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Original Research Article

CLINICAL SIGNIFICANCE OF SERUM LDH LEVELS IN PREECLAMPSIA AND ECLAMPSIA: A TERTIARY CARE STUDY

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Abstract

Background: Preeclampsia and eclampsia are major hypertensive disorders of pregnancy associated with significant maternal and perinatal morbidity and mortality. Endothelial dysfunction, tissue ischemia, and cellular injury in these conditions result in elevated serum lactate dehydrogenase (LDH) levels. The present study aimed to evaluate serum LDH as an early diagnostic biomarker in preeclampsia and eclampsia and to assess its association with disease severity and maternal–fetal outcomes.

Methods: This hospital-based observational study included 167 pregnant women divided into three groups: normotensive controls (n=56), preeclampsia (n=56), and eclampsia (n=55). Detailed clinical evaluation and laboratory investigations were performed in all participants. Serum LDH estimation was carried out using automated biochemical analysis. Maternal complications, fetal outcomes, and correlation of LDH with clinical and biochemical parameters were assessed. Statistical analysis was performed using SPSS software, and $p<0.05$ was considered statistically significant.

Results: Mean serum LDH levels were significantly higher in preeclampsia (691.8 ± 188.6 U/L) and eclampsia (1184.6 ± 346.7 U/L) compared to normotensive women (382.5 ± 96.4 U/L) ($p<0.001$). Elevated LDH levels were significantly associated with severe hypertension, thrombocytopenia, elevated liver enzymes, renal dysfunction, HELLP syndrome, and ICU admission ($p<0.001$). Adverse fetal outcomes including preterm delivery, low birth weight, NICU admission, fetal distress, and perinatal mortality were significantly more common among women with elevated LDH levels. Serum LDH showed significant positive correlation with systolic blood pressure ($r=0.71$), diastolic blood pressure ($r=0.68$), serum uric acid ($r=0.62$), and serum creatinine ($r=0.58$), while negative correlation was observed with platelet count, gestational age at delivery, and birth weight ($p<0.001$).

Conclusion: Serum LDH is a reliable and cost-effective biomarker for early diagnosis and severity assessment of preeclampsia and eclampsia. Elevated LDH levels are strongly associated with maternal complications and adverse perinatal outcomes, making LDH a useful tool for early risk stratification and timely clinical intervention in hypertensive disorders of pregnancy.

Keywords: Lactate dehydrogenase; Hypertensive disorders of pregnancy; Biomarker; Maternal outcome; Perinatal outcome.

Introduction

Hypertensive disorders of pregnancy remain one of the leading causes of maternal and perinatal morbidity and mortality worldwide, contributing substantially to adverse obstetric outcomes, particularly in developing countries [1]. Among these disorders, preeclampsia and eclampsia are major complications characterized by widespread endothelial dysfunction, vasospasm, and multiorgan involvement [2]. Preeclampsia is generally defined as new-onset hypertension occurring after 20 weeks of gestation associated with proteinuria and/or evidence of end-organ dysfunction, whereas eclampsia refers to the occurrence of generalized tonic–clonic seizures in a woman with preeclampsia that cannot be attributed to other neurological causes [3].

Globally, preeclampsia complicates approximately 2–8% of all pregnancies and is responsible for nearly 10–15% of maternal deaths worldwide [4].

In India, the incidence of preeclampsia ranges from 5–15% among pregnant women, with eclampsia

accounting for a significant proportion of maternal intensive care admissions and perinatal loss [5]. The exact etiopathogenesis of preeclampsia and eclampsia remains incompletely understood; however, abnormal placentation, impaired trophoblastic invasion, oxidative stress, exaggerated inflammatory response, endothelial dysfunction, and placental ischemia are considered central mechanisms [6]. These pathophysiological alterations result in generalized vascular injury, increased capillary permeability, intravascular coagulation, and cellular hypoxia leading to multisystem involvement including hepatic, renal, neurological, and hematological complications [7]. Early identification of disease severity is therefore crucial for timely intervention and prevention of maternal and fetal complications such as HELLP syndrome, placental abruption, disseminated intravascular coagulation, acute kidney injury, preterm birth, fetal growth restriction, and intrauterine fetal demise [8].

