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Original Article

Association of Serum Alkaline Phosphatase with Preeclampsia among Pregnant Women Attended at a Tertiary Care Hospital of Bangladesh

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Abstract

Background: Preclampsia is a multisystem disorder that can induce damage cardiovascular system, kidney, brain and liver. It is responsible for significant maternal and perinatal morbidity. **Objectives**: The purpose of the present study was to find an association of serum alkaline phosphatase and preeclampsia. **Methodology:** This cross-sectional study was carried out in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Bangladesh from July 2009 to June 2010. In this study, 60 pregnant women with preeclampsia, aged 18-39 years with a gestational period of more than 20th weeks, were included in the study (group B). For comparison age and gestational period-matched 30 normotensive pregnant women (group A) were also studied. Both control and preeclamptic women were selected from Obstetric and Gynae in and out-patient Departments of BSMMU and Dhaka Medical College Hospital. Serum alkaline phosphatase was estimated by quantitative determination method. **Results:** The mean serum ALP level increased from 79.87 ± 12.69 U/L in the control group (Group A) to 87.77 ± 13.63 U/L in women with mild preeclampsia (Group B1), and this difference was statistically significant ($p < 0.05$). A further marked increase was observed in women with severe preeclampsia (Group B2), where the mean serum ALP level reached 104.47 ± 10.12 U/L. The difference between the severe preeclampsia group and the control group was highly significant ($p < 0.001$). Moreover, comparison between mild and severe preeclamptic women showed that serum ALP levels were significantly higher in Group B2 than in Group B1 ($p < 0.001$). These findings indicate a progressive rise in serum ALP with increasing severity of preeclampsia, suggesting that elevated ALP levels may reflect disease progression and could serve as a useful biochemical marker for assessing the severity of preeclampsia. **Conclusion:** The result of this parameter plays an important role in preeclampsia; hence, early detection may help to improve maternal and fetal outcomes. *[Journal of Current and Advance Medical Research, January 2025;12(1):3-9]*

Keywords: Serum alkaline phosphatase, preeclamptic women; pregnant women

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Introduction

Preeclampsia is defined as the triad of hypertension to the extent of 140/90 mm Hg or more with proteinuria and with or without edema occurring after 20th weeks of gestation in previously normotensive and non-proteinuric women[1]. Severe preeclampsia is associated with one or more of elevated blood pressure160/110 mm Hg with proteinuria along with other functional symptoms such as headache, hyperreflexia, oliguria, epigastric or right upper quadrant pain, impaired liver function, and thrombocytopenia[2]. Hepatic damage seen in severe preeclampsia[3].

Preeclampsia is a multisystem disorder of pregnancy characterized by new-onset hypertension (blood pressure ≥140/90 mmHg) after 20 weeks of gestation in a previously normotensive woman, accompanied by proteinuria and, in some cases, edema. It results from abnormal placentation, endothelial dysfunction, and widespread vasospasm, leading to impaired perfusion of multiple maternal organs. Although mild preeclampsia may remain stable with appropriate monitoring, progression to severe disease significantly increases the risk of maternal and fetal morbidity and mortality.

Severe preeclampsia is defined by sustained blood pressure of ≥160/110 mmHg and may be associated with severe proteinuria or evidence of end-organ dysfunction. Clinical manifestations include persistent headache, visual disturbances, hyperreflexia, oliguria, epigastric or right upper quadrant pain, elevated liver enzymes, thrombocytopenia, renal impairment, and pulmonary edema. These findings reflect widespread vascular injury and microangiopathy affecting several organ systems.

Hepatic involvement is a hallmark of severe preeclampsia and ranges from mild elevation of liver enzymes to extensive hepatocellular injury. Reduced hepatic blood flow and fibrin deposition within the hepatic microvasculature contribute to ischemia, periportal necrosis, and subcapsular hemorrhage. Patients frequently present with right upper quadrant or epigastric pain due to stretching of the liver capsule. In advanced cases, hepatic complications may progress to the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count), hepatic hematoma, or, rarely, liver rupture, all of which require urgent recognition and multidisciplinary management to reduce adverse maternal and fetal outcomes.

