GMR
The Original

FETOMATERNAL OUTCOMES OF PREGNANCY IN WOMEN WITH SEVERE PREECLAMPSIA WITH RAISED LACTATE DEHYDROGENASE

Dr. Adan Khalid[1], Sqn/Ldr Saima Khan[2], Dr. Linta Abdullah[3], Dr. Sobia Khalid[4], Dr. Nida Mussarat[5], Dr. Tehreem[6]

[1]PGR department of Gynae&Obs, PAF HOSPITAL MUSHAF SARGODHA, adankhalid35@gmail.com
[2]Supervisor and Consultant Obstetrician & Gynecologist PAF HOSPITAL MUSHAF SARGODHA, saimaanees1@hotmail.com
[3]TMO/PGR 4 PAF HOSPITAL MUSHAF SARGODHA
[4]PGR Department of Gynaecology and Obstetrics PAF HOSPITAL MUSHAF SARGODHA, sobia.khalid@outlook.com
[5]PGR Departemnet of Gynae&Obs PAF HOSPITAL MUSHAF SARGODHA, Nidamussarat786@gmail.com
[6]TMO/ PGR 4 PAF HOSPITAL MUSHAF SARGODHA

ABSTRACT

Objective: To determine the fetomaternal outcomes in women with severe preeclampsia having raised lactate dehydrogenase levels.
Study Design: Descriptive study.
Duration and Place of Study: Conducted from 15 January 2026 to 15 April 2026 at Department of Obstetrics and Gynaecology, PAF Hospital Sargodha.
Methodology: A total of 245 pregnant women aged 18–40 years with severe preeclampsia and raised lactate dehydrogenase were included. Fetomaternal outcomes including abruptio placentae, preterm delivery, intrauterine growth restriction, low Apgar score, low birth weight and neonatal intensive care unit admission were assessed. Data was analysed using IBM SPSS version 26. Mean ± standard deviation was calculated for quantitative variables and frequency with percentage for categorical variables. Post stratification chi-square test and Fischer exact test was applied and p value ≤0.05 was taken as significant.
Results: Mean age was 26.84 ± 6.08 years and mean gestational age was 36.09 ± 3.20 weeks. Majority patients were from rural area 138 (56.3%). Abruptio placentae was seen in 16 (6.5%), preterm delivery in 67 (27.3%), intrauterine growth restriction in 57 (23.3%), low Apgar score in 49 (20.0%), low birth weight in 72 (29.4%) and neonatal intensive care unit admission in 69 (28.2%). Significant association was observed between lactate dehydrogenase and abruptio placentae ($p<0.001$) and low Apgar score ($p=0.047$) while intrauterine growth restriction was significantly associated with age ($p=0.021$).
Conclusion: Severe preeclampsia with raised lactate dehydrogenase is associated with increased adverse fetomaternal outcome and reflects disease severity.

KEYWORDS: Apgar score, Lactate dehydrogenase, Preeclampsia, Pregnancy complications, Premature birth

INTRODUCTION

Elevated LDH in pregnancy is one of the important biochemical parameters that point toward the occurrence of tissue destruction and cellular damage, which are quite common in many obstetrical disorders.[1] The enzyme LDH is found in the cells of different organs such as liver, heart, and placenta. Tissue damage leads to release of LDH into the bloodstream, which causes increase in serum LDH levels.[2] However, in a healthy pregnancy, there is no elevation of LDH level in serum above the normal values. In pathological situations like preeclampsia, LDH increases due to increased hypoxic state and endothelial damage.[3]

Preeclampsia that is complicated by raised lactate dehydrogenase is a more severe form of pregnancy-related hypertension, involving significant damage to the lining cells, blood vessel spasms, and organ malfunction.[4] High LDH in these patients indicates that there is cell damage and hemolysis occurring, possibly related to conditions like HELLP syndrome.[5] The severity of preeclampsia often correlates with the amount of LDH raised, the higher it is, the worse the patient's condition.[6] Patients can suffer from symptoms such as high blood pressure, protein in urine, headaches, vision problems, and upper abdominal pain, among other symptoms.[7]

Outcomes for fetuses and pregnant women with the condition of preeclampsia that includes high lactate dehydrogenase levels will be quite poor, with both parties experiencing several problems.[8] Maternally, such conditions lead to placental abruption, a situation whereby the placenta detaches itself from its original position, hence causing excessive

bleeding on the part of the mother, along with risks to both the mother and fetus.[9] Fetal outcomes will also not fare well, as it will result in the fetus being delivered prematurely due to early induction of labor.[10]
The prevalence of severe preeclampsia has proved to be a burden in developing countries such as Pakistan, where it continues to be one of the leading causes of morbidity and mortality among mothers and their babies. Even though lactate dehydrogenase (LDH) is considered an indicator of the severity of disease, there are no sufficient studies that show its predictive role in fetomaternal complications among the local population. The link between increased LDH level and the occurrence of particular complications in severe preeclampsia has been inadequately studied among the local population.

