Section: Biochemistry

Original Research Article

ATHEROGENIC DYSLIPIDAEMIA IN PREECLAMPSIA: A COMPARATIVE CROSS-SECTIONAL STUDY OF THE SERUM LIPID PROFILE

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ABSTRACT

Background: Preeclampsia is a major cause of maternal and perinatal morbidity and mortality, in which endothelial dysfunction, oxidative stress and a heightened inflammatory response are central. Altered lipid metabolism (dyslipidaemia) is increasingly recognised as a contributor to this endothelial injury. This study compared the serum lipid profile of preeclamptic women with that of normotensive pregnant women.

Materials and Methods: An analytical cross-sectional study was conducted over one year (September 2023–September 2024) in the Department of Biochemistry in collaboration with the Department of Obstetrics & Gynaecology, Government General Hospital, Kurnool. Using stratified random sampling, 120 pregnant women (>20 weeks gestation, 19–35 years) were enrolled—60 preeclamptic cases and 60 normotensive controls. Total cholesterol, triglycerides, LDL-C, HDL-C and VLDL-C were estimated by enzymatic methods on a Beckman Coulter AU480 analyser. Groups were compared using the independent samples t-test; associations with blood pressure were assessed by Pearson's correlation.

Results: Preeclamptic women had significantly higher total cholesterol (223.1 ± 62.1 vs 193.5 ± 34.7 mg/dL; $p = 0.002$), triglycerides (263.8 ± 80.5 vs 197.9 ± 76.0 mg/dL; $p < 0.001$) and VLDL-C (55.9 ± 25.0 vs 39.7 ± 15.2 mg/dL; $p < 0.001$). LDL-C was higher but not statistically significant (112.4 ± 47.7 vs 99.0 ± 26.9 mg/dL; $p = 0.059$), and HDL-C did not differ between groups (55.6 ± 14.5 vs 55.8 ± 9.1 mg/dL; $p = 0.18$). High triglycerides (≥200 mg/dL) were seen in 78.3% of cases versus 46.8% of controls, and high VLDL-C (>50 mg/dL) in 55% versus 26.8%. Total cholesterol and triglycerides correlated positively with blood pressure ($p < 0.05$).

Conclusion: Preeclampsia is characterised by an atherogenic dyslipidaemia—raised triglycerides, VLDL-C and total cholesterol—that parallels the severity of hypertension, while HDL-C and LDL-C are relatively preserved. Serum lipid profiling may aid risk stratification when combined with clinical assessment. Larger prospective studies with population-specific cut-offs are warranted.

Keywords: Preeclampsia, Dyslipidaemia, Serum lipid profile, Triglycerides, VLDL-cholesterol, Pregnancy hypertension.

INTRODUCTION

Hypertensive disorders of pregnancy are among the foremost causes of maternal and perinatal death worldwide. Preeclampsia—new-onset hypertension (systolic ≥140 mmHg or diastolic ≥90 mmHg) developing after 20 weeks of gestation, usually with proteinuria or other evidence of end-organ dysfunction—complicates an estimated 2–8% of pregnancies globally.[8] In India, its prevalence

ranges from about 3.8% to 10%, and it contributes to roughly 8–10% of maternal deaths.

Although the precise aetiology remains incompletely understood, defective trophoblastic invasion, poor placental perfusion, endothelial dysfunction, oxidative stress and a heightened systemic inflammatory response are recognised as key mechanisms. Normal pregnancy is itself accompanied by a physiological, oestrogen-driven rise in circulating lipids that supports foetal growth and steroidogenesis. In preeclampsia, however, this adaptive hyperlipidaemia appears to be exaggerated into a frankly atherogenic pattern—rising triglycerides and very-low-density lipoprotein (VLDL) with variable changes in cholesterol fractions.

Excess triglyceride-rich lipoproteins and small dense LDL can accumulate in the vascular wall, promote the generation of reactive oxygen species and impair endothelial nitric-oxide function, thereby amplifying the endothelial dysfunction that underlies preeclampsia. Several investigators have reported significantly higher triglycerides, cholesterol and LDL, with lower HDL, in preeclamptic women, and some have proposed the lipid profile as a marker of disease presence and severity.[1–5] The present study was therefore undertaken to estimate the serum lipid profile in preeclamptic women, to compare it with normotensive pregnant women, and to examine its relationship with blood pressure.

MATERIALS AND METHODS

Study Design and Setting: This analytical cross-sectional study was carried out over one year (September 2023–September 2024) in the 24-hour clinical laboratory of the Department of Biochemistry, in collaboration with the Department of Obstetrics & Gynaecology, Government General Hospital, Kurnool. Ethical committee approval was obtained, and written informed consent was taken from every participant in her local language (Telugu).

Participants: A total of 120 singleton pregnant women aged 19–35 years and beyond 20 weeks of gestation were recruited by stratified random sampling into two equal strata: 60 preeclamptic cases (SBP ≥140 and/or DBP ≥90 mmHg on two occasions six hours apart, with or without proteinuria) and 60 normotensive controls. Women with chronic or secondary hypertension, chronic liver or kidney disease, diabetes mellitus, gout or other metabolic disorders were excluded. The sample size of 120 was derived from $n = Z^2p(1-p)/d^2$ ($Z = 1.96$, $p = 0.5$, $d = 0.09$).

Laboratory analysis: A 5 mL fasting venous sample was collected under aseptic conditions and centrifuged, and the serum was analysed on a fully automated Beckman Coulter AU480 analyser. Total cholesterol was measured by the enzymatic cholesterol oxidase–peroxidase (CHOD-PAP) method, triglycerides by the enzymatic glycerol-phosphate-oxidase (GPO-PAP) method, and HDL-cholesterol by a direct enzymatic method. VLDL-cholesterol and LDL-cholesterol were derived using the Friedewald relationship. Blood pressure was recorded by standardised sphygmomanometry, with diastolic pressure taken at Korotkoff phase V.

Statistical analysis: Data were expressed as mean ± standard deviation. The Shapiro–Wilk test was used to assess normality. Group means were compared using the independent samples t-test, and the association between lipid parameters and blood pressure was assessed using Pearson's correlation coefficient. A p-value < 0.05 was considered statistically significant. Analyses were performed in SPSS.

RESULTS

The two groups were comparable in gestational age. A full comparison of the lipid parameters is presented in [Table 1]. Total cholesterol, triglycerides and VLDL-cholesterol were all significantly higher in preeclamptic women ($p < 0.05$ for each), whereas the difference in LDL-cholesterol did not reach significance and HDL-cholesterol was essentially unchanged between the groups.

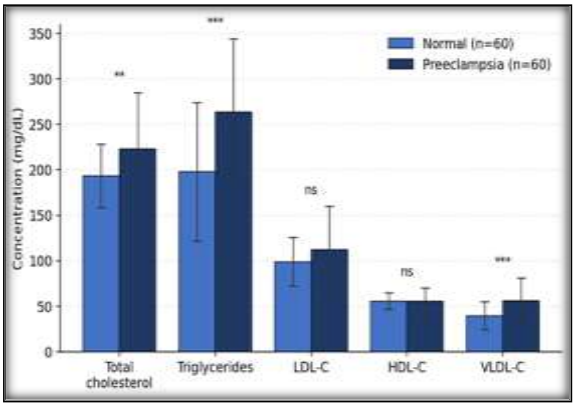

Figure 1: Mean serum lipid values (± SD) in normotensive and preeclamptic women. Total cholesterol, triglycerides and VLDL-C were significantly higher in preeclampsia (p < 0.01, ***p < 0.001); LDL-C and HDL-C did not differ significantly (ns).**

The categorical distribution of lipid abnormalities [Table 2] reinforced this pattern. High triglycerides and high VLDL-cholesterol predominated among cases, and low HDL-cholesterol (<40 mg/dL) was confined to the preeclampsia group, whereas nearly a third of controls maintained normal triglyceride and VLDL levels.

Table 1: Comparison of serum lipid parameters (mean ± SD) between normotensive and preeclamptic women (independent samples t-test, df = 118)

Parameter	Normal (n=60)	Preeclampsia (n=60)	p-value
Total cholesterol (mg/dL)	193.5 ± 34.7	223.1 ± 62.1	0.002
Triglycerides (mg/dL)	197.9 ± 76.0	263.8 ± 80.5	<0.001
LDL-C (mg/dL)	99.0 ± 26.9	112.4 ± 47.7	0.059
HDL-C (mg/dL)	55.8 ± 9.1	55.6 ± 14.5	0.18
VLDL-C (mg/dL)	39.7 ± 15.2	55.9 ± 25.0	<0.001

Table 2: Distribution (%) of abnormal lipid categories among normotensive and preeclamptic women

Lipid category	Normal (n=60)	Preeclampsia (n=60)
Total cholesterol ≥240 mg/dL (high)	11.8%	30.0%
Triglycerides ≥200 mg/dL (high)	46.8%	78.3%
LDL-C ≥160 mg/dL (high)	6.4%	11.7%
HDL-C <40 mg/dL (low)	0%	16.6%
VLDL-C >50 mg/dL (high)	26.8%	55.0%

