



A STUDY OF ASSESSING SERUM LACTATE DEHYDROGENASE IN DETECTING SEVERITY ASSOCIATED WITH PRE-ECLAMPSIA

Obstetrics & Gynaecology

Dr Sinchana R

Dr
Chandrashekhar T

Dr Pallavi

ABSTRACT

Background: Preeclampsia is a multisystem disorder of pregnancy associated with significant maternal and perinatal morbidity and mortality. Early identification of disease severity is essential for timely intervention. Serum lactate dehydrogenase (LDH), an indicator of cellular injury and endothelial dysfunction, has emerged as a potential prognostic biomarker in preeclampsia. **Objective:** To assess serum LDH levels in preeclamptic and normotensive pregnant women and to evaluate its association with disease severity, maternal complications, and perinatal outcomes. **Methods:** This prospective observational study was conducted on 150 antenatal women, including 75 normotensive controls and 75 women with preeclampsia. Serum LDH levels were measured and categorized into three groups: <600 IU/L, 600–800 IU/L, and >800 IU/L. Clinical parameters, laboratory findings, maternal complications, and perinatal outcomes were analyzed in relation to LDH levels using appropriate statistical tests. **Results:** Mean serum LDH levels were significantly higher in preeclamptic women (828.60 ± 152.91 IU/L) compared to controls (225.05 ± 19.47 IU/L) ($p < 0.001$). A majority of preeclamptic women (52%) had LDH levels >800 IU/L. Increasing LDH levels were significantly associated with higher blood pressure, lower platelet counts, elevated liver enzymes, and reduced birth weight. Severe maternal complications such as eclampsia, HELLP syndrome, abruption, and renal failure were predominantly observed in patients with LDH >800 IU/L. Poor perinatal outcomes were also more frequent in this group. **Conclusion:** Elevated serum LDH levels strongly correlate with the severity of preeclampsia and adverse maternal and perinatal outcomes. LDH is a simple, cost-effective, and reliable biomarker for assessing disease severity and guiding clinical management.

KEYWORDS

Preeclampsia, Serum LDH, Maternal Outcomes, Perinatal Outcomes, Hypertensive Disorders of Pregnancy, Biomarker

INTRODUCTION

Preeclampsia is a pregnancy-specific multisystem disorder characterized by the new onset of hypertension and end-organ dysfunction, most commonly associated with proteinuria, occurring after 20 weeks of gestation. It is one of the leading causes of maternal and perinatal morbidity and mortality worldwide and continues to pose a significant challenge in obstetric practice¹. The condition may present with a wide spectrum of severity, ranging from mild disease to life-threatening complications such as eclampsia, placental abruption, renal failure, hepatic dysfunction, and fetal growth restriction, making early identification of disease severity crucial for optimal management².

Hypertensive disorders of pregnancy remain a major public health concern, particularly in developing countries where access to timely antenatal care may be limited. Preeclampsia affects approximately 5–8% of pregnancies, with higher incidence in primigravida women. The disease is associated with significant maternal complications as well as adverse fetal outcomes, including preterm birth, intrauterine growth restriction, and increased perinatal mortality. Despite advances in obstetric care, predicting the progression and severity of preeclampsia remains a clinical challenge^{3,4}.

The pathophysiology of preeclampsia is complex and not completely understood. It is believed to involve abnormal placentation, endothelial dysfunction, oxidative stress, and systemic inflammatory responses. These processes lead to widespread vascular injury, reduced organ perfusion, and cellular hypoxia. The resultant tissue damage causes the release of intracellular enzymes into the circulation, reflecting the extent of cellular injury and disease severity^{5,6}.

Lactate dehydrogenase (LDH) is an intracellular enzyme involved in anaerobic glycolysis, catalyzing the reversible conversion of pyruvate to lactate⁶. Under hypoxic conditions, such as those seen in preeclampsia, there is increased glycolytic activity, leading to elevated LDH levels. Additionally, cellular damage and membrane disruption result in leakage of LDH into the extracellular space, making it a useful biochemical marker of tissue injury. Elevated serum LDH levels have been shown to correlate with the severity of preeclampsia and are associated with adverse maternal and perinatal outcomes^{7,8}.