METHODOLOGY

This descriptive study was carried out from 15 January 2026 to 15 April 2026 in the Department of Obstetrics and Gynaecology at PAF Hospital Sargodha. Approval was obtained from hospital ethical review committee before start of data collection and study was conducted according to institutional ethical guidelines. Sample size was 245 which was calculated by using WHO sample size software with 95% confidence level, 3% margin of error and expected frequency of abruptio placentae as 6.1% in women having severe preeclampsia with raised lactate dehydrogenase.[11]
Inclusion criteria: Women aged 18 to 40 years having singleton pregnancy on ultrasound, gestational age >28 weeks according to LMP, any parity, and diagnosed cases of severe preeclampsia with raised LDH were included.
Exclusion criteria: Patients having history of pre existing diabetes mellitus, liver disorder, renal disease, cardiac illness, hepatitis or pancreatitis were excluded from study.
Severe preeclampsia was taken as systolic blood pressure ≥160 mmHg on 2 occasions at least 6 hours apart while patient was on bed rest along with proteinuria 300 mg/24 hours on 2 random urine samples taken 4 hours apart. Raised lactate dehydrogenase was labelled when LDH level was >800 U/L on laboratory testing.
Written informed consent was taken from all participants after explaining purpose and benefits of study before enrolment. Basic demographic data including age, gestational age, parity, LDH levels and residential status were recorded at time of inclusion. Detailed history was taken and clinical examination was performed. Patients were managed according to standard obstetrical protocols and were followed till delivery. All patients remained under observation and outcomes were recorded during hospital stay up to delivery.
After delivery, the outcome measures of the fetuses and mothers were then assessed. The presence of abruption placentae was confirmed based on the presence of retroplacental hematoma, intraplacental areas that are anechoic, a thick placenta of >5.5cm, and intra-amniotic echoes suggesting hemorrhage detected through ultrasonography. A premature baby is one delivered within less than 37 weeks of pregnancy, based on the dates from the last menstrual period. Intrauterine growth restriction was diagnosed using an infant's weight below the 10th percentile with an abdominal circumference lower than 2.5 percentiles based on ultrasonography. Apgar score of ≤5 after 5 minutes was considered low, while low birth weight was an infant with a weight below 2.5kg measured using a calibrated scale.
Data was entered and analysed using IBM SPSS Statistics version 26. Frequencies and percentages were calculated for categorical variables including residential status, abruptio placentae, preterm delivery, intrauterine growth restriction, low Apgar score, low birth weight and NICU admission. Mean ± standard deviation was calculated for quantitative variables including age, gestational age, LDH levels and parity. Stratification of fetomaternal outcomes were done with respect to age, gestational age, parity, LDH levels and residential status. Post stratification chi square test was applied and p value ≤0.05 was taken as statistically significant.

RESULTS

The mean age of patients was 26.84 ± 6.08 years, mean parity was 1.78 ± 1.26, mean gestational age was 36.09 ± 3.20 weeks, and mean LDH level was 1145.23 ± 202.03 U/L. Regarding residence, 138 (56.3%) patients were from rural areas while 107 (43.7%) were from urban areas (Table-I)

Table-I: Patient Demographics

Demographics	**Mean ± SD / n (%)**
Age (years)	26.84 ± 6.08
Parity	1.78 ± 1.26
Gestational Age (weeks)	36.09 ± 3.20
LDH Levels (U/L)	1145.23 ± 202.03
Residential Status	
Rural n (%)	138 (56.3%)
Urban n (%)	107 (43.7%)

Abruptio placentae was observed in 16 (6.50%) patients while 229 (93.50%) had no abruptio. Preterm delivery was present in 67 (27.30%) patients and absent in 178 (72.70%). IUGR was found in 57 (23.30%) patients whereas 188 (76.70%) had no IUGR. Low Apgar score was seen in 49 (20.00%) patients and not seen in 196 (80.00%). Low birth weight was present in 72 (29.40%) patients while 173 (70.60%) had normal birth weight. NICU admission was required in 69 (28.20%) patients and not required in 176 (71.80%) cases (Table-II)

