Maternal and Cord Serum Lipid Profiles of Preterm Neonates with Respiratory Distress Syndrome

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Abstract

Introduction: Respiratory Distress Syndrome (RDS) occurs almost exclusively in preterm infants. Preterm with RDS has low amount of surfactant which contains low amount of desaturated phosphatidylcholine, phosphatidylglycerol, and less of surfactant proteins than surfactant from mature lung.

The Aim of this Study: Was to evaluate the level of lipid profiles in maternal and cord blood of preterm infants with respiratory distress syndrome and to assess the effect of maternal nutritional status and lipid profile on cord blood lipid profile of preterm infants.

Methods and Results: The study was conducted on 30 neonates with RDS with gestational age 27-36 week, birth weight 750-2600gm, and 20 neonates without RDS, their gestational age range from 31-36 weeks and birth weights range 1200-2700gm were enrolled as a control group. All newborn and their mothers were subjected to detailed medical history, thorough clinical examination and laboratory, radiological, and imaging investigations. Pre-gravid weight, pre-gravid BMI, weight gain during pregnancy, were significantly lower in mothers of RDS patients. Birth weight, length, head circumference, Apgar score at 5 minutes were significantly lower in RDS group compared to control group. Total serum cholesterol, HDL-C, LDL-C were significantly lower in RDS neonates, while serum triglycerides, VLDL-C, showed no significant difference when compared to control group. For the mothers of affected neonates serum total cholesterol, HDL-C, LDL-C, triglycerides, VLDL-C, showed no significant difference when compared to control group. Total serum cholesterol, HDL-C, LDL-C were significantly lower in RDS neonates, while serum triglycerides, VLDL-C, showed no significant difference when compared to control group. For the mothers of affected neonates serum total cholesterol, HDL-C, LDL-C, triglycerides, VLDL-C, were significantly lower when compared with mothers of non affected neonates. There was a positive correlation between maternal lipid profile and their infant’s lipid profile in infants with RDS. Bi parietal diameter, abdominal circumference, femoral length, and ultrasono graphic weight one week before delivery were significantly lower in RDS neonates compared to control group.

Conclusion: Low plasma lipid during gestation appears to have negative effects on the fetal lung development. Mean total HDL-C, LDL-C were significantly lower in infants with RDS and their mothers than in control group.

Key Words: Lipid profile – RDS – Preterm infant – HDL-C, LDL-C – VLDL-C.

Introduction

RESPIRATORY distress syndrome (RDS) one of the most common causes of neonatal morbidity, occurs almost exclusively in premature infants, incidence and severity are related inversely to gestational age [1]. Patient presents shortly after birth with respiratory symptoms. Radiological finding includes diffuse reticulogranular appearance with superimposed air bronchogram. Preterm with RDS has low amount of surfactant which contains lower percent of desaturated phosphatidylcholine, phosphatidylglycerol, and less of surfactant proteins than surfactant from mature lung. Surfactant is synthesized and secreted from pneumocytes at 24 -34 week of gestation, formed of 80% phospholipids, 10% neutral lipid mainly cholesterol, 10% proteins [2]

Lung cholesterol is regulated by both LDL-C, and HDL-C [3], which stimulates alveolar cell type II to secrete surfactant [4]. So lipid metabolism is important in lung development with indication that triglyceride for VLDL is essential for surfactant synthesis [5]. Maternal lipoproteins provide the free fatty acid substrate required for fetal surfactant synthesis in vivo [6]. Inadequate total fatty acid supply in utero could interfere with normal fetal growth and maