

Research Article

Volume 14 Issue 3 - November 2024
DOI: 10.19080/AJPN.2024.14.555945

Acad J Ped Neonatol

Copyright © All rights are reserved by Nehad Refhat Mostafa Sallam

Studies of Lipid Profiles among Infants with Respiratory Distress Syndrome



Nehad Refhat Mostafa Sallam*, Shymaa El-Sayed Ali Mohamed, Lamyaa Hussain Abdulrahman Seliem, Hebatallah Shoukry Mohamed Soliman, Marwa Foad Abdelrahman Asker and Ibrahim Mohamed Eladel Ibrahim Abouelkhair

Pediatrics department, Shebin Elkom Teaching Hospital, Shebin Elkom, Menoufia, Egypt

Submission: October 17, 2024; **Published:** November 04, 2024

***Corresponding author:** Nehad Refhat Mostafa Sallam, Pediatrics department, Shebin Elkom Teaching Hospital, Shebin Elkom, Menoufia, Egypt

Abstract

Background: Respiratory distress syndrome (RDS) is known as hyaline membrane disease (HMD). It is the major cause of neonatal respiratory distress, especially in preterm infants. Infants with RDS present with tachypnea, cyanosis, grunting, subcostal and intercostal retractions and nasal flaring. Oliguria with mild generalized edema may be present. Oxygen requirement may increase rapidly and is typically higher than that seen in infants with transient tachypnea of the newborn (TTN).

Objective: To evaluate the relationship between the maternal and cord blood lipid profiles in preterm babies with respiratory distress syndrome.

Methods: This is a case control study which was conducted on 50 preterm neonates admitted to the neonatal intensive care unit of Shebin El-Kom teaching hospital between January 2020 and October 2021.

Results: The mean of total cholesterol was significantly higher in infants whose mothers had received antenatal steroid for lung maturity than those whose mothers had not ($P<0.05$). But there was no significant difference in the mean of levels of TG, HDL-cholesterol, LDL-cholesterol and VLDL cholesterol between both groups. Also, There is a positive correlation between weight gain during pregnancy and neonatal cord blood levels of (Total cholesterol, TG, HDL-C, LDL-C and VLDL-C). There is a positive correlation between maternal blood levels of (total cholesterol, TG, LDL-C and VLDL-C) and their neonatal cord blood levels of (total cholesterol, TG, HDL-C, LDL-C and VLDL-C). There is a positive correlation between the maternal blood level of HDL-C and their neonatal cord blood levels of (total cholesterol and HDL-C). Also, the cutoff values of cord blood (total Cholesterol, HDL-C, and LDL-C) below which RDS can be predicted are 80, 45.5 and 23.2 mg/dl respectively with a sensitivity of 83.3%, 86.7% and 86.7% and with specificity of 95%, 80% and 70% respectively.

Conclusion: RDS is accompanied with lipid alteration in the infants and their mothers. The results of this study point to the importance of measuring maternal serum and cord blood lipid profile as a predictor for the occurrence of RDS.

Keywords: Cord Serum; Hyaline Membrane Disease; Respiratory Distress Syndrome; Preterm Infants

Abbreviation: RDS: Respiratory Distress Syndrome; HMD: Hyaline Membrane Disease; TTN: Transient Tachypnea of the Newborn; VLDL: Very Low-Density Lipoproteins; LDL: Low-Density Lipoproteins; HDL: High Density Lipoproteins; LDL-C: Low-Density Lipoprotein-Cholesterol; TC: Total Cholesterol Concentration; TG: Triglycerides Concentration; VLDL-C: Very Low-Density Lipoprotein-Cholesterol; LPL: Lipoprotein Lipase; G3P: Glycerol-3-Phosphate; ADP: Adenosine-5-Diphosphate; ATP: Adenosine-Triphosphate; GPO: Glycerol Phosphate Dehydrogenase; DAP: Dihydroxyacetone Phosphate; H2O2: Hydrogen Peroxide; POD: Presence Of Peroxidase

Introduction

Respiratory distress syndrome (RDS) is also known as hyaline membrane disease (HMD). It is the major cause of neonatal respiratory distress, especially in preterm infants [1]. Typically, RDS affects preterm infants below 35 weeks of gestational age, however, older infants with delayed lung maturation of different

etiologies can also be affected [2]. The outcome of RDS has improved in recent years with the increased use of antenatal steroids to improve pulmonary maturity, early postnatal surfactant therapy to replace surfactant deficiency, and proper advanced techniques of ventilation to minimize damage to the immature

lungs [3]. Infants with RDS present with tachypnea, cyanosis, grunting, subcostal and intercostal retractions and nasal flaring. Oliguria with mild generalized edema may be present. Oxygen requirement may increase rapidly and is typically higher than that seen in infant with transient tachypnea of the newborn (TTN) [4].

