



Original Article

Comparative Evaluation of Serum Uric Acid and Selected Biochemical Parameters in Preeclamptic and Normotensive Pregnancies and Their Association with Maternal–Fetal Outcomes

Manpreet Saini^{1*}, Sarat Chandra Nag², Debendra Kumar Naik³, Vijay Kumar⁴

¹Associate Professor, Department of Biochemistry, Laxmi Chandravansi Medical College & Hospital, Bishrampur, Palamu, Jharkhand, India.

²Assistant professor, Department of Community Medicine, Bhima Bhoi Medical College, Balangir, Odisha, India.

³Assistant professor, Department of Obstetrics & Gynaecology, Bhima Bhoi Medical College, Balangir, Odisha, India.

⁴Professor, Department of Community Medicine, Laxmi Chandravansi Medical College & Hospital, Bishrampur, Palamu, Jharkhand, India.

 OPEN ACCESS

Corresponding Author:

Manpreet Saini

Associate Professor, Department of Biochemistry, Laxmi Chandravansi Medical College & Hospital, Bishrampur, Palamu, Jharkhand, India.

Received: 03-01-2026

Accepted: 25-01-2026

Available online: 28-02-2026

Copyright © International Journal of Medical and Pharmaceutical Research

ABSTRACT

Introduction- Preeclampsia, a pregnancy-specific hypertensive disorder, causes significant maternal and fetal complications. Endothelial dysfunction, oxidative stress, and inflammation underlie its pathophysiology. Biochemical markers, including serum uric acid, lactate dehydrogenase (LDH), and C-reactive protein (CRP), reflect disease severity and adverse outcomes. This study evaluates these markers in preeclamptic versus normotensive pregnancies to predict maternal–fetal risks.

Material and Method- The present hospital-based comparative cross-sectional study enrolled 120 pregnant women (60 preeclampsia, 60 normotensive). Clinical, obstetric, and biochemical data (uric acid, LDH, CRP, urea, creatinine, AST, ALT) were collected. Maternal and fetal outcomes were assessed. Statistical analyses included t-tests, Chi-square, and multivariate logistic regression to identify predictors of adverse outcomes. A p-value <0.05 was considered statistically significant.

Result- In this study of 120 pregnant women, preeclampsia was associated with higher BMI, blood pressure, proteinuria, and cesarean delivery. Preeclamptic women showed elevated uric acid, LDH, CRP, urea, creatinine, AST, and ALT, with levels rising in severe cases. Adverse maternal–fetal outcomes were more frequent in preeclampsia, and multivariate analysis identified uric acid, LDH, creatinine, and CRP as independent predictors of complications.

Conclusion- Preeclampsia is associated with elevated uric acid, LDH, CRP, creatinine, and hepatic enzymes, correlating with adverse maternal–fetal outcomes. Uric acid, LDH, creatinine, and CRP are key predictors, supporting their use in early risk stratification and clinical monitoring.

Keywords: Preeclampsia, maternal–fetal outcomes, pregnancy, uric acid, LDH, CRP etc.

INTRODUCTION

Preeclampsia is a pregnancy-specific hypertensive disorder characterized by new-onset hypertension and proteinuria after 20 weeks of gestation, contributing significantly to maternal and perinatal morbidity and mortality worldwide. It affects approximately 3–5% of pregnancies and remains a major public health concern, particularly in developing countries [1]. The condition is associated with serious maternal complications such as eclampsia, HELLP (hemolysis, elevated liver enzymes and low platelet count) syndrome, renal failure, and increased risk of cardiovascular disease later in life, while fetal complications include intrauterine growth restriction (IUGR), prematurity, and perinatal death [1,2]. The exact

pathophysiology of preeclampsia remains incompletely understood; however, endothelial dysfunction, oxidative stress, abnormal placentation, and systemic inflammation are considered central mechanisms [2,3]. In this context, biochemical markers reflecting these underlying processes have gained considerable attention for early detection and prognostication. Among these, serum uric acid has emerged as a potential marker due to its association with impaired renal function, oxidative stress, and endothelial injury [4]. Elevated maternal uric acid levels have been shown to correlate with the severity of preeclampsia and adverse maternal–fetal outcomes, including low birth weight and preterm delivery [5,6].