Preeclampsia is the third pregnancy-related cause of death after hemorrhage and embolism. It is the cause of 790 maternal deaths per 100,000 live births[4]. Cerebral haemorrhage and adult respiratory distress are the common causes of death in preeclampsia[5]. The incidence in primigravidae is about 10.0% and in multigravidae about 5.0% cases[6] It is associated with increased risk of placental abruption, acute renal failure, cerebrovascular and cardiovascular complications, disseminated intravascular coagulation and maternal death[7]. Pathophysiology of preeclampsia is not clearly known.

Maternal endothelial dysfunction mediated by excess placenta-derived soluble VEGF receptor I is an emerging prominent component in disease pathogenesis[8]. Placental synthesis of alkaline phosphatase during pregnancy has a role in division of normal and transformed cells. This enzyme is responsible for active transport of phosphate. Lowering of serum ALP in pregnancy may be an indicator of IUGR. ALP was noticed to be increased in preeclampsia[9]. Liver function tests abnormalities occur in 3% of the pregnancies and preeclampsia is the most frequent cause[10]. Some investigators reported increase serum alkaline phosphatase in preeclampsia is linked with liver injury and hemolysis[11].

Some groups of investigators observed higher serum alkaline phosphatase in preeclamptic women[6, 8,11,12 14, 15, 16]. In Bangladesh, about 16% of maternal deaths are caused by preeclampsia and Eclampsia[13]. Therefore, this study was undertaken to compare serum alkaline phosphatase in normal pregnancy and preeclampsia.

Methodology

Study Design and Setting: This analytical cross-sectional study was conducted in the Department of Physiology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, over a one-year period from July 2009 to June 2010. The study was designed to evaluate serum alkaline phosphatase levels in women with preeclampsia and compare them with those of normotensive pregnant women. Ethical approval for the study was obtained from the Central Ethical Review Committee of Bangabandhu Sheikh Mujib Medical University before the commencement of data collection. All study procedures were performed in accordance with the ethical principles outlined in the Declaration of Helsinki for research involving human participants.

Study Population: A total of 90 pregnant women aged between 18 and 39 years with gestational age greater than 20 weeks were enrolled in the study. Among them, 60 clinically diagnosed cases of preeclampsia constituted the study group (Group B), while 30 age- and gestational age-matched normotensive pregnant women served as the control group (Group A). Preeclampsia was diagnosed by obstetricians based on standard clinical criteria, including blood pressure of ≥140/90 mmHg measured on at least two occasions after 20 weeks of gestation in previously normotensive women, accompanied by proteinuria.

The participants were recruited from both the inpatient and outpatient departments of Obstetrics and Gynaecology at Bangabandhu Sheikh Mujib Medical University and Dhaka Medical College Hospital. A simple random sampling technique was employed to minimize selection bias and to ensure that every eligible participant had an equal chance of inclusion in the study.

Selection Criteria: Pregnant women aged 18–39 years with singleton pregnancies beyond 20 weeks of gestation were considered eligible for participation. The study group included women diagnosed with preeclampsia, whereas healthy normotensive pregnant women with uncomplicated pregnancies were enrolled as controls. Women with conditions that could influence serum alkaline phosphatase levels or interfere with the interpretation of biochemical findings were excluded.

These included pre-existing diabetes mellitus, chronic hypertension, renal disease, cardiovascular disease, chronic liver disease, endocrine disorders, malignancy, hemophilia, hydatidiform mole, and other chronic systemic illnesses. Women with multiple pregnancies or known fetal congenital anomalies were also excluded whenever identified to minimize potential confounding factors.

Data Collection: Eligible participants were approached individually, and the objectives, procedures, potential benefits, and possible risks of the study were explained in detail in a language they could understand. Written informed consent was obtained from each participant before enrollment. A structured and pretested questionnaire was used to collect demographic and clinical information. Data included maternal age, parity, gestational age, socioeconomic status, previous obstetric history, family history of hypertension or preeclampsia, and relevant medical history. Information regarding current pregnancy, medication use, and antenatal care was also recorded whenever applicable.

Clinical Assessment: Each participant underwent a thorough physical and obstetric examination. Height and weight were measured using standardized equipment, and body mass index (BMI) was calculated where appropriate. Blood pressure was measured using a calibrated mercury sphygmomanometer with the participant in a seated position after at least 10 minutes of rest. An appropriately sized cuff was used, and measurements were taken from the right arm supported at heart level.