The severity of respiratory distress is assessed by Silverman-Anderson Score and Downes' Score. While the Silverman-Anderson Retraction Score is more suited for preterms with HMD, the Downes' Score is more comprehensive and can be applied to any gestational age and condition. Scoring should be done at half hourly intervals and a chart maintained to determine progress. A progressively increasing FiO_2 requirement to maintain a saturation of 90-92% in a preterm and 94- 96% in a term baby is also a sensitive indicator of the severity and progress of distress [5]. Lipids are heterogeneous group of water insoluble (hydrophobic) organic molecules that can be extracted from tissues by non-polar solvent. Because of their property of insolubility in water they are mainly found in different compartments, mostly associated with membranes enclosing various cell organelles. In adiposites as droplets of triglycerides, when transported in cytosol, they are in the form of lipoprotein particles [6]. This study aims to evaluate the relationship between the maternal and cord blood lipid profiles in preterm babies with respiratory distress syndrome.

Patients and Methods

A case control study which was conducted on 50 preterm neonates admitted to the neonatal intensive care unit of Shebin El-Kom teaching hospital between January 2020 and October 2021. The newborns were divided into two groups: Group A: (STUDY GROUP) included 30 preterm newborns that developed RDS and their mothers, Group B: (CONTROL GROUP) It included 20 preterm newborns that did not develop RDS and their mothers.

Patients' Criteria

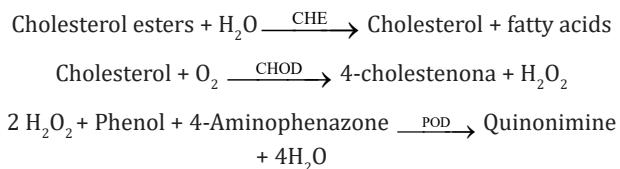
We included Preterm neonates \leq 36 weeks gestational age, Mature neonates $>$ 36 weeks gestational age, Small /large for gestational age, Neonatal asphyxia, Major congenital anomalies, Mothers whose pregnancies were complicated by hypertension and pre-eclampsia, or with history of endocrine disorders as diabetes, thyroid or adrenal problems, Infant whose mothers were taking drugs affecting lipid metabolites such as steroid (except for fetal lung maturation) and ritodrine.

All the Included Newborns Were Subjected to the Following

Maternal history: Maternal age, Menstrual history including the first day of last menstrual period. The diagnosis of RDS was established by meeting all the following criteria: Physical examination notable for chest wall retractions and cyanosis on room air, Compatible X-ray demonstrating diffuse alveolar atelectasis, Arterial blood gas documentation of metabolic acidosis, hypoxemia and hypercapnia. **Cord blood sample:** Cord blood sample (2ml) was withdrawn from the umbilical vein

immediately after delivery in a plain tube for the measurement of lipid profile. Maternal and cord blood samples were left to clot at room temperature for 20 minutes then centrifuged for 20 minutes at 2500 round per minute and samples stored at -20°C until analysis within 15 days, for estimation of the following elements: Serum total cholesterol, serum HDL-cholesterol, serum LDL-cholesterol, serum VLDL-cholesterol and serum Triglyceride.

- **Serum Total Cholesterol:** The cholesterol present in the sample originates a colored complex, according to the following reaction:



The intensity of the color formed is proportional to the cholesterol concentration in the sample.

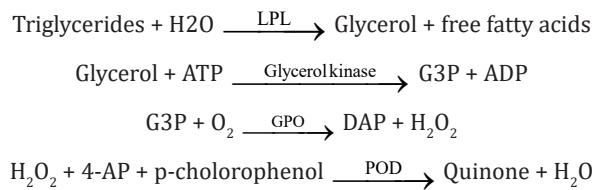
- **Serum High Density Lipoprotein-Cholesterol (HDL-C):** The chylomicrons, very low-density Lipoproteins (VLDL) and low-density lipoproteins (LDL) are precipitated by phosphotungstic acid and magnesium chloride. After centrifugation the clear supernatant containing high density lipoproteins (HDL) is used for the determination of HDL cholesterol.