In addition to uric acid, other biochemical parameters such as lactate dehydrogenase (LDH) and inflammatory markers like C-reactive protein (CRP) have been investigated. LDH, an intracellular enzyme released during cellular damage, reflects tissue injury and hemolysis, both of which are prominent in severe preeclampsia [2,7]. Studies have demonstrated that elevated LDH levels are significantly associated with disease severity and poor fetomaternal outcomes [7,8]. Similarly, CRP, an acute-phase reactant, indicates systemic inflammation and has been linked with the pathogenesis and progression of preeclampsia [3]. Recent systematic reviews and meta-analyses have further strengthened the role of biochemical markers, particularly uric acid, in predicting adverse pregnancy outcomes and aiding in risk stratification [6,9]. Despite these advances, there remains variability in findings across populations, and the combined evaluation of multiple biochemical parameters may provide better clinical insight compared to a single marker. Therefore, the present study aims to comparatively evaluate serum uric acid and selected biochemical parameters in preeclamptic and normotensive pregnancies and to determine their association with maternal and fetal outcomes. Such an approach may help in identifying reliable, cost-effective biomarkers for early diagnosis, severity assessment, and improved management of preeclampsia.

MATERIAL AND METHOD-

This hospital-based comparative cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynecology at Laxmi Chandravansi Medical College and Hospital, in Palamu region of Jharkhand over a six-month period from May 2025 to October 2025. Ethical clearance was obtained from the Institutional Ethics Committee prior to the study.

A total of 120 pregnant women were enrolled and divided into two groups: 60 women diagnosed with preeclampsia and 60 age- and gestational age-matched normotensive pregnant women serving as controls. Preeclampsia was defined as blood pressure $\geq 140/90$ mmHg on two occasions at least four hours apart after 20 weeks of gestation, along with proteinuria ($\geq 1+$ on dipstick or ≥ 300 mg/24 hours). Women with chronic hypertension, multiple pregnancies, systemic illnesses, or those unwilling to participate were excluded from the study.

After obtaining informed consent, detailed clinical and obstetric data were collected using a predesigned proforma. Parameters recorded included maternal age, body mass index (BMI), parity, family history of hypertension, previous history of preeclampsia, gestational age, blood pressure, proteinuria, and mode of delivery. Maternal outcomes such as severity of preeclampsia and complications (eclampsia, HELLP syndrome) were noted. Fetal outcomes assessed included birth weight, preterm delivery, APGAR score at 5 minutes, and neonatal intensive care unit (NICU) admission.

Approximately 5 mL of venous blood was collected under aseptic conditions. Serum was separated by centrifugation and analyzed for biochemical parameters including serum uric acid, lactate dehydrogenase (LDH), C-reactive protein (CRP), urea, creatinine, aspartate aminotransferase (AST), and alanine aminotransferase (ALT). Biochemical parameters were estimated using standard kinetic enzymatic methods, while CRP was measured by immunoturbidimetric assay on an automated analyzer.

Preeclamptic women were further categorized into mild ($n=32$) and severe ($n=28$) groups based on ACOG 2020 criteria. Biochemical parameters were compared between study groups and according to disease severity. Adverse maternal–fetal outcomes evaluated included severe preeclampsia, eclampsia, HELLP syndrome, preterm delivery, low birth weight (<2.5 kg), NICU admission, and APGAR score <7 at 5 minutes.

Statistical analysis was performed using SPSS version 20. Continuous variables were expressed as mean \pm standard deviation and compared using Student's t-test, while categorical variables were analyzed using the Chi-square test. Multivariate logistic regression analysis was performed to identify independent biochemical predictors of adverse maternal–fetal outcomes, with adjusted odds ratios (OR) and 95% confidence intervals (CI). A p-value <0.05 was considered statistically significant.

