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Original article

Serum lactate dehydrogenase levels in preeclampsia: Association with maternal and fetal outcomes

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Abstract

Background: Preeclampsia, a hypertensive disorder of pregnancy, is associated with adverse maternal and fetal outcomes. Serum lactate dehydrogenase (LDH) levels have been proposed as a marker of disease severity. This study investigates the association between LDH levels and maternal and fetal outcomes in preeclampsia.

Methods: This prospective study included 150 pregnant women beyond 28 weeks of gestation diagnosed with preeclampsia. Exclusion criteria included pre-existing conditions, multiple pregnancies, and substance use. Participants were stratified into mild (n = 100) and severe preeclampsia groups (n = 50) and further categorized by LDH levels: Group 1 (< 600 IU/L), Group 2 (600–800 IU/L), and Group 3 (> 800 IU/L). Clinical, laboratory, and fetal outcomes were assessed. Statistical analyses were performed using SPSS version 21.0, with a p-value <0.05 considered significant.

Results: Elevated LDH levels were significantly associated with severe preeclampsia (mean LDH: 966.0 ± 11.1 IU in severe vs. 567.6 ± 208.6 IU in mild cases; p< 0.001). Maternal complications, including disseminated intravascular coagulation (1.3%), eclampsia (2.7%), HELLP syndrome (2.0%), and acute renal failure (3.3%), were most frequent in group 3. Adverse fetal outcomes were more common in higher LDH groups, including low APGAR scores, low birth weight (≤ 2.5 kg in 28.7%), and increased neonatal intensive care unit (NICU) admissions (17.3% in Group 3).

Conclusion: Serum LDH levels correlate with preeclampsia severity and maternal-fetal complications. LDH> 800 IU serves as a valuable biomarker for predicting adverse outcomes, emphasizing its role in guiding clinical management. Further multicenter studies are warranted to validate these findings and improve care for preeclampsia patients.

Keywords: Biomarkers, Fetal outcomes, Maternal outcomes, Preeclampsia, Serum LDH

Introduction

Pregnancy is a physiological state characterized by extensive alterations in metabolic, biochemical, physiological, hematological, and immunological processes. In the absence of complications, these changes are typically reversible within a few days to months postpartum (1). However, hypertensive disorders during pregnancy represent a significant public health challenge, being one of the leading causes of maternal, fetal, and perinatal morbidity and mortality (2). Preeclampsia is a clinical condition unique to pregnancy, defined by the onset of hypertension, proteinuria, and, in some cases, edema after 20 weeks of gestation (3). This multisystem disorder affects approximately 5–8% of pregnancies and is associated with substantial maternal and fetal complications. It has been described as a two-stage disease: the initial stage involves inadequate placental invasion, development, and remodeling, while the subsequent stage manifests clinically through maternal hypertension, proteinuria, and end-organ dysfunction. The disorder impacts multiple maternal organ systems, including the liver, kidneys, brain, coagulation pathways, and placenta (4).

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Severe maternal complications include placental abruption, disseminated intravascular coagulation (DIC), intracranial hemorrhage, hepatic failure, acute renal failure, and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count). Fetal complications encompass growth restriction, reduced amniotic fluid, abnormal oxygenation, and preterm delivery, all of which contribute significantly to perinatal morbidity and mortality (5). Approximately 15% of pregnancies are complicated by mild preeclampsia, while severe preeclampsia occurs in 1–2% of cases (6).

Although the etiology of preeclampsia remains elusive, increasing evidence suggests that placental hypoxia plays a pivotal role in its pathogenesis, leading to endothelial dysfunction and cellular death. Defective placentation and endothelial injury are hallmark features of the condition (7). Endothelial cell dysfunction promotes vasoconstriction, platelet aggregation, and coagulation system activation, ultimately resulting in decreased blood flow to vital maternal and fetal organs. These changes are early indicators of atherosclerosis, hypertension, and coronary vasospasm (8).

