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ASSOCIATION BETWEEN MATERNAL SERUM LIPID PROFILE AND NEONATAL MACROSOMIA IN HEALTHY PREGNANT WOMEN: A PROSPECTIVE COHORT STUDY FROM SOUTH INDIA

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ABSTRACT

Background: Neonatal macrosomia (birth weight ≥ 4000 g) complicates 5–20% of pregnancies worldwide and is associated with substantial maternal and neonatal morbidity. Although gestational diabetes mellitus is the classical risk factor, 50–70% of macrosomic infants are born to non-diabetic mothers, suggesting that maternal dyslipidaemia — independent of glycaemic status — may contribute to excessive fetal growth. Data from healthy, rigorously screened Indian women remain limited.

Objective: To determine the association between maternal serum lipid profile and neonatal macrosomia in healthy pregnant women, and to compare lipid parameters between mothers of macrosomic and non-macrosomic neonates between 24 and 34 weeks of gestation.

Methods: This prospective cohort study enrolled 400 antenatal women aged 24–34 weeks of gestation attending the Obstetrics and Gynaecology outpatient department of a tertiary care hospital in Chennai, India, between July 2024 and December 2025. Women with singleton pregnancies and pre-pregnancy BMI 18–24 kg/m² were included after rigorous exclusion of gestational/pre-gestational diabetes, hypertensive and thyroid disorders, and other metabolic comorbidities. Fasting total cholesterol (TC), triglycerides (TG), HDL-C, LDL-C and VLDL-C were measured, and participants were followed until delivery. Neonatal macrosomia was defined as birth weight ≥ 4000 g. Associations were assessed using the chi-square test, Spearman's correlation and multivariable logistic regression with bootstrap-derived 95% confidence intervals; receiver operating characteristic (ROC) curve analysis evaluated diagnostic performance.

Results: The prevalence of macrosomia was 19.0% (76/400). Elevated TC (≥250 mg/dL; OR 1.77, 95% CI 1.07–2.93, p=0.035) and elevated TG (≥200 mg/dL; OR 1.85, 95% CI 1.04–3.28, p=0.048) were significantly associated with macrosomia. Triglycerides showed the strongest correlation with birth weight (Spearman ρ=0.315, p<0.001) and the highest diagnostic accuracy (AUC 0.638).

Conclusion: Maternal hypertriglyceridaemia in the second and early third trimesters is significantly and independently associated with neonatal macrosomia in healthy, euglycaemic women, even after rigorous exclusion of gestational diabetes. Routine mid-trimester triglyceride screening may help identify pregnancies at risk of fetal overgrowth in settings where ultrasonography is not readily available.

KEYWORDS: Macrosomia; Maternal lipid profile; Triglycerides; Dyslipidaemia; Pregnancy; Birth weight

INTRODUCTION

Neonatal macrosomia, conventionally defined as a birth weight exceeding 4000 g regardless of gestational age, is one of the most clinically significant complications encountered in modern obstetric practice. The World Health Organization and the American College of Obstetricians and Gynecologists (ACOG) recognise this threshold as the most widely accepted diagnostic criterion, with ACOG further designating 4500 g as the threshold for clinical macrosomia.[1,2] The related concept of "large for gestational age" (LGA) — birth weight above the 90th percentile for gestational age. The global prevalence of macrosomia ranges from 5% to 20%, with rates of 8–10% reported in Western nations and 2–6% in developing countries, including India.[3–6]

The aetiology of macrosomia is multifactorial. Gestational diabetes mellitus (GDM) has traditionally been regarded as the most important cause,[7] operating through the Pedersen hypothesis of maternal hyperglycaemia-induced fetal hyperinsulinism.[8] However, 50–70% of macrosomic neonates are born to non-diabetic mothers, indicating that metabolic pathways unrelated to glycaemic dysregulation contribute substantially to excessive fetal growth.[9,10] Among candidate mechanisms, maternal dyslipidaemia — particularly hypertriglyceridaemia. Pregnancy is characterised by progressive physiological alterations in maternal lipid metabolism. By the third trimester, plasma triglyceride concentrations rise two- to three-fold above non-pregnant baselines, driven by enhanced hepatic VLDL secretion, insulin-resistance-mediated reduction in lipoprotein lipase (LPL) activity, and rising human placental lactogen.[11] These

triglyceride-rich lipoproteins are hydrolysed at the placental syncytiotrophoblast by placental LPL, liberating non-esterified fatty acids that cross the placenta via specific fatty acid transport and binding proteins and are re-esterified as subcutaneous fetal adipose tissue — the principal mechanism of fetal fat accretion in the third trimester.[12–14] Landmark studies by Kitajima et al. and Di Cianni et al. demonstrated that maternal triglycerides were the single strongest predictor of neonatal birth weight, even surpassing glucose levels in women with GDM.[15,16,17,18]

