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Original Research Article

# Comparison of Lipid Profile Parameters in Gestational Hypertension, Preeclampsia, and Eclampsia with Normal Pregnancy

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**Abstract**

**Background & Objectives:** Hypertensive disorders of pregnancy (HDP) remain a major cause of maternal and fetal morbidity and mortality worldwide. While physiological hyperlipidemia occurs in normal pregnancy under hormonal regulation, defective lipid adaptation can induce oxidative stress and endothelial damage. This study was designed to evaluate serum lipid profile parameters in cases of HDP (gestational hypertension, preeclampsia, and eclampsia) in comparison to normal healthy pregnant controls.

**Materials & Methods:** A prospective case-control study was conducted comprising 200 pregnant women with gestation >20 weeks. The study population was divided into two groups: 100 cases diagnosed with HDP (45 Gestational Hypertension, 50 Preeclampsia, 5 Eclampsia) and 100 age-matched normotensive pregnant controls. Fasting blood samples (12-hour fast) were evaluated for Total Cholesterol (TC), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), Very Low-Density Lipoprotein (VLDL), and Triglycerides (TG). Statistical analysis was carried out using Student's t-test, One-Way ANOVA, and Pearson/Kendall correlation coefficients.

**Results:** The mean serum Total Cholesterol (TC) was significantly elevated in the HDP group compared to controls (237.32 ± 60.73 mg/dL vs. 215.20 ± 43.99 mg/dL, $p < 0.05$). Serum HDL was significantly reduced in cases compared to controls (49.42 ± 10.76 mg/dL vs. 53.99 ± 18.47 mg/dL, $p = 0.034$). The HDL/TC ratio was significantly lower in the hypertensive group (0.22 ± 0.07 vs. 0.26 ± 0.10, $p = 0.002$). A positive correlation was observed between blood pressure (SBP & DBP) and lipid parameters (TC & TG), whereas HDL exhibited a negative correlation with blood pressure. No significant correlation was observed between lipid parameters and the degree of proteinuria.

**Conclusion:** Dyslipidemia characterized by elevated Total Cholesterol and decreased HDL levels plays an essential role in mediating endothelial dysfunction in HDP. Serum lipid profiling during pregnancy may serve as a valuable biochemical tool for early detection and assessment of disease severity in hypertensive pregnancy.

**Keywords:** Hypertensive Disorders of Pregnancy, Preeclampsia, Gestational Hypertension, Eclampsia, Lipid Profile, Total Cholesterol, HDL.

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## Introduction

Hypertensive disorders of pregnancy (HDP) represent a major global clinical challenge, serving as one of the primary components of the classic deadly triad—alongside hemorrhage and infection—that accounts for substantial maternal and perinatal morbidity and mortality worldwide.

Preeclampsia, a principal manifestation within this disease spectrum, is defined as a multisystem disorder of unknown etiology characterized by the new onset of hypertension (systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg) accompanied by significant proteinuria (≥300 mg per 24-hour collection or ≥1+ on dipstick testing) developing after 20 weeks of gestation in previously normotensive and non-proteinuric women. In its most severe and fulminant manifestation, preeclampsia progresses to eclampsia, characterized by the catastrophic onset of generalized tonic-clonic seizures in the absence of pre-existing neurological disease.

Epidemiologically, preeclampsia complicates approximately 3% to 5% of all pregnancies

globally, with a vastly disproportionate burden falling upon low- and middle-income countries. In India, the overall incidence of hypertensive disorders of pregnancy is estimated between 5% and 15%. The disease demonstrates a strong predilection for nulliparous women, among whom the incidence may reach up to 10–15%, compared to approximately 5% in multiparous women. Preeclampsia is directly responsible for over 60,000 maternal deaths annually on a global scale. Furthermore, in developing nations, fetal and perinatal mortality associated with hypertensive gestations remains alarmingly high, exceeding 10% due to complications such as intrauterine growth restriction (IUGR), placental abruption, severe birth asphyxia, and iatrogenic prematurity.

