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Atherogenic Dyslipidemia and Severity of Preeclampsia: Evidence from a Prospective Comparative Study

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ABSTRACT

Background: Preeclampsia is a pregnancy-specific hypertensive disorder and a major contributor to maternal and perinatal morbidity worldwide. Emerging evidence suggests that dysregulated lipid metabolism plays a key role in its pathophysiology. Atherogenic lipid ratios, particularly triglyceride to high-density lipoprotein (TG/HDL) and low-density lipoprotein to high-density lipoprotein (LDL/HDL), may provide better predictive value than individual lipid parameters. However, evidence from South Asian populations remains limited. This study evaluated the association between atherogenic lipid ratios and the severity of preeclampsia among pregnant women in Eastern India.

Methods: A prospective observational comparative cohort study was conducted at Rampurhat Government Medical College, West Bengal, India, from May 2023 to November 2024. A total of 180 pregnant women (≥20 week's gestation) were enrolled and categorized into three groups: normotensive controls (n=60), mild preeclampsia (n=60), and severe preeclampsia (n=60). Fasting lipid profiles, including total cholesterol, triglycerides, LDL-C, and HDL-C, were measured using standard enzymatic assays. Atherogenic lipid ratios (TG/HDL and LDL/HDL) were calculated. Statistical analyses included one-way ANOVA, Pearson correlation, multivariate logistic regression, and receiver operating characteristic (ROC) curve analysis to assess predictive performance.

Results: Women with preeclampsia exhibited significantly higher triglycerides and LDL-C levels and lower HDL-C levels compared with normotensive controls ($p<0.001$). The TG/HDL ratio increased progressively from controls (2.09) to mild preeclampsia (2.97) and severe preeclampsia (3.73), while the LDL/HDL ratio increased from 1.44 to 2.28 across the groups ($p<0.001$). The TG/HDL ratio showed a strong positive correlation with systolic ($r=0.87$) and diastolic blood pressure ($r=0.85$). Multivariate logistic regression identified TG/HDL as an independent predictor of pregnancy-induced hypertension ($p=0.013$). ROC curve analysis demonstrated excellent diagnostic performance for TG/HDL ratio (AUC=0.97).

Conclusions: Atherogenic lipid ratios, particularly TG/HDL, are strongly associated with the presence and severity of preeclampsia and may serve as reliable, low-cost biomarkers for

early risk stratification in pregnancy. Incorporating these ratios into routine antenatal screening could improve early identification of high-risk women and support timely clinical intervention.

Keywords: Preeclampsia, Atherogenic lipid ratios, TG/HDL ratio, LDL/HDL ratio, Dyslipidemia, Pregnancy-induced hypertension.

INTRODUCTION

Preeclampsia is a pregnancy-specific hypertensive disorder characterized by the onset of hypertension and multisystem involvement after 20 weeks of gestation. It remains a significant contributor to maternal and perinatal morbidity and mortality worldwide, particularly in low- and middle-income settings [1]. Affecting approximately 5–8% of pregnancies globally, preeclampsia is a major factor in adverse maternal and perinatal outcomes [2]. The burden is especially pronounced in low- and middle-income countries, where challenges in early detection, monitoring, and access to timely obstetric care exacerbate adverse outcomes. As a hypertensive disorder marked by widespread endothelial dysfunction and multi-organ involvement, preeclampsia poses serious risks to both maternal and fetal health. Given its substantial global impact, there is a critical need to enhance early identification of at-risk pregnancies through reliable biomarkers and to develop effective prevention and management strategies aimed at reducing disease-related complications. The pathophysiology of preeclampsia is complex and multifactorial, involving abnormal placentation, systemic endothelial dysfunction, and widespread inflammation. Beyond these classical mechanisms, accumulating evidence implicates dysregulated maternal lipid metabolism as a significant contributor to both disease onset and severity[3], [4], [5]. Multiple observational studies and meta-analyses have consistently demonstrated that women who develop preeclampsia exhibit a characteristic pattern of atherogenic dyslipidemia, marked by elevated triglycerides (TG), total cholesterol, and low-density lipoprotein cholesterol (LDL-C), alongside reduced high-density lipoprotein cholesterol (HDL-C) compared with normotensive pregnant controls[4], [6], [7]. These lipid alterations have been observed across diverse populations and gestational time points, suggesting that impaired lipid homeostasis may both reflect and exacerbate the vascular and inflammatory derangements central to preeclampsia. Elevated TG and LDL particles are prone to oxidative modification, promoting endothelial injury and systemic inflammation - key features of the disorder - while reduced HDL limits reverse cholesterol transport and antioxidant protection [6], [7], [8]. Meta-analytic evidence further confirms significantly higher TG, LDL-C, and total cholesterol and lower HDL-C in women with preeclampsia, underscoring the clinical relevance of lipid abnormalities in this condition[4], [7].