Lactate dehydrogenase (LDH) is an intracellular enzyme found in tissues such as the heart, liver, kidneys, skeletal muscle, brain, and blood (9). It plays a critical role in the interconversion of pyruvate and lactate during cellular metabolism (10). Elevated LDH levels in the serum indicate increased cellular membrane permeability, hemolysis, and cellular death—processes that are prominent in preeclampsia (11).

Serum LDH levels have been proposed as a marker for assessing disease severity and predicting multi organ involvement. Elevated LDH levels correlate with disease severity, adverse maternal and fetal outcomes, and increased risk of complications (12, 13). Given that LDH is released early during hypoxia and oxidative stress, it serves as a valuable biochemical marker for evaluating the progression of preeclampsia and eclampsia (14).

Its ease of testing, combined with its diagnostic and prognostic utility, makes LDH a practical tool for guiding clinical management and improving maternal and fetal outcomes (15). LDH testing is particularly useful in the third trimester, where high serum levels are strongly associated with disease severity and poor outcomes (4, 16). This study aimed to evaluate serum LDH levels in patients with preeclampsia and explore their relationship with maternal and fetal outcomes.

Materials & Methods

This prospective study was conducted at Deen Dayal Upadhyay Hospital, New Delhi, following approval from the institutional ethics committee. Pregnant women beyond 28 weeks of gestation diagnosed with preeclampsia were included in the study.

Exclusion criteria encompassed individuals with essential hypertension or hypertension diagnosed before 20 weeks of gestation, as well as those with pre-existing conditions such as renal disease, diabetes mellitus, liver disorders, epilepsy, hemolytic anemia, coronary artery disease, chronic lung disease, multiple pregnancies, smoking, and alcoholism.

The sample size was determined based on a prior study conducted by Jharia et al., (17) which assessed the prognostic value of LDH as a marker of preeclampsia severity. Assuming a sensitivity (p) of 70% for LDH levels >600 in predicting maternal and perinatal outcomes with a 10% margin of error, the minimum required sample size at a 5% level of significance was calculated to be 150 participants. Written informed consent was obtained from all participants or their legal representatives.

A total of 150 pregnant women beyond 28 weeks of gestation with preeclampsia who attended the antenatal clinic or emergency services were enrolled in the study. Among these, 100 participants were diagnosed with mild preeclampsia, while 50 had severe preeclampsia. Data collection was performed using a pre-designed and pretested proforma, which captured clinical history, baseline characteristics, and findings from physical examinations, including baseline blood pressure measurements using the auscultatory method. Participants underwent laboratory and diagnostic investigations, including a complete blood count, blood grouping and typing, platelet count, random blood sugar levels, LDH levels, blood urea, serum creatinine, direct and indirect bilirubin levels, alanine transaminase (ALT) and aspartate transaminase (AST) levels, urine analysis (albumin, sugar, and microscopy), and ultrasound assessment for fetal weight, amniotic fluid index, placental location, and maturity. Additional investigations included color

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Doppler studies, fundoscopy, prothrombin time, bleeding time (BT), clotting time (CT), and international normalized ratio (INR). Blood samples for LDH levels were collected under aseptic conditions, and participants were stratified into three groups based on LDH levels: group 1 (<600 IU/L), group 2 (600–800 IU/L), and group 3 (>800 IU/L).

Mild preeclampsia was defined as a pregnant individual beyond 20 weeks of gestation presenting with blood pressure readings of ≥ 140/90 mmHg but <160/110 mmHg on at least two occasions, measured six hours apart, in conjunction with proteinuria of ≥ 1+ (≥30 mg/dL) detected via dipstick method in a random urine sample, after excluding urinary tract infections. Severe preeclampsia was defined as the presence of any of the following:

Systolic blood pressure ≥ 160 mmHg or diastolic blood pressure ≥ 110 mmHg on two occasions, six hours apart, while at rest; proteinuria ≥ 5 g in a 24-hour urine specimen or ≥ 3+ on two random samples collected at least four hours apart; oliguria < 500 mL in 24 hours; cerebral or visual disturbances; pulmonary edema or cyanosis; epigastric or right upper quadrant pain; impaired liver function; thrombocytopenia (platelet count < 100,000/mm³); or intrauterine growth restriction.

