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Original Research

Association of Maternal Hypertriglyceridemia with Preeclampsia in Pregnant Females

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Abstract: **Background:** To determine the association between maternal hypertriglyceridemia and preeclampsia among pregnant females and assess the frequencies of both conditions. **Study Design, Place and Duration:** Analytical cross-sectional study conducted at Lahore General Hospital, Lahore, from August 2025 to November 2025. **Materials and Methods:** A total of 235 pregnant females aged 18–45 years with singleton pregnancies ≥20 weeks were enrolled through non-probability consecutive sampling. Females with chronic hypertension, eclampsia, diabetes mellitus, renal disease, autoimmune disease, or use of lipid-altering medicines were excluded. Maternal age, gestational age, parity, residence, body mass index (BMI), blood pressure, proteinuria, and fasting serum triglycerides were recorded. Hypertriglyceridemia was defined as triglycerides ≥150 mg/dL. Preeclampsia was diagnosed by new-onset hypertension after 20 weeks with proteinuria. Data were analysed using SPSS version 26. Mann–Whitney U, Welch's t, Pearson's chi-square, Fisher's exact, and Mantel–Haenszel tests were applied. **Results:** Preeclampsia was present in 21 (8.9%) participants and hypertriglyceridemia in 116 (49.4%). Median triglycerides were higher in females with preeclampsia than in those without preeclampsia (194 vs 144 mg/dL; p<0.001). Preeclampsia occurred in 13.8% of females with hypertriglyceridemia and 4.2% of those with normal triglycerides. Hypertriglyceridemia was significantly associated with preeclampsia (OR 3.648, 95% CI 1.290–10.315; p=0.010). The association remained significant after adjustment for age, gestational age, parity, and BMI. **Conclusion:** Maternal hypertriglyceridemia was associated with higher odds of preeclampsia. Fasting triglyceride measurement may assist risk assessment, although prospective studies are required before clinical use.

Keywords: Hypertriglyceridemia; preeclampsia; pregnancy; triglycerides; maternal BMI.

INTRODUCTION

Preeclampsia is a pregnancy-specific multisystem disorder defined by new-onset hypertension after 20 weeks of gestation, accompanied by proteinuria and/or evidence of maternal organ dysfunction.1 It remains an important cause of maternal and perinatal illness and death, with a recent meta-analysis estimating a global prevalence of 4.43% (95% CI: 3.73–5.20%).2 In Pakistan, recent evidence is mainly derived from individual hospitals, and a reliable nationally representative prevalence estimate is not currently available; however, local studies continue to report a substantial burden of preeclampsia and eclampsia on maternal and neonatal outcomes.3 Maternal complications include eclampsia, stroke, acute kidney injury, hepatic dysfunction, pulmonary oedema and placental abruption, while fetal complications include growth restriction, medically indicated preterm delivery, low birth weight and perinatal death.4 Delivery of the fetus and placenta remains the definitive treatment, creating a difficult clinical decision when the condition develops before fetal maturity.1 These serious outcomes support the need to identify measurable maternal factors associated with preeclampsia.

Maternal lipid concentrations normally increase during pregnancy to meet maternal energy requirements and support placental function and fetal growth, with triglyceride levels rising more markedly than other lipid fractions.5 However, triglyceride

elevation beyond the expected physiological increase may indicate disturbed lipid metabolism and has been linked with a greater likelihood of preeclampsia. In a large study of women with normal early-pregnancy BMI, high triglyceride levels were associated with preeclampsia after adjustment for potential confounders, with an adjusted odds ratio of 1.70 (95% CI: 1.38–2.11).6 Higher four-hour postprandial triglyceride concentrations during the second trimester were also observed among women who later developed preeclampsia.7 Similarly, fasting triglyceride levels were significantly higher in preeclamptic than normotensive pregnancies.8 High early-pregnancy triglycerides further increased preeclampsia risk when accompanied by elevated blood pressure, while third-trimester triglyceride concentration was independently associated with preeclampsia among women with normal pre-pregnancy BMI.9,10 In contrast, a multiancestry Mendelian randomization analysis found no consistent causal effect of genetically predicted triglyceride levels on preeclampsia, suggesting that some observed associations may be influenced by shared metabolic factors or residual confounding.11 Comparison between studies remains difficult because triglycerides were measured at different gestational stages, fasting and postprandial samples were used, thresholds varied, and adjustment for maternal characteristics was inconsistent.

