Journal of Cardiovascular Disease Research

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 05, 2023

Original research article

Evaluation of role of gestational age and weight on lipid profile in neonates

[1]Dr. Jogi Satya Sree Tata, [2]Dr. Sarat Chandra Jalagadugula, [3]Dr. V. Thrishi Sagna, [4]Dr. Jaya Lakshmi Nalavath

[1,2,4]Assistant Professor, Department of Paediatrics, Gayatri Vidya Parishad Institute of Health Care and Medical Technology, Visakhapatnam, Andhra Pradesh, India

[3]Associate Professor, Department of Paediatrics, Gayatri Vidya Parishad Institute of Health Care and Medical Technology, Visakhapatnam, Andhra Pradesh, India

Corresponding Author:

Dr. Jaya Lakshmi Nalavath (drjaya3105@gmail.com)

Abstract

Background and objectives: To calculate the levels of triglycerides, HDL and LDL cholesterol, as well as total cholesterol, in the cord blood of term and preterm neonates. To calculate the levels of triglycerides, total cholesterol, high-density lipoprotein (HDL) cholesterol and low-density lipoprotein (LDL) cholesterol in the cord blood of neonates.

Method: In the proposed study, which was conducted at the Gayatri Vidya Parishad Institute of Health Care and Medical Technology in Visakhapatnam, Andhra Pradesh, India, 100 infants from booked and unbooked expectant mothers underwent a thorough clinical evaluation. Diabetes, hypertension, and heart disease not in the family. The deciding factor, according to New Ballard's scale, will be gestational age.

Result: The p value for triglycerides was less than 0.05 when compared to LDL and VLDL. Mean lipid profiles of term (n=62) and preterm (n=38) neonates were compared. LDL and VLDL values in preterm neonates differ ($p<0.013$ and 0.007). The atherogenic index, triglycerides, VLDL, and total cholesterol all have statistically significant p values. 40 SGA terms as well as 22 AGA terms. Significant proportional disparities in total cholesterol and very low density lipoprotein ($p<0.05$) show that newborns who are small for gestational age are at risk. Significant p values can be seen for birth weight, head circumference, and HDL. 38 premature neonates had SGA or AGA. AGA and SGA in preterms had different HDL fractions ($p<0.05$).

Conclusion: Preterm SGA neonates had greater TC, TG, LDL, and VLDL readings than preterm AGA neonates did. Compared to term AGA newborns, term SGA neonates had higher readings for TC, TG, and VLDL. AI was significantly higher among people who weighed less than 1.5 kg ($P<0.001$), which suggested an increased risk of atherosclerosis.

Keywords: Gestational age, lipid profile, HDL, LDL, AGA, SGA, TC, TG

Introduction

In the majority of industrialised nations, particularly in the United States, there is a significant reason to be concerned about the rising morbidity and death from coronary heart disorders in males and females in early middle age. After the age of 40, coronary heart problems start to become a major cause of death. The prevalence of hereditary and environmental risk factors influences coronary artery disease incidence. The intrauterine environment has an impact on the emergence of cardiovascular disease risk factors, according to recent human and animal studies. The idea that coronary heart disease starts in childhood has drawn more and more attention over the past 25 years, especially since Enos *et al*. published their postmortem investigations on an American serviceman slain in Korea [1, 2].

Researchers like Glueck, Berenson, and Lauer, who have presented strong evidence that established risk factors for coronary heart illnesses in adult life can be discovered in children, further encouraged these study efforts in the year 1970. The assumption that prophylaxis, intended to delay the formation of the atherosclerotic plaque, is better to therapies adopted later in life at plaque regression has led a number of professionals to advocate that risk factors discovered in children should be treated. The prevalence of diabetes mellitus (DM), hypertension, and coronary heart disease (CHD) is epidemic worldwide. Although unhealthy lifestyle choices and behaviours are widely acknowledged as risk factors, the fundamental origins of these diseases may actually be detected during pregnancy. These discoveries gave rise to the "foetal origin of cardiovascular disease hypothesis", which contends that an unfavourable

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 05, 2023

intrauterine environment during a crucial developmental stage may have programmed or imprinted the development of foetal tissues and organs and influenced the responses that resulted in later coronary artery disease. Therefore, foetal growth restriction and low birth weight are linked to adult onset arterial hypertension, dyslipidemia, and type 2 diabetes, all of which have their genesis in the foetus. The "Indian prevalence of dyslipidemia" was found to be more common in men than in women. 38.7% of participants with a total cholesterol (TC) value under 200 mg/dl were men, whereas 23.3% were women. 64.2% of men and 33.8% of women had unusually low levels of high density lipoprotein cholesterol (HDL-C) [3, 4, 5].

