

A STUDY OF FASTING SERUM LIPID PROFILE IN THE SECOND TRIMESTER AND ITS ASSOCIATION WITH PRE-ECLAMPSIA AND MATERNAL AND FETAL OUTCOMES

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ABSTRACT

Background: Preeclampsia is a major cause of maternal and perinatal morbidity and mortality. Identification of simple and inexpensive biomarkers during pregnancy may facilitate early recognition of women at risk. The aim and objective is to evaluate the association between fasting serum lipid profile measured during the second trimester (20–22 weeks of gestation) and the development of preeclampsia. **Materials and Methods:** A prospective cohort study was conducted among 120 singleton pregnant women at 20–22 weeks of gestation in a tertiary care hospital. Fasting serum lipid profile (TC, TG, LDL, HDL, and VLDL) was measured, and participants were followed until delivery to assess maternal and fetal outcomes. Data were analysed using the independent sample t-test and chi-square test, with p-value<0.05 considered statistically significant. **Result:** Women with preeclampsia had significantly higher mean TC (293.95 vs. 199.40 mg/dL), TG (258.90 vs. 208.08 mg/dL), LDL (132.33 vs. 109.70 mg/dL), and VLDL (29.98 vs. 19.83 mg/dL), whereas HDL was significantly lower (43.45 vs. 47.98 mg/dL) than women without preeclampsia (all p≤0.005). Elevated TC and TG were observed in all non-severe (n=42) and severe (n=18) preeclampsia cases. Women with preeclampsia had higher rates of misoprostol-induced delivery (23), emergency LSCS (17), abruptio placenta (8), eclampsia (6), preterm birth (27), and intrauterine growth restriction (15) than women without preeclampsia. **Conclusion:** Second-trimester fasting serum lipid profile showed significant associations with preeclampsia, disease severity, and adverse maternal and fetal outcomes. It may serve as a useful marker for identifying women at increased risk and facilitate closer antenatal surveillance.

INTRODUCTION

Preeclampsia is a multisystem disorder unique to pregnancy and remains one of the leading causes of maternal and perinatal morbidity and mortality worldwide. It is characterized by new-onset hypertension after 20 weeks of gestation, often accompanied by proteinuria or evidence of maternal organ dysfunction. Globally, preeclampsia affects approximately 2–8% of all pregnancies and accounts for nearly 10–15% of maternal deaths. In India, the reported prevalence ranges from 5% to 15%, making it a major public health concern and a significant contributor to adverse maternal and neonatal outcomes.^[1-4]

The exact pathogenesis of preeclampsia is not completely understood; however, abnormal placentation, endothelial dysfunction, oxidative stress, and exaggerated inflammatory responses are

considered key mechanisms. These changes lead to impaired uteroplacental perfusion and widespread vascular injury, increasing the risk of maternal complications such as eclampsia, placental abruption, HELLP syndrome, and postpartum haemorrhage. Fetal complications include intrauterine growth restriction, preterm birth, low birth weight, fetal distress, stillbirth, and increased neonatal intensive care unit admission.^[4,5]

Pregnancy is associated with physiological alterations in lipid metabolism to meet the nutritional demands of the growing fetus. Serum triglycerides, total cholesterol, and lipoprotein concentrations progressively increase as gestation advances. In women who subsequently develop preeclampsia, these physiological changes become exaggerated, resulting in dyslipidaemia characterized by elevated triglycerides, total cholesterol, low-density lipoprotein (LDL), and very low-density lipoprotein

(VLDL), along with reduced high-density lipoprotein (HDL). These lipid abnormalities may contribute to endothelial injury and placental dysfunction, thereby increasing the risk of preeclampsia.^[6-8]

Several biochemical and biophysical markers have been investigated for the early prediction of preeclampsia. Although uterine artery Doppler, placental biomarkers, and angiogenic factors have shown promising results, their routine use is often limited by cost and availability, particularly in resource-limited settings. In contrast, fasting serum lipid profile is an inexpensive, widely available, and minimally invasive investigation that can be incorporated into routine antenatal care. Identifying abnormal lipid levels during the second trimester may facilitate early recognition of women at increased risk and enable timely surveillance and intervention.^[8,9]