Despite this, much of the extant evidence originates from cohorts in Western and East Asian contexts, with limited data available from South Asia and India. In India, the burden of hypertensive disorders during pregnancy is substantial, yet there is a paucity of research examining maternal lipid metabolism and its association with disease severity in this population[9]. South Asian populations, including those in India, are recognized for their distinct cardio metabolic risk profiles and a high prevalence of dyslipidemia, which may influence the development and progression of preeclampsia[10]. Understanding the role of lipid parameters in this region could, therefore, provide region-specific biomarkers for early risk stratification and targeted clinical intervention.

Atherogenic lipid ratios, such as TG/HDL and LDL/HDL, integrate key aspects of lipid imbalance and may enhance predictive

accuracy compared to individual lipid measurements alone. However, their utility as predictors of preeclampsia severity has not been systematically evaluated in India, particularly in Eastern India. Therefore, the primary objective of this study was to evaluate the association between these atherogenic lipid ratios and the severity of preeclampsia. Secondary objectives included comparing maternal lipid profiles between normotensive pregnant women and those with preeclampsia, determining whether TG/HDL and LDL/HDL ratios can predict severe disease, and assessing the diagnostic performance of these ratios using receiver operating characteristic (ROC) curve analysis

MATERIALS & METHODS

This study was a prospective observational comparative cohort study conducted in the Department of Gynaecology & Obstetrics at Rampurhat Government Medical College, West Bengal, India. The study was carried out from May 2023 to November 2024, with an additional one-year period dedicated to follow-up and comprehensive data analysis. The primary objective was to evaluate the association between atherogenic lipid ratios (TG/HDL and LDL/HDL) and the severity of preeclampsia, while secondary objectives included comparing maternal lipid profiles between groups and assessing the predictive value of these ratios using receiver operating characteristic (ROC) curve analysis.

All procedures were conducted in accordance with the ethical principles outlined in the declaration of Helsinki and adhered to applicable institutional and national guidelines for research involving human participants. Prior to participation, all subjects were provided with detailed information regarding the study objectives, procedures, potential risks, and anticipated benefits. Written informed consent was obtained from each participant before enrollment.

The study population included pregnant women attending antenatal clinics or admitted to obstetric wards at Rampurhat Government Medical College. Participants were enrolled after applying the following criteria:

Inclusion Criteria

- Singleton pregnancy
- Gestational age ≥20 weeks
- Diagnosis of preeclampsia based on standard clinical criteria, including new-onset hypertension and proteinuria or evidence of end-organ dysfunction

Exclusion Criteria

- Chronic hypertension prior to pregnancy
- Diabetes mellitus
- Renal or hepatic disease
- Multiple pregnancy (e.g., twins or higher-order gestation)

Participants were categorized into three groups according to clinical status:

1. Normotensive pregnant women (control group): Women with normal blood pressure and no evidence of proteinuria or hypertensive disorders.
2. Mild preeclampsia group: Women diagnosed with preeclampsia presenting with elevated blood pressure and proteinuria without severe clinical features.
3. Severe preeclampsia group: Women with preeclampsia exhibiting severe clinical features, including markedly elevated blood pressure and/or evidence of maternal organ dysfunction.