- **Estimation of Low-Density Lipoprotein-Cholesterol (LDL-C):** The LDL cholesterol concentration (LDL-C) is calculated from the total cholesterol concentration (TC), the HDL cholesterol concentration (HDL-C) and the triglycerides concentration (TG) according to Friedwald equation. $\text{LDL-C} = \text{total cholesterol} - \text{HDL} - \text{triglyceride}/5$ [mg/dl]. This formula is not used for triglycerides levels above 400 mg/dl

- **Estimation of Very Low-Density Lipoprotein-Cholesterol (VLDL-C):**

This will be calculated from the equation: $\text{VLDL} = \frac{\text{TG}}{5}$

- **Estimation of Serum Triglycerides:** Sample of triglycerides incubated with lipoprotein lipase (LPL), liberate glycerol and free fatty acids. Glycerol is converted to glycerol-3-phosphate (G3P) and adenosine-5-diphosphate (ADP) by glycerol kinase and adenosine-triphosphate (ATP). G3P is then converted by glycerol phosphate dehydrogenase (GPO) to dihydroxyacetone phosphate (DAP) and hydrogen peroxide (H_2O_2). In the last reaction, hydrogen peroxide (H_2O_2) reacts with 4-aminophenazone (4-AP) and p-chlorophenol in presence of peroxidase (POD) to give a red colored dye:



The intensity of the color formed is proportional to the triglyceride's concentration in the sample.

Statistical Analysis

All data were collected, tabulated, and statistically analyzed using SPSS 26.0 for Windows (SPSS Inc., Chicago, IL, USA). Qualitative data were described using numbers and percentages. Quantitative data were described using range (minimum and maximum), mean, standard deviation, and median. Student's t-test, Pearson correlation coefficient test, chi square test (χ^2 -value), P value, probability of chance, indicates significance when P value ≤ 0.05 , highly significant when P value ≤ 0.01 and P value

>0.05 was considered not statistically significant.

Results

In the current study, the studied mothers and their newborns includes 15 neonates whose gestational age from 34 to 36 weeks; they represent 13.3% of infants with RDS and 55.0% of infants without RDS, 22 neonates whose gestational age from 31 to 33 weeks; they represent 43.3% of infants with RDS and 45.0% of infants without RDS, 13 neonates whose gestational age from 28 to 30 weeks; they represent 43.3% of infants with RDS and 0% of infants without RDS (Table 1).

Table 1: Gestational age among the studied groups.

	Gestational Age in Weeks	Group (A) Preterm with RDS		Group (B) Control Preterm	
		No.	%	No.	%
GA groups	28 - 30	13	43.30%	0	0.00%
	31 - 33	13	43.30%	9	45.00%
	34 - 36	4	13.30%	11	55.00%

Also, significant difference was observed as regarding the mean of levels of cord blood lipid profile between male and female ($P > 0.05$). (Table 2) Also, there was no significant difference in the

mean of levels of cord blood lipid profile between infants born vaginally and those born by cesarean section ($P > 0.05$) (Table 3).

Table 2: Cord blood lipid profile as regard neonatal sex.

	Sex				t-test	P-value		
	Male		Female					
	Mean	SD	Mean	SD				
Total Cholesterol	77	13	79	13	-0.515	>0.05		
TG	59	14	63	11	-1.146	>0.05		
HDL	42	10	43	10	-0.143	>0.05		
LDL	23.1	3.5	23.8	3.9	-0.642	>0.05		
VLDL	11.8	2.8	12.6	2.2	-1.13	>0.05		

Table 3: The effect of mode of delivery on the cord blood lipid profile.

	Delivery				t-test	p-value		
	VD		CS					
	Mean	SD	Mean	SD				
Total Cholesterol	75	15	81	10	-1.747	>0.05		
TG	58	13	63	13	-1.259	>0.05		
HDL	40	11	44	8	-1.319	>0.05		
LDL	22.5	3.1	24.2	4	-1.689	>0.05		
VLDL	11.7	2.6	12.6	2.6	-1.239	>0.05		

Additionally, the mean of total cholesterol was significantly higher in infants whose mothers had received antenatal steroid for lung maturity than those whose mothers had not ($P < 0.05$). But there was no significant difference in the mean of levels of TG, HDL-cholesterol, LDL-cholesterol and VLDL cholesterol between

both groups (Table 4). Additionally, there is a positive correlation between gestational age, birth weight, length, Apgar score at 1 and 5 min and the cord blood levels of (total cholesterol, triglyceride, HDL-C, LDL-C and VLDL-C). There is a positive correlation between ponderal index and each of total cholesterol and LDL-C (Table 5).

Table 4: The effect of antenatal steroid as a drug used for lung maturity on cord blood lipid profile.