Despite increasing evidence linking maternal dyslipidaemia with preeclampsia, limited data are available regarding the predictive value of fasting serum lipid profile measured during the second trimester in the Indian population. The relationship between lipid profile abnormalities, the severity of preeclampsia, and subsequent maternal and fetal outcomes has not been adequately explored. Therefore, the present study was undertaken to evaluate fasting serum lipid profile during the second trimester as a predictor of preeclampsia, to assess its association with disease severity, and to determine its relationship with maternal and fetal outcomes.

Aims and Objectives

Aims

To evaluate the association between fasting serum lipid profile measured during the second trimester (20–22 weeks of gestation) and the development of preeclampsia.

Objectives

- To evaluate whether fasting serum lipid profile measured during the second trimester (20–22 weeks of gestation) is associated with an increased risk of developing preeclampsia.
- To assess the association of second-trimester fasting serum lipid profile with the severity of preeclampsia and maternal and fetal outcomes.

MATERIALS AND METHODS

This prospective cohort study was conducted in the Department of Obstetrics and Gynaecology, Institute of Social Obstetrics Government Kasturba Gandhi Hospital, under Madras Medical College, Chennai, over a period of one year.

Study population: The study included antenatal women attending the antenatal outpatient department during the second trimester (20–22 weeks of gestation) who fulfilled the eligibility criteria.

Sample Size Calculation

The sample size was calculated using the formula:

$$N = Z^2 P(100-P)/d^2$$

The sample size is calculated based on the

- P- prevalence of preeclampsia as 8%

- d - desired precision $\pm 5\%$

- Z=1.96 at 95% confident interval

Using the formula $N = Z^2 P(100-P)/d^2$

$$N = 1.96 \times 1.96 \times 8 \times 92 / 5 \times 5$$

$$N = 113.0496$$

The Sample size calculated to be 113.04 (rounded off to near 10 value)

Inclusion Criteria

Pregnant women aged 18 years and above with a singleton pregnancy at 20–22 weeks of gestation who were willing to provide written informed consent were included in the study.

Exclusion Criteria

Pregnant women who did not provide informed consent, had a history of chronic hypertension, pre-existing diabetes mellitus, known hyperlipidaemia, multiple pregnancy, or medical disorders such as renal disease, liver disease, or thyroid disorders were excluded from the study.

Methods: Eligible participants were recruited after obtaining Institutional Ethics Committee approval and screened according to the inclusion and exclusion criteria. After obtaining written informed consent, demographic and obstetric information, including age, ethnicity, socioeconomic status, educational level, previous pregnancies, family history of preeclampsia, history of complications in previous pregnancies, dietary habits, and physical activity, were recorded using a structured proforma.

Clinical evaluation was performed during the second trimester (20–22 weeks of gestation). Following an overnight fast, approximately 5 mL of venous blood was collected in plain red vacutainer tubes for fasting serum lipid profile estimation. The lipid parameters analysed included total cholesterol (TC), triglycerides (TG), LDL, HDL, and VLDL.

The suggested cut-off values used for lipid profile assessment during the second trimester were: triglycerides >250 mg/dL, total cholesterol >280 mg/dL, LDL >130 mg/dL, HDL <50 mg/dL, and VLDL >30 mg/dL, which were considered indicative of an increased risk of developing preeclampsia.

Participants were followed prospectively through regular antenatal visits until delivery and subsequently up to 6 weeks postpartum. Blood pressure was measured at each antenatal visit, and urine analysis was performed periodically to detect proteinuria. The gestational age at delivery and indication for delivery were documented.

Maternal outcomes assessed included the development of preeclampsia, which was classified as non-severe or severe according to standard diagnostic criteria, gestational age at onset of preeclampsia, requirement for antihypertensive medication, indication for termination of pregnancy, mode of delivery, and maternal complications including HELLP syndrome, eclampsia, placental abruption, postpartum haemorrhage, and postpartum eclampsia.

