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

**Abstract**

**Background:** Preeclampsia, a key cause of maternal and perinatal morbidity and mortality worldwide and extremely in regions like the Asian subcontinent, insists effective biomarkers for threat stratum and outcome prediction. Lactate dehydrogenase, a sign of cellular injury, has been explored for its eventual association with the importance and harmful outcomes of preeclampsia. **Aim:** To evaluate the LDH level and correlate the LDH to the preeclampsia outcomes in mother and fetus. **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 from June 1st, 2022, to May 31st, 2024. Data on socio-demographics, maternal and fetal outcomes, and laboratory LDH levels were collected from medical records. The association between LDH levels and maternal edema, as well as neonatal obstacles, was assessed using Fisher's Exact Test. A *p*-value <0.05 was denoted the level of significance. Ethical approval was acquired, and informed consent was confirmed by all contributors. **Results:** In 68 preeclamptic women, no significant association was noticed between LDH levels and maternal edema (*p*=0.625) or neonatal difficulties (low birth weight: *p*=0.802; prematurity: *p*=0.396; NICU admission: *p*=0.728). A significant association materialized between the history of preeclampsia and the existence of edema, proteinuria, and convulsion (*p*<0.001). The study population predominantly protected 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 specified harmful effects in this study, extra research with big samples are needed to screen its prognostic criteria in preeclampsia.

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

**Introduction:**

Preeclampsia is a significant hypertensive complaint of pregnancy, presents significant dangers together mothers and their fetuses [1]. Completely, it continues to be a top cause of maternal and perinatal morbidity and mortality [2]. In the South Asian zone, having Bangladesh and India, the obstacle of preeclampsia is remarkably extreme, causal especially to bad pregnancy outcomes [3]. High levels of serum LDH, an intracellular enzyme, have been suspect as a potential marker of cellular damage and disease seriousness in preeclampsia [4].

A study in Bangladesh found elevated LDH levels to be associated with improved disease seriousness in preeclamptic women [5]. Also, research from India has denoted that higher LDH levels correlate with harmful maternal outcomes such as eclampsia and HELLP syndrome [6, 7]. Universally, systematic reviews and meta-analyses have hinted a relationship between advanced LDH and an enhanced risk of harmful maternal and fetal outcomes in preeclampsia, encompassing preterm birth, low birth weight, and fetal distress [8, 9].

Preeclampsia is a multisystem hypertensive syndrome of pregnancy that notably causes to maternal and perinatal morbidity and mortality globally. It is exemplified by new-beginning hypertension and proteinuria or proof of end-organ dysfunction after 20 weeks of gestation. Among several biochemical markers examined for initial discovery and harshness assessment of preeclampsia, serum LDH has developed as a bright due to its association with tissue analysis, oxidative stress, and endothelial dysfunction.

LDH is an intracellular enzyme observed in near all body tissues and is published into the bloodstream in cellular damage. In preeclampsia, placental hypoxia and systemic endothelial damage findings in grown LDH levels. Higher LDH shows hemolysis, hepatocellular damage, and renal injury, making it a possible biomarker for illness seriousness. Studies have recommended numerous cutoff ethics for LDH in expecting problems, with values >600 IU/L repeatedly connected with severe illness and opposing outcomes, with eclampsia, HELLP syndrome, placental abruption, and IUGR [10, 11, 12].

At the molecular level, preeclampsia is credited to rise from abnormal placentation indicating oxidative stress and systemic inflammatory reactions. These practices injure vascular endothelium and uphold cell lysis, ensuing in the proclamation of LDH and added intracellular substances into distribution. The raised LDH levels reflect tissue hypoxia, hemolysis, and raised cell turnover, assisting as an implied marker of illness pathophysiology [13, 14].

Current findings from South Asia, involving Bangladesh and India, follow the use of LDH as a prognostic marker. Prospective research in Bangladesh originates a substantial correlation between LDH levels and preeclampsia severity, with advanced levels associated to improved maternal problems such as acute kidney injury, liver dysfunction, and the necessity for premature delivery [10]. Equally, Indian studies informed associations between elevated LDH and placental abruption, preterm birth, and perinatal mortality [11, 12]. A study by Qublan et al. informed that LDH levels >800 IU/L related to poor maternal and fetal results [12].

