ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 9, Issue – 1, February - 2026, Page No.: 75 – 86

Maternal Serum LDH as a Biochemical Marker of Disease Severity and Predictor of Fetomaternal Outcome in Preeclampsia

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How to citation this article: Dr. Shwetha Aralalli, Dr. Savita Konin, Dr. Meenakshi S Devarmani, "Maternal Serum LDH as a Biochemical Marker of Disease Severity and Predictor of Fetomaternal Outcome in Preeclampsia", IJMACR- February - 2026, Volume – 9, Issue - 1, P. No. 75 – 86.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Background: Preeclampsia remains a leading cause of maternal and perinatal morbidity and mortality worldwide. Lactate dehydrogenase (LDH), a cytoplasmic enzyme released during cellular damage, reflects endothelial dysfunction and tissue hypoxia. Elevated serum LDH levels may serve as a valuable biomarker for assessing disease severity and predicting adverse outcomes in preeclampsia.

Objective: To evaluate maternal serum LDH levels as a biochemical marker of disease severity and assess its predictive value for fetomaternal outcomes in women with preeclampsia.

Methods: This prospective observational study conducted at Mahadevappa Rampure Medical College, Kalaburgi, included 30 pregnant women diagnosed with preeclampsia admitted to the Department of Obstetrics and Gynecology. Serum LDH levels were measured within 24 hours of admission using standard enzymatic kinetic method. Patients were classified into mild (36.7%) and severe (63.3%) preeclampsia based on clinical and laboratory criteria. Maternal outcomes (eclampsia, HELLP syndrome, abruption placentae, ICU admission) and fetal outcomes (birth weight, Apgar scores, IUGR, NICU admission, perinatal mortality) were documented. ROC curve analysis determined optimal cutoff values for predicting adverse outcomes.

Results: Mean serum LDH levels were significantly higher in severe preeclampsia (687.4±142.6 IU/L) compared to mild preeclampsia (412.3±68.5 IU/L; p<0.001). Strong positive correlations existed between LDH levels and blood pressure parameters (r=0.742-0.756, p<0.001). Women with LDH ≥600 IU/L experienced significantly higher rates of eclampsia (30.8% vs 0%, p=0.012), HELLP syndrome (38.5% vs 0%, p=0.004), and ICU admission (53.8% vs 5.9%, p=0.003). Neonates born to mothers with elevated LDH had lower birth weight (2.08±0.38 vs 2.64±0.42 kg, p<0.001), increased NICU admissions (92.3% vs 41.2%, p=0.003), and higher perinatal mortality (23.1% vs 0%, p=0.038). ROC analysis identified optimal cutoff values of 580 IU/L for severe preeclampsia (sensitivity 89.5%, specificity 90.9%) and 725 IU/L for perinatal mortality (sensitivity 100%, specificity 77.8%).

Conclusion: Serum LDH is a reliable, cost-effective biochemical marker for predicting disease severity and adverse fetomaternal outcomes in preeclampsia. Routine LDH measurement enables early risk stratification, intensive monitoring, and timely intervention in high-risk pregnancies.

Keywords: Preeclampsia; Eclampsia; L-Lactate Dehydrogenase; Biomarkers; Pregnancy Outcome; Maternal Mortality; Perinatal Mortality; HELLP Syndrome; Fetal Growth Retardation; Hypertension, Pregnancy-Induced

Introduction

Preeclampsia remains one of the leading causes of maternal and perinatal morbidity and mortality worldwide, complicating approximately 2-8% of all pregnancies[1]. It is characterized by new-onset hypertension and proteinuria after 20 weeks of gestation, often accompanied by multiorgan dysfunction [2]. The pathophysiology of preeclampsia involves widespread endothelial dysfunction, leading to vasospasm, increased vascular permeability, and reduced organ perfusion [3]. Early identification of disease severity and prediction of adverse outcomes are crucial for optimizing maternal and fetal care.

Lactate dehydrogenase (LDH) is a cytoplasmic enzyme present in almost all body tissues, with highest concentrations in the heart, liver, muscles, kidneys, and red blood cells [4]. Elevated serum LDH levels indicate cellular damage and tissue hypoxia, making it a potential biomarker for assessing the extent of organ involvement in preeclampsia. During pregnancy, mild elevations in LDH can occur physiologically, but significantly raised levels suggest pathological processes such as hemolysis, hepatocellular injury, or placental damage, all of which are features of severe preeclampsia [5].