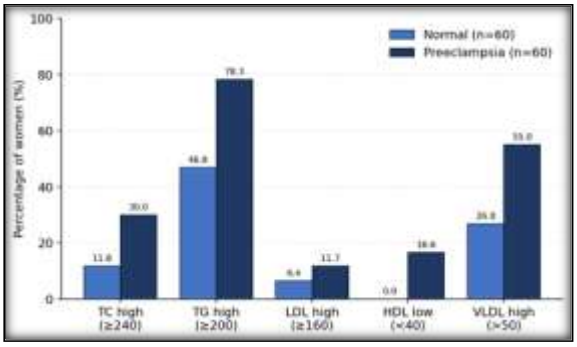

Figure 2: Proportion (%) of women with abnormal lipid values in each group. High triglycerides and high VLDL-C predominated in preeclampsia, and low HDL-C was confined to the preeclampsia group.

On quantitative comparison, mean triglycerides in preeclamptic women (263.8 ± 80.5 mg/dL) were about a third higher than in controls (197.9 ± 76.0 mg/dL; $t = -4.11$, $p < 0.001$), and mean VLDL-cholesterol was correspondingly raised (55.9 ± 25.0 vs 39.7 ± 15.2 mg/dL; $t = -4.27$, $p < 0.001$). Total cholesterol was also significantly higher (223.1 ± 62.1 vs 193.5 ± 34.7 mg/dL; $t = -3.22$, $p = 0.002$). LDL-cholesterol showed a higher mean in cases but did not reach significance ($t = -1.90$, $p = 0.059$), while HDL-cholesterol was virtually identical in the two groups ($t = 0.08$, $p = 0.18$). Total cholesterol and triglycerides correlated positively with systolic and diastolic blood pressure ($p < 0.05$), indicating that the atherogenic shift intensified with the severity of hypertension; LDL-C and HDL-C showed no significant correlation with blood pressure.

DISCUSSION

This study demonstrates a distinct atherogenic dyslipidaemia in preeclampsia. Compared with normotensive pregnant controls, preeclamptic women had significantly higher triglycerides, VLDL-cholesterol and total cholesterol, while HDL-cholesterol was preserved and LDL-cholesterol rose only modestly. The predominance of hypertriglyceridaemia—present in more than three-quarters of cases—together with the positive correlation of triglycerides and cholesterol with blood pressure, points to triglyceride-rich lipoproteins as the most consistent lipid disturbance in this disorder.

The findings are concordant with the wider literature. Enaruna et al. (2014) reported significant differences in triglycerides, LDL and HDL between preeclamptics and normotensive controls,[1] and Agarwal et al. (2014) found significantly raised total cholesterol, LDL and VLDL in third-trimester nulliparous preeclamptics.[2] Kumari et al. (2023) and Salma (2022) similarly documented significantly higher total cholesterol, triglycerides and LDL, with lower HDL, in preeclampsia,[3,4] while a meta-analysis by Spracklen et al. and later Mendelian-randomisation work by Hosier et al. support a causal contribution of maternal hyperlipidaemia to preeclampsia risk.[5,7] The relative preservation of HDL and the non-significant LDL change in the present cohort are consistent with reports that triglyceride and VLDL elevations are the earliest and most reproducible lipid abnormalities of the disorder.

Mechanistically, the exaggerated hypertriglyceridaemia of preeclampsia deposits triglyceride in endothelial and other tissues and favours the formation of small, dense, readily oxidised LDL particles. The resulting oxidative stress impairs endothelial nitric-oxide-dependent vasodilation, promotes a prothrombotic, vasoconstrictive state and contributes to the hypertension and end-organ injury that define the syndrome. This provides a plausible link between the lipid profile observed here and the clinical severity of disease.

Certain limitations should be acknowledged. The cross-sectional, single-centre design precludes inference of causality, and lipid values are influenced by gestational age, diet, body-mass index and the physiological hyperlipidaemia of normal pregnancy—factors that themselves contributed to the high proportion of controls with borderline or raised triglycerides. Lipid measurements are best interpreted alongside clinical assessment and other biochemical markers, and population-specific

reference values are needed for pregnant women in this region.

CONCLUSION

Preeclampsia is associated with a significant atherogenic dyslipidaemia—raised triglycerides, VLDL-cholesterol and total cholesterol—that rises with the severity of hypertension, while HDL-cholesterol and LDL-cholesterol remain relatively unchanged. Measuring the serum lipid profile, particularly triglycerides and VLDL, may aid risk stratification when integrated with clinical evaluation and other laboratory parameters. Larger, multi-centre prospective studies with population-specific cut-off values are recommended to define its predictive and clinical utility.

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