



Original Article

Serum LDH And GGT as A Prognostic Factor in Preeclampsia: A Case-Control Study in A Tertiary Care Hospital

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ABSTRACT

Pre-eclampsia is affecting 2% – 8% of the pregnancies worldwide (Lo et al., 2013). Defective placentation and endothelial dysfunction are considered to be the core features that aggravate hypertension in pregnancy (Var et al., 2003), (Friedman et al., 1995). Serum lactate dehydrogenase (LDH) and serum gamma-glutamyl transferase (GGT) are most often measured to evaluate the presence of tissue damage associated with Pre-eclampsia.

Aim: The aim of this study was to estimate serum LDH and GGT levels in the normal antenatal women and women with preeclampsia in the antepartum period and to correlate maternal and perinatal outcomes with these biomarkers across the study groups in a tertiary care hospital. **Materials and Methods:** The study sample comprised of 147 Antenatal women with pre-eclampsia as cases and 147 Antenatal women without pre-eclampsia as controls. Serum LDH and GGT were measured and compared and correlated with maternal and perinatal outcome in both groups and data were analyzed using SPSS. Independent Sample t-tests, Chi-square, correlation, were employed. $p < 0.05$ considered as significant. **Results:** Serum LDH and GGT level were significantly elevated in Antenatal women with pre-eclampsia (498.91 ± 147.125) when compared to Antenatal women without pre-eclampsia (259.84 ± 45.725). Elevated LDH and GGT levels were associated with worse perinatal outcome

Conclusion: This study elucidated that serum LDH and GGT levels were significantly increased in Antenatal women with pre-eclampsia when compared to Antenatal women without pre-eclampsia. Hence, serum LDH and GGT can be used as a novel biomarker for predicting early preeclampsia in pregnancy.

Keywords: Preeclampsia, LDH, GGT, endothelial dysfunction, maternal morbidity.

INTRODUCTION

Preeclampsia remains major contributors to maternal morbidity and mortality worldwide. It is the second leading direct cause of maternal mortality next to hemorrhage (Lo et al., 2013). It contributes to around 46,000 maternal deaths per year and around 5,00,000 fetal or newborn deaths (Lain et al., 2002). In India, eclampsia accounts for approximately 24% of maternal deaths during pregnancy, according to a study by the Federation of Obstetric and Gynecological Societies of India

(FOGSI)3(Friedman et al., 1995). Furthermore, 10–15% of maternal deaths are directly attributed to preeclampsia4(Lain et al., 2002).

Preeclampsia is a multisystem disorder of pregnancy, characterized by widespread endothelial dysfunction, inflammation, and cellular injury. Though it is more than simply gestational hypertension with proteinuria the appearance of protein in urine still remains a primary diagnostic criterion. This objective marker reflects the widespread endothelial leak that characterizes the preeclampsia syndrome. Early diagnosis of preeclampsia remains still challenging because these markers detect preeclampsia only after significant damage has occurred. Several biomolecules have shown potential for development as promising diagnostic markers in the early detection of pre-eclampsia.

Lactate dehydrogenase (LDH) is an intracellular enzyme released into the bloodstream following cellular damage5 (Murthy et al., 2019). Functionally, LDH plays a role in glycolysis by catalyzing the conversion of pyruvic acid to lactic acid. In the hypoxic environment associated with preeclampsia, glycolytic activity is enhanced, leading to increased LDH production and activity. Studies have demonstrated that LDH activity and gene expression are significantly elevated in the placentas of women with preeclampsia compared to those with normal pregnancies6, 7(Kay et al., 2007), (Burd et al., 1975). Among the five isoforms of LDH, the LDH4 isoform—predominantly expressed in the placenta has been shown to be highly responsive to hypoxic conditions in preeclamptic pregnancies8, 9(Bougnères et al., 1995),(Markert et al., 1975).. Elevated serum LDH levels in women with preeclampsia reflect the extent of cellular injury and tissue breakdown. Therefore, LDH serves as a valuable biochemical marker for assessing disease severity and systemic involvement in these patients.

Gamma-glutamyl transferase (GGT), an ectoenzyme linked to oxidative stress10 (Whitfield, 2001) and free radical production, has been associated with endothelial dysfunction and may play a role in the progression of preeclampsia11 (Chen et al., 2021).. We hypothesized that elevated levels of these enzymes may correlate with disease severity and the occurrence of complications. Early prediction of severity in preeclampsia is crucial for timely intervention and preventing adverse maternal and perinatal outcomes. Due to limited regional data, this study aimed to assess the association between serum LDH and GGT levels, disease severity, and complications of preeclampsia in South Indian women.

