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Serum LDH as a biochemical marker for the prediction of hypertensive disorders in pregnancy and the severity of the maternal and foetal outcome: A prospective study in a tertiary centre

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Abstract

Objective: This hospital-based case-control study explored the association between serum lactate dehydrogenase (LDH) levels and the severity of hypertensive disorders in pregnancy.

Materials and Methods: Two hundred thirty pregnant women with hypertensive disorders in pregnancy were enrolled in the study, with a 1:1 distribution of cases and controls. The investigation specifically focused on singleton pregnancies after 20 weeks of pregnancy. Participants were divided into case and control groups, the case group was further classified as preeclampsia without severe features, preeclampsia with severe features and eclampsia groups. Parameters such as blood pressure, and heart rate were recorded and blood and urine samples were collected for laboratory analysis.

Results: Most cases in preeclampsia with severe features group (30.18%) and eclampsia (15.72%) group occurred in the 21-30 age group. Preeclampsia with severe features (74.2%) and eclampsia (57.3%) cases were most prevalent between 34 and 37 weeks. Elevated proteinuria after diastolic blood pressure between 101 and 120 mmHg was observed in over half of preeclampsia with the severe feature group and eclampsia group. All eclampsia cases exhibited elevated blood LDH levels; only 19.1% of preeclampsia with severe features group had LDH below 600 IU. Adverse outcomes, including placental abruption, HELLP syndrome, intrauterine growth retardation (IUGR), and emergency lower segment caesarean section (LSCS), were noted across all the case groups. As serum LDH levels increased, the incidence of HELLP syndrome, IUGR, mortality, and placental abruption rose significantly.

Conclusion: Serum LDH emerges as a promising biochemical marker for predicting hypertensive disorders in pregnancy and assessing the severity of both maternal and foetal outcomes.

Keywords: Pregnancy, serum lactate dehydrogenase, eclampsia, proteinuria, maternal outcome, neonatal outcome

Introduction

The process of pregnancy induces significant modifications in the morphological, physiological, and metabolic characteristics of maternal tissues. However, challenges can arise at any point during pregnancy, affecting both the developing embryo and the mother. Hypertension, characterized by high blood pressure, emerges as a primary concern during pregnancy, presenting as preeclampsia or gestational hypertension, both linked to adverse outcomes [1]. Of particular concern is the potential progression of hypertension to eclampsia, a severe complication that significantly contributes to maternal mortality rate in developing nations [2]. Hypertension affects about 10% of pregnancies, with pulmonary embolism and deep vein thrombosis accounting for roughly 50% of cases globally [3, 4]. Despite extensive studies on these conditions, there still remains a gap in understanding the comprehensive nature of these ailments. Research efforts spanning decades have explored the aetiology and escalation of hypertension during pregnancy, yet definitive resolutions to these complex issues remain elusive [5]. Problems with placentation and endothelial function characterize this medical condition. Genes, race, immune system traits, dietary habits, higher insulin resistance, oxidative stress, low oxygen levels, and imbalanced prostaglandin levels can cause these problems [6]. Lactate dehydrogenase (LDH), a key enzyme converting pyruvic acid into lactic acid, is vital in glycolysis.

The placenta, crucial for providing nutrients and oxygen to the developing foetus, relies on metabolic pathways, with glycolysis being paramount. Previous studies have noted a significant increase in LDH activity and corresponding gene expression in placentas from women with preeclampsia compared to those with uncomplicated pregnancies [8-10]. Estimating LDH levels can be a useful way to check for cellular damage and dysfunction. It can also be used as a biochemical marker to show how severe the disease is if there are any complications, and what might happen to the developing baby.

Our study aimed to bridge the existing research gap by investigating the correlation between blood LDH levels and the severity of hypertensive disorders in pregnant women. The main goals were to (a) measure serum LDH levels in pregnant women with hypertensive disorders in pregnancy; (b) to find out the association between serum LDH and severity of hypertensive disorders in pregnancy; and (c) to assess the foetal-maternal outcome in hypertensive disorders in pregnancy. Through this research, we aspire to provide nuanced insights that contribute to improved predictive and diagnostic strategies, ultimately enhancing maternal and foetal health outcomes in hypertensive disorders in pregnancy.

Materials and Methods

The present study was conducted in the Department of Obstetrics and Gynaecology at SCB Medical College and Hospital, Cuttack, as a hospital-based case-control study from March 2021 to February 2022. Ethical approval for the study was obtained from the hospital ethical committee ensuring adherence to ethical standards including the Declaration of Helsinki. The investigation focused on pregnant women diagnosed with hypertensive disorders in pregnancy, all carrying a singleton pregnancy. Data were collected after 20 weeks onwards of pregnancy. All enrolled subjects were evenly divided into cases and controls, with cases further categorized into three subgroups: A, B, and C. Subgroup A included patients with preeclampsia without severe features that is blood pressure greater than or equal to 140/90 mm Hg on two occasions at least 4 hours apart without severe features with proteinuria. Subgroup B included patients with preeclampsia with severe features that is blood pressure greater than or equal to 160/110 mm Hg on two occasions at least 4 hours apart and proteinuria or in the absence of proteinuria thrombocytopenia, renal insufficiency, impaired liver function, pulmonary oedema, new onset headache unresponsive to medication and not accounted for by alternative diagnoses or visual symptoms and subgroup C included patients with eclampsia that is new onset tonic-clonic, focal or multifactorial seizures in the absence of causative condition such as epilepsy, cerebral arterial ischemia and infarction, intracranial haemorrhage, or drug use [11]. We kept patients with eclampsia in separate groups considering its completely different clinical presentation. None of the patients with eclampsia had a history of previous seizure disorders. They were categorised as eclampsia at the time of diagnosis considering the fact any convulsion in pregnancy until otherwise proven is considered as eclampsia. After stabilisation, further CT brain was done after consultation with a neurologist. They were included in eclampsia group only if CT brain did not show any signs of haemorrhage or infarction. Pregnant women meeting any of the following criteria were excluded from the study: primary hypertension, kidney disease, idiopathic epilepsy, thyroid conditions, multiple pregnancies or molar pregnancies, haemolytic diseases, and patients not willing. The target sample size was determined to be 240, evenly