RESULT-

A total of 120 pregnant women were enrolled in the study, including 60 women with preeclampsia and 60 normotensive controls. The demographic and clinical characteristics of the study participants are summarized in Table 1. The mean age of women in the preeclampsia group was 26.8 ± 3.5 years, which was comparable to 25.9 ± 3.2 years in the normotensive group ($p > 0.05$). Gestational age was also similar between the two groups (35.2 ± 2.4 weeks vs. 36.1 ± 2.1 weeks; $p >$

0.05). Body mass index (BMI) was significantly higher in women with preeclampsia ($27.1 \pm 3.2 \text{ kg/m}^2$) compared to normotensive pregnant women ($25.8 \pm 2.9 \text{ kg/m}^2$; $p < 0.05$). As expected, both systolic and diastolic blood pressures were significantly elevated in the preeclampsia group ($154.6 \pm 10.2 \text{ mmHg}$ and $98.3 \pm 6.5 \text{ mmHg}$, respectively) compared to controls ($118.4 \pm 8.6 \text{ mmHg}$ and $76.2 \pm 5.4 \text{ mmHg}$; $p < 0.001$). Proteinuria was markedly higher in preeclamptic women ($312 \pm 85 \text{ mg/dL}$) than in normotensive participants ($58 \pm 12 \text{ mg/dL}$; $p < 0.001$). A higher proportion of women in the preeclampsia group had a family history of hypertension (41.7% vs. 20%; $p < 0.05$). Mode of delivery also differed significantly, with cesarean delivery being more frequent in preeclamptic women (38/60) compared to normotensive women (22/60; $p < 0.01$).

Table 1- Demographic and clinical characteristics of participants

Parameter	Preeclampsia (n=60)	Normotensive (n=60)	p-value
Age (years)	26.8 ± 3.5	25.9 ± 3.2	>0.05
Gestational age (weeks)	35.2 ± 2.4	36.1 ± 2.1	>0.05
BMI (kg/m^2)	27.1 ± 3.2	25.8 ± 2.9	<0.05
Systolic BP (mmHg)	154.6 ± 10.2	118.4 ± 8.6	<0.001
Diastolic BP (mmHg)	98.3 ± 6.5	76.2 ± 5.4	<0.001
Proteinuria (mg/dL)	312 ± 85	58 ± 12	<0.001
Family history of hypertension n (%)	25 (41.7%)	12 (20%)	<0.05
Mode of delivery (Cesarean / Vaginal) n	38 / 22	22 / 38	<0.01

Table 2 compares the biochemical parameters between preeclamptic and normotensive pregnant women. Serum uric acid levels were significantly higher in the preeclampsia group ($6.8 \pm 1.2 \text{ mg/dL}$) compared to controls ($4.2 \pm 0.8 \text{ mg/dL}$; $p < 0.001$). Markers of tissue injury and inflammation, including lactate dehydrogenase (LDH) and C-reactive protein (CRP), were also markedly elevated in preeclamptic women (LDH: $620.5 \pm 140.3 \text{ U/L}$ vs. $320.4 \pm 85.6 \text{ U/L}$; CRP: $9.6 \pm 3.1 \text{ mg/L}$ vs. $3.8 \pm 1.5 \text{ mg/L}$; $p < 0.001$). Renal function parameters showed significant derangement, with higher serum urea ($38.6 \pm 10.4 \text{ mg/dL}$ vs. $24.8 \pm 6.2 \text{ mg/dL}$) and creatinine levels ($1.02 \pm 0.28 \text{ mg/dL}$ vs. $0.72 \pm 0.15 \text{ mg/dL}$; $p < 0.001$). Similarly, hepatic enzymes were significantly elevated in the preeclampsia group (AST: $58.4 \pm 20.6 \text{ U/L}$ vs. $28.3 \pm 10.2 \text{ U/L}$; ALT: $52.1 \pm 18.4 \text{ U/L}$ vs. $26.5 \pm 9.6 \text{ U/L}$; $p < 0.001$). Overall, all biochemical parameters were significantly higher in preeclamptic women, suggesting significant biochemical derangement in preeclampsia

