained.

Study Limitations

The study enrolled only 60 participants from out-patients settings and the distribution between cases and controls was uneven. There was no a priori justification of sample size. Recruitment and gestational age matching were not fully described, maternal age and other potential confounders were not adjusted for in the analysis and no correction was made for multiple comparisons. Selection of ROC threshold and independent validation were not described. The cross-sectional design precludes conclusions of temporal or causal relationships. These limitations limit generalisability and interpretation of estimated biomarker performance.

CONCLUSION

In this comparative cross-sectional sample, serum MDA, renin, ACE and KLK1 were higher and CoQ10 and BK were lower in women with preeclampsia compared to healthy pregnant controls. In preeclampsia, KLK1 and BK showed a positive correlation, while CoQ10 and BK showed a negative correlation in the control group. MDA needs to be replicated, not used clinically right away. Good discrimination in-sample. Larger prospective studies with confounder adjustment and independent validation are needed to establish whether these biomarkers provide clinically meaningful information .

ETHICS AND DECLARATIONS

Ethical Approval and Consent

The study followed the Declaration of Helsinki. Ethical approval was reported under No. 2390 (31 January 2026), and informed consent was obtained before specimen collection.

Data Availability

Data may be requested from the corresponding author at the email address provided above.

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MULTI SCIENCE JOURNAL
PISSN 1693-668X
EISSN 2829-3975

Competing Interests
The author declared no competing interests.

Funding
No funding was declared.

Author Contributions
Shahad Idrees Mohammed was responsible for the study concept, design, implementation, analysis, and manuscript preparation.

Acknowledgments
No acknowledgments were reported.

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JURNAL SAINTIFIK
MULTI SCIENCE JOURNAL
PISSN 1693-668X
EISSN 2829-3975

ABBREVIATIONS

Abbreviation	Full name
ACOG	American College of Obstetricians and Gynecologists
ACE	Angiotensin-converting enzyme
AUC	Area under the curve
BK	Bradykinin
BMI	Body mass index
CoQ10	Coenzyme Q10
ELISA	Enzyme-linked immunosorbent assay
KKS	Kallikrein–kinin system
KLK1	Kallikrein-1
MDA	Malondialdehyde
PE	Preeclampsia
RAS	Renin–angiotensin system
ROC	Receiver operating characteristic
SPSS	Statistical Package for the Social Sciences
STARD	Standards for Reporting Diagnostic Accuracy Studies