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Original Research Article

Association of insulin resistance with pre-eclampsia: a case-control study

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ABSTRACT

Background: Preeclampsia, characterized by new-onset hypertension and proteinuria after 20 weeks of gestation, is a leading cause of maternal and perinatal death. Its etiology involves vascular endothelial dysfunction, oxidative stress, and metabolic dysregulation. Evidence suggests that insulin resistance may contribute to its development by promoting vasoconstriction and endothelial injuries. This study aimed to evaluate the association between insulin resistance and preeclampsia and its correlation with blood pressure in pregnant women.
Methods: A hospital-based case-control study was conducted at Department of Obstetrics and Gynaecology, Sir Salimullah Medical College Mitford Hospital, Dhaka, Bangladesh, from February 2022 to January 2023, including 50 preeclamptic and 50 normotensive pregnant women (gestational age, 20-40 weeks). Fasting plasma glucose and serum insulin were measured, and insulin resistance was calculated using the HOMA-IR index. Data were analyzed using SPSS version 24, and statistical significance was set at $p<0.05$.
Results: Preeclamptic women had significantly higher fasting glucose (4.48±0.51 mmol/l), serum insulin (29.0±5.97 µU/ml), and HOMA-IR (3.52±0.74) compared to controls (4.24±0.47 mmol/l, 13.94±3.61 µU/ml, 1.73±0.40; $p<0.001$). Women with HOMA-IR ≥1.7 had a 7.94-fold increased risk of preeclampsia (95% CI: 2.87–21.98). Insulin resistance was strongly correlated with systolic ($r=0.755$) and diastolic ($r=0.774$) blood pressure ($p<0.001$).
Conclusions: Elevated insulin resistance is significantly associated with preeclampsia and blood pressure elevation, indicating its potential utility as an early biomarker for the risk assessment and prevention of preeclampsia.

Keywords: Fasting insulin, HOMA-IR, Insulin resistance, Preeclampsia, Pregnancy hypertension

INTRODUCTION

Preeclampsia remains a major contributor to maternal and perinatal morbidity and mortality worldwide. It is characterized by new-onset hypertension and proteinuria after 20 weeks of gestation and is associated with endothelial dysfunction and systemic inflammation. The global incidence is estimated at approximately 4.6%, with considerable regional variations ranging from 1.8% to 16.7% in developing countries.[1] In Bangladesh, the burden is higher, with studies reporting a prevalence of 14.4% in

some regions.[2] Despite extensive research, the precise pathophysiology of preeclampsia remains elusive, complicating early diagnosis and effective prevention.

Recent evidence has highlighted the role of insulin resistance (IR) in the pathogenesis of preeclampsia. Insulin resistance, defined as a reduced biological response of target tissues to insulin, results in compensatory hyperinsulinemia and metabolic disturbances.[3] During normal pregnancy, physiological IR increases progressively due to placental hormones such as human placental lactogen, progesterone, and cortisol.[4] However, in preeclampsia, IR is significantly elevated beyond physiological levels, suggesting a potential pathogenic link. Elevated insulin levels may contribute to hypertension through sympathetic nervous system activation, sodium retention, and vascular smooth muscle hypertrophy.[5]

The endothelial dysfunction observed in preeclampsia may also be exacerbated by insulin-mediated vascular changes. Studies have shown that IR may impair nitric oxide synthesis and promote oxidative stress, both central to the vascular pathology of preeclampsia.[6] Given its measurable biochemical basis, insulin resistance can serve as a relatively accessible biomarker for identifying women at higher risk, particularly in low-resource settings where genetic or molecular testing is impractical.

Several case-control and cohort studies have demonstrated that fasting plasma glucose, fasting serum insulin, and HOMA-IR values are significantly higher in preeclamptic women than in normotensive pregnant controls.[7,8] A study by Abhari et al reported that the mean HOMA-IR was nearly double in preeclamptic women compared to normotensive pregnancies.[7] Similar findings were echoed by Lakshmi et al, who found a strong positive correlation between IR and blood pressure.[8] These observations suggest that insulin resistance may not only be a secondary feature but could also play a causal role in disease progression.