Excessive triglyceride-rich lipoproteins may release free fatty acids, increasing lipid peroxidation and reactive oxygen species. This oxidative stress may reduce nitric oxide availability, impair vascular relaxation, and promote endothelial injury, vasoconstriction, inflammation, and vascular permeability. These changes may worsen placental hypoperfusion and contribute to preeclampsia. Fasting triglyceride measurement provides standardised assessment, while a fixed cutoff allows classification. Excluding confounders improves validity, and local evaluation is needed because metabolic profiles vary between populations. This study therefore assessed the association and frequencies of maternal hypertriglyceridemia and preeclampsia at Lahore General Hospital, with stratification by maternal age, gestational age, parity, and body mass index.

MATERIALS AND METHODS

A hospital-based analytical cross-sectional study was conducted in the Department of Obstetrics and Gynecology, Lahore General Hospital, Lahore, from August 2025 to November 2025. Ethical approval was obtained from the Institutional Review Board under approval number 2025/IRB/143. The sample size was calculated as 235 pregnant females using a 95% confidence level, a 3.5% margin of error, and an expected preeclampsia frequency of 8%. Participants were enrolled through non-probability consecutive sampling after obtaining written informed consent.
Pregnant females aged 18–45 years with a singleton pregnancy, gestational age of at least 20 weeks, and attendance for routine antenatal assessment were included. Females with chronic hypertension, eclampsia, diabetes mellitus, renal disease, autoimmune disease, or use of medicines affecting lipid metabolism were excluded. Females who declined participation were also excluded. Maternal age, gestational age, parity, place of residence, height, weight, body mass index, systolic blood pressure, diastolic blood pressure, and proteinuria status were recorded on a structured data collection form.

Blood pressure was measured using a calibrated sphygmomanometer after the participant had rested for at least five minutes. Preeclampsia was diagnosed when new-onset systolic blood pressure was at least 140 mmHg and/or diastolic blood pressure was at least 90 mmHg after 20 weeks of gestation, confirmed on two readings taken at least four hours apart, together with proteinuria. Proteinuria was identified by urinary protein of at least 300 mg in 24 hours or a dipstick result of at least 1+ when quantitative measurement was unavailable. A 5-mL fasting venous blood sample was collected after an overnight fast of 8–12 hours. Serum triglyceride concentration was measured by an enzymatic colorimetric method in the hospital laboratory. Maternal hypertriglyceridemia was labelled as a fasting triglyceride concentration of at least 150 mg/dL. Participants were classified as having or not having preeclampsia and as having or not having hypertriglyceridemia. The main outcome was the association between maternal hypertriglyceridemia and preeclampsia.

Data were analysed using SPSS version 26. Normality of continuous variables was assessed with the Shapiro–Wilk test. Normally distributed data were presented as mean and standard deviation, while non-normally distributed data were reported as median and interquartile range. Systolic blood pressure was compared using Welch's independent-samples t test because the group variances were unequal. Maternal age, gestational age, body mass index, diastolic blood pressure, and triglyceride concentration were compared using the Mann–Whitney U test. Categorical variables were presented as frequency and percentage. Pearson's chi-square test was used when expected cell counts were adequate, whereas Fisher's exact test was applied when expected counts were below five. Odds ratios with 95% confidence intervals were calculated. Stratified analyses were performed for maternal age, gestational age, parity, and body mass index, and Mantel–Haenszel common odds ratios were reported. A p-value below 0.05 was considered statistically significant.

RESULTS

A total of 235 pregnant females were included, with no missing observations in the analysed variables. Of these, 158 (67.2%) were aged 18–30 years, 137 (58.3%) were assessed at 32–40 weeks of gestation, 129 (54.9%) were multiparous, and 141 (60.0%) lived in urban areas. Preeclampsia was identified in 21 participants (8.9%; 95% CI, 5.9%–13.3%), while maternal hypertriglyceridemia was present in 116 (49.4%; 95% CI, 43.0%–55.7%).

