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EVALUATION OF SERUM LIPID LEVELS IN PRETERM AND TERM APPROPRIATE-FOR-GESTATIONAL-AGE NEONATES IN AN INDIAN POPULATION.

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Abstract

Background: The developmental origins of health and disease hypothesis suggests that adverse intrauterine conditions may permanently program metabolic functions, predisposing individuals to cardiovascular and metabolic disorders in later life. Cord blood lipid profile may serve as an early indicator of such metabolic programming in neonates, particularly in preterm and small-for-gestational-age (SGA) infants. ***Methodology:*** This prospective observational study was conducted in the Department of Paediatrics, Sree Mookambika Institute of Medical Sciences, Kulasekharam, from November 2025 to May 2026. Neonates were classified as preterm or term and as SGA or appropriate-for-gestational-age (AGA) using New Ballard scoring and AIIMS intrauterine growth charts. Cord blood samples were collected immediately after delivery for lipid profile estimation. Statistical analysis was performed using Student's t-test and Chi-square test, and results were expressed as mean ± SD and percentages. ***Results:*** SGA neonates demonstrated significantly higher cord blood lipid profile values compared to AGA neonates. Preterm neonates also showed significantly elevated total cholesterol, triglycerides, LDL, HDL, and VLDL levels compared to term neonates ($p<0.05$). Birth weight and ponderal index were significantly lower in SGA infants. Overall, lipid profile alterations were more pronounced in SGA and preterm groups. ***Conclusion:*** Cord blood lipid profile is significantly influenced by gestational age and fetal growth status. SGA and preterm neonates exhibit altered lipid metabolism, indicating early metabolic programming and potential future cardiovascular risk.

Keywords: Cord blood, lipid profile, preterm neonates, small for gestational age, AGA, metabolic programming, neonatal lipids.

INTRODUCTION

Cardiovascular diseases remain a leading cause of morbidity and mortality worldwide, with coronary artery disease (CAD) being a major contributor to adult deaths, particularly after the fourth decade of life in many industrialized and developing countries [1]. The development of coronary heart disease is strongly influenced by a combination of genetic predisposition and modifiable environmental risk factors such as diet, obesity, and physical inactivity. However, increasing evidence suggests that the origins of cardiovascular disease may extend far earlier than adulthood, beginning as early as intrauterine life.

The "fetal origin of adult disease" or "developmental origins of health and disease (DOHaD)" hypothesis proposes that adverse intrauterine conditions during critical periods of fetal growth can permanently program the structure and function of organs and metabolic pathways, thereby predisposing individuals to chronic diseases in later life [2]. Nutritional deprivation, placental insufficiency, and intrauterine growth restriction (IUGR) can lead to fetal adaptations that, while beneficial for short-term survival, may result in long-term metabolic and cardiovascular consequences.

A growing body of evidence links low birth weight and fetal growth restriction with an increased risk of hypertension, type 2 diabetes mellitus, and dyslipidaemia in adulthood [3]. Among these, dyslipidaemia plays a central role in the pathogenesis of atherosclerosis and coronary artery disease. Serum lipid profile, which includes total cholesterol, triglycerides, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and derived atherogenic indices, serves as an important marker of cardiovascular risk and underlying metabolic status.

Several studies have demonstrated that small-for-gestational-age (SGA) newborns exhibit altered lipid metabolism at birth compared to appropriate-for-gestational-age (AGA) infants, suggesting early metabolic programming [4]. These lipid abnormalities may persist into later life, thereby contributing to early onset atherosclerosis and increased cardiovascular risk. Furthermore, preterm infants may also demonstrate altered lipid profiles due to immature hepatic function and disrupted intrauterine nutrient supply, further increasing their vulnerability to metabolic disorders.

Evidence also indicates a direct association between abnormal neonatal lipid profiles and future cardiovascular morbidity, reinforcing the importance of early identification of high-risk neonates [5]. Early assessment of lipid profile in newborns, particularly those born preterm or SGA, may therefore provide valuable insight into future cardiovascular risk stratification and enable early preventive strategies.