Systolic blood pressure was determined by the appearance of the first Korotkoff sound (Phase I), whereas diastolic blood pressure was recorded at the disappearance of the Korotkoff sounds (Phase V). Two readings were obtained at an interval of at least five minutes, and the average value was recorded for analysis. Proteinuria was assessed using routine urinary examination performed in the respective hospital laboratories as part of the clinical evaluation.

Blood Sample Collection and Biochemical Analysis: Following clinical assessment, approximately 5 mL of venous blood was collected aseptically from the antecubital vein of each participant using a sterile disposable syringe. Blood samples were transferred into plain tubes and allowed to clot at room temperature before centrifugation at approximately 3000 rpm for 10 minutes to separate serum. The separated serum was stored under appropriate laboratory conditions until biochemical analysis.

Measurement of Serum Alkaline Phosphatase: Serum alkaline phosphatase (ALP) concentration was measured quantitatively in the Department of Physiology laboratory at BSMMU using a standardized enzymatic colorimetric method according to the manufacturer's instructions. Internal quality control procedures were followed throughout the study to ensure the accuracy, precision, and reproducibility of laboratory measurements. All analyses were performed under identical laboratory conditions by trained personnel who adhered to standard operating procedures.

Statistical Analysis: Statistical analyses was performed with SPSS software, versions 22.0 (IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.). Continuous data that were normally distributed were summarized in

terms of the mean, standard deviation, median, minimum, maximum and number of observations. Categorical or discrete data were summarized in terms of frequency counts and percentages. When values are missing, the denominator was stated. Chi-square test was used for comparison of categorical variables. Every effort was made to obtain missing data. A two-sided P value of less than 0.05 was considered to indicate statistical significance. Data were expressed as Mean + SD. Data analysis was done with SPSS version-12. For statistical significance, ANOVA, and Independent-samples t test was used.

Ethical Clearance: All procedures of the present study were carried out in accordance with the principles for human investigations (i.e., Helsinki Declaration) and also with the ethical guidelines of the Institutional Research Ethics Committee. Formal ethics approval was granted. Participants in the study were informed about the procedure and purpose of the study and confidentiality of information provided. All participants consented willingly to be a part of the study during the data collection periods. All data were collected anonymously and analyzed using the coding system.

Results

Table 1 presents the baseline demographic and clinical characteristics of the study participants across three groups (Group A, Group B1, and Group B2), with each group comprising 30 subjects (total *n* = 90). The variables assessed included age, body mass index (BMI), gestational period, systolic blood pressure (SBP), and diastolic blood pressure (DBP). The mean age of the participants was comparable among the three groups. Group A had a mean age of 25.13 ± 4.97 years, while the mean age was 24.63 ± 5.70 years in Group B1 and 25.60 ± 5.70 years in Group B2. The small differences indicate that the study groups were well matched with respect to age.

The mean BMI showed a gradual increase across the groups. Group A had the lowest mean BMI at 23.22 ± 1.88 kg/m², followed by 25.05 ± 2.13 kg/m² in Group B1 and 26.63 ± 2.50 kg/m² in Group B2. Thus, participants in Groups B1 and B2 had higher BMI values than those in the control group, with the highest BMI observed in Group B2.

The mean gestational period was also similar across the study groups, indicating that participants were enrolled at comparable stages of pregnancy. Group A had a mean gestational age of 31.00 ± 4.39 weeks, whereas the mean gestational age was 32.67 ± 0.64 weeks in Group B1 and 32.03 ± 0.45 weeks in Group B2. A marked difference was observed in blood pressure measurements among the groups. The mean systolic blood pressure (SBP) in Group A was 108.17 ± 8.95 mmHg, representing normotensive pregnant women.

In comparison, the mean SBP increased substantially to 144.33 ± 6.79 mmHg in Group B1 and further to 166.67 ± 10.28 mmHg in Group B2, demonstrating progressively higher systolic blood pressure among the hypertensive groups. Similarly, the mean diastolic blood pressure (DBP) was lowest in Group A at 68.67 ± 8.19 mmHg. It increased to 94.50 ± 4.01 mmHg in Group B1 and reached 113.17 ± 5.79 mmHg in Group B2. The progressive rise in both systolic and diastolic blood pressure from Group A to Group B2 reflects increasing severity of hypertension among the study participants.