Maternal demographic and clinical data were collected using a structured data collection form, including maternal age, parity, body mass index (BMI), gestational age, and relevant obstetric history. Clinical evaluation included:

- Blood pressure measurement using a standardized sphygmomanometer
- Assessment of urinary protein using dipstick or quantitative methods
- Fasting venous blood samples for lipid profile analysis

Fasting blood samples were analyzed in the hospital laboratory for the following lipid parameters using standard enzymatic assays:

- Total cholesterol (TC)
- Triglycerides (TG)
- Low-density lipoprotein cholesterol (LDL-C)
- High-density lipoprotein cholesterol (HDL-C)

Atherogenic lipid ratios were calculated as follows:

- TG/HDL ratio = Triglycerides ÷ HDL cholesterol
- LDL/HDL ratio = LDL cholesterol ÷ HDL cholesterol

These ratios were evaluated as potential predictors of severe preeclampsia.

All data were recorded in a predesigned, standardized proforma and subsequently entered into Microsoft Excel (Microsoft Corp., Redmond, WA, USA) for data management. Statistical analyses were conducted using Graphpad Prism (version 10.6.1) and IBM SPSS Statistics (version 31). Data was analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. Statistical methods included:

- One-way ANOVA or Kruskal–Wallis test for comparing lipid parameters and ratios among study groups
- Pearson or Spearman correlation analysis to assess associations between lipid ratios and clinical variables
- Multivariate logistic regression analysis to determine independent predictors of severe preeclampsia
- Receiver operating characteristic (ROC) curve analysis to evaluate diagnostic performance of TG/HDL and LDL/HDL ratios

A p-value <0.05 was considered statistically significant.

RESULT

A total of 180 pregnant women were included in the study, with 60 participants in each group: normotensive controls, mild preeclampsia (PE), and severe PE. The baseline demographic characteristics, including maternal age, gestational age, and body mass index (BMI), were comparable among the three groups, with no statistically significant differences ($p > 0.05$). As expected, both systolic and diastolic blood pressures were significantly higher in women with severe preeclampsia compared with the mild PE and control groups (systolic BP: 164 ± 11 mmHg vs. 149 ± 8 mmHg and 108 ± 9 mmHg; diastolic BP: 110 ± 6 mmHg vs. 98 ± 6 mmHg and 74 ± 6 mmHg; $p < 0.001$), confirming the clinical classification of disease severity (Table 1). These findings demonstrate that the study groups were well-matched for demographic variables, while the hemodynamic parameters appropriately reflected the progression from normotension to severe preeclampsia.

Table1. Baseline Demographic and Clinical Characteristics

Parameter	Control (n=60)	Mild PE (n=60)	Severe PE (n=60)	p-value
Maternal age (years)	25.05 ± 3.36	26.93 ± 4.13	25.93 ± 3.58	>0.05
Gestational age (weeks)	37.95 ± 1.64	36.74 ± 2.51	36.43 ± 1.91	>0.05
Systolic BP (mmHg)	108 ± 9	149 ± 8	164 ± 11	<0.001
Diastolic BP (mmHg)	74 ± 6	98 ± 6	110 ± 6	<0.001
BMI	23.43±3.43	24.06 ± 2.49	24.67± 3.09	>0.05

The comparison of lipid profile parameters among the study groups is presented in Table 2. Women with severe preeclampsia showed significantly higher levels of triglycerides and LDL cholesterol, while HDL cholesterol levels were significantly lower compared with normotensive controls ($p < 0.001$).

Women with preeclampsia demonstrated a significantly more atherogenic lipid profile

compared with normotensive controls. Mean total cholesterol, triglycerides, and LDL cholesterol increased progressively from the control group to mild and severe preeclampsia, while HDL cholesterol decreased significantly with increasing disease severity. These differences were statistically highly significant across groups ($p < 0.001$, one-way ANOVA), indicating a strong association between dyslipidemia and the severity of preeclampsia.