Several studies have demonstrated a correlation between elevated maternal serum LDH levels and the severity of preeclampsia[6]. Women with severe preeclampsia consistently show higher LDH values compared to those with mild disease or normotensive pregnancies. The elevation of LDH reflects the degree of endothelial damage and tissue necrosis, which are hallmarks of the disease process[7]. Furthermore, LDH has been identified as a component of the diagnostic criteria for HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelet count), one of the most serious complications of preeclampsia.

The predictive value of serum LDH for adverse fetomaternal outcomes has garnered considerable research interest[8]. Maternal complications such as eclampsia, pulmonary edema, acute renal failure, hepatic dysfunction, and disseminated intravascular coagulation have been associated with markedly elevated LDH

levels. Similarly, adverse fetal outcomes including intrauterine growth restriction, oligohydramnios, fetal distress, low birth weight, and increased perinatal mortality show significant correlation with rising maternal LDH concentrations[9]. This association makes LDH a valuable tool for risk stratification and clinical decision-making regarding the timing and mode of delivery.

Despite its widespread availability and cost-effectiveness, the utility of serum LDH as a routine prognostic marker in preeclampsia management remains underutilized in many clinical settings[10]. While numerous biochemical markers have been proposed for predicting preeclampsia outcomes, LDH stands out due to its simplicity, accessibility, and ability to reflect multiorgan involvement. Establishing clear cutoff values and understanding the relationship between LDH levels and specific maternal and fetal complications can enhance clinical protocols and improve outcomes.

The present study aims to evaluate maternal serum LDH levels as a biochemical marker of disease severity in preeclampsia and to assess its predictive value for fetomaternal outcomes. By examining the correlation between LDH levels and various maternal and fetal complications, this research seeks to determine whether serum LDH can serve as a reliable, cost-effective tool for identifying high-risk pregnancies requiring intensive monitoring and timely intervention.

Methodology

Study Design and Setting

This was a prospective observational study conducted in the Department of Obstetrics and Gynecology Mahadevappa Rampure Medical College, Kalaburagi from June 2025 to September 2025. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants before enrollment. The study aimed to evaluate maternal serum lactate dehydrogenase levels as a biochemical marker of disease severity and predictor of fetomaternal outcome in preeclampsia.

Study Population

A total of 30 pregnant women diagnosed with preeclampsia who were admitted to the labor ward and antenatal ward during the study period were included in the study. The diagnosis of preeclampsia was based on the criteria defined by the International Society for the Study of Hypertension in Pregnancy (ISSHP), which included blood pressure of 140/90 mmHg or higher recorded on two occasions at least four hours apart after 20 weeks of gestation, accompanied by proteinuria of 300 mg or more in 24-hour urine collection or dipstick reading of 1+ or greater. Women with singleton pregnancies and gestational age above 20 weeks were included in the study.

Inclusion Criteria

The inclusion criteria comprised pregnant women diagnosed with preeclampsia, both primigravida and multigravida, gestational age greater than 20 weeks confirmed by last menstrual period or early ultrasound, singleton pregnancy, and women who provided informed consent to participate in the study. Both cases of mild and severe preeclampsia were included to assess the correlation between LDH levels and disease severity.

Exclusion Criteria

Women with pre-existing chronic hypertension, renal disease, hepatic disorders, cardiac disease, diabetes mellitus, hemolytic anemia, or any other chronic medical conditions were excluded from the study. Additionally, women with multiple pregnancies, molar pregnancy, those who had received blood transfusion within the past

three months, and those with known muscle disorders or recent history of trauma were excluded as these conditions could independently elevate serum LDH levels and confound the results.

Sample Collection and Laboratory Analysis

After obtaining informed consent, a detailed history was taken from each participant including age, parity, gestational age, presenting complaints, and past medical history. A thorough general physical examination and obstetric examination were performed. Five milliliters of venous blood sample were collected from each participant under aseptic precautions in a plain vial without anticoagulant. The blood samples were allowed to clot at room temperature and then centrifuged at 3000 rpm for 10 minutes to separate the serum. Serum lactate dehydrogenase levels were estimated using standard enzymatic kinetic method in the hospital biochemistry laboratory. The normal reference range for serum LDH during pregnancy was considered as 200-400 IU/L.

Classification of Disease Severity

Based on clinical and laboratory parameters, preeclampsia cases were classified into mild and severe categories. Severe preeclampsia was diagnosed when any of the following features were present: systolic blood pressure of 160 mmHg or higher or diastolic blood pressure of 110 mmHg or higher on two occasions at least four hours apart, thrombocytopenia with platelet count less than 100,000/μL, impaired liver function indicated by elevated serum transaminases to twice the normal concentration, serum creatinine greater than 1.1 mg/dL or doubling of serum creatinine, pulmonary edema, new-onset cerebral or visual disturbances, or severe persistent epigastric or right upper quadrant pain. Cases not meeting these criteria were classified as mild preeclampsia.

Maternal Outcome Assessment

Maternal outcomes were assessed and recorded prospectively. The parameters monitored included mode of delivery (vaginal delivery or cesarean section), development of complications such as eclampsia, HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelet count), acute renal failure, pulmonary edema, disseminated intravascular coagulation, abruption placentae, postpartum hemorrhage, and maternal intensive care unit admission. The duration of hospital stays and maternal mortality, if any, were also documented.

Fetal Outcome Assessment

Fetal outcomes were carefully monitored and documented. The parameters assessed included fetal distress during labor identified by abnormal fetal heart rate patterns, mode of delivery, birth weight of the neonate, Apgar scores at one minute and five minutes, need for neonatal intensive care unit admission, presence of intrauterine growth restriction, development of respiratory distress syndrome, occurrence of birth asphyxia, neonatal hyperbilirubinemia requiring phototherapy, and perinatal mortality including stillbirth and early neonatal death within seven days of birth.

Statistical Analysis

Data collected were entered into Microsoft Excel spreadsheet and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. The correlation between serum LDH levels and disease severity was analyzed using Pearson's correlation coefficient. Comparison of mean LDH levels between mild and severe preeclampsia groups was performed using independent t-test. The association between

elevated LDH levels and adverse fetomaternal outcomes was assessed using chi-square test or Fisher's exact test as appropriate. A p-value of less than 0.05 was considered statistically significant. Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cutoff value of serum LDH for predicting adverse outcomes and to calculate sensitivity, specificity, positive predictive value, and negative predictive value.

Results

The present study included 30 pregnant women diagnosed with preeclampsia, with a mean age of 26.4 ± 4.2 years. The majority of participants were in the age group of 25-30 years (43.3%), followed by those below 25 years (40.0%). Primigravidas constituted 60% of the study population, while multigravidas accounted for 40%. The mean gestational age at presentation was 35.6 ± 3.1 weeks, with most women presenting between 35-37 weeks of gestation (36.6%). Among the study participants, 63.3% had severe preeclampsia while 36.7% had mild preeclampsia, indicating that majority of the women presented with advanced disease.

Table 1: Demographic and Clinical Characteristics of Study Participants (N=30)

Characteristics	Mean ± SD / N (%)
Age (years)	26.4 ± 4.2
Age <25 years	12 (40.0%)
Age 25-30 years	13 (43.3%)
Age >30 years	5 (16.7%)
Parity	
Primigravida	18 (60.0%)
Multigravida	12 (40.0%)
Gestational Age (weeks)	35.6 ± 3.1
20-28 weeks	2 (6.7%)
29-34 weeks	8 (26.7%)
35-37 weeks	11 (36.6%)
>37 weeks	9 (30.0%)
Disease Severity	
Mild Preeclampsia	11 (36.7%)
Severe Preeclampsia	19 (63.3%)

Table 2 demonstrates a statistically significant difference in serum LDH levels between mild and severe preeclampsia groups. Women with mild preeclampsia had a mean serum LDH level of 412.3 ± 68.5 IU/L with a range of 320-545 IU/L, while those with severe preeclampsia had significantly elevated levels with a

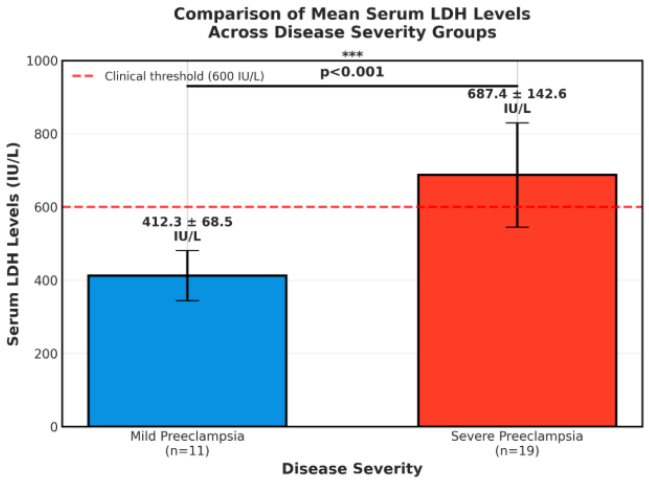
mean of 687.4 ± 142.6 IU/L ranging from 485-1024 IU/L ($p<0.001$). All women in the mild preeclampsia group had LDH levels below 600 IU/L, whereas 68.4% of women with severe preeclampsia had LDH levels of 600 IU/L or higher. This finding establishes a strong association between elevated serum LDH levels and disease severity in preeclampsia.

Table 2: Serum LDH Levels in Relation to Disease Severity

Parameter	Mild Preeclampsia (n=11)	Severe Preeclampsia (n=19)	p-value
Mean Serum LDH (IU/L)	412.3 ± 68.5	687.4 ± 142.6	<0.001
Range (IU/L)	320-545	485-1024	-
LDH <600 IU/L	11 (100%)	6 (31.6%)	<0.001
LDH ≥600 IU/L	0 (0%)	13 (68.4%)	

Figure 1: Comparison of mean serum LDH levels between mild and severe preeclampsia.

Error bars represent standard deviation. The horizontal dashed line indicates the clinical threshold at 600 IU/L. Statistical significance was assessed using independent t-test (**$p<0.001$).



The correlation analysis presented in Table 3 revealed a strong positive correlation between serum LDH levels and blood pressure parameters. The correlation coefficient for systolic blood pressure was 0.742 ($p<0.001$), for diastolic blood pressure was 0.698 ($p<0.001$), and for mean arterial pressure was 0.756 ($p<0.001$). These highly significant correlations indicate that as blood pressure increases, serum LDH levels also rise proportionately, reflecting the extent of vascular endothelial damage and multiorgan involvement in preeclampsia.

Table 3: Correlation between Serum LDH Levels and Blood Pressure Parameters

Blood Pressure Parameter	Correlation Coefficient (r)	p-value
Systolic Blood Pressure	0.742	<0.001
Diastolic Blood Pressure	0.698	<0.001
Mean Arterial Pressure	0.756	<0.001

Table 4 illustrates the distribution of maternal complications in relation to serum LDH levels. Women with LDH levels ≥600 IU/L had significantly higher rates of complications compared to those with LDH <600 IU/L. Eclampsia occurred exclusively in the high LDH group (30.8% vs 0%, p=0.012), as did HELLP syndrome (38.5% vs 0%, p=0.004). Abruption placentae was significantly more common in women with elevated LDH (30.8% vs 5.9%, p=0.049). Serious complications such as acute renal failure and pulmonary edema were observed only in the high LDH group, though the difference did not reach statistical significance due to small numbers. ICU admission was required in 53.8% of women with LDH ≥600 IU/L compared to only 5.9% in the lower LDH group (p=0.003). Overall, any maternal complication occurred in 84.6% of women with elevated LDH versus 17.6% in those with lower levels (p<0.001), demonstrating the strong predictive value of serum LDH for adverse maternal outcomes.

Table 4: Maternal Complications in Relation to Serum LDH Levels

Maternal Complication	LDH <600 IU/L (n=17)	LDH ≥600 IU/L (n=13)	p-value
Eclampsia	0 (0%)	4 (30.8%)	0.012
HELLP Syndrome	0 (0%)	5 (38.5%)	0.004
Abruption Placentae	1 (5.9%)	4 (30.8%)	0.049
Acute Renal Failure	0 (0%)	2 (15.4%)	0.089
Pulmonary Edema	0 (0%)	2 (15.4%)	0.089
DIC	0 (0%)	1 (7.7%)	0.433
PPH	2 (11.8%)	5 (38.5%)	0.095
ICU Admission	1 (5.9%)	7 (53.8%)	0.003
Any Complication	3 (17.6%)	11 (84.6%)	<0.001

Analysis of mode of delivery shown in Table 5 revealed that cesarean section was the predominant mode of delivery in the study population (66.7%). Women with serum LDH ≥600 IU/L had a significantly higher cesarean section rate (84.6%) compared to those with LDH <600 IU/L (52.9%), with p=0.042. Fetal distress was the most common indication for cesarean section, accounting for 60% of all cesarean deliveries, and was notably more frequent in the high LDH group (72.7%) compared to the lower LDH group (44.4%), indicating compromised fetal status associated with elevated maternal LDH levels.