In this context, the present study was undertaken to evaluate serum lipid profile in preterm and term appropriate-for-gestational-age (AGA) Indian newborns. The aim is to identify early metabolic alterations at birth and to understand potential differences in lipid parameters among different gestational groups, thereby contributing to early risk prediction and long-term preventive cardiovascular care.

Aim and Objectives

Aim

To evaluate and compare serum lipid profile in preterm and term appropriate-for-gestational-age (AGA) Indian newborns.

Objectives

1. To assess serum lipid profile parameters (total cholesterol, triglycerides, LDL, HDL) in preterm AGA newborns.
2. To assess serum lipid profile parameters in term AGA newborns.
3. To compare serum lipid profile between preterm and term AGA newborns.

METHEDOLOGY

The present study was conducted in the Department of Paediatrics, Sree Mookambika Institute of Medical Sciences, Kulasekharam, during the study period from November 2025 to May 2026. The study included both term and preterm neonates who were classified as small for gestational age (SGA) or appropriate for gestational age (AGA) and delivered in the study setting. Neonates with congenital malformations, large for gestational age (LGA), and post-term newborns were excluded from the study. Written informed consent was obtained from the parents or guardians of all participants prior to enrolment.

Classification of neonates as SGA and AGA was done using AIIMS intrauterine growth charts. Neonates with birth weight less than the 10th percentile for gestational age were classified as SGA, while those between the 10th and 90th percentiles were classified as AGA. Detailed demographic and clinical data were recorded for all enrolled neonates.

Statistical analysis was performed using appropriate methods. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency and percentages. Since the data were assumed to follow a normal distribution, parametric tests were applied for analysis. Comparison of serum lipid profile parameters between SGA and AGA neonates, as well as between preterm and term neonates and between male and female neonates, was performed using the Student's t-test. The association between categorical variables was assessed using the Chi-square test. A p-value of less than 0.05 was considered statistically significant for all analyses.

RESULTS

Table1: Gestational age distribution according to weeks.

GA (in weeks)	Term	Percent (%)	Pre- term	Percent (%)
<34	0	0	34	15.3
34-37	0	0	33	14.8
37-42	155	69.8	0	0

Termneonateswithgestationalagemorethan37weekswerearound69.8percentandpreterm neonates with 34- 37 weeks gestational age were around 14.8 percent and Pre term neonates with gestational age less than 34 weeks were around 15.3 percent.

Table2: Comparison of mean values and standard deviation of cord lipid rofile among term AGA and preterm AGA neonates.

Lipidprofile	TC	TG	HDL	LDL	VLDL
Term AGA	71.70±21.25	66.28±19.95	27.95±5.34	34.26±16.05	13.24±03.98
PTAGA	105.82±27.21	77.50±24.69	41.32±9.49	60.10±24.01	15.50±04.53
P value	<0.001	<0.05	<0.001	<0.001	<0.01

Table3: Comparison of mean values and standard deviation of cord lipid profile among term SGA and preterm SGA neonates.

Lipidprofile	TC	TG	HDL	LDL	VLDL
Term SGA	101.11±27.03	81.95±22.70	29.02±07.90	45.64±08.48	16.39±04.54
PTSGA	92.00±24.40	84.00±17.81	27.80±07.39	48.45±21.26	16.80±03.56
P Value	0.1176	0.6545	0.4763	0.4456	0.6545

Table4: Comparison of mean values and standard deviation of cord lipid profile according to gain in weeks.

Lipid profile	**Gestationalage(in weeks)**				**Pvalue**
	<34	34-37	37-40	40	
TC	123.41±25.15	92.03±23.03	82.97±27.18	83.50±09.57	<0.001
TG	100.58±14.86	75.39±20.07	71.27±22.17	86.75±14.03	<0.001
HDL	40.11±05.66	33.06±11.44	29.50±06.35	36.75±05.85	<0.001
LDL	67.52±25.99	50.69±29.33	38.91±50.02	42.75±05.82	<0.001
VLDL	20.11±02.97	15.07±04.01	14.27±04.43	17.35±02.80	<0.001
AI	3.11±0.71	3.07±1.15	3.04±1.29	2.29±0.18	0.643

DISCUSSION

The present study was undertaken to evaluate variations in cord blood lipid profile among preterm and term neonates, as well as between small-for-gestational-age (SGA) and appropriate-for-gestational-age (AGA) newborns. Cord blood lipid estimation provides an early metabolic snapshot and reflects intrauterine programming influences on neonatal lipid metabolism, which may have implications for future cardiovascular risk.

