RESEARCH
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ASSOCIATION OF LDH LEVEL WITH PERINATAL OUTCOME AMONG WOMEN WITH GESTATIONAL HYPERTENSION, PREECLAMPSIA AND ECLAMPSIA - A TERTIARY HOSPITAL BASED CASE CONTROL STUDY

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ABSTRACT

Introduction: Gestational hypertension, preeclampsia, and eclampsia are significant contributors to maternal and perinatal morbidity and mortality. Lactate dehydrogenase (LDH) is an intracellular enzyme that increases in response to cellular damage and stress, and its elevated levels are associated with adverse maternal and fetal outcomes in hypertensive disorders of pregnancy. This study aims to evaluate the association of serum LDH levels with perinatal outcomes among women diagnosed with gestational hypertension, preeclampsia, and eclampsia.

Materials and Methods: This prospective case control study, conducted over a period of 1 year at TEZPUR MEDICAL COLLEGE AND HOSPITAL, included 130 pregnant women categorized into two groups: 62 with normal pregnancies and 68 patients with PIH.

Results: Elevated LDH levels were significantly associated with adverse perinatal outcomes in all three groups. Women with eclampsia had the highest mean LDH levels, followed by those with preeclampsia and gestational hypertension. High LDH levels correlated with an increased risk of preterm birth, low birth weight, IUGR, and higher rates of NICU admission. The association between elevated LDH levels and poor perinatal outcomes was more pronounced in the preeclampsia and eclampsia groups compared to the gestational hypertension group.

Conclusion: This study suggests that elevated serum LDH levels in pregnant women with gestational hypertension, preeclampsia, and eclampsia are significantly associated with adverse perinatal outcomes. LDH can serve as a useful biochemical marker for predicting perinatal complications in hypertensive disorders of pregnancy, allowing for timely intervention and improved maternal and fetal prognosis.

Keywords: LDH, gestational hypertension, preeclampsia, eclampsia, perinatal outcome, case-control study.

INTRODUCTION

Hypertensive disorders continue to be one of the most critical and unresolved challenges in obstetrics [(1)]. among the lethal triads that contribute to maternal mortality and morbidity during pregnancy are hypertension, Hemorrhage, and infection. [(2)] It causes complications in as many as 10% of pregnancies. Hypertensive diseases account for 14% of deaths making pregnancy more complicated. Hypertensive disorders remain one of the most significant factors contributing to maternal and perinatal mortality and morbidity.

Hypertension is a symptom of an underlying condition rather than a disease on its own. It implies a rise in peripheral vascular resistance or, more commonly, an increase in cardiac output. Hypertension is diagnosed based on clinical criteria when systolic blood pressure exceeds 140 mm Hg and diastolic pressure exceeds 90 mm Hg. Preeclampsia is defined by elevated blood pressure and the onset of proteinuria. The core pathophysiology is thought to be a defective placentation that triggers an inflammatory response that results in endothelial dysfunction [(3)]. This dysfunction results in extensive endothelial leakage, which can lead to potentially life-threatening conditions like, hepatocellular necrosis, disseminated intravascular coagulation (DIC), eclampsia, placental abruption, and severe renal failure [(4)]. The human placenta frequently produces lactate and consumes large amounts of glucose and glycolysis is a crucial energy mechanism. Lactate dehydrogenase (LDH), which transforms pyruvate into lactate, is activated by hypoxia, which also enhances glycolysis. The secreted LDH is an intracellular enzyme that has a high degree of sensitivity and is useful in the diagnosis of several diseases involving damaged cells. Compared to a normal pregnancy, the placenta in preeclampsia has greater levels of gene expression and lactate dehydrogenase activity [(5)]. Hypoxia causes trophoblasts LDH isoenzyme activity to rise, which raises lactate production. LDH comes in five different varieties, with type 4 being the most common in the placenta. It is present in preeclamptic individual's placenta [(6)]. The early identification, close observation, and treatment of these high-risk individuals with higher LDH levels may reduce maternal and fatal morbidity and death by preventing these problems.

MATERIALS AND METHODS

This was a case control study conducted at the department of obstetrics and gynecology TEZPUR MEDICAL COLLEGE AND HOSPITAL from September 2022– OCT 2023. Written informed consent was obtained from all participants before their participations. A certificate from the institutional ethical committee was obtained before starting this study. The method of sampling was purposive sampling.