Aims & Objectives

1. To estimate serum LDH and GGT levels in the normal antenatal women and women with preeclampsia in the antepartum period.
2. To correlate maternal and perinatal outcomes with serum LDH and GGT levels in the normal antenatal women and women with preeclampsia.

MATERIALS AND METHODS

Study Design: Case-Control Study

Study setting & study duration:

This study was conducted in a tertiary care center in South India during the period from December 2023 to January 2024, after getting approval from the Institutional Research Ethics Committee, protocol id 20232818 on 10.11.2023.

Sample size and sampling technique:

Based on the values retrieved from a study by Lo JO et al1(Lo et al., 2013), using LDH levels in cases and controls, mean \pm SD: 13.8 \pm 6.84 and mean \pm SD: 26.9 \pm 56.2, sample size was estimated in open epi software version 3 with 80% power and for 95% confidence level the sample size was calculated to be 147cases and 147 controls.

Study population:

Cases:

Antenatal women diagnosed with Preeclampsia 12 (Cunningham et al., 2022)

1. Systolic Blood Pressure (SBP) \geq 140 mmHg or Diastolic Blood Pressure (DBP) \geq 90 mmHg on two occasions \geq 4 hours apart, with proteinuria (dipstick reading of 1+).
2. In the absence of proteinuria, when hypertension is accompanied by any severe features such as thrombocytopenia, elevated liver enzymes, renal insufficiency, pulmonary edema, and new-onset cerebral or visual symptoms

Inclusion Criteria

No history of hypertension before 20 weeks gestation (Blood Pressure <120/80 mmHg during early pregnancy) and no prior diagnosis of chronic hypertension (SBP >140 mmHg or DBP >90 mmHg before pregnancy)

Controls:

Antenatal women with normal blood pressure (< 120/80mmHg) during the third trimester.12 (Cunningham et al., 2022)

Inclusion Criteria (cases & controls)

Singleton pregnancy in the age between 20-30 years during third trimester of pregnancy (>28 weeks of gestation)

Exclusion Criteria (cases & controls)

Antenatal women with diabetes, renal failure, hemolytic anemias, chronic hypertension, gestational diabetes, multiple pregnancy, smoking and alcoholism, liver disease, hepatotoxic drugs, stroke, coronary artery disease, chronic lung diseases, connective tissue disorders, disseminated intravascular coagulation and seizures were excluded

Cases and controls were age-matched. Written informed consent was obtained from all the patients prior to the study

Study tool: A semi-structured questionnaire was used to collect the socio-demographic variables like age, gestational age, marital status, employment status, residence, educational status, parity, personal and family history of preeclampsia and the following clinical and biochemical parameters were collected.

Clinical and Biochemical Parameters collected

Recumbent Blood Pressure and Pulse were recorded for each patient. 5ml of venous blood was collected from each patient in both groups after fasting for 8 - 12 hours. LDH, GGT were performed in the serum immediately after separation. Serum LDH is measured by fully automated analyser EM 640 using enzymatic method based on the recommendations of DGKCH (Deutsche Gesellschaft für Klinische Chemie, or German Society for Clinical Chemistry). The reference interval for LDH is 125-220U/L13 (Pagani et al ., 2003) . Serum GGT is measured by semiautomated analyser using carboxy substrate method. The reference interval for GGT is <40U/L14 (Ceriotti et al ., 2010). Urine protein is analysed using dipstick test.

Method of statistical analysis

The statistical analysis was done using MS Excel and IBM Package Version 26. Categorical variables like demographic details and clinical profile (symptoms & signs) of the participants were expressed in frequency and percentages. Continuous variables like lab investigation parameters (LDH, GGT) were presented as the mean ± SD. Association between categorical variables like demographic characteristics, clinical profile and prognosis were tested using chi square test. Independent Sample t test was used to test the association between continuous variables among preeclamptic antenatal women and normal antenatal women. For all statistical analysis, p-value < 0.05 was assumed as statistically significant

Data availability statement: We declare our willingness to share the data upon request.