The pathophysiology of preeclampsia has historically been described as a 'disease of theories' due to its complex and multifaceted nature. Current consensus models view preeclampsia as a two-stage disorder originating in the placenta and manifesting systemically in the maternal vascular endothelium. During normal pregnancy, specialized fetal cytotrophoblasts invade the maternal decidua and myometrium, transforming the high-resistance, narrow-caliber maternal spiral arteries into large-caliber, low-resistance capacitance vessels.

This process of 'pseudovasculogenesis' ensures continuous, unimpeded blood flow to the fetoplacental unit. In preeclampsia, however, endovascular trophoblastic invasion is strikingly incomplete and shallow, restricted primarily to the decidual segments. Consequently, maternal spiral arteries retain their smooth muscle coat and high-resistance responsiveness, leading to severe uteroplacental ischemia, hypoxia, and repetitive reperfusion injury. The ischemic placenta reacts by releasing a plethora of antiangiogenic proteins, pro-inflammatory cytokines, microparticles, and biochemical mediators into the maternal systemic circulation. Key among these are soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng), which neutralize vascular endothelial growth factor (VEGF) and transforming growth factor-beta (TGF-β), respectively. The resulting systemic antiangiogenic state triggers widespread maternal endothelial dysfunction, characterized by impaired synthesis of endogenous vasodilators such as prostacyclin (PGI2) and nitric oxide (NO), alongside heightened production of potent vasoconstrictors including thromboxane A2 (TxA2) and endothelin-1.

In recent years, the pivotal role of maternal lipid metabolism in the pathogenesis and systemic endothelial damage of preeclampsia has drawn immense scientific interest. Under normal physiological conditions, pregnancy is characterized by marked, progressive hyperlipidemia driven by elevated levels of estrogen, progesterone, and human placental lactogen. During early gestation, maternal metabolic adaptations favor lipogenesis and fat deposition to construct energy reserves. In late gestation, metabolic control shifts toward lipolysis, accelerating the breakdown of adipose depots to supply the growing fetus with essential fatty acids and glycerol. Crucially, while physiological hyperlipidemia in normal pregnancy is non-atherogenic due to balanced hormonal regulation and robust antioxidant defenses, complicated pregnancies exhibit a fundamental failure in adjusting lipid homeostasis. Pathological dyslipidemia is not merely a secondary epiphenomenon or biomarker of preeclampsia; rather, it is directly involved in driving the disease process. Abnormal circulating concentrations of free fatty acids (FFAs) and triglycerides (TGs) provide abundant substrates for free radical-mediated lipid peroxidation. Reactive oxygen species (ROS) interact with polyunsaturated fatty acids to generate lipid peroxides, which possess a prolonged half-life in the systemic circulation.

Elevated lipid peroxides directly impair endothelial function through several destructive pathways: they selectively inhibit endothelial prostacyclin synthase while simultaneously stimulating platelet thromboxane A2 production, impair endothelial nitric oxide synthase (eNOS) activity, increase microvascular permeability leading to protein leakage and edema, and upregulate vascular cell adhesion molecules (VCAM-1, ICAM-1) that promote leukocyte adhesion and arterial wall inflammation. Moreover, dyslipidemia in preeclampsia is characterized by a shift toward small, dense low-density lipoprotein (sdLDL) particles, which are uniquely susceptible to oxidative modification and readily form atherogenic foam cells. Although altering serum lipid profile parameters during pregnancy offers significant potential for early biochemical prediction and risk stratification of preeclampsia, data comparing full lipid profiles (TC, HDL, LDL, VLDL, TG, and risk ratios) across the complete spectrum of hypertensive pregnancy—from gestational hypertension to severe preeclampsia and eclampsia—remain limited in local populations. Therefore, this prospective case-control study was conducted to comprehensively evaluate and compare serum lipid parameters in women with gestational hypertension, preeclampsia, and eclampsia against age- and parity-matched normotensive pregnant controls, to determine their relationship with blood pressure severity and proteinuria.

## Materials and Methods

**Study Design & Selection Criteria**: This prospective case-control study enrolled a total of 200 pregnant women at the tertiary care teaching

hospital BRIMS Bidar from January 2024 to March 2025. The study criteria were defined as follows:

**Inclusion Criteria:** Singleton pregnancy with gestational age >20 weeks, categorized into normotensive controls (n=100) and diagnosed cases of HDP (n=100).