Considering the clinical and biochemical interrelations, investigating insulin resistance as a potential diagnostic marker for preeclampsia is of considerable relevance. The present case-control study aimed to determine the association between insulin resistance and preeclampsia by comparing fasting plasma glucose, fasting serum insulin, and HOMA-IR levels in preeclamptic and normotensive pregnant women and by exploring the correlation of IR with blood pressure parameters.

METHODS

This was a hospital-based case-control study conducted in the Department of Obstetrics and Gynaecology at Sir Salimullah Medical College Mitford Hospital, Dhaka, Bangladesh, from February 2022 to January 2023. A total of 100 pregnant women were enrolled, comprising 50 preeclamptic cases and 50 normotensive pregnant controls. All participants were between 18 and 38 years of age and had singleton pregnancies with gestational ages ranging from 20 to 40 weeks.

Inclusion criteria

Pregnant women aged between 18 and 38 years with a gestational age of 20 to 40 weeks and a singleton pregnancy were eligible for inclusion in the study. Women diagnosed with preeclampsia were included in the case group, while normotensive pregnant women were included in the control group.

Exclusion criteria

Participants were excluded from the study if they had chronic hypertension or renal disease, multiple pregnancy, congenital fetal anomalies, diabetes mellitus or hyperglycemia in pregnancy, or obesity (body mass index ≥30 kg/m²).

Data collection procedure

After obtaining ethical clearance and informed consent, each participant underwent a structured clinical assessment. Sociodemographic and obstetric data were collected through interviews. Blood pressure was measured using a standard sphygmomanometer after 10 minutes of rest, applying Korotkoff phase I and V sounds to determine systolic and diastolic pressures, respectively. Fasting blood samples (5 mL) were collected after an overnight fast: 2 mL in EDTA vacutainers for plasma glucose (estimated by glucose oxidase method) and 3 mL in clot activator tubes for serum insulin (measured by Siemens Atellica IM IRI lite reagent). Urinary protein was determined by the dipstick method. All laboratory analyses were conducted at the Biochemistry Department of Bangabandhu Sheikh Mujib Medical University (BSMMU), ensuring standardized laboratory protocols.

Ethical considerations

The study received ethical approval from the Institutional Review Board of Sir Salimullah Medical College, Mitford Hospital. Written informed consent was obtained from all participants after explaining study objectives, potential risks, and confidentiality assurances. Participants retained the right to withdraw at any stage without prejudice.

Statistical analysis

Data were coded, checked for completeness, and analyzed using SPSS version 24. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The independent sample t-test was applied to compare continuous variables, and the chi-square test was used for categorical variables. Pearson's correlation coefficient determined the relationship between HOMA-IR and blood

pressure. A p value <0.05 was considered statistically significant.

RESULTS

There were no significant differences between the two groups of patients regarding age (p value>0.05). The highest percentage of patients from both cases and control groups belonged to 25 to 31 years (48% in both groups respectively). BMI was found to be similar between the two groups (Table 1).

Table 1: Comparison of age and BMI in preeclamptic cases and controls (n=100).

Characteristics	Case, N (%)	Control, N (%)	P value
Age (in years)			
18-24	18 (36)	17 (34)	
25-31	24 (48)	24 (48)	0.957
32-38	8 (16)	9 (18)	
Mean±SD	26.74±4.41	26.84±4.62	0.912
BMI (kg/m^2)	23.46±2.29	23.35±1.98	0.794

Table 2: Comparison of fasting plasma glucose, serum insulin and insulin resistance in preeclamptic cases and controls (n=100).

Parameter	Case, Mean±SD	Control, Mean±SD	P value
Fasting plasma glucose (mmol/l)	4.48±0.51	4.24±0.47	0.019
Fasting serum insulin (µU/ml)	29.0±5.97	13.94±3.61	<0.001
Insulin resistance (HOMA-IR)	3.52±0.74	1.73±0.40	<0.001

The mean value of fasting plasma glucose in preeclamptic women was significantly higher than in control women and was statistically significant (p<0.05). Similarly, the mean value of fasting insulin and insulin resistance by HOMA-IR were also found significantly higher among the preeclamptic group than the control group, respectively (p<0.001) (Table 2).

There was a significant difference regarding insulin resistance between the case and control groups (p<0.001). Respondents with HOMA IR ≥1.7 had 7.94 times more chance to develop preeclampsia compared to that of respondents with HOMA IR <1.7 (OR=7.94, 95% CI=2.87-21.98) (Table 3).