Statistical Analysis: For statistical analysis, categorical variables were expressed as frequencies and percentages, while continuous variables were reported as means ± standard deviations (SD) or medians. Comparisons of normally distributed continuous variables between groups were conducted using the Student's t-test. A p-value < 0.05 was considered statistically significant. Data entry was performed using Microsoft Excel, and statistical analyses were conducted using SPSS software version 21.0.

Results

Total of 150 subjects were enrolled of which 100 were mild preeclampsia and 50 were severe preeclampsia. Table 1 presents the distribution of mild and severe preeclampsia patients according to age, parity, gestational period, and serum LDH levels.

Regarding age, the majority of patients in both mild and severe preeclampsia groups were between 21 and 30 years old, accounting for 52.7% of mild preeclampsia cases and 26.7% of severe preeclampsia cases. The youngest group (< 20 years) comprised 8.7% of mild preeclampsia cases and 4.7% of severe preeclampsia cases, while patients over 40 years represented 0.7% in both mild and severe preeclampsia groups.

Table 1. Distribution of mild and severe preeclampsia patients by age, parity, gestational period, and LDH levels (n= 50)

	Mild preeclampsia		Severe preeclampsia		Total	
	No.	%	No.	%	No.	%
Age (years)						
<20	13	8.7	7	4.7	20	13.3
21-30	79	52.7	40	26.7	119	79.3
31-40	7	4.7	2	1.3	9	6.0
>40	1	0.7	1	0.7	2	1.3
Parity						
Primigravida	50	33.3	30	20.0	80	53.3
Secondary gravida	42	28.0	17	11.3	59	39.3
Multigravida	8	5.3	3	2.00	11	7.3
Period of gestation(weeks)						
28 -30	2	1.3	3	2.0	5	3.3
31-33	3	2.0	1	0.6	4	2.6
34-36	24	16.0	16	10.6	40	26.6
37-40	71	47.3	30	20.0	101	67.3
>40	00	0.0	00	0.0	00	0.0
LDH						
<600 IU	50	33.3	03	2.0	53	35.3
600-800 IU	41	27.3	09	6.0	50	33.3
>800 IU	09	06.0	38	25.3	47	31.3

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Based on the information provided in the search in terms of parity, primigravida patients made up 33.3% of the mild preeclampsia group and 20.0% of the severe preeclampsia group. Secondary gravida patients comprised 28.0% and 11.3%, while multigravida patients represented 5.3% and 2.0% of the mild and severe preeclampsia groups, respectively. In terms of parity, primigravida patients made up 33.3% of the mild preeclampsia group and 20.0% of the severe preeclampsia group. Secondary gravida patients comprised 28.0% and 11.3%, while multigravida patients represented 5.3% and 2.0% of the mild and severe preeclampsia groups, respectively. Finally, regarding serum LDH levels, the majority of mild preeclampsia patients had LDH levels below 600 IU (33.3%), followed by those with levels between 600–800 IU (27.3%). In contrast, the severe preeclampsia group showed a higher proportion of patients with LDH levels greater than 800 IU (25.3%), compared to those with levels below 600 IU (2.0%). In this study, the mean systolic BP was significantly higher in the severe preeclampsia group (181.2 ± 12.3 mmHg) compared to the mild preeclampsia group (147.8 ± 6.7 mmHg) (P < 0.001). Similarly, the mean diastolic BP was also significantly higher in the severe preeclampsia group (114.2 ± 7.2 mmHg) compared to the mild preeclampsia group (94.9 ± 4.7 mmHg) (P < 0.001). Serum LDH levels were notably higher in the severe preeclampsia group, with a mean of 966.0 ± 211.1 IU, compared to the mild preeclampsia group, which had a mean of 567.6 ± 208.6 IU (P < 0.001) (Table 2). Serum LDH levels were notably higher in the severe preeclampsia group, with a mean of 966.0 ± 211.1 IU, compared to the mild preeclampsia group, which had a mean of 567.6 ± 208.6 IU (P < 0.001) (Table 2). In this study out of total 150 babies, the distribution of preeclamptic women by LDH levels reveals worsening fetal outcomes with higher LDH. APGAR scores ≤7 at 1 minute were observed in 1.3% (<600 IU), 4.0% (600–800 IU), and 8.7% (>800 IU), while scores ≥8 totaled 86.0%. At 5 minutes, scores ≤7 occurred only in the >800 IU group (4.0%), with scores ≥8 in 96.0% overall. Low birth weight (≤2.5 kg) was more common with higher LDH: 19.3% (<600 IU), 18.0% (600–800 IU), and 28.7% (>800 IU). NICU admissions increased with LDH, including respiratory distress (5.3%), prematurity (6.0%), low birth weight (6.7%), and intrauterine growth restriction (IUGR) (8.0%), with total neonatal intensive care unit (NICU) admissions at 2.6%, 6.0%, and 17.3% for LDH <600 IU, 600–800 IU, and >800 IU, respectively. Higher LDH levels were strongly associated with adverse fetal outcomes (Table 4).

Table 2. Distribution of mild preeclampsia and severe preeclampsia patients based on systolic blood pressure, diastolic blood pressure, and serum lactate dehydrogenase (LDH) levels

Parameters	Mild preeclampsia (n=100) Mean ± SD	Severe preeclampsia (n=50) Mean ± SD	p value
Systolic BP* (mmHg)	147.8 ± 6.7	181.1 ± 12.2	<0.001
Diastolic BP (mmHg)	94.9 ± 4.7	114.1 ± 7.1	<0.001
LDH level (IU)	567.5 ± 208.5	966.0 ± 211.0	<0.001

* BP: Blood pressure

Table 3. Distribution of preeclampsia women according to the serum lactate dehydrogenase (LDH) levels and correlation with its maternal outcomes.

Maternal Outcomes	LDH levels <600(IU)		600-800(IU)		>800 (IU)		Total	
	No.	%	No.	%	No.	%	No.	%
Disseminated intravascular coagulation	0	0.0	1	0.6	2	1.3	3	2.0
Eclampsia	1	0.6	1	0.6	4	2.6	6	4.0
Shock	0	0.0	1	0.6	2	1.3	3	2.0
Postpartum Hemorrhage	0	0.0	0	0.0	3	2.0	3	2.0
Acute rheumatic fever	1	0.6	0	0.0	5	3.3	6	4.0
HELLP syndrome	1	0.6	0	0.0	3	2.0	4	2.6
Abruption	0	0.0	1	0.6	3	2.0	4	2.6
Pulmonary edema	0	0.0	0	0.0	0	0.0	0	0.0
Retinopathy	0	0.0	0	0.0	0	0.0	0	0.0
Intracranial hemorrhage	0	0.0	0	0.0	0	0.0	0	0.0
Total	3	1.9	4	2.6	22	14.6	29	19.3

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Table 4: Distribution of preeclampsia women according to the serum LDH levels and correlation with its fetal outcomes

Fetal outcomes	LDH levels							
	<600 (IU)		600-800 (IU)		>800 (IU)		Total	
	No.	%	No.	%	No.	%	No.	%
APGAR at 1 minute ≤7	2	1.3	6	4.00	13	8.7	21	14.0
APGAR at 1 minute ≥8	51	34.0	44	29.3	34	22.6	129	86.0
Total	53	35.3	50	33.3	47	31.3	150	100.0
APGAR at 5 minute ≤7	0	0.0	0	0.0	6	4.0	6	4.0
APGAR at 1 minute ≥8	53	35 .3	50	33.3	41	27.3	144	96.0
Total	53	35.3	50	33.3	47	31.3	150	100.0
Fetal weight ≤ 2.5 kg	29	19.3	27	18	43	28.6	99	66.0
Fetal weight ≥ 2.6 kg	24	16.0	23	15.3	4	2.6	51	34.0
Total	53	35.3	50	33.3	47	31.3	150	100.0
NICU admission								
Respiratory distress	1	0.6	2	1.3	5	3.3	8	5.3
Prematurity	1	0.6	3	2.0	5	3.3	9	6.0
Low birth weight	1	0.6	2	1.3	7	4.6	10	6.6
Intrauterine growth restriction	1	0.6	2	1.3	9	6.0	12	8.0
Total	4	2.6	9	5.9	26	17.3	39	26.0