Altogether, continuing studies aim to enhance the ideal LDH level values and investigate their projecting utility in altered residents [15, 16]. Conversely, deviations in laboratory assays, population appearances, and clinical explanations promote heterogeneity in results.

Regardless of extending evidence, regional findings stay restricted, exclusively in Bangladesh, wherever context-specific data are needed for increasing maternal and fetal care policies. This study seeks to assess the correlation between maternal serum LDH levels and harmful pregnancy outcomes in women with preeclampsia. An improvement knowing of this correlation could improve early danger stratification, accelerate well-timed involvement, and recover maternal-perinatal effects [17-22].

**Methodology:**

This cross-sectional study was conducted at BMU, Dhaka, encompassing 68 pregnant women diagnosed with preeclampsia. Participants were nominated 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 situations (gestational age, parity, delivery mode, gestational diabetes, edema, convulsions), fetal outcomes (birth weight, Apgar scores, gestational age at birth, neonatal complications), and laboratory results (LDH, proteinuria, uric acid, serum creatinine), with the highest LDH level near delivery documented, were collected using a structured questionnaire and medical material review. Diagnostic Standards for Preeclampsia: Preeclampsia was detected based on the respected clinical and laboratory parameters, reliable with proven standards:

Systolic BP ≥ 140 mmHg and/or Diastolic BP ≥ 90 mmHg considered on two divide grounds at least four hours distant after 20 weeks of gestation in an earlier normotensive woman.

Urine dipstick test showing ≥1+ protein; Though not mandatory for elementary diagnosis, yet Platelet count <100,000/mm³. Elevated liver transaminases to twice the usual attention. Serum creatinine >1.1 mg/dL or doubling up of serum creatinine in the lack of other renal illnesses. Cerebral or visual symptoms, Persistent headache, visual disturbances, Generalized edema stated on clinical examination. Women completing the standards were categorized as ensuring preeclampsia and involved in the study if they met the extra inclusion criteria.

Statistical analysis: Data was analyzed using SPSS version 26. using descriptive statistics and Fisher's Exact Test to assess associations between LDH and categorical maternal/fetal outcomes with a significance level of *p*< 0.05. The study adhered to the Declaration of Helsinki, received IRB consent and ethical consent from the authority of BMU, and confirmed participant anonymity and secrecy through recorded informed consent.

**Results:**

A cross-sectional study was led at the Department of Obstetrics and Gynecology, BMU, Dhaka, among 68 pregnant women diagnosed with preeclampsia to discovery the association between maternal and fetal outcomes in preeclampsia and LDH level. The resulting tables display the results by descriptive statistics. In all tables, the "frequency" column describes the number of respondents in all categories, whereas the "percent" column signifies the consistent ratio stated as a percentage. Every table go with an interpretation sum up the key results.

**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%, specifically 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 appeared in 35.3% of cases. Common neonatal problems involved 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 demonstrated significant proteinuria ++ in 63.2% and advanced LDH levels, with 29.4% proving 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, correspondingly.

 **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% necessity 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 explored the association between maternal and fetal outcomes and LDH levels in 68 pregnant women detected with preeclampsia at BMU. The study decisions showed no significant association between maternal edema and LDH levels *p*=0.625, nor between neonatal difficulties (low birth weight, prematurity, NICU admission) and LDH levels (*p*>0.05 for all comparisons). Conversely, a significant association was noticed between the history of preeclampsia and the existence of edema, proteinuria, and convulsion *p*<0.001.

The lack of a significant association between LDH levels and the exact maternal and fetal outcomes considered in the study contrasts with selected previous research. Numerous studies have advocated that elevated LDH levels correlate with enhanced disease seriousness and opposing outcomes in preeclampsia [5, 7, 8, 10]. For example, a study by Rahman et al. (2018) in Bangladesh observed LDH to be a marker of disease seriousness [5]. Equally, studies from India have shown a connection between higher LDH and bad maternal and perinatal outcomes [6, 7, 8, 9]. A realistic justification for the discrepancy in the results could be the reasonably insignificant sample size of study, which might have insufficient statistical power to detect subtle but actual associations. Besides, the heterogeneity in the justification of outcomes and the timing of LDH sizes across different studies could pay to these inconsistencies [15, 16].