distributed between 120 cases and 120 controls. Due to non-response and following the loss to follow-up and missing data, 10 patients were eliminated, resulting in a final sample size of 230 participants. Informed consent was obtained from all participants before collecting relevant data on their socio-demographic profile, principal complain, medical history, menstruation history, and any other pertinent information. Subsequently, each participant underwent a comprehensive clinical and obstetric evaluation, including recording essential physiological parameters such as heart rate and blood pressure. Blood and urine specimens were collected for laboratory analyses and aseptic measures were followed during collection of venous blood for serum lactate dehydrogenase (LDH) measurement. The LDH levels were determined using the International Federation of Clinical Chemistry (IFCC) guidelines [12]. All collected data were entered into Microsoft Excel and analysed using SPSS version 23.0. Descriptive information was expressed as a percentage, and associations were measured using Pearson's chi-square test, with statistical significance set at $P < 0.05$.

Results

In this research, patients were divided into various study groups. A total of 115 cases and 115 controls were enlisted in the study (Tables 1 & 2). The preponderance of cases (68; 59.13%) were due to preeclampsia with severe features group, while eclampsia (31; 26.96%) ranked second (Table 2). Most cases, i.e., 80 (25 eclampsia, 7 preeclampsia without severe features, and 48 preeclampsia with severe features), and controls, i.e., 79, were aged between 21 and 30 (Fig. 1). We further investigated the parity of the study participants (Table 2). The relative frequency of parity for control and cases in multigravidas and primigravidas were 47.83%, 52.17%, 35.65%, and 64.35%, respectively. The distribution of research participants based on their gestational age is illustrated in Figure 2. The predominant proportion of controls fell between the gestational age range of 38 and 40 weeks. However, a higher percentage of cases with preeclampsia with severe features group (74.2%, 39) and eclampsia (57.3%, 23) occurred during the gestational period of 34 to 37 weeks.

Next, we studied the blood pressure ranges of the controls and cases. Both SBP and DBP significantly increased in all the patients (Fig. 3). On an average, for preeclampsia without severe features, preeclampsia with severe features, and eclampsia groups, SBP and DBP ranged from 143.7 mmHg, 76.88 mmHg, 164.47 mmHg, 97.09 mmHg, 180.06 mmHg, and 99.03 mmHg, respectively (Fig. 4). Further, a positive correlation (Fig. 3) was discovered between the severity of the hypertensive disorder and the likelihood of raised systolic blood pressure (SBP). The finding was additionally corroborated by doing an LDH-dependent analysis of variance (ANOVA), which revealed a statistically significant ($P < 0.05$) association of all the parameters with LDH except for the mode of delivery and obstetrics score (Table 2).

The levels of albumin in the urine of the study groups were subsequently assessed (Fig. 5). It was observed that a considerable proportion of controls, precisely 94 individuals, or 81.7% of the total, did not exhibit any presence of albumin excretion in their urine. At the same time, 21 (18.26%) exhibited a trace amount of urine albumin. All the patients in the cases exhibited a remarkable amount of proteinuria. In contrast, there was a notable rise in proteinuria with the escalation of severity grading in hypertensive diseases. Further, the levels of lactate dehydrogenase (LDH) remarkably increased in all instances of

eclampsia and pre-eclampsia with severe features group (Fig. 5). Besides, we observed a significant correlation between the severity of hypertensive diseases and the considerable elevation of serum LDH levels (Fig.3).

Additionally, a range of adverse maternal outcomes were identified across the different study groups (Fig. 6). In comparison to the control group, a higher proportion of women diagnosed with preeclampsia without severe features (2, 10.52%), preeclampsia with severe features (16, 84.21%), and eclampsia (1, 5.26%) encountered placental abruption. Moreover, it was observed that 3 out of 31 eclamptic women (9.7%) and 5 out of 68 preeclampsia with severe features women (7.4%) had HELLP syndrome. Further investigation was conducted to examine the perinatal outcomes observed in the research groups. The average birth weight of the infants decreased considerably in preeclampsia without severe features group, compared to eclampsia and preeclampsia with severe features group (Fig. 7). Subsequently, an analysis was conducted to investigate the mode of delivery within different research cohorts. A total of 127 (55.21%) of the female participants including control and cases underwent vaginal delivery out of which 11(8.66%) were diagnosed with preeclampsia without severe features and preeclampsia with severe features each (fig.8). Nevertheless, a much higher proportion of women diagnosed with hypertensive disorders in pregnancy had an emergency lower segment caesarean section (LSCS). Specifically, 57 women (83.8%) in the preeclampsia with severe features group and 27 women (87.1%) in the eclampsia group delivered via LSCS (Fig.8).

Additionally, a study was undertaken to investigate the association between serum lactate dehydrogenase (LDH) levels and adverse maternal and perinatal outcomes (Fig. 9). When serum LDH levels were high, there was a noticeable rise in the chances of developing HELLP syndrome and placental abruption. Further, our study revealed an association between elevated LDH levels and the incidence of pulmonary oedema and mortality. In addition, a statistically significant variation (Table 2) was seen between an increase in blood LDH levels and both intrauterine growth retardation and intrauterine death.