	Antenatal Steroid							
	Positive		Negative					
	Mean	Standard Deviation	Mean	Standard Deviation				
Total cholesterol	81	12	77	15	2.6	<0.05*		
TG	63	13	59	13	1.132	>0.05		
HDL	43	9	42	11	0.25	>0.05		
LDL	23.8	3.9	23.1	3.5	0.663	>0.05		
VLDL	12.6	2.5	11.7	2.7	1.158	>0.05		

Table 5: Correlations between neonatal clinical characteristics and their cord blood lipid profile.

Correlations						
		Total Cholesterol	TG	HDL	LDL	VLDL
GA	r	.727**	.500**	.693**	.353*	.503**
	P	<0.01**	<0.01**	<0.01**	<0.05*	<0.01**
wt (kg)	r	.790**	.555**	.748**	.388**	.557**
	P	<0.01**	<0.01**	<0.01**	<0.01**	<0.01**
Length (cm)	r	.702**	.467**	.698**	0.276	.468**
	P	<0.01**	<0.01**	<0.01**	<0.05*	<0.01**
Ponderal Index	r	0.328	0.231	0.115	.339*	0.234
	P	<0.05*	>0.05	>0.05	<0.05*	0.101
Apgar1	r	.547**	.351*	.478**	.402**	.354*
	P	<0.01**	<0.05*	<0.01**	<0.01**	<0.05*
Apgar5	r	.598**	.418**	.530**	.396**	.422**
	P	<0.01**	<0.01**	<0.01**	<0.01**	<0.01**

Also, there is a positive correlation between weight gain during pregnancy and neonatal cord blood levels of (Total cholesterol, TG, HDL-C, LDL-C and VLDL-C). There is a positive correlation between maternal blood levels of (total cholesterol, TG, LDL-C and VLDL-C) and their neonatal cord blood levels of (total cholesterol, TG, HDL-C, LDL-C and VLDL-C). There is a positive correlation between the maternal blood level of HDL-C and their neonatal cord blood levels of (total cholesterol and HDL-C) (Table 6). Additionally, the cutoff values of cord blood (total Cholesterol, HDL-C, and LDL-C) below which RDS can be predicted are 80, 45.5 and 23.2 mg/dl respectively with a sensitivity of 83.3%, 86.7% and 86.7% and with specificity of 95%, 80% and 70% respectively (Table 7, Figure 1).

Discussion

Respiratory distress syndrome (RDS) in premature infants remains the major life-threatening neonatal disease affecting 1 to 2% of newborn infants despite antenatal steroid and surfactant treatments. RDS is principally associated with developmental deficiency in synthesis, intracellular processing, and secretion of pulmonary surfactant, required to reduce surface tension at the air-liquid interface of the distal conducting airways and alveoli [7]. It is most common in infants born at fewer than 28 weeks' gestation and affects one third of infants born at 28 to 34 weeks' gestation, but occurs in less than 5 percent of those born after 34 weeks' gestation [8]. Lipids play a major role in fetal development.

Changes in the availability of lipid components, like those produced by changes in dietary fatty acids, are known to have implications in fetal and postnatal development [9].

Cholesterol is the second most abundant lipid component of pulmonary surfactant, composing from 10 to 25% (by mole fraction) of total surfactant lipid. Cholesterol represents over

50% of the neutral lipid of both the surfactant and the lamellar body fractions. De novo synthesis of cholesterol accounts for only 1% of the surfactant cholesterol, the remainder is derived from lipoprotein [10]. Fetal metabolism and consequently fetal growth directly depend on the nutrients crossing the placenta, so lower maternal lipid profile could delay fetal lung maturation and increase the risk for RDS [11].

Table 6: Correlations between maternal (clinical and laboratory) data and their neonatal cord blood lipid profile.

Correlations						
		Total Cholesterol	TG	HDL	LDL	VLDL
Mother Age (years)	r	0.134	0.153	0.105	0.085	0.151
	P	>0.05	>0.05	>0.05	>0.05	>0.05
Prepregnant (wt)	r	0.107	0.024	0.142	-0.021	0.021
	P	>0.05	>0.05	>0.05	>0.05	>0.05
Mother Height	r	0.05	-0.022	0.058	0.038	-0.023
	P	>0.05	>0.05	>0.05	>0.05	>0.05
Mother BMI	r	0.12	0.04	0.16	-0.037	0.037
	P	>0.05	>0.05	>0.05	>0.05	>0.05
Weight Gain During Pregnant	r	.468**	.380**	.418**	0.361	.381**
	P	<0.01**	<0.01**	<0.01**	<0.05*	<0.01**
Mother Total Cholesterol	r	.896**	.679**	.840**	.430**	.679**
	P	<0.01**	<0.01**	<0.01**	<0.01**	<0.01**
Mother TG	r	.821**	.707**	.750**	.388**	.710**
	P	<0.01**	<0.01**	<0.01**	<0.01**	<0.01**
Mother HDL	r	-0.334	-0.177	-0.336	-0.069	-0.163
	P	<0.05*	>0.05	<0.05*	>0.05	>0.05
Mother LDL	r	.865**	.636**	.817**	.412**	.634**
	P	<0.01**	<0.01**	<0.01**	<0.01**	<0.01**
Mother VLDL	r	.821**	.707**	.750**	.388**	.710**
	P	<0.01**	<0.01**	<0.01**	<0.01**	<0.01**

