**Maternal and Fetal Outcomes in Preeclampsia and Lactate Dehydrogenase (LDH) level**

**Abstract**

**Background:** Preeclampsia, a key cause of maternal and perinatal morbidity and mortality universally and remarkably in regions like the Asian subcontinent, imposes efficient biomarkers for risk stratum and outcome forecast. Lactate dehydrogenase, an indicator of cellular injury, has been examined for its prospective association with the seriousness and harmful outcomes of preeclampsia. **Aim:** To find out the association between maternal and fetal outcomes in preeclampsia and LDH level. **Methods:** This cross-sectional study was implied among 68 pregnant women diagnosed with preeclampsia at the Department of Obstetrics and Gynaecology at Bangladesh Medical University (BMU), Dhaka. Data on socio-demographics, maternal and fetal outcomes, and laboratory LDH levels were collected from medical documents. The association between LDH levels and maternal edema, as well as neonatal obstacles, was evaluated using Fisher's Exact Test. A *p*-value <0.05 was indicated the level of significance. Ethical approval was acquired, and informed consent was ensured from all contributors. **Results:** In 68 preeclamptic women, no significant association was noticed between LDH levels and maternal edema (*p*=0.625) or neonatal problems (low birth weight: *p*=0.802; prematurity: *p*=0.396; NICU admission: *p*=0.728). Conversely, a significant association happened between the history of preeclampsia and the existence of edema, proteinuria, and convulsion (*p*<0.001). The study population predominantly covered women aged 20-30 years, living in urban areas, with at minimum secondary education, gestational age ≥25 weeks, and multiparity. High rates of gestational diabetes 79.4%, moderate edema 70.6%, low birth weight newborns 47.1%, and significant proteinuria 63.2% were declared. Higher LDH (>524 U/L) appeared in 29.4% of cases.**Conclusion:**The history of preeclampsia was significantly associated with maternal edema, proteinuria, and convulsion. While the results do not support LDH as a direct predictor of these certain adverse effects in this study, further research with big samples are required to filter its prognostic criteria in preeclampsia.

**Keywords:** Preeclampsia, LDH level, Maternal outcome, Fetal outcome, History of preeclampsia.

**Introduction:**

Preeclampsia is a significant hypertensive condition of pregnancy, presentssignificanthazardstogether mothers and their fetuses [1]. Universally, it continuesto be a top cause of maternal and perinatal morbidity and mortality [2]. In the South Asian region, containing Bangladesh and India, the problem of preeclampsia is remarkablyextreme, causalnotably to bad pregnancy outcomes [3]. High levels of serum LDH, an intracellular enzyme, have been accused as a potential marker of cellular damage and disease severity in preeclampsia [4].

Several studies have explored the association between LDH levels and the clinical manifestations of preeclampsia. A study in Bangladesh found elevated LDH levels to be associated with increased disease severity in preeclamptic women [5]. Similarly, research from India has indicated that higher LDH levels correlate with adverse maternal outcomes such as eclampsia and HELLP syndrome [6, 7]. Globally, systematic reviews and meta-analyses have suggested a link between elevated LDH and an increased risk of adverse maternal and fetal outcomes in preeclampsia, incorporating preterm birth, low birth weight, and fetal distress [8, 9].

Latest research remains to examine the role of LDH as a predictive biomarker in preeclampsia. A prospective study in a tertiary care center in Bangladesh seen a significant correlation between LDH levels and the severity of preeclampsia, advocating its prospectiveefficiency in risk stratification [10]. Moreover, studies in the Indian subcontinent have discovered the correlation between LDH and definite maternal difficulties like acute kidney injury and placental abruption in preeclampsia [11, 12]. Global, investigations are continuing to determine the optimal LDH cutoff estimates for predicting adverse outcomes and to understand the underlying pathophysiological mechanisms linking LDH elevation to disease progression in preeclampsia [13, 14].

Despite the growing body of evidence, the precise role of LDH in predicting the spectrum of maternal and fetal outcomes in preeclampsia remains an area of active research. Variations in study populations, methodologies, and LDH assays contribute to the heterogeneity in findings [15, 16]. Therefore, further investigation, particularly within specific regional contexts like Bangladesh, is warranted to clarify the association between LDH levels and the diverse range of maternal and fetal outcomes in preeclampsia [17]. Understanding this relationship better could aid in the early identification of high-risk pregnancies and the implementation of timely interventions to improve maternal and perinatal health [18, 19]. This study aims to contribute to this understanding by examining the association between LDH levels and various maternal and fetal outcomes in a cohort of preeclamptic women in Bangladesh [20, 21, 22].