Table 2- Comparison of biochemical parameters among study groups

Parameter	Preeclampsia (n=60)	Normotensive (n=60)	p-value
Serum Uric Acid (mg/dL)	6.8 ± 1.2	4.2 ± 0.8	<0.001
LDH (U/L)	620.5 ± 140.3	320.4 ± 85.6	<0.001
CRP (mg/L)	9.6 ± 3.1	3.8 ± 1.5	<0.001
Serum Urea (mg/dL)	38.6 ± 10.4	24.8 ± 6.2	<0.001
Serum Creatinine (mg/dL)	1.02 ± 0.28	0.72 ± 0.15	<0.001
AST (U/L)	58.4 ± 20.6	28.3 ± 10.2	<0.001
ALT (U/L)	52.1 ± 18.4	26.5 ± 9.6	<0.001

Table 3 presents the severity-wise comparison of biochemical parameters among women with preeclampsia. Women with severe preeclampsia (n=28) had significantly higher serum uric acid levels ($7.8 \pm 1.1 \text{ mg/dL}$) compared to those with mild preeclampsia ($5.9 \pm 0.8 \text{ mg/dL}$; $p < 0.001$). Similarly, markers of tissue injury and inflammation, including lactate dehydrogenase (LDH) and C-reactive protein (CRP), were significantly elevated in the severe group (LDH: $740.6 \pm 150.4 \text{ U/L}$ vs. $520.3 \pm 110.2 \text{ U/L}$; CRP: $12.1 \pm 3.2 \text{ mg/L}$ vs. $7.2 \pm 2.4 \text{ mg/L}$; $p < 0.001$). Renal function parameters (urea and creatinine) and hepatic enzymes (AST and ALT) were also significantly higher in severe preeclampsia ($p < 0.01$). Overall, increasing severity of preeclampsia was associated with progressive elevation of all biochemical parameters.

Table 3- Severity-wise comparison of biochemical parameters in preeclampsia (n=60)

Parameter	Mild Preeclampsia (n=32)	Severe Preeclampsia (n=28)	p-value
Uric Acid (mg/dL)	5.9 ± 0.8	7.8 ± 1.1	<0.001
LDH (U/L)	520.3 ± 110.2	740.6 ± 150.4	<0.001
CRP (mg/L)	7.2 ± 2.4	12.1 ± 3.2	<0.001
Urea (mg/dL)	17.8 ± 4.8	27.1 ± 6.2	<0.01
Creatinine (mg/dL)	0.88 ± 0.20	1.18 ± 0.30	<0.01
AST (U/L)	48.2 ± 15.3	70.5 ± 22.4	<0.01
ALT (U/L)	39.8 ± 11.9	59.3 ± 17.6	<0.01

The distribution of participants based on the occurrence of adverse maternal–fetal outcomes is shown in Figure 1. Overall, 56 out of 120 participants (46.7%) experienced at least one adverse outcome, while 64 participants (53.3%) did not ($p < 0.001$). Among women with preeclampsia, a higher proportion had adverse outcomes, with 40 out of 60 (66.7%) affected compared to 20 (33.3%) without adverse outcomes. In contrast, only 16 of 60 normotensive women (26.7%) experienced adverse maternal–fetal outcomes, while the majority (44, 73.3%) remained unaffected. These findings indicate a significantly increased risk of adverse maternal–fetal events in preeclamptic pregnancies compared to normotensive controls.