Table 7: Cord blood lipid profile in predicting RDS.

Test Result Variable(s)	Area Under the Curve (AUC)	RDS if Less Than or Equal To	Sensitivity	Specificity
Total Cholesterol	0.946	80	83.30%	95%
HDL-C	0.888	45.5	86.70%	80%
LDL-C	0.807	23.2	86.70%	70%

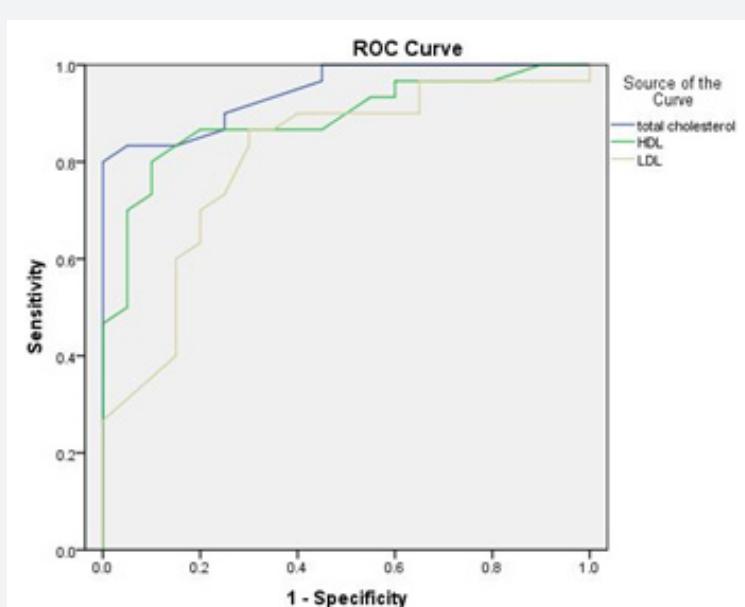


Figure 1: Receiver Operating Characteristic (ROC) curve.

The aim of this study was to compare the maternal and cord lipid profiles of preterm infants with respiratory distress syndrome and a control group without respiratory distress syndrome. The study groups consisted of 30 preterm neonates with RDS, their gestational ages ranging from 28 to 36 weeks, birth weights ranging from (850 to 2400 g) and their mothers; and 20 preterm neonates without RDS, their gestational ages ranging from 31 to 36 weeks, birth weights ranging from (1500 to 2400 g) and their mothers were enrolled in this study as a control group. The present study revealed that weight gain during pregnancy was significantly lower in mothers of preterm with RDS, and also there was significant difference regarding mode of delivery and antenatal steroid, however; no statistically significant difference regarding maternal age, maternal weight, maternal BMI, pregravid height, the ruptured membrane > 24 hr. and parity between the mothers of RDS neonates and mothers of the control group.

In agreement with these results Teslova et al. [12] who reported that inadequate maternal weight gain during pregnancy may play a significant role in immaturity of the pulmonary surfactant system and may thus offer a predictor of respiratory distress syndrome in premature babies. Butte et al. [13] Suggested that appropriate, but not excessive, gestational weight gain is needed to optimize infant birth weight and minimize maternal postpartum fat retention. So, appropriate weight gain during pregnancy is related to the neonatal outcome. Sollid et al. [14] observed that poor weight gain during pregnancy, associated with LBW, prematurity, and maternal delivery complications. On the other hand, Gunes et al. [15] found no significant difference in weight gain of mothers of preterm infants with or without RDS. As regard mode of delivery, our findings are in agreement with Hansen et al. [16] and Kolas et

al. [17] who reported that newborn delivered by elective cesarean section around term have an increased risk of overall and serious respiratory morbidity than infant delivered vaginally. The relative risk increased with decreasing gestational age.

