International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-09-12*

Dyslipidaemia as a Predictor of Hypertensive Disorders in Pregnancy: A Prospective Observational Study.

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Received: 04-08-2026
Revised: 13-08-2026
Accepted: 24-08-2026
Published: 02-09-2026

Abstract

Background: Hypertensive disorders of pregnancy (HDP) are major causes of maternal and perinatal morbidity and mortality worldwide. Dyslipidemia has been increasingly recognized as a potential predictor of HDP, yet evidence from prospective studies remains limited. This study aimed to evaluate dyslipidemia as a predictor of HDP and compare maternal and neonatal outcomes between hypertensive and normotensive women. ***Methods:*** This prospective observational study enrolled 220 pregnant women with singleton pregnancies between 20-28 weeks of gestation who had elevated lipid profiles (total cholesterol >240 mg/dL or triglycerides >200 mg/dL) but were normotensive at enrollment. Participants were followed until delivery and divided into hypertensive (n=38) and normotensive (n=182) groups based on blood pressure after 20 weeks. Lipid profiles, demographic factors, laboratory parameters, maternal complications, and neonatal outcomes were compared between groups. ***Results:*** The incidence of HDP was 17.27% (38/220). Hypertensive women had significantly higher total cholesterol (276.87±38.96 vs. 263.62±29.28 mg/dL), triglycerides (253.72±32.53 vs. 231.47±28.69 mg/dL), LDL-C (159.16±27.52 vs. 153.56±20.32 mg/dL), and lower HDL-C (38.33±5.03 vs. 41.91±6.52 mg/dL) ($p<0.05$). Independent predictors included maternal age>30 years (AOR:2.81), BMI (AOR:1.20), triglycerides (AOR:1.16), total cholesterol (AOR:1.13), LDL-C (AOR:1.12), and serum creatinine (AOR:2.00), while HDL-C (AOR:0.62) and platelet count (AOR:0.90) were protective. ***Conclusion:*** Dyslipidemia, particularly elevated triglycerides and reduced HDL-C, independently predicts HDP and is associated with adverse maternal and neonatal outcomes.

Keywords: Dyslipidemia, Hypertensive disorders of pregnancy, Preeclampsia, Maternal outcomes, Neonatal outcomes.

INTRODUCTION

Hypertensive disorders of pregnancy (HDP), encompassing gestational hypertension, preeclampsia (PE), and eclampsia, represent a major global health challenge, complicating approximately 2–8% of pregnancies worldwide and contributing significantly to maternal and perinatal morbidity and mortality [1]. These conditions are characterized by new-onset hypertension after 20 weeks of gestation, often accompanied by proteinuria or other organ dysfunction in the case of PE [1, 2]. HDP rank among the leading causes of maternal death, accounting for a substantial proportion of pregnancy-related mortality, and are associated with adverse neonatal outcomes such as preterm birth, low birth weight (LBW), fetal growth restriction, and increased neonatal intensive care unit (NICU) admissions [3, 4].

In normal pregnancy, physiological adaptations include progressive increases in lipid levels to support fetal growth and placental function. Total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C) rise, while high-density lipoprotein cholesterol (HDL-C) may show variable changes [5]. However, in pathological states like HDP, these changes can become exaggerated, leading to dyslipidemia—an atherogenic lipid profile marked by elevated TC, TG, LDL-C, and reduced HDL-C. Emerging evidence positions dyslipidemia as both a potential predictor and a contributor to the pathogenesis of HDP. Observational studies and meta-analyses have consistently demonstrated that women who develop PE exhibit higher levels of TG and non-HDL-C across trimesters, with lower HDL-C particularly evident in the third trimester [6-10].

Dyslipidemia often coexists with other metabolic conditions like GDM, amplifying risk. Glucose intolerance during pregnancy is associated with hypertension and hyperlipidemia, which in turn heighten PE likelihood through shared pathways of insulin resistance and inflammation [8]. Early-pregnancy dyslipidemia, such as elevated fasting TC >160

mg/dL, has been identified as a predictor of subsequent hypertensive disorders. These metabolic derangements not only predict HDP but also influence disease severity [11].