Discussion

This study underscores a robust association between elevated serum LDH levels, severe preeclampsia, and adverse maternal and fetal outcomes. Patients with severe preeclampsia exhibited significantly higher blood pressure and LDH levels. Notably, LDH >800 IU was linked to complications such as DIC (1.3%), eclampsia (2.7%), HELLP syndrome (2.0%), and acute rheumatic fever (ARF) (3.3%). Higher LDH levels also correlated with poorer fetal outcomes, including lower APGAR scores (≤7 at 1 minute in 8.7% of cases with LDH >800 IU) and increased NICU admissions due to respiratory distress (3.3%) and low birth weight (4.7%). Additionally, infants with birth weights ≤2.5 kg were more prevalent in the high LDH group.

These findings emphasize LDH as a valuable biomarker for predicting preeclampsia severity and guiding management to improve maternal and neonatal outcomes. Among the 150 patients studied, 53.3% were primigravida, with primigravidity observed in 33.3% of mild preeclampsia cases and 20% of severe cases. This aligns with prior research, such as Sajith et al., (18), who reported 53.8% of hypertensive pregnancies occurring in primigravida. Similar trends were documented by Aabidha et al., (19), Chan P et al., and Mjahed et al., (20, 21) who noted primiparity rates of 52–73% in preeclampsia cases. This study underscores a robust association between elevated serum LDH levels, severe preeclampsia, and adverse maternal and fetal outcomes. Patients with severe preeclampsia exhibited significantly higher blood pressure and LDH levels. Notably, LDH >800 IU was linked to complications such as DIC (1.3%), eclampsia (2.7%), HELLP syndrome (2.0%), and ARF (3.3%). Higher LDH levels also correlated with poorer fetal outcomes, including lower APGAR scores (≤7 at 1 minute in 8.7% of cases with LDH >800 IU) and increased NICU admissions due to respiratory distress (3.3%) and low birth weight (4.7%). Additionally, infants with birth weights ≤2.5 kg were more prevalent in the high LDH group.

These findings emphasize LDH as a valuable biomarker for predicting preeclampsia severity and guiding management to improve maternal and neonatal outcomes. Among the 150 patients studied, 53.3% were primigravida, with primigravidity observed in 33.3% of mild preeclampsia cases and 20% of severe cases. This aligns with prior research, such as Sajith et al., (18), who reported 53.8% of hypertensive pregnancies occurring in primigravida. Similar trends were documented by Aabidha et al., (19), Chan P et al.,

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and Mjahed et al. (20, 21) who noted primiparity rates of 52–73% in preeclampsia cases. Serum LDH levels were significantly elevated in severe preeclampsia, with mean values of 966.0 ± 211.1 IU compared to 567.6±208.6 IU in mild cases (P< 0.001). This is consistent with studies by Qublan et al. (23) and Andrews L et al. (22) which demonstrated a significant rise in LDH levels with disease severity (P<0.001). Higher LDH levels were strongly associated with increased maternal complications, including DIC, eclampsia, shock, PPH, renal failure, HELLP syndrome, and placental abruption. Maternal morbidity was reported in 19.3% of patients, with 14.7% of these cases having LDH >800 IU. Similar findings were noted in studies by Umasatyasri et al,(24) Martin JN et al., (25) and Catanzerite (26) et al., which highlighted the predictive value of elevated LDH for maternal morbidity.

Low birth weight was also associated with elevated LDH levels. Among 150 deliveries, 66% of infants weighed ≤ 2.5 kg, with LDH levels < 600 IU in 19.3%, 600–800 IU in 18%, and > 800 IU in 28.7%. This correlation mirrors findings by He et al. and Jaiswar (16) et al., who also observed decreasing birth weights with increasing LDH levels (P<0.001). APGAR scores at 1 and 5 minutes were significantly lower in newborns of mothers with higher LDH levels, with 14% of babies scoring ≤7 at 1 minute. NICU admissions for respiratory distress, prematurity, low birth weight, and IUGR were also more frequent in cases with LDH >800 IU, consistent with findings by Jaiswar et al. (16).