Discussion

The current investigation, conducted within the Department of Obstetrics and Gynaecology at SCB Medical College and Hospital in Cuttack, sheds light on the prevalence and characteristics of hypertensive disorders in pregnancy. Notably, preeclampsia with severe features emerged as the predominant presentation among the patients examined, constituting 59.13% of the total cases. Eclampsia followed this in 26.96% of cases, with preeclampsia without severe features accounting for 13.91% (Table 2). Our findings align with studies conducted by Andrews *et al.* (2016), which reported a similar prevalence of severe preeclampsia but a slightly higher incidence of eclampsia [13]. A significant portion of cases occurred among people in their 20s and 30s, which highlights the vulnerability of teenagers and young females. This finding underscores the heightened susceptibility to illness and mortality in this demography of women, a concern particularly relevant in the context of limited access to education and healthcare resources, which is characteristic of developing nations like India [14, 15].

Examining the parity distribution, our results indicate a higher proportion of primigravida (64.3%) cases than in previous studies [16]. This contrasts with studies that reported a lower percentage of primigravidas in cases of preeclampsia and eclampsia [16, 19]. This prompts further exploration into the relationship between parity and the severity of hypertensive

disorders during pregnancy [21-23].

While our investigation found no statistically significant correlation between obstetrics score and diastolic blood pressure (DBP), a significant association was observed with systolic blood pressure (SBP) in some cases (Fig. 3). This aligns with previous studies that similarly reported no substantial association between obstetrics score and the prevalence of hypertensive complications during pregnancy [21, 22]. It is essential to note that in our study individuals were in the third trimester, which is consistent with earlier research findings [23, 24, 25].

Gestational age emerged as a critical factor, with a notable percentage of cases occurring between 34 and 37 weeks of gestation. This finding is consistent with prior research indicating a lesser gestational age in individuals with more pronounced hypertension issues [18]. As expected, the severity of hypertensive disease in pregnancy correlated with an increase in both systolic and diastolic blood pressure, with eclampsia cases exhibiting the highest average SBP and DBP. This aligns with findings reported by Garg *et al.* (2012) [21].

Turning attention to serum LDH levels, our study demonstrated a substantial link with the severity of hypertensive disorders in pregnancy (Fig. 9). All eclampsia cases exhibited elevated serum LDH levels, reaching 1328.39 IU, a significant elevation compared to preeclampsia without severe features cases. This finding is consistent with studies reporting increased LDH levels in preeclampsia and eclampsia patients, emphasising the potential of LDH as a valuable biomarker in assessing disease severity [26-28]. Furthermore, a positive correlation between LDH levels and birth weight was observed, adding another layer to the intricate relationship between LDH and maternal-fetal outcomes. Our study aimed to look into LDH as a possible biomarker for predicting negative outcomes. Maternal outcomes, including placental abruption and HELLP syndrome, demonstrated a clear association with the severity of hypertensive disorders. In contrast to other studies, our investigation revealed additional severe consequences, namely eclampsia, detected when LDH levels exceeded 800 IU/L. This highlights the potential of LDH in predicting established complications and identifying emerging risks, providing clinicians with valuable insights for timely intervention. Moreover, the association between elevated LDH levels and adverse perinatal outcomes, including intrauterine growth retardation (IUGR) and intrauterine death (IUD) underscores the multi-faceted implications of LDH in maternal and neonatal health [29-30]. Overall, our study contributes nuanced insights into the complex interplay between hypertensive disorders in pregnancy, LDH levels, and foetal-maternal outcomes. The prevalence of preeclampsia, the role of demographic factors, and the association between LDH and disease severity underscore the need for continued research in this field. While our findings align with existing literature, the unique aspects of our investigation, such as the focus on LDH as a biomarker, provide a valuable perspective for future studies. Moving forward, exploring the mechanistic underpinnings of LDH in hypertensive disorders and conducting larger, multi-centre studies will contribute to a more comprehensive understanding of this intricate relationship.

Though there we gained a lot from the study which can be beneficial for the patient, still we acknowledge few limitations of the study. Firstly sample size was small due to limited duration of time. Secondly this study was conducted in a single centre. So further multi centric approach with larger sample size is required to determine its applicability to larger population with different demographic profile.

Table 1: Descriptive Statistics of Quantitative and qualitative data of controls and cases