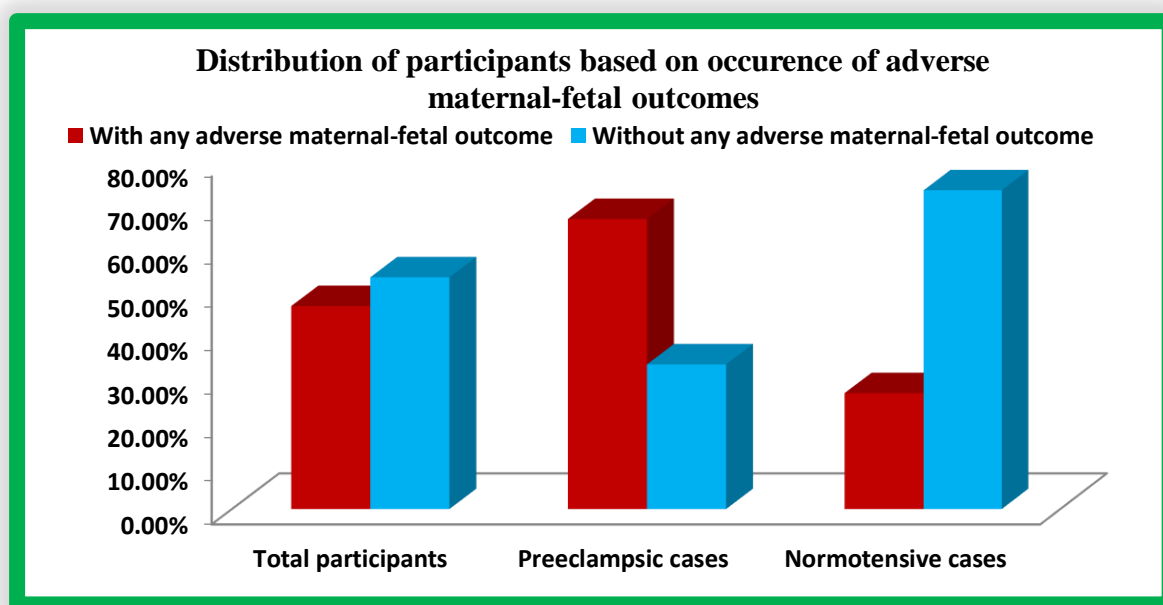


Figure 1- Distribution of participants based on occurrence of adverse maternal-fetal outcomes

Table 4 shows the distribution of adverse maternal and fetal outcomes among the study participants. Among maternal outcomes, severe preeclampsia was observed in 28 women (46.7%) in the preeclampsia group and was absent in normotensive controls ($p < 0.001$). Eclampsia occurred in 6 (10%) and HELLP syndrome in 4 (6.7%) preeclamptic women, with no cases in the control group ($p < 0.05$). Fetal outcomes were also significantly more frequent in the preeclampsia group. Preterm delivery was noted in 26 (43.3%) versus 10 (16.7%) cases ($p < 0.01$), low birth weight in 32 (53.3%) versus 14 (23.3%) ($p < 0.01$), and NICU admission in 24 (40%) versus 8 (13.3%) ($p < 0.01$). An APGAR score <7 at 5 minutes was observed in 12 (20%) preeclamptic neonates compared to 4 (6.7%) in controls ($p < 0.05$). These findings indicate a significantly higher incidence of both maternal and fetal complications in preeclamptic pregnancies.

Table 4- Distribution of adverse maternal–fetal outcomes among study participants

Outcome		Preeclampsia (n=60)	Normotensive (n=60)	Total (n=120)	p-value
Maternal outcomes	Severe preeclampsia	28 (46.7%)	0	28 (23.3%)	<0.001
	Eclampsia	6 (10%)	0	6 (5%)	<0.05
	HELLP syndrome	4 (6.7%)	0	4 (3.3%)	<0.05
Fetal outcomes	Preterm delivery	26 (43.3%)	10 (16.7%)	36 (30%)	<0.01
	Low birth weight	32 (53.3%)	14 (23.3%)	46 (38.3%)	<0.01
	NICU admission	24 (40%)	8 (13.3%)	32 (26.7%)	<0.01
	APGAR score <7 at 5 min	12 (20%)	4 (6.7%)	16 (13.3%)	<0.05