Table 2. Lipid Profile among Study Groups

Parameter (mg/dl)	Control(n=60)	Mild PE(n=60)	Severe PE(n=60)	p-value
Total Cholesterol	192.25 ± 8.35	206.12 ± 12.29	232.45 ± 19.26	<0.001
Triglycerides	141.36 ± 10.38	172.27± 11.45	190.33 ± 12.30	<0.001
LDL Cholesterol	96.96 ± 9.95	126 ± 24	116.22 ± 18.87	<0.001
HDL Cholesterol	67.54 ± 1.41	57.98 ± 3.21	51 ± 2.58	<0.001

Atherogenic lipid ratios were calculated to evaluate their association with the severity of preeclampsia (Table 3). The TG/HDL ratio increased progressively from 2.09 in the control group to 2.97 in mild preeclampsia and 3.73 in severe preeclampsia. Similarly, the LDL/HDL ratio was significantly higher among women with preeclampsia compared with normotensive controls (1.44 vs. 2.17 and 2.28 for mild and severe preeclampsia, respectively). Both ratios demonstrated highly significant differences among the study groups ($p < 0.001$), suggesting that elevated atherogenic lipid indices are strongly associated with the severity of preeclampsia.

Table 3. Comparison of Atherogenic Lipid Ratios among Study Groups

Parameter	Control (n=60)	Mild PE (n=60)	Severe PE (n=60)	p-value
TG/HDL ratio	2.09	2.97	3.73	<0.001
LDL/HDL ratio	1.44	2.17	2.28	<0.001

Post-hoc analysis revealed significant differences between all three groups ($p <0.01$).

There is a strong positive linear relationship between the TG/HDL ratio and both systolic and diastolic blood pressure in our sample. This suggests that as the TG/HDL ratio increases, both SBP and DBP tend to increase, supporting the link between lipid profile alterations and hypertension severity in preeclampsia (Table 4).

Table 4: Correlation between TG/HDL and SBP, DBP

Variables	Pearson r	p-value	Interpretation
TG/HDL ratio vs SBP	0.87	<0.001	Strong positive correlation, highly significant
TG/HDL ratio vs DBP	0.85	<0.001	Strong positive correlation, highly significant

Pearson correlation analysis between systolic blood pressure and LDL/HDL ratio showed a weak positive correlation ($r = 0.186$), indicating that increases in systolic blood pressure are associated with only a slight increase in LDL/HDL ratio (Fig 1).