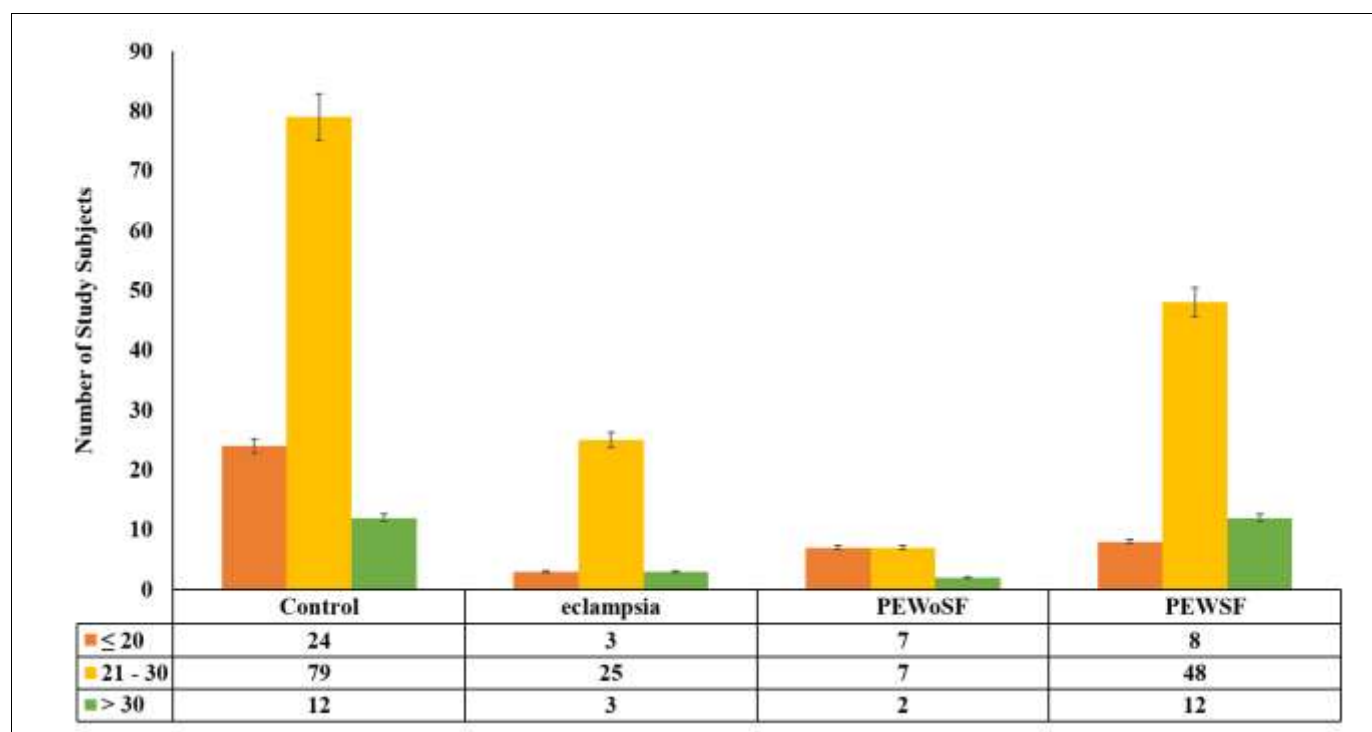
Statistic	Age (in years)		Gravida		GA(in years)		SBP(mmHg)		DBP(mmHg)		LDH(IU/L)		Baby Weight(kg)	
	Control	Cases	Control	Cases	Control	Cases	Control	Cases	Control	Cases	Control	Cases	Control	Cases
No. of observations	115	115	115	115	115	115	115	115	115	115	115	115	115	115
Minimum	19.00	18.00	1.00	1.00	34.00	30.00	110.00	140.00	60.00	60.00	104.00	302.00	1.00	1.10
Maximum	37.00	36.00	3.00	4.00	40.00	40.00	130.00	200.00	88.00	130.00	482.00	1980.00	3.20	3.75
Mean	24.31	24.56	1.51	1.44	38.38	36.67	119.88	165.79	77.76	94.80	225.67	925.36	2.52	2.48
Variance (n-1)	18.97	21.60	0.32	0.46	1.85	4.47	52.93	221.99	123.87	341.74	6129.71	152064.78	0.27	0.38
Standard deviation (n-1)	4.36	4.65	0.57	0.68	1.36	2.11	7.28	14.90	11.13	18.49	78.29	389.95	0.52	0.62
Standard error of the variance	2.51	2.86	0.04	0.06	0.25	0.59	7.01	29.40	16.41	45.26	811.90	20141.47	0.04	0.05

GA: Gestation age, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, LDH: Lactate dehydrogenase, IUGR: Intrauterine growth restriction, IUD: Intrauterine death.

Table 2: Descriptive Statistics of Quantitative and qualitative data of controls and cases

	Parity				Study groups				Urine albumin					
Sample	control		Cases		Control	Cases			Control		Cases			
No. of observations	115		115		115	115			115		115			
Category	multi	primi	multi	primi	control	eclampsia	PEWoSF	PEWSF	nil	traces	1+	2+	3+	4+
Frequency per category	55.00	60.00	41.00	74.00	115.00	31.00	16.00	68.00	94.00	21.00	12.00	35.00	56.00	12.00
Rel. frequency per category (%)	47.83	52.17	35.65	64.35	100.00	26.96	13.91	59.13	81.74	18.26	10.43	30.43	48.70	10.43
	Abortion				Delivery mode				Hellp		Abrupton			
Sample	Control	cases		Control		Cases		Control	Cases		Control	Cases		
No. of observations	115	115		115		115		115			115	115		
Category	no	no	yes	emergency lscs	normal	emergency lscs	normal	no	no	yes	no	no	yes	
Frequency per category	115.00	108.00	7.00	14.00	101.00	89.00	26.00	115.00	107.00	8.00	115.00	97.00	18.00	
Rel. frequency per category (%)	100.00	93.91	6.09	12.17	87.83	77.39	22.61	100.00	77.39	22.61	100.00	84.35	15.65	
	Pulmonary oedema				Death			IUGR			IUD			
Sample	Control	Cases		Control	Cases		Control		Cases		Control	Cases		
No. of observations	115	115		115	115		115		115		115	115		
Category	no	no	yes	no	no	yes	no	yes	no	yes	no	no	yes	
Frequency per category	115.00	114.00	1.00	115.00	114.00	1.00	111.00	4.00	71.00	44.00	115.00	110.00	5.00	
Rel. frequency per category (%)	100.00	99.13	0.87	100.00	99.13	0.87	96.52	3.48	61.74	38.26	100.00	95.65	4.35	

GA: Gestation age, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, LDH: Lactate dehydrogenase, IUGR: Intrauterine growth restriction, IUD: Intrauterine death, Pre-eclampsia without severe features (PEWoSF), pre-eclampsia with severe features (PEWSF).