Overall, the three groups were comparable with respect to age and gestational age, while BMI was moderately higher in the hypertensive groups. The most prominent differences were observed in SBP and DBP, with Group B2 exhibiting the highest blood pressure values, followed by Group B1, whereas Group A maintained blood pressure within the normal range (Table 1).

Table 1: Age, BMI, Gestational Period, SBP and DBP in Different Groups of Subjects (n=90)

Groups	n	Mean Age (years)	BMI (kg/m^2)	Ges. Period (Weeks)	SBP (mm of Hg)	DBP (mm of Hg)
Group A	30	25.13 ± 4.97	23.22 ± 1.88	31 ± 4.39	108.17 ± 8.95	68.67 ± 8.19
Group B_1	30	24.63 ± 5.70	25.05 ± 2.13	32.67 ± 0.64	144.33 ± 6.79	94.50 ± 4.01
Group B_2	30	25.60 ± 5.70	26.63 ± 2.50	32.03 ± 0.45	166.67 ± 10.28	113.17 ± 5.79

Data are expressed as Mean ± SD. For statistical analysis, one-way ANOVA was performed for comparison among the groups and independent sample t test was done for comparison between the groups. Figures in parentheses indicate ranges; SBP=Systolic blood pressure; DBP=Diastolic blood pressure; Group A = Normotensive pregnant women (control); Group B_1 = Mild preeclamptic women; Group B_2 = Severe preeclamptic women

Table 2: Comparison of different groups to calculate P value

Groups	P value				
A vs B_1 vs B_2	0.787^{ns}	0.000^{***}	0.237^{ns}	0.000^{***}	0.000^{***}
A vs B_1	0.680^{ns}	0.001^{***}	0.115^{ns}	0.000^{***}	0.000^{***}
A vs B_2	0.771^{ns}	0.000^{***}	0.295^{ns}	0.000^{***}	0.000^{***}
B_1 vs B_2	0.514^{ns}	0.01^{**}	0.510^{ns}	0.000^{***}	0.000^{***}

Group A = Normotensive pregnant women (control); Group B_1 = Mild preeclamptic women; Group B_2 = Severe preeclamptic women

Table 2 presents the statistical comparison of age, body mass index (BMI), gestational period, systolic blood pressure (SBP), and diastolic blood pressure (DBP) among the three study groups using one-way ANOVA and independent sample *t*-tests. When all three groups (Group A, Group B1, and Group B2) were compared simultaneously, age did not differ significantly among the groups ($p = 0.787$), indicating comparable age distribution. In contrast, BMI differed highly significantly among the groups ($p < 0.001$), suggesting an increase in BMI with the severity of preeclampsia. Gestational period also showed no significant difference ($p = 0.237$), indicating that participants were enrolled at similar gestational ages. However, both SBP and DBP demonstrated highly significant differences among the groups (both $p < 0.001$), reflecting marked variation in blood pressure according to disease severity. Pairwise comparison between Group A (normotensive pregnant women) and Group B1 (mild preeclamptic women) revealed no significant difference in age ($p = 0.680$) or gestational period ($p = 0.115$). However, BMI was significantly higher in Group B1 than in Group A ($p = 0.001$). Both SBP and DBP were also highly significantly increased in Group B1 compared with the control group (both $p < 0.001$). Comparison between Group A and Group B2 (severe preeclamptic women) similarly showed no significant difference in age ($p = 0.771$) or gestational period ($p = 0.295$). In contrast, BMI was highly significantly higher in Group B2 than in Group A ($p < 0.001$). Moreover, both SBP and DBP were markedly elevated in Group B2 compared with the control group, with highly significant differences for both parameters (both $p < 0.001$). When Group B1 was compared with Group B2, age remained comparable between the two groups ($p = 0.514$), and gestational period also did not differ significantly ($p = 0.510$). BMI, however, was significantly higher in severe preeclamptic women than in mild preeclamptic women ($p = 0.010$). Additionally, both SBP and DBP were highly significantly greater in Group B2 than in Group B1 (both $p < 0.001$), indicating progressively increasing blood pressure with worsening severity of preeclampsia. Overall, the statistical analysis demonstrates that the three groups were well matched with respect to age and gestational period, whereas BMI increased significantly with the severity of preeclampsia. The most pronounced differences were observed for systolic and diastolic blood pressure, both of which increased progressively from normotensive controls to mild preeclampsia and further to severe preeclampsia, with highly significant differences in all relevant comparisons.