In comparison to those under 30, those between 31 and 40 years old experienced a greater increase in the prevalence of hypercholesterolemia and hypertriglyceridemia. Since the past 20 years, coronary artery disease has been on the rise in India, while it has been declining in Western nations. It is generally known that the demographic shift in Western nations was accompanied by a decline in infectious disease deaths and an increase in non-communicable disease mortality. Compared to the national average of 41.2 years between 1951 and 1961, India's average life expectancy at birth is 63.7 years, 63.1 years for men and 64.4 years for women. Between 1942 and 1972, there was a decrease in the mortality rate, which was then followed by a decrease in the birth rate. The improvement in India's life expectancy, which is significant in and of itself, is linked to a decline in parasitic, nutritional and infectious diseases. Many people have reached an age where cardio vascular disease begins to show symptoms as a result of the increase in life expectancy. In industrialised Western nations, rates of coronary artery disease have been on the decline for the past 30 years, but have climbed in India. Because there has not been a significant prospective study of this kind, it is impossible to determine the exact prevalence of coronary artery disease in India [6, 7, 8].

The estimate of the actual burden of cardio vascular disease is also hampered by the lack of a centralised death registry for these illnesses and errors in the preparation of death certificates. The prevalence of coronary artery disease in the urban population has increased, nevertheless, from 1% in 1960 to 10.5% in 1998, according to a number of independent epidemiological studies carried out in North India. From 11.0% to 14.2% more cases of coronary artery disease have been documented in South India. These prevalence rates, when converted to numbers and applied to the size of the Indian population, show that coronary artery disease is a major cause of mortality in a significant portion of cases. There is already evidence from community research in India that there is a growing correlation between poorer income or educational attainment and the number of deaths from cardiovascular disease may exceed projections because of these social groups' poorer economic status. It is imperative to halt the spread of this epidemic since the current healthcare system is ill-equipped to handle the tremendous load that cardiovascular disease will impose [9, 10].

The long-term effects of these metabolic changes will increase the prevalence of cardiovascular illnesses, hypertension, and type 2 diabetes in this group of infants. Since the origins of coronary heart disease may be traced to infant and early childhood, it is generally agreed that preventative measures should start in this age group. Hypertension and dyslipoproteinemia appear to be the main risk factors that are treatable by diagnostic and therapeutic intervention. In order to identify the group most at risk of developing atherosclerosis, the pathological characteristic of coronary heart disease, cord blood screening has mostly been used. Additionally, lipid fractional analysis, which has been used for screening and diagnostic evaluation of a number of different illnesses, is acknowledged for its value. The alternate energy source is free fatty acids. Free fatty acids have a significant role as a source of energy during the first few hours of life after birth when the maternal blood supply stops. Free fatty acid and triglyceride levels in cord blood during birth are typically low. In the first few weeks following delivery, levels of free fatty acids and triglycerides rise quickly. By roughly 6 to 12 hours of age, it has been shown that their levels had increased by two to ten fold, respectively [11, 12].

This increase in the levels of free fatty acids and triglycerides is most likely the result of the mobilisation of free fatty acids from mobile fat stores and an increase in the production of hepatic glycerides, which were then released into the bloodstream. If this physiological reaction is exacerbated by a number of unfavourable circumstances and an unfavourable environment, it interferes with the foetal oxygenation and leads to antepartum and/or intrapartum hypoxia. Serum free fatty acid and triglyceride levels significantly increase in response to this stressful circumstance. Numerous high-risk factors for both mothers and foetus have a detrimental effect on the fetal-placental unit and compromise its functions. Ante-partum haemorrhage, prolonged labour, foetal distress due to various causes, cord compression and low Apgar score, low birth weight, and pregnancy-induced hypertension are some of the well-known maternal as well as foetal factors. A person's lipid profile is a reliable indicator of their cardiovascular health. Numerous atherogenic indices and measurements of cholesterol and its derivatives are included in the lipid profile. According to studies, SGA new-borns lipid profiles were aberrant as compared to AGA neonates. Numerous studies have shown a clear link between the incidence of cardiovascular illnesses and the aberrant lipid profiles found in SGA new-borns. The goal of the current study was to identify lipid profile anomalies as soon as possible (at birth), particularly in preterm and SGA infants, in order to monitor these high-risk infants closely in the future. In these high risk new-borns, early diagnosis, careful

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 05, 2023

nutritional supplementation, and pharmacological therapy may offer a chance for long-term primary amelioration of risk factors that lead to the development of cardio vascular disorders in adulthood [12, 13].