RESULTS

The present study was done on 147 preeclamptic pregnant women (cases) and 147 normotensive pregnant women (controls). All subjects were in their third trimester and in the age group 20-30 years. The demographic and clinical profile of the cases and controls were given in Table 1. The mean age of the cases was 25.19 ± 4.19 years, while the controls had a mean age of 25.59 ± 4.23 years. This difference is not statistically significant

Table 1: Demographic and clinical profile of the cases and controls

Variable	Controls (n=147)	Cases (n=147)	Statistical test applied	Test statistic (value)	p-value
Age (years) (Mean ± SD)	25.59 ± 4.23	25.19 ± 4.19	Independent t-test	t=0.79,	0.43
Gravidity	Primi: 48 Multi: 99	Primi: 32 Multi: 115	Chi-square test	χ ² =6.42,	0.011
Systolic BP (mmHg) (Mean ± SD)	103.47 ± 5.47	145.21 ± 9.50	Independent t-test	t=40.1	<0.001
Diastolic BP (mmHg) (Mean ± SD)	72.05 ± 3.03	93.45 ± 7.01	Independent t-test	t=31.3	<0.001

There was no significant difference in the mean age between cases and controls (t = 0.79, p = 0.43), indicating that the two groups were comparable with respect to age. A significantly higher proportion of multigravida women were observed among cases compared to controls (χ² = 6.42, p = 0.011), indicating that gravidity was unevenly distributed between the

two groups. The mean systolic blood pressure was significantly higher in cases than in controls ($t = 40.1$, $p < 0.001$), demonstrating a strong association between systolic hypertension and case status. The mean diastolic blood pressure was also significantly higher in cases compared to controls ($t = 31.3$, $p < 0.001$), further indicating that elevated blood pressure is strongly associated with the case group.

Table 2: Comparison of LDH and GGT value among controls and cases

Variable	Controls (n = 147)	Cases (n = 147)	Statistical test applied	Test statistic (t value)	p-value
Serum LDH (U/L)					
Mean \pm SD	259.84 \pm 45.72	498.91 \pm 147.12	Independent t-test	t = 19.01	< 0.001
Range	147–523	237–974	—	—	—
Serum GGT (U/L)					
Mean \pm SD	10.95 \pm 6.69	31.17 \pm 48.88	Independent t-test	t = 5.02	< 0.001
Range	1–37	2–56.4	—	—	—

The mean \pm SD serum LDH value was 498.91 \pm 147.12 among cases while it was 259.84 \pm 45.72 among controls. This difference was found to be statistically significant ($p < 0.001$). The mean \pm SD serum GGT value was 31.17 \pm 48.88 among cases while it was 10.95 \pm 6.698 among controls. This difference was found to be statistically significant ($p < 0.001$).

Table 3: Pregnancy outcome according to the level of LDH in cases and controls

Outcome	Cases LDH <600 U/L (N=116)	Cases LDH >600 U/L (N=31)	Statistical Test (Cases)	p-value	Controls LDH <600 U/L (N=147)	Controls LDH >600 U/L (N=0)
Perinatal death, n (%)	3 (2.6%)	7 (22.6%)	Fisher's exact test	<0.001	1 (0.7%)	-
Birth weight (g), Mean \pm SD	2217 \pm 404	1836 \pm 343	Independent t-test (t=4.93)	<0.001	2680 \pm 444	-
Mode of delivery						
Normal Vaginal Delivery, N(%)	16 (14%)	0 (0%)			126 (85.7%)	-
Caesarean, N(%)	100 (86%)	31 (100%)	Fisher's exact test	0.03	21 (14.3%)	-

Controls had no participants with LDH >600 U/L, so comparisons within the control group cannot be made. Fisher's exact test was used for categorical variables due to small and zero cell counts. Independent t-test was used for continuous variables (birth weight).

In the present study, none of the control participants had LDH levels >600 U/L, whereas 21% of cases (31/147) were in this category. This difference was statistically significant (Fisher's exact test, $p < 0.001$), demonstrating that elevated LDH values were predominantly associated with the diseased group. Among the cases, perinatal death occurred more frequently in the LDH >600 U/L group (7/31; 22.6%) compared to cases with LDH <600 U/L group (3/116; 2.6%). This difference was statistically significant (Fisher's exact test, $p < 0.001$). In controls, perinatal death occurred only in the LDH <600 U/L group (1/147), and none had LDH >600 U/L. Thus, no statistical comparison was required for controls.