Sample Size: 130

Study population-

All antenatal women after 20 weeks of gestation who came for admission in Tezpur medical college and hospital.

Inclusion criteria

All antenatal women after 20 weeks of gestation.

Exclusion criteria

1. Essential hypertension
2. preexisting renal disease
3. liver disease
4. diabetes mellitus
5. Multiple pregnancy
6. Epilepsy
7. pregnancy with fetal anomalies
8. HIV

After excluding above cases total 130 antenatal women taken. Out of which 62 patients were normotensive and 68 patients were those having BP >140/ 90 mmHg who were admitted in Tezpur medical college.

After completing the informed consent process properly, a pre-established proforma was filled out, which included a comprehensive history taking and clinical examination.

Statistical Analysis:

For statistical analysis, data were initially entered into a Microsoft Excel spreadsheet and then analyzed using SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and Graph Pad Prism (version 5). Numerical variables were summarized using means and standard deviations, while categorical variables were described with counts and percentages. Two-sample t-tests, which compare the means of independent or unpaired samples, were used to assess differences between groups. Paired t-tests, which account for the correlation between paired observations, offer greater power than unpaired tests. Chi-square tests (χ^2 tests) were employed to evaluate hypotheses where the sampling distribution of the test statistic follows a chi-squared distribution under the null hypothesis; Pearson's chi-squared test is often referred to simply as the chi-squared test. P-values were determined from Student's t-distribution tables. A p-value ≤ 0.05 was considered statistically significant, leading to the rejection of the null hypothesis in favour of the alternative hypothesis.

RESULT AND ANALYSIS

Table: 1 Association between Groups with all parameters

	Group				
		Case	**Control**	**Total**	**P value**
Parity	**Primi**	25	12	37	0.241
	(%)	36.8	19.4	28.5	
	Multi	43	50	93	
	(%)	63.2	80.6	71.5	
	Total	68	62	130	
	(%)	100	100	100	
Mode Of Delivery	**C-Sec**	56	12	68	<0.0001
	(%)	82.4	19.4	52.3	
	VD	12	50	62	
	(%)	17.6	80.6	47.7	
	Total	68	62	130	
	(%)	100	100	100	
Signs Of Prematurity	**YES**	42	10	52	<0.0001
	(%)	66.7	16.1	41.6	
	No	21	52	73	
	(%)	33.3	83.9	58.4	
	TOTAL	63	62	125	
	(%)	100	100	100	
IUGR	**YES**	38	10	48	<0.0001
	(%)	60.3	16.1	38.4	
	NO	25	52	77	
	(%)	39.7	83.9	61.6	
	TOTAL	63	62	125	
	(%)	100	100	100	
NICU Admission	**Yes**	55	3	58	<0.0001
	(%)	87.3	4.8	46.4	
	NO	8	59	67	

	(%)	12.7	95.2	53.6	
	TOTAL	63	62	125	
	(%)	100	100	100	
Death	**IUFD**	5	0	5	
	(%)	7.4	0	3.8	
	Neonatal Death	8	0	8	
	(%)	11.8	0	6.2	
	Live Birth	55	62	117	
	(%)	80.9	100	90	
	TOTAL	68	62	130	
	(%)	100	100	100	0.0014

Table: 2 Association between LDH Category with all parameters

	LDH Category					
		LDH (<600)	**LDH-2 (600-800)**	**LDH-3 (>800)**	**TOTAL**	**P VALUE**
Mode Of Delivery	**C-SEC**	13.00	13.00	42.00	68.00	<0.0001
	(%)	21.00	76.50	82.40	52.30	
	VD	49.00	4.00	9.00	62.00	
	(%)	79.00	23.50	17.60	47.70	
	TOTAL	62.00	17.00	51.00	130.00	
	(%)	100.00	100.00	100.00	100.00	
Signs Of Prematurity	**Yes**	10.00	10.00	32.00	52.00	<0.0001
	(%)	16.10	58.80	69.60	41.60	
	No	52.00	7.00	14.00	73.00	
	(%)	83.90	41.20	30.40	58.40	
	TOTAL	62.00	17.00	46.00	125.00	
	(%)	100.00	100.00	100.00	100.00	
IUGR	**Yes**	10.00	6.00	32.00	48.00	<0.0001
	(%)	16.10	35.30	69.60	38.40	
	No	52.00	11.00	14.00	77.00	
	(%)	83.90	64.70	30.40	61.60	
	TOTAL	62.00	17.00	46.00	125.00	
	(%)	100.00	100.00	100.00	100.00	
NICU Admission	**Yes**	3.00	13.00	42.00		<0.0001
	(%)	4.80	76.50	91.30	58	
	No	59.00	4.00	4.00	46.4	
	(%)	95.20	23.50	8.70	67	
	TOTAL	62.00	17.00	46.00	53.6	
	(%)	100.00	100.00	100.00	125 100.0	

