Original Article

A Study on Serum Lactate Dehydrogenase and Uric Acid in Preeclampsia and Eclampsia: Can they Predict Adverse Fetomaternal Outcome?

Abstract

Background: Hypertensive disorders of pregnancy affect 3%–5% of all pregnancies, contributing immensely to maternal morbidity and mortality. According to the WHO, the incidence of deaths due to preeclampsia and eclampsia in developing and developed countries is 2.8% and 0.4%, respectively. Lactate dehydrogenase (LDH) and uric acid are good predictors of disease severity. **Aim:** This study aims to determine the fetomaternal outcome in relation to abnormal serum levels of LDH and uric acid. **Materials and Methods:** A cross-sectional study was carried out in 1200 patients with preeclampsia and eclampsia at a tertiary care center over 2 years. Patients were divided into – Group A: patients with normal LDH (≤300 IU/L) and uric acid (<6 mg/dl) (n = 300). Group B: patients with abnormal LDH and uric acid (n = 900), who were further divided into mild and severe preeclampsia and eclampsia. Abnormal serum values were stratified into groups for easier comparison. The results were compared in terms of maternal and perinatal outcomes. **Results:** The incidence of preeclampsia and eclampsia in our study is 3.14% and 1.57%, respectively. Significant changes in LDH and uric acid were associated with increased severity of the disease (LDH – 1116.94 ± 4.78; uric acid – 9.2 ± 2.89). Higher incidence of maternal and fetal complications was seen with severe preeclampsia and eclampsia with LDH >800 IU/L and uric acid >6 mg/dl. **Conclusion:** Standard antenatal follow-up should be carried out for early detection and prevention of preeclampsia, with strict monitoring of serum uric acid level and LDH. This may reduce the maternal and fetal complications due to preeclampsia.

Keywords: *Complications, lactate dehydrogenase, preeclampsia, uric acid*

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Introduction

Hypertensive disorders of pregnancy affect 3%-5% of all pregnancies,[1] forming the deadly triad along with haemorrhage and infection, which contribute substantially to maternal morbidity and mortality.[2,3] According to the National Eclampsia Registry, 2013, the incidence of preeclampsia was 10.3%, while that of eclampsia was 1.9% (antepartum being more than 50%, approximately 13% postpartum). Although maternal deaths have been largely reduced by early diagnosis and management in developed countries, it is still responsible for 19% of annual deaths in developing countries including India.[4] Maternal mortality attributed to eclampsia, alone, is 4%–6%. Perinatal mortality occurs in 20% of cases, of which 50% are stillbirths.

The systemic inflammatory response of preeclampsia results in endothelial dysfunction which leads to increased vascular reactivity, that precedes the onset of symptomatic clinical disease. Loss of endothelial integrity contributes to an imbalance of sodium-volume homeostasis and reversal of cardiovascular changes of normal pregnancy.[5]

Such generalized alterations in the maternal organ systems lead to complications such as eclampsia, placental abruption, disseminated intravascular coagulation (DIC), intracranial hemorrhage, heart failure, hepatic failure, and renal failure, which are usually lethal. Fetal complications include fetal growth restriction, reduced birth weight, spontaneous or iatrogenic premature delivery, hyaline membrane disease, and overall, more admissions to the neonatal intensive care unit (NICU).

Hyperuricemia is one of the earliest (seen as early as 10 weeks of gestation) observations noted in preeclamptic pregnancies, preceding hypertension, and proteinuria.[2] This increased level suggests serious impending damage to kidney

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function.[6] While elevated concentrations of circulating uric acid are not uniformly seen in every woman with preeclampsia, they do appear to identify a subset of preeclamptic women who are at greater risk for maternal and fetal morbidities, thereby suggesting prognostic and also diagnostic significance.[7] It is as important as proteinuria in identifying the risk of renal involvement and fetal compromise.

Lactate dehydrogenase (LDH) is an intracellular enzyme; thus, elevated levels are indicative of cellular dysfunction, hemolysis, and cell death.[8] Hypoxia in preeclampsia increases both glycolysis and LDH activity, resulting in higher lactate production.[5] LDH has five isoforms, of which LDH-A (4) isoenzyme is immunolocalized in the fetal endothelial cells, while LDH-B (4) isoenzyme is predominantly present in syncytiotrophoblasts. Some studies show an increased serum levels of LDH as a predictive factor in the onset of hemolysis, elevated liver enzyme levels, and low platelet levels (HELLP) syndrome, as well as for small for gestational age babies.[8]

Materials and Methods

The study aims to observe the changes in serum LDH and uric acid levels in patients with preeclampsia and eclampsia and to determine the fetomaternal outcome in relation to abnormal serum levels of LDH and uric acid.