Table 3: Distribution of the respondents by HOMA IR with preeclamptic cases and control groups (n=100).

HOMA-IR	Case, N (%)	Control N (%)	P value	Odds Ratio (95% CI)
<1.7	6 (12.0)	26 (52.0)	<0.001	7.94 (2.87-21.98)
≥1.7	44 (88.0)	24 (48.0)		

Table 4: Correlation of insulin resistance with systolic BP and diastolic BP(n=100).

Parameter	R value	P value
Systolic BP	0.755	<0.001
Diastolic BP	0.774	<0.001

Insulin resistance was directly proportional to systolic BP and diastolic BP, and was found statistically significant in both parameters (Table 4).

According to Pearson's correlation test, it was found that systolic blood pressure and HOMA-IR had statistically significant positive correlation in pregnant women (r=0.755, p<0.001) (Figure 1).

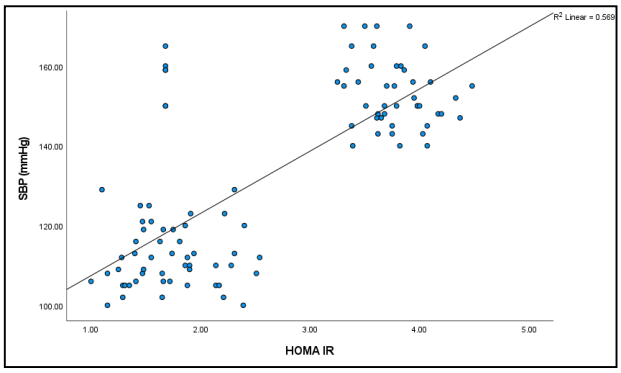

Figure 1: The scattered plot diagram showing the relationship between the systolic blood pressure and HOMA-IR of the participants (n=100).

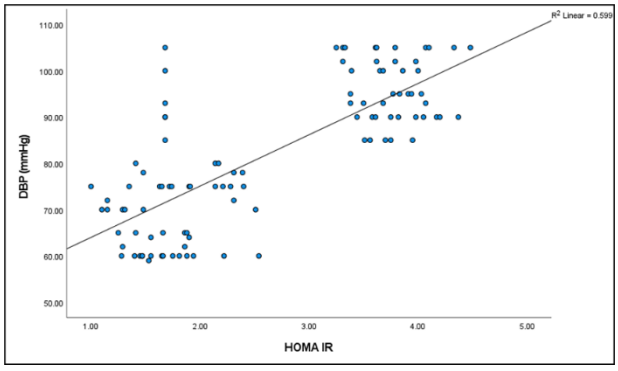

Figure 2: The scattered plot diagram showing the relationship between the diastolic blood pressure and HOMA-IR of the participants (n=100).

According to Pearson's correlation test, it was found that diastolic blood pressure and HOMA-IR had statistically significant positive correlation in pregnant women ($r=0.774$, $p<0.001$) (Figure 2).

DISCUSSION

This study aimed to determine the association of insulin resistance with preeclampsia and to explore the correlation between HOMA-IR and blood pressure among pregnant women. The findings revealed a markedly higher fasting plasma glucose, fasting serum insulin, and HOMA-IR in preeclamptic women compared with normotensive controls. Insulin resistance also showed a strong positive correlation with both systolic and diastolic blood pressure, reinforcing the role of metabolic dysregulation in the pathophysiology of preeclampsia.

The observed difference in insulin resistance between preeclamptic and normotensive women aligns with the findings of Abhari et al, who reported significantly elevated fasting insulin and HOMA-IR values in women with preeclampsia.[7] Similarly, Lakshmi et al found that preeclamptic participants had mean HOMA-IR nearly twice that of healthy pregnant women, emphasizing that insulin resistance is intricately linked to the development of preeclampsia.[8] The current study also supports the hypothesis that insulin resistance may precede the clinical onset of hypertension in pregnancy, thereby serving as a potential early biomarker.