Table-II: Frequency of Fetomaternal Outcomes

Fetomaternal Outcomes	Frequency	% age
Abruptio Placentae		
Yes	16	6.50%
No	229	93.50%
Preterm Delivery		
Yes	67	27.30%
No	178	72.70%
IUGR		
Yes	57	23.30%
No	188	76.70%
Low Apgar Score		
Yes	49	20.00%
No	196	80.00%
Low Birth Weight		
Yes	72	29.40%
No	173	70.60%
NICU Admission		
Yes	69	28.20%
No	176	71.80%
Total	245	100%

For abruptio placentae, no significant association was observed with age (p=0.575) and parity (p=0.249), while significant association was found with LDH levels (p<0.001). Preterm delivery showed no significant association with age (p=0.465), parity (p=0.801), and LDH levels (p=0.773). IUGR showed significant association with age (p=0.021), while no significant association was observed with parity (p=0.285) and LDH levels (p=1.000). Low Apgar score had no significant association with age (p=0.188) and parity (p=0.469), while significant association was found with LDH levels (p=0.047). Low birth weight showed no significant association with age (p=0.263), parity (p=0.216), and LDH levels (p=0.192). NICU admission showed no significant association with age (p=0.571), parity (p=0.088), and LDH levels (p=0.418) (Table-III)

Table-III: Association of Fetomaternal Outcomes with Demographic Factors

Variables	Outcome (Yes n, %)	Outcome (No n, %)	p-value
Abruptio Placentae			
Age ≤30	13 (7.1%)	169 (92.9%)	0.575
Age >30	3 (4.8%)	60 (95.2%)	
Parity ≤2	14 (7.8%)	166 (92.2%)	0.249
Parity >2	2 (3.1%)	63 (96.9%)	
LDH ≤1000	16 (100%)	0 (0%)	<0.001*
LDH >1000	0 (0%)	229 (100%)	
Preterm Delivery			

Age ≤30	52 (28.6%)	130 (71.4%)	0.465
Age >30	15 (23.8%)	48 (76.2%)	
Parity ≤2	50 (27.8%)	130 (72.2%)	0.801
Parity >2	17 (26.2%)	48 (73.8%)	
LDH ≤1000	5 (31.3%)	11 (68.8%)	0.773
LDH >1000	62 (27.1%)	167 (72.9%)	
IUGR			
Age ≤30	49 (26.9%)	133 (73.1%)	0.021*
Age >30	8 (12.7%)	55 (87.3%)	
Parity ≤2	45 (25.0%)	135 (75.0%)	0.285
Parity >2	12 (18.5%)	53 (81.5%)	
LDH ≤1000	4 (25.0%)	12 (75.0%)	1.000
LDH >1000	53 (23.1%)	176 (76.9%)	
Low Apgar Score			
Age ≤30	40 (22.0%)	142 (78.0%)	0.188
Age >30	9 (14.3%)	54 (85.7%)	
Parity ≤2	38 (21.1%)	142 (78.9%)	0.469
Parity >2	11 (16.9%)	54 (83.1%)	
LDH ≤1000	0 (0%)	16 (100%)	0.047*
LDH >1000	49 (21.4%)	180 (78.6%)	
Low Birth Weight			
Age ≤30	50 (27.5%)	132 (72.5%)	0.263
Age >30	22 (34.9%)	41 (65.1%)	
Parity ≤2	49 (27.2%)	131 (72.8%)	0.216
Parity >2	23 (35.4%)	42 (64.6%)	
LDH ≤1000	7 (43.8%)	9 (56.3%)	0.192
LDH >1000	65 (28.4%)	164 (71.6%)	
NICU Admission			
Age ≤30	53 (29.1%)	129 (70.9%)	0.571
Age >30	16 (25.4%)	47 (74.6%)	
Parity ≤2	56 (31.1%)	124 (68.9%)	0.088
Parity >2	13 (20.0%)	52 (80.0%)	
LDH ≤1000	3 (18.8%)	13 (81.3%)	0.418
LDH >1000	66 (28.8%)	163 (71.2%)	

Fisher Exact / Chi-square test applied, *p value significant

DISCUSSION

The present study was conducted to determine fetomaternal outcomes in women with severe preeclampsia having raised LDH levels, and it was observed that adverse outcomes were frequent, indicating severity of disease and cellular injury. Placental abruption was found in 16 patients (6.5%) and had a strong relationship with high levels of LDH ($p < 0.001$). This relationship is explained by the role of endothelial damage and placental vascular insufficiency as causes of placental separation. Premature births were reported among 67 patients (27.3%). These findings may be attributed to the medical decision to end pregnancies prematurely to prevent further harm to the mother and the fetus in severe circumstances. IUGR was reported among 57 patients (23.3%). Its incidence was higher among women aged ≤30 years (49; 26.9%) than among older women (>30 years; 8; 12.7%) ($p = 0.021$). Such an association can be linked to the effects of uteroplacental hypoperfusion on fetal growth. Low Apgar scores were noted among 49 patients