In recent years, considerable attention has been directed toward identifying reliable biochemical markers that can aid in the early diagnosis and assessment of severity of preeclampsia and eclampsia. Among various biomarkers, serum lactate dehydrogenase (LDH) has emerged as a potentially valuable indicator because it reflects cellular injury, tissue breakdown, and hypoxia [9]. LDH is an intracellular glycolytic enzyme present in almost all body tissues and is released into the circulation following cellular damage and membrane disruption [10]. Elevated serum LDH levels in preeclampsia are believed to occur due to endothelial damage, hemolysis, hepatic dysfunction, and hypoxic injury associated with placental insufficiency and microangiopathic changes [11].

Literatures have demonstrated a positive association between elevated serum LDH levels and the severity of hypertensive disorders in pregnancy. Higher LDH concentrations have been correlated with severe preeclampsia, eclampsia, maternal complications, poor fetal outcomes, and increased perinatal mortality [12]. Women with severe disease frequently exhibit markedly elevated LDH levels compared to normotensive pregnant women, suggesting that LDH may serve not only as a diagnostic biomarker but also as a prognostic indicator of disease progression and adverse outcomes [13].

Furthermore, serum LDH estimation is inexpensive, widely available, rapid, and technically simple, making it particularly useful in resource-limited healthcare settings where advanced diagnostic modalities may not be readily accessible. Therefore, further evaluation is necessary to establish the clinical significance of serum LDH as an early diagnostic biomarker in preeclampsia and eclampsia. The present study was aimed to evaluate serum lactate dehydrogenase levels in pregnancies complicated by preeclampsia and eclampsia and to assess its role as an early diagnostic marker for identifying disease severity and associated maternal–fetal complications.

Materials and Methods

Study Design and Setting: This hospital-based observational case–control study was conducted in the Department of Obstetrics and Gynaecology in collaboration with the Department of Biochemistry at a tertiary care teaching hospital over a period of 24 months. The study was undertaken to evaluate the role of serum lactate dehydrogenase (LDH) as an early diagnostic biomarker in pregnancies complicated by preeclampsia and eclampsia. Pregnant women admitted to the antenatal ward, labour room, and obstetric emergency services during the study period were screened for eligibility and enrolled after obtaining informed written consent. The study protocol was reviewed and approved by the Institutional Ethics Committee prior to commencement, and the study was conducted in accordance with ethical principles outlined in the Declaration of Helsinki.

Study Population and Sample Size: The study population comprised pregnant women with gestational age greater than or equal to 20 weeks who were categorized into three groups: normotensive pregnant women serving as controls, women diagnosed with preeclampsia, and women diagnosed with eclampsia. Diagnosis of preeclampsia was made according to standard obstetric guidelines and was defined as blood pressure ≥140/90 mmHg measured on two occasions at least 4 hours apart after 20 weeks of gestation in a previously normotensive woman, associated with proteinuria of ≥300 mg in 24-hour urine collection or ≥1+ proteinuria on dipstick examination. Severe preeclampsia was identified in the presence of blood pressure ≥160/110 mmHg, significant proteinuria, thrombocytopenia, elevated liver enzymes, renal impairment, pulmonary edema, or neurological symptoms. Eclampsia was diagnosed when generalized tonic–clonic seizures occurred in association with preeclampsia in the absence of any other identifiable neurological cause. Pregnant women with chronic hypertension, diabetes mellitus, renal disease, chronic liver disease, cardiovascular disorders, epilepsy, hemolyticanemia, multiple pregnancy, active infection, malignancy, or any condition known to influence serum LDH levels were excluded from the study. Women unwilling to participate were also excluded. A total of 167 participants were included in the study using consecutive sampling technique. These included 56 normotensive pregnant women as controls, 56 women diagnosed with preeclampsia, and 55 women with eclampsia.

Data Collection and Clinical Assessment: Detailed demographic and obstetric information including age, parity, socioeconomic status, gestational age, booking status, and previous obstetric history were recorded using a predesigned proforma. A thorough clinical examination was performed in all participants. Blood pressure was measured using a standard mercury sphygmomanometer with the patient in sitting position after adequate rest, and the average of two readings was recorded. Presence and degree of edema, neurological symptoms, visual disturbances, epigastric pain, and other clinical manifestations were noted. Routine obstetric examination and fetal assessment were carried out in all cases.