The study was carried out after approval from the Institutional Ethical Committee (Letter No: 410/2018). It was conducted over 2 years, with data collection from the patients admitted to the labor room of a Tertiary Hospital in Southern Odisha.

Inclusion criteria

Singleton pregnancies beyond 20 weeks of gestation, with a diagnosis of preeclampsia or eclampsia, on admission.

Exclusion criteria

Patients with chronic hypertension (without superimposed preeclampsia), existing liver or renal disease, alcoholism, thyrotoxicosis, connective tissue disorder, other convulsive disorders, hemolytic disorders, and multiple pregnancy.

A researcher-made questionnaire and laboratory tests were used to collect data. After obtaining informed written consent, the questionnaire was completed by the researcher by interview method. Patients underwent thorough clinical examinations. Blood pressure (BP) was measured with a sphygmomanometer in the right arm sitting position. Korotkoff phase V readings were used for diastolic readings. BP equal to or more than 140/90 mmHg, with 2 such readings 6 h apart along with proteinuria assessed by dipstick method with reading of 1+ or more were classified as preeclampsia. They were further divided into mild and severe preeclampsia based on BP more than 160/110 mmHg, presence of thrombocytopenia (total Platelet count <1 lakh/mm^3), impaired liver function tests (elevated liver enzymes), progressive renal insufficiency (serum creatinine ≥1.1 mg/dl or doubling of baseline), pulmonary edema, new onset cerebral/visual disturbance. Eclampsia was diagnosed in patients with new onset of generalized tonic–clonic seizures (GTCS) beyond 20 weeks of gestation or preeclampsia complicated by such convulsions.

Patients were subjected to routine and specific investigations. The uric acid level was estimated by a commercial kit in the autoanalyzer (uricase method) and LDH estimation through the LDH isoenzyme kit in the autoanalyzer. Normal levels were taken as serum uric acid: 3.1–6.3 mg/dl and serum LDH: 140–280 U/L.

Patients were divided into two groups. Group A: preeclampsia and eclampsia with normal LDH (<300 IU/L) and uric acid (<6 mg/dl) and Group B: Preeclampsia and eclampsia with abnormal LDH and uric acid. Group B was divided further into mild preeclampsia, severe preeclampsia, and eclampsia. Those having serum LDH ≥300 were divided into three subgroups – (a) 300–600 IU/L; (b) 600–800 IU/L; and (c) >800 IU/L. Those having serum uric acid ≥6 mg/dl were divided into two subgroups – (a) 6–8 mg/dl; and (b) >8 mg/dl.

The two groups and subgroups were studied based on age, gravidity, socio-economic status, gestational age, presenting complaints, investigations, and maternal and perinatal outcomes.

Statistical analysis

The data were tabulated into an MS Excel spreadsheet. Statistical analysis was performed by the SPSS program (Released 2017, IBM SPSS Statistics for Windows, version 25.0. Armonk; NY: IBM Corp, United States). Continuous data (age, body mass index, serum LDH, and serum uric acid) was expressed as mean ± standard deviation (SD), and discrete variables (presenting complaints, mode of delivery, and maternal and fetal complications) were expressed as frequencies. Comparison of continuous data was performed using the Independent samples Student's *t*-test (mean ± SD) and proportion test in *R* (frequencies) to find predictive association of variables. Levene's test of equality of variance was applied to compare the mean values between groups. A $P < 0.05$ was considered statistically significant.

Results

Group A had 300 study samples while Group B had 900 study samples. The incidence of preeclampsia and eclampsia in the hospital of study, over the study period was 3.14% and 1.57%, respectively.

The majority of our subjects belonged to the age group of 21–30 years. The mean age of women with mild preeclampsia was 24.7 years, severe preeclampsia was 24.9 years and eclampsia was 23.9 years. The highest incidence was seen among primigravidae, belonging to

lower socioeconomic status (Modified Kuppuswamy Scale) with irregular antenatal check-ups.