This study, however, has limitations. One potential limitation of this study is the inclusion of participants from a single medical center, which may limit the generalizability of the findings to broader populations. Additionally, the study excluded individuals with pre-existing conditions, multiple pregnancies, and lifestyle factors such as smoking and alcoholism, which may influence the severity of preeclampsia. This selective inclusion criterion could underestimate the complexity of real-world scenarios where such comorbidities are prevalent. Finally, while LDH levels were analyzed as a marker of preeclampsia severity, the study did not account for potential confounding factors that might influence LDH levels, such as undiagnosed metabolic or hematologic disorders, which could impact the interpretation of the results. Future research with a larger, more diverse cohort and control for confounders is necessary to validate and expand upon these findings.

Conclusion

This study highlights the significant correlation between elevated serum LDH levels, the severity of preeclampsia, and adverse maternal and fetal outcomes. Higher LDH levels (> 800 IU) were strongly associated with severe complications, such as DIC, eclampsia, HELLP syndrome, and adverse neonatal outcomes, including low birth weight, lower APGAR scores, and increased NICU admissions. These findings support the role of LDH as a valuable biomarker in predicting disease severity and guiding clinical management. While the results align with existing literature, further research with larger, multicenter cohorts is warranted to validate these observations and improve maternal and neonatal care in preeclampsia.

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Conflicts of Interest

The authors declare that no conflict of interest.

References

1. Saleem HM, Muhammed TM, Al-Hetty HRAK, Salman DA. Physiological, hematological and some biochemical alterations during pregnancy. Int J Health Sci 2022; 6(S6): 7156-69.
2. Dzakpasu S, Nelson C, Darling EK, et al. Trends in rate of hypertensive disorders of pregnancy and associated morbidities in Canada: a population-based study (2012–2021). Cmaj 2024; 196(26): E897-E904.
3. Bartal MF, Sibai BM. Gestational Hypertension, Preeclampsia, and Eclampsia. Queenan's Management of High-Risk Pregnancy: An Evidence-Based Approach 2024: 281-7.
4. Sarkar PD, Sogani S. Evaluation of serum lactate dehydrogenase and gamma glutamyl transferase in preeclamptic pregnancy and its comparison with normal pregnancy in third trimester. 2013.
5. Panda S, Das R, Sharma N, Das A, Deb P, Singh K. Maternal and perinatal outcomes in hypertensive

[Downloaded from caspjrm.ir on 2026-11-11]

[DOI: 10.22088/caspjrm.10.2.3]