**Fig 1:** Age distribution of the cases includes control, eclampsia, pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

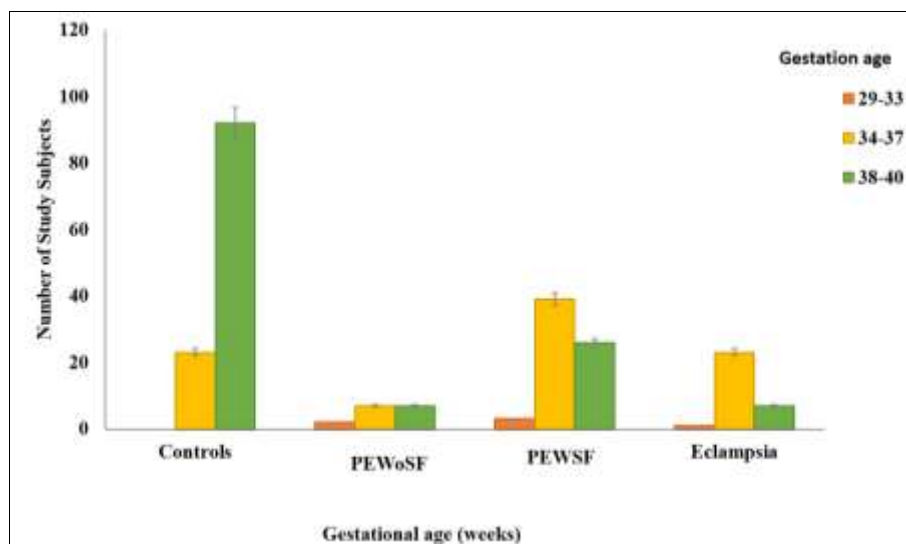


Fig 2: The distribution of study subjects based on gestational age (GA) in weeks. The data are presented as a histogram, with the frequency of subjects in each GA category on the y-axis and the GA category on the x-axis. The figure also includes a vertical line indicating the mean GA for the study population. Pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

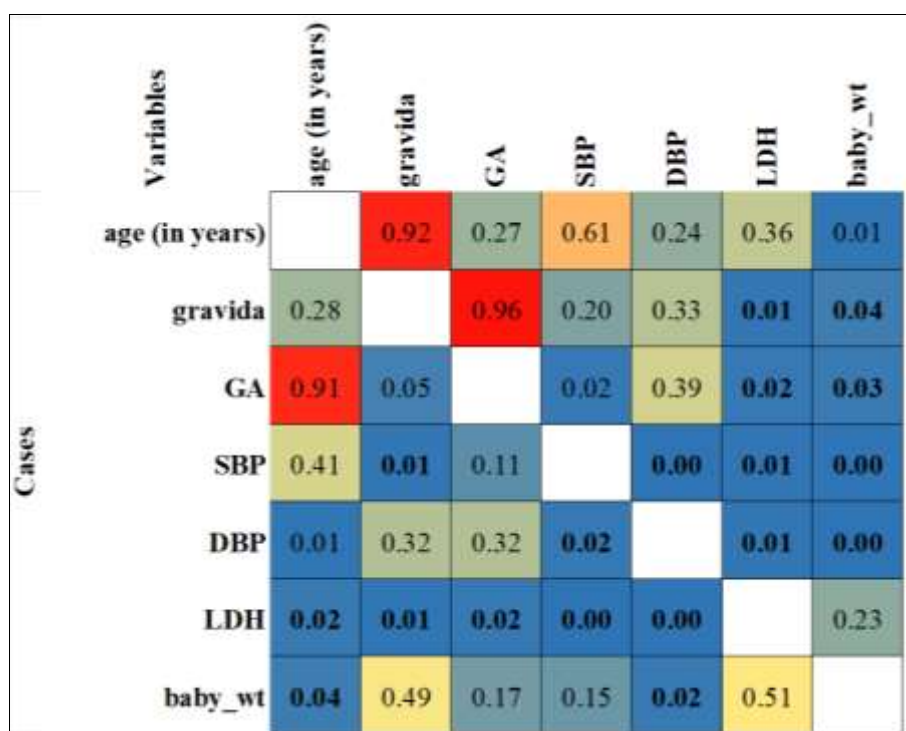


Fig 3: Correlation matrix plot showing Pearson's correlation coefficient between the studied parameters.

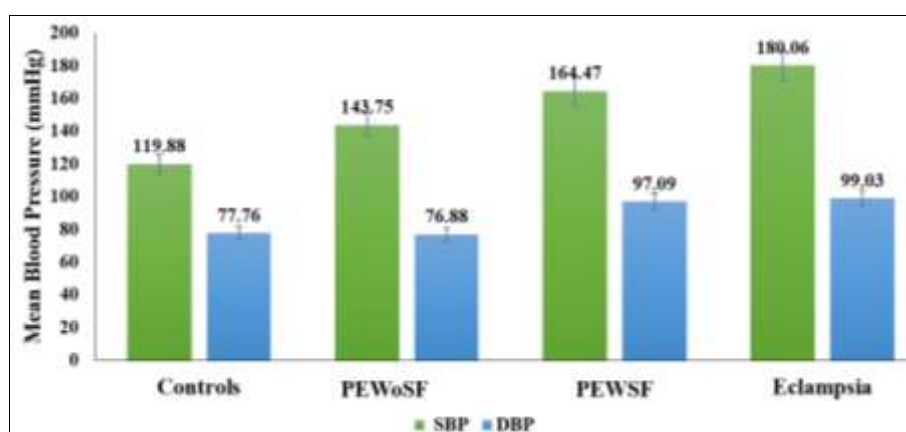


Fig 4: Distribution of mean systolic and diastolic blood pressures (in mmHg) across various study groups. Pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