Despite this evidence, important gaps remain. Most existing studies either include women with GDM or fail to rigorously exclude metabolic comorbidities, confounding the independent contribution of lipids, and population-specific lipid cut-offs predictive of macrosomia have not been established for healthy Indian women. We therefore conducted this prospective cohort study to determine the association between maternal serum lipid profile and neonatal macrosomia in healthy, euglycaemic pregnant women, and to compare the maternal lipid profile — total cholesterol, triglycerides, LDL-C, HDL-C and VLDL-C.

MATERIALS AND METHODS

Study Design and Setting : This prospective cohort study was conducted in the Department of Obstetrics and Gynaecology, Sree Balaji Medical College and Hospital, Chennai, India, between July 2024 and December 2025.

Study Population and Sample Size : All antenatal women aged 24–34 weeks of gestation attending the outpatient department during the study period were screened for eligibility and enrolled at their first eligible visit. A total of 400 women meeting the eligibility criteria were recruited and followed prospectively until delivery, with neonatal birth weight recorded as the primary outcome.

Inclusion criteria: gestational age 24–34 weeks, singleton pregnancy, pre-pregnancy body mass index (BMI) 18–24 kg/m².

Exclusion criteria: maternal age >35 years; multifetal pregnancy; delivery before 37 weeks; previous history of preterm labour; prior history of diabetes in pregnancy; pre-gestational or gestational diabetes mellitus, hypertensive disorders, thyroid disorders, systemic lupus erythematosus, antiphospholipid antibody syndrome, or pancreatic/hepatic disorders; pre-pregnancy BMI >25 kg/m²; and any congenital fetal malformation.

Data Collection : After obtaining written informed consent, a structured, pre-tested proforma was used to record demographic and obstetric history, including age, parity, pre-pregnancy weight, gestational age (confirmed by last menstrual period and first-trimester ultrasound), and relevant medical and family history. Anthropometric measurements (height, weight, BMI) and routine antenatal investigations were performed to confirm eligibility. Socioeconomic status was classified using the Modified B.G. Prasad Classification.

Fasting venous blood samples (4 mL) were collected in the morning after an overnight fast of 8–10 hours. Serum total cholesterol (TC) and triglycerides (TG) were measured by enzymatic colorimetric methods, and HDL-C by a direct homogeneous assay, using an automated biochemistry analyser. LDL-C was calculated using the Friedewald equation (LDL-C = TC − HDL-C − [TG/5]), and VLDL-C was estimated as TG/5. All values were expressed in mg/dl

Participants were followed until delivery. Neonatal birth weight was recorded immediately at birth using a calibrated electronic weighing scale. Neonatal macrosomia was defined as birth weight ≥4000 g, in keeping with WHO criteria.[2] Gestational age at delivery, mode of delivery, and immediate neonatal condition were also recorded.

Statistical Analysis : Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics (version 28.0; IBM Corp., Armonk, NY, USA); R software (version 4.3.0) was used for ROC curve analysis. Normality of continuous variables was assessed using the Shapiro-Wilk test; lipid parameters were summarised as mean ± standard deviation, and categorical variables as frequency (percentage). Maternal lipid parameters were compared between macrosomic (birth weight ≥4000 g) and non-macrosomic groups using the chi-square test after categorisation at pre-defined clinical cut-offs (TC ≥250 mg/dL, TG ≥200 mg/dL, LDL-C ≥150 mg/dL, HDL-C <60 mg/dL, VLDL-C ≥40 mg/dL); Spearman's rank correlation was used Multivariable binary logistic regression was performed with macrosomia as the dependent variable and z-score-standardised lipid parameters as predictors; odds ratios (OR) and 95% confidence intervals (CI). Model discrimination was assessed by the area under the receiver operating characteristic curve (AUC) and Nagelkerke R^2. Optimal cut-offs for individual parameters were identified by maximising the Youden index (sensitivity + specificity − 1). A two-tailed p-value <0.05 was considered statistically significant throughout.