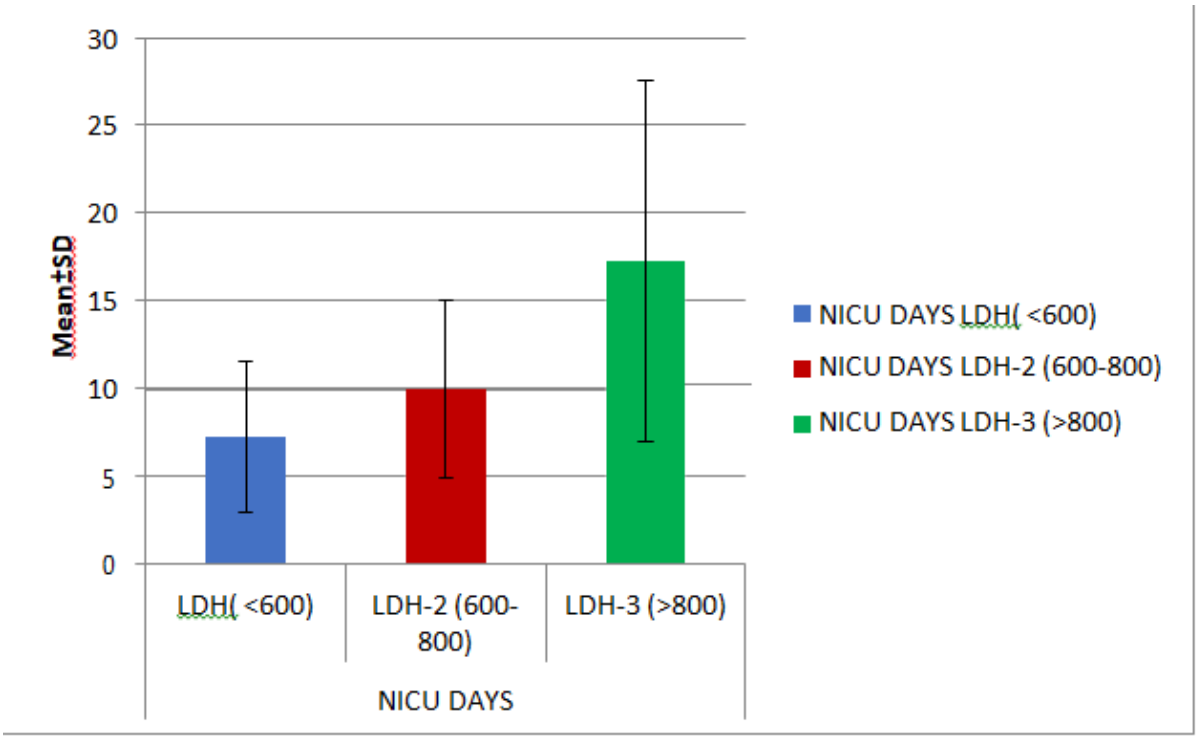
Table: 3 Distribution of mean GESTATIONAL AGE (AT WEEKS) and BIRTH WEIGHT

		Number	Mean	SD	Minimum	Maximum	Median	p-value
GESTATIONAL AGE (AT WEEKS)	LDH(<6 00)	62.00	37.55	1.92	33.00	40.00	38.00	<0.0001
	LDH-2 (600-800)	17.00	36.35	1.93	32.00	40.00	37.00	
	LDH-3 (>800)	51.00	34.14	3.41	24.00	41.00	34.00	
BIRTH WEIGHT	LDH(<600)	62.00	2662.26	361.65	1900.00	3600.00	26500.00	
	LDH-2 (600-800)	17.00	2329.41	308.25	1700.00	2850.00	2400.00	<0.0001
	LDH-3(>800)	51.00	1855.88	504.15	800.00	2650.00	2050.00	