Relevant laboratory investigations including complete blood count, urine protein estimation, liver function tests, renal function tests, platelet count, and serum uric acid were performed wherever indicated. Maternal complications such as HELLP syndrome, placental abruption, disseminated intravascular coagulation, acute renal failure, pulmonary edema, and intensive care unit admission were documented. Fetal outcomes including birth weight, prematurity, fetal distress, NICU admission, stillbirth, and perinatal mortality were also recorded.

Estimation of Serum Lactate Dehydrogenase: Under aseptic precautions, approximately 5 mL of venous blood was collected from each participant before initiation of definitive treatment. The blood samples were allowed to clot and subsequently centrifuged at 3000 rpm for 10 minutes to separate serum. Serum LDH levels were estimated using an automated biochemical analyzer by kinetic UV assay method based on the International Federation of Clinical Chemistry (IFCC) protocol. Internal quality control measures were maintained throughout the study to ensure reliability and accuracy of biochemical analysis. Serum LDH values were expressed in units per liter (U/L).

For analytical purposes, serum LDH levels were compared among normotensive pregnant women, preeclamptic women, and eclamptic women. Correlation between serum LDH levels and severity of disease as well as maternal and fetal outcomes was also assessed.

Statistical Analysis: The collected data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 25.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were represented as frequencies and percentages. Comparison of continuous variables among study groups was performed using Student's t-test or one-way analysis of variance (ANOVA) as appropriate. Chi-square test or Fisher's exact test was used for comparison of categorical variables. Correlation between serum LDH levels and severity parameters was assessed using Pearson's correlation coefficient depending upon data distribution. A p-value of less than 0.05 was considered statistically significant.

Results

The mean age of participants was comparable among the three groups, with no statistically significant difference observed (p=0.214). Women aged less than 25 years constituted the majority in all groups. Primigravida women were significantly more common in the preeclampsia (62.5%) and eclampsia groups (70.9%) compared to normotensive women (42.9%) (p=0.006). Mean gestational age at admission was significantly lower among women with preeclampsia and eclampsia compared to normotensive controls (p<0.001). A higher proportion of women with hypertensive disorders belonged to rural areas and were unbooked pregnancies, with unbooked status showing significant association with disease severity (p<0.001). Both systolic and diastolic blood pressures were markedly elevated in preeclamptic and eclamptic women compared to controls (p<0.001). Pedal edema and significant proteinuria (≥2+) were also significantly more prevalent among women with preeclampsia and eclampsia (p<0.001) (Table 1).

Table 1: Baseline demographic, obstetric, and clinical characteristics among normotensive, preeclamptic, and eclamptic pregnant women

Variable	Normotensive (n=56)	Preeclampsia (n=56)	Eclampsia (n=55)	p-value
	Frequency (%)/Mean±SD			
Age (years)	24.8 ± 3.6	25.9 ± 4.1	24.7 ± 4.4	0.214
Age group				
<25 years	31 (55.4%)	28 (50.0%)	34 (61.8%)	0.412
>25 years	25 (44.6%)	28 (50.0%)	21 (38.2%)	
Gravida				
Mutligravida	32 (57.1%)	21 (37.5%)	16 (29.1%)	0.006
Primigravida	24 (42.9%)	35 (62.5%)	39 (70.9%)	
Gestational age at admission	37.8 ± 1.9	35.9 ± 2.8	34.7 ± 3.1	<0.001

(weeks)				
Residence				
Urban	26 (46.4%)	20 (35.7%)	15 (27.3%)	0.083
Rural	30 (53.6%)	36 (64.3%)	40 (72.7%)	
Booking status				
Booked	44 (78.6%)	32 (57.1%)	17 (30.9%)	
Unbooked	12 (21.4%)	24 (42.9%)	38 (69.1%)	<0.001
Systolic BP (mmHg)	114.6 ± 8.2	154.8 ± 13.5	170.2 ± 18.7	<0.001
Diastolic BP (mmHg)	73.5 ± 6.1	102.4 ± 9.8	112.7 ± 11.6	<0.001
Pedal edema				
Absent	47 (83.9%)	17 (30.4%)	11 (20.0%)	<0.001
Present	9 (16.1%)	39 (69.6%)	44 (80.0%)	
Proteinuria ≥2+	0 (0%)	37 (66.1%)	47 (85.5%)	<0.001

BP: Blood pressure.