Fetal outcomes evaluated included birth weight, preterm birth, intrauterine growth restriction (IUGR),

oligohydramnios, birth asphyxia, intrauterine fetal demise, and stillbirth.

The primary outcome of the study was to determine the predictive value of fasting serum lipid profile measured during the second trimester for the subsequent development of preeclampsia and its association with maternal and fetal outcomes.

Ethical considerations: Institutional Ethics Committee approval was obtained from the Institutional Ethics Committee, Madras Medical College, Chennai (IEC Registration No. ECR/1492/Inst/TN/2021) prior to study initiation. Written informed consent was obtained from all participants.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ standard deviation (SD), and

categorical variables were presented as frequencies and percentages. The independent sample t-test was used to compare continuous variables, while the Chi-square test was used to analyse categorical variables. A p-value of <0.05 was considered statistically significant.

RESULTS

Among the 120 participants, 53 (44.2%) were aged 21–25 years, 77 (64.2%) were primigravida, 61 (50.8%) were overweight, and 64 (53.3%) followed a vegetarian diet. The mean age was 25.14 ± 4.80 years, gestational age was 31.67 ± 5.65 weeks, weight was 65.88 ± 5.54 kg, height was 156.74 ± 7.69 cm, BMI was 26.87 ± 3.19 kg/m², platelet count was $178731.88 \pm 115771.99/\mu\text{L}$, and mean birth weight was 2.69 ± 0.52 kg [Table 1].

Table 1: Baseline demographic and clinical characteristics of the study participants

Variable	Category	n (%)
Age (years)	18–20	23 (19.2%)
	21–25	53 (44.2%)
	26–30	22 (18.3%)
	>30	22 (18.3%)
Parity	Primigravida	77 (64.2%)
	Multigravida	43 (35.8%)
Body Mass Index (BMI)	Normal	36 (30.0%)
	Overweight	61 (50.8%)
	Obese	23 (19.2%)
Dietary habit	Vegetarian	64 (53.3%)
	Mixed	56 (46.7%)

Women with preeclampsia had higher mean age (26.10 vs. 24.18 years, $p=0.028$), SBP (166.20 vs. 123.47 mmHg, $p<0.001$), and DBP (110.13 vs. 79.83 mmHg, $p<0.001$), while platelet count was lower

(77623.32 vs. 279840.45/ μL , $p<0.001$). Gestational age, BMI, and birth weight were comparable between the groups ($p>0.05$) [Table 2].

Table 2: Comparison of maternal characteristics between women with and without preeclampsia

Variable	No preeclampsia	Preeclampsia	p value
Age (years)	24.183 ± 4.0316	26.100 ± 5.3257	0.028
Gestational age (weeks)	31.883 ± 5.5360	31.450 ± 5.8089	0.676
SBP (mmHg)	123.467 ± 20.0317	166.200 ± 5.8536	<0.001
DBP (mmHg)	79.833 ± 14.3977	110.133 ± 3.1914	<0.001
BMI (kg/m ²)	27.01 ± 3.46	26.74 ± 2.91	0.644
Platelet count (/ μL)	279840.45 ± 77413.349	77623.32 ± 15755.306	<0.001
Birth weight (kg)	2.6423 ± 0.54152	2.7370 ± 0.49325	0.319

Mean total cholesterol (293.95 vs. 199.40 mg/dL), triglycerides (258.90 vs. 208.08 mg/dL), LDL (132.33 vs. 109.70 mg/dL), and VLDL (29.98 vs.

19.83 mg/dL) were higher, whereas HDL was lower (43.45 vs. 47.98 mg/dL) in women with preeclampsia (all $p \leq 0.005$) [Table 3].