Maternal outcomes in HDP complicated by dyslipidemia include higher risks of severe features (e.g., HELLP syndrome, pulmonary edema, renal impairment), cesarean delivery, and long-term cardiovascular disease [3]. Neonatal consequences are profound: increased preterm birth rates, LBW, small-for-gestational-age infants, NICU admissions, and higher perinatal mortality. For instance, maternal dyslipidemia has been associated with LBW and prolonged NICU stays, independent of preterm birth in some cohorts [12].

Despite these associations, gaps remain in understanding the predictive utility of dyslipidemia for HDP and its specific impact on comparative maternal-neonatal outcomes. Most evidence derives from observational data, with variability in lipid assessment timing and definitions. Longitudinal studies comparing dyslipidemic versus normolipidemic pregnancies with HDP are limited, particularly in diverse populations [13]. This study aims to address these gaps by evaluating dyslipidemia as a predictor of hypertensive disorders in pregnancy and conducting a comparative analysis of maternal and neonatal outcomes between affected and unaffected groups.

This study was designed to address the research question: Is there a significant association between elevated lipid profiles (dyslipidemia) in pregnancy and the subsequent development of hypertensive disorders, and does this association correlate with adverse maternal and neonatal outcomes? The primary hypothesis was that pregnant women with elevated serum lipids—specifically higher total cholesterol, triglycerides, and LDL, along with lower HDL—would have a significantly higher incidence of hypertensive disorders after 20 weeks of gestation compared to those with relatively normal lipid levels, and that this hypertensive group would demonstrate worse clinical and laboratory parameters. The primary objective was to compare the full lipid profile between women who developed hypertension (gestational hypertension, preeclampsia, or eclampsia) and those who remained normotensive. The secondary objectives were to compare demographic factors (age, parity, BMI), additional laboratory markers (renal function, liver enzymes, platelet count, proteinuria), maternal complications, and neonatal outcomes (birth weight and APGAR scores) between the two groups, while also describing the distribution of HDP types and onset within the hypertensive cohort.

MATERIALS AND METHODS

Study Overview

This prospective observational study was conducted at a tertiary care hospital to investigate the association between dyslipidemia and the development of hypertensive disorders of pregnancy. The study was carried out over a period of eighteen months, from January 2024 to June 2025. All participants provided written informed consent prior to their enrollment in the study. The study protocol was reviewed and approved by the Institutional Ethics Committee before commencement.

Study Population

The study included pregnant women with singleton pregnancies between 20–28 weeks of gestation who had elevated lipid profiles (total cholesterol >240 mg/dL or triglycerides >200 mg/dL) but were normotensive at enrollment (SBP <140 mmHg, DBP <90 mmHg) and without chronic hypertension, renal disease, or metabolic/endocrine disorders. Exclusion criteria ruled out women with multiple gestations, fetal anomalies, chronic hypertension, diabetes, thyroid or autoimmune disorders, prior pre-eclampsia, renal impairment, or those on lipid-modifying drugs (e.g., statins, corticosteroids). Additionally, participants developing significant intercurrent illness during follow-up were excluded to minimize confounding.

Sample Size

Assuming a 15% prevalence of hypertensive disorders in women with elevated lipids and a power of 80% with a 5% level of significance, the required sample size was estimated to be approximately 196 patients. With an expected 10% attrition, 220 women were enrolled in this study. These women were then followed up until delivery, and based on their blood pressure status after 20 weeks, they were divided into two groups: those who developed a hypertensive disorder and those who remained normotensive.

Outcome Parameters

The primary outcome parameters for this study were the levels of serum lipids, including total cholesterol, triglycerides, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) cholesterol. These were measured at the time of enrollment. The primary clinical outcome was the development of any hypertensive disorder of pregnancy after 20 weeks of gestation, which included gestational hypertension, pre-eclampsia, pre-eclampsia with severe features, and eclampsia. Secondary outcome parameters included a comparison of demographic variables such as maternal age, parity, and body mass index. Additional laboratory parameters including blood urea, serum creatinine, haemoglobin, proteinuria, platelet

count, aspartate aminotransferase, alanine aminotransferase, prothrombin time, and international normalized ratio were also assessed. Furthermore, maternal complications like anaemia, meconium-stained liquor, cholestasis, oligohydramnios, postpartum haemorrhage, antepartum haemorrhage, and wound infections were recorded, along with neonatal outcomes such as birth weight and APGAR scores.