Mean hemoglobin and platelet count progressively decreased from normotensive women to preeclamptic and eclamptic patients, with statistically significant differences across groups (p=0.002 and p<0.001, respectively). Renal function parameters including serum creatinine and serum uric acid were significantly elevated in women with hypertensive disorders, particularly among eclamptic patients (p<0.001). Similarly, hepatic enzymes AST and ALT showed a significant rise with increasing disease severity (p<0.001). Serum LDH levels demonstrated a marked and progressive elevation from normotensive women (382.5 ± 96.4 U/L) to preeclampsia (691.8 ± 188.6 U/L) and eclampsia groups (1184.6 ± 346.7 U/L), with highly significant statistical association (p<0.001). Most normotensive pregnant women (92.9%) had serum LDH levels below 600 U/L, whereas elevated LDH levels were predominantly observed among women with preeclampsia and eclampsia. In the preeclampsia group, the majority of women had LDH levels between 600–800 U/L (44.6%), while 17.9% and 7.1% had LDH levels of 801–1000 U/L and >1000 U/L, respectively. In contrast, women with eclampsia showed markedly elevated LDH levels, with 58.2% having serum LDH levels exceeding 1000 U/L. The distribution of LDH levels differed significantly among the study groups (p<0.001) (Table 2).

Table 2: Comparison of hematological, renal, hepatic, and serum LDH parameters among study groups

Laboratory Parameter	Normotensive (n=56)	Preeclampsia (n=56)	Eclampsia (n=55)	p-value
	Mean±SD			
Hemoglobin (g/dL)	10.9 ± 1.2	10.4 ± 1.5	9.8 ± 1.6	0.002
Platelet count (×10³/μL)	2.41 ± 0.52	1.86 ± 0.61	1.42 ± 0.58	<0.001
Serum creatinine (mg/dL)	0.72 ± 0.14	0.94 ± 0.26	1.28 ± 0.44	<0.001
Serum uric acid (mg/dL)	4.1 ± 0.8	6.3 ± 1.2	7.8 ± 1.7	<0.001
AST (IU/L)	24.6 ± 8.4	46.2 ± 19.8	71.3 ± 31.4	<0.001
ALT (IU/L)	22.8 ± 7.6	41.7 ± 16.9	65.4 ± 28.6	<0.001
Serum LDH (U/L)	382.5 ± 96.4	691.8 ± 188.6	1184.6 ± 346.7	<0.001
Serum LDH Level (U/L)				
<600	52 (92.9%)	17 (30.4%)	2 (3.6%)	<0.001
600–800	4 (7.1%)	25 (44.6%)	7 (12.7%)	
801–1000	0 (0%)	10 (17.9%)	14 (25.5%)	
>1000	0 (0%)	4 (7.1%)	32 (58.2%)	

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; LDH: Lactate dehydrogenase.

Higher serum LDH levels were significantly associated with worsening clinical severity and organ dysfunction. Severe hypertension was observed in only 11.3% of women with LDH <600 U/L compared to 94.4% among those with LDH >1000 U/L (p<0.001).

Similarly, thrombocytopenia, elevated liver enzymes, and serum creatinine >1.2 mg/dL were significantly more frequent in women with higher LDH levels (p<0.001 for all). HELLP syndrome and ICU admission also showed a progressive increase with rising LDH concentrations.

Women with LDH >1000 U/L had substantially higher frequencies of HELLP syndrome (27.8%) and ICU admission (38.9%) compared to those with lower LDH levels, indicating a strong association between elevated serum LDH and disease severity (Table 3).