**Methodology:**

This cross-sectional study was conducted at BMU, Dhaka, from June 1st, 2022, to May 31st, 2024, involving 68 pregnant women diagnosed with preeclampsia. Participants were selected based on the following inclusion criteria: clinical diagnosis of preeclampsia, gestational age between 16-25+ weeks, singleton pregnancy, and age between 20-30+ years. Women with essential hypertension, diabetes mellitus, thyroid disorder, connective tissue disorder, epilepsy, hepatic disease, chronic kidney disease, history of stroke, primary history of coronary artery disease, chronic infections, or those who refused to enroll were excluded. Data encompassing socio-demographics, maternal medical conditions (gestational age, parity, delivery mode, gestational diabetes, edema, convulsions), fetal outcomes (birth weight, Apgar scores, gestational age at birth, neonatal complications), and laboratory findings (LDH, proteinuria, uric acid, serum creatinine), with the highest LDH level near delivery recorded, were collected using a structured questionnaire and medical record review. Statistical analysis, employing descriptive statistics and Fisher's Exact Test to assess associations between LDH and categorical maternal/fetal outcomes, was performed using SPSS, with a significance level of *p*< 0.05. The study adhered to the Declaration of Helsinki, received IRB approval and ethical clearance from the authority of BMU, and ensured participant anonymity and confidentiality through written informed consent.

**Results:**

A cross-sectional study was conducted at the Department of Obstetrics and Gynecology, BMU, Dhaka, among 68 pregnant women diagnosed with preeclampsia to find out the association between maternal and fetal outcomes in preeclampsia and LDH level.

**Table 1:** Distribution of the respondents by socio-demographic factors (n=68)

|  |  |  |
| --- | --- | --- |
| **Age category** | **Frequency** | **Percent** |
| 20-30 | 41 | 60.3 |
| 30+ | 27 | 39.7 |
| Mean±SD | 28.09±5.924 | |
| **Residence** | | |
| Urban | 41 | 60.3 |
| Rural | 27 | 39.7 |
| **Educational level** | | |
| No formal education | 06 | 8.8 |
| Primary | 09 | 13.2 |
| Secondary | 21 | 30.9 |
| Higher Secondary | 22 | 32.4 |
| Graduate and above | 10 | 14.7 |
| **Occupation** | | |
| Housewife | 11 | 16.2 |
| Service holder | 22 | 32.4 |
| Businesswoman | 10 | 14.7 |
| Other | 25 | 36.8 |
| **Monthly family income (BDT)** | | |
| <30,000 | 8 | 11.8 |
| 30,000–50,000 | 45 | 66.2 |
| >50,000 | 15 | 22.1 |
| **Total** | **68** | **100.0** |

Table 1 shows majority of the respondents (60.3%) were aged 20-30 years with a mean age of 28.09±5.92 years. Among them 60.3% resided in urban areas and 78% had at least secondary level of education. Service holders 32.4% and other occupations 36.8% made up the largest employment groups. Most families, 66.2% reported monthly incomes between BDT 30,000-50,000.

**Table 2:** Distribution of the respondents by maternal medical conditions (n=68)

|  |  |  |
| --- | --- | --- |
| **Variables** | **Frequency** | **Percent** |
| **Gestational age** | | |
| 16-24 | 23 | 33.8 |
| 25+ | 45 | 66.2 |
| **Mode of delivery** | | |
| Normal vaginal delivery | 19 | 27.9 |
| Elective cesarean section | 35 | 51.5 |
| Emergency cesarean section | 14 | 20.6 |
| **Parity** | | |
| Primipara | 27 | 39.7 |
| Multipara | 41 | 60.3 |
| **Gestational diabetes** | | |
| Yes | 54 | 79.4 |
| No | 14 | 20.6 |
| **Presence of edema** | | |
| Mild | 10 | 14.7 |
| Moderate | 48 | 70.6 |
| Severe | 10 | 14.7 |
| **Convulsion** | | |
| Yes | 01 | 1.5 |
| No | 67 | 98.5 |
| **Total** | **68** | **100.0** |

Table 2 displays most participants 66.2% had a gestational age of 25 weeks or more and 60.3%. were multiparous. Cesarean delivery 51.5%, particularly elective, was more common than vaginal delivery. A significant proportion of 79.4% had gestational diabetes, and moderate edema was prevalent in 70.6% of cases. Convulsions were rare, only 1.5%.