Material and Method

A hospital-based simple random sampling prospective case control study with case controls was conducted at the Gayatri Vidya Parishad Institute of Health Care and Medical Technology, Visakhapatnam, Andhra Pradesh, India, on one hundred new-borns who came from booked and unbooked pregnant women. Diabetes, high blood pressure, or cardiovascular disease not run in the family. When applying the New Ballard scale, the child's gestational age will be the determining factor.

Inclusion criteria

1. Both booked and unbooked infant cases are included in the total number of neonates delivered at Hospital.
2. Mother and family are low socioeconomic status.
3. A mother who takes IFA supplements.

Exclusion criteria

Neonates with

1. Congenital disorders
2. Newborns born to mothers who have conditions such gestational diabetes, diabetes mellitus, insulin dependent diabetes mellitus, tuberculosis, asthma, or pregnancy-induced hypertension.
3. Hypercholesterolemia and coronary heart disease run in the family.
4. All maternal drugs, excluding iron and vitamin supplements.
5. Prenatal drugs and drug abuse by mothers.
6. Twin birth with instrumental delivery, including extraction.
7. Newborns with Sepsis and Hypoxic-Ischemic Encephalopathy (HIE).

Methodology

Following formal parental or guardian approval, all neonates who meet the criteria and are eligible will be enrolled. The newborn will get a comprehensive clinical checkup and a newborn weight will be determined using an electronic weighing scale. New Ballard's rating will be used to categorise newborn as term or preterm based on gestational age. According to gestational age, the neonates will be split into two groups based on birth weight: those weighing 2.5 kg or more and those weighing less than 2.5 kg. The neonates were once more separated into groups according to their gestational age: SGA and AGA. On the basis of the intra uterine growth charts advised by AIIMS, birth weights (in kg) that are less than the 10th percentile for their gestational age are classified as small for gestational age. Measurements include length (in cm) and head circumference (in cm). Additionally, measurements of the chest and abdominal circumferences are taken, and the ponderal index is computed. We will do a Chi-square test, t-test and contingency coefficient analysis for continuous variables using SPSS for Windows.

Result

Table 1: Displaying Lipid Profile Differences by Sex

	Male N=54	**Female N=46**	**P value**
Total Cholesterol	97.80±25.3	95.23±28.9	0.643
Triglycerides	76.17±24.08	77.2±22.9	0.825
HDL	31.7±9.6	28.5±6.9	0.067
LDL	51.7±21.2	46.5±12.5	0.147
VLDL	15.2±4.8	15.4±4.5	0.825

Lipid profiles of male and female are compared in the table. Total cholesterol in male averages 97.80±25.3, whereas in female it averages 95.23± 28.9, and the difference is not statistically significant (p<0.63). Triglyceride levels were not significantly different between sexes (76.17± 24.08 and 77.2 ±22.9). The p value was 0.825.

HDL levels are not significantly different between sexes, with males having HDL levels of 31.7±9.6 and females having HDL levels of 28.5 ±6.9 (p<0.067). The p value of 0.147 between male and female LDL levels indicates there is no statistically significant difference between the sexes.

There was no statistically significant difference between male and female in terms of their VLDL levels (15.2±4.8, 15.4±4.5).

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 05, 2023

Table 2: Lipid Profile Demonstration in AGA and SGA

LIPID	AGA (N=30)	SGA (N=70)	P value
Total Cholesterol	91.7±26.9	98.7±26.8	P=0.238
Triglycerides	63.9±23.5	82.0±21.3	P=0.0001
HDL	34.2±9.1	28.6±7.8	P =0.003
LDL	51.4±20.4	48.5±16.7	P=0.456
VLDL	12.7±4.7	3.7±1.4	P=0.0001

There are a total of 30 AGA and 70 SGA. Triglycerides, high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL) all have p values lower than 0.05, while total cholesterol and low-density lipoprotein (LDL) do not.