In the present study, SGA neonates demonstrated higher cord blood lipid profile values compared to AGA neonates. This finding is consistent with the metabolic adaptation hypothesis, wherein growth-restricted fetuses utilize alternative energy sources such as lipids and amino acids due to relative glucose deficiency. Increased hepatic lipogenesis and reduced peripheral utilization of lipids, secondary to decreased lipoprotein lipase activity, may contribute to elevated circulating lipid levels in SGA neonates [6,8]. Similar observations were reported by Kelishadi et al. and Pardo et al., who demonstrated significantly lower HDL levels and altered lipid fractions in SGA neonates compared to AGA controls, supporting early metabolic derangement in growth-restricted infants [4,5].

The Barker hypothesis further strengthens these findings by proposing that adverse intrauterine conditions lead to permanent metabolic "programming," predisposing individuals to hypertension, type 2 diabetes mellitus, and dyslipidaemia in later life [11,12]. In the present study, term SGA neonates exhibited higher lipid levels compared to term AGA neonates, reinforcing the concept that fetal growth restriction itself, rather than gestational age alone, influences neonatal lipid metabolism. Oba et al. similarly reported significantly elevated total cholesterol, triglycerides, LDL, and HDL levels in both term and preterm SGA neonates compared to AGA neonates, with strong statistical significance ($p < 0.0001$), which closely aligns with the present findings [10].

In addition, preterm neonates in the present study showed significantly higher levels of total cholesterol, triglycerides, LDL, HDL, and VLDL compared to term neonates. This suggests that prematurity itself is associated with altered lipid metabolism, possibly due to immature hepatic enzymatic systems and incomplete intrauterine nutrient transfer. These findings are supported by Haridas et al., who reported higher triglyceride and cholesterol levels in preterm neonates, although statistical significance was noted only for total cholesterol [15]. Mathur et al. also demonstrated significantly elevated cholesterol levels in preterm infants, further supporting altered lipid handling in this group [16]. However, contrasting results have been reported by Kalra et al., who observed lower lipid levels in preterm neonates compared to term neonates, with significance mainly in total cholesterol [13]. Similarly, Singh et al. reported higher total cholesterol in term neonates compared to preterm infants [6], while Mishra et al. found no significant difference in triglyceride levels

between the groups [14]. These variations may be attributed to differences in sample size, gestational age distribution, maternal factors, and methodological variations.

In the present study, the mean birth weight and ponderal index were significantly lower in SGA neonates, reflecting intrauterine growth restriction and supporting their classification. Comparable findings have been reported in previous studies, although differences exist in inclusion criteria and gestational age grouping [4,5].

Overall, the present study reinforces the concept that both prematurity and intrauterine growth restriction significantly influence neonatal lipid metabolism. Elevated cord blood lipid levels in SGA and preterm neonates may represent early metabolic adaptations that predispose to long-term cardiovascular risk. Early identification of these alterations may help in risk stratification and long-term preventive strategies.

CONCLUSION

The present study demonstrates that cord blood lipid profile varies significantly with gestational age and fetal growth pattern. Both small-for-gestational-age (SGA) and preterm neonates exhibit altered lipid parameters compared to appropriate-for-gestational-age (AGA) and term neonates, suggesting early metabolic adaptation to intrauterine conditions.

SGA neonates showed higher lipid profile values, which may be attributed to intrauterine growth restriction leading to altered lipid utilization and increased hepatic lipid synthesis. Preterm neonates also demonstrated significant alterations in lipid fractions, likely due to immature metabolic and enzymatic systems. These findings support the concept of fetal metabolic programming and the developmental origins of adult diseases.

The observed differences in lipid profile highlight the importance of early metabolic assessment in high-risk neonates such as preterm and SGA infants. Identification of such early biochemical alterations may help in risk stratification for future cardiovascular and metabolic disorders. Therefore, cord blood lipid profile may serve as a useful early biomarker for identifying neonates at increased risk, enabling long-term follow-up and preventive health strategies.

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