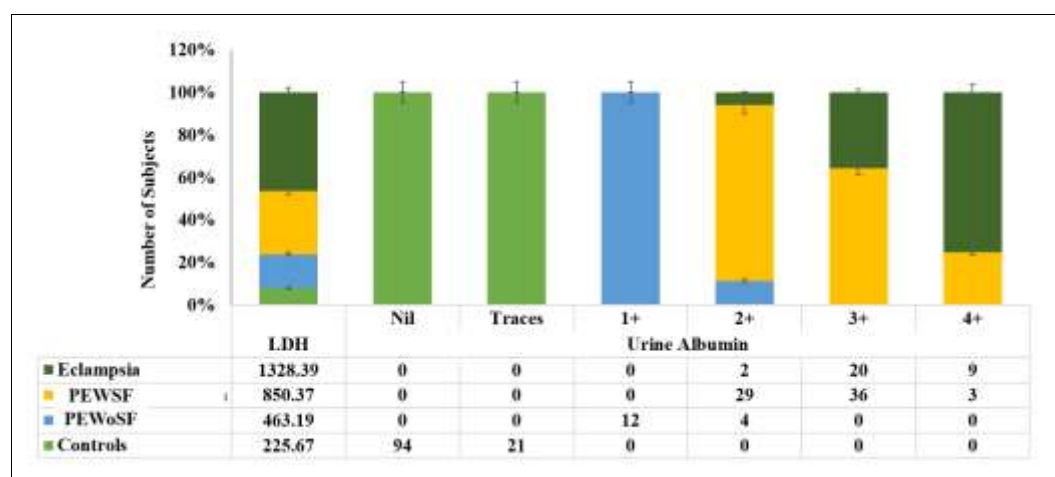


Fig 5: Distribution of LDH (IU/ml) levels among various study groups in association with urine albumin levels. Pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

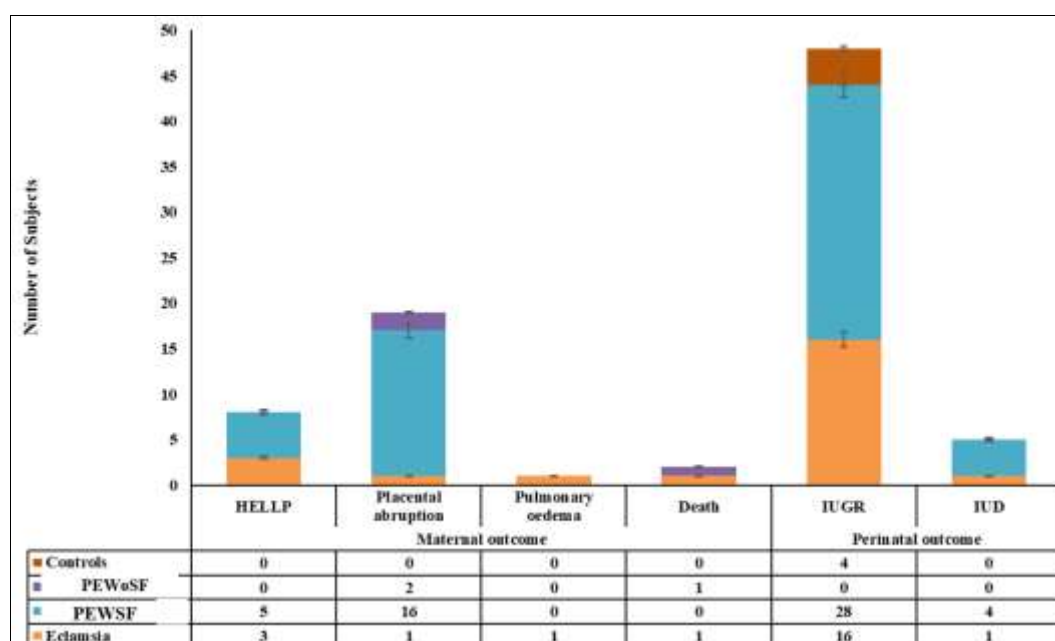


Fig 6: Maternal and perinatal outcome across different study groups. Pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

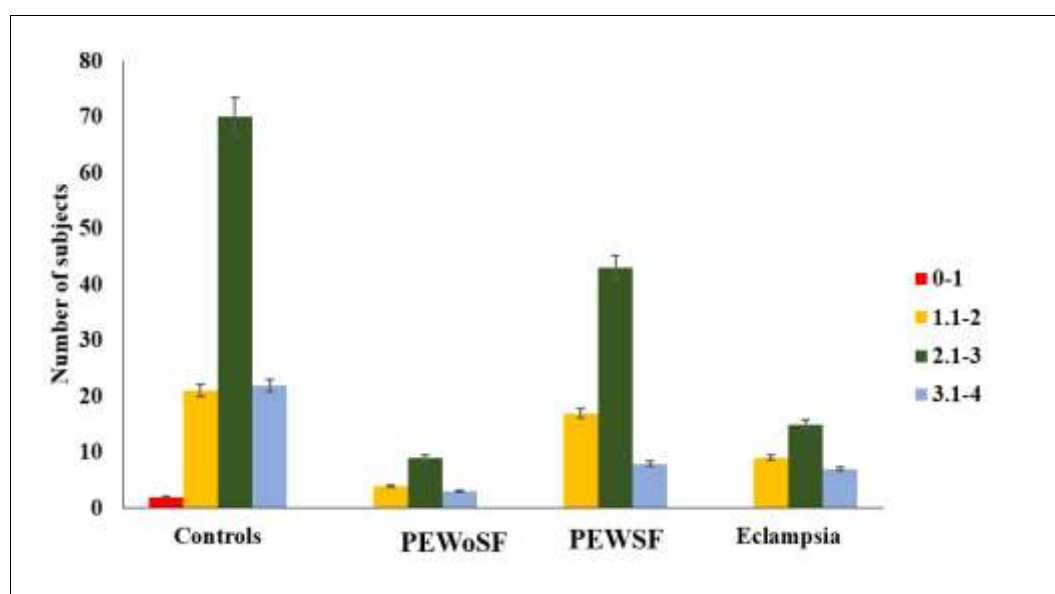


Fig 7: Birth weight across different study groups. Pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