Table 3: Comparison of Term and Preterm Neonates Lipid Profiles

LIPID	Term (N=62)	Preterm (N=38)	P Value
Total Cholesterol	95±26.9	99.2±27.1	0.455
Triglycerides	71.7±24.1	84.6±20	0.007
HDL	29.5±7.2	31.5±10.4	0.26
LDL	45.9±10.9	54.9±24.6	0.013
VLDL	14.3±4.83	16.9±4	0.007

There are 62 full-term newborns and 38 premature. When comparing triglycerides to LDL and VLDL, a p value of less than 0.05 indicates statistical significance. Mean lipid profiles were compared between term (n=62) and preterm (n=38) births. Preterm LDL and VLDL levels are significantly different from each other (p = 0.013 and 0.007, respectively).

Table 4: Comparison of Lipid Profile in Term AGA vs. SGA Based on Neonatal Parameter

	Term AGA (n=22)	Term SGA (n=40)	P value
Total Cholesterol	83..45±23.23	101.43±26.9	0.011
Triglyceride	55.3±16.1	80.7±23.2	0.0001
HDL	30.2±5.9	29.1±7.8	0.579
LDL	45.5±14.3	46.1±8.7	0.830
VLDL	11.0±3.2	16.1±4.6	0.0001
AI	2.8±0.91	3.7±1.6	0.014
Birth weight	2.8±0.3	2.0±0.3	0.0001
Ponderal Index	1.6±0.3	2.0±0.26	0.001
Head circumference	34.1±0.95	33.0±0.8	0.0001

Total Cholesterol, Triglyceride, Very Low Density Lipoprotein, and the Atherogenic Index all have statistically significant p values. There were 22 AGA terms and 40 SGA terms in total. Total cholesterol (mean 83.45$\pm$23 in term aga and 101.45$\pm$ 26.9 in term sga) and very low density lipoprotein (mean 11.0$\pm$3.2 in term aga and 16.1$\pm$4.6 in term sga) have significant proportional differences (p<0.05), indicating that neonates born small for gestational age are at increased risk.

Table 5: Preterm Aga vs. SGA: A comparison of lipid profile and neonatal parameters

	Preterm AGA (n =8)	Preterm SGA(n=30)	P-value
Total Cholesterol	114.6±23.9	95.1±26.8	0.071
Triglycerides	87.7±25.0	83.8±18.8	0.632
HDL	45.1±7.4	27.9±7.8	0.0001
LDL	67.6±26.4	51.6±23.4	0.103
VLDL	17.5±5.0	16.7±3.7	0.632
AI	2.6±0.84	3.6±1.1	0.040
Length	46.1±0.6	44.4±1.3	0.001
Head circumference	33.7±0.7	31.5±1.4	0.001
Ponderal Index	2.0±0.4	1.7±0.2	0.002
Birth weight	2.0±0.45	1.4±0.1	0.001

Birth weight, ponderal index, head circumference, and HDL all have significant p values. Of the total number of premature infants analysed in the study, 38 were classified as either small for gestational age (SGA) or appropriate for gestational age (AGA). High density lipoproteins (HDL) had a significant proportional difference between preterm AGA and preterm SGA (p<0.05).

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 05, 2023

Discussion

Researchers have recently become increasingly interested in cord lipids since atherogenic changes are assumed to have their origins in childhood and lipid issues can be traced back to childhood. Lipid profile is a helpful predictor of cardiovascular health since abnormalities in it are significantly associated with an increased risk of cardiovascular disease and death. Levels of lipids and lipoproteins in the cord should be indicative of the new born's plasma lipid metabolism at birth because the majority of foetal lipids are produced de novo through the conversion of glucose to different fatty acid-containing molecules. Investigating the composition of cord blood lipids is similar to investigating foetal and new born lipid metabolism because only a small portion of them are obtained through placental circulation.

Atherosclerosis is thought to begin as a result of high plasma levels of cholesterol and/or triglycerides. In addition, only low-density lipoprotein (LDL) and high-density lipoprotein (HDL) are currently regarded by many researchers as major risk factors for the development of atherosclerotic vascular illnesses. Analyses of the cord lipid profile are a promising method for the early detection of infants at higher risk because it has been hypothesised by a number of studies that atherosclerotic lesions have their origins in infancy [13, 14].