Table 3: Comparison of fasting lipid profile between women with and without preeclampsia

Lipid parameter	No preeclampsia	Preeclampsia	p value
Total cholesterol (mg/dL)	199.400 ± 57.1276	293.950 ± 10.1304	<0.001
Triglycerides (mg/dL)	208.083 ± 50.7257	258.900 ± 7.8841	<0.001
HDL (mg/dL)	47.983 ± 11.2151	43.450 ± 4.8448	0.005
LDL (mg/dL)	109.700 ± 29.2165	132.333 ± 7.2103	<0.001
VLDL (mg/dL)	19.833 ± 7.3258	29.983 ± 3.0281	<0.001

Abnormal lipid values included elevated total cholesterol in 76 women (63.3%), elevated triglycerides in 92 (76.7%), low HDL in 87 (72.5%), and elevated LDL in 48 (40.0%). Among these, non-

severe and severe preeclampsia cases were 42 (100%) and 18 (100%) for total cholesterol and triglycerides, 37 (88.1%) and 15 (83.3%) for HDL, and 21 (50.0%) and 11 (61.1%) for LDL [Table 4].

Table 4: Association of abnormal lipid parameters with preeclampsia

Lipid parameter	Total abnormal	Without PE	Non-severe	Severe
Total cholesterol ↑	76 (63.3%)	16 (26.7%)	42 (100%)	18 (100%)
Triglycerides ↑	92 (76.7%)	32 (53.3%)	42 (100%)	18 (100%)
HDL ↓	87 (72.5%)	35 (58.3%)	37 (88.1%)	15 (83.3%)
LDL ↑	48 (40.0%)	16 (26.7%)	21 (50.0%)	11 (61.1%)

Elevated total cholesterol and triglycerides were observed in all non-severe (42) and severe (18) preeclampsia cases ($p<0.01$). Reduced HDL was

present in 37 non-severe and 15 severe cases ($p<0.05$), while borderline LDL was observed in 21 non-severe and 11 severe cases ($p<0.05$) [Table 5].

Table 5: Lipid profile categories according to preeclampsia severity

Parameter	Category	No PE	Non-severe	Severe	p value
Total cholesterol	Normal	32 (53.3%)	0	0	<0.01
	Borderline	12 (20.0%)	0	0	
	Elevated	16 (26.7%)	42 (100%)	18 (100%)	
Triglycerides	Normal	8 (13.3%)	0	0	<0.01
	Borderline	20 (33.3%)	0	0	
	Elevated	32 (53.3%)	42 (100%)	18 (100%)	
HDL	Normal	25 (41.7%)	5 (11.9%)	3 (16.7%)	<0.05
	Reduced	35 (58.3%)	37 (88.1%)	15 (83.3%)	
LDL	Normal	44 (73.3%)	21 (50.0%)	7 (38.9%)	<0.05
	Borderline	15 (25.0%)	21 (50.0%)	11 (61.1%)	
	Elevated	1 (1.7%)	0	0	

Among women with preeclampsia, 23 (38.3%) underwent misoprostol-induced delivery, 17 (28.3%) emergency LSCS, 10 (16.7%) elective LSCS, and 10 (16.7%) normal vaginal delivery. Maternal complications included severe anaemia in 7 (11.7%),

abruptio placenta in 8 (13.3%), and eclampsia in 6 (10.0%). Fetal outcomes included 27 (45.0%) preterm births, 15 (25.0%) IUGR cases, and 18 (30.0%) term deliveries [Table 6].

Table 6: Maternal and fetal outcomes according to preeclampsia status

Variable	Category	No PE	PE
Mode of delivery	NVD	26 (43.3%)	10 (16.7%)
	Misoprostol induced	19 (31.7%)	23 (38.3%)
	Emergency LSCS	13 (21.7%)	17 (28.3%)
	Elective LSCS	2 (3.3%)	10 (16.7%)
Maternal complications	Nil	56 (93.3%)	39 (65.0%)
	Severe anaemia	3 (5.0%)	7 (11.7%)
	Abruptio placenta	1 (1.7%)	8 (13.3%)
	Eclampsia	0 (0%)	6 (10.0%)
Fetal outcome	Term	45 (75.0%)	18 (30.0%)
	Preterm	10 (16.7%)	27 (45.0%)
	IUGR	5 (8.3%)	15 (25.0%)

Among women with non-severe and severe preeclampsia, maternal complications included abruptio placenta in 7 (16.7%) and 1 (5.6%), eclampsia in 5 (11.9%) and 1 (5.6%), and PPH in 4

(9.5%) and 3 (16.7%), respectively. Fetal outcomes comprised preterm births in 18 (42.9%) and 9 (50.0%), IUGR in 9 (21.4%) and 6 (33.3%), and term deliveries in 15 (35.7%) and 3 (16.7%) [Table 7].

Table 7: Maternal and fetal outcomes according to preeclampsia severity

Variable	Category	No PE	Non-severe	Severe	p value
Maternal complications	Nil	56 (93.3%)	26 (61.9%)	13 (72.2%)	<0.05*
	Abruptio placenta	1 (1.7%)	7 (16.7%)	1 (5.6%)	
	Eclampsia	0 (0%)	5 (11.9%)	1 (5.6%)	
	PPH	3 (5.0%)	4 (9.5%)	3 (16.7%)	
Fetal outcome	Term	45 (75.0%)	15 (35.7%)	3 (16.7%)	<0.05*
	Preterm	10 (16.7%)	18 (42.9%)	9 (50.0%)	
	IUGR	5 (8.3%)	9 (21.4%)	6 (33.3%)	

DISCUSSION

Preeclampsia remains a major cause of maternal and perinatal morbidity, making early identification important for improving pregnancy outcomes.^[1,2] The present study was undertaken to evaluate the role of fasting serum lipid profile in the second trimester

in predicting preeclampsia, its relationship with disease severity, and maternal and fetal outcomes. The study demonstrated differences in lipid profile parameters between women with and without preeclampsia, along with variations in maternal and fetal outcomes. Maternal dyslipidaemia may contribute to the development of preeclampsia

through several biological mechanisms. Elevated triglycerides and LDL can promote oxidative stress and endothelial dysfunction, while reduced HDL may impair antioxidant and anti-inflammatory functions. These alterations can affect placental vascular development and uteroplacental perfusion, contributing to the development and progression of preeclampsia.^[4-6]

In the present study, the study population predominantly comprised women aged 21–25 years, with most participants being primigravida, overweight, and following a vegetarian diet. Baseline maternal anthropometric and clinical characteristics were documented. Women with preeclampsia differed from those without preeclampsia in terms of maternal age, blood pressure, and platelet count, whereas gestational age, body mass index, and birth weight were comparable between the two groups. Similar findings were reported by Li et al., who observed that women with early-onset preeclampsia had a higher mean maternal age (32.6 ± 4.2 vs. 28.8 ± 3.2 years). However, unlike the present study, they also reported a significantly higher pre-pregnancy BMI (23.2 ± 3.9 vs. 20.4 ± 2.5 kg/m²) and lower neonatal birth weight (1870.6 ± 638.7 vs. 3360.8 ± 387.3 g) among women with early-onset preeclampsia. This difference may be related to differences in study population, disease phenotype, and baseline maternal characteristics.^[10]

Chitteti et al. also observed a significant association between serum triglyceride levels and the development of preeclampsia among women aged 21–29 years ($p=0.001$), in both primigravida ($p=0.000003$) and multigravida ($p=0.0369$), and across BMI categories of 18.5–24.5 kg/m² ($p=0.0017$) and 24.5–29.5 kg/m² ($p=0.0094$).^[11] Nithya reported that most participants were aged 20–25 years, and 47% were primigravidae. Women who developed preeclampsia had significantly higher systolic blood pressure (152.5 ± 9.2 mmHg) and diastolic blood pressure (107.6 ± 4.5 mmHg) than normotensive women (110.9 ± 9.1 mmHg and 72.9 ± 5.9 mmHg, respectively). Women with preeclampsia had a higher body mass index, with most falling into the overweight and class II obesity categories ($p=0.01$).¹² These findings are consistent with previous studies and suggest that maternal demographic characteristics, adiposity, and elevated blood pressure are commonly associated with the development of preeclampsia.

Our findings demonstrated that women with preeclampsia had higher total cholesterol, triglycerides, LDL, and VLDL levels, along with lower HDL levels, than women without preeclampsia. Abnormal lipid parameters, including elevated total cholesterol, triglycerides, and LDL levels and reduced HDL levels, were observed among women with preeclampsia, with lipid profile categories varying according to disease severity. Similar findings were reported by Li et al., who observed significantly higher total cholesterol, triglycerides, and LDL-C, along with lower HDL-C

levels, in women with both early- and late-onset preeclampsia than in healthy pregnant women ($p<0.05$). They further identified elevated triglycerides (OR=2.059, 95% CI: 1.073–3.949) and reduced HDL-C (OR=7.314, 95% CI: 3.593–14.893) as independent predictors of early-onset preeclampsia.¹⁰ Nithya M.P. also reported significantly higher total cholesterol, triglycerides, and LDL-C, together with lower HDL-C levels, in women with preeclampsia than in normotensive women (all $p=0.01$), with more pronounced dyslipidaemia among women with preeclampsia than those with gestational hypertension.^[12]

Gupta et al. demonstrated significantly higher total cholesterol, triglycerides, LDL, and VLDL levels in women with dyslipidaemia and reported a higher incidence of preeclampsia in women with abnormal lipid profiles than in those with normal lipid profiles (27 vs. 8 cases, $p=0.003$).^[13] Qin et al. confirmed that women with preeclampsia consistently exhibited higher total cholesterol, triglycerides, and LDL levels, together with lower HDL levels than healthy pregnant women, supporting the association between maternal dyslipidaemia and preeclampsia.^[14] These findings are consistent with previous evidence and suggest that second-trimester dyslipidaemia is closely associated with the development of preeclampsia and increasing disease severity.

Our findings showed that women with preeclampsia had higher rates of operative delivery, maternal complications, preterm birth, and intrauterine growth restriction, with these adverse outcomes being more frequent in severe than in non-severe preeclampsia. Li et al. similarly reported poorer maternal and neonatal outcomes in women with preeclampsia, particularly early-onset disease, including higher cesarean section rates (92.7%), placental abruption (7.3%), fetal growth restriction (21.8%), lower birth weight (1870.6 ± 638.7 g), earlier delivery (32.1 ± 2.3 weeks), and higher NICU admission (88.0%).¹⁰ Nithya M.P. also observed that emergency lower segment caesarean section was more common among women with preeclampsia (8/11), whereas normal vaginal delivery predominated in normotensive women (42/82).^[12] Yue and Ying demonstrated that abnormal second-trimester maternal lipid levels were significantly associated with adverse pregnancy outcomes, including preeclampsia, postpartum hemorrhage, fetal distress, and macrosomia, with second-trimester BMI, lipoprotein(a), and apolipoprotein-B identified as independent predictors of preeclampsia.^[15] These findings suggest that maternal dyslipidaemia is associated with adverse maternal and fetal outcomes, highlighting the importance of early risk identification and appropriate antenatal management.

Limitations

This study was conducted at a single tertiary care centre with a relatively small sample size, which may limit the generalizability of the findings. In addition, the study assessed the association between second-trimester fasting lipid profile and preeclampsia but

did not evaluate predictive performance using receiver operating characteristic analysis or determine optimal lipid cut-off values.

CONCLUSION

Fasting serum lipid profile measured during the second trimester showed significant differences between women with and without preeclampsia and varied according to disease severity. Women with preeclampsia also experienced poorer maternal and fetal outcomes. These findings suggest that second-trimester fasting lipid profile may serve as a useful marker for identifying women at increased risk of preeclampsia and facilitating closer antenatal surveillance.

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