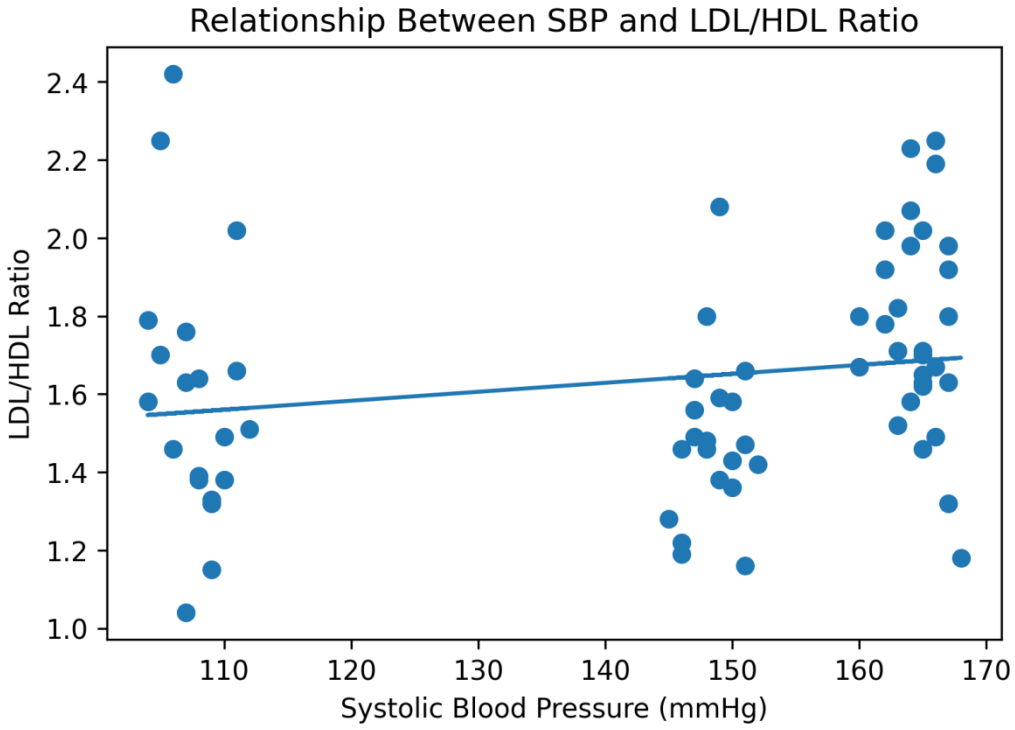

Figure 1: Correlation between LDL/HDL ratios with systolic blood pressure

A multivariate logistic regression analysis was performed to identify independent predictors of pregnancy-induced hypertension (PIH) using age, body mass index (BMI), TG/HDL ratio, and LDL/HDL ratio as covariates. The model demonstrated a good overall fit and was statistically significant (Likelihood ratio test, $p < 0.001$; pseudo $R^2 = 0.799$). Among the variables included in the model, the TG/HDL ratio emerged as a significant independent predictor of PIH (adjusted OR = 6.66×10^6, 95% CI: 27.3 – 1.62×10^{12}, $p = 0.013$), indicating that higher TG/HDL ratios were associated with increased odds of developing PIH. In contrast, age (adjusted OR = 1.25, 95% CI: 0.86 – 1.81, $p = 0.245$) and BMI (adjusted OR = 1.47, 95% CI: 0.73 – 2.94, $p = 0.277$) were not significantly associated with PIH after adjustment for other variables. The LDL/HDL ratio showed a borderline association with PIH (adjusted OR = 0.00019, 95% CI: 1.63×10^{-8} – 2.23, $p = 0.073$), but did not reach statistical significance. These findings suggest that the TG/HDL ratio may serve as an important lipid marker associated with the risk of PIH in the studied population (Table 5).

Table 5: Multivariate Logistic Regression Analysis for Predictors of PIH

Variable	β Coefficient	Adjusted OR	95% CI for OR	p-value
Age	0.221	1.25	0.86 – 1.81	0.245
BMI	0.384	1.47	0.73 – 2.94	0.277
TG/HDL ratio	15.71	6.66×10^6	27.30 – 1.62×10^{12}	0.013
LDL/HDL ratio	−8.57	0.00019	1.63×10^{-8} – 2.23	0.073

Receiver operating characteristic (ROC) curve analysis (Fig 2) was performed to evaluate the ability of the TG/HDL ratio to predict pregnancy-induced hypertension (PIH). The analysis demonstrated an area under the curve (AUC) of 0.97, indicating excellent discriminatory performance of the TG/HDL ratio in distinguishing women with PIH from those without the condition. An AUC value close to 1.0 suggests a high diagnostic accuracy, with the TG/HDL ratio showing strong sensitivity and specificity for identifying PIH cases. These findings suggest that the TG/HDL ratio may serve as

a useful biochemical marker for predicting the risk of PIH, supporting its potential role in early risk stratification and clinical assessment of pregnant women.