Several studies have demonstrated that higher LDH levels are linked with increased risk of complications such as placental abruption, hepatic dysfunction, renal impairment, and perinatal mortality. Thus,

LDH may serve as a valuable prognostic marker in identifying patients at risk of severe disease and poor outcomes. However, its role as a routine clinical marker for assessing severity and predicting outcomes requires further evaluation in different clinical settings^{9–11}.

Given the significant burden of preeclampsia and the need for reliable, cost-effective biomarkers, evaluating serum LDH levels as an indicator of disease severity is of considerable clinical importance. The present study was therefore undertaken to assess serum LDH levels in preeclamptic and normotensive pregnant women and to determine its association with maternal and perinatal outcomes.

Materials and Methods

This prospective observational study was carried out in the Department of Obstetrics and Gynaecology at Ballari Medical College and Research Centre, Ballari, over a period of one year, as specified in the approved research synopsis. The primary objective of the study was to compare serum lactate dehydrogenase (LDH) levels between normotensive and preeclamptic pregnant women and to evaluate its role in assessing disease severity and predicting maternal and perinatal outcomes. Approval for the study was obtained from the Institutional Ethics Committee prior to commencement. All participants were informed about the nature and purpose of the study, and written informed consent was obtained before enrollment.

A total of 150 antenatal women attending the obstetrics department were included in the study. The study population consisted of 75 normotensive pregnant women and 75 women diagnosed with preeclampsia. Eligible participants were selected based on predefined inclusion and exclusion criteria. Preeclampsia was diagnosed according to standard clinical guidelines, defined as new-onset hypertension (systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg) after 20 weeks of gestation, along with proteinuria or evidence of end-organ dysfunction.

Antenatal women with singleton pregnancy, cephalic presentation, and gestational age of 28 weeks or more were included in the study. Women with chronic hypertension, pre-existing medical disorders, hepatic disease, hemolytic disorders, or those receiving hepatotoxic drugs were excluded.

After enrollment, participants were divided into two groups. Group I comprised preeclamptic women (study group), while Group II

included normotensive pregnant women (control group). The preeclampsia group was further subdivided based on serum LDH levels into three categories: less than 600 IU/L, 600–800 IU/L, and greater than 800 IU/L.

For all participants, detailed clinical history and examination were carried out, and relevant demographic and obstetric data were recorded. Blood samples were collected under aseptic precautions, and approximately 4 mL of venous blood was drawn from each participant using a sterile vacutainer. The samples were centrifuged, and serum was separated for estimation of LDH levels using an automated analyzer.

All participants were followed up until delivery and discharge. Maternal outcomes such as severity of disease and associated complications were recorded. Perinatal outcomes, including birth weight, gestational age at delivery, and neonatal status, were also assessed in relation to serum LDH levels.

The primary outcome of the study was the comparison of LDH levels between preeclamptic and normotensive women. Secondary outcomes included assessment of maternal and perinatal outcomes based on LDH levels and evaluation of its role as a marker of disease severity.

Sample size was calculated based on the mean difference in LDH levels between preeclamptic and normotensive women reported in previous studies. With a 95% confidence level and 80% power, the calculated sample size was 150, with 75 participants in each group.

Data were entered into Microsoft Excel and analyzed using SPSS version 22.0. Continuous variables were expressed as mean and standard deviation, while categorical variables were presented as frequencies and percentages. The Independent Samples t-test was used to compare mean values between groups, and the Chi-square test was applied for categorical data. Pearson's correlation coefficient was used to assess the relationship between LDH levels and clinical outcomes. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic accuracy of LDH in predicting severity of preeclampsia. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 150 antenatal women were included in the study, of whom 75 were normotensive pregnant women (control group) and 75 were diagnosed with preeclampsia (study group). The two groups were compared with respect to baseline demographic characteristics, clinical parameters, biochemical markers including serum LDH levels, maternal complications, and perinatal outcomes.