RESULTS

A total of 400 pregnant women were enrolled and completed follow-up until delivery. The mean maternal age was approximately 25 years, with the majority of participants belonging to the 21- 30 year age group, reflecting the typical reproductive age of the study population. Most women were primigravidae (77.5%), while 22.5% were multigravidae. The mean gestational age at lipid estimation was 28.5±3.1 weeks. The distribution of participants across gestational age groups was relatively balanced, with 152 women (38%) evaluated at 24-27 weeks, 105 (26.3%) at 28-30 weeks, and 143 (35.8%) at 31 – 34 weeks. (Fig. 1)

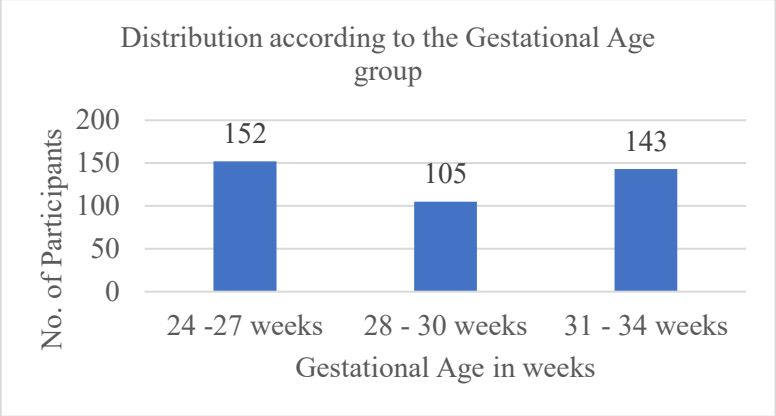

Fig 1. Gestational age group among the study participants

The mean maternal lipid values were: total cholesterol 239.10 ± 37.38 mg/dL, triglycerides 220.11 ± 56.77 mg/dL, LDL-C 142.63 ± 37.97 mg/dL, HDL-C 51.95 ± 8.30 mg/dL, and VLDL-C 44.03 ± 11.36 mg/dL. Using pre-defined clinical cut-offs, hypertriglyceridaemia was the most prevalent abnormality (66.0%), followed by low HDL-C (83.3% — reflecting the physiological trimester-related decline), elevated VLDL-C (67.0%), elevated LDL-C (41.3%), and elevated total cholesterol (37.5%) (Table 1).

Table 1. Maternal lipid profile and prevalence of dyslipidaemia (N = 400)

Lipid parameter	Mean ± SD (mg/dL)	Cut-off used	Abnormal, n (%)
Total cholesterol	239.10 ± 37.38	≥250 mg/dL	150 (37.5)
Triglycerides	220.11 ± 56.77	≥200 mg/dL	264 (66.0)
LDL-C	142.63 ± 37.97	≥150 mg/dL	165 (41.3)
HDL-C	51.95 ± 8.30	<60 mg/dL	333 (83.3)
VLDL-C	44.03 ± 11.36	≥40 mg/dL	268 (67.0)

SD = standard deviation. "Abnormal" denotes elevated values for TC, TG, LDL-C and VLDL-C, and low values for HDL-C.

Prevalence of Neonatal Macrosomia was neonatal macrosomia (birth weight ≥4000 g) occurred in 76 of 400 neonates (19.0%). On categorical analysis, elevated total cholesterol (OR 1.77, 95% CI 1.07–2.93, p=0.035) and elevated triglycerides (OR 1.85, 95% CI 1.04–3.28, p=0.048) were significantly associated with macrosomia. Elevated VLDL-C showed a borderline association (OR 1.75, 95% CI 0.98–3.11, p=0.075). LDL-C (OR 1.04, p=0.969) and HDL-C (OR 1.09, p=0.937) showed no significant categorical association with macrosomia (Table 2).

Spearman's rank correlation showed that triglycerides (ρ=0.315, p<0.001) and VLDL-C (ρ=0.315, p<0.001) had the strongest positive correlation with birth weight, followed by total cholesterol (ρ=0.194, p<0.001) and LDL-C (ρ=0.109, p=0.029); HDL-C showed a weak inverse correlation (ρ=−0.119, p=0.018) (Table 3).