The association of biochemical parameters with maternal–fetal outcomes is presented in Table 5. Participants with adverse outcomes ($n=56$) had significantly higher serum uric acid levels (7.8 ± 1.1 mg/dL) compared to those without adverse outcomes (5.9 ± 0.8 mg/dL; $p < 0.001$). Similarly, markers of tissue injury and inflammation, including lactate dehydrogenase (LDH) and C-reactive protein (CRP), were significantly elevated in the adverse outcome group (LDH: 710.4 ± 140.2 U/L vs. 520.3 ± 110.5 U/L; CRP: 11.4 ± 3.1 mg/L vs. 7.2 ± 2.0 mg/L; $p < 0.001$). Renal function parameters, including serum urea and creatinine, were also significantly higher ($p < 0.01$). Hepatic enzymes (AST and ALT) were elevated in participants with adverse outcomes compared to those without ($p < 0.01$). Overall, higher levels of all biochemical parameters were significantly associated with the occurrence of adverse maternal–fetal outcomes.

Table 5- Association of biochemical parameters with maternal–fetal outcomes (n=120)

Parameter	No Adverse Outcome (n=64)	Adverse Outcome (n=56)	p-value
Uric Acid (mg/dL)	5.9 ± 0.8	7.8 ± 1.1	<0.001
LDH (U/L)	520.3 ± 110.5	710.4 ± 140.2	<0.001
CRP (mg/L)	7.2 ± 2.0	11.4 ± 3.1	<0.001
Urea (mg/dL)	18.5 ± 5.2	27.3 ± 6.4	<0.01
Creatinine (mg/dL)	0.85 ± 0.18	1.17 ± 0.29	<0.01
AST (U/L)	48.9 ± 15.7	69.1 ± 20.8	<0.01
ALT (U/L)	40.5 ± 12.3	58.6 ± 18.2	<0.01

Table 6 presents the multivariate logistic regression analysis identifying independent predictors of adverse maternal–fetal outcomes. Elevated serum uric acid emerged as the strongest predictor (adjusted OR: 2.8; 95% CI: 1.6–4.9; $p < 0.001$). Lactate dehydrogenase (LDH) was also significantly associated with adverse outcomes (OR: 2.3; 95% CI: 1.4–3.8; $p < 0.01$). Serum creatinine (OR: 1.9; 95% CI: 1.2–3.2; $p < 0.05$) and C-reactive protein (CRP) (OR: 1.5; 95% CI: 1.1–2.6; $p < 0.05$) were identified as additional independent predictors. In contrast, serum urea, AST, and ALT did not show statistically significant independent associations ($p > 0.05$). These findings indicate that serum uric acid, LDH, creatinine, and CRP are key biochemical predictors of adverse maternal–fetal outcomes in preeclampsia.

Table 6- Multivariate association – key predictors of adverse maternal–fetal outcomes (n=120)

Parameter	Adjusted OR	95% CI	p-value
Serum Uric acid (mg/dL)	2.8	1.6–4.9	<0.001
LDH (U/L)	2.3	1.4–3.8	<0.01
Urea (mg/dL)	1.2	0.8–1.7	0.15
Serum Creatinine (mg/dL)	1.9	1.2–3.2	<0.05
CRP (mg/L)	1.5	1.1–2.6	<0.05
AST (U/L)	1.4	0.9–2.1	0.08
ALT (U/L)	1.3	0.9–1.9	0.12

DISCUSSION-

The present study demonstrates that preeclampsia is associated with significant alterations in biochemical parameters, including serum uric acid, LDH, CRP, renal function tests, and liver enzymes, all of which correlate strongly with disease severity and adverse maternal–fetal outcomes. These findings reinforce the role of biochemical markers in understanding disease progression and predicting complications.