**Table 3:** Distribution of the respondents by fetal medical conditions (n=68)

|  |  |  |
| --- | --- | --- |
| **Variables** | **Frequency** | **Percent** |
| **Birth weight** | | |
| <2 kg | 32 | 47.1 |
| >2 kg | 36 | 52.9 |
| **Apgar score at 1 minute** | | |
| Good | 32 | 47.1 |
| Poor | 36 | 52.9 |
| **Apgar score at 5 minutes** | | |
| Good | 55 | 80.9 |
| Poor | 13 | 19.1 |
| **Gestational age at birth** | | |
| <37 weeks | 24 | 35.3 |
| >37 weeks | 44 | 64.7 |
| **Neonatal complications** | | |
| Prematurity | 24 | 35.3 |
| Low birth weight | 34 | 50.0 |
| Respiratory distress | 04 | 5.9 |
| NICU admission | 05 | 7.4 |
| Stillbirth | 01 | 1.5 |
| **Total** | **68** | **100.0** |

Table 3 reveals nearly half of the newborns 47.1% had low birth weight <2 kg, and 52.9% had poor Apgar scores at 1 minute, though most improved (80.9%) by 5 minutes had good scores. Preterm birth <37 weeks occurred in 35.3% of cases. Common neonatal complications included low birth weight 50% and prematurity 35.3%.

**Table 4:** Distribution of the respondents by lab investigations (n=68)

|  |  |  |
| --- | --- | --- |
| **Variables** | **Frequency** | **Percent** |
| **Proteinuria** | | |
| Trace | 05 | 7.4 |
| + | 15 | 22.1 |
| ++ | 43 | 63.2 |
| +++ | 05 | 7.4 |
| **LDH level** | | |
| 80-447 | 48 | 70.6 |
| 82-524 | 10 | 14.7 |
| 524+ | 10 | 14.7 |
| **Uric acidlevel** | | |
| 2.4-4.9 | 20 | 29.4 |
| 3.1-6.3 | 24 | 35.3 |
| 6.3+ | 24 | 35.3 |
| **Serum creatinine level** | | |
| 0.4-0.8 | 27 | 39.7 |
| 0.4-0.9 | 04 | 5.9 |
| 0.9+ | 37 | 54.4 |
| **Total** | **68** | **100.0** |

Table 4 displays most participants exhibited significant proteinuria ++ in 63.2% and elevated LDH levels, with 29.4% showing levels above 524 U/L. Elevated uric acid (≥6.3 mg/dL) and serum creatinine (>0.9 mg/dL) were present in 35.3% and 54.4% of cases, respectively.

**Figure 1:**Length of hospital stay

Figure 1 confirms most patients 73.5% had a hospital stay of less than two weeks, while 26.5% required hospitalization for more than two weeks.

**Table 5:** Association between presence of edemaand LDH levels.

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Presence of edema** | **LDH level** | | | ***p*-value** |
| **80-447** | **82-524** | **524+** |
| Mild | 06 | 02 | 02 | .625f |
| Moderate | 34 | 06 | 08 |
| Severe | 08 | 02 | 00 |
| **Total** | **48** | **10** | **10** | **68** |

\*fFisher's Exact Test,

Table 5 shows that there was no significant association between maternal outcome and LDH levels.

(*p*=.625)

**Table 6:** Association between (maternal outcome) presence of edema, proteinuria status, convulsion and history of preeclampsia

|  |  |  |  |
| --- | --- | --- | --- |
| **Presence of edema** | **History of preeclampsia** | | ***p*-value** |
| **Yes** | **No** |
| Mild | 05 | 05 | .000 f |
| Moderate | 10 | 00 |
| Severe | 48 | 00 |
| **Proteinuria test** | | | |
| Trace | 05 | 00 | .000 f |
| + | 10 | 05 |
| ++ | 43 | 00 |
| +++ | 05 | 00 |
| **Convulsion** | | | |
| Yes | 63 | 01 | .000 f |
| No | 00 | 04 |
| **Total** | **63** | **05** | **68** |

\*f -Fisher's Exact Test,

Table 6 shows that there was significant association between presence of edema, proteinuria status, convulsion and history of preeclampsia(*p*=.000).