On multivariable logistic regression with all five z-standardised lipid parameters entered simultaneously, only triglycerides (OR 1.32, 95% CI 1.07–1.68, p=0.017) and VLDL-C (OR 1.32, 95% CI 1.03–1.68, p=0.029) remained independent predictors of macrosomia. (Table 4) The model showed acceptable discrimination (AUC 0.649, Nagelkerke R^2 0.098) (Table 5).

Table 2. Association between maternal lipid parameters and neonatal macrosomia (N = 400)

Parameter (category)	Macrosomia n (%)	No macrosomia n (%)	OR (95% CI)	p-value
Total cholesterol ≥250 mg/dL (n=150)	37 (24.7)	113 (75.3)	1.77 (1.07–2.93)	0.035*
Total cholesterol <250 mg/dL (n=250, Ref)	39 (15.6)	211 (84.4)	1.00	—

Parameter (category)	Macrosomia n (%)	No macrosomia n (%)	OR (95% CI)	p-value
Triglycerides ≥200 mg/dL (n=264)	58 (22.0)	206 (78.0)	1.85 (1.04–3.28)	0.048*
Triglycerides <200 mg/dL (n=136, Ref)	18 (13.2)	118 (86.8)	1.00	—
LDL-C ≥150 mg/dL (n=165)	32 (19.4)	133 (80.6)	1.04 (0.63–1.73)	0.969
LDL-C <150 mg/dL (n=235, Ref)	44 (18.7)	191 (81.3)	1.00	—
HDL-C <60 mg/dL (n=333)	64 (19.2)	269 (80.8)	1.09 (0.55–2.16)	0.937
HDL-C ≥60 mg/dL (n=67, Ref)	12 (17.9)	55 (82.1)	1.00	—
VLDL-C ≥40 mg/dL (n=268)	58 (21.6)	210 (78.4)	1.75 (0.98–3.11)	0.075
VLDL-C <40 mg/dL (n=132, Ref)	18 (13.6)	114 (86.4)	1.00	—

OR = odds ratio; CI = confidence interval; Ref = reference category. *p<0.05, chi-square test.

Table 3. Spearman's rank correlation between maternal lipid parameters and neonatal birth weight (N = 400)

Parameter	Correlation coefficient (ρ)	p-value
Total cholesterol	0.194	<0.001*
Triglycerides	0.315	<0.001*
LDL-C	0.109	0.029*
HDL-C	−0.119	0.018*

*p<0.05, two-tailed.

Table 4. Multivariable logistic regression for prediction of neonatal macrosomia (N = 400)

Predictor (z-standardised)	β	OR	95% CI	p-value
Triglycerides	0.278	1.321	1.070–1.683	0.017*
VLDL-C	0.279	1.322	1.025–1.678	0.029*
LDL-C	0.177	1.194	0.721–1.877	0.473
HDL-C	0.077	1.080	0.825–1.427	0.579
Total cholesterol	0.005	1.005	0.704–1.673	0.982

Model AUC = 0.649; Nagelkerke R^2 = 0.098. 95% CI derived by bootstrap resampling (1000 iterations). *p<0.05.

Diagnostic Performance (ROC Analysis)

Triglycerides (AUC 0.638) and VLDL-C (AUC 0.637) showed the best individual discriminatory performance (both p<0.05 vs. AUC 0.5), with optimal cut-offs of 253 mg/dL and 51 mg/dL respectively. Total cholesterol showed modest performance (AUC 0.564), while LDL-C and HDL-C performed close to chance (AUC 0.514 and 0.517, respectively). The combined logistic model achieved the highest AUC (0.649), the highest sensitivity (71.1%) and the highest negative predictive value (88.6%), at the cost of lower specificity (52.8%) (Table 6).