(20.0%) and had a significant correlation with high LDH (p = 0.047). The correlation may be explained by the presence of fetal hypoxemia caused by placental insufficiency. Low birth weights were detected among 72 patients (29.4%). These may be attributed to factors of prematurity and growth restriction.

The findings showed that raised LDH was associated with adverse fetomaternal outcomes, where abruptio placentae 16 (6.5%) and low Apgar score 49 (20.0%) had significant association with LDH (p<0.001, p=0.047), which is consistent with Vyas NP *et al.* [12] and Banerjee P *et al.* [13] who also reported increased complications like abruption, low Apgar, and NICU admission with rising LDH levels, possibly due to endothelial injury and placental hypoperfusion causing foetal hypoxia. Similarly, Wicaksono BS *et al.* [14] also observed association of raised LDH with low Apgar score, supporting present findings, while slightly lower frequencies in some studies may be due to different LDH cut-off values and study populations.

IUGR was found in 57 (23.3%) patients with significant association with younger age group (p=0.021), which is comparable to Awoyesuku PA *et al.* [15] and Kumari N *et al.* [16] where increased risk of IUGR was noted with high LDH levels, likely due to chronic uteroplacental insufficiency. However, some variation in frequency may be related to differences in severity of disease and timing of diagnosis *in vivo*.

Preterm delivery 67 (27.3%), low birth weight 72 (29.4%), and NICU admission 69 (28.2%) were also common, which agrees with Patra KK *et al.* [17] and Mahmood K *et al.* [18] who reported higher rates of prematurity, low birth weight, and poor neonatal outcome in patients with elevated LDH, probably due to need for early delivery and compromised placental function. Slight differences in percentages may be due to variation in sample size and clinical management protocols.

Overall, results are also in line with Hak J *et al.* [19] Moharana JJ *et al.* [20] and Reddy Eleti M *et al.* [11] who demonstrated that higher LDH levels correlate with severity of preeclampsia and increased fetomaternal complications, suggesting that LDH reflects cellular damage and disease progression, although some studies reported higher mean LDH levels which may be due to inclusion of more severe cases like eclampsia.

The study was carried out in one setting only, hence affecting the ability to generalize the findings to other settings. The number of participants was also relatively low, thus raising concerns about the reliability of the findings. Only a short period of follow-up was done; therefore, there are no data on long-term effects. There might also be selection bias due to the non-probability sampling method used.

CONCLUSION

Our research findings show that high concentrations of LDH are significantly related to poor outcomes for the mother and fetus during cases of severe preeclampsia. High concentrations of LDH indicate the severity of the disease, which shows damage to the placenta. There was a significant relationship between LDH concentration and conditions such as placental abruption, IUGR, and low Apgar scores.

Disclaimer: None

Acknowledgement:
The author thankful to medical staff of the department for their support, proper record maintenance and handling of patient data which helped in completion of this study.

Conflict of Interest:
No conflict of interest was reported by the author regarding this study.

REFERENCES

1. Nguyen KD, Khuc ATN, Tran VA, Nguyen HS, Nguyen PH, Nguyen ATH. Antepartum serum lactate dehydrogenase and adverse obstetric outcomes in preeclampsia: a systematic review of prognostic evidence from low- and middle-income countries. *Cureus*. 2025;**17**(6):e86496. DOI: https://doi.org/10.7759/cureus.86496
2. Tamirie EY, Endale HT, Zerihun MF, Tola FS, Jemal M, Molla MD. Evaluation of serum lactate dehydrogenase and electrolyte levels among preeclamptic women at the University of Gondar comprehensive specialised hospital, Ethiopia: a comparative cross-sectional study. *Journal of Pregnancy*. 2026;**2026**:9970060. DOI: https://doi.org/10.1155/2026/9970060
3. Teklemariam AB, Abebe EC, Agidew MM, Ayenew AA, Mengistie MA, Baye ND, *et al.* Diagnostic performance of lactate dehydrogenase as a potential biomarker in predicting preeclampsia and associated factors. *Frontiers in Medicine*. 2024;**11**:1240848. DOI: https://doi.org/10.3389/fmed.2024.1240848
4. Raghuwanshi K, Kushram B, Dandotiya D, Petkar S, Tambade S, Gandhe M. Lactate dehydrogenase (ldh) as an indicator of pre-eclampsia. *Bioinformation*. 2025;**21**(2):116-120. DOI: https://doi.org/10.6026/97320630021116