Birth weight was significantly lower among cases with LDH >600 U/L (1836 \pm 343 g) compared to cases with LDH <600 U/L (2217 \pm 404 g). This difference was statistically significant (Independent t-test, $t = 4.93$, $p < 0.001$). In controls, all participants were in the LDH <600 U/L group; therefore, comparative statistics were not applicable.

Among the cases, the rate of Caesarean section was significantly higher in women with LDH >600 U/L (31/31; 100%) compared to cases with LDH <600 U/L (100/116; 86%). This difference was statistically significant (Fisher's exact test, $p = 0.03$). In the control group, 85.7% had normal vaginal delivery and 14.3% underwent Caesarean section. Since no control participant had LDH >600 U/L, statistical comparison was not possible.

Overall, the results indicate that LDH levels above 600 U/L are strongly associated with poor maternal and perinatal outcomes, including higher perinatal mortality, lower birth weight, and a higher requirement for Caesarean delivery. The complete absence of elevated LDH in the control group further strengthens potential role of LDH as a biomarker of disease severity in pregnancy related complications.

Table 4: Pregnancy outcome according to the level of serum GGT in cases and Controls

Outcome	Cases GGT <40 IU/L (N=124)	Cases GGT >40 IU/L (N=23)	Statistical Test (Cases)	p-value	Controls GGT <40 IU/L (N=147)	Controls GGT >40 IU/L (N=0)
Perinatal death, n (%)	2 (1.36%)	8 (5.4%)	Fisher's exact test	<0.001	1 (0.68%)	-
Birth weight (g), Mean ± SD	2197 ± 384	1711 ± 478	Independent t-test	<0.001	2680 ± 444	-
Mode of delivery						
Normal Vaginal Delivery N(%)	17 (14%)	0			126 (85.7%)	-
Caesarean section, N(%)	107 (88%)	23 (100%)	Fisher's exact test	0.04	21 (14.3%)	-

No control participant had GGT >40 IU/L, so group comparisons within controls are not statistically feasible. Fisher's exact test used for categorical variables due to small/zero cell counts. Independent t-test used for continuous variable (birth weight).

Serum GGT levels were found to be significantly associated with adverse pregnancy outcomes. Among the cases, women with GGT >40 IU/L had a substantially higher incidence of perinatal death (5.4%) compared to cases with GGT <40 IU/L (1.36%). This difference was statistically significant (Fisher's exact test, $p < 0.001$), suggesting that elevated GGT levels are associated with increased fetal risk.

Birth weight was also significantly lower among cases with elevated GGT (1711 ± 478 g) compared to cases with normal GGT values (2197 ± 384 g). This difference was statistically significant (Independent t-test, $p < 0.001$), indicating poor fetal growth outcomes in the high GGT group.

Mode of delivery differed markedly between the two groups. All women with GGT >40 IU/L underwent Caesarean section (100%), compared with 88% in the GGT <40 IU/L group. This association was statistically significant (Fisher's exact test, $p = 0.04$), reflecting the increased need for obstetric intervention in women with elevated GGT levels.

In contrast, among controls, all participants had GGT levels <40 IU/L, and none had elevated GGT. Perinatal death and delivery outcomes in the control group followed normal patterns, with higher birth weights and a predominance of normal vaginal deliveries. Since no control participant had high GGT values, statistical comparisons within the control group were not possible.

Overall, these findings suggest that elevated serum GGT (>40 IU/L) is strongly associated with adverse pregnancy outcomes such as higher perinatal mortality, lower birth weight, and increased Caesarean delivery among cases.

DISCUSSION

Preeclampsia is a hypertensive disorder of pregnancy with multisystem complications. This study was done in a tertiary care centre. 294 pregnant women were included in the study. The cases (n=147) and controls (n=147) were age matched. In our study mean serum LDH level is higher (498.91±147.125) in preeclamptic women compared to normal pregnant women (259.84 ± 45.725) and it was statistically significant. This finding was in agreement with the study conducted by Qublan HS et al 15 (Qublan HS et al., 2005) Saleem FR et al 16 (Saleem FR et al., 2020) and Reddy Eleti M et al 17 (Reddy Eleti M et al., 2023)

Mean GGT level was significantly higher (31.17± 48.886) ($p < 0.001$) in preeclamptic women than that of healthy pregnant women (10.95±6.698). Our study is also comparable to studies reported by Zhang Y et al 18 (Zhang, Y et al., 2022), Chen Y et al 11 (Chen Y et al., 2021) and Wu J et al 19 (Wu J et al., 2017)

Our study observed a trend towards lower birth weights in babies born to mothers with higher LDH and GGT levels. This finding suggests a potential link between elevated LDH, GGT and increased risk of preterm delivery, which aligns with the

work of Jaiswar SP et al20 (Jaiswar et al ., 2011) and Lu Y Yang et al21 (Lu Y Yang et al .,2025). There was a significant difference in the mode of delivery between the preeclampsia group and the control group. Pre-eclamptic pregnant women with LDH <600, (N=116) 86% delivered by LSCS while 14% by normal vaginal delivery. Pre- eclamptic pregnant women with LDH >600, (N=31) 100% delivered by LSCS(Lower Segment Caesarean Section).