Figure 2. ROC curves for lipid profile parameters and combined model in predicting fetal macrosomia (n=400)

Table 5. Diagnostic performance of maternal lipid parameters for predicting neonatal macrosomia (ROC analysis, N = 400)

Parameter	Optimal cut-off	Sensitivity	Specificity	PPV	NPV	AUC
Triglycerides	253 mg/dL	51.3%	75.9%	33.3%	86.9%	0.638*
VLDL-C	51 mg/dL	51.3%	75.9%	33.3%	86.9%	0.637*
Total cholesterol	253 mg/dL	47.4%	67.3%	25.4%	84.5%	0.564
LDL-C	166 mg/dL	34.2%	75.3%	24.5%	83.0%	0.514
HDL-C	51.3 mg/dL	56.6%	54.6%	22.6%	84.3%	0.517
Combined logistic model	Prob. ≥0.171	71.1%	52.8%	26.1%	88.6%	0.649*

AUC = area under the receiver operating characteristic curve; PPV = positive predictive value; NPV = negative predictive value. Optimal cut-offs determined by maximising the Youden index (sensitivity + specificity − 1). *AUC significantly greater than 0.5 ($p<0.05$). The HDL-C cut-off reflects the inverse relationship (lower HDL-C associated with macrosomia).

DISCUSSION

This prospective cohort study examined the relationship between maternal lipid parameters measured at 24–34 weeks of gestation and neonatal macrosomia in 400 healthy, euglycaemic pregnant women. The overall prevalence of macrosomia was 19.0%, considerably higher than the 5–10% typically reported from Western cohorts but consistent with figures from South Asian settings.[3,4] Mishra et al., in a prospective Indian study, reported macrosomia rates of 15–20% among women with dyslipidaemia, comparable to our findings,[19] and Koyanagi et al., in a multicountry analysis of 23 developing countries, noted that South Asian countries reported rates between 12% and 21%.[4] The relatively high rate observed in this cohort may reflect the marked prevalence of physiological hypertriglyceridaemia (66% of participants had TG ≥200 mg/dL) and the predominance of primigravidae (77.5%), who may lack the metabolic adaptation associated with prior pregnancies.

On categorical analysis, elevated total cholesterol (≥250 mg/dL) was significantly associated with macrosomia (OR 1.77, $p=0.035$), consistent with cohort data from Vrijkotte et al., who reported an association between first-trimester lipid elevation and large-for-gestational-age infants.[20] Total cholesterol rises progressively during pregnancy owing to oestrogen-driven increases in hepatic lipid synthesis and reduced catabolism,[11,21] and enhanced transplacental cholesterol transport via LDL-receptor-mediated pathways on the syncytiotrophoblast may augment fetal substrate availability and promote adipogenesis.[22,23] Woollett has shown that maternal cholesterol is a critical determinant of fetal cholesterol availability during organogenesis and growth,[22] consistent with the significant correlation we observed between total cholesterol and birth weight ($\rho=0.194$, $p<0.001$); however, the modest ROC AUC (0.564) suggests limited value as a stand-alone screening marker.

Triglycerides emerged as the most consistent and clinically important lipid predictor of macrosomia in this study — significantly associated on categorical analysis (OR 1.85, $p=0.048$), showing the strongest correlation with birth weight ($\rho=0.315$, $p<0.001$), the highest AUC on ROC analysis (0.638), and remaining an independent predictor on multivariable analysis (OR 1.32, $p=0.017$). This mirrors earlier work by Kitajima et al., who found that maternal triglycerides at 24–32 weeks were the single strongest predictor of neonatal birth weight even in women with GDM,[15] and by Di Cianni et al., who reported an independent, positive correlation between third-trimester triglycerides and birth weight in euglycaemic women.[16] Schaefer-Graf et al. similarly identified maternal triglycerides as a key determinant of the fetal environment in pregnancies complicated by GDM,[24] and Roy et al., in an Eastern Indian cohort, reported a comparable positive association between maternal triglycerides and neonatal birth weight.[25] The mean triglyceride level in our cohort

(220.11 ± 56.77 mg/dL) and the high prevalence of hypertriglyceridaemia (66.0%) reflect the marked physiological lipid shift of the second and third trimesters.[26] Mechanistically, maternal VLDL-triglyceride particles are hydrolysed at the syncytiotrophoblast by placental lipoprotein lipase, releasing non-esterified fatty acids that are transported across the placenta by fatty acid transport and binding proteins, fuelling fetal lipogenesis and adipose deposition.[12,13,14] VLDL-C mirrored the triglyceride findings almost exactly (ρ=0.315, AUC=0.637) — expected, given that VLDL-C is calculated as TG/5 — and both remained independent predictors on multivariable analysis, though given this collinearity they most likely represent a single underlying biological signal rather than two independent pathways.