Table – 8: Distribution between APGAR at 0 min, 1 min and 5 min vs Group

GROUP						
		CASE	**CONTROL**	**TOTAL**	**Chi-square**	**P-value**
APGAR AT 0	**≤7**	62	38	100	26.9137	<0.0001
	Col %	98	61.3	80		
	>7	1	24	25		
	Col %	1.6	38.7	20		
	TOTAL	63	62	125		
	Col %	100	100	100		
APGAR AT 1MIN	**≤7**	46	14	60	31.8453	<0.0001
	Col %	73	22.6	48		
	>7	17	48	65		
	Col %	27	77.4	52		
	TOTAL	63	62	125		
	Col %	100	100	100		
APGAR AT 5 MIN	**≤7**	24	8	32	10.412	0.0012
	Col %	38	12.9	25.6		
	>7	39	54	93		
	Col %	62	87.1	74.4		
	TOTAL	63	62	125		
	Col %	100	100	100		

Mean NICU DAYS: LDH category



Above table shows in Cases, 43 (63.2%) patients were multigravida and 25 (36.8%) patients were Primigravida. In Control, 50 (80.62%) patients were Primigravida and 12 (19.4%) patients were Primigravida. Between the two groups, there was no discernible difference in the parity distribution. (p = 0.2410) Table shows in Case, 56 (82.4%) patients had C-Sec Mode of Delivery and 12 (17.6%) patients had VD Mode Of Delivery.

In Control, 12 (19.4%) patients had C-Sec Mode of Delivery and 50 (80.6%) patients had VD Mode of Delivery and it was statistically significant (P<0.0001).

Above table illustrates that in Case, 42 (66.7%) babies had Signs of prematurity while in Control group, 10 (16.1%) babies had Signs of Prematurity was significantly more common in cases compared to controls(P <0.001).

Table shows in Case, 38 (60.3%) babies had IUGR. While in Control, 10 (16.1%) babies had IUGR. IUGR statistically significantly more common in cases compared to control groups. (p<0.0001)

Above table shows 68 (52.3%) patients were delivered by lower segment c- sec. out of which 13(21%) patients had LDH <600, 13 (76.5%) had LDH 600-800, 42 (82.4%) had LDH > 800. while 62 (47.7%) had normal vaginal delivery out of which 49 (79%) had LDH level <600, 4 (23.5%) had LDH level (600-800) and 9(17.6) had LDH level > 800. C-sec was significantly increased with higher LDH level. (p<0.0001

Above table shows out of 125 babies, 52 (41.6%) babies had prematurity. out of which 10 (16.1%) babies had LDH <600, 10 (58.8%) babies had LDH between 600-800, 32 (69.6%) babies had LDH >800. While 73(58.4%) had no signs of prematurity 62 (83.9%) had LDH <600, 7 (41.2 %) had LDH (600-800) and 14(30.4%) had LDH > 800.Statistical analysis revealed that the likelihood of premature births was significantly higher in women with elevated LDH levels (p<0.0001).

Out of 125 babies 48 (38.4 %) had IUGR. out of which 10 (16.1%) babies had LDH<600, 6 (35.3 %) babies had LDH between 600-800, 14 (30.4%) had LDH >800 .On statistical analysis chances of IUGR babies are more with women having higher LDH levels. (p<0.0001).Above table shows 58 (46.4%) required NICU admission. out of which 3 (4.8%), 13 (76.5%) had LDH 600-800. 42(91.3%) had LDH >800.On statistical

analysis NICU admission are higher in women with increased LDH levels (p<0.0001).