About 25% of the study subjects presented with GTCS and were diagnosed with eclampsia. Among the women with preeclampsia, majority (23%) presented with bilateral pedal edema. 5% had pedal edema with features of imminent eclampsia. 8.6% of women were asymptomatic. 39.8% of patients were preterm.

Figure 1 correlates the severity of preeclampsia with levels of LDH. Among those with normal LDH, an average of 92% of patients had mild preeclampsia. However, LDH >800 IU/L was seen in 11.5% of patients with mild preeclampsia as compared to 26% of patients with severe preeclampsia. This was statistically significant ($P < 0.05$).

Figure 2 correlates the level of Serum Uric acid with the severity of preeclampsia. With normal uric acid levels, around 90% were diagnosed with mild preeclampsia. At uric acid >8 mg/dl, only 15.5% had mild preeclampsia as compared to 43% with severe preeclampsia. This was statistically significant ($P = 0.000$).

Table 1 shows an increasing trend in the mean LDH and mean uric acid values with the increase in severity of the disease, with the highest values in pregnancies complicated by eclampsia (1116.94 ± 4.78 and 9.2 ± 2.89, respectively).

Maternal complications were present in 44 cases of Group A and 200 cases of Group B. In Group B, postpartum hemorrhage (PPH) was the biggest complication (39.5%) followed by abruption placenta (20.5%). DIC was observed in 5%. Twenty patients in this group had multiple complications (PPH + abruption + DIC). Death occurred in 6.2% of cases in this group.

When we compared the maternal complications with the LDH and serum uric acid [Table 2] levels, it was observed that complications were more (45.9% and 53.6%, respectively) in patients with LDH >800 IU/L and uric acid >8 mg/dl.

Table 3 correlates fetal outcome with elevations in uric acid and LDH levels, which shows a higher frequency of low birth weight (LBW) babies (8.2%) and birth asphyxia leading to hypoxic-ischemic encephalopathy (HIE) (3.7%) when uric acid >8 mg/dl. NICU admission increased from 8.3% to 12.2% with a deranged uric acid >8 mg/dl. On comparing LDH levels with fetal outcome, the percentage of LBW babies increases as the serum LDH levels rise. The number of NICU admissions, HIE, intra-uterine death, and early neonatal death among the babies is higher in the group with LDH >800 IU/L.

Table 1: Comparison of mean lactate dehydrogenase and uric acid among the study groups

Group	Clinical profile (*n*=number of cases)	Mean LDH (IU/L)	Mean uric acid (mg/dL)
Group A	*n*=300	298.56±2.87	3.7±2.76
Group B	Mild preeclampsia (*n*=300)	678.43±3.65	6.5±2.81
	Severe preeclampsia (*n*=300)	1089.74±2.57	7.8±3.69
	Eclampsia (*n*=300)	1116.94±4.78	9.2±2.89

LDH: Lactate dehydrogenase

Discussion

Preeclampsia is a multi-system disease which, when left untreated, progresses to eclampsia, characterized by GTCS requiring immediate termination of pregnancy. While the degree of hypertension is a clinically important guide for predicting the severity of preeclampsia, certain biochemical markers aid in determining the prognosis and management.

Our study demonstrated an increase in the severity of hypertension with increasing serum uric acid and LDH levels. A solid correlation of systolic BP with serum LDH as well as uric acid was seen in a study by Vechalapu and Raj Kumari[9] whereas a similar study by Singh *et al.* showed a stronger correlation of diastolic BP with the rise in LDH.[10] Mehta *et al.* found increased systolic and diastolic BP with higher serum LDH levels.[11]

The mean LDH and uric acid level in our study showed a rising trend with the increasing severity of the disease.