Serum uric acid levels were significantly elevated in preeclamptic women and showed a progressive increase with severity. This observation is consistent with earlier reports highlighting its association with endothelial dysfunction and oxidative stress [2,4]. In addition, studies by Roberts et al. [10] and Powers et al. [11] have shown that hyperuricemia reflects impaired renal clearance and placental dysfunction. Bainbridge and Roberts [12] further suggested that uric acid may act as a pathogenic mediator rather than just a marker. In the present study, uric acid also emerged as the strongest independent predictor of adverse outcomes, emphasizing its clinical relevance.

LDH, a marker of cellular injury and hemolysis, was significantly elevated in preeclamptic women, particularly in severe cases. This finding is in agreement with previous studies demonstrating its association with tissue damage and disease severity [2,7]. Similar observations were reported by Qublan et al. [13] and Jaiswar et al. [14], who found that elevated LDH levels are strongly associated with complications such as HELLP syndrome and eclampsia.

CRP, an acute-phase reactant, was also significantly elevated in preeclampsia and in patients with adverse outcomes. This supports the role of systemic inflammation in disease pathogenesis, as highlighted in earlier studies by Arias-Sánchez C et al. [3], Tjoa et al. [15] and Kushner et al. [16].

Renal involvement, a key feature of preeclampsia, was evident in the form of significantly elevated serum urea and creatinine levels. These findings are consistent with studies describing impaired renal function due to endothelial injury and altered glomerular filtration [17,18]. However, in multivariate analysis, only serum creatinine remained an independent predictor, suggesting a stronger association with adverse outcomes.

Liver enzyme abnormalities (AST and ALT) observed in the present study indicate hepatic involvement, particularly in severe preeclampsia. Previous studies have reported similar findings, attributing elevated liver enzymes to hepatic ischemia and microvascular injury [19,20]. However, these parameters did not show independent predictive value after adjustment, indicating their limited role as standalone predictors.

The present study also demonstrated a significantly higher incidence of adverse maternal and fetal outcomes in preeclamptic pregnancies, including preterm delivery, low birth weight, NICU admission, and low APGAR scores. These findings are consistent with earlier reports highlighting increased perinatal morbidity and mortality in such cases [1,2]. Studies by Mol et al. [21] and von Dadelszen et al. [22] further support these observations.

Importantly, multivariate logistic regression analysis identified serum uric acid, LDH, creatinine, and CRP as independent predictors of adverse maternal–fetal outcomes. These findings are in line with previous studies [23,24], which have emphasized the role of biochemical markers in risk stratification and prognostication. The lack of independent significance of urea and liver enzymes suggests that, although they reflect disease severity, they may not independently predict adverse outcomes.

The strength of this study lies in its comparative design and comprehensive evaluation of multiple biochemical parameters in relation to both disease severity and outcomes. However, limitations include its single-center nature and relatively small sample size. Further multicentric studies with larger cohorts are recommended to validate these findings and explore additional biomarkers.

CONCLUSION-

Preeclampsia is associated with significant biochemical derangements, including elevated serum uric acid, LDH, CRP, creatinine, and hepatic enzymes, which correlate with disease severity and adverse maternal–fetal outcomes. Among these, serum uric acid, LDH, creatinine, and CRP emerged as independent predictors of complications. These findings highlight the utility of routine biochemical assessment in early risk stratification and clinical monitoring of preeclamptic pregnancies. Timely identification of high-risk patients using these markers may facilitate improved clinical decision-making, optimize maternal management, and enhance neonatal outcomes.

