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Original Article

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Early Second Trimester Serum Lipid Profile as a Predictor of Gestational Hypertension and Preeclampsia: A Prospective Hospital-Based Study

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Abstract

Objective: To evaluate the association between early second-trimester serum lipid concentrations and the subsequent risk of developing gestational hypertension and preeclampsia. ***Design:*** A prospective hospital-based observational study. ***Subjects/Patients:*** A total of 100 antenatal women (both primigravidae and multigravidae) with singleton pregnancies between 14 and 20 weeks of gestation who had no preexisting medical illnesses, with normal baseline blood pressure, were enrolled. ***Methods:*** Serum lipid profiles of participants were measured, and participants were followed until delivery. Based on blood pressure and proteinuria, participants were classified as normotensive, having gestational hypertension, or preeclampsia. Data were statistically analysed. ***Results:*** Among the participants, 82% antenatal women remained normotensive (Group A), 7% developed gestational hypertension (Group B), and 11% developed preeclampsia (Group C). Higher serum lipid profiles were seen in women who subsequently developed hypertensive disorders compared to normotensive women ($p < 0.05$). Elevated lipid levels were more pronounced in preeclamptic women, indicating an atherogenic trend with increasing disease severity. ***Conclusion:*** Overall, the study demonstrated that the association of altered lipid profiles during the early second trimester resulted in increased risk of gestational hypertension and preeclampsia. Prompt recognition of lipid screening is recommended for identifying high-risk pregnancies and enabling timely preventive interventions.

Keywords: ***Dyslipidemia, Endothelial dysfunction, Gestational hypertension, Lipid profile, Pregnancy, singleton pregnancies.***

1. Introduction

Hypertensive disorders of pregnancy, including gestational hypertension (GH) and preeclampsia (PE), constitute one of the leading causes of maternal mortality worldwide. Nearly 5 to 10% of pregnant women have a diagnosis of hypertension, while PE complicates approximately 2–8% of pregnancies [1,2]. A diagnosis is made if hypertension (≥140/90 mmHg) develops after 20 weeks of gestation, regardless of the presence of proteinuria or signs of organ impairment [3]. Various maternal risk factors, including nulliparity, advanced maternal age, obesity, pre-existing hypertension, diabetes mellitus, chronic kidney disease, and a family history of preeclampsia, have been associated with an increased risk of developing hypertensive disorders. This can lead to a heightened risk of cardiovascular disease, and prompt recognition and modification of associated risk factors is crucial.

According to the National High Blood Pressure Education Program (NHBPEP) working Group, hypertensive disorders of pregnancy are categorised into four groups: chronic hypertension, preeclampsia–eclampsia, preeclampsia superimposed on chronic hypertension, and gestational hypertension [4]. Gestational hypertension is defined as systolic blood pressure of ≥140 mm Hg, or a diastolic pressure of ≥ 90 mmHg, on two occasions at least 4 hours apart after 20 weeks of gestation in the absence of proteinuria or features of preeclampsia. Preeclampsia, in contrast, is characterised by hypertension with proteinuria (≥300 mg/24 h or ≥1+ dipstick) or, in its absence, with maternal organ dysfunction such as thrombocytopenia, renal or hepatic impairment, pulmonary edema, or new-onset neurological symptoms [5].

Preeclampsia typically develops from abnormal placentation and inadequate remodelling of spiral arteries, which can lead to placental hypoxia and ischemia. These abnormalities can trigger the release of anti-angiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) and inflammatory cytokines, which result in endothelial dysfunction, oxidative stress, and vasospasm. The endothelial dysfunction may result in hypertension, proteinuria and multi-organ involvement, which are key contributors to preeclampsia [6] Dyslipidemia has been increasingly implicated in this process, as lipid abnormalities can enhance oxidative stress, impair nitric oxide bioavailability, and promote vascular inflammation, further exacerbating endothelial dysfunction [7].

For effective risk stratification, early identification of biomarkers is crucial, but most available predictors show limited accuracy. Emerging evidence suggests that maternal lipid metabolism alterations may play a key role in the pathophysiology and early prediction of hypertensive disorders [8]. Recent meta-analyses provide evidence of consistent lipid abnormalities in hypertensive pregnancies [9-13]. These changes may occur as early as the first or second trimester, preceding clinical manifestation. Despite the growing evidence linking hypertension in pregnancy, the

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clinical utility of lipid parameters as a predictive tool remains underexplored, particularly in the early second trimester. Early diagnosis, during 14 and 20 weeks of gestation, may offer a practical and cost-effective approach for early risk identification, especially in low-resource settings where advanced biomarker assays are not routinely available.

Therefore, the present hospital-based prospective study was conducted to examine the relationship between early second-trimester serum lipid levels and the subsequent risk of gestational hypertension and preeclampsia. It also aimed to compare serum lipid parameters (Triglycerides, Total cholesterol, LDL, HDL, VLDL) among normal pregnant women, those with gestational hypertension, and preeclampsia in primigravidae and multigravidae with singleton pregnancies.