Above table shows the mean Gestational Age (AT WEEKS) (Mean± SD) of patients with LDH <600 was 37.5484± 1.9221.the mean GESTATIONAL AGE (AT WEEKS) (Mean± SD) of patients with LDH 600-800 was 36.3529± 1.9346.the mean GESTATIONAL AGE (AT WEEKS) (Mean± SD) of patients with LDH>800 was 34.1373± 3.4119. on statistically analysis mean gestational age were lower in babies with higher serum LDH levels. Chances of premature babies born were higher in LDH levels. (p <0.0001)

According to the table, mother with LDH levels more than 600 IU/L had baby with mean birth weight (Mean± SD) of 2662.26 ± 361.65 grams. Mean BIRTH WEIGHT (Mean± SD) of baby of mother with LDH 600-800 was 2329.4118± 308.2505.Mean BIRTH WEIGHT (Mean± SD) of baby of mother with LDH >800 was 1855.8824± 504.1475.Mean birth weight was lower in babies with higher LDH levels. Chances of low-birth- weight babies born were higher in women with higher LDH levels (p<0.0001).

Above table shows in CASE, at 0 min 62 (98.4%) patients had APGAR score <7 and 1 (1.6%) patient had APGAR score >7. In CONTROL, at 0 min 38 (61.3%) patients had APGAR score <7 and 24 (38.7%) patients had APGAR score >7. There was a statistically significant (p<0.0001) correlation between the group and the Apgar scores at 0 minutes.

At 1 min in CASE, 46 (73.0%) patients had APGAR score < 7 and 17 (27.0%) patients had APGAR score >7. In CONTROL, 14 (22.6%) patients had APGAR score <7 and 48 (77.4%) patients had APGAR score >7. There was a statistically significant (p<0.0001) correlation between the group and the Apgar scores at 1 minutes.

At 5 min in CASE, 24 (38.1%) patients had APGAR AT score <7 and 39 (61.9%) patients had APGAR >7. In CONTROL, 8 (12.9%) patients had APGAR AT <7 and 54 (87.1%) patients had APGAR >7. There was a statistically significant (p<0.0001) correlation between the group and the Apgar scores at 5 minute.

DISCUSSION

In our study of 130 patients, the control group had a higher proportion of multigravida patients [50 (80.6%)] compared to the case group [43 (63.2%)]. However, this difference was not statistically significant (p=0.280), indicating that parity distribution between the control and case groups was comparable. This suggests that parity was unlikely to have influenced the outcomes being studied, allowing for a more balanced comparison between the two groups.

In the study by **Reddy Eleti et al.[7]**, among both the normotensive and preeclamptic-eclamptic groups, 107 patients (46.2%) were primigravida, 92 (40%) were second gravida, 24 (10.43%) were third gravida, and 7 (3.04%) were fourth gravida or higher. The distribution of parity was comparable across all groups. The difference may not have been significant (p = 0.9849), despite the fact that primigravida were more numerous than multigravida in the instances. Our investigation revealed that multigravida was more prevalent than primigravida.

In our study, preterm births were notably higher in the case group, where 52 out of 62 infants (66.7%) were preterm, compared to only 10 (16.1%) in the control group. Among these preterm infants, there was a significant association with elevated maternal LDH levels: 32 had levels above 800 IU/ml, 10 had levels between 600-800 IU/ml, and 10 had levels below 600 IU/ml (p < 0.0001). The odds ratio of 10.4 suggests that newborns of mothers with preeclampsia-eclampsia and elevated LDH levels are 10.4 times more likely to be born preterm than those of normotensive mothers.

The finding consistent with the study conducted by **Reddy Eleti et al. [7]** and **Gupta et al** [8]. who found that pre-term birth was more common in patients with preeclampsia with LDH level >800 IU/L.

Our study establishes a significant link between hypertensive disorders in pregnancy (preeclampsia/eclampsia) and increased rates of intrauterine growth restriction (IUGR) in neonates. The incidence of IUGR was notably higher in infants born to mothers with preeclampsia/eclampsia (60.3%) compared to those born to normotensive mothers (6.1%). Additionally, maternal lactate dehydrogenase (LDH) levels above 800 IU/L were associated with a substantial percentage of IUGR cases (69.6%), showing strong statistical significance ($p < 0.0001$) .

Reddy Eleti et al. [7]. found 11 cases of IUGR in the normotensive group with serum LDH levels below 600 IU/L, compared to 47 cases in the preeclamptic-eclamptic group with serum LDH levels between 600 and 800 IU/L. **Lavanya et al [9]** and **Prajapati et al [10]** found strong correlation seen between elevated LDH levels and IUGR in patients with preeclampsia and eclampsia.