Abnormal lipid profiles in preterm and SGA new borns have been linked in numerous studies to the onset of cardiovascular disease. The goal of the current study was to identify abnormal lipid profiles as soon as feasible after birth, especially in preterm and SGA neonates, in order to monitor high-risk new borns. There was no statistically significant difference between the term boy and girl cord lipid profile levels in the current study. Preterm infants in this study had LDL levels that were significantly higher than term infants (preterm 54.91+0.9 vs. term 24.6, p = 0.013). Between the distributions of TC, TG, HDL, VLDL, and AI at term and preterm, there was no statistically significant change. According to a study by N.Harias and P.T. Acharya *et al*. [15, 16], preterm newborns exhibited higher TG and TC levels, however the difference is only statistically significant for TC ($p<0.001$).

In preterms, the TC value was significantly greater ($p<0.001$), according to research by Mathur *et al*. Preterm newborns had much higher TC, LDL and HDL readings than full-term infants, but far lower TG readings, according to research by Jane Oba *et al*. Total cholesterol (TC), low-density lipoprotein (LDL), and high-density lipoprotein (HDL) levels were higher in preterm neonates than term neonates ($p<0.001$), however HDL levels did not differ statistically between the two groups. The AI readings for preterm and full-term were not significantly different. According to a study by A K Kalra *et al*., all cord lipid profile values were lower in preterm neonates than in term neonates, albeit the difference was only statistically significant for TC levels ($p<0.001$) and not for HDL or LDL. The TC of term and preterm infants differ statistically significantly ($p<0.05$), according to the study by Jagadish Singh *et al*. No statistically significant change between TG levels before and after delivery was discovered by Mishra *et al*. Our results suggest that higher cord lipid levels in preterm neonates may reflect a deficiency in circulating energy resulting from a deficiency in both hepatic glucose and subcutaneous adipose storage. An increase in cholesterol levels in cord blood may be a sign of the metabolic adjustment needed to provide enough energy, particularly to organs like the brain [17, 18].

In the current study, Term SGA neonates had substantially higher total cholesterol and very low density lipoprotein (VLDL) levels than TERM AGA neonates (mean aga term 83.4 +23.2 and SGA term mean $=101.42\pm6.9$, p value 0.0001 and 11.06 ± 3.2, respectively). Due to decreased lipoprotein lipase activity in SGA neonates, triglyceride and very low density lipoprotein (VLDL) synthesis are increased, and metabolism is decreased. Compared to the current inquiry, similar increases in triglyceride levels were observed in the other trials. The study by Daniel *et al*. had the highest triglyceride rise, with a value of 85.6 mg/dl. The results of earlier studies by Kelishadi *et al*. and Pardo *et al*., which revealed a comparable decline in HDL levels in cord blood, were supported by the findings of the current investigation. Investigations by Jones *et al*., Daniel *et al*., Wang *et al*., and Hossain *et al*. [19, 20] produced findings that were similar.

Because SGA neonates do not have access to glucose as a fuel source, alternative sources such as amino acids and lipids are used instead, and glucose is produced (gluconeogenesis), leading to increased hepatic lipid production (especially VLDL and chylomicrons) in SGA compared to AGA in the current study. Lower peripheral lipid utilisation and decreased lipoprotein lipase enzyme activity in SGA infants can be used to explain the higher plasma lipid levels. The programming phenomenon, in which stimulation or insult during a crucial period of intrauterine life could also result in changes to physiology and metabolism in later life, was proposed by the Barker hypothesis, which showed a correlation between low birth weight and an increased prevalence of cardiovascular disease, hypertension, and type 2 diabetes mellitus. In the current study, it was discovered that the Term SGA group had greater cord blood lipid profile values than the Term AGA group. Preterm SGA new borns had greater TC, LDL, VLDL, and TG, although these differences were not statistically significant compared to Preterm AGA neonates lower cord lipid profiles in terms of HDL and LDL. As compared to preterm AGA, preterm SGA was associated with higher levels of total cholesterol, triglycerides, low-density lipoprotein, and high-density lipoprotein (P 0.0001, 0.01, 0.0001, and 0.0001, respectively) [20, 21]. Likewise, as compared to term AGA, term SGA was associated with higher levels of total cholesterol, triglycerides, LDL and HDL.

