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Comparative Analysis of Serum Iron Levels Between Preeclamptic and Normotensive Pregnant Women: A Case-Control Study

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Abstract

Preeclampsia is a multifaceted, systemic hypertensive disorder of pregnancy, the exact etiopathogenesis of which remains incompletely understood. With a global prevalence of 5–10%, it ranks as the third most frequent cause of pregnancy-related maternal fatalities. The present cross-sectional study was designed to assess the differential serum iron status between preeclamptic patients and their normotensive counterparts. Conducted over 12 months (July 2016–June 2017) at the Department of Biochemistry, Mymensingh Medical College, Bangladesh, the study enrolled 100 pregnant women, evenly divided into a case group (n=50) with preeclampsia and a control group (n=50) with uncomplicated pregnancies. Serum iron levels were measured via a standardized colorimetric method, and statistical evaluation was carried out using SPSS version 21, employing Student's unpaired t-test for intergroup comparisons. The results demonstrated a substantially higher mean serum iron concentration in the preeclamptic group (190.65 ±22.77 µg/dl) compared to the normal pregnancy group (108.85 ±23.91 µg/dl), with the difference achieving high statistical significance (P<0.001).

Keywords: Preeclampsia, Serum iron.

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Introduction

Pregnancy represents one of the most significant physiological phenomena in reproductive-aged women, accompanied by extensive adaptations across physiological, metabolic, and biochemical domains.[1] Among the various complications that may arise during gestation, preeclampsia remains a predominant contributor to both maternal and perinatal morbidity and mortality worldwide. Each year, a substantial number of maternal deaths are attributed to this condition, and the risk of neonatal mortality in preeclamptic pregnancies is estimated to be approximately fourfold higher than that observed in uncomplicated gestations.[2] Accumulating evidence from multiple investigations suggests that alterations in serum iron concentrations among preeclamptic patients may be implicated in the underlying pathophysiological mechanisms of the disease. Disruption of the normal homeostatic balance of essential trace elements, particularly iron, can adversely influence critical biological processes, potentially predisposing to the development of preeclampsia.[3] Elevated circulating iron levels have been associated with an increased susceptibility to adverse pregnancy outcomes, including preeclampsia.[4] Consequently, fluctuations in serum iron concentration may serve as a contributory factor in the etiopathogenesis of this disorder. Comparative assessment of serum iron status between normotensive pregnant women and those with preeclampsia could facilitate the identification of whether deviations in this parameter play a causal role in disease development.

Iron is a fundamental trace element that serves as an essential cofactor for numerous proteins and enzymes involved in vital physiological functions, including oxygen transport, cellular energy production, and deoxyribonucleic acid (DNA) synthesis.[5] This metal has been shown to promote lipid peroxidation, a process potentially facilitated by the hyperlipidemic state that accompanies the substantial lipid mobilization occurring during the latter half of human gestation.[6] When maternal iron stores are inadequate, iron supplementation may contribute to improved pregnancy outcomes. In preeclamptic women, elevated serum iron levels have been attributed to several interrelated mechanisms, including hemolysis, hepatocellular injury, and hemoconcentration. The hemolytic process, in particular, is considered a significant factor contributing to the hyperferremia observed in this condition. Placental ischemia or hypoxia is widely recognized as a central pathogenic feature.[7] Under ischemic conditions, reactive oxygen species such as superoxide and hydrogen peroxide are generated; however, these species lack sufficient reactivity to directly induce cellular injury. Nevertheless, in the presence of catalytic quantities of transition metals, particularly iron—which may accumulate in the ischemic placenta through erythrocyte destruction within thrombotic, necrotic, and hemorrhagic foci—the highly reactive hydroxyl radical (OH-) and ferryl ion (FeO^{2+}) can be generated via Fenton chemistry.[8] These potent radicals are capable of initiating lipid peroxidation cascades, which, if unchecked, may culminate in endothelial cell damage. Some investigators have proposed that the elevated serum iron levels observed in preeclampsia may deviate from hemolysis, which could arise from mechanical disruption of erythrocytes secondary to intense vasospasm or aberrant endothelial cell-erythrocyte interactions.[9] Oxidative stress is widely acknowledged to play a pivotal role in the pathogenesis of preeclampsia. An imbalance between pro-oxidant and antioxidant defense mechanisms results in oxidative stress, thereby augmenting the potential for preeclampsia development.[10] Given that preeclampsia is a genetically and phenotypically heterogeneous disorder, it is improbable that a single gene exerts a dominant influence on its pathogenesis.[11] Furthermore, the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count) may manifest in 0.1–0.8% of all pregnancies and complicates 10–20% of pregnancies ultimately diagnosed with preeclampsia featuring severe characteristics.[12]

Materials and methods

This cross-sectional comparative investigation was conducted over one year, from July 2016 to June 2017, at the Department of Biochemistry, Mymensingh Medical College, in conjunction with the Department of Obstetrics and Gynecology at Mymensingh Medical College Hospital, Bangladesh. The study enrolled 100 pregnant women, who were categorized into two equal cohorts: a case group (Group II, n=50) comprising preeclamptic patients, and a control group (Group I, n=50) consisting of normotensive healthy gravidas. All participants were between 20 and 40 years of age. Subject selection was performed according to predefined inclusion and exclusion criteria, and each participant provided written informed consent before inclusion. Venous blood specimens were drawn under aseptic conditions, and serum iron levels were subsequently quantified via a colorimetric assay using a commercially available diagnostic kit at the Department of Biochemistry. The following demographic and clinical

parameters were systematically documented for every subject: maternal chronological age (years), gestational duration (weeks), and both systolic (SBP) and diastolic (DBP) blood pressures (mmHg). All quantitative data are expressed as mean ± standard deviation. Comparative analyses between groups were carried out using Student's unpaired t-test, with a P-value of less than 0.05 (P<0.05) designated as the threshold for statistical significance. All statistical computations were performed utilizing the Statistical Package for Social Sciences (SPSS), version 21.0 for Windows.