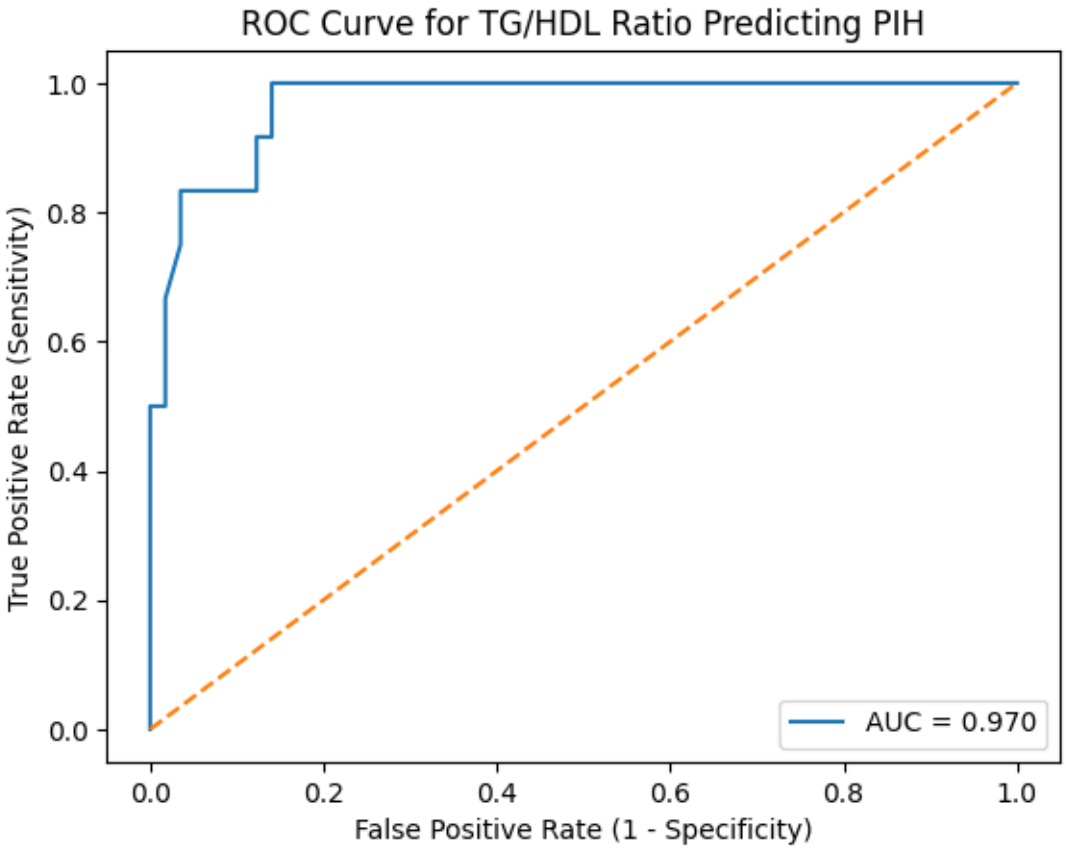

Figure 2: ROC curve for TG/HDL ratio predicting PIH

DISCUSSION

Atherogenic lipid ratios (TG/HDL, LDL/HDL) as predictors of severity of preeclampsia

In our study, the TG/HDL ratio exhibited a progressive increase from the control group to those with severe preeclampsia, while the LDL/HDL ratio increased from 1.44 to 2.28 across the groups.

These ratios encapsulate the interaction between heightened oxidative stress and impaired lipid transport, which are pivotal factors driving the systemic inflammatory response in preeclamptic pregnancies, consistent with the findings of previous studies [5]. Our study corroborates earlier research by establishing the role of elevated very-low-density lipoprotein and oxidized LDL levels in exacerbating endothelial injury. Integrating these ratios into clinical assessments may offer a more nuanced stratification of patients at risk for severe adverse outcomes [5], [11].

A principal finding of this study was the progressive increase in the TG/HDL ratio (from 2.09 in controls to 3.73 in severe PE) and the LDL/HDL ratio (from 1.44 to 2.28). This observation is consistent with existing literature, which suggests that individual lipid parameters are less predictive than their ratios, as these ratios encapsulate the balance between pro-atherogenic and anti-atherogenic forces [5]. The observed progressive increase in total cholesterol, TG, and LDL, alongside a decrease in HDL, aligns with systematic reviews identifying early pregnancy dyslipidemia as a significant risk factor for PE [11]. Notably, the "outstanding" diagnostic performance of the TG/HDL ratio in our cohort (AUC = 0.97) surpasses several existing models, such as the second-trimester lipid

nomogram reported by Li et al., which achieved an AUC of 0.912 using a combination of five risk factors [11], [13].

The strong positive linear relationship observed between the TG/HDL ratio and both systolic (r = 0.87) and diastolic (r = 0.85) blood pressure suggests that lipid metabolic shifts are closely linked to the hemodynamic manifestations of this disease. This correlation supports the hypothesis that the accumulation of triglyceride-rich lipoproteins directly contributes to endothelial dysfunction [12], [13]. Unlike normal pregnancies, in which hyperlipidemia is a physiological adaptation to support fetal growth, the hypertriglyceridemia observed in PE appears to be pathological, potentially reflecting impaired placental uptake and decreased lipoprotein lipolysis [12], [14]. The resulting accumulation of TG-rich remnant lipoproteins can induce platelet activation and further damage the vascular endothelium [12], [15]. The association between elevated LDL/HDL ratios and PE severity likely arises from the susceptibility of LDL and HDL particles to oxidative modification [16]. In PE, the placenta acts as a major source of reactive oxygen species, leading to the formation of oxidized LDL, which is highly toxic to the vascular endothelium [7], [16]. Our findings of significantly lower HDL levels in severe PE (51 mg/dL vs. 67.54 mg/dl in controls) are particularly critical, as HDL typically protects the maternal vascular endothelium from oxidative damage [16]. A decrease in HDL leads to the inactivation of paraoxonase 1, an antioxidant enzyme that prevents the peroxidation of LDL [13]. This loss of antioxidant capacity, combined with elevated atherogenic lipids, exacerbates systemic inflammation and contributes to the characteristic vascular lesions of PE, often described as "acute atherosis" of the spiral arteries [17].