As regard the effect of sex of the newborn on the cord blood lipid profile, no significant difference was found between male and females in our study. This agrees with the studies done by Descamps et al. [18], Rodie et al. [19] and Saleh et al. [20] that reported no significant differences in cord blood lipid levels were seen between male and female newborns. However, significant gender differences in cord lipids have been noted by Kelishadi et al. [21] and Kharb et al. [22] who found that Cord blood of female newborns had higher total cholesterol, HDL-C, LDL-C, Apo A as compared to male newborns. Aasvee et al. [23] speculated that female fetuses need different hormonal stimulation, and a higher level of blood cholesterol, compared to males.

As regards the effect of mode of delivery on the cord blood lipid profile, no significant difference was found in the present study. This agrees with Gunes et al. [15] who demonstrated that the neonatal lipid profiles are not related to the mode of delivery. However, Bansal et al. [24] observed that methods of delivery that associated with higher fetal distress, such as vaginal delivery, appear to be associated with higher cord lipids compared with elective caesarean section. Also, Rodie et al. [19] reported that emergency caesareans increase fetal stress and lipids more than that of elective section.

As regard the effect of antenatal steroid on the cord blood lipid profile, this study revealed that there was significant difference in total cholesterol, but no significant difference in TG, HDL-

cholesterol, LDL-cholesterol and VLDL- cholesterol between both groups. Parker et al. [25] reported that antenatal steroids stimulate hepatic production and secretion of lipids and certain lipoproteins in the fetus. In the present study there was a positive correlation between gestational age, birth weight, length, Apgar score at 1 & 5 min and each of total cholesterol, triglyceride, HDL-cholesterol, VLDL- cholesterol and LDL- cholesterol levels. Also, significant positive correlations were found between ponderal index, and each of total cholesterol and LDL- cholesterol levels.

In agreement with these results Lane et al. [26] who reported that cord blood lipid profile in infants with RDS depends on their birth weight. They found that cord serum lipid and apolipoprotein levels were significantly elevated in large (2,000-2,499 g) RDS infants, but lower levels were found in smaller (1,000-1,999 g) RDS infants. Also, Kelishadi et al. [21] found a significant correlation between birth weight and cord serum lipid. Moreover, Huxley et al. [27] documented a retrospective association between birth weight and lipid levels and suggested that birth weight and body size have a great effect on blood cholesterol levels. But Jain et al. [28] demonstrate that birth weight was inversely correlated with TC, LDL-C, VLDL-C, TG, Apo B. Moreover, Badiee and Kelishadi R [29] found that There were no significant correlations between infant's ponderal index, birth weight, length, and TC, LDL-C, HDL-C, apolipoprotein A, apolipoprotein B. This difference between the results of these studies may be explained by the difference in the mean of gestational ages of the newborns selected in these studies; some may have included very near-term or very preterm newborns.

In the present study a positive correlation was detected between maternal weight gain during pregnancy and neonatal cord blood Total cholesterol, TG, HDL-C, LDL-C and VLDL-C. In agreement with our findings, Gunes et al. [15] documented that weight gain during pregnancy was related to both maternal and infants' lipid profile and added the necessity of adequate gestational weight gain for optimal pregnancy outcome is recognized; however, the specific components of gestational weight gain that are critical for fetal growth and development are not delineated clearly. Kaser et al. [30] suggested that both increased and decreased nourishment in utero affect the neonatal lipoprotein profile. However, Kelishadi et al. [21] observed that maternal weight can affect fetal growth and maturation, but their impact on cord lipid profile remains controversial. Normal fetal growth is a result of complex interactions among the three components of the maternal-placental-fetal unit. Nutritional status of the mother is the most important maternal factor leading to intrauterine growth retardation [21].

In this study a positive correlation was found between all maternal serum (total cholesterol, TG, LDL-C and VLDL-C) and each of cord blood (total cholesterol, TG, HDL-C, LDL-C, and VLDL-C). Also, a positive correlation between maternal HDL-C and neonatal cord blood (total cholesterol and HDL-C) was detected.

In agreement with the present study Bansal et al. [24] and Gunes et al. [15] found that there was significant positive correlation between maternal and cord lipid profile of preterm infants that could be explained by the fact that maternal cholesterol can cross the placenta, and cholesterol concentrations in maternal serum affect concentrations in neonates. Rodie et al. [19] found a strong correlation between maternal total cholesterol, HDL cholesterol and newborn HDL cholesterol. This could be explained by Studies done by Burke et al. [31] and Yoshida and Wada [32] who demonstrated the ability of maternal cholesterol to be transported to the fetus even if fetuses can synthesize cholesterol de novo.