Table 3 shows the serum alkaline phosphatase (ALP) levels among the three study groups, each consisting of 30 participants ($n = 90$). The results are presented as mean ± standard deviation (SD), with the corresponding range in parentheses. The normotensive pregnant women (Group A) had the lowest mean serum ALP level, measuring 79.87 ± 12.69 U/L, with values ranging from 60 to 104 U/L. These findings represent the baseline serum ALP levels in healthy pregnant women. Among mild preeclamptic women (Group B1), the mean serum ALP level increased to 87.77 ± 13.63 U/L, with a range of 64 to 114 U/L. Compared with the control group, mild preeclamptic women demonstrated a higher average serum ALP concentration, although there was some overlap in the observed ranges between the two groups. The highest serum ALP levels were observed in severe preeclamptic women (Group B2). The mean serum ALP concentration was 104.47 ± 10.12 U/L, with values ranging from 83 to 120 U/L. This group exhibited an increase of approximately 24.6 U/L compared with the normotensive control group and about 16.7 U/L compared with the mild preeclamptic group. The relatively lower standard deviation in Group B2 indicates less variability in serum ALP values despite the higher mean concentration. Overall, the findings demonstrate a progressive increase in serum alkaline phosphatase levels with increasing severity of preeclampsia. The mean serum ALP was lowest in normotensive pregnant women, moderately elevated in women with mild preeclampsia, and highest in those with severe

preeclampsia, suggesting a positive association between serum ALP levels and the severity of the disease.

Table 3: Serum ALP levels in Different Groups (n =90)

Groups	n	Serum Alkaline Phosphatase (U/L)
Group A	30	79.87± 12.69 (60-104)
Group B_1	30	87.77± 13.63 (64-114)
Group B_2	30	104.47± 10.12 (83-120)

Data are expressed as Mean ± SDFigures in parentheses indicate ranges; Group A = Normotensive pregnant women (control); Group B_1 = Mild preeclamptic women; Group B_2 = Severe preeclamptic women

Table 4 presents the statistical comparison of serum alkaline phosphatase (ALP) levels among the three study groups using one-way ANOVA for overall group comparison and independent sample *t*-tests for pairwise comparisons. The overall comparison among Group A (normotensive pregnant women), Group B1 (mild preeclamptic women), and Group B2 (severe preeclamptic women) demonstrated a highly statistically significant difference in serum ALP levels ($p < 0.001$). This finding indicates that serum ALP levels varied significantly across the three groups and were associated with the severity of preeclampsia. Pairwise comparison between Group A and Group B1 showed a statistically significant difference in serum ALP levels ($p = 0.02$).

As shown in Table 3, the mean serum ALP increased from 79.87 ± 12.69 U/L in normotensive pregnant women to 87.77 ± 13.63 U/L in women with mild preeclampsia, indicating a significant elevation even in the early stage of the disease. Comparison between Group A and Group B2 revealed a highly statistically significant difference ($p < 0.001$). The mean serum ALP level was markedly higher in severe preeclamptic women (104.47 ± 10.12 U/L) than in normotensive controls (79.87 ± 12.69 U/L), demonstrating a substantial increase in serum ALP with severe disease. Similarly, comparison between Group B1 and Group B2 also demonstrated a highly statistically significant difference ($p < 0.001$). The mean serum ALP increased from 87.77 ± 13.63 U/L in mild preeclamptic women to 104.47 ± 10.12 U/L in severe preeclamptic women, indicating that serum ALP levels rise significantly as preeclampsia progresses from mild to severe.

Overall, the statistical analysis demonstrates a progressive and statistically significant increase in serum alkaline phosphatase levels with increasing severity of preeclampsia. While the difference between normotensive and mild preeclamptic women was significant ($p = 0.02$), the comparisons involving severe preeclampsia showed highly significant differences ($p < 0.001$), suggesting that serum ALP may serve as a useful biochemical marker reflecting the severity of preeclampsia.