Table 3: Association between serum LDH levels and severity-related clinical variables in hypertensive disorders of pregnancy

Clinical Variable	LDH <600 U/L (n=71)	LDH 600–1000 U/L (n=60)	LDH >1000 U/L (n=36)	p-value
	Frequency (%)			
Severe hypertension	8 (11.3%)	33 (55.0%)	34 (94.4%)	<0.001
Platelet count <1 lakh/μL	1 (1.4%)	9 (15.0%)	16 (44.4%)	<0.001
Elevated liver enzymes	3 (4.2%)	18 (30.0%)	24 (66.7%)	<0.001
Serum creatinine >1.2 mg/dL	0 (0%)	7 (11.7%)	15 (41.7%)	<0.001
HELLP syndrome	0 (0%)	4 (6.7%)	10 (27.8%)	<0.001
ICU admission	1 (1.4%)	6 (10.0%)	14 (38.9%)	<0.001

LDH: Lactate dehydrogenase; HELLP: Hemolysis, Elevated Liver enzymes, and Low Platelet count; ICU: Intensive Care Unit.

Maternal complications were more frequently observed among women with eclampsia compared to those with preeclampsia. Placental abruption, acute kidney injury, pulmonary edema, disseminated intravascular coagulation (DIC), and ICU admission were significantly higher in the eclampsia group ($p<0.05$). HELLP syndrome was also more common among eclamptic women (18.2%) than preeclamptic women (7.1%), although the difference did not reach statistical significance ($p=0.079$). Maternal mortality occurred only in the eclampsia group (3.6%), but this difference was statistically non-significant (Table 4).

Table 4: Maternal complications among women with preeclampsia and eclampsia

Maternal Complication	Preeclampsia (n=56)	Eclampsia (n=55)	p-value
	Frequency (%)		
HELLP syndrome	4 (7.1%)	10 (18.2%)	0.079
Placental abruption	2 (3.6%)	8 (14.5%)	0.041
Acute kidney injury	3 (5.4%)	11 (20.0%)	0.021
Pulmonary edema	1 (1.8%)	6 (10.9%)	0.048
DIC	0 (0%)	4 (7.3%)	0.039
ICU admission	5 (8.9%)	16 (29.1%)	0.007
Maternal mortality	0 (0%)	2 (3.6%)	0.145

DIC: Disseminated intravascular coagulation; HELLP: Hemolysis, Elevated Liver enzymes, and Low Platelet count; ICU: Intensive Care Unit.

Adverse fetal and neonatal outcomes were significantly more common among pregnancies complicated by preeclampsia and eclampsia. The incidence of preterm delivery progressively increased from normotensive women (10.7%) to preeclampsia (41.1%) and eclampsia groups (61.8%) ($p<0.001$). Low birth weight, NICU admission, and fetal distress were also significantly higher among hypertensive pregnancies, particularly in eclampsia ($p<0.001$). Intrauterine fetal death and perinatal mortality were markedly increased in women with eclampsia compared to normotensive controls and preeclamptic women ($p=0.006$ and $p=0.009$, respectively), indicating poorer perinatal outcomes with increasing disease severity (Table 5).

Table 5: Comparison of fetal and neonatal outcomes among study groups

Fetal/Neonatal Outcome	Normotensive (n=56)	Preeclampsia (n=56)	Eclampsia (n=55)	p-value
	Frequency (%)			
Preterm delivery	6 (10.7%)	23 (41.1%)	34 (61.8%)	<0.001
Low birth weight (<2.5 kg)	8 (14.3%)	29 (51.8%)	39 (70.9%)	<0.001
NICU admission	5 (8.9%)	18 (32.1%)	29 (52.7%)	<0.001
Fetal distress	4 (7.1%)	15 (26.8%)	21 (38.2%)	<0.001
Intrauterine fetal death	0 (0%)	3 (5.4%)	8 (14.5%)	0.006
Perinatal mortality	1 (1.8%)	4 (7.1%)	10 (18.2%)	0.009

NICU: Neonatal Intensive Care Unit.

Serum LDH levels showed significant positive correlation with systolic blood pressure ($r=0.71$), diastolic blood pressure ($r=0.68$), serum uric acid ($r=0.62$), and serum creatinine ($r=0.58$), indicating increasing LDH levels with worsening hypertension and renal dysfunction ($p<0.001$ for all). Conversely, serum LDH demonstrated significant negative correlation with platelet count

(r=−0.54), gestational age at delivery (r=−0.49), and birth weight (r=−0.57) (p<0.001), suggesting that elevated LDH levels were associated with thrombocytopenia, earlier delivery, and poorer fetal growth outcomes (Table 6).