Results

In this study, the case group exhibited a mean maternal age of 26.56±4.67 years and a mean gestational age of 32.28±4.11 weeks, whereas the corresponding values for the control group were 25.82±4.45 years and 34.00±4.74 weeks. Intergroup comparisons of these baseline characteristics revealed no statistically significant differences (P>0.05), confirming appropriate matching between the two study groups. In contrast, both SBP and DBP were substantially elevated in the preeclamptic cohort, with mean values of 165.60±16.06 mmHg and 110.60±14.0 mmHg, respectively, compared to 107.40±7.77 mmHg and 68.20±7.19 mmHg in the control group.

Table 01: Clinical parameters of the study population

Variables	Group-I (Control) Mean ±SD	Group-II (Case) Mean ±SD	P-value
Maternal age (years)	25.82±4.45	26.56±4.67	0.419[NS]
Gestational age (weeks)	34±4.74	32.28±4.11	0.292[NS]
Systolic Blood Pressure (mmHg)	107.40±7.77	165.60±16.06	P<0.001**
Diastolic Blood Pressure (mmHg)	68.20±7.19	110.60±14.0	P<0.001**

In this study, it was observed that the mean values of serum iron levels were 190.65±22.77 and 108.85±23.91 µg/dl in Group-II (case) and Group-I (control), respectively. A highly significant (P<0.001) increase in serum iron levels was observed in preeclamptic patients (case) when compared to that of the normal healthy pregnant women. Analysis of mean serum iron levels of the study population is presented in Table 02 and Figure 01.

Table 02: Comparison of mean serum iron levels in the study population

	Group-I (control) Mean ±SD	Group-II (case) Mean ±SD	P-value
Serum iron (µg/dl)	108.85 ±23.91	190.65 ±22.77	<0.001**

**P less than 0.05 taken as the level of significance.
P<0.001 considered as a highly significant result.
SD=Standard deviation

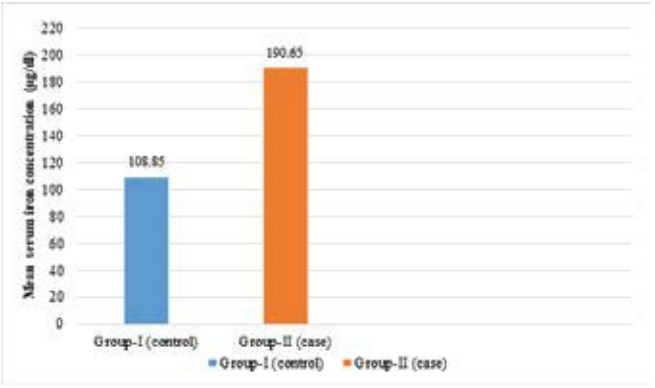

Figure 01: Comparison of mean serum iron levels in the study population

Discussion

In the current investigation, the mean serum iron concentration recorded for the control group (Group I) was 108.85±23.91 µg/dL, whereas the preeclamptic cohort (Group II) demonstrated a substantially higher mean value of 190.65±22.77 µg/dL. This observed intergroup difference was statistically highly significant (P<0.001), indicating a marked elevation in circulating iron levels among preeclamptic patients relative to their normotensive counterparts. These findings align closely with previously reported observations by Bansode et al.[13], Masti and Dattatreya[1], Sultana et al.[14], Zafar and Iqbal[10], Rayman[15], and Hubel et al.[16]

The work of Masti and Dattatreya[1] proposed a mechanistic explanation, suggesting that tissue ischemia occurring in preeclampsia may lead to the accumulation of iron within the ischemic placenta, potentially arising from erythrocyte destruction in thrombotic, necrotic, and hemorrhagic foci. This locally released iron can subsequently catalyze the generation of free radicals, initiating lipid peroxidation cascades and ultimately resulting in endothelial injury. It has further been postulated that iron or its reactive species may serve as a contributing factor in the propagation of oxidative stress characteristic of preeclamptic states.[14] Iron-mediated oxidative damage has been documented across various tissues, including the intestinal mucosa, liver, spleen, bone marrow, and placenta. The detection of hydroxyl and methoxyl radical formation within both the luminal and mucosal compartments of the gastrointestinal tract provides additional evidence for the involvement of iron in free radical-mediated tissue injury.[10] Several interrelated pathological processes, including hemolysis, hepatic dysfunction (which may facilitate the release of stored iron), and diminished erythropoietic activity, have been suggested as potential contributors to the elevated serum iron levels observed in preeclampsia.[13]

Nevertheless, conflicting evidence exists within the literature. In contrast to the present findings, investigations conducted by Sarwar et al.[3] and Rathore et al.[17] reported significantly diminished serum iron concentrations in preeclamptic women when compared to healthy pregnant controls. This discrepancy may be attributable to differential maternal iron status, as increased fetal iron requirements or maternal iron deficiency have been associated with both upregulation of placental transferrin receptor expression on the syncytiotrophoblast and enhanced ferritin receptor expression on the placental microvillar membrane.[18]

Conclusion

This study was done within the context of the facilities available to us. In this comparative study, serum iron levels were significantly higher than those in normal healthy pregnant women. Excess serum iron may have had a role in the pathogenesis of preeclampsia. Therefore, serum iron level could be assessed before giving iron supplements in preeclamptic patients.

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