**Exclusion Criteria:** Pre-existing hypertension prior to 20 weeks, chronic renal, hepatic, or cardiac diseases, pre-existing diabetes mellitus or gestational diabetes, multiple gestation, and molar pregnancy/abruptio placentae.

**Methodology & Laboratory Estimation**: All participants underwent thorough clinical examination, maternal blood pressure assessment, and obstetric ultrasound. Spot urine tests for albuminuria were graded. Following a 12-hour overnight fast, 5 mL of venous blood was collected under aseptic conditions. Serum was separated via centrifugation and analyzed for Total Cholesterol (TC) via CHOD-PAP Enzymatic method, HDL-Cholesterol via direct method, and Triglycerides (TG) via Glycerol Phosphate Oxidase (GPO-PAP) method. VLDL-Cholesterol was calculated as TG / 5, and LDL-Cholesterol was calculated using Friedewald's equation: LDL = TC - HDL - (TG / 5).

**Statistical Analysis:** Continuous data are expressed as Mean ± Standard Deviation (SD). Unpaired Student's t-test was used to evaluate differences between two independent continuous groups. One-Way ANOVA was utilized for multi-group comparisons. Categorical variables were assessed via Chi-Square test. Non-parametric testing (Mann-Whitney U) and Kendall's tau-b correlation coefficients were used where applicable. A p-value < 0.05 was considered statistically significant.

## Results

The mean age was 25.34 ± 3.88 years in cases and 24.11 ± 3.44 years in controls. Nulliparous women accounted for 64% of the hypertensive group, compared to 43% in the control group (p = 0.003). The mean Gestational Age at presentation was 35.86 ± 4.89 weeks in cases compared to 38.13 ± 1.81 weeks in controls (p < 0.05).

**Table 1: Baseline Demographics and Hemodynamic Characteristics**

| Parameter | Cases (n=100) | Controls (n=100) | t-value / $\chi^2$ | p-value |
|---|---|---|---|---|
| Age (years) | 25.34 ± 3.88 | 24.11 ± 3.44 | t = 2.374 | 0.02* |
| Primigravida (%) | 64% | 43% | $\chi^2$ = 8.86 | 0.003* |
| Systolic BP (mmHg) | 153.10 ± 13.12 | 115.34 ± 7.54 | t = 24.95 | < 0.05* |
| Diastolic BP (mmHg) | 99.52 ± 9.27 | 75.52 ± 5.42 | t = 22.35 | < 0.05* |
| Mean Arterial Pressure (mmHg) | 126.31 ± 10.25 | 95.43 ± 5.56 | t = 26.46 | < 0.05* |
| Mean Birth Weight (kg) | 2.64 ± 0.68 | 2.98 ± 0.47 | t = -4.01 | < 0.05* |

*** Statistically significant (p < 0.05)**

Among the 100 case subjects, 50% were diagnosed with Pre-Eclampsia, 45% with Gestational Hypertension, and 5% with Eclampsia. Pedal edema of Grade II was present in 53% of cases. Proteinuria was present as 1+ in 20%, 2+ in 15%, 3+ in 21%, and 4+ in 2% of hypertensive patients.

**Table 2: Comparison of Lipid Parameters between Hypertensives and Controls**

| Lipid Parameter (mg/dL) | Hypertensives (n=100) | Controls (n=100) | t-value | p-value |
|---|---|---|---|---|
| Total Cholesterol (TC) | 237.32 ± 60.73 | 215.20 ± 43.99 | 2.95 | 0.004* |
| HDL-Cholesterol | 49.42 ± 10.76 | 53.99 ± 18.47 | -2.14 | 0.034* |
| LDL-Cholesterol | 124.51 ± 53.68 | 122.51 ± 49.44 | 0.27 | 0.784 |
| VLDL-Cholesterol | 52.96 ± 16.78 | 52.14 ± 15.03 | 0.36 | 0.716 |
| Triglycerides (TG) | 278.60 ± 102.87 | 263.29 ± 81.67 | 1.16 | 0.245 |
| HDL / TC Ratio | 0.22 ± 0.07 | 0.26 ± 0.10 | -3.14 | 0.002* |
| HDL / LDL Ratio | 0.54 ± 0.55 | 0.56 ± 0.42 | — | 0.520 |