The mean fasting plasma glucose level in preeclamptic women (4.48 mmol/l) was significantly higher than in controls (4.24 mmol/l). Although both values fall within the normal glycemic range, this subtle elevation may indicate impaired glucose metabolism secondary to insulin resistance. Hyperinsulinemia, as observed here, could reflect compensatory pancreatic activity aimed at maintaining normoglycemia despite reduced peripheral insulin sensitivity. Previous studies have described similar findings, suggesting that these alterations in glucose-insulin homeostasis may occur early in preeclampsia's pathogenesis.[9,10]

Increased serum insulin levels and resultant insulin resistance may contribute to vascular dysfunction through multiple mechanisms. Elevated insulin enhances sympathetic nervous system activity, promotes renal sodium reabsorption, and induces vascular smooth muscle proliferation, ultimately increasing peripheral vascular resistance and blood pressure.[5] Moreover, insulin resistance is associated with impaired endothelial nitric oxide synthesis and increased oxidative stress, which further aggravate vasoconstriction and endothelial injury-hallmark features of preeclampsia.[6,11] The strong correlation between HOMA-IR and both systolic and diastolic blood pressure observed in this study ($r=0.755$ and 0.774, respectively) substantiates this mechanistic link.

The odds ratio of 7.94 indicates that women with HOMA-IR ≥ 1.7 had nearly eight times the risk of developing preeclampsia compared to those with lower insulin resistance. This finding is consistent with the report of Sonagra et al, who observed that as the disease advances, insulin resistance increases proportionally.[12] These results underscore the potential clinical relevance of HOMA-IR as a predictive tool in antenatal screening, particularly in resource-limited settings where early identification of high-risk pregnancies could guide monitoring and intervention strategies.

The relationship between insulin resistance and preeclampsia is also supported by studies linking IR to angiogenic imbalance. Ghosh et al. demonstrated that microvascular dysfunction in preeclamptic women was influenced by insulin resistance and dysregulated angiogenic mediators such as VEGF and sFlt-1.[13] Elevated insulin levels may indirectly alter placental vascularization and nutrient transport, predisposing to placental ischemia-a central feature of preeclampsia's early stage. Similarly, Hauth et al reported that maternal insulin resistance correlated with preeclampsia severity, suggesting that the degree of metabolic dysfunction could predict clinical outcomes.[14]

Contradictory findings exist in the literature, with some studies reporting weak or no association between insulin resistance and preeclampsia. Balani et al found that maternal history of chronic hypertension and gestational weight gain, rather than HOMA-IR, were stronger predictors of preeclampsia among obese women.[15] Likewise, Teimoori et al did not observe significant differences in insulin sensitivity between preeclamptic and normotensive women.[16] These discrepancies may arise from differences in study design, population characteristics, or the timing of measurement, as insulin sensitivity varies throughout pregnancy. Nonetheless, the consistent pattern across multiple studies including the present one suggests that insulin resistance remains a key pathophysiological factor, especially in non-obese, normoglycemic populations.

In Bangladesh, where access to advanced diagnostic modalities is limited, the use of simple indices such as fasting glucose, insulin, and HOMA-IR could facilitate early identification of women at risk. The current study's results, derived from a well-defined cohort and standardized biochemical assessment, reinforce the feasibility of integrating metabolic screening into routine antenatal care. While this study was not designed to establish causality, the robust association observed provides a foundation for longitudinal studies exploring temporal relationships between early-pregnancy insulin resistance and subsequent development of preeclampsia.

Overall, the study demonstrates that increased insulin resistance is not merely an accompanying feature but likely plays a mechanistic role in preeclampsia. The results suggest that HOMA-IR may serve as a cost-effective

biomarker to identify women predisposed to hypertensive disorders of pregnancy, complementing traditional clinical assessments.

This study has few limitations. The study was conducted in a single hospital using a purposive sampling method, and the sample size was small. So, the results may not represent the whole community.

CONCLUSION

The study demonstrated a strong association between insulin resistance and preeclampsia. Preeclamptic women exhibited significantly higher fasting glucose, serum insulin, and HOMA-IR levels than normotensive controls. Insulin resistance was positively correlated with both systolic and diastolic blood pressure, suggesting its mechanistic involvement in disease progression. These findings highlight the potential utility of HOMA-IR as an accessible biomarker for early detection and risk stratification of preeclampsia, particularly in low-resource clinical settings.

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