LDL-C showed no significant categorical association with macrosomia (OR 1.04, p=0.969), despite LDL particles being the principal carriers of cholesterol to the placenta via LDL-receptor-mediated uptake.[22,23] This is consistent with Mudd et al., who reported that LDL-C associations with birth weight were weak and largely confined to the third trimester;[27] as lipid sampling in the present cohort spanned 24–34 weeks, with more than 60% of samples drawn before 31 weeks. HDL-C likewise showed no significant categorical association with macrosomia (OR 1.09, p=0.937), although a weak inverse correlation with birth weight was noted (ρ=−0.119, p=0.018) — consistent with the expected biological direction, in which lower HDL-C reflects a more pro-lipogenic state. These findings align with those of Di Cianni et al.[28] and Herrera,[29] who described a progressive decline in HDL-C from the second trimester as hepatic lipase activity rises; Schaefer-Graf et al. similarly found that HDL-C did not independently predict macrosomia in women with GDM.[24] Notably, 83.3% of participants in this cohort had HDL-C below the study cut-off of 60 mg/dL, a near-universal finding that substantially limits the discriminatory value of this particular threshold in South Asian pregnant populations.

On multivariable logistic regression incorporating all five lipid parameters simultaneously, only triglycerides and VLDL-C remained independent predictors of macrosomia (model AUC 0.649, Nagelkerke R^2 0.098). It is comparable to previously published single-biomarker models, including the sensitivity and specificity reported by Roy et al. for triglyceride-based prediction of macrosomia.[25] Given the recognised limitations of ultrasound-based fetal weight estimation, particularly its wide margin of error and inconsistent availability in resource-limited settings,[30–33] the incorporation of a low-cost, widely available biomarker such as fasting triglycerides into composite antenatal risk assessment — alongside anthropometric and glycaemic indices — may meaningfully improve macrosomia risk stratification, particularly in South Asian populations where physiological hypertriglyceridaemia is common.

When stratified descriptively by gestational-age window, the proportion of women with macrosomic neonates among those with elevated total cholesterol and triglycerides was numerically highest in the 28–30-week subgroup, though formal statistical comparison across gestational-age strata was not performed in this analysis; this pattern nonetheless suggests that the mid-trimester window may warrant particular attention for lipid-based risk assessment, a hypothesis that would benefit from confirmation in serial-sampling studies.

This study's principal strength is the rigorous exclusion of gestational diabetes and other metabolic comorbidities, allowing the independent contribution of maternal lipids to be examined without the confounding effect of glycaemic dysregulation. Several limitations should be considered when interpreting these findings. First, the study was conducted at a single tertiary care institution, which may limit generalisability to primary care or rural settings with different dietary patterns and baseline lipid profiles. Second, lipid measurement was performed at a single time point within the 24–34-week window rather than serially, precluding assessment of the trajectory of lipid change across gestation. Third, composite atherogenic indices (TC/HDL-C and TG/HDL-C ratios) were not formally analysed. Finally, advanced lipid biomarkers such as apolipoprotein fractions and lipoprotein particle size were not measured and may offer additional predictive value in future studies.

CONCLUSION

Maternal hypertriglyceridaemia in the second and early third trimesters was significantly and independently associated with neonatal macrosomia in this cohort of healthy, euglycaemic Indian women, even after rigorous exclusion of gestational diabetes and other metabolic comorbidities. Among the five lipid parameters assessed, triglycerides were the most consistent predictor across categorical, correlational, multivariable and ROC-based analyses, with VLDL-C — biochemically derived from triglycerides — showing an almost identical pattern. Total cholesterol was associated with macrosomia on categorical analysis but did not retain independent significance after adjustment, while LDL-C and HDL-C showed no clinically meaningful association. Although the overall discriminatory performance of the lipid profile was modest (AUC 0.649), reflecting the multifactorial nature of fetal overgrowth, these findings support the incorporation of fasting triglyceride measurement at 24–32 weeks of gestation into routine antenatal risk assessment, particularly in South Asian populations where physiological hypertriglyceridaemia and macrosomia rates are both high. Such an approach could offer a simple, low-cost adjunct to ultrasonographic surveillance for identifying pregnancies at risk of fetal overgrowth, and warrants further evaluation in multicentric and interventional studies.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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ETHICAL APPROVAL

This study was approved by the Institutional Ethics Committee, Sree Balaji Medical College and Hospital [IEC approval number and date: to be inserted]. Written informed consent was obtained from all participants prior to enrolment.

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