In this study, 11 babies born to normotensive mothers with serum LDH levels below 600 IU/L experienced complications such as jaundice, hypoxia, respiratory distress syndrome (RDS), hypoxic-ischemic encephalopathy (HIE), sepsis, and necrotizing enterocolitis (NEC), while 51 babies did not face any issues. In comparison, among mothers with preeclampsia or eclampsia and serum LDH levels between 600 and 800 IU/L, 8 newborns encountered neonatal complications. For mothers with serum LDH levels exceeding 800 IU/L, 46 newborns developed complications, with RDS being the most commonly observed problem. The differences noted were statistically significant ($p < 0.0001$).

This finding is consistent with the study by **Reddy Eleti et al [7]**. found three infants delivered by normotensive mothers with serum LDH levels under 600 IU/L had asphyxia, whereas 37 infants born to mothers with preeclampsia or eclampsia had asphyxia.

Studies by **Jaiswar et al** [11]**.** reported that elevated LDH levels were significantly linked to an increase in prenatal problems.

Our study found a clear correlation between elevated LDH levels and the likelihood of NICU admission. Specifically, a significant majority of newborns with LDH levels greater than 800 IU/ml required NICU care, compared to a smaller percentage of those with LDH levels between 600-800 IU/ml and an even smaller percentage with LDH levels below 600 IU/ml. The p-value of less than 0.0001 indicates a statistically significant association between LDH levels and the requirement for NICU admission. This suggests that the differences observed in NICU admission rates across the different LDH groups are unlikely to be due to random chance.

This finding is consistent to the study by **Vyas NP et al [12]**. In 2021, who reported that 54.4% of newborns having serum LDH levels above 600 IU/L were admitted to the NICU.

In our study, the average birth weight of newborns was found to be 2.66 kg for those born to mothers with LDH levels above 600 IU/ml. In contrast, newborns with maternal LDH levels between 600-800 IU/ml had an average weight of 2.3 kg, while those with levels exceeding 800 IU/ml weighed only 1.85 kg. These findings indicate that as maternal blood LDH levels increase, particularly in cases of preeclampsia and eclampsia, there is a corresponding decrease in mean birth weight. This relationship was statistically significant ($p < 0.0001$).

This is similar to the findings reported by **Ajila D** et al [13]. where pregnant hypertensive women with serum LDH levels above 800 IU/L had the lowest average birth weight of 2.16 ± 0.39 kg.

Bhave NV et al. [14] found that at one minute and five minutes, mothers with blood LDH levels < 600 IU/L had APGAR scores of 8.29 and 8.45, respectively. For moms with blood LDH levels ranging from 600 to 800 IU/L, APGAR scores were 8.09 at one minute and 8.41

at five minutes. Mothers with blood LDH levels more than 800 IU/L had an APGAR score of 5.22 at one minute and 5.59 at five minutes.

In our study, the occurrence of intrauterine fetal death (IUFD) was notably observed, with five cases identified among preeclamptic and eclamptic patients whose maternal LDH levels exceeded 800 IU/L. The presence of IUFD in this context emphasizes the need for close monitoring and management of pregnant patients with significantly elevated LDH levels, as they may be at an increased risk for adverse fetal outcomes.

Reddy Eleti et al [7]found that there were five cases of intrauterine fetal death (IUFD) among preeclamptic and eclamptic patients with maternal LDH levels greater than 800 IU/L.

In our study, eight newborns (15.7%) whose mothers had LDH levels exceeding 800 IU/L succumbed while receiving care in the NICU. This finding indicates a significant association between elevated maternal LDH levels and neonatal mortality, with a p-value of less than 0.0002. The results highlight the critical impact of maternal LDH levels on neonatal outcomes, suggesting that higher LDH levels may serve as a warning signal for increased risk of mortality in affected newborns.

Bhave NV et al[14]. noted that there were six instances of newborn deaths (14.6%) among mothers whose blood LDH levels were higher than 800 IU/L.