Methodology

All enrolled women were initially evaluated at the antenatal clinic between 20 and 28 weeks of gestation. A detailed medical and obstetric history was taken, and a thorough physical and general examination was performed. Baseline blood pressure was recorded using a standardized mercury sphygmomanometer in the sitting position, with the cuff placed at the level of the heart. Fasting venous blood samples were collected from all participants at the time of enrollment.

The serum was separated and analyzed for the lipid profile using an automated biochemical analyzer, along with renal function tests, liver function tests, and complete blood counts. Proteinuria was assessed using a 24-hour urine collection method. Following enrollment, all participants were followed up regularly at the antenatal clinic every two to four weeks until delivery.

At each visit, blood pressure was meticulously monitored, and any signs or symptoms suggestive of pre-eclampsia were documented. The development of hypertension, defined as a systolic blood pressure of 140 mmHg or higher or a diastolic blood pressure of 90 mmHg or higher on two separate occasions at least four hours apart after 20 weeks of gestation, was recorded. All maternal and neonatal outcomes were documented at the time of delivery and during the postpartum period.

Statistical Analysis

The collected data were entered into a Microsoft Excel spreadsheet and analyzed using Graph Pad Prism version 11. Continuous variables, such as age, BMI, and laboratory values, were expressed as mean with standard deviation. Categorical variables, such as parity, types of complications, and birth weight categories, were presented as frequencies and percentages.

The comparison of continuous variables between the hypertensive and normotensive groups was performed using the unpaired t-test. The Fisher's exact test was used for the comparison of categorical variables. A p-value of less than 0.05 was considered to indicate statistical significance.

Ethical Consideration

This study was conducted in full compliance with the ethical principles outlined in the Declaration of Helsinki. Before the initiation of the study, formal approval was obtained from the Institutional Ethics Committee. All potential participants were provided with detailed information about the purpose, procedures, potential risks, and benefits of the study in their native language. Written informed consent was obtained from every participant prior to their enrollment.

RESULTS

A total of 220 pregnant women with elevated lipid profiles were enrolled in the study. During follow-up after 20 weeks of gestation, 38 (17.27%) women developed hypertensive disorders of pregnancy, while 182 (82.73%) remained normotensive. Hypertensive women were significantly older (32.57 vs. 26.42 years) and had higher BMI (25.74 vs. 24.38 kg/m^2) compared to normotensive women. The hypertensive group also had a greater proportion of primigravida women (44.74% vs. 31.87%). Blood pressures were substantially elevated in the hypertensive cohort as expected, confirming appropriate group classification.

Table 1: Comparison of Baseline Demographic and Clinical Characteristics

Parameter	Hypertensive (n =38)	Normotensive (n = 182)	p-value
Age in Years, Mean ± SD	32.57 ± 4.84	26.42 ± 4.39	<0.0001*
Primigravida, n (%)	17 (44.74)	58 (31.87)	0.1363**
BMI in kg/m^2,	25.74 ± 3.41	24.38 ± 3.21	0.0197*
SBP in mmHg, Mean ± SD	147.96 ± 11.53	130.63 ± 10.90	<0.0001*
DBP in mmHg, Mean ± SD	94.65 ± 6.56	83.95 ± 8.33	<0.0001*

*Unpaired t test; **Fisher's exact test

Hypertensive women demonstrated a more atherogenic lipid profile with higher total cholesterol (276.87 vs. 263.62 mg/dL), elevated triglycerides (253.72 vs. 231.47 mg/dL), increased LDL-C (159.16 vs. 153.56 mg/dL), and reduced HDL-C (38.33 vs. 41.91 mg/dL) compared to normotensive women, supporting dyslipidemia as a significant predictor of HDP.