Table 1: Overall Demographic and Clinical Profile of the Study Participants

Characteristic	**Category**	**n (%)**
Maternal age	18–30 years	158 (67.2%)
	31–45 years	77 (32.8%)
Gestational age	20–31 weeks	98 (41.7%)
	32–40 weeks	137 (58.3%)
Parity	Nulliparous	106 (45.1%)
	Multiparous	129 (54.9%)
Residence	Rural	94 (40.0%)
	Urban	141 (60.0%)
BMI category	<25 kg/m²	116 (49.4%)
	≥25 kg/m²	119 (50.6%)
Hypertriglyceridemia	Absent	119 (50.6%)
	Present	116 (49.4%)
Preeclampsia	Absent	214 (91.1%)
	Present	21 (8.9%)
Proteinuria	Absent	214 (91.1%)
	Present	21 (8.9%)

Note. Data are presented as frequency (percentage). Percentages were calculated using the total sample of 235 participants. No inferential test was applicable to this descriptive table. Proteinuria formed part of the operational definition of preeclampsia.

Maternal age and gestational age did not differ between participants with and without preeclampsia: the corresponding medians were 29.00 versus 28.00 years (U = 2209.50, p = 0.899) and 33.00 versus 33.50 weeks (U = 2010.50, p = 0.425). In contrast, the preeclampsia group had a higher median BMI [28.20 versus 25.05 kg/m²; U = 1494.00, p = 0.011] and a higher median serum triglyceride concentration [194.00 versus 144.00 mg/dL; U = 910.00, p < 0.001]. Mean systolic blood pressure was 155.86 ± 6.22 mmHg in the preeclampsia group compared with 115.65 ± 9.10 mmHg in the unaffected group, giving a mean difference of 40.21 mmHg (95% CI, 37.16–43.26; Welch t = 26.95, p < 0.001). Median diastolic blood pressure was also higher (97.00 versus 74.00 mmHg; U = 0.00, p < 0.001). Hypertriglyceridemia occurred in 16 (76.2%) participants with preeclampsia and 100 (46.7%) without preeclampsia, corresponding to an odds ratio of 3.65 (95% CI, 1.29–10.32; p = 0.010).

Table 2: **Comparison of Participant Characteristics According to Preeclampsia Status**

Variable	**No preeclampsia (n = 214)**	**Preeclampsia (n = 21)**	**Test statistic**	**Effect estimate (95% CI)**	**p**
Maternal age, years	28.00 (8.00)	29.00 (9.00)	U = 2209.50; Z = −0.13	r = 0.01	0.899[a]
Gestational age, weeks	33.50 (10.00)	33.00 (9.00)	U = 2010.50; Z = −0.80	r = 0.05	0.425[a]
BMI, kg/m²	25.05 (5.00)	28.20 (6.60)	U = 1494.00; Z = −2.53	r = 0.17	0.011[a]
Systolic blood pressure, mmHg	115.65 ± 9.10	155.86 ± 6.22	t(29.18) = 26.95	MD = 40.21 (37.16–43.26)	<0.001[b]
Diastolic blood pressure, mmHg	74.00 (10.00)	97.00 (7.00)	U = 0.00; Z = −7.57	r = 0.49	<0.001[a]
Serum triglycerides, mg/dL	144.00 (68.00)	194.00 (92.00)	U = 910.00; Z = −4.50	r = 0.29	<0.001[a]
Age 31–45 years	68 (31.8%)	9 (42.9%)	$\chi^2(1) = 1.07$	OR = 1.61 (0.65–4.00)	0.302[c]
Gestational age 32–40 weeks	125 (58.4%)	12 (57.1%)	$\chi^2(1) = 0.01$	OR = 0.95 (0.38–2.35)	0.910[c]
Nulliparous	93 (43.5%)	13 (61.9%)	$\chi^2(1) = 2.63$	OR = 2.11 (0.84–5.31)	0.105[c]
BMI ≥25 kg/m²	108 (50.5%)	11 (52.4%)	$\chi^2(1) = 0.03$	OR = 1.08 (0.44–2.65)	0.867[c]

Variable	No preeclampsia (n = 214)	Preeclampsia (n = 21)	Test statistic	Effect estimate (95% CI)	p
Hypertriglyceridemia	100 (46.7%)	16 (76.2%)	$\chi^2(1) = 6.64$	OR = 3.65 (1.29–10.32)	0.010[c]

Note. Non-normal continuous variables are median (interquartile range); systolic blood pressure is mean ± standard deviation; categorical variables are n (column percentage). Superscript a denotes the Mann–Whitney U test; b, Welch's t test; and c, Pearson's chi-square test. Pearson's test was used only when expected counts were adequate. MD = mean difference; OR = odds ratio; CI = confidence interval. Blood pressure and proteinuria were diagnostic components, not independent predictors.