The fetus is dependent for its growth and development on the nutrients provided by the mother. The number of substrates reaching the placenta strictly depends on maternal diet and metabolism [33]. Although lipids cross the placental barrier with difficulty, changes in lipid metabolism taking place on the maternal side could also contribute to the fetal development [34,35]. Dietary deviations in maternal fatty acids intake throughout pregnancy may affect the nature of fatty acids crossing the placenta, having consequences to post-natal development.

In this study, ROC analysis and area under the curve showed that cord blood total cholesterol of < 80 mg/dL, HDL-C of < 45.5 mg/dL and LDL-C of < 23.2 mg/dL were chosen as cutoff point below which RDS can be predicted in preterm infants, with the best sensitivity and specificity in predicting RDS. Our study has its limitation that the dietary intakes of the mothers during pregnancy were not evaluated. So that, more studies are needed to evaluate its reflection on serum lipid profile of mothers and their infants.

Conclusion

RDS is accompanied by lipid alteration in the infants and their mothers. The results of this study point to the importance of measuring maternal serum and cord blood lipid profile as a predictor for the occurrence of RDS. Consent for publication: all authors have read and revised well for the manuscript and agree to publish.

References

1. Kim SY (2010) Neonatal respiratory distress: recent progress in understanding pathogenesis and treatment outcomes. *Korean Journal of Pediatrics* (53): 1-6.
2. Rodriguez RJ (2003) Management of respiratory distress syndrome: an update. *Respir Care* 48(3): 279-286.
3. Mantan S, Arulkumaran S (2006) Neonatal RDS. *Lancet* 367(9526): 1878-1879.
4. Keszler M, Abubakar KM (2012) The Respiratory System. In: *Textbook of Clinical Pediatrics*. 2nd ed. Elzouki AY, Harfi HA, Nazer HM, Stapleton FB, William OH and Whitley RJ (eds) 15: 195-215.
5. Mathai SS, Raju CU, Kanitkar CM (2007) Management of Respiratory Distress in the Newborn. *Medical Journal Armed Forces India* 63(3): 269-272.
6. Pamela CC, Richard AH, Denise RF (2008) Biochemistry 4th edition. Lippincott's Illustrated reviews. Wolter's Kluwer's, USA, pp. 178-179.