2. Materials and Methods

2.1 Study Design and Ethical Considerations

The present study was conducted in the Department of Obstetrics and Gynaecology, Mydhili Hospital, Gudur, Andhra Pradesh, which caters predominantly to women from the class II socioeconomic group, in rural settings. The number of participants (n=96) was statistically derived based on the sample size calculation. (Annexure I)

2.2 Study Population

A total of 100 pregnant women between 14–20 weeks of gestation were recruited and monitored until delivery. The study population included primigravidae and multigravidae with singleton, uncomplicated pregnancies between 14 and 20 weeks of gestation, confirmed by last menstrual period (LMP) or ultrasound. The inclusion criteria decided were primigravidae and multigravidae women with singleton pregnancies of 14–20 weeks' gestation who were normotensive at recruitment (systolic blood pressure <140 mmHg and diastolic blood pressure <90 mmHg), and free of pre-existing medical disorders. However, women with multiple pregnancies, diabetes mellitus, renal disease, hepatic dysfunction, dyslipidemia, congenital anomalies, or pre-existing hypertension were excluded.

2.3 Data collection

The total study duration was 3 months. Informed consent in English and in the local language in a designated form was obtained from the participants before allocation. Participants were informed regarding the safety of venous blood collection. Data was collected in a pre-designed proforma which included identification data, demographic characteristics, and general physical and obstetrics examination. Maternal height, weight, and body mass index (BMI) in the first trimester, as well as gestational weight gain, were noted from antenatal records.

For biochemical analysis, 5 mL of venous blood was collected from the antecubital vein under aseptic precautions after an overnight fasting period of 12 hours, using a red-capped vacutainer with clot activator. After clotting at room temperature for 30 minutes, samples were centrifuged at 1500 rpm to separate serum, which was analysed using the Siemens Dimension RXL Max system. Serum triglycerides, total cholesterol, and HDL-C were measured by enzymatic methods with Ozosense kits and Med Source Ozone fully automated biochemistry analyser. Serum LDL-C was calculated using the Friedewald formula [14]:

LDL-C = Total cholesterol – (HDL-C + VLDL-C), with VLDL-C = 1/5th of triglycerides

The serial blood pressure measurements were done for all enrolled women until delivery. Based on pregnancy outcomes, participants were classified into three groups: Group A (normotensive), Group B (gestational hypertension), and Group C (preeclampsia). Comparative analysis was performed between groups for maternal age, obstetric score, previous history of pregnancy-induced hypertension (PIH), BMI, and lipid parameters (total cholesterol, HDL-C, LDL-C, VLDL-C, and triglycerides).

2.4 Study analysis

Statistical analysis was carried out using SPSS version 20. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Statistical significance was defined at $p < 0.05$.

3. Results

One hundred antenatal women at 14–20 weeks of gestation were enrolled in the study and followed through to delivery. Based on blood pressure records and proteinuria status, participants were categorised into three groups: 82 women remained normotensive (Group A), 7 developed gestational hypertension (Group B), and 11 developed preeclampsia (Group C), as represented in Table I.

3.1 Demographics of participants

3.1.1 Age

The mean age of all participants was 23.9 ± 3.6 years (range 19–34). Figure 1 represents a histogram that shows that most women were within the 20–25-year age group, with the highest frequency observed between 21–23 years. The age distribution showed a slight right skew, with fewer participants in the younger age group, and a minor peak observed around 34–35 years.

The study population was categorised into three age groups: <25 years, 26–30 years, and >30 years, and their distribution was compared across the study groups, as shown in Figure 2. The majority of normotensive women (68) were below 25 years old, 11 from 26-30 years, and only one woman was older than 30 years. From the gestational hypertensive women group, 2 women were younger than 25 years, and 5 were between 26–30 years. In the preeclampsia group, 2 women were younger than 25 years, 2 were between 26–30 years, and 5 were older than 30 years.

3.1.2 Socioeconomic status

The socioeconomic status of the study population, assessed according to the modified Kuppuswamy's classification, is shown in Table II and Figure 3. More than half of the participants were from Class 4, while 31% belonged to Class III, 10% to Class V, and 7% to Class II, while none were from Class I. Among normotensive women (Group A), the majority were in Class IV (42), while most women with gestational hypertension (Group B) also fell into Class IV (4). In the preeclampsia group, 6 women (Group C) were in Class III, 3 in Class IV, and 1 each in Classes II and V.

3.1.3 Obstetric score

The distribution of the obstetric score among the study population is shown in Table III. Of the 100 antenatal women, 47% were primigravidae (n=47) and 53% were multigravidae (n=53). However, distribution across groups is represented in Figure 4. Group A (Normotensive, n=78) included 36 primigravidae and 47 multigravidae. Group B (Gestational Hypertension, n=7) comprised 6 primigravidae and 1 multigravida. Group C (Preeclampsia, n=11) included 4 primigravidae and 6 multigravidae.

3.1.4 Family history of hypertension

The participants were asked about their family history of hypertension. Of these, 11% had a positive family history, while 89 women (89%) had no such history. (**Figure 5**) Across groups, 7 women in Group A (Normotensive) and 4 women in Group B (Gestational Hypertension) had a positive family history, whereas none in Group C (Preeclampsia) reported a family history of hypertension.

3.2 Clinical parameters

Clinical parameters such as blood pressure measurements (systolic and diastolic) and body mass index (BMI) were compared across the study groups to assess differences and their association with gestational hypertension and preeclampsia.

3.2.1 Blood pressure

Table IV represents the comparison of systolic blood pressure (SBP) and Diastolic blood pressure (DBP) across groups.

The SBP in Group A (Normotensive) was 110.9 ± 9.1 mmHg, in Group B (Gestational Hypertension) was 144.6 ± 6.8 mmHg, and in Group C (Preeclampsia) was 152.5 ± 9.2 mmHg. ANOVA revealed a statistically significant difference in systolic blood pressure across the groups ($p = 0.01$). On comparison of mean values, significantly higher in women from Group B (Gestational Hypertension) and Group C (Preeclampsia) compared to Group A (Normotensive) ($p < 0.05$). In contrast, there was no significant association between preeclampsia and a positive family history of hypertension in this study. The mean DBP in Group A (Normotensive) was 72.9 ± 5.9 mmHg, and in Group B (Gestational Hypertension) was 96.7 ± 2.1 mmHg. However, a higher DBP, 107.6 ± 4.5 mmHg, was observed in Group C (Preeclampsia).

3.2.2 BMI

The distribution of BMI across the study groups is shown in Table V. In Group A (Normotensive, n=82), 55 women had normal BMI (18.5–24.9 kg/m²), 23 were in Class I obesity (25–29.9 kg/m²), and 4 were in Class II obesity (30–39.9 kg/m²). In Group B (Gestational Hypertension, n=7), 1 woman had normal BMI, 5 were in Class I obesity, and 1 was in Class II obesity. In Group C (Preeclampsia, n=11), none had normal BMI; 8 were in Class I obesity and 3 in Class II obesity. No participants in any group were underweight (<18.5 kg/m²) or in Class III/extreme obesity (>40 kg/m²). ANOVA revealed a significant difference in BMI across the groups ($p = 0.01$), with post-hoc analysis showing that women in Groups B and C had significantly higher BMI compared to Group A.

On comparison of the mean level of blood pressure and BMI between the groups, there is a significant difference in blood pressure and BMI in group B and group C compared to group A participants ($p<0.05$)

3.2.3 Lipid profile parameters

Lipid profile parameters, including total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C), and triglycerides (TG), were compared across the three study groups to evaluate their association with gestational hypertension and preeclampsia. (Table VI).

The mean TC level was 167.1 ± 12.9 mg/dL in Group A (Normotensive), 200.0 ± 5.2 mg/dL in Group B (Gestational Hypertension), and 210.6 ± 9.6 mg/dL in Group C (Preeclampsia). ANOVA revealed a statistically significant difference in total cholesterol across the groups ($p = 0.01$), with post-hoc analysis indicating significantly higher cholesterol levels in Groups B and C compared to Group A.

Similarly, HDL-C levels were lower in hypertensive groups, with mean values of 42.7 ± 4.3 mg/dL in Group A, 37.0 ± 4.1 mg/dL in Group B, and 35.2 ± 5.1 mg/dL in Group C ($p = 0.01$), showing a significant reduction in Groups B and C compared to Group A.

LDL-C levels were also significantly different across the groups ($p = 0.01$), with mean values of 98.4 ± 10.2 mg/dL in Group A, 126.4 ± 8.5 mg/dL in Group B, and 127.5 ± 7.1 mg/dL in Group C.

In contrast, VLDL-C did not show a significant difference ($p >0.05$), with mean values of 28.9 ± 3.0 mg/dL, 29.4 ± 1.6 mg/dL, and 29.6 ± 2.8 mg/dL in Groups A, B, and C, respectively.

Triglycerides (TG) were significantly higher in hypertensive groups ($p = 0.01$), with mean values of 139.3 ± 14.4 mg/dL in Group A, 175.6 ± 9.7 mg/dL in Group B, and 184.8 ± 13.1 mg/dL in Group C.

3.2.4 Proteinuria

Proteinuria was evaluated using urine albumin levels and compared across the groups to differentiate between gestational hypertension and preeclampsia.

Urine albumin levels were assessed to evaluate proteinuria in the study population. Out of 100 women, 89 had no proteinuria, while 11 showed positive findings. Among these, 8 women had proteinuria of 1+, and 3 women (3%) had proteinuria of 2+. (Table VII). All women from Groups A and B had negative urine albumin (nil). In contrast, in Group C (Preeclampsia, n=11), proteinuria was present in all participants, with 8 women (72.7%) showing 1+ proteinuria and 3 women (27.3%) showing 2+ proteinuria. (Table VIII). Statistically significant differences in proteinuria distribution across the groups ($p = 0.01$) were seen.

3.3 Mode of Delivery

Pregnancy outcomes, including the mode of delivery (normal vaginal delivery, emergency lower segment caesarean section, and elective caesarean section), were assessed and compared among the study groups.

Out of 100 women, 51 underwent emergency lower segment caesarean section (LSCS), 5 had an elective LSCS, and 44 delivered by normal vaginal delivery (NVD). (Table IX)

When the mode of delivery across the study groups was analysed (**Figure 6**), in Group A (Normotensive, n=82), 42 women had NVD, 36 required emergency LSCS, and 4 underwent elective LSCS. In Group B (Gestational Hypertension, n=7), 2 women delivered vaginally, 4 by emergency LSCS, and 1 by elective LSCS. In Group C (Preeclampsia, n=11), 3 women delivered vaginally and 8 required emergency LSCS. There were no Elective LSCS among Group C.

Table I: Classification of Study Groups Based on Blood Pressure and Proteinuria

Group	No of participants	Category	Definition
Group A	82	Normotensive	SBP<140 mm hg and DBP <90 mm hg
Group B	7	Gestational Hypertension	SBP >140 mm hg and DBP >90 mm hg without proteinuria
Group C	11	Pre-eclampsia	SBP >140 mm hg and DBP >90 mm hg with proteinuria

Table II: Socioeconomic Status of Study Participants

Classes	Frequency	Percent
Class I	0	0
Class II	7	7.0
Class III	31	31.0
Class IV	52	52.0
Class V	10	10.0
Total	100	100.0

Table III: Obstetric Score Distribution

Obstetric score	Frequency	Percent
Multigravida	53	53
Primigravida	47	47
Total	100	100

Table IV: Comparison of SBP and DBP between the groups

	Group A		Group B		Group C		ANOVA (p-value)
	Mean	SD	Mean	SD	Mean	SD	
SBP	110.9	9.1	144.6	6.8	152.5	9.2	0.01*
DBP	72.9	5.9	96.7	2.1	107.6	4.5	0.01

Table V: Comparison of BMI among the groups

	Underweight (BMI <18.5 $Kg/m^{2)}$	Normal BMI (18.5-24.9 $Kg/m^{2)}$	Class I obesity (overweight) (25-29.9 $kg/m^{2)}$	Class II Obesity (30-39.9$kg/m^{2)}$	Class III Extreme obesity (>40 $kg/m^{2)}$
Group A (Normotensive)	0	55	23	4	0
Group B (Gestational Hypertension)	0	1	5	1	0
Group C (Pre-eclampsia)	0	0	8	3	0

Table VI: Comparison of Mean Total Cholesterol Levels Across Study Groups

Parameter	Group A (Normotensive) Mean ± SD	Group B (Gestational Hypertension) Mean ± SD	Group C (Preeclampsia) Mean ± SD	p-value (ANOVA)
Total Cholesterol (mg/dL)	167.1 ± 12.9	200.0 ± 5.2	210.6 ± 9.6	0.01*
HDL-C (mg/dL)	42.7 ± 4.3	37.0 ± 4.1	35.2 ± 5.1	0.01*
LDL-C (mg/dL)	98.4 ± 10.2	126.4 ± 8.5	127.5 ± 7.1	0.01*
VLDL-C (mg/dL)	28.9 ± 3.0	29.4 ± 1.6	29.6 ± 2.8	0.699 (NS)
Triglycerides (mg/dL)	139.3 ± 14.4	175.6 ± 9.7	184.8 ± 13.1	0.01*

Table VII: Distribution of Proteinuria among participants

Urine albumin	Frequency	Percent
1+	8	8.0
2+	3	3.0
Nil	89	89.0
Total	100	100.0

Table VIII: Distribution of urine albumin across groups

Urine albumin	Group A		Group B		Group C		(p-value)
	Count	N %	Count	N %	Count	N %	
1+	0	0.0%	0	0.0%	8	72.7%	0.01
2+	0	0.0%	0	0.0%	3	27.3%	
Nil	82	100.0%	7	100.0%	0	0.0%	

Table IX: Association of the mode of delivery among the groups

Mode of Delivery	Frequency	Percent
Elective LSCS	5	5.0
Emergency LSCS	51	51.0
NVD	44	44.0
Total	100	100.0

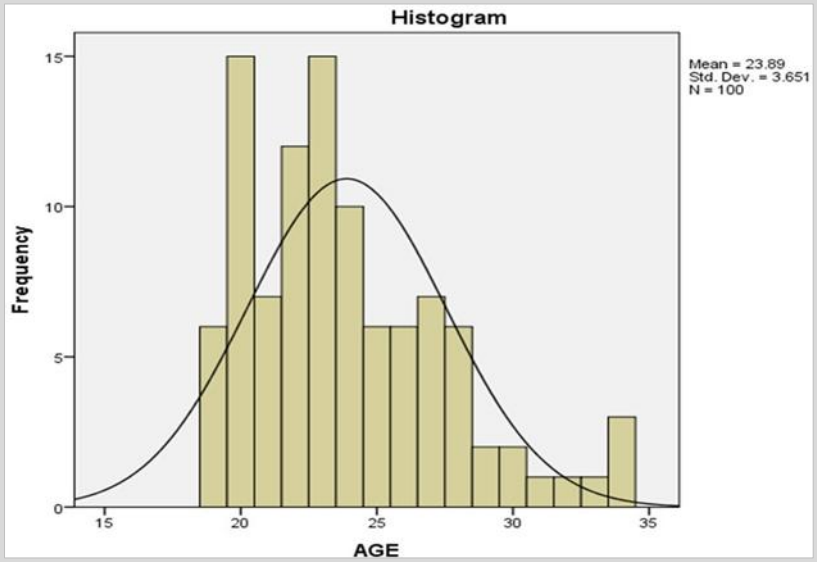

Figure 1: Histogram showing mean age of patients

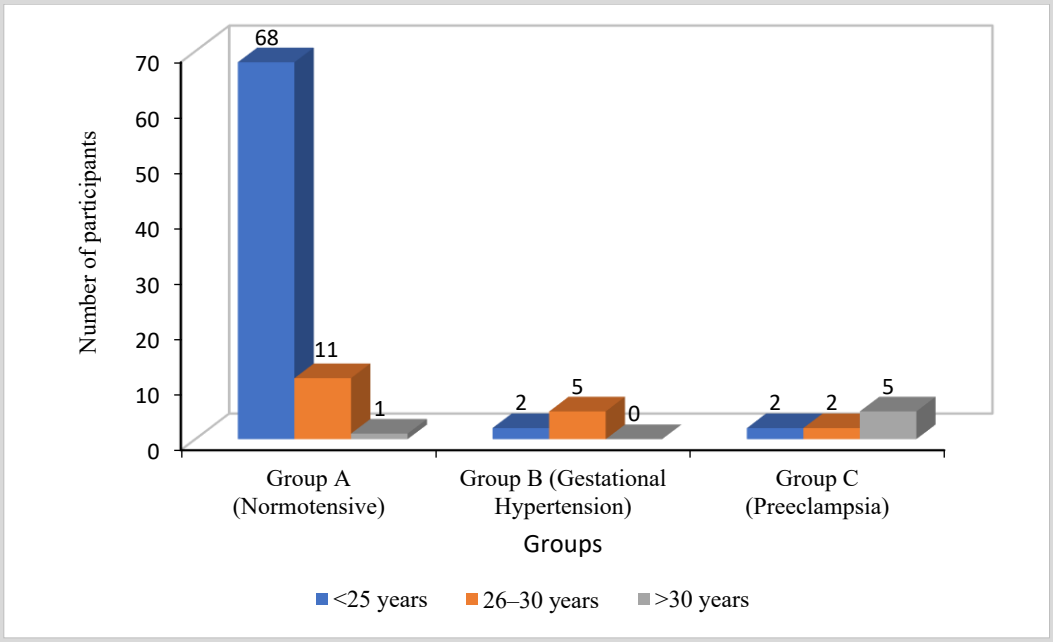

Figure 2: Age Distribution of participants between Groups

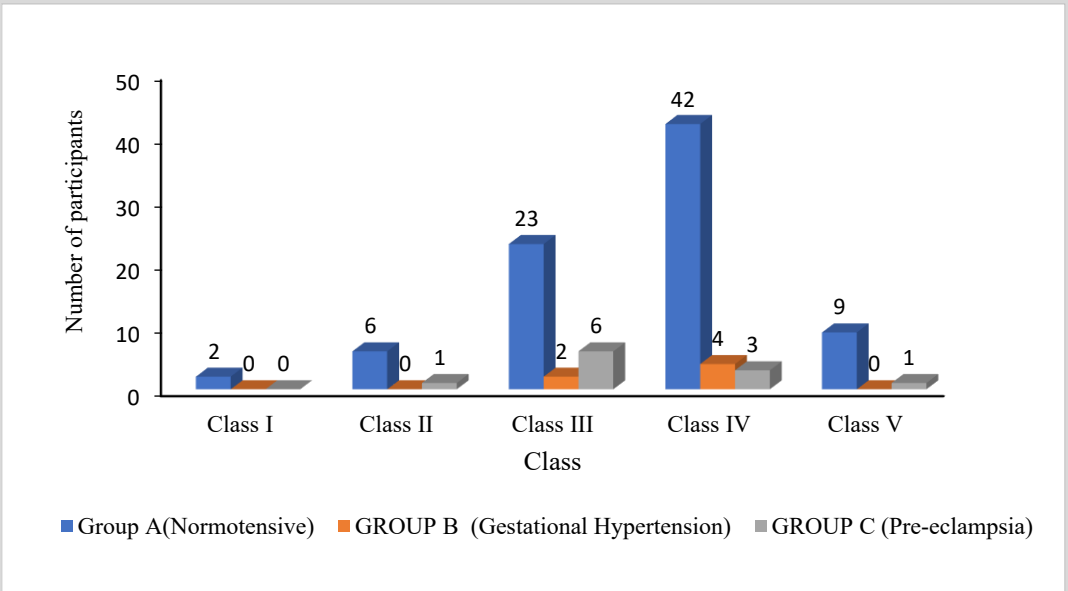

Figure 3: Socioeconomic Status Distribution Across Groups

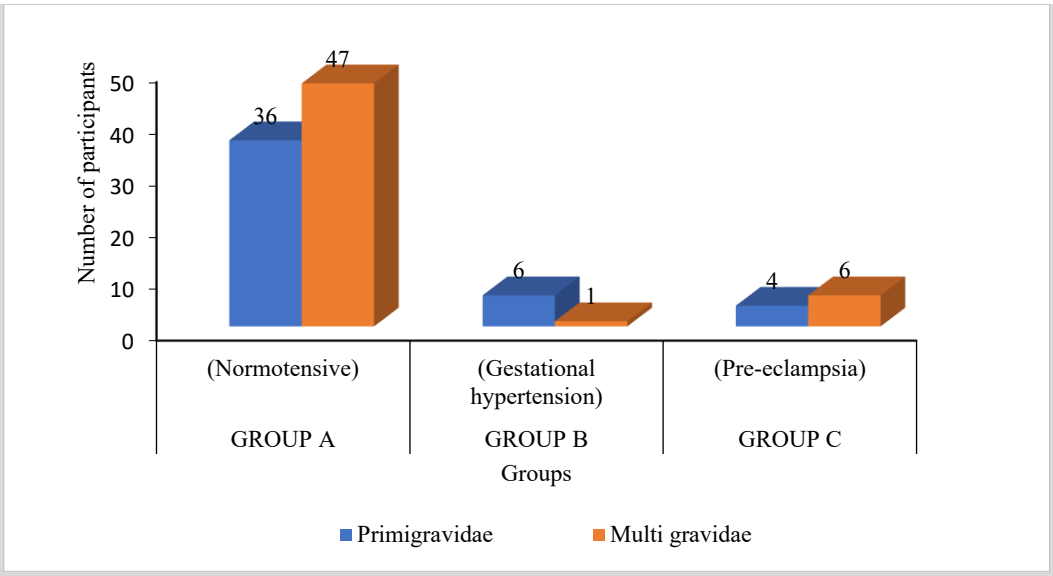

Figure 4: Obstetric Score Across Groups

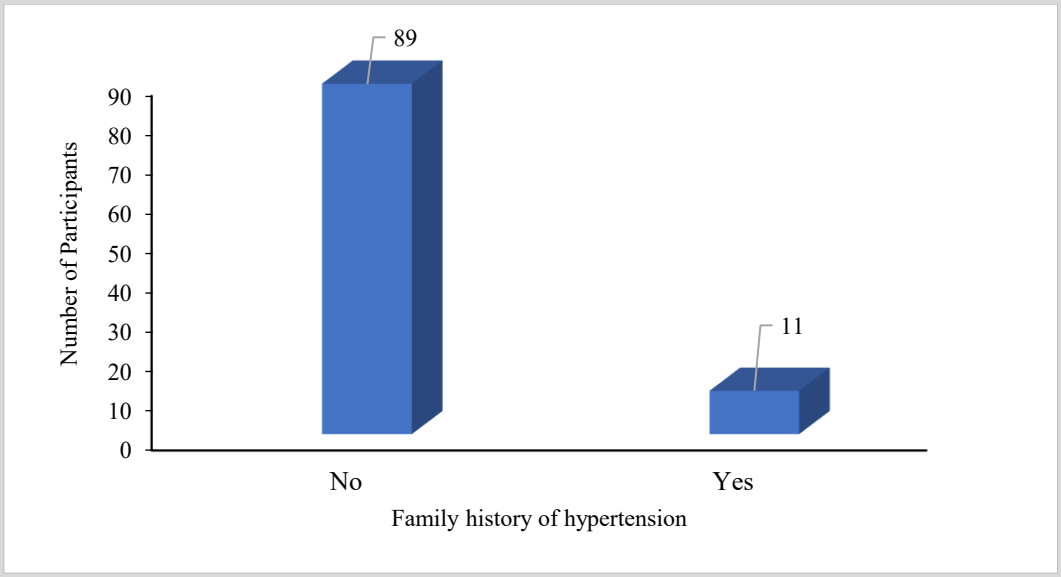

Figure 5: Family History of Hypertension across participants

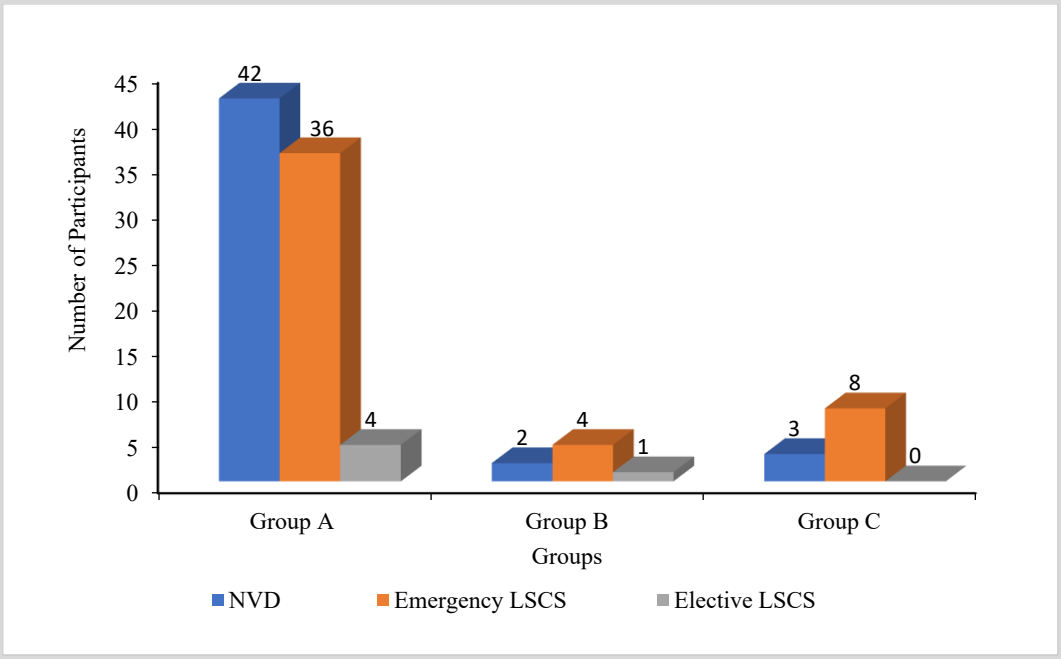

Figure 6: Mode of Delivery Distribution by Groups

4. Discussion

The exact cause of pregnancy-induced hypertension (PIH) and its severe form, preeclampsia, remains unclear. It is mainly characterised by high blood pressure and often involves proteinuria and systemic disturbances. Abnormal placentation, inadequate spiral artery remodelling, and subsequent placental ischemia are central to its development. Placental ischemia triggers the release of anti-angiogenic factors and inflammatory mediators, leading to endothelial dysfunction, oxidative stress, and systemic inflammation, which ultimately cause proteinuria, edema, and multi-organ effects [15].

A significant contributing factor to preeclampsia is dyslipidemia, marked by abnormal lipid metabolism. Studies indicate that women with preeclampsia have elevated levels of TG, TC, and low-density lipoprotein cholesterol (LDL-C), along with reduced high-density lipoprotein cholesterol (HDL-C), which contribute to endothelial dysfunction and aggravate vascular injury and disease severity [7,8]. The mechanism behind the relationship between dyslipidaemia with hypertensive disorders of pregnancy was well established in earlier studies. Dyslipidemia causes endothelial dysfunction, which leads to vasoconstriction, inflammation, and thrombosis, all of which contribute to hypertension. Oxidative stress results in, generation of reactive oxygen species that damage vascular endothelium. Dyslipidemia is also linked with increased production of inflammatory cytokines, which can further exacerbate endothelial dysfunction and hypertension [16].

The present study included 100 participants, who were included and divided into three groups as Group A: Normotensive pregnant women (82); Group B: pregnant ladies with Gestational hypertension (11); Group C: Pregnant ladies with Preeclampsia (7). The mean age of participants was approximately 24 years, representative that most women were in their early reproductive age group. Also, most of the participants belonged to lower socioeconomic strata, which may reflect limited access to antenatal care. The high disease burden of gestational hypertension and preeclampsia has been linked to nutritional imbalance, inadequate prenatal follow-up, and delayed detection of complications. A positive family history of hypertension was observed in 11% of the study participants, although no statistically significant association was found with the development of preeclampsia. Also, women with multigravidae were marginally more in number than primigravidae. Recent evidence indicated that parity by itself does not offer protection, and the condition may recur in later pregnancies, particularly in women with underlying metabolic risk factors such as obesity, insulin resistance, or dyslipidemia [17,18].

In this study, mean systolic and diastolic pressures were significantly higher among women with gestational hypertension and preeclampsia compared to normotensive women, indicating progressive vascular involvement with disease severity. This result was consistent with previous findings where both systolic and diastolic blood pressures increase significantly with the progression of preeclampsia, affecting systemic vascular injury and endothelial dysfunction [19]. BMI was also significantly higher among women who developed preeclampsia compared to other normotensive women, indicating that excess maternal weight is an important risk factor for hypertensive disorder. Studies reported that higher BMI is associated with increased risk of preeclampsia and that obesity and metabolic derangements (insulin resistance, dyslipidemia) worsen endothelial dysfunction and hypertension. Mao Z, et al.[20] also highlighted the correlation between obesity and hypertensive disorder of pregnancy. These findings collectively suggest that excessive maternal adiposity contributes to lipid imbalance.

The present study also revealed that a significant rise in total cholesterol, LDL-C, and triglycerides, with a reduction in HDL-C in both gestational hypertension and preeclampsia groups compared to normotensive women ($p < 0.05$). These findings are supported by various studies representing higher TC, TG, and LDL-C and lower HDL-C among preeclamptic patients. The increased levels of cholesterol may accelerate oxidative stress and lipid peroxidation, leading to vascular stiffness and atherosclerotic-like changes in the uteroplacental circulation. However, increased levels of TGs could reflect increased hepatic lipase activity and decreased lipoprotein lipase function, contributing to delayed triglyceride clearance and endothelial damage. Similar findings have been consistently reported in previous literature. Recently, Qin et al.[21] reported that LDL, total cholesterol, and triglycerides were significantly increased, while HDL was consistently reduced in women with hypertensive disorders of pregnancy compared to normotensive controls. However, Stadler et al.[22] demonstrated that both early- and late-onset preeclampsia are associated with atherogenic dyslipidemia, marked by elevated TG levels, reduced HDL-C, and a shift from large to smaller, less functional HDL particles. These findings across diverse cohorts highlight the role of lipid dysregulation in the pathogenesis of preeclampsia, indicating both quantitative and qualitative alterations in lipoproteins to contribute to endothelial dysfunction and oxidative stress.

Proteinuria remains one of the important features distinguishing preeclampsia from gestational hypertension. In this study, all women in Groups A (normotensive) and B (gestational hypertension) showed negative urine albumin, whereas all participants in Group C (preeclampsia) demonstrated proteinuria (72.7%) with 1+ and (27.3%) with 2+ levels, demonstrating significant renal involvement in preeclamptic women ($p = 0.01$). These findings align with the diagnostic criteria outlined by the American College of Obstetricians and Gynaecologists (ACOG), which recognises proteinuria ≥1+ as a defining marker of preeclampsia [5]. Studies also reported that elevated maternal urine albumin and albumin-to-creatinine ratio in early pregnancy were significantly associated with a higher risk of developing preeclampsia [23]. The findings from the current study and recent evidence revealed the strong association of proteinuria with preeclampsia as an indicator of endothelial dysfunction and renal impairment.

In the present study, hypertensive disorders of pregnancy were also associated with adverse obstetric outcomes. About 51% of women underwent emergency LSCS, while 44% delivered vaginally, and only 5% had elective caesarean sections. Similar findings were reported by literature studies, which reported significantly higher caesarean delivery rates among women with preeclampsia and gestational hypertension compared to normotensive pregnancies, primarily due to fetal distress, poor cervical progress, or maternal complications such as uncontrolled blood pressure or impending eclampsia.

Overall, findings of the study and growing evidence support the incidence of preeclampsia due to metabolic disturbance, especially dyslipidemia and obesity, that may predispose to hypertensive disorders of pregnancy. Further, large-scale studies with mechanistic investigations are necessary to establish lipid biomarkers as reliable predictors and therapeutic targets in hypertensive disorders of pregnancy.

5. Conclusion

The current hospital-based study identified a clear link between lipid profile changes during the early second trimester and the development of gestational hypertension and preeclampsia. Elevated levels of TGs, TC, and LDL-C, along with decreased HDL-C, were observed in women who later experienced hypertensive disorders. These results indicate that dyslipidemia may not just be a consequence but also an early metabolic disturbance that contributes to the development of endothelial dysfunction in preeclampsia. Therefore, lipid profiles in the early second trimester can serve as a useful, affordable predictor to identify high-risk pregnancies and allow for timely preventive measures. Further research with larger groups and long-term follow-up is recommended to confirm these findings and evaluate the role of lipid biomarkers in risk assessment and preventive care for preeclampsia and gestational hypertension.

Declarations

Acknowledgments

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Conflict of interest

The author declares no conflict of interest.

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Author contributions

Dr. Nithya M.P. was solely responsible for the conception and design of the study, data acquisition, statistical analysis, interpretation of results, drafting, and final approval of the manuscript.

EC details

The study received approval from the Institutional Ethics Committee of Narayana Medical College, Nellore, Andhra Pradesh (Approval date: 26 April 2022). Written informed consent was obtained from all study participants.

Data availability statement

The datasets generated and/or analysed during this study are available from the corresponding author upon reasonable request. Data sharing will adhere to institutional policies and applicable ethical guidelines to ensure patient confidentiality.

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