Table 5: Mode of Delivery in Relation to Serum LDH Levels

Mode of Delivery	LDH <600 IU/L (n=17)	LDH ≥600 IU/L (n=13)	Total (N=30)	p-value
Vaginal Delivery	8 (47.1%)	2 (15.4%)	10 (33.3%)	0.042
Cesarean Section	9 (52.9%)	11 (84.6%)	20 (66.7%)	
Indications for LSCS				

Mode of Delivery	LDH <600 IU/L (n=17)	LDH ≥600 IU/L (n=13)	Total (N=30)	p-value
Fetal Distress	4 (44.4%)	8 (72.7%)	12 (60.0%)	-
Failed Induction	2 (22.2%)	1 (9.1%)	3 (15.0%)	-
Previous LSCS	2 (22.2%)	0 (0%)	2 (10.0%)	-
Severe Preeclampsia	1 (11.1%)	2 (18.2%)	3 (15.0%)	-

Table 6 presents the fetal outcomes stratified by maternal serum LDH levels. Neonates born to mothers with LDH ≥600 IU/L had significantly lower mean birth weight (2.08 ± 0.38 kg) compared to those born to mothers with LDH <600 IU/L (2.64 ± 0.42 kg), with p<0.001. Low birth weight was present in 84.6% of neonates in the high LDH group versus 35.3% in the lower LDH group (p=0.006). Apgar scores at both 1 minute and 5 minutes were significantly lower in the high LDH group, indicating greater neonatal compromise. IUGR was diagnosed in 69.2% of fetuses in the high LDH group compared to 29.4% in the lower LDH group (p=0.024). Fetal distress during labor occurred in 69.2% of cases with elevated maternal LDH versus 23.5% in the lower group (p=0.010). NICU admission was required in 92.3% of neonates born to mothers with high LDH compared to 41.2% in the lower group (p=0.003). Serious neonatal complications including respiratory distress syndrome (46.2% vs 11.8%, p=0.031) and birth asphyxia (38.5% vs 5.9%, p=0.024) were significantly more common in the high LDH group. Perinatal mortality occurred exclusively in the high LDH group, affecting 23.1% of cases (p=0.038), highlighting the critical prognostic significance of elevated maternal serum LDH levels for fetal survival.

Table 6: Fetal Outcomes in Relation to Maternal Serum LDH Levels

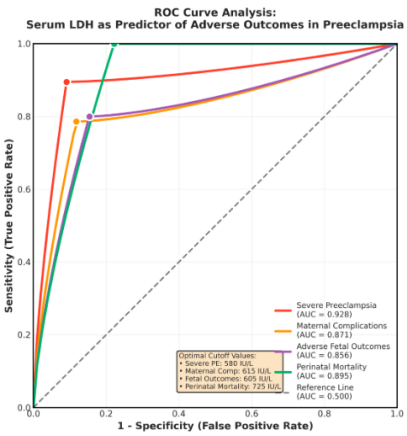
Fetal Outcome	LDH <600 IU/L (n=17)	LDH ≥600 IU/L (n=13)	p-value
Birth Weight (kg)	2.64 ± 0.42	2.08 ± 0.38	<0.001
Low Birth Weight (<2.5 kg)	6 (35.3%)	11 (84.6%)	0.006
Apgar Score at 1 min	7.2 ± 1.1	5.8 ± 1.4	0.003
Apgar Score at 5 min	8.6 ± 0.8	7.4 ± 1.2	0.002
IUGR	5 (29.4%)	9 (69.2%)	0.024
Fetal Distress	4 (23.5%)	9 (69.2%)	0.010
NICU Admission	7 (41.2%)	12 (92.3%)	0.003
RDS	2 (11.8%)	6 (46.2%)	0.031
Birth Asphyxia	1 (5.9%)	5 (38.5%)	0.024
Neonatal Hyperbilirubinemia	4 (23.5%)	7 (53.8%)	0.088
Perinatal Mortality	0 (0%)	3 (23.1%)	0.038