**Table 7:** Association between (fetal outcomes) Neonatal complications and LDH level

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Neonatal complications** | **LDH level category** | | | ***p*-value** |
| **80-447** | **82-524** | **524+** |
| Prematurity | 18 | 03 | 03 | .802f |
| Low birth weight | 21 | 06 | 07 |
| Respiratory distress | 04 | 00 | 00 |
| NICU admission | 04 | 01 | 00 |
| Stillbirth | 01 | 00 | 00 |
| **Apgar score at 1 minute** | | | | |
| Good | 25 | 04 | 03 | .396f |
| Poor | 23 | 06 | 07 |
| **Apgar score at 5 minutes** | | | | |
| Good | 38 | 08 | 09 | .728f |
| Poor | 10 | 02 | 01 |
| **Total** | **48** | **10** | **10** | **68** |

\*fFisher's Exact Test

Table 7 shows that there was no significant association between Neonatal complications and LDH levels (*p*=.802,.396,.728)

**Discussion:**

This cross-sectional study investigated the association between maternal and fetal outcomes and LDH levels in 68 pregnant women diagnosed with preeclampsia at BMU. The study findings revealed no significant association between maternal edema and LDH levels *p*=0.625, nor between neonatal complications (low birth weight, prematurity, NICU admission) and LDH levels (*p*>0.05 for all comparisons). However, a significant association was observed between the history of preeclampsia and the presence of edema, proteinuria, and convulsion *p*<0.001.

The lack of a significant association between LDH levels and the specific maternal and fetal outcomes examined in the study contrasts with some previous research. Several studies have suggested that elevated LDH levels correlate with increased disease severity and adverse outcomes in preeclampsia [5, 7, 8, 10]. For instance, a study by Rahman et al. (2018) in Bangladesh found LDH to be a marker of disease severity [5]. Similarly, studies from India have indicated a link between higher LDH and adverse maternal and perinatal outcomes [6, 7, 8, 9]. A feasible explanation for the inconsistency in the results could be the moderately small sample size of study, which might have inadequate the statistical power to detect subtle but real associations. Furthermore, the heterogeneity in the explanation of outcomes and the timing of LDH sizes across different studies could contribute to these discrepancies [15, 16].

The significant association originate between a history of preeclampsia and certain maternal complications (edema, proteinuria, and convulsion) aligns with the considerate that prior preeclampsia is a strong risk factor for recurrence and possibly more severe manifestations of the disease in subsequent pregnancies [1]. This focuses the importance of a full obstetric history in measuring the risk outline of preeclamptic women.

The study population predominantly contained of women aged 20-30+ years with a gestational age of ≥25 weeks, which is consistent with the typical appearance of preeclampsia [1]. The high prevalence of gestational diabetes 79.4% in the study is notable and warrants more study into its possible adjusting consequence on the relationship between LDH and preeclampsia outcomes, as hyperglycemia can also inspire cellular stress and LDH levels [26]. The perceived high rates of low-birth-weight newborns 47.1% and preterm birth 35.3% emphasize the significant fetal risks correlated with preeclampsia, regardless of the directly correlation with LDH in our analysis [2].

The observing that most patients had a hospital stay of less than two weeks 73.5% might consider the management protocols at our setting up for the spectrum of preeclampsia severity detected in our cohort. Conversely, 26.5% needing longer hospitalization probably represented more severe cases with possible complications.

Whilst the study did not confirm a direct link between LDH and the specific adverse outcomes examined, LDH remains an extensively studied biomarker in preeclampsia [11, 12, 13, 14, 15]. Its elevation indicates cellular damage, specifically in the liver and erythrocytes, which are identified to be affected in preeclampsia [4]. Forthcoming research with larger, multi-center studies and standardized outcome definitions is desired to further elucidate the role of LDH as a predictive marker for maternal and fetal complications in preeclampsia, specially within the Bangladeshi context [18]. Longitudinal studies assessing LDH trends through the course of the disease might also offereffective insights into its prognostic utility [16].

Though this study did not find a significant association between LDH levels and maternal edema or specific neonatal complications in our cohort of preeclamptic women, it did authorize the recognized link between a history of preeclampsia and certain maternal complications. Supplementary research is needed to clarify the clinical utility of LDH in predicting adverse results in preeclampsia and to investigate the impact of issues like gestational diabetes on this association [20, 21, 22, 23].

**Conclusion:**

This study of 68 preeclamptic women in Bangladesh noticed no significant independent association between maternal LDH levels and edema, or between LDH levels and definite neonatal complications. Nevertheless, a history of preeclampsia was significantly associated with maternal edema, proteinuria, and convulsion. The inadequate sample size and cross-sectional design require larger, longitudinal studies, mainly in the Bangladeshi population, to explain LDH's prognostic value and the influence of factors like gestational diabetes on maternal and fetal outcomes in preeclampsia. Potential research should confirm optimal LDH cutoffs and analyze its dynamic changes all through the disease.

**Declaration of Interest:** The authors affirm that they have no known disagreements of interest that could have persuaded the conduct or writing of this study.

Conflict of Interest: The authors have no conflict of interest.

**COMPETING INTERESTS DISCLAIMER:**

Authors have declared that they have no known competing financial interests OR non-financial interests OR personal relationships that could have appeared to influence the work reported in this paper.

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