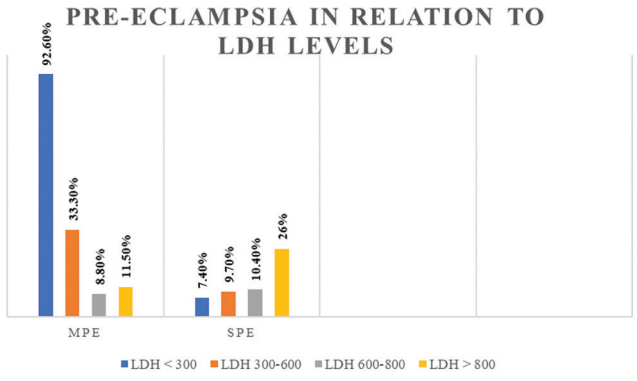

Figure 1: Percentage prevalence of patients having normal and abnormal lactate dehydrogenase levels in correlation with severity of preeclampsia. MPE: Mild preeclampsia, SPE: Severe preeclampsia, LDH: Lactate dehydrogenase

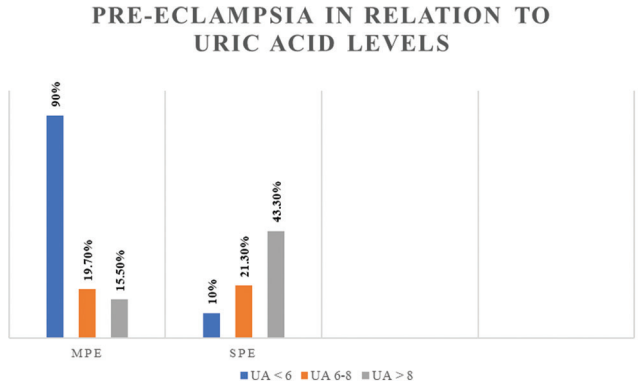

Figure 2: Percentage prevalence of patients having normal and abnormal uric acid levels in correlation with severity of preeclampsia. MPE: Mild preeclampsia, SPE: Severe preeclampsia, UA: Uric acid

Table 2: Correlation of maternal complications with percentage of women having normal and abnormal (including subgroups) values of lactate dehydrogenase and uric acid

Complications (maternal)	Group A		Group B					*P*
	LDH <300 IU/L	UA <6 mg/dL	LDH 300–600	LDH 600–800 IU/L	LDH >800 IU/L	UA 6–8 mg/dL	UA >8 mg/dL	
Present (*n*=244), *n* (%)	44 (18)	44 (18)	20 (8.1)	68 (27.8)	112 (45.9)	69 (28.2)	131 (53.6)	<0.001
Absent (*n*=956), *n* (%)	256 (26.7)	256 (25.6)	348 (36.4)	126 (13.1)	226 (23.6)	301 (31.4)	399 (41.7)	

LDH: Lactate dehydrogenase; UA: Uric acid

Table 3: Correlation of fetal complications with percentage of women having normal and abnormal (including subgroups) values of uric acid and lactate dehydrogenase

Fetal outcome	Group A		Group B					*P*
	UA <6 mg/dL	LDH <300 IU/L	UA 6–8 mg/dL	UA >8 mg/dL	LDH <600 IU/L	LDH 600–800 IU/L	LDH >800 IU/L	
Healthy, *n* (%)	244 (81.3)	246 (82)	292 (32.4)	238 (26.4)	190 (21.1)	20 (2.2)	80 (8.8)	<0.001
LBW, *n* (%)	04 (1.3)	2 (0.6)	22 (2.4)	74 (8.2)	2 (0.2)	12 (1.3)	41 (4.5)	
NICU admission, *n* (%)	25 (8.3)	30 (10)	20 (2.2)	110 (12.2)	84 (9.3)	78 (8.6)	125 (13.8)	
HIE, *n* (%)	5 (1.6)	2 (0.6)	20 (2.2)	34 (3.7)	2 (0.2)	24 (2.6)	29 (3.2)	
IUFD, *n* (%)	10 (3.3)	4 (1.3)	18 (2)	34 (3.7)	26 (2.8)	12 (1.3)	40 (4.4)	
Neonatal death, *n* (%)	12 (4)	10 (3.3)	18 (2)	20 (2.2)	20 (2.2)	12 (1.3)	37 (4.1)	

LDH: Lactate dehydrogenase; UA: Uric acid; LBW: Low birth weight; NICU: Neonatal intensive care unit; HIE: Hypoxic ischemic encephalopathy; IUFD: Intrauterine fetal death

Findings by Renu *et al.*[12] showed around 85% of subjects with eclampsia and 75% of subjects with severe preeclampsia have uric acid >5.7 mg/dl; 91% eclampsia and 34% of subjects with severe preeclampsia have serum LDH >800 U/L. Studies by Saha *et al.*,[13] Singh *et al.*,[10] Mary *et al.*,[14] Sharma and Hariharan[15] confirmed the findings with serum LDH levels. Dhungana *et al.*[16] demonstrated similar results with serum uric acid.