Multivariate logistic regression analysis identified the TG/HDL ratio as a powerful independent predictor of pregnancy-induced hypertension (adjusted OR = 6.66 × 10^6, P = 0.013). Although individual factors, such as body mass index and maternal age, were not significant after adjustment in our model, the lipid ratio remained a robust indicator of risk. This suggests that the TG/HDL ratio could serve as a vital clinical tool for early risk stratification, particularly in resource-limited settings, where it can be calculated from standard, inexpensive lipid panels [5], [11].

CONCLUSION

The present study provides compelling evidence that atherogenic lipid ratios, particularly the triglyceride-to-high-density lipoprotein (TG/HDL) and low-density lipoprotein-to-high-density lipoprotein (LDL/HDL) ratios, serve as robust independent predictors of both the occurrence and severity of preeclampsia. The progressive elevation of these ratios with increasing disease severity indicates a pronounced shift toward a pro-atherogenic metabolic profile, supporting the notion that dysregulated lipid metabolism plays a central role in the pathophysiology of preeclampsia. Among the evaluated markers, the TG/HDL ratio demonstrated exceptional diagnostic accuracy (area under the curve [AUC] = 0.97) and exhibited strong positive correlations with both systolic and diastolic blood pressure, underscoring its close association with the hemodynamic burden of the disease.

Multivariate analysis identified the TG/HDL ratio as a powerful independent predictor of preeclampsia, surpassing conventional demographic risk indicators, such as maternal age and body mass index. These findings reinforce emerging evidence that lipid-derived indices may provide clinically meaningful insights into endothelial dysfunction and cardiometabolic stress during pregnancy.

As TG/HDL and LDL/HDL ratios can be readily derived from routine lipid profile testing, their incorporation into antenatal screening frameworks could offer a practical, low-cost, and scalable strategy for the early identification of women at an

elevated risk of preeclampsia. Integrating atherogenic lipid ratios into existing obstetric risk-assessment models may enhance early risk stratification, guide closer clinical monitoring, and facilitate timely preventive or therapeutic interventions, thereby improving maternal and fetal outcomes.

Future research should focus on large, multicenter prospective studies to validate these findings and establish clinically standardized threshold values for atherogenic lipid ratios during pregnancy. Additionally, integrating lipid ratios with emerging biomarkers and machine learning–based predictive models may further refine early-detection strategies and strengthen precision obstetric care.

The findings of this study should be interpreted with caution because of several limitations. The relatively small sample size and recruitment from a single clinical center may constrain the generalizability of the results, as the study population may not fully represent the metabolic, genetic, and socioeconomic diversity of pregnant women across different regions. Although the TG/HDL ratio demonstrated strong discriminatory performance, universally accepted clinical cutoff values for atherogenic lipid ratios during pregnancy have not yet been established, limiting their immediate application in routine obstetric screening. Additionally, the observational design of the study precludes establishing a causal relationship between dyslipidemia and the development of preeclampsia. While multivariate regression was employed to adjust for major confounders, other factors, such as dietary habits, physical activity, socioeconomic status, and genetic predisposition to lipid metabolism disorders, were not comprehensively assessed and may have influenced maternal lipid profiles. Furthermore, the extremely high odds ratio observed for the TG/HDL ratio may reflect potential model instability related to the relatively small sample size; therefore, the magnitude of this estimate should be interpreted cautiously and validated in larger multicentre studies.

Declaration by Authors

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