Preeclampsia occurred in 16 of 116 women with hypertriglyceridemia (13.8%) compared with 5 of 119 women without hypertriglyceridemia (4.2%). This difference was statistically significant (Pearson $\chi^2(1) = 6.64$, p = 0.010), and hypertriglyceridemia was associated with 3.65-fold higher odds of preeclampsia (95% CI 1.29–10.32). The direction of association remained positive in every maternal-age, gestational-age, parity, and BMI stratum. The strongest stratum-specific association was observed among women with BMI <25 kg/m², in whom preeclampsia occurred in 16.7% of women with hypertriglyceridemia and 2.9% of those without it (OR 6.60, 95% CI 1.34–32.64; Fisher's exact p = 0.016). Although the individual age, gestational-age, and parity strata did not reach statistical significance, the Mantel–Haenszel common odds ratios remained significant after separate adjustment for maternal age, gestational age, parity, and BMI. Breslow–Day tests were non-significant for all four stratifying variables, indicating no evidence that the odds ratios differed across strata.

Table 3: Overall and Stratified Association between Maternal Hypertriglyceridemia and Preeclampsia

variable	Stratum	Preeclampsia without hypertriglyceridemia, n (%)	Preeclampsia with hypertriglyceridemia, n (%)	Statistical test and value	OR (95% CI)	p
Maternal age	18–30 years	3 (3.7)	9 (11.8)	Pearson $\chi^2(1) = 3.76$	3.54 (0.92–13.60)	0.052[a]
	31–45 years	2 (5.4)	7 (17.5)	Fisher's exact test	3.71 (0.72–19.17)	0.156[b]
Gestational age	20–31 weeks	2 (4.0)	7 (14.6)	Fisher's exact test	4.10 (0.81–20.83)	0.088[b]
	32–40 weeks	3 (4.3)	9 (13.2)	Pearson $\chi^2(1) = 3.39$	3.36 (0.87–12.98)	0.066[a]
Parity	Nulliparous	3 (6.0)	10 (17.9)	Pearson $\chi^2(1) = 3.45$	3.41 (0.88–13.17)	0.063[a]
	Multiparous	2 (2.9)	6 (10.0)	Fisher's exact test	3.72 (0.72–19.19)	0.144[b]
BMI	<25 kg/m²	2 (2.9)	8 (16.7)	Fisher's exact test	6.60 (1.34–32.64)	0.016[b]
	≥25 kg/m²	3 (5.9)	8 (11.8)	Fisher's exact test	2.13 (0.54–8.48)	0.348[b]

Note. Values are presented as the number of women with preeclampsia within each triglyceride category, with row percentages in parentheses. ORs compare women with hypertriglyceridemia with those without hypertriglyceridemia. a Pearson's chi-square test was used when all expected cell counts were ≥5. b Two-sided Fisher's exact test was used when at least one expected cell count was <5.

DISCUSSION

The present study showed that maternal hypertriglyceridemia was associated with higher odds of preeclampsia in pregnant females attending a tertiary hospital. Preeclampsia occurred in 8.9% of participants, and affected females had higher fasting triglyceride concentrations than those without preeclampsia. This frequency exceeded the 2.0% reported in a large prospective study and the 2.83–5.54% reported in recent Chinese hospital-based analyses, but was lower than the 21.6% reported in an Indian study with a high frequency of dyslipidemia.9,12–14 Differences may reflect referral patterns, gestational age at enrolment, diagnostic criteria, and the proportion of high-risk pregnancies included.