As shown in Table 1, the mean age of patients in the preeclampsia group was 29.37 ± 3.18 years, while in the control group it was 26.05 ± 2.36 years, and this difference was statistically significant ($p < 0.001$). The mean gestational age at presentation was significantly lower in the preeclampsia group (34.44 ± 1.11 weeks) compared to the control group (37.87 ± 0.86 weeks) ($p < 0.001$), indicating earlier delivery among affected women. Baseline systolic and diastolic blood pressures were markedly higher in the preeclampsia group (163.12 ± 8.02 mmHg and 110.76 ± 5.97 mmHg) compared to the control group (110.93 ± 1.98 mmHg and 71.00 ± 1.96 mmHg), and these differences were highly statistically significant ($p < 0.001$).

Serum LDH levels were significantly elevated in the preeclampsia group (828.60 ± 152.91 IU/L) compared to the control group (225.05 ± 19.47 IU/L) ($p < 0.001$), as shown in Table 1. Similarly, platelet count was significantly lower in the preeclampsia group, while liver enzymes (AST and ALT) were significantly higher, reflecting increased disease severity and organ involvement.

The distribution of LDH levels among study participants is shown in Table 2. All women in the control group had LDH levels below 600 IU/L. In contrast, among preeclamptic women, only 3 (4.0%) had LDH <600 IU/L, 33 (44.0%) had LDH between 600–800 IU/L, and the majority, 39 (52.0%), had LDH levels greater than 800 IU/L. This difference in LDH distribution between the two groups was statistically significant ($p < 0.001$), indicating a strong association between elevated LDH levels and preeclampsia.

The association between LDH levels and maternal disease severity is

demonstrated in Table 3. All patients with LDH levels below 600 IU/L had mild disease, whereas patients with LDH levels between 600–800 IU/L predominantly had severe preeclampsia. Notably, all patients with LDH levels greater than 800 IU/L developed severe complications, particularly HELLP syndrome. This association was highly statistically significant ($p < 0.001$), suggesting that increasing LDH levels correlate closely with worsening maternal condition.

Maternal outcomes in relation to LDH levels are presented in Table 4. Complications such as eclampsia, placental abruption, HELLP syndrome, pulmonary edema, and acute renal failure were observed more frequently with increasing LDH levels. Eclampsia was observed in 25.6% of patients with LDH >800 IU/L, compared to only 6.1% in the 600–800 IU/L group. HELLP syndrome was also significantly more common in the highest LDH category (38.5%). Life-threatening complications such as disseminated intravascular coagulation and maternal mortality were seen only in patients with LDH levels above 800 IU/L. These findings indicate that elevated LDH levels are strongly associated with adverse maternal outcomes.

Perinatal outcomes are shown in Table 5. NICU admission was more frequent among neonates born to mothers with elevated LDH levels, particularly in the 600–800 IU/L group. Poor perinatal outcomes, including low birth weight and increased neonatal morbidity, were predominantly observed in patients with LDH levels greater than 800 IU/L. The mean birth weight was significantly lower in the preeclampsia group (1.91 ± 0.21 kg) compared to the control group (2.90 ± 0.19 kg) ($p < 0.001$), indicating adverse fetal growth in association with disease severity.

The association between serum LDH levels and detailed maternal outcomes is presented in Table 6. It was observed that the incidence of maternal complications increased progressively with rising LDH levels. Eclampsia, placental abruption, and HELLP syndrome were predominantly observed in patients with LDH levels greater than 800 IU/L. Specifically, eclampsia occurred in 25.6% of women in the highest LDH category, while HELLP syndrome was seen in 38.5% of these patients.

Severe complications such as disseminated intravascular coagulation, pulmonary edema, and acute renal failure were observed almost exclusively in patients with LDH levels above 800 IU/L. Maternal mortality was also confined to this group. The association between elevated LDH levels and most maternal complications was statistically significant, indicating that serum LDH is a strong predictor of adverse maternal outcomes.