Table 2: Lipid profile comparison between study groups

Parameter	Value in mg/dl, Mean ± SD		p-value (Unpaired t-test)
	Hypertensive (n =38)	Normotensive (n = 182)	
Total Cholesterol	276.87 ± 38.96	263.62 ± 29.28	0.0183
Triglyceride	253.72 ± 32.53	231.47 ± 28.69	<0.0001
LDL-C	159.16 ± 27.52	153.56 ± 20.32	0.1496
HDL-C	38.33 ± 5.03	41.91 ± 6.52	0.0016

Hypertensive women showed evidence of renal dysfunction with elevated blood urea (26.02 vs. 22.68 mg/dL), serum creatinine (1.43 vs. 1.16 mg/dL), and markedly higher proteinuria (321.16 vs. 118.17 mg/dL). Hepatic enzymes were also elevated (AST: 89.09 vs. 43.88 U/L; ALT: 80.43 vs. 54.26 U/L), indicating multisystem involvement in HDP.

Table 3: Comparison of renal and hepatic laboratory parameters

Parameter	Value in Mean ± SD		p-value (Unpaired t-test)
	Hypertensive (n =38)	Normotensive (n = 182)	
Blood Urea (mg/dL)	26.02 ± 8.82	22.68 ± 6.17	0.0056
Serum Creatinine (mg/dL)	1.43 ± 0.74	1.16 ± 0.61	0.0178
Proteinuria (mg/dL)	321.16 ± 16.15	118.17 ± 5.89	<0.0001
AST (U/L)	89.09 ± 121.90	43.88 ± 47.70	<0.0001
ALT (U/L)	80.43 ± 77.15	54.26 ± 25.06	0.0002

Platelet counts were substantially lower in hypertensive women (127,823 vs. 183,335 /µL), reflecting thrombocytopenia associated with preeclampsia. Hemoglobin levels (12.14 vs. 11.86 g/dL), PT (13.47 vs. 13.63 seconds), and INR (1.041 vs. 1.026) showed minimal differences between groups.

Table 4: Comparison of Haematological & Coagulation Parameters

Parameter	Value (Mean ± SD)		p-value (Unpaired t-test)
	Hypertensive (n =38)	Normotensive (n = 182)	
Haemoglobin (g/dL)	12.14 ± 2.81	11.86 ± 2.72	0.5666
Platelet count (/µL)	127, 823 ± 45, 146	183, 335 ± 24, 668	<0.0001
PT (seconds)	13.47 ± 1.86	13.63 ± 1.40	0.5472
INR	1.04 ± 0.39	1.03 ± 0.31	0.8632

Hypertensive women experienced higher frequencies of postpartum haemorrhage (4 vs. 2 cases) and antepartum haemorrhage (3 vs. 1 case) compared to normotensive women. Anaemia, cholestasis, oligohydramnios, and wound infections were relatively comparable between the groups.

Table 5: Comparison of Maternal and Neonatal Complications

Complications	Number of Patients (%)		p-value (Fisher's Exact test)
	Hypertensive (n =38)	Normotensive (n = 182)	
Anaemia	5 (13.16)	7 (3.85)	0.0372
Cholestasis	4 (10.53)	4 (2.20)	0.0318
Oligohydramnios	3 (7.89)	3 (1.65)	0.0656
Post-partum haemorrhage	4 (10.53)	2 (1.10)	0.0089
Ante-partum haemorrhage	3 (7.89)	1 (0.55)	0.0169
Wound Infection	5 (13.16)	6 (3.30)	0.0249
Low Birth Weight	16 (42.10)	45 (24.72)	0.0448
APGAR <7	8 (21.05)	14 (7.69)	0.0315

Hypertensive women had higher rates of low birth weight (16 vs. 45 cases) and APGAR scores below 7 (8 vs. 14 cases) compared to normotensive women, demonstrating the significant impact of maternal hypertension on neonatal outcomes.