Table 6: Correlation of serum LDH levels with clinical and laboratory parameters

Variable	Correlation Coefficient (r)	p-value
Systolic blood pressure	0.71	<0.001
Diastolic blood pressure	0.68	<0.001
Serum uric acid	0.62	<0.001
Serum creatinine	0.58	<0.001
Platelet count	-0.54	<0.001
Gestational age at delivery	-0.49	<0.001
Birth weight	-0.57	<0.001

Pearson's correlation analysis was applied. LDH: Lactate dehydrogenase.

Discussion

Hypertensive disorders of pregnancy remain a major contributor to maternal and perinatal morbidity and mortality, particularly in developing countries where delayed diagnosis and inadequate antenatal surveillance are common [2,3]. The present study evaluated serum lactate dehydrogenase (LDH) as an early diagnostic biomarker in pregnancies complicated by preeclampsia and eclampsia and demonstrated a strong association between elevated serum LDH levels, disease severity, maternal complications, and adverse fetal outcomes. The findings of the present study support the growing evidence that serum LDH serves as a reliable indicator of cellular injury and multiorgan dysfunction in hypertensive disorders of pregnancy [13].

In the present study, the majority of women with preeclampsia and eclampsia were primigravidas, accounting for 62.5% and 70.9% of cases respectively, which was significantly higher than the normotensive group (42.9%; p=0.006). Similar observations have been reported by Nasir et al., and Hu et al., who documented increased susceptibility of primigravida women to preeclampsia due to immunological maladaptation and abnormal placentation during first pregnancies [14,15]. Reduced gestational age at admission among hypertensive women in the current study further reflects the increased likelihood of preterm obstetric intervention necessitated by disease severity. The significantly higher proportion of unbooked pregnancies among eclamptic women (69.1%; p<0.001) also highlights the role of inadequate antenatal care in delayed detection and progression of hypertensive disease, which is consistent with reports from several Indian tertiary-care studies by Moharana et al., and Dave et al., [16,17].

The present study demonstrated significantly elevated systolic and diastolic blood pressure levels in preeclamptic and eclamptic women compared to normotensive controls (p<0.001). These findings reflect widespread endothelial dysfunction and vasospasm, which are hallmark pathophysiological features of preeclampsia. Endothelial injury leads to increased vascular permeability, intravascular coagulation, and tissue ischemia, resulting in multiorgan involvement [11]. The significantly increased frequency of pedal edema and heavy proteinuria in the hypertensive groups further supports the presence of endothelial damage and glomerular endotheliosis [9].

With regard to laboratory parameters, progressive deterioration in hematological, renal, and hepatic indices was observed with increasing disease severity. Mean platelet counts significantly decreased from normotensive pregnancies to eclampsia (2.41 ± 0.52 vs 1.42 ± 0.58 $\times 10^3/\mu L$; p<0.001), indicating platelet consumption secondary to endothelial injury and microangiopathichemolysis. Similar reductions in platelet counts have been documented by Thang et al., and Deeksha et al. Elevated serum creatinine and uric acid levels among eclamptic women in the present study indicate worsening renal impairment due to reduced renal perfusion, glomerular capillary endotheliosis, and ischemic injury [18,19]. Hepatic enzymes AST and ALT were also markedly elevated in severe disease, suggesting hepatocellular injury caused by hepatic vasospasm and periportal necrosis. Comparable findings have been described in studies by Omkara Murthy et al., and Bhati et al., who reported significant hepatic dysfunction among women with severe preeclampsia and eclampsia [20,21]. A major finding of the present study was the progressive and statistically significant rise in serum LDH levels with increasing disease severity. Mean serum LDH levels increased from 382.5 ± 96.4 U/L in normotensive women to 691.8 ± 188.6 U/L in preeclampsia and 1184.6 ± 346.7 U/L in eclampsia (p<0.001). Furthermore, 58.2% of eclamptic women had LDH levels >1000 U/L, whereas most normotensive women had LDH levels below 600 U/L. These observations are in agreement with studies by Kulkarni et al., Mehta et al., and

Pergialiotis et al., who also demonstrated significantly elevated LDH levels in severe hypertensive disorders of pregnancy [22,23,24]. LDH is an intracellular enzyme released during cellular necrosis, hemolysis, and hypoxic tissue damage. In preeclampsia and eclampsia, placental ischemia and generalized endothelial dysfunction produce widespread cellular injury leading to leakage of LDH into the circulation. Therefore, rising serum LDH levels directly reflect the extent of tissue damage and disease severity [25].