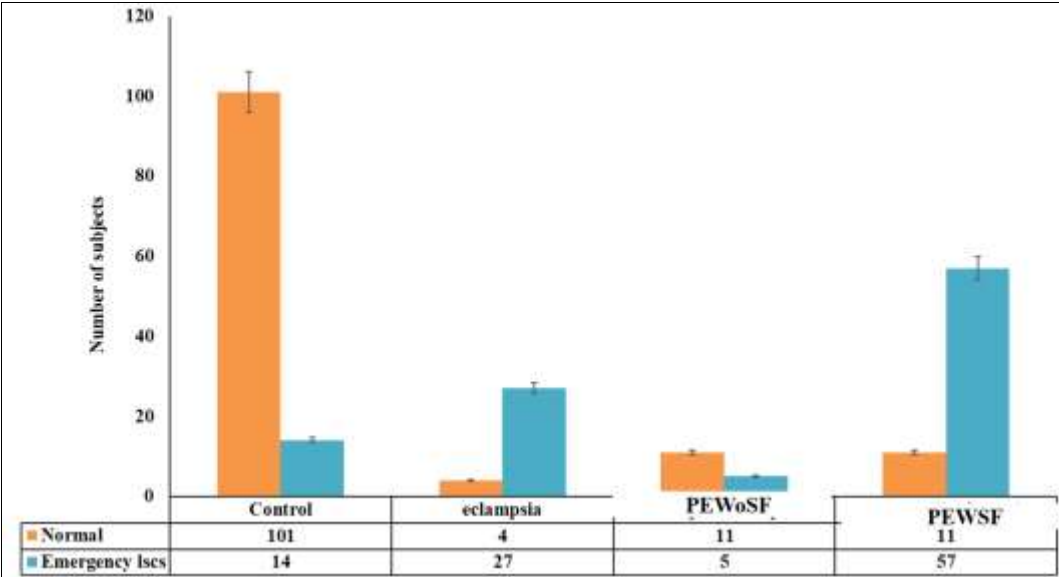


Fig 8: Mode of delivery methods was classified as normal or emergency (LSCS) in control, eclampsia, pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

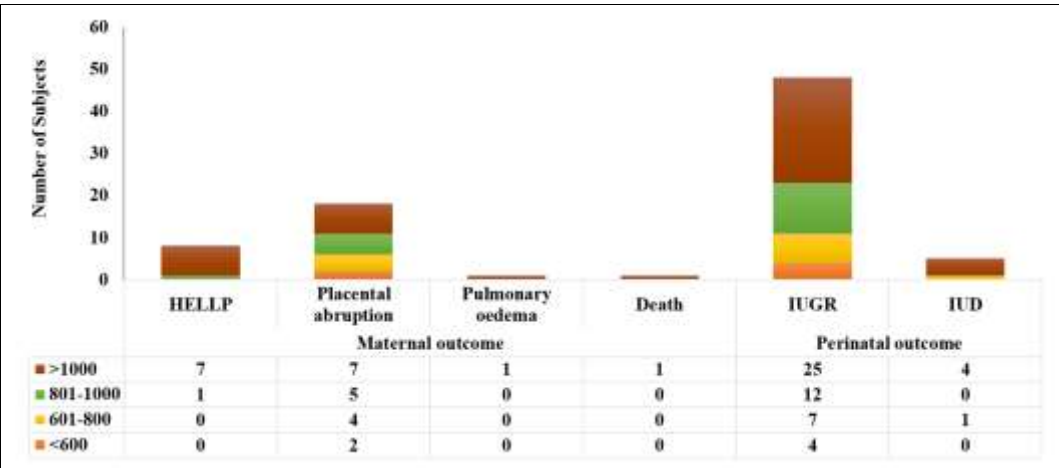


Fig 9: Association of serum LDH (IU) levels with Maternal and Perinatal outcome. Pre-eclampsia without severe features (PEWoSF) and pre-eclampsia with severe features (PEWSF).

Conclusion

Our study underscores the potential of serum lactate dehydrogenase (LDH) as a valuable biochemical indicator for predicting hypertensive disorders in pregnancy and assessing the severity of maternal and foetal outcomes. Despite the temporal limitations and a modest sample size inherent in this study, the consistent correlations observed between LDH levels and critical parameters, including blood pressure, proteinuria, adverse maternal outcomes, and perinatal complications, highlight the promising role of LDH as a comprehensive prognostic tool. The routine use, cost-effectiveness, and widespread availability of LDH assays position them as a pragmatic candidate for integration into routine antenatal assessments.

As we navigate the complexities of hypertensive disorders in pregnancy, LDH emerges as a focal point for future research endeavours. This study provides a foundational understanding of the potential applications of LDH, paving the way for more extensive investigations with larger and more diverse cohorts. Further exploration in varied populations can deepen our insights into the predictive capabilities of LDH, refining its clinical utility and contributing to the development of targeted interventions and personalized management strategies for pregnant individuals at risk of hypertensive complications. This

research opens avenues for advancing antenatal care precision and effectiveness, encouraging continued exploration within the scientific community.

We do not have any conflict of interest.