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 05, 2023

No statistically significant difference was discovered between the TC values for term AGA and preterm AGA, or between term SGA and preterm SGA, according to research by Ajay Kumar *et al*. The TG values for term SGA and preterm SGA as well as term AGA and preterm AGA differed statistically significantly ($p<0.05$). Neonates born as a result of intra uterine malnutrition have low birth weights. Due to situations that encourage the breakdown of foetal adipose tissue and the release of free fatty acids that cannot be oxidised for energy but are instead synthesised into triglyceride in the liver, low birth weight neonates frequently have high levels of triglyceride. Stunting inside the uterus can result from placental insufficiency. The widespread perception is that unhealthy adult behaviours like smoking, eating a high-fat diet, and inactivity cause heart disease. The foetal hypothesis implies that, in addition to inherited determinants and lifestyle factors, foetal nutrition should be considered a risk factor in the aetiology of the disease. It does not downplay the importance of these adult factors. According to study, coronary heart disease can be linked to early development. A risk factor for developing coronary artery disease later in life is hyperlipoprotenemia at birth. According to our research, low birth weight infants have greater TG, TC, and VLDL levels in cord blood while preterm new borns have higher LDL levels. This demonstrates that an undernourished foetus must adapt in order to survive, and that as a result, it may be susceptible to hyperlipidemia. To prevent hyperlipidemia from causing heart problems later in life, improve foetal growth and check for it in infancy and early childhood. The investigation going on now the gestational age was found to be 36.78$\pm$2.1 weeks, which is similar to the study by Pardo *et al*., which included both term and preterm SGA neonates in its study group and discovered a gestational age of 35.5$\pm$70.11 weeks. Only full-term SGA neonates were examined in other investigations, including those by Kelishadi *et al*. and Wang *et al*. [21, 22].

The table shows that the birth weights for the investigations by Pardo *et al*. and Jones *et al*. were 2.04$\pm$0.76 and 2.07$\pm$0.53, respectively; both of these results were statistically significant at the $p<0.01$ level. However, because Wang *et al*. and Kelishadi *et al*. only included term SGA neonates in their sample, the mean birth weight in both studies was larger than it was in the present study and the study by Pardo *et al*., which included both term and preterm SGA. Very similar results (44.58$\pm$0.48) that were statistically significant ($p<0.01$) were obtained by Pardo *et al*. With a p value of $p<0.001$, the average Ponderal index in the current study was 1.83$\pm$0.23, making it statistically significant. In contrast, the study by Kelishadi *et al*. had an average Ponderal index of 2.18, which was not statistically significant. The gestational age was found to be 36.6$\pm$2.1 weeks, which is comparable to the research by Pardo *et al*., which included both term and preterm SGA neonates in its study group and discovered a gestational age of 35.57$\pm$0.11 weeks. Only full-term SGA neonates were examined in another research, including those by Kelishadi *et al*. and Wang *et al*. [23, 24].

Conclusion

There was no statistically significant difference in cord lipid profile between males and females in both term and preterm neonates. Preterm neonates had higher values of LDL compared to Term neonates and values were statistically significant ($p<0.01$). SGA neonates had significantly higher mean value (82.08 ±21.3) ($p<0.001$) TG compared to AGA neonates, and HDL, VLDL, AI, P values were significantly higher in SGA neonates. Term SGA had significantly higher cord blood lipid profile values ($p<0.001$) compared to the Term AGA, except, LDL ($p=0.8$) HDL ($p=0.3995$) levels. Preterm AGA had higher cord lipid profile values of HDL compared to the Term AGA neonates and values were significantly significant ($P <0.0001$). Preterm SGA neonates had higher values of TC, TG, LDL, VLDL compared to preterm AGA. Term SGA neonates had higher values of TC, TG, VLDL had higher values compared to term AGA neonates. AI was significantly higher in <1.5 kg ($P<0.001$) which indicated increased risk for atherosclerosis.

Funding support: None.

Conflict of interest: None.

References

1. Joint statement by the American Heart Association. The Cholesterol Facts. Circulation; c1980. p. 81.
2. Glueck CJ, Tsang RC. Pediatric type-II hyperlipoproteinemia. Effect of diet on plasma cholesterol in the first year of life. Am J Clin Nutr. 1972;25:224-230.
3. Berenson GS, Blonde CV, Farris RP, Foster TA, Frank GC, Srinivasan SR, *et al*. Cardiovascular disease risk factor variables during the first year of life. Am J Dis Child. 1979,133:1049-1058.
4. Lauer RM, Connor WE, Leaverton PE, Reiter NA, Clake MS. Coronary heart disease risk factors in school children: The Muscatine study J Pediatr. 1975,86:697-706.
5. Kent L, Thornburg. Fetal Origins of Cardiovascular Disease. American Academy of Pediatrics. 2004 Dec;(5):527-34.
6. Sawant AM, Shetty D, Mankeshwar R, Ashavaid TF. Prevalence of dyslipidemia in young adult Indian population. J Assoc Physicians India. 2008 Feb;56:99-102.