Dave *et al.*,[17] Lincy *et al.*,[18] Gurugunti and Sarah[19] showed a significant increase in serum LDH and uric acid levels in hypertensive women as compared to normotensive pregnant women.

The screening efficacy of LDH levels was evaluated by Kulkarni and Shaikh[20] to predict the progression to preeclampsia and eclampsia. Sensitivity of 83.8% and specificity of 71.4% were seen at a cutoff level of 250 U/L, while at LDH levels of 400 U/L, sensitivity was 58% with 44% specificity.

In our study, maternal complications were present in 44 cases in Group A and 200 cases in Group B (with abnormal uric acid and LDH). Gurugunti and Sarah[19] showed that mean uric acid and LDH were high in mothers with maternal complications. It was observed that complications were more (53.6%) with uric acid >8 mg/dl and 45.9% patients with LDH >800 IU/L. The study by Singh *et al.*[10] showed an increased prevalence of complications such as eclampsia, abruption, HELLP syndrome in patients with LDH >800 IU/L. Sharma and Hariharan[15] also found a correlation of higher serum LDH levels with maternal complications such as abruption (3%), renal failure (11%), HELLP syndrome (10%), PPH (2%), and DIC (2%). Similar findings were observed by Dave *et al.*[17] and Mary *et al.*[14]

Other than perinatal mortality, complications such as retinopathy, acute kidney injury, abruption, DIC, Cerebrovascular accident (CVA), multiple organ dysfunction syndrome, and even shock was seen in cases where the serum LDH level was in excess of 800 IU/L, in a study by Bhati *et al.* (2020).[21] HELLP, psychosis, and jaundice were the added maternal complications seen by Mehta *et al.*[11]

Lincy *et al.*[18] found preeclampsia associated with increased serum uric acid levels were at greater risk for complications and adverse outcomes. When the serum LDH was <600, there were less number of complications as compared to LDH > 600.

Our study reflects higher fetal morbidity, in terms of LBW babies and HIE, in the group with uric acid >8 mg/dl. NICU admissions were more in babies born to mothers with deranged uric acid levels. The percentage of LBW babies increase as the serum LDH levels rise as well as the incidence of perinatal death. It is comparable with the findings of Singh *et al.*[10] in terms of neonatal complications with LDH >800 IU/L, where 44% of babies were LBW babies, 4% were stillborn, with neonatal death of 2%, and NICU admission rate of 16%.A similar conclusion was drawn by Mary *et al.*[14]

In the postnatal period, a study by Omkara Murthy *et al.*[22] showed an increased incidence of Neonatal pathological jaundice and neonatal sepsis among babies born to severe preeclamptic women with serum LDH levels >800 IU/l.

Gurugunti and Sarah[19] also found mean LDH and Uric acid was higher among mothers giving birth to stillborn,

followed by those with intrauterine growth restriction and preterm babies. Maternal hyperuricemia is, thus, a strong predictor of maternal disease progression and fetal outcome.

The strength of our study lies in its sample size, as it has been performed in a tertiary care center. The limitation is the lack of a control group of normotensive patients.

Systematic reviews have mostly focused on predicting the onset of preeclampsia, which also incorporates ultrasound Doppler studies. The role of uric acid in the early detection of preeclampsia needs large-scale studies. As the concentrations may also rise in systemic diseases, it cannot be used as a suitable index for the management of preeclampsia.[23] According to Hall *et al*., higher LDH level before delivery was associated with early onset of severe preeclampsia.[24]

Future research should aim to establish a cut-off value that is associated with increased specificity (and if possible, sensitivity) for developing severe morbidity in these patients. This will help in the evaluation of the optimal interval for termination of pregnancy.

Conclusion

Understanding the pathological process of preeclampsia is important in developing effective strategies for prevention of preeclampsia. A close monitoring of serum uric acid level and LDH during the period of pregnancy may help in the early detection and intervention of gestational hypertension and preeclampsia and thereby preventing disease progression and maternal and fetal complications due to preeclampsia.

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Ethical statement

Institutional Ethics Committee, MKCG Medical College & Hospital, Brahmapur, Ganjam, Odisha, India (Letter No: 410/ 2018).

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Conflicts of interest

There are no conflicts of interest.

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