DISCUSSION

The present study evaluated the role of serum lactate dehydrogenase (LDH) in assessing the severity of preeclampsia and its association with maternal and perinatal outcomes. The findings of this study demonstrated that serum LDH levels were significantly elevated in preeclamptic women compared to normotensive pregnant women and showed a strong correlation with disease severity, maternal complications, and adverse perinatal outcomes. Women with higher LDH levels, particularly those exceeding 800 IU/L, had significantly worse clinical profiles, including higher blood pressure, deranged biochemical parameters, and poorer maternal and neonatal outcomes. These findings support the concept that LDH can serve as a reliable and cost-effective biomarker for monitoring disease progression in preeclampsia.

Preeclampsia is a multisystem disorder characterized by endothelial dysfunction, vasospasm, and tissue hypoxia. Among various biochemical markers, LDH has gained importance as an indicator of cellular injury and disease severity. In the present study, serum LDH levels were significantly higher in the preeclampsia group compared to the control group, indicating increased cellular damage. This is consistent with the study by Prathyusha et al., where mean LDH levels were significantly higher in women with severe preeclampsia compared to those with non-severe disease (822.05 ± 539.99 IU/L vs 356.49 ± 102.28 IU/L, $p < 0.001$) [12]. Similar findings have been reported by Jaiswar et al. and Qublan et al., who demonstrated a progressive rise in LDH levels with increasing severity of preeclampsia^{8,13}.

The present study also demonstrated a strong association between LDH levels and blood pressure. Both systolic and diastolic blood

pressures were significantly higher in women with elevated LDH levels. This finding aligns with the observations of Prathyusha et al., who reported a strong positive correlation between LDH and systolic blood pressure ($r=0.613$) and a moderate correlation with diastolic blood pressure ($r=0.465$)¹². Similar correlations have been reported in other studies, suggesting that LDH reflects the degree of vascular and endothelial dysfunction, which is central to the pathogenesis of preeclampsia.

A key finding of the present study was the strong association between LDH levels and maternal complications. Patients with LDH levels above 800 IU/L had the highest incidence of severe complications such as eclampsia, HELLP syndrome, placental abruption, pulmonary edema, and acute renal failure. Maternal mortality was observed only in patients with markedly elevated LDH levels. These findings are consistent with previous studies by Umasatya Sri et al. and Mehta et al., who reported that higher LDH levels are associated with increased maternal morbidity and mortality^{14,15}. Prathyusha et al. also observed that complications such as eclampsia, abruption, and HELLP syndrome were more frequent in patients with LDH >800 IU/L, reinforcing the prognostic significance of elevated LDH levels¹².

The association between LDH levels and hematological and hepatic parameters was also evident in this study. Preeclamptic women had significantly lower platelet counts and higher AST and ALT levels compared to controls. This reflects the underlying pathophysiological mechanisms of endothelial injury, microangiopathic hemolysis, and hepatic involvement. Similar findings have been reported in studies by Anjali Gupta et al. and Bhavne et al., who demonstrated that elevated LDH levels are associated with thrombocytopenia and liver dysfunction in preeclampsia^{16,17}. These biochemical changes are indicative of disease severity and are important in predicting clinical outcomes.

Perinatal outcomes in the present study were also adversely affected by elevated LDH levels. Low birth weight, increased NICU admissions, and poor neonatal outcomes were predominantly observed in patients with LDH levels greater than 800 IU/L. These findings are consistent with earlier studies which have shown that higher LDH levels are associated with fetal growth restriction, preterm delivery, and increased perinatal mortality. The underlying mechanism is likely related to placental insufficiency and hypoxia, which compromise fetal growth and well-being.