7. Marttila R, Haataja R, Guttentag S, Hallman M (2003) Surfactant Protein A and B Genetic Variants in Respiratory Distress Syndrome in Singletons and Twins. *Am J Respir Crit Care Med* 168(10): 1216-1222.
8. Hermansen CL, Lorah KN (2007) Respiratory distress in the newborn. *American Family Physician* 76(7): 987-994.
9. Herrera E (2002) Implications of dietary fatty acids during pregnancy on placental, fetal and postnatal development. *Placenta* 23(A): S9-S19.
10. Madsen EM, Lindegaard MLS, Andersen CB, Damm PL, Nielsen LB (2004) Human placenta secretes apolipoprotein B-100-containing lipoproteins. *J Biol Chem* 279: 55271-55276.
11. Chapman C, Morgan LM, Murphy MC (2000) Maternal and early dietary fatty acid intake: Changes Lipid Metabolism and enzymes in adult rats. *J Nutr* 130(2): 146-151.
12. Teslova OA, Hiroma T, I Baranovskaya EI, Nakamura T (2012) Evaluation of factors influencing stable microbubble test results in preterm neonates at 30-33 gestational weeks. *Research and Reports in Neonatology* 2: 5-10.
13. Butte NF, Ellis KJ, Wong WW, Hopkinson JM, Smith EO (2003) Composition of gestational weight gains impacts maternal fat retention and infant birth weight. *Am J Obstet Gynecol* 189(5): 1423-1432.
14. Sollid CP, Wisborg K, Hjort J, Secher NJ (2004) Eating disorder that was diagnosed before pregnancy and pregnancy outcome. *American Journal of Obstetrics and Gynecology* 190(1): 206-210.
15. Gunes T, Koklu E, Ozturk MA (2007) Maternal and cord serum lipid profiles of preterm infants with respiratory distress syndrome. *Journal of perinatology* 27(7): 415-421.
16. Hansen AK, Wisborg K, Uldbjerg N, Henriksen TB (2008) Risk of respiratory morbidity in term infants delivered by elective caesarean section: Cohort study. *BMJ* 336(7635): 85-87.
17. Kolås T, Saugstad OD, Daltveit AK, Nilsen ST, Øian P (2006): Planned cesarean versus planned vaginal delivery at term: comparison of newborn infant outcomes. *Am J Obstet Gynecol* 195(6): 1538-1543.
18. Descamps OS, Bruniaux M, Guilmot PF, Tonglet R, Heller FR (2004) Lipoprotein concentrations in newborns are associated with allelic variations in their mothers. *Atherosclerosis* 172(2): 287-298.
19. Rodie VA, Caslake MJ, Stewart F, Sattar N, Ramsay JE, et al. (2004) Fetal cord plasma lipoprotein status in uncomplicated human pregnancies and in pregnancies complicated by pre-eclampsia and intrauterine growth restriction. *Atherosclerosis* 176: 181-187.
20. Saleh J, Al-Riyami HD, Chaudhary TA, Cianflone K (2008) Cord Blood ASP Is Predicted by Maternal Lipids and Correlates with Fetal Birth Weight. *Obesity* 16(6): 1193-1198.
21. Kelishadi R, Badiee Z, Adeli K (2007) Cord blood lipid profile and associated factors: baseline data of a birth cohort study. *Paediatric and Perinatal Epidemiology* 21(6): 518-524.
22. Kharb S, Kaur A, Nanda S (2010) Comparison of cord blood atherogenic index in males and females. *Iranian Cardiovascular Research Journal* 4(1): 35-38.
23. Aasvee K, Kurvinen E, Jordania R, Jauhainen M, Sundvall J (2004) Lipoprotein parameters in relation to other risk factors of atherosclerosis in adults and newborns: Tallinn Young Family Study. *Scand J Clin Lab Invest* 64(3): 245-253.
24. Bansal N, Cruickshank JK, McElduff P, Durrington PN (2005) Cord blood lipoproteins and prenatal influences. *Curr Opin Lipidol* 16(4): 400-408.
25. Parker CR, Atkinson MW, Owen J, Andrews WW (2006) Dynamics of the fetal adrenal, cholesterol, and apolipoprotein B responses to antenatal betamethasone therapy. *Am J Obstet Gynecol* 174: 562-565.
26. Lane DM, McConathy WJ, McCaffree MA, Hall M (2002) Cord serum lipid and apolipoprotein levels in preterm infants with the neonatal respiratory distress syndrome. *J Matern Fetal neonatal Med* 11: 118-125.
27. Huxley R, Owen CG, Whincup PH, Cook DG, Colman S, et al. (2004) Birth weight and subsequent cholesterol levels: exploration of the 'fetal origins' hypothesis. *JAMA* 292: 2755-2764.
28. Jain R, Tripathi VN, Singh RD, Pandey K (2011) Lipid Profile and Apolipoproteins in Neonates in relation to Birth Weight and Gestational Maturity. *Journal of Pediatric Sciences* 3(2): e80.
29. Badiee Z, Kelishadi R (2008) Lipoprotein and apolipoprotein levels in Iranian newborns. *Paediatr Cardiol* 29(3): 574-579.
30. Kaser S, Ebenbichler CF, Wolf HJ, Sandhofer A, Stanzl U, Ritsch A and Patsch JR (2001): Lipoprotein profile and cholesterol ester transfer protein in neonates. *Metabolism*; 50:723-728.
31. Burke KT, Colvin PL, Myatt L, Graf GA, Schroeder F, et al; (2009) Transport of maternal cholesterol to the fetus is affected by maternal plasma cholesterol concentrations in the Golden Syrian hamster. *J Lipid Res* 50: 1146-1155.
32. Yoshida S, Wada Y (2005) Transfer of maternal cholesterol to embryo and fetus in pregnant mice. *J Lipid Res* 46(10): 2168-2174.
33. Cetin I, Alvino G, Cardelluccio M (2009) Long chain fatty acids and dietary fats in fetal nutrition. *J Physiol* 587(14): 3441-3451.
34. Herrera E, Ortega-senovilla H (2010) Maternal lipid metabolism during normal pregnancy and its implications to fetal development. *Clin Lipidol* 5(6): 899-911.
35. Herrera E, Amusquivar E, Lopez-Soldado I, Ortega-senovilla H (2006) Maternal lipid metabolism and placental lipid transfer. *Horm Res* 65(3): 59-64.



This work is licensed under Creative Commons Attribution 4.0 License
DOI: [10.19080/AJPN.2024.14.555945](https://doi.org/10.19080/AJPN.2024.14.555945)

**Your next submission with Juniper Publishers
will reach you the below assets**

- Quality Editorial service
- Swift Peer Review
- Reprints availability
- E-prints Service
- Manuscript Podcast for convenient understanding
- Global attainment for your research
- Manuscript accessibility in different formats
(**Pdf, E-pub, Full Text, Audio**)
- Unceasing customer service

Track the below URL for one-step submission

<https://juniperpublishers.com/online-submission.php>