Contrast to our study in **Pillai SK et al** [15] study, five cases of intrauterine fetal death were noted among hypertensive patients with normal LDH levels, compared to only one case in those with elevated LDH levels. The primary causes included severe oligohydramnios, intrauterine growth restriction, and Doppler abnormalities. Additionally, one neonatal death due to respiratory depression was observed in the hypertensive group with normal LDH levels. In the hypertensive group with high LDH levels, a single stillbirth was attributed to eclampsia, though this result did not reach statistical significance. These findings suggest that hypertensive pregnancies may have varied fetal and neonatal outcomes depending on LDH levels, underscoring the complex role of LDH as a potential marker in predicting specific perinatal risks.

CONCLUSION

Preeclampsia is a significant cause of maternal mortality in India and other developing countries, highlighting the urgent need for reliable predictors and prognostic markers. Elevated serum LDH levels are strongly associated with preeclampsia, with higher levels indicating an increased risk of complications. Regular monitoring of serum LDH in pregnant women, especially those at high risk, can aid in the early detection and timely intervention of preeclampsia. Since serum LDH correlates with disease severity, it serves as a valuable biochemical marker for identifying high-risk patients who require close monitoring and effective management.

REFERENCES

1. Kumari D. An observational study on the association of biomarkers such as maternal C-reactive protein and serum uric acid with preeclampsia in Andhra Pradesh. International Journal of Clinical Obstetrics and Gynecology. 2021;5(4).
2. Arias F, Bhide AG, Arulkumaran S, Damania K, Daftary SN, editors. Arias' practical guide to high-risk pregnancy and delivery: a South Asian perspective. Elsevier Health Sciences; 2019 Oct 16.
3. Corton MM, Leveno KJ. Williams Obstetrics 24/E (EBOOK): Williams Obstetrics 24/E (EBOOK). Apple Books; 2014 Apr 28.
4. Mba, E. a. R. M. P., & Hobbins, J. C. (2008). Handbook of Clinical Obstetrics: The Fetes and Mother. John Wiley & Sons.
5. Velegrakis, A., Kouvidi, E., Fragkiadaki, P., & Sifakis, S. (2023). Predictive value of the sFlt-1/PlGF ratio in women with suspected

preeclampsia: An update (Review). International Journal of Molecular Medicine, 52(4).

6. Van De Velde, M., Scholefield, H., & Plante, L. A. (2013). Maternal critical care: A Multidisciplinary Approach. Cambridge University Press.
7. Reddy Eleti M, Agrawal M, Dewani D, et al. (April 06, 2023) 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
8. Gupta A, Bhandari N, Kharb S, Chauhan M: Lactate dehydrogenase levels in preeclampsia and its correlation with maternal and perinatal outcome. Int J Reprod Contracept Obstet Gynecol. 2019, 8:1505-10.
9. Lavanya B, Ullagaddi R, Pavani M, Rao KS: Evaluation of serum lactate dehydrogenase as early diagnostic biomarker in pregnancy with preeclampsia and eclampsia. Indian J Obstet Gynecol Res. 2022, 15:83-7.
10. Prajapati SR, Maitra N: Prediction of pre-eclampsia by a combination of maternal history, uterine artery Doppler, and mean arterial pressure (a prospective study of 200 cases). J Obste Gynaecol India. 2013, 63:32-
11. Jaiswar SP, Gupta A, Sachan R, Natu SN, Shaili M: Lactic dehydrogenase: a biochemical marker for preeclampsia-eclampsia. J Obstet Gynaecol India. 2011, 61:645-8. 10.1007/s13224-011-0093-9
12. International Journal of Reproduction, Contraception, Obstetrics and Gynecology Vyas NP et al. Int J Reprod Contracept Obstet Gynecol. 2021 Nov;10(11):4107-4112
13. International Journal of Reproduction, Contraception, Obstetrics and Gynecology Ajila D et al. Int J Reprod Contracept Obstet Gynecol. 2022
14. International Journal of Reproduction, Contraception, Obstetrics and Gynecology Bhave NV et al. Int J Reprod Contracept Obstet Gynecol. 2017
15. Sivarajan S, Pillai SK. Lactate dehydrogenase - a biochemical marker for the prediction of adverse outcomes in pre- eclampsia. J Evolution Med Dent Sci 2020;9(43):3218-3222, DOI: 10.14260/jemds/2020/70