disorders of pregnancy and factors influencing it: a prospective hospital-based study in Northeast India. Cureus 2021; 13(3).
6. Saleem FR, Chandru S, Biswas M. Evaluation of total LDH and its isoenzymes as markers in preeclampsia. J Med Biochem 2020;39(3):392.
7. Chiang Y-T, Seow K-M, Chen K-H. The pathophysiological, genetic, and hormonal changes in preeclampsia: A systematic review of the molecular mechanisms. Int J Mol Sci 2024; 25(8): 4532.
8. Zhao Y, Yang M, Liu Y, Wan Z, Chen M, He Q, et al. Pathogenesis of cardiovascular diseases: effects of mitochondrial CF6 on endothelial cell function. Mol Cell Biochem 2024: 1-13.
9. Meng X, Wu W, Tang Y, , et al. Lactate/Hydroxycarboxylic Acid Receptor 1 in Alzheimer's Disease: Mechanisms and Therapeutic Implications-Exercise Perspective. Molecular Neurobiology 2024:1-15.
10. Barrak M, Dawood F, Abed Shubar S, Al-fahham A. Pathophysiology, The Biochemical and Clinical Significance of Lactate Dehydrogenase. Med Res Med Res 2024; 3(07): 440-3.
11. Burwick RM, Feinberg BB. Complement activation and regulation in preeclampsia and hemolysis, elevated liver enzymes, and low platelet count syndrome. Am J Obstet Gynecol 2022; 226(2): S1059-S70.
12. Lavanya B, Ullagaddi R, Pavani M, Rao KS. Evaluation of serum lactate dehydrogenase as early diagnostic biomarker in pregnancy with preeclampsia and eclampsia. India J Obstet Gynecol Res 2023; 9(1): 83-7.
13. Bhandari N, Gupta A, Kharb S, Chauhan M. Lactate dehydrogenase levels in preeclampsia and its correlation with maternal and perinatal outcome. Int J Reprod Contracept Obstet Gynecol 2019; 8(4): 1505.
14. Simon S, Krishnan V, Ramachandran L. A Hospital-based Study on assessing the significance of Serum Lactate dehydrogenase level in Preeclampsia and its association with Maternal and Fetal outcome. Res J Pharm Tech 2024; 17(5): 2109-13.
15. Singh P, Gaikwad HS, Marwah S, Mittal P, Kaur C. Role of serum lactate dehydrogenase in pregnancy induced hypertension with its adverse feto-maternal outcome-a case-control study. J Clin Diagn Resc 2018; 12(5).
16. Jaiswar S, Gupta A, Sachan R, Natu S, Shaili M. Lactic dehydrogenase: a biochemical marker for preeclampsia–eclampsia. J obstetet Gynecol India 2011; 61: 645-8.
17. Jharia J, Mathur P, Dave A, Mathur P. A prospective study to assess role of serum lactate dehydrogenase in prediction of adverse outcomes of pre-eclampsia and eclampsia. Int J Reprod Contracep Obstet Gynecol 2016; 5: 2522-9.
18. Sajith M, Nimbargi V, Modi A, Sumariya R, Pawar A. Incidence of pregnancy induced hypertension and prescription pattern of antihypertensive drugs in pregnancy. Int J Pharma Sci Res 2014; 23: 4.
19. Aabidha PM, Cherian AG, Paul E, Helan J. Maternal and fetal outcome in pre-eclampsia in a secondary care hospital in South India. Journal of family medicine and primary care 2015; 4(2): 257-60.
20. Chan P, Brown M, Simpson JM, Davis G. Proteinuria in pre-eclampsia: how much matters? BJOG: An International Journal of Obstetrics & Gynaecology 2005; 112(3): 280-5.
21. Mjahed K, Alaoui SY, Barrou L. Acute renal failure during eclampsia: incidence risks factors and outcome in intensive care unit. Renal failure 2004; 26(3): 215-21.
22. 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. Medical science monitor: international medical journal of experimental and clinical research 2005; 11(8): CR393-7.
23. Umasatyasri Y, Vani I, Shamita P. Role of LDH (Lactate dehydrogenase) in preeclampsia-eclampsia as a prognostic marker: An observational study. International Archives of Integrated Medicine 2015; 2(9).
24. Martin Jr JN, May WL, Magann EF, Terrone DA, Rinehart BK, Blake PG. Early risk assessment of severe preeclampsia: admission battery of symptoms and laboratory tests to predict likelihood of subsequent significant maternal morbidity. American journal of obstetrics and gynecology. 1999; 180(6): 1407-14.

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[DOI: 10.22088/caspjrm.10.2.3]

25. Catanzarite VA, Steinberg SM, Mosley CA, Landers CF, Cousins LM, Schneider JM. Severe preeclampsia with fulminant and extreme elevation of aspartate aminotransferase and lactate dehydrogenase levels: high risk for maternal death. American journal of perinatology. 1995; 12(05): 310-3.

26. Demir SC, Evruke C, Ozgunen FT, Urunsak IF, Candan E, Kadayifci O. Factors that influence morbidity and mortality in severe preeclampsia, eclampsia and hemolysis, elevated liver enzymes, and low platelet count syndrome. Saudi medical journal. 2006; 27(7): 1015.

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