Pre-eclamptic pregnant women with GGT<40, (N=124) 88% delivered by LSCS while 12% by normal vaginal delivery. Pre-eclamptic pregnant women with GGT >40, (N=23) 100% delivered by LSCS.

In the present study, we observed a significant rise in the LDH and GGT levels with increasing severity of the disease (P<0.001–statistically significant). Mean LDH level in control group is (259.84± 45.725) and mean LDH level in preeclampsia is (498.91 ± 147.125). This finding is comparable to study conducted by Moharana JJ et al22(Moharana et al., 2023). Mean GGT level in control group is (10.95 ± 6.698) and mean GGT level in preeclampsia is (31.17±48.88).

When LDH levels were <600 IU/L (116 cases), 3 early neonatal deaths were reported and there were no still births. In women with LDH levels >600 IU/L (31 cases), 7 early neonatal deaths were reported. When GGT levels were <40 IU/L (124 cases), 2 early neonatal deaths were reported. In women with GGT levels >40 IU/L (23 cases), 8 early neonatal deaths were reported. In our study maternal complications such as Eclampsia, abruptio placenta, Intracranial hemorrhage and HELLP Syndrome were not reported.

CONCLUSION

The mean systolic BP, diastolic BP, mean serum LDH and GGT values were elevated among cases compared to controls and this difference was found to be statistically significant. Elevated levels of LDH and GGT were associated with an increased risk of C-section delivery (LSCS), perinatal death and low birth weight babies. Serum LDH and GGT can be considered as important prognostic biomarker in the third trimester because they reflect the severity of the disease, occurrence of complications and fetal outcome. Deciphering the specific isoforms of LDH and gamma-glutamyl transferase (GGT) holds promise for unveiling the precise nature of organ injury in women with severe preeclampsia

Summary

The mean systolic BP (145.21± 9.5) and diastolic BP (93.45± 7.014) were elevated in cases compared to controls. The mean serum LDH and GGT were elevated in cases compared to controls. Elevated serum LDH (498.91± 147.125) and GGT (31.17±48.88) level reflects the severity of preeclampsia and predictive biomarkers to assess maternal and perinatal outcome.

Since elevated LDH and GGT levels are associated with worse perinatal outcome identifying pregnant patients with elevated LDH and GGT levels triggers more frequent monitoring and proactive management to ensure the best possible outcomes for both mother and fetus.

REFERENCES:

1. Lo, J. O., Mission, J. F., & Caughey, A. B. (2013). Hypertensive disease of pregnancy and maternal mortality. *Current Opinion in Obstetrics and Gynecology*, 25(2), 124–132. <https://doi.org/10.1097/GCO.0b013e32835e0ef5>
2. Var, A., Yildirim, Y., & Onur, E. (2003). Endothelial dysfunction in preeclampsia: Increased homocysteine and decreased nitric oxide levels. *Gynecologic and Obstetric Investigation*, 56, 221–224.
3. Friedman, S. A., Schiff, E., Emeis, J. J., Dekker, G. A., & Sibai, B. M. (1995). Biochemical corroboration of endothelial involvement in severe preeclampsia. *American Journal of Obstetrics and Gynecology*, 172(1), 202–203.
4. Lain, K. Y., & Roberts, J. M. (2002). Contemporary concepts of the pathogenesis and management of preeclampsia. *JAMA*, 287, 3183–3186.
5. Murthy, K. O., Dhananjaya, B. S., & Shazi, S. (2019). Serum lactate dehydrogenase levels in preeclampsia and maternal and perinatal outcomes. *Obstetrics and Gynecology Review*, 5(1), 19–25.
6. Kay, H. H., Zhu, S., & Tsoi, S. (2007). Hypoxia and lactate production in trophoblast cells. *Placenta*, 28(8–9), 854–860.
7. Burd, L. I., Jones, M. D., Jr, Simmons, M. A., Makowski, E. L., Meschia, G., & Battaglia, F. C. (1975). Placental production and foetal utilisation of lactate and pyruvate. *Nature*, 254(5502), 710–711.
8. Bournères, P. F., Rocchiccioli, F., Nurjhan, N., & Zeller, J. (1995). Plasma lactate conversion into glucose in fasting infants. *American Journal of Physiology*, 268(4), E652–E659.
9. Markert, C. L., Shaklee, J. B., & Whitt, G. S. (1975). Evolution of a gene: Multiple genes for LDH isozymes. *Science*, 189(4197), 102–114.
10. Whitfield, J. B. (2001). Gamma glutamyl transferase. *Critical Reviews in Clinical Laboratory Sciences*, 38(4),