At the molecular level, LDH in preeclamptic patients shows cellular injury, oxidative stress, and endothelial dysfunction focal elements in the pathophysiology of preeclampsia. Preeclampsia is illustrated by short trophoblastic invasion of the maternal spiral arteries, indicating to placental hypoxia and the subsequent issue of anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) [4, 14]. These factors antagonize vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), interrupting standard endothelial function and improving vascular permeability. This cascade influences extensive cellular damage and hemolysis, mainly in the liver, kidneys, and erythrocytes. LDH, being a cytoplasmic enzyme issued in cellular lysis, becomes elevated in the maternal circulation because of this systemic damage [11, 12, 14]. Moreover, hypoxic settings in the placenta favor anaerobic glycolysis, advance accelerating LDH fabrication as the enzyme catalyzes the conversion of pyruvate to lactate [13]. So, enhanced LDH levels in preeclampsia not only act as a marker of tissue injury but also indicate the metabolic and inflammatory disorders inherent to the illness.

The significant association between a history of preeclampsia and convinced maternal worries (edema, proteinuria, and convulsion) is associated with the considerate that prior preeclampsia is a robust danger factor for recurrence and perhaps more severe manifestations of the disease in following pregnancies [1]. These applications the importance of a full obstetric history in measuring the risk outline of preeclamptic women.

The study population principally covered of women aged 20-30+ years with a gestational age of ≥25 weeks, which is reliable with the normal appearance of preeclampsia [1]. The high prevalence of gestational diabetes 79.4% in the study is distinguished and permits more study into its likely correcting significance on the relationship between LDH and preeclampsia outcomes, as hyperglycemia can also encourage cellular stress and LDH levels [26]. The noticed high rates of low-birth-weight newborns 47.1% and preterm birth 35.3% highlight the significant fetal dangers related with pre-eclampsia, whatever the complete correlation with LDH in our analysis [2].

The detection that most patients had a hospital stay of less than two weeks 73.5% might consider the managing protocols at our setting up for the spectrum of preeclampsia seriousness identified in our cohort. Equally, 26.5% wanting longer hospitalization possibly represented more dangerous cases with probable difficulties.

LDH stays one of the utmost greatly explored biomarkers in preeclampsia [11-15]. Elevated LDH levels are broadly acknowledged to consider cellular injury, especially implying hepatocytes and erythrocytes organs and techniques normally associated in the pathophysiology of preeclampsia [4].

Some prior reports have instructed a significant correlation between elevated LDH levels and the seriousness of preeclampsia, as well as weak perinatal outcomes. For example, research by Ghimire et al. (2020) and Kanagal et al. (2014) stated that excessive LDH levels were associated with heightened maternal problems (e.g., HELLP syndrome, eclampsia) and neonatal morbidity. Remarkably, our study had a smaller sample size and was conducted at a single center, which may have limited the statistical power to detect significant associations. Furthermore, differences in laboratory methods and levels used to explain elevated LDH stages might have caused the noticed differences.

The strong points of our study include an exact patient population, careful stratification of preeclampsia cases, and accurate data collection techniques. But limitations such as a moderately small sample size, lack of successive LDH size, and limited generalizability due to the single-center design may have affected the results.

Forthcoming research concerning larger, multicenter cohorts and standardized outcome characterizations is essential to clarify the prognostic role of LDH in preeclampsia, notably within the Bangladeshi context [18]. Longitudinal studies judging LDH trends over time may offer effective insights into its utility as a projecting biomarker [16].

However, this study did not discovery a significant association between LDH levels and maternal edema or exact neonatal complications in our cohort of preeclamptic women, it did authorize the standard connection between a history of preeclampsia and certain maternal difficulties. Additional research is required to explain the clinical utility of LDH in expecting adverse findings in preeclampsia and to examine the impact of concerns like gestational diabetes on this association [20, 21, 22, 23].

**Conclusion:**

This study of 68 preeclamptic women in Bangladesh detected no significant independent association between maternal LDH levels and edema, or between LDH levels and stated neonatal complications. Nonetheless, a history of preeclampsia was extensively associated with maternal edema, proteinuria, and convulsion. The insufficient sample size and cross-sectional design needed to larger, longitudinal studies, principally in the Bangladeshi people, to explain LDH's prognostic value and the encourage of issues like gestational diabetes on maternal and fetal outcomes in preeclampsia. Prospective research should confirm optimal LDH cutoffs and analyze its dynamic changes all through the disease.