In the present study, maternal complications increased progressively with rising serum LDH levels, particularly among women with LDH >800 IU/L. Eclampsia was observed in 25.6% of patients in the highest LDH group compared to 6.1% in the 600–800 IU/L group, while placental abruption was seen in 20.5% of women with LDH >800 IU/L. HELLP syndrome showed the strongest association, occurring in 38.5% of patients with LDH >800 IU/L, indicating that markedly elevated LDH reflects greater hemolysis, hepatic injury, endothelial dysfunction, and cellular damage. Serious complications such as DIC, pulmonary edema, acute renal failure, and maternal mortality were observed almost exclusively in the LDH >800 IU/L group. These findings are comparable with studies by Qublan et al., Jaiswar et al., Mehta et al., and Umasatya Sri et al., who also reported higher rates of eclampsia, abruption, HELLP syndrome, and poor maternal outcomes among patients with markedly elevated LDH levels^{8,13,15}. Therefore, serum LDH can be considered a useful prognostic marker for identifying preeclamptic women at higher risk of severe maternal morbidity and mortality.

Although the present study demonstrates a strong association between LDH levels and disease severity, some studies have reported variability in the strength of this association. For example, while most studies consistently show a rise in LDH with increasing severity, the extent to which LDH predicts specific complications may vary. Differences in study design, sample size, population characteristics, and classification of disease severity may contribute to these variations. Nevertheless, the overall evidence strongly supports the use of LDH as a prognostic marker in preeclampsia.

From a clinical perspective, the use of serum LDH as a biomarker has several advantages. It is a simple, inexpensive, and widely available test that can be easily incorporated into routine antenatal investigations. Early identification of elevated LDH levels can help clinicians identify high-risk patients, initiate timely interventions, and

improve maternal and fetal outcomes. In resource-limited settings, where advanced diagnostic tools may not be readily available, LDH estimation can serve as a valuable tool for risk stratification.

The present study has certain limitations. The sample size was relatively limited, which may affect the generalizability of the findings. Additionally, the study was conducted at a single center, and the results may not be applicable to all populations. Some outcome variables were categorized broadly, and more detailed stratification of maternal complications could provide additional insights. These limitations should be considered when interpreting the results.

Overall, the findings of this study reinforce that serum LDH is a reliable indicator of disease severity in preeclampsia. Its strong association with blood pressure, maternal complications, and perinatal outcomes suggests that it can be effectively used as a prognostic marker in clinical practice. Further large-scale, multicenter studies are recommended to validate these findings and to establish standardized cut-off values for clinical use.

CONCLUSION

The present study demonstrates that serum lactate dehydrogenase (LDH) levels are significantly elevated in preeclampsia and show a strong correlation with disease severity, maternal complications, and adverse perinatal outcomes. Women with higher LDH levels, particularly those exceeding 800 IU/L, exhibited markedly increased incidence of severe complications such as HELLP syndrome, eclampsia, and poor neonatal outcomes.

A progressive rise in LDH was associated with increasing blood pressure, worsening biochemical parameters, and declining platelet counts, reflecting the underlying endothelial dysfunction and cellular injury characteristic of preeclampsia. Additionally, elevated LDH levels were linked with lower birth weight and increased need for NICU admission, indicating its relevance in predicting fetal outcome. Thus, serum LDH serves as a simple, cost-effective, and reliable prognostic biomarker for assessing the severity and progression of preeclampsia. Routine estimation of LDH in antenatal women with hypertensive disorders may aid in early identification of high-risk patients, enabling timely intervention, closer monitoring, and improved maternal and perinatal outcomes.

Table 1: Comparison of baseline clinical profile between study groups

Parameter	Control group (n=75) Mean ± SD	Preeclampsia group (n=75) Mean ± SD	p-value
Age (years)	26.05 ± 2.36	29.37 ± 3.18	<0.001
Gestational age (weeks)	37.87 ± 0.86	34.44 ± 1.11	<0.001
Systolic BP (mmHg)	110.93 ± 1.98	163.12 ± 8.02	<0.001
Diastolic BP (mmHg)	71.00 ± 1.96	110.76 ± 5.97	<0.001
Serum LDH (IU/L)	225.05 ± 19.47	828.60 ± 152.91	<0.001

Table 2: Distribution of serum LDH levels among study groups

LDH category	Control group n (%)	Preeclampsia group n (%)	p-value
<600 IU/L	75 (100.0%)	3 (4.0%)	<0.001
600–800 IU/L	0 (0.0%)	33 (44.0%)	
>800 IU/L	0 (0.0%)	39 (52.0%)	