*** Statistically significant (p < 0.05)**

**Table 3: One-Way ANOVA Comparison of Total Cholesterol Across Subgroups**

| Subgroup Comparison | Mean Difference | Standard Error | p-value |
|---|---|---|---|
| Pre-Eclampsia vs. Normal Pregnancy | -29.14 | 9.17 | 0.01* |
| Gestational HTN vs. Normal Pregnancy | -17.40 | 9.50 | 0.41 |
| Eclampsia vs. Normal Pregnancy | 5.60 | 24.26 | 1.00 |

*** Statistically significant (p < 0.05)**

**Correlation Analysis**: Blood Pressure Correlation: Systolic Blood Pressure (SBP) demonstrated a significant positive correlation with Total Cholesterol (r = 0.22, p = 0.02) and Triglycerides (r = 0.40). HDL exhibited a negative correlation with both SBP (r = -0.04) and DBP (r = -0.06).

Proteinuria Correlation: Kendall's tau-b rank correlation demonstrated no statistically significant correlation between urine albumin grades and lipid parameters (TC: p=0.35; HDL: p=0.12; TG: p=0.48).

**Discussion**

Hypertensive disorders of pregnancy represent a complex multisystem syndrome characterized by impaired placental perfusion, systemic vascular constriction, and generalized maternal endothelial dysfunction. In the present study, maternal dyslipidemia was clearly demonstrated as a hallmark feature in pregnant women suffering from hypertensive disorders compared to healthy normotensive controls. The findings provide critical insights into the biochemical alterations accompanying gestational hypertension, preeclampsia, and eclampsia.

Primary demographic analysis revealed that 64% of women in the hypertensive group were nulliparous (primigravidae), compared to 43% in the normotensive group ($p = 0.003$). This strong association between primigravidity and hypertensive disorders is thoroughly documented in obstetric literature. Primigravidae are particularly prone to preeclampsia due to lack of prior maternal immune adaptation to paternal fetal alloantigens, leading to an abnormal maternal immune reaction against invasive cytotrophoblasts and subsequent defective spiral artery remodeling.

Serum Total Cholesterol (TC) levels were significantly elevated in the hypertensive cohort compared to normotensive controls ($237.32 \pm 60.73$ mg/dL vs. $215.20 \pm 43.99$ mg/dL, $p = 0.004$). On subgroup analysis using One-Way ANOVA, the increase in total cholesterol was most prominent in preeclamptic women compared to normal pregnant controls (mean difference = 29.14 mg/dL, $p = 0.01$). Elevated total cholesterol promotes oxidative stress by increasing circulating lipoperoxides and lowering total antioxidant capacity.

Excess circulating cholesterol enhances endothelial cell permeability, rendering vascular walls vulnerable to lipid deposition and macrophage uptake. These findings are in strong alignment with previous studies conducted by Sahu et al., Adiga et al., and Mohanty et al., who reported significant hypercholesterolemia in preeclamptic gestations.

High-Density Lipoprotein (HDL) cholesterol serves a vital protective anti-atherogenic role by facilitating reverse cholesterol transport from peripheral tissues back to the liver for excretion and by inhibiting the oxidation of low-density lipoproteins. In our study, serum HDL levels were significantly reduced in hypertensive cases compared to healthy normotensive gestations ($49.42 \pm 10.76$ mg/dL vs. $53.99 \pm 18.47$ mg/dL, $p = 0.034$). Furthermore, the HDL/TC ratio was markedly diminished in the hypertensive group ($0.22 \pm 0.07$ vs. $0.26 \pm 0.10$, $p = 0.002$).

The profound reduction in protective HDL cholesterol in preeclamptic women can be explained by several metabolic mechanisms. In preeclampsia, insulin resistance and altered estrogen levels impair hepatic Apo A-I synthesis and suppress lecithin-cholesterol acyltransferase (LCAT) activity.