Source of funding- Nil

Conflict of interest- None

REFERENCES-

1. Moharana JJ, Mishra R, Nayak AK. A study on serum LDH and uric acid in preeclampsia. *Int J Appl Basic Med Res.* 2023;13(2):95–100.
2. Deeksha HS, et al. Serum LDH and uric acid in preeclampsia: implications for maternal health. *Cureus.* 2024;16(3):e56928.
3. Arias-Sánchez C, et al. Uric acid and inflammasome activation in preeclampsia. *Antioxidants.* 2025;14(8):928.
4. Chen X, et al. Serum uric acid as a diagnostic biomarker for preeclampsia. *Front Endocrinol.* 2026.
5. Colmenares-Mejia CC, et al. Uric acid and risk of preeclampsia. *Sci Rep.* 2023;13:3018.
6. Abu-Zaid A, et al. Maternal hyperuricemia and adverse maternal–fetal outcomes: meta-analysis. *Front Med.* 2026.
7. Nasir SK, et al. LDH levels in preeclampsia and outcomes. *BMC Women's Health.* 2025;25:108.
8. Kunder M, et al. Correlation of LDH and uric acid with severity of preeclampsia. *Int J Clin Biochem Res.* 2019;6:315–320.
9. Dong L, et al. Uric acid, lipid levels and pregnancy outcomes in preeclampsia. *Am J Transl Res.* 2025;17(4):2800–2808.
10. Roberts JM, Bodnar LM, Lain KY, et al. Uric acid is as important as proteinuria in identifying fetal risk in women with gestational hypertension. *Hypertension.* 2005;46(6):1263–9.
11. Powers RW, Bodnar LM, Ness RB, et al. Uric acid concentrations in early pregnancy among preeclamptic women. *Am J Obstet Gynecol.* 2006;194(1):160.e1–8.
12. Bainbridge SA, Roberts JM. Uric acid as a pathogenic factor in preeclampsia. *Placenta.* 2008;29(Suppl A):S67–72.
13. Qublan HS, Amarun V, Bateinen O, et al. Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe preeclampsia. *Med Sci Monit.* 2005;11(8):CR393–7.
14. Jaiswar SP, Gupta A, Sachan R, et al. Lactic dehydrogenase: A biochemical marker for preeclampsia–eclampsia. *J Obstet Gynaecol India.* 2011;61(6):645–8.
15. Tjoa ML, van Vugt JM, Go AT, et al. Elevated C-reactive protein levels in preeclampsia. *J Reprod Immunol.* 2003;59(2):231–9.
16. Kushner I, Rzewnicki D, Samols D. What does minor elevation of C-reactive protein signify? *Am J Med.* 2006;119(2):166.e17–28.
17. Hladunewich MA, Karumanchi SA, Lafayette RA. Pathophysiology of preeclampsia: renal perspective. *Kidney Int.* 2007;71(10):1007–14.
18. Karumanchi SA, Maynard SE. The pathogenesis of preeclampsia: antiangiogenic factors. *J Clin Invest.* 2008;118(6):2152–7.

19. Sibai BM. Diagnosis and management of gestational hypertension and preeclampsia. *Obstet Gynecol.* 2003;102(1):181–92.
20. Rana S, Lemoine E, Granger JP, Karumanchi SA. Preeclampsia: pathophysiology and clinical implications. *Circulation.* 2019;140(14):1177–93.
21. Mol BWJ, Roberts CT, Thangaratinam S, Magee LA, de Groot CJM, Hofmeyr GJ. Pre-eclampsia. *Lancet.* 2016;387(10022):999–1011.
22. von Dadelszen P, Magee LA, Roberts JM. Subclassification of preeclampsia. *Hypertens Pregnancy.* 2003;22(2):143–8.
23. Thangaratinam S, Ismail KMK, Sharp S, Coomarasamy A, Khan KS. Accuracy of serum uric acid in predicting complications of pre-eclampsia. *BMJ.* 2006;333(7561):1–6.
24. Bellomo G, Venanzi S, Saronio P, Verdura C, Narducci PL. Prognostic significance of serum uric acid in women with gestational hypertension. *Hypertension.* 2011;58(4):704–8.