Table 4: Comparison of Different Groups to Calculate P value

Groups	P value
A vs B_1 vs B_2	0.000^{***}
A vs B_1	0.02^{*}
A vs B_2	0.000^{***}
B_1 vs B_2	0.000^{***}

For statistical analysis, one-way ANOVA was performed for comparison among the groups and independent sample t test was done for comparison between the groups.

Figure I illustrates the mean serum alkaline phosphatase (ALP) levels among the three study groups: Group A (normotensive pregnant women), Group B1 (mild preeclamptic women), and Group B2 (severe preeclamptic women). The figure demonstrates a progressive increase in serum ALP levels with increasing severity of preeclampsia. The lowest mean serum ALP level was observed in Group A, measuring 79.87 U/L. In Group B1, the mean serum ALP increased to 87.77 U/L, representing a statistically significant increase compared with the control group ($p < 0.05$), as indicated by the single asterisk (*).

The highest mean serum ALP level was recorded in Group B2, with a value of 104.47 U/L. This increase was highly statistically significant compared with the other groups ($p < 0.001$), as denoted by the three asterisks (***). Compared with the control group, serum ALP increased by approximately 10.0% in mild preeclampsia and by about 31.0% in severe preeclampsia. Overall, the bar diagram clearly shows a stepwise elevation in serum alkaline phosphatase concentrations from normotensive pregnancy to mild and severe preeclampsia.

These findings suggest a positive association between increasing serum ALP levels and the severity of preeclampsia, supporting the potential role of serum ALP as a biochemical marker for disease severity.

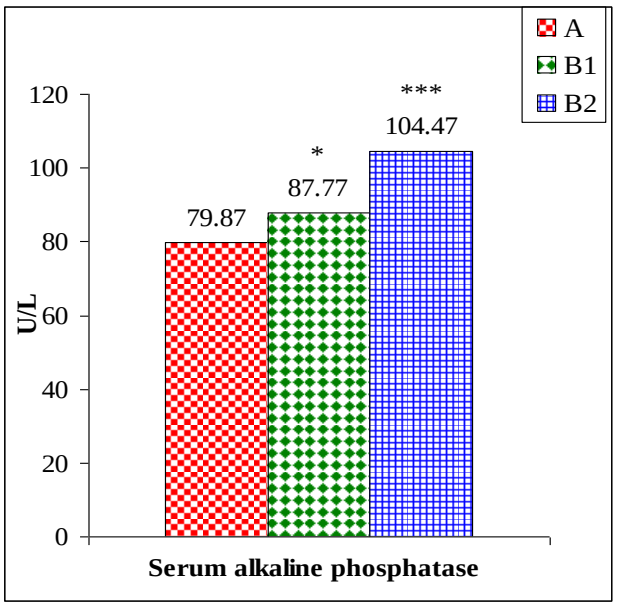

Figure I: Mean serum alkaline phosphatase in different groups (Group A = Normotensive pregnant women; Group B_1 = Mild preeclamptic women; Group B_2 = Severe preeclamptic women)

Discussion

Quality of life for mother and newborn has become our most important concern nowadays. Preeclampsia and eclampsia are associated with poor maternal and fetal outcomes. If left untreated, preeclampsia can progress to a convulsive state known as eclampsia[6]. This study showed increased serum alkaline phosphatase levels in preeclamptic women compared to normotensive pregnant women. Similar observations were reported by other investigators of different countries[12, 14-17, 19-20].

Again, there was a significant ($p<0.001$) difference in serum alkaline phosphatase level between two preeclamptic groups. But no published data was found to compare these findings. Increased level of alkaline phosphatase in PIH is attributed to the increase blood pressure. The increasing hypertension causing increased alkaline phosphatase activity can be explained by ischemia resulting due to maternal hypertension[18].

Mechanism of raised liver enzymes is hypervascularization and vasoconstriction of liver leading to cell injury, alteration of membrane permeability, and damage to hepatocyte[8]. Placental ischemia and endothelial dysfunction, both of which are linked to the pathophysiology of preeclampsia, could also contribute to the elevation of ALP observed in this study[19]. Human placental alkaline phosphatase is synthesized in the placenta during pregnancy. PALP was localized in the outer and inner layers of the syncytiotrophoblast. There is increased phosphatase activity in the outer and inner layers of the syncytiotrophoblast of preeclamptic placenta. Placenta of preeclamptic women on chronic hypertension has maximum intensity of PALP in the outer layers of the syncytiotrophoblasto[20].