**Ethical approval and Consent:**

Ethical approval was acquired, and informed consent was confirmed by authors. The study adhered to the Declaration of Helsinki, received IRB consent and ethical consent from the authority of BMU, and confirmed participant anonymity and secrecy through recorded informed consent.

**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.

**Disclaimer (Artificial intelligence)**

Option 1:

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

Option 2:

Author(s) hereby declare that generative AI technologies such as Large Language Models, etc. have been used during the writing or editing of manuscripts. This explanation will include the name, version, model, and source of the generative AI technology and as well as all input prompts provided to the generative AI technology

Details of the AI usage are given below:

1.

2.

3.

**References:**

1. Magee, L. A., Pels, A., Helewa, M., Rey, E., von Dadelszen, P., Canadian Hypertensive Disorders of Pregnancy (HDP) Working Group, & Canadian Hypertensive Disorders of Pregnancy HDP Working Group (2015). The hypertensive disorders of pregnancy (29.3). Best practice & research. Clinical obstetrics &gynaecology, 29(5), 643–657. <https://doi.org/10.1016/j.bpobgyn.2015.04.001>
2. World Health Organization. (2018). WHO recommendations for prevention and treatment of pre-eclampsia and eclampsia. World Health Organization.<https://www.who.int/news-room/fact-sheets/detail/pre-eclampsia>
3. Khan, K. S., Wojdyla, D., Say, L., Gülmezoglu, A. M., & Van Look, P. F. (2006). WHO analysis of causes of maternal death: a systematic review. Lancet (London, England), 367(9516), 1066–1074. [https://doi.org/10.1016/S0140-6736(06)68397-9](https://doi.org/10.1016/S0140-6736%2806%2968397-9)
4. Maynard, S. E., Venkatesha, S., Thadhani, R., &Karumanchi, S. A. (2005). Soluble Fms-like tyrosine kinase 1 and endothelial dysfunction in the pathogenesis of preeclampsia. Pediatric research, 57(5 Pt 2), 1R–7R. <https://doi.org/10.1203/01.PDR.0000159567.85157.B7>
5. Rahman, M. M., Hossain, M. B., Akter, S., Jesmin, S., & Khan, M. N. I. (2018). Serum lactate dehydrogenase as a marker of disease severity in preeclampsia. Bangladesh Journal of Obstetrics and Gynaecology, 33(1), 15-19.DOI: 10.52711/0974-360X.2024.00334
6. Jaiswar, S. P., Gupta, A., Sachan, R., Natu, S. N., & Shaili, M. (2011). Lactic dehydrogenase: a biochemical marker for preeclampsia-eclampsia. Journal of obstetrics and gynaecology of India, 61(6), 645–648. <https://doi.org/10.1007/s13224-011-0093-9>
7. Bhave, N. V., & Shah, P. K. (2017). A correlation of lactate dehydrogenase enzyme levels in pregnancy induced hypertensive disorders with severity of disease, maternal and perinatal outcome. International Journal of Reproduction, Contraception, Obstetrics and Gynecology, 6(10), 4302–4308. <https://doi.org/10.18203/2320-1770.ijrcog20174132>
8. Gupta, A., Bhandari, N., Kharb, S., & Chauhan, M. (2019). Lactate dehydrogenase levels in preeclampsia and its correlation with maternal and perinatal outcome. International Journal of Reproduction, Contraception, Obstetrics and Gynecology, 8(4), 1505–1510. <https://doi.org/10.18203/2320-1770.ijrcog20191208>
9. Hemalatha. K .r, Sahaja.kittur, Serum Lactate Dehydrogenase as a prognostic marker in preeclampsia and eclampsia. Indian J Obstet Gynecol Res 2018;5(1):31-36.<https://doi.org/10.18231/2394-2754.2018.0007>
10. 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 FetomaternalOutcome?. International journal of applied & basic medical research, 13(2), 95–100. <https://doi.org/10.4103/ijabmr.ijabmr_626_22>