References

1. Lindheimer MD, Taler SJ, Cunningham FG. Hypertension in pregnancy. *J Am Soc Hypertens.* 2010;4(2):68-78.
2. Dimitriadis E, Rolnik DL, Zhou W, Estrada-Gutierrez G, Koga K, Francisco RPV, *et al.* Pre-eclampsia. *Nat Rev Dis Primers.* 2023 Feb 16;9(1).
3. Langley-Evans SC, Pearce J, Ellis S. Overweight, obesity and excessive weight gain in pregnancy as risk factors for adverse pregnancy outcomes: a narrative review. *J Hum Nutr Diet.* 2022 Apr;35(2):250-264.
4. Lutsey PL, Zakai NA. Epidemiology and prevention of venous thromboembolism. *Nat Rev Cardiol.* 2023 Apr;20(4):248-262.
5. Habli M, Sibai BM. Hypertensive disorders of pregnancy. In: Danforth DN, editor. *Danforth's obstetrics and gynecology.* 10th ed. Philadelphia: Lippincott Williams & Wilkins; 2008. p. 530-573.
6. Barry MJ, Nicholson WK, Silverstein M, Cabana MD,

- Chelmow D, Coker TR, *et al.* Screening for hypertensive disorders of pregnancy: US Preventive Services Task Force final recommendation statement. *JAMA*. 2023 Sep 19;330(11):1074-1082.
7. Khan AA, Allemailem KS, Alhumaydhi FA, Gowder SJT, Rahmani AH. The biochemical and clinical perspectives of lactate dehydrogenase: an enzyme of active metabolism. *Endocr Metab Immune Disord Drug Targets*. 2020;20(6):855-868.
 8. Castro KR, Prado KM, Lorenzon AR, Hoshida MS, Alves EA, Francisco RP, *et al.* Serum from preeclamptic women triggers endoplasmic reticulum stress pathway and expression of angiogenic factors in trophoblast cells. *Front Physiol*. 2022 Feb 4;12:799653.
 9. Menkhorst E, Santos LL, Zhou W, Yang G, Winship AL, Rainczuk KE, *et al.* IL11 activates the placental inflammasome to drive preeclampsia. *Front Immunol*. 2023 May 24;14:1175926.
 10. Madendag Y, Sahin E, Madenda IC, Sahin M, Kirlangic MM, Muhtaroglu S. Maternal serum telomerase levels increase in pregnancies with mild and severe preeclampsia. *Placenta*. 2022 Jun 1;123:41-45.
 11. American College of Obstetricians and Gynecologists. Gestational hypertension and preeclampsia. *ACOG Practice Bulletin No. 222*. *Obstet Gynecol*. 2020;135:e237-e260.
 12. Vanderlinde RE. Measurement of total lactate dehydrogenase activity. *Ann Clin Lab Sci*. 1985 Jan-Feb;15(1):13-31.
 13. Andrews L, Patel N. Correlation of serum lactate dehydrogenase and pregnancy-induced hypertension with its adverse outcomes. *Int J Res Med Sci*. 2016;4(5):1347-1350.
 14. Salazar JM, Villegas NC, Rojas-Zumaran V, Zaña M, Chicoma-Flores K, Campos G, *et al.* Serum levels of LDH and protein/creatinine index in pregnant women with preeclampsia: a single-center retrospective study. *Electron J Gen Med*. 2022;19(4):em378.
 15. Conde-Agudelo A, Belizan JM, Lammers C. Maternal-perinatal morbidity and mortality associated with adolescent pregnancy in Latin America: cross-sectional study. *Am J Obstet Gynecol*. 2005 Feb;192(2):342-349.
 16. Kulkarni VV, Shaikh B. Levels of LDH in normal pregnancy, pre-eclampsia, and eclampsia. *J Evol Med Dent Sci*. 2019;8(35):2768-2772.
 17. Sarkar PD, Soganani S. Evaluation of serum lactate dehydrogenase and gamma-glutamyl transferase in preeclamptic pregnancy and its comparison with normal pregnancy in third trimester. *Int J Res Med Sci*. 2013;1(4):365-368.
 18. Saleem FR, Chandru S, Biswas M. Evaluation of total LDH and its isoenzymes as markers in preeclampsia. *J Med Biochem*. 2020 Sep 2;39(3):392-398.
 19. Nadkarni J, Bahl JP. Perinatal outcome in pregnancy-associated hypertension. *Indian Pediatr*. 2001;38(2):174-178.
 20. 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 Gynecol India*. 2016 Feb;66(1):23-29.
 21. Mallika A, Rajapriya S, Sethupathy S. High serum lactate dehydrogenase levels as a prognostic marker for morbid perinatal outcome in pre-eclampsia. *Int J Clin Obstet*. 2018;2(5):88-91.
 22. Vinitha PM, Chellatamizh M, Padmanaban S. Role of serum LDH in preeclampsia as a prognostic factor: a cross-sectional case-control study in a tertiary care hospital. *Int J Reprod Contracept Obstet Gynecol*. 2017;6:595-598.
 23. Qublan HS, Amarín V, Bataineh O, Al-Shraideh Z, Tahat Y, Awamleh I. Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe preeclampsia. *Med Sci Monit*. 2005 Aug;11(8):CR393-CR397.
 24. 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*. 2023 Apr 6;15(4):e37220.
 25. Singh P, Gaikwad HS, Marwah S, Mittal PR, Kaur C. Role of serum lactate dehydrogenase in pregnancy-induced hypertension with its adverse fetomaternal outcome: a case-control study. *J Clin Diagn Res*. 2018 May 1;12(5).
 26. Mammaro A, Carrara S, Cavaliere A, Ermito S, Dinatale A, Pappalardo EM, *et al.* Hypertensive disorders of pregnancy. *J Prenat Med*. 2009 Jan;3(1):1-5.
 27. Mary VP, Chellatamizh M, Padmanaban S. Role of serum LDH in preeclampsia as a prognostic factor: a cross-sectional case-control study in a tertiary care hospital. *Int J Reprod Contracept Obstet Gynecol*. 2017 Feb;6(2):595-598.
 28. 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):645-648.
 29. Hak J, Nayar-Un-Nisa, Gupta S. LDH levels in pregnancy and its association with severity of the disease and fetomaternal outcome in pre-eclampsia and eclampsia. *JK Sci*. 2015;17(3):110-113.
 30. 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 Apr;8(4):1505-1510.

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