Alkaline phosphatase of the placenta appears to be moderately resistant to hypoxia. There is considerable increase in lysosomal activity in hypertensive placenta, presumably a response to placental ischemia which occurs in preeclampsia and which by altering the tissue P^H of trophoblast, stimulates lysosomal activity. This leads to syncytial damage and destruction in preeclampsia, which releases PALP from vesicles into cytoplasm[20]. Information about this in patients with preeclampsia may allow appropriate obstetric planning, including delivery timing.

The present study demonstrated a progressive and statistically significant increase in serum alkaline phosphatase (ALP) levels with increasing severity of preeclampsia. The mean serum ALP level was 79.87 ± 12.69 U/L in normotensive pregnant women (Group A), which increased significantly to 87.77 ± 13.63 U/L in women with mild preeclampsia (Group B1) ($p < 0.05$) and further to 104.47 ± 10.12 U/L in women with severe preeclampsia (Group B2) ($p < 0.001$). The highly significant difference between mild and severe preeclamptic women ($p < 0.001$) suggests that serum ALP levels increase in parallel with the severity of the disease. These findings indicate that serum ALP may be a useful biochemical marker for assessing the progression and severity of preeclampsia.

The findings of the present study are consistent with those of Nargis et al[21] who reported significantly elevated serum ALP levels in women with preeclampsia compared with normotensive pregnant women. Their study also demonstrated that ALP concentrations increased with disease severity, suggesting that placental dysfunction and hepatic involvement contribute to elevated enzyme levels. Similarly, another study[22] observed significantly higher serum ALP levels among women with severe preeclampsia than those with mild disease and healthy controls, supporting the role of ALP as an indicator of disease progression.

Comparable findings were also reported in a study[23] found a significant rise in serum ALP among preeclamptic women, particularly in severe cases.

They proposed that increased placental production of ALP, together with hepatic endothelial injury resulting from preeclampsia, accounts for the elevated enzyme activity. Likewise, another study[23] has demonstrated that serum ALP levels were significantly increased in preeclamptic pregnancies and showed a positive correlation with blood pressure, further emphasizing its association with disease severity.

The biological basis for increased serum ALP in preeclampsia is multifactorial. Pregnancy itself is associated with a physiological increase in ALP due to placental isoenzyme production. However, in preeclampsia, placental ischemia, trophoblastic damage, oxidative stress, and endothelial dysfunction may lead to increased release of placental ALP into the maternal circulation. In addition, hepatic involvement caused by vasospasm and microvascular injury may contribute to elevated liver-derived ALP. Therefore, higher serum ALP levels may reflect both placental dysfunction and maternal organ involvement, particularly in severe preeclampsia.

However, a few studies have reported findings that differ from the present study. In a study[24], it has been observed that higher serum ALP levels in preeclamptic women than in controls, but the differences between mild and severe preeclampsia were not statistically significant. Similarly, another study[17] has reported only modest elevations in serum ALP and suggested that physiological variations during late pregnancy, nutritional status, and differences in placental isoenzyme activity might limit the diagnostic value of total ALP. These discrepancies may be explained by differences in sample size, gestational age, disease severity, laboratory assay methods, ethnicity, and study design.

Overall, the present findings support the growing body of evidence that serum ALP increases progressively with the severity of preeclampsia. Although serum ALP lacks specificity because it originates from multiple tissues, its significant elevation in severe disease suggests that it may serve as an inexpensive, widely available adjunctive biochemical marker for evaluating disease severity when interpreted alongside clinical findings and other laboratory investigations. Further multicenter prospective studies incorporating placental ALP isoenzyme estimation are warranted to better establish its prognostic and diagnostic utility in preeclampsia.

Conclusion

Serum alkaline phosphatase was increased in preeclamptic women compared to normal pregnant women. Hence at this juncture it can be suggested that Serum alkaline phosphatase could be used as predictor of severity of preeclampsia.

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None

Conflict of Interest
The authors have no conflicts of interest to disclose

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Data Availability
Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate
Ethical approval for the study was obtained from the Institutional Review Board. As this was a prospective study the written informed consent was obtained from all study participants. All methods were performed in accordance with the relevant guidelines and regulations.

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