11. Mehta, M., Parashar, M., & Kumar, R. (2019). Serum lactate dehydrogenase: a prognostic factor in pre-eclampsia. International Journal of Reproduction, Contraception, Obstetrics and Gynecology, 8(7), 2792–2798.<https://doi.org/10.18203/2320-1770.ijrcog20193044>
12. Qublan, H. S., Ammarin, V., Bataineh, O., Al-Shraideh, Z., Tahat, Y., Awamleh, I., Khreisat, B., Nussair, B., & Amarin, Z. O. (2005). Lactic dehydrogenase as a biochemical marker of adverse pregnancy outcome in severe pre-eclampsia. Medical science monitor : international medical journal of experimental and clinical research, 11(8), CR393–CR397.
13. He, S., Bremme, K., Kallner, A., &Blombäck, M. (1995). Increased concentrations of lactate dehydrogenase in pregnancy with preeclampsia: a predictor for the birth of small-for-gestational-age infants. Gynecologic and obstetric investigation, 39(4), 234–238. <https://doi.org/10.1159/000292417>
14. Rana, S., Lemoine, E., Granger, J. P., &Karumanchi, S. A. (2019). Preeclampsia: Pathophysiology, Challenges, and Perspectives. Circulation research, 124(7), 1094–1112. <https://doi.org/10.1161/CIRCRESAHA.118.313276>
15. Teklemariam, A. B., Abebe, E. C., Agidew, M. M., Ayenew, A. A., Mengistie, M. A., Baye, N. D., & Muche, Z. T. (2024). Diagnostic performance of lactate dehydrogenase as a potential biomarker in predicting preeclampsia and associated factors. Frontiers in medicine, 11, 1240848. <https://doi.org/10.3389/fmed.2024.1240848>
16. Tomkiewicz, J., &Darmochwał-Kolarz, D. A. (2024). Biomarkers for Early Prediction and Management of Preeclampsia: A Comprehensive Review. Medical science monitor : international medical journal of experimental and clinical research, 30, e944104.<https://doi.org/10.12659/MSM.944104>
17. Irene, K., Amubuomombe, P.P., Mogeni, R. et al. Maternal and perinatal outcomes in women with eclampsia by mode of delivery at Riley mother baby hospital: a longitudinal case-series study. BMC Pregnancy Childbirth 21, 439 (2021). <https://doi.org/10.1186/s12884-021-03875-6>
18. Thi Huyen Anh, N., Manh Thang, N., & Thanh Huong, T. (2024). Maternal and perinatal outcomes of hypertensive disorders in pregnancy: Insights from the National Hospital of Obstetrics and Gynecology in Vietnam. PloS one, 19(1), e0297302. <https://doi.org/10.1371/journal.pone.0297302>
19. Shah, A., Faundes, A., Machoki, M., Bataglia, V., Amokrane, F., Donner, A., Mugerwa, K., Carroli, G., Fawole, B., Langer, A., Wolomby, J. J., Naravaez, A., Nafiou, I., Kublickas, M., Valladares, E., Velasco, A., Zavaleta, N., Neves, I., & Villar, J. (2008). Methodological considerations in implementing the WHO Global Survey for Monitoring Maternal and Perinatal Health. Bulletin of the World Health Organization, 86(2), 126–131. <https://doi.org/10.2471/blt.06.039842>
20. Steegers, E. A., von Dadelszen, P., Duvekot, J. J., &Pijnenborg, R. (2010). Pre-eclampsia. Lancet (London, England), 376(9741), 631–644. [https://doi.org/10.1016/S0140-6736(10)60279-6](https://doi.org/10.1016/S0140-6736%2810%2960279-6)
21. Duley, L., Henderson-Smart, D. J., Meher, S., & King, J. F. (2007). Antiplatelet agents for preventing pre-eclampsia and its complications. The Cochrane database of systematic reviews, (2), CD004659. <https://doi.org/10.1002/14651858.CD004659.pub2>
22. Sibai B. M. (2003). Diagnosis and management of gestational hypertension and preeclampsia. Obstetrics and gynecology, 102(1), 181–192. [https://doi.org/10.1016/s0029-7844(03)00475-7](https://doi.org/10.1016/s0029-7844%2803%2900475-7)
23. Mustary, F., Chowdhury, T. A., Begum, F., & Mahjabeen, N. (2019). Maternal and perinatal outcome in gestational diabetes mellitus compared to pregestational diabetes mellitus. BIRDEM Medical Journal, 9(2), 127-132.