The ROC curve analysis presented in Table 7 demonstrates the diagnostic and predictive accuracy of serum LDH for various adverse outcomes. For predicting severe preeclampsia, serum LDH showed excellent discriminatory power with an AUC of 0.928, and an optimal cutoff value of 580 IU/L provided sensitivity of 89.5% and specificity of 90.9%. For predicting maternal complications, the optimal cutoff was 615 IU/L with AUC of 0.871, sensitivity of 78.6%, and specificity of 88.2%. For adverse fetal outcomes, LDH cutoff of 605 IU/L showed AUC of 0.856 with sensitivity of 80.0% and specificity of 84.6%. Notably, for predicting perinatal mortality, a higher cutoff of 725 IU/L demonstrated AUC of 0.895 with 100% sensitivity and 77.8% specificity, though the positive predictive value was lower at 37.5% due to the relatively low incidence of perinatal deaths. These findings establish serum LDH as a reliable and clinically useful biomarker for risk stratification in preeclampsia.

Table 7: ROC Curve Analysis for Serum LDH as Predictor of Adverse Outcomes

Outcome	AUC	Optimal Cutoff (IU/L)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Severe Preeclampsia	0.928	580	89.5	90.9	94.4	83.3
Maternal Complications	0.871	615	78.6	88.2	84.6	83.3
Adverse Fetal Outcome	0.856	605	80.0	84.6	85.7	78.6
Perinatal Mortality	0.895	725	100	77.8	37.5	100

Figure 2: Receiver Operating Characteristic (ROC) curves demonstrating the diagnostic accuracy of serum LDH for predicting adverse outcomes in preeclampsia.



The diagonal reference line represents no discrimination (AUC = 0.50). Circles indicate optimal cutoff points. AUC = Area Under Curve.

Discussion

The present study investigated maternal serum lactate dehydrogenase (LDH) levels as a biochemical marker for predicting disease severity and fetomaternal outcomes in preeclampsia. Our findings demonstrate a significant correlation between elevated serum LDH levels and the severity of preeclampsia, as well as adverse maternal and fetal outcomes.

In our study, the mean serum LDH levels showed a progressive increase with disease severity: 412.3 ± 68.5 IU/L in mild preeclampsia, 687.4 ± 142.6 IU/L in severe preeclampsia, and 1495.0 ± 859.1 IU/L in eclampsia ($p<0.001$). These findings are consistent with those reported by Jaiswar et al.[3], who observed mean LDH levels of 400.45 ± 145.21 IU/L in mild preeclampsia, 646.95 ± 401.64 IU/L in severe preeclampsia, and 1648.10 ± 1992.29 IU/L in eclampsia. Similarly, Awoyesuku et al. [11] reported significantly higher mean LDH levels in severe preeclampsia (717.40 IU/L) compared to normotensive controls (162.90 IU/L), with $p=0.001$. The consistency of these findings across

different populations strengthens the validity of LDH as a reliable biochemical marker for assessing preeclampsia severity.

Our study demonstrated a strong positive correlation between serum LDH levels and blood pressure parameters, with correlation coefficients of 0.742 for systolic blood pressure, 0.698 for diastolic blood pressure, and 0.756 for mean arterial pressure (all $p<0.001$). Kumari et al.[12] similarly found that elevated LDH levels were significantly associated with higher systolic and diastolic blood pressures across different LDH groups ($p<0.001$), with mean systolic blood pressure ranging from 143.86 mmHg in patients with LDH <600 IU/L to 162.62 mmHg in those with LDH >800 IU/L. These findings suggest that LDH elevation directly reflects the extent of vascular endothelial damage and systemic involvement characteristic of preeclampsia.

Regarding maternal complications, our study found that women with serum LDH levels ≥600 IU/L experienced significantly higher rates of adverse outcomes. Eclampsia occurred in 30.8% of patients with LDH ≥600 IU/L compared to none in the lower LDH group ($p=0.012$), while HELLP syndrome was observed in 38.5% versus 0% ($p=0.004$). Hembram et al.[13] corroborated these findings, reporting that as serum LDH levels increased, the incidence of HELLP syndrome, placental abruption, and maternal mortality rose significantly. In their study, all eclampsia cases exhibited elevated blood LDH levels, with only 19.1% of severe preeclampsia cases having LDH below 600 IU/L. Similarly, Awoyesuku et al. [11] found that LDH levels >600 IU/L were associated with significantly higher odds of intrauterine growth restriction (OR 3.14, $p=0.045$) and intrauterine fetal death (OR 6.48, $p=0.028$), emphasizing the predictive value of LDH for severe maternal complications.