Additionally, hypertriglyceridemia drives Cholesteryl Ester Transfer Protein (CETP) activity, which exchanges triglycerides from VLDL for cholesteryl esters in HDL particles. The resulting triglyceride-enriched HDL particles undergo rapid hydrolysis and clearance by hepatic lipase, substantially lowering circulating HDL concentrations. Decreased HDL levels impair endothelial nitric oxide synthase (eNOS) activation and leave vascular endothelium exposed to unchecked oxidative damage. Similar significant reductions in HDL were documented by Carlos Antonio Negrato et al., Q Lei et al., and Begum et al. In the present study, serum Triglycerides (TG) and Very Low-Density Lipoprotein (VLDL) cholesterol levels were elevated in the hypertensive group (TG: $278.60 \pm 102.87$ mg/dL vs. $263.29 \pm 81.67$ mg/dL; VLDL: $52.96 \pm 16.78$ mg/dL vs. $52.14 \pm 15.03$ mg/dL), although the difference did not achieve statistical significance.

Hypertriglyceridemia in preeclampsia is primarily attributable to enhanced hepatic TG synthesis driven by hypoestrogenemia and insulin resistance, combined with reduced clearing activity of peripheral endothelial lipoprotein lipase (LPL). Delayed vascular clearance of TG-rich remnant particles leads to their accumulation in maternal blood, fostering the formation of highly atherogenic small, dense low-density lipoprotein (sdLDL) particles.

Unlike large, buoyant LDL particles, sdLDL easily penetrates the endothelial barrier, possesses a high affinity for arterial proteoglycans, and is highly susceptible to free radical peroxidation. Oxidized sdLDL is avidly scavenged by tissue macrophages to form foam cells, while simultaneously upregulating endothelin-1 and downregulating endothelial prostacyclin production. This shifts the local vascular balance heavily toward intense vasoconstriction and platelet aggregation, directly triggering the clinical manifestations of hypertension and tissue edema. Correlation analysis in our study revealed a significant positive correlation between Systolic Blood Pressure (SBP) and Total Cholesterol ($r = 0.22$, $p = 0.02$) as well as Triglycerides ($r = 0.40$). Conversely, HDL exhibited a consistent negative correlation with both SBP ($r = -0.04$) and Diastolic Blood Pressure

(DBP) (r = -0.06). These correlations indicate that as blood pressure escalates, lipid abnormalities become progressively more exaggerated. Interestingly, Kendall's tau-b rank correlation showed no significant correlation between urine albumin severity and individual lipid profile values, indicating that dyslipidemia is primarily driven by systemic vascular endothelial injury and hepatic metabolic dysregulation rather than secondary to renal protein loss. Clinical and Perinatal Outcomes: Infant birth weights were significantly lower in the hypertensive group compared to the control group (2.64 ± 0.68 kg vs. 2.98 ± 0.47 kg, $p < 0.05$). This reduction in birth weight reflects severe uteroplacental insufficiency, structural spiral artery narrowing, and early delivery requirements (35.86 weeks in cases vs. 38.13 weeks in controls) inherent to preeclampsia.

In summary, pathological dyslipidemia characterized by elevated total cholesterol, suppressed HDL levels, reduced HDL/TC ratios, and hypertriglyceridemia plays a pivotal role in the pathophysiological cascade of hypertensive disorders of pregnancy. Monitoring maternal lipid profiles during early and mid-gestation provides a valuable, accessible biochemical tool to identify high-risk pregnancies, assess disease severity, and implement timely clinical management strategies to improve maternal and neonatal outcomes.

## Conclusion

This study demonstrates that hypertensive disorders of pregnancy are associated with significant pathological dyslipidemia, characterized by elevated Total Cholesterol and decreased HDL-C levels, alongside reduced HDL/TC ratios. The altered lipid parameters correlate directly with systemic blood pressure elevations and play a vital role in mediating vascular endothelial damage. Incorporating fasting serum lipid profile screening into routine antenatal evaluation can assist clinicians in early risk stratification, monitoring disease progression, and initiating timely therapeutic interventions to minimize maternal and fetal complications.

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