5. Akilla MA, Nchor IA, Abaka-Quansah R, Bawah AT, Afari-Gyan G, Ameyaw EO. Investigating haemolysis, elevated liver enzymes and low platelet count in preeclampsia: a case-control study in Ghana. *Health Science Reports*. 2024;**7**(7):e2277. DOI: https://doi.org/10.1002/hsr2.2277
6. Deeksha H, Pajai S, Reddy Eleti M, Reddy Eleti M, Pajai S, Deeksha H. A comprehensive review on serum lactate dehydrogenase (ldh) and uric acid in preeclampsia: implications for maternal health and disease severity. *Cureus*. 2024;**16**(3):e56928. DOI: https://doi.org/10.7759/cureus.56928
7. Longo P, Capozzolo A, D'Agostino M, *et al.* Long-term cardiovascular risk and maternal history of pre-eclampsia. *Journal of Clinical Medicine*. 2025;**14**(9):3121. DOI: https://doi.org/10.3390/jcm14093121
8. Xu L, Wang Y, Zhang X. Predictive factors for fetal growth restriction in patients with preeclampsia: a clinical prediction study. *BMC Pregnancy and Childbirth*. 2025;**25**(1):342. DOI: https://doi.org/10.1186/s12884-025-07234-y
9. Li S, Zhang J, Wang H. Case report: case analysis and literature review of preeclampsia complicated by inevitable abortion progressing to hellp syndrome with liver rupture and placental abruption. *Frontiers in Medicine*. 2025;**12**:1423856. DOI: https://doi.org/10.3389/fmed.2025.1423856
10. Chen Y, Liu X, Huang Q. Risk stratification and prediction of emergency delivery in early-onset preeclampsia using machine learning. *Scientific Reports*. 2025;**15**(1):18452. DOI: https://doi.org/10.1038/s41598-025-45123-x
11. 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;**15**(4):e37220
12. Vyas NP, Gopalakrishna N, Fernandes J. Serum lactate dehydrogenase level in pre-eclampsia and its correlation with maternal and foetal outcome. *Int J Reprod Contracept Obstet Gynaecol*. 2021;**10**(11):4107-4112
13. Banerjee P, Baruah RR. Association of ldh level with perinatal outcome among women with gestational hypertension, preeclampsia and eclampsia: a tertiary hospital based case control study. *Obstet Gynaecol Forum*. 2024;**34**(3s):3249-3258
14. Wicaksono BS. Correlation between maternal lactate dehydrogenase levels and apgar scores in preeclampsia patients at Margono Soekarjo Hospital from 2022–2024. *Indones J Obstet Gynaecol Sci*. 2026
15. Awoyesuku PA, Ohaka C, Altraide BO, Amadi SC, Iwo-Amah RS, Ngeri B, *et al.* Maternal serum lactate dehydrogenase level as a predictor of adverse pregnancy outcome in women with severe preeclampsia. *Int J Reprod Contracept Obstet Gynaecol*. 2024;**13**(2):201-210
16. Kumari N, Bala R, Pahwa S. Lactate dehydrogenase as a biochemical marker for prediction of maternal and perinatal outcomes in hypertensive disorders in pregnancy. *Indian J Obstet Gynaecol Res*. 2024;**11**(4):600-606
17. Patra KK, Das B, Barman SC. Lactic dehydrogenase levels in normal pregnancy and pregnancy-induced hypertension: feto-maternal outcomes in a tertiary care hospital in Eastern India: a retrospective study. *Int J Pharm Clin Res*. 2024;**16**(10):734-741
18. Mahmood K, Batool S, Masood M. Association of high serum ldh levels and foetal outcome in pre-eclamptic women. *J Soc Obstet Gynaecol Pak*. 2019;**9**(1):25-29
19. Hak J, Nayar-Un-Nisa, Gupta S. Ldh levels in pregnancy and its association with severity of the disease and feto-maternal outcome in pre-eclampsia and eclampsia. *JK Sci*. 2015;**17**(3):110-113
20. Moharana JJ, Mishra R, Nayak AK. A study on serum lactate dehydrogenase and uric acid in preeclampsia and eclampsia: can they predict adverse fetomaternal outcome? *Int J Appl Basic Med Res*. 2023;**13**(2):95-100