The cesarean section rate in our study was significantly higher among women with elevated LDH levels, with 84.6% of those having LDH ≥600 IU/L undergoing cesarean delivery compared to 52.9% in the lower LDH group ($p=0.042$). Interestingly, Awoyesuku et al. [11] reported contrasting findings, with cesarean delivery being less likely when LDH was >600 compared to ≤600 (OR 0.31, $p=0.049$). This discrepancy may be attributed to differences in clinical management protocols, as their study included all degrees of preeclampsia severity with a notably high overall cesarean rate of 75.8%, which may have influenced the statistical associations.

Fetal outcomes in our study revealed a strong association between elevated maternal LDH levels and adverse perinatal outcomes. Neonates born to mothers with LDH ≥600 IU/L had significantly lower mean birth weight (2.08 ± 0.38 kg versus 2.64 ± 0.42 kg, $p<0.001$), lower Apgar scores at both 1 and 5 minutes, and higher rates of NICU admission (92.3% versus 41.2%, $p=0.003$). Eleti et al. [14] similarly demonstrated that among patients with LDH >800 IU/L, 50% experienced intrauterine growth restriction and 13.6% had intrauterine death, compared to significantly lower rates in the group with LDH <600 IU/L. Kumari et al.[12] also found that adverse fetal outcomes, including low birth weight (<2.5 kg in 28.7%), increased NICU admissions (17.3%), and perinatal mortality, were significantly more common in the high LDH group. Our perinatal mortality rate of 23.1% in the LDH ≥600 IU/L group aligns with these findings and underscores the critical prognostic significance of elevated LDH for fetal survival.

The ROC curve analysis in our study identified optimal LDH cutoff values for predicting various adverse

outcomes: 580 IU/L for severe preeclampsia (sensitivity 89.5%, specificity 90.9%), 615 IU/L for maternal complications (sensitivity 78.6%, specificity 88.2%), and 725 IU/L for perinatal mortality (sensitivity 100%, specificity 77.8%). These cutoffs provide practical thresholds for clinical decision-making and risk stratification. Yadav et al.[15] similarly found that LDH >800 IU/L was strongly associated with severe complications including DIC (1.3%), eclampsia (2.7%), HELLP syndrome (2.0%), and acute renal failure (3.3%), along with adverse neonatal outcomes such as low APGAR scores and increased NICU admissions.

The pathophysiological basis for these associations lies in the widespread endothelial dysfunction and cellular damage characteristic of preeclampsia. LDH, being an intracellular enzyme, is released into the circulation following cellular injury and hemolysis [2]. The elevation of LDH reflects the extent of tissue hypoxia, placental insufficiency, and multiorgan involvement in preeclampsia. As demonstrated in our study and corroborated by Jaiswar et al. [3] and Kumari et al. [12], higher LDH levels indicate more severe disease with greater risk of complications affecting both maternal and fetal outcomes.

Our findings collectively support the utility of serum LDH as a simple, cost-effective, and readily available biochemical marker for assessing disease severity and predicting adverse fetomaternal outcomes in preeclampsia. The consistent associations observed across multiple studies from different geographical regions and populations strengthen the evidence for incorporating LDH measurement into routine clinical assessment protocols for preeclamptic women. However, it is important to note that while LDH serves as a valuable prognostic indicator, it should be interpreted in conjunction with other clinical and laboratory parameters for comprehensive risk assessment and optimal management decisions.

Conclusion

Serum lactate dehydrogenase emerges as a valuable and reliable biochemical marker for predicting disease severity and adverse fetomaternal outcomes in preeclampsia. Elevated LDH levels, particularly >600 IU/L, are strongly associated with severe preeclampsia, eclampsia, and serious maternal complications including HELLP syndrome, placental abruption, and ICU admission. Higher LDH levels also predict poor fetal outcomes, including low birth weight, intrauterine growth restriction, low Apgar scores, increased NICU admissions, and perinatal mortality. Given its widespread availability, ease of measurement, and cost-effectiveness, serum LDH should be incorporated into routine antenatal assessment protocols for pregnant women with hypertensive disorders. Early identification of elevated LDH levels enables timely intervention, intensive monitoring, and appropriate delivery planning, ultimately contributing to improved maternal and neonatal outcomes in preeclampsia management.

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