ISSN:0975 -3583,0976-2833 VOL14, ISSUE 05, 2023

7. Meenakshi Sharma, Nirmal Kumar Ganguly. Premature coronary artery disease in Indians and its associated risk factors, Vascular Health and Risk Management, Dove Medical Press Limited. 2005;1(3):217-225.
8. Rasa Verkauskiene, Paul Czernichow, Claire LM. Long-term metabolic consequences of being born small for gestational Age. Expert Review of Endocrinology and Metabolism. 2006 May;(1):439-447.
9. Jones JN, Taylor Gercel, Taylor Doughlas D. Altered cord serum lipid levels associated with small for gestational age infants. Journal of American College of Obstetricians and Gynecology. 1999 Oct;93:527-531.
10. Molina M, Casnueva V, Cid X, Chiang MT, Ferrada MC, Perez R, *et al*. Lipid profile in new borns with intrauterine growth retardation. Rev Med Chil. 2000 July;128(7):741-748.
11. Pardo IMCG, Geloneze B, Tambascia MA, Barros Filho AA. Atherogenic lipid profile of Brazalian near term new borns. Brazilian Journal of Medical and Biological Research. 2005 May;38(5):755-760.
12. Hossain MA, Islam MN, Shahidullah M, Akhter H. Serum triglyceride level in IUGR babies and its comparison with preterm AGA and term normal babies. My Men Singh Med J. 2006 Jul;15(2):180-182.
13. Daniel M Lane, Walter J. Mcconathy. Factors affecting the lipid and Apo-lipoprotein levels of cord sera. Pediatr. Res. 1983;17:83-91.
14. Misra PK, Tiwari DK, Sharma B, Kaul R. Free fatty acids and Triglycerides in normoglycemic low birth weight newborns in early neonatal period. Indian J Pediatr. 1984;51:637-641.
15. Haridas N, Acharya PT. Serum Lipid Status in neonates. Indian Pediatr. 1984;21:327-334.
16. Mathur PP, Prasad R, Jain SK, Pandey DH, Singh SP. Cord blood Cholesterol in term and preterm new-borns. Indian Pediatr. 1986;23:103-106.
17. Kalra A, Kalra K, Agarwal MC, Prasad R, Pant MC, Bhatia R, *et al*. Serum lipid profile in term and preterm infants in early neonatal period, Indian Pediatr. 1988;25:977-981.
18. Dogra J, Sengar GS, Mathur HC, Misra SN. Serum lipids in neonatal cord blood in families with diabetes mellitus Type-1. Indian Pediatr. 1988;25:267-271.
19. Kumar A, Gupta A, Malhotra VK, Thirupuram S, Gaind B. Cord blood lipid levels in low birth weight new-borns. Indian Paediatr. 1989;26:571-574.
20. Van-Der-Schouw YT, ALMD, Hornstra G, Bullstraw, Ramkers MT, Huisjes HJ. Fatty acids composition of serum lipids of mother and their babies after normal and hypertensive pregnancies. Prostaglandins-Leucot-Essent-Fatty-acids. 1991;44:247-252.
21. Kherkeulidze P, Johansson J, Carlson LA. High density lipoprotein particles size distribution in cord blood. Acta Paediatr Scand. 1991;80:770-779.
22. Averna MR, Barbagallo CM, Dipaola G, Labisi M, Pinna G, Marino G, *et al*. Total cholesterol, IDL-Cholesterol and lipoprotein B in umbilical cord blood: cross sectional study. Minerva Pediatr. 1992;44:395-399.
23. Xinli Wang, Yunpu Cui, Xiaomei Tong, Hongmao Ye, Song Li. Glucose and Lipid metabolism in Small for gestational age infants. The Journal of Clinical Endocrinology & Metabolism. 1992 Feb;(2):681-684.
24. Averna MR, Barbagallo CM, Di-Paola G, La-Placa FP, Scafidi V, Dimita U, *et al*. Transient Neonatal Hypercholesterolemia, Lack of relationships with dehydroepiandrosterone sulphate. Horm-Metab-Res. 1993,25:271-274.