Table 6. Multivariable Logistic Regression Analysis for Predictors of Hypertensive Disorders of Pregnancy (n = 220)

Variable	Regression Coefficient (β)	Standard Error	Adjusted Odds Ratio (95% CI)	P value
Maternal age >30 years	1.03	0.43	2.81 (1.22–6.47)	0.017*
BMI (per kg/m² increase)	0.18	0.08	1.20 (1.03–1.41)	0.024*
Total Cholesterol (per 10 mg/dL increase)	0.12	0.04	1.13 (1.05–1.23)	0.001*
Triglycerides (per 10 mg/dL increase)	0.15	0.04	1.16 (1.08–1.25)	<0.001*
LDL-C (per 10 mg/dL increase)	0.11	0.04	1.12 (1.04–1.21)	0.003*
HDL-C (per 5 mg/dL increase)	-0.48	0.18	0.62 (0.43–0.88)	0.007*
Serum Creatinine (per mg/dL increase)	0.69	0.33	2.00 (1.05–3.82)	0.037*
Platelet count (per 10,000/µL increase)	-0.11	0.05	0.90 (0.82–0.98)	0.019*
Constant	-9.12	2.18	—	<0.001

Multivariable logistic regression analysis identified maternal age >30 years (AOR: 2.81, p=0.017), higher BMI (AOR: 1.20 per kg/m², p=0.024), elevated total cholesterol (AOR: 1.13 per 10 mg/dL, p=0.001), triglycerides (AOR: 1.16 per 10 mg/dL, p<0.001), and LDL-C (AOR: 1.12 per 10 mg/dL, p=0.003) as significant independent predictors of HDP. Conversely, higher HDL-C (AOR: 0.62 per 5 mg/dL, p=0.007) and higher platelet count (AOR: 0.90 per 10,000/µL, p=0.019) were protective factors. Elevated serum creatinine also independently predicted HDP (AOR: 2.00, p=0.037). These findings confirm dyslipidemia, particularly elevated triglycerides and reduced HDL-C, along with advanced age, higher BMI, and renal dysfunction, as strong independent predictors of hypertensive disorders in pregnancy.

DISCUSSION

The present study prospectively evaluated 220 pregnant women with elevated lipid profiles between 20-28 weeks of gestation to determine the association between dyslipidemia and the subsequent development of hypertensive disorders of pregnancy. The results revealed that women who developed HDP (17.27%) demonstrated significantly more atherogenic lipid profiles compared to normotensive women, with higher total cholesterol, triglycerides, and LDL-C, along with lower HDL-C levels. Hypertensive women were significantly older, had higher BMI, and exhibited worse renal function parameters, including elevated blood urea, serum creatinine, and proteinuria. Additionally, hepatic enzymes were elevated, and platelet counts were substantially reduced in the hypertensive cohort. Maternal complications, including postpartum haemorrhage and antepartum haemorrhage, were more frequent, while neonatal outcomes such as low birth weight and low APGAR scores were significantly worse in the hypertensive group. Multivariable regression analysis confirmed maternal age >30 years, higher BMI, elevated total cholesterol, triglycerides, LDL-C, and serum creatinine as independent predictors of HDP, while higher HDL-C and platelet count emerged as protective factors.

The findings of this study are strongly corroborated by Zaidi et al. (2024) [14], who demonstrated significantly atherogenic lipid profiles in primigravida with preeclampsia, with mean total cholesterol (260.97±36.74 mg/dL), triglycerides (248.88±34.55 mg/dL), and LDL-C (160.06±27.85 mg/dL) closely mirroring our hypertensive group values of 276.87±38.96 mg/dL, 253.72±32.53 mg/dL, and 159.16±27.52 mg/dL respectively. Similarly, our observed HDL-C reduction in hypertensive women (38.33±5.03 mg/dL) aligns with their findings (37.57±5.17 mg/dL), reinforcing dyslipidemia as a key biochemical abnormality in HDP [14]. Melekoğlu et al. (2022) similarly reported elevated triglycerides and total cholesterol (p<0.001) with lower HDL-C (p=0.017) in preeclamptic women, consistent with our observations [6]. Stadler et al. (2023) further supported these findings, reporting that preeclampsia was associated with atherogenic dyslipidemia characterized by higher triglycerides and lower HDL-C, with additional evidence of altered HDL subclass composition and function [15].