Importantly, the present study demonstrated strong associations between elevated LDH levels and severe clinical manifestations. Women with LDH >1000 U/L had significantly higher rates of severe hypertension (94.4%), thrombocytopenia (44.4%), elevated liver enzymes (66.7%), renal dysfunction (41.7%), HELLP syndrome (27.8%), and ICU admission (38.9%) compared to women with lower LDH levels ($p<0.001$ for all). These findings are biologically plausible because elevated LDH represents ongoing hemolysis, hepatic injury, and cellular hypoxia, which are central components of severe preeclampsia and HELLP syndrome [26]. Similar associations between elevated LDH and maternal complications have been reported by Jaiswar et al., who found that LDH levels above 800 U/L were significantly associated with maternal morbidity and adverse outcomes [26]. Prajapati et al., also demonstrated that LDH levels correlated positively with disease severity and maternal complications [27]. Maternal complications in the current study were considerably higher among women with eclampsia. Acute kidney injury, placental abruption, pulmonary edema, DIC, and ICU admission were significantly more frequent in eclampsia than preeclampsia ($p<0.05$). Maternal mortality occurred only among eclamptic women. These findings are consistent with previous studies by Monte et al., and Raghuwanshi et al., demonstrating that uncontrolled endothelial dysfunction and severe vasospasm can rapidly progress to multiorgan failure in eclampsia [9,11]. Elevated LDH levels may therefore serve as an early warning indicator for impending maternal complications and help identify patients requiring intensive monitoring and aggressive management [27]. The present study also demonstrated significantly poorer fetal and neonatal outcomes among women with hypertensive disorders. Preterm delivery, low birth weight, NICU admission, fetal distress, intrauterine fetal death, and perinatal mortality were markedly increased in the eclampsia group ($p<0.001$). These findings correlate with the known pathophysiology of placental insufficiency and uteroplacental hypoperfusion in preeclampsia. Chronic placental ischemia compromises fetal oxygen and nutrient supply, leading to fetal growth restriction, prematurity, and increased perinatal morbidity. Similar observations have been reported by Nguyen et al., and Teklemariam et al., who found elevated LDH levels to be associated with adverse fetal outcomes including low birth weight and increased NICU admissions [28,29].

Correlation analysis in the present study further strengthened the diagnostic utility of serum LDH. Serum LDH showed strong positive correlation with systolic blood pressure ($r=0.71$), diastolic blood pressure ($r=0.68$), serum uric acid ($r=0.62$), and serum creatinine ($r=0.58$), while demonstrating significant negative correlation with platelet count ($r=-0.54$), gestational age at delivery ($r=-0.49$), and birth weight ($r=-0.57$) ($p<0.001$ for all). These findings indicate that increasing LDH levels are associated with worsening maternal systemic involvement and poorer perinatal outcomes. The observed correlations support the hypothesis that serum LDH reflects the degree of endothelial dysfunction, hemolysis, tissue ischemia, and multiorgan damage occurring in severe hypertensive disorders of pregnancy [30].

Limitations

The present study was conducted at a single tertiary care center with a relatively limited sample size, which may restrict generalizability of the findings to the wider population. Serial monitoring of serum LDH levels during the course of pregnancy was not performed. Additionally, other biomarkers of endothelial dysfunction and oxidative stress were not evaluated. Long-term maternal and neonatal follow-up outcomes were also beyond the scope of the study.

Conclusion

The present study demonstrated that serum lactate dehydrogenase levels were significantly elevated in women with preeclampsia and eclampsia compared to normotensive pregnant women, with progressive rise corresponding to increasing disease severity. Elevated LDH levels showed strong association with severe hypertension, hepatic and renal dysfunction, thrombocytopenia, maternal complications, and adverse fetal outcomes. Significant positive correlation of LDH with blood pressure and biochemical parameters further supports its role as a marker of systemic involvement and cellular injury. Serum LDH estimation is inexpensive, rapid, and widely available, making it a valuable early diagnostic and prognostic biomarker for risk stratification and timely management of hypertensive disorders of pregnancy.

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