- 263–355. <https://doi.org/10.1080/20014091084227>
11. Chen, Y., Ou, W., Lin, D., Lin, M., Huang, X., Ni, S., Chen, S., Yong, J., O’Gara, M. C., Tan, X., & Liu, R. (2021). Early pregnancy biomarkers and risk of preeclampsia. *Frontiers in Cardiovascular Medicine*, 8, 756140. <https://doi.org/10.3389/fcvm.2021.756140>
 12. Cunningham, F. G., Leveno, K. J., Bloom, S. L., Spong, C. Y., Dashe, J. S., & Hoffman, B. L. (2022). *Williams obstetrics* (26th ed.). McGraw-Hill Education.
 13. Pagani, F., Bonora, R., & Panteghini, M. (2003). Reference interval for LDH activity. *Clinical Chemistry and Laboratory Medicine*, 41, 970–971.
 14. Ceriotti F, Henny J, Queraltó J, Ziyu S, Özarda Y, Chen B, Boyd JC, Panteghini M; IFCC Committee on Reference Intervals and Decision Limits (C-RIDL); Committee on Reference Systems for Enzymes (C-RSE). Common reference intervals for aspartate aminotransferase (AST), alanine aminotransferase (ALT) and γ -glutamyl transferase (GGT) in serum: results from an IFCC multicenter study. *Clin Chem Lab Med*. 2010 Nov;48(11):1593-601. doi: 10.1515/CCLM.2010.315. Epub 2010 Oct 29. PMID: 21034260.
 15. Qublan, H. S., Ammarin, V., Bataineh, O., et al. (2005). LDH as a marker of adverse pregnancy outcome. *Medical Science Monitor*, 11(8), 393–397.
 16. Saleem, F. R., Chandru, S., & Biswas, M. (2020). LDH isoenzymes in preeclampsia. *Journal of Medical Biochemistry*, 39(3), 392–398. <https://doi.org/10.2478/jomb-2019-0045>
 17. Reddy Eleti, M., Agrawal, M., Dewani, D., & Goyal, N. (2023). Serum LDH levels and fetomaternal outcomes. *Cureus*, 15(4), e37220. <https://doi.org/10.7759/cureus.37220>
 18. Zhang, Y., Sheng, C., Wang, D., et al. (2022). Liver enzyme levels and risk of preeclampsia. *Frontiers in Cardiovascular Medicine*, 9, 963957. <https://doi.org/10.3389/fcvm.2022.963957>
 19. Wu, J., Zhou, W., Li, Q., et al. (2017). GGT and prediction of perinatal outcomes. *Clinica Chimica Acta*, 475, 97–101. <https://doi.org/10.1016/j.cca.2017.09.018>
 20. Jaiswar, S. P., Gupta, A., Sachan, R., et al. (2011). LDH as a marker in preeclampsia-eclampsia. *Journal of Obstetrics and Gynaecology of India*, 61(6), 645–648.
 21. Lu, Y., Yang, L., Li, X., Kuai, D., Tian, W., & Zhang, H. (2025). A prediction model of superimposed preeclampsia in women with chronic hypertension. *Frontiers in Cardiovascular Medicine*, 12, 1641662. <https://doi.org/10.3389/fcvm.2025.1641662>
 22. Moharana, J. J., Mishra, R., & Nayak, A. K. (2023). A study on serum lactate dehydrogenase and uric acid in preeclampsia and eclampsia: Can they predict adverse fetomaternal outcome? *International Journal of Applied and Basic Medical Research*, 13(2), 95–100. https://doi.org/10.4103/ijabmr.ijabmr_626_22