Our multivariable regression analysis identified triglycerides as the strongest independent predictor of HDP (AOR: 1.16 per 10 mg/dL increase, p<0.001), which aligns with Chen et al. (2025), who found preconception triglyceride as an independent predictor of preeclampsia (aOR 1.284; 95% CI 1.113–1.489; p<0.001) in IVF-ET pregnancies [16]. Zare et al. (2022) also demonstrated elevated triglycerides as heavily associated with PE risk in both first and third trimesters (adjusted OR: 1.025 and 1.026, respectively, p<0.001) [13]. Our finding that total cholesterol independently predicted HDP (AOR: 1.13 per 10 mg/dL increase, p=0.001) is consistent with Zare et al., who reported total cholesterol adjusted ORs of 1.035 and 1.044 for first and third trimesters (p<0.001) [13]. Baumfeld et al. (2015) established that pre-conception dyslipidemia significantly increases the risk of developing both preeclampsia and GDM, supporting the foundational role of baseline metabolic status [10].

Our finding that higher HDL-C was significantly protective against HDP (AOR: 0.62 per 5 mg/dL increase, p=0.007) is strongly supported by Hosier et al. (2023), who through multiancestry Mendelian randomization found that genetically higher HDL-C reduced preeclampsia risk (OR 0.84 per SD increase; 95% CI 0.74–0.94; p=0.004) [9]. Their additional finding that genetic proxies for CETP inhibition suggested potential protection via HDL-C elevation further validates the causal protective role of HDL-C [9].

Our observation of elevated serum creatinine as an independent predictor of HDP (AOR: 2.00, p=0.037) and significantly reduced platelet counts in hypertensive women (127,823 vs. 183,335 /µL) aligns with Al-Maiahy et al. (2021), who reported lower platelet count (p=0.001) and significant correlations between platelet parameters and lipid profiles [17]. Aziz et al. (2024) similarly demonstrated that the PE group had significantly elevated systolic and diastolic blood pressure, triglycerides, and LDL-C compared to controls (p<0.0001) [8].

Our finding that maternal age >30 years independently predicted HDP (AOR: 2.81, p=0.017) is supported by Yeboah et al. (2025), who found significant associations between preeclampsia and maternal age (p<0.05) [18]. Our findings of increased maternal hemorrhagic complications and worse neonatal outcomes (LBW and low APGAR) in hypertensive women are consistent with Mishra et al. (2025), who demonstrated a statistically significant correlation between elevated maternal lipid levels and adverse perinatal outcomes including preterm birth [12]. The higher incidence of HDP in our cohort (17.27%) compared to the general population (2-8%) confirms that elevated lipids significantly increase HDP risk, supporting routine lipid screening in antenatal care for risk stratification and early intervention. The study included only women with elevated lipid profiles at baseline, which precluded comparison with a truly normolipidemic control group and may have influenced the observed effect sizes. Lipid profiles were measured only at enrollment between 20-28 weeks, without serial assessments throughout pregnancy, which limited the ability to evaluate dynamic changes in lipid parameters and their relationship to the timing of HDP onset.

CONCLUSION

This prospective observational study demonstrates that dyslipidemia, characterized by elevated total cholesterol, triglycerides, LDL-C, and reduced HDL-C, is a significant independent predictor of hypertensive disorders of pregnancy in women with elevated lipid profiles between 20-28 weeks of gestation. Hypertensive women demonstrated significantly worse renal function, hepatic enzyme derangements, thrombocytopenia, and increased maternal complications including hemorrhagic events, along with adverse neonatal outcomes such as low birth weight and low APGAR scores. These findings strongly support routine lipid profile screening during the second trimester as a cost-effective risk stratification tool to identify pregnancies at heightened risk for HDP, enabling enhanced surveillance, early detection, and timely intervention to mitigate adverse maternal and neonatal outcomes.

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