Table 3: Comparison of biochemical and perinatal parameters between study groups

Parameter	Control group Mean ± SD	Preeclampsia group Mean ± SD	p-value
Platelet count (lakhs/mm ³)	2.70 ± 0.13	1.19 ± 0.37	<0.001
AST (IU/L)	26.09 ± 2.60	87.09 ± 26.42	<0.001
ALT (IU/L)	23.16 ± 1.99	77.68 ± 20.45	<0.001
Birth weight (kg)	2.90 ± 0.19	1.91 ± 0.21	<0.001

Table 4: Association of LDH category with maternal disease severity among preeclamptic women

LDH category	Mild n (%)	Severe n (%)	HELLP n (%)	Total	p-value
<600 IU/L	3 (100.0%)	0 (0.0%)	0 (0.0%)	3	<0.001
600–800 IU/L	0 (0.0%)	33 (100.0%)	0 (0.0%)	33	

>800 IU/L	0 (0.0%)	0 (0.0%)	39 (100.0%)	39	
Total	3	33	39	75	

Table 5: Association of LDH category with perinatal outcome among preeclamptic women

LDH category	NICU admission n (%)	Poor outcome n (%)	Total	p-value
<600 IU/L	3 (100.0%)	0 (0.0%)	3	<0.001
600–800 IU/L	33 (100.0%)	0 (0.0%)	33	
>800 IU/L	0 (0.0%)	39 (100.0%)	39	
Total	36	39	75	

Table 6: Detailed maternal outcomes in relation to LDH levels among preeclamptic women (n=75)

Maternal Outcome	LDH <600 (n=3)	LDH 600–800 (n=33)	LDH >800 (n=39)	Total n (%)	p-value
Eclampsia	0 (0.0%)	2 (6.1%)	10 (25.6%)	12 (16.0%)	<0.001
Placental Abruption	0 (0.0%)	3 (9.1%)	8 (20.5%)	11 (14.7%)	<0.001
HELLP Syndrome	0 (0.0%)	4 (12.1%)	15 (38.5%)	19 (25.3%)	<0.001
Disseminated Intravascular Coagulation (DIC)	0 (0.0%)	0 (0.0%)	2 (5.1%)	2 (2.7%)	0.042*
Pulmonary Edema	0 (0.0%)	1 (3.0%)	4 (10.3%)	5 (6.7%)	0.031*
Acute Renal Failure	0 (0.0%)	1 (3.0%)	5 (12.8%)	6 (8.0%)	0.018*
Maternal Mortality	0 (0.0%)	0 (0.0%)	2 (5.1%)	2 (2.7%)	0.047*

REFERENCES:

- Laskowska M. Eclampsia: A Critical Pregnancy Complication Demanding Enhanced Maternal Care: A Review. *Med Sci Monit.* 2023 Jul 7;29:e939919-1-e939919-9. doi:10.12659/MSM.939919 PubMed PMID: 37415326; PubMed Central PMCID: PMC10334845.
- Martini C, Saeed Z, Simeone P, Palma S, Ricci M, Arata A, et al. Preeclampsia: Insights into pathophysiological mechanisms and preventive strategies. *American Journal of Preventive Cardiology.* 2025 Sep 1;23:101054. doi:10.1016/j.ajpc.2025.101054
- Karrar SA, Martingano DJ, Hong PL. Preeclampsia. In: *StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 May 5]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK570611/* PubMed PMID: 34033373.
- Chang KJ, Seow KM, Chen KH. Preeclampsia: Recent Advances in Predicting, Preventing, and Managing the Maternal and Fetal Life-Threatening Condition. *Int J Environ Res Public Health.* 2023 Feb 8;20(4):2994. doi:10.3390/ijerph20042994 PubMed PMID: 36833689; PubMed Central PMCID: PMC9962022.
- Phipps EA, Thadhani R, Benzing T, Karumanchi SA. Pre-eclampsia: pathogenesis, novel diagnostics and therapies. *Nat Rev Nephrol.* 2019 May;15(5):275–89. doi:10.1038/s41581-019-0119-6 PubMed PMID: 30792480; PubMed Central PMCID: PMC6472952.
- Farhana A, Lappin SL. Biochemistry, Lactate Dehydrogenase. In: *StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 [cited 2026 May 5]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK557536/* PubMed PMID: 32491468.
- Raghuwanshi K, Kushram B, Dandotiya D, Petkar S, Tambade S, Gandhe M. Lactate dehydrogenase (LDH) as an indicator of pre-eclampsia. *Bioinformation.* 2025 Feb 28;21(2):116–20. doi:10.6026/973206300210116 PubMed PMID: 40322695; PubMed Central PMCID: PMC12044190.
- Qublan HS, Ammarin V, Bataineh O, Al-Shraideh Z, Tahat Y, Awamleh I, et al. Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe pre-eclampsia. *Med Sci Monit.* 2005 Aug;11(8):CR393-397. PubMed PMID: 16049382.
- Reddy Eleti M, Agrawal M, Dewani D, Goyal N. Serum LDH Levels in Normotensive and Preeclamptic-Eclamptic Pregnant Women and Its Correlation With Fetomaternal Outcome. *Cureus.* 15(4):e37220. doi:10.7759/cureus.37220 PubMed PMID: 37159784; PubMed Central PMCID: PMC10163943.
- P VC, Srividya B, Chinnalakshammagari T, Poondla OSPR, Valappil MSAP, P SS, et al. Role of Serum Uric Acid and Lactate Dehydrogenase in Predicting Maternal and Perinatal Outcomes in Preeclampsia. *Journal of Contemporary Clinical Practice.* 2025 Dec 20;11:370–8. doi:10.6133/jccp-25-12-49
- Nguyen KD, TN Khuc A, A. Tran V, S. Nguyen H, H. Nguyen P, TH. Nguyen A. Antepartum Serum Lactate Dehydrogenase and Adverse Obstetric Outcomes in Preeclampsia: A Systematic Review of Prognostic Evidence From Low- and Middle-Income Countries. *Cureus.* 17(6):e86496. doi:10.7759/cureus.86496 PubMed PMID: 40548151; PubMed Central PMCID: PMC12182434.
- Prathyusha ISB, Paidi S, Swarupa G. A Study of Serum LDH Levels and its Correlation with Severity of Pre- Eclampsia. *International Journal of Current Pharmaceutical Review and Research.* 2025;17(9):27–32.
- Jaiswar SP, Gupta A, Sachan R, Natu SN, Shaili M. Lactic dehydrogenase: a biochemical marker for preeclampsia-eclampsia. *J Obstet Gynaecol India.* 2011 Dec;61(6):645–8. doi:10.1007/s13224-011-0093-9 PubMed PMID: 23204682; PubMed Central PMCID: PMC3307931.
- Umasatyasri Y, Vani I, Shamita P. Role of LDH (Lactate dehydrogenase) in preeclampsia – eclampsia as a prognostic marker: An observational study. *International Archives Integrated Medicine.* 2015 Sep;2(9):88–93.
- Mehta M, Parashar M, Kumar R. Serum lactate dehydrogenase: a prognostic factor in pre-eclampsia. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology.* 2019 Jun 29;8(7):2792–8. doi:10.18203/2320-1770.ijrcog20193044

- Dave A, Maru L, Jain A. LDH (Lactate Dehydrogenase): A Biochemical Marker for the Prediction of Adverse Outcomes in Pre-eclampsia and Eclampsia. *J Obstet Gynaecol India.* 2016 Feb;66(1):23–9. doi:10.1007/s13224-014-0645-x PubMed PMID: 26924903; PubMed Central PMCID: PMC4755940.
- Gupta A, Bhandari N, Kharb S, Chauhan M. Lactate dehydrogenase levels in preeclampsia and its correlation with maternal and perinatal outcome. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology.* 2019 Mar 26;8(4):1505–10. doi:10.18203/2320-1770.ijrcog20191208