Maternal hypertriglyceridemia was present in 49.4% of participants. This high proportion requires caution because triglyceride concentrations rise during pregnancy, and a fixed threshold of 150 mg/dL may classify some physiological increases as abnormal, particularly later in gestation (Jiang et al., 2025). Previous studies used thresholds ranging from 150 or 200 mg/dL to the 90th percentile or trimester-specific values, limiting direct comparison.9,15,16 Nevertheless, preeclampsia occurred in 13.8% of females with hypertriglyceridemia compared with 4.2% of those without it, corresponding to 3.65-fold higher odds.

This finding agrees with several recent reports. High early-pregnancy triglycerides were associated with a 70% increase in adjusted odds of preeclampsia, while

higher four-hour postprandial triglycerides were found among women who later developed the condition.6,7 Higher fasting triglyceride levels in preeclamptic than normotensive pregnancy have also been reported in Nigeria, Bangladesh, Indonesia, India, Egypt, and Pakistan.8,17–20 Categorical analyses from Bangladesh and India also showed increased odds of preeclampsia among women with raised triglycerides.15,16 Third-trimester triglycerides remained associated with preeclampsia among women with normal pre-pregnancy BMI, and raised triglycerides strengthened the association between early-pregnancy hypertension and preeclampsia.9,10

A biological link is plausible. Excess triglyceride-rich lipoproteins may release free fatty acids that promote lipid peroxidation and reactive oxygen species formation. Oxidative stress may reduce nitric oxide availability, impair vascular relaxation, and increase endothelial injury, vasoconstriction, inflammation, and vascular permeability. These changes may worsen placental blood flow and contribute to preeclampsia.8,21 However, the cross-sectional design cannot establish whether triglyceride elevation preceded preeclampsia. A multiancestry Mendelian randomization analysis found no consistent causal effect of genetically predicted triglycerides, suggesting that shared metabolic factors, reverse causation, or residual confounding may partly explain the observed relationship.11 Variation in fasting status, gestational timing, ancestry, BMI, thresholds, and statistical adjustment may also account for conflicting results.

Median BMI was higher among females with preeclampsia, although BMI category was not significantly associated with the outcome. Similar relationships between higher BMI and preeclampsia or dyslipidemia have been reported elsewhere.16,22,23 Converting BMI into two categories may have reduced information and statistical power. Maternal age, gestational age, parity, and residence were not significantly different according to preeclampsia status.

Stratified odds ratios remained above one across age, gestational age, and parity categories, but most subgroup results were not significant and had wide confidence intervals. This likely reflected reduced precision after dividing only 21 preeclampsia cases. The stronger association in females with BMI below 25 kg/m² resembles findings in which the triglyceride–preeclampsia relationship was most evident among women with normal pre-pregnancy BMI.10 However, the difference between BMI strata does not prove interaction without a formal interaction test. A large multicentre analysis similarly found no significant modification by age, BMI, parity, or testing week.13

Strengths included fasting blood sampling, a predefined triglyceride cutoff, explicit eligibility criteria, exclusion of major disorders affecting lipid metabolism, consecutive enrolment, complete data, and use of statistical tests suited to data distribution and expected cell counts. Limitations included the single-centre setting, cross-sectional design, one-time triglyceride measurement, lack of trimester-specific reference ranges, few preeclampsia cases, wide subgroup intervals, and absence of simultaneous multivariable logistic regression. Diet, gestational weight gain, socioeconomic status, family history, physical activity, and other lipid fractions were not fully controlled. Generalisability beyond this tertiary hospital is therefore limited. Fasting triglyceride measurement may contribute to maternal risk assessment, but it should not be regarded as a diagnostic test, independent predictor, or basis for treatment without prospective confirmation.

CONCLUSION

Maternal hypertriglyceridemia was significantly associated with preeclampsia among pregnant females attending Lahore General Hospital. Higher fasting triglyceride concentrations were observed in females with preeclampsia, and the direction of association remained consistent after stratification by maternal age, gestational age, parity, and body mass index. These findings suggest that fasting triglyceride measurement may have a supportive role in maternal risk assessment. However, the association should not be interpreted as causal, and triglyceride testing should not replace standard clinical assessment. Prospective multicentre studies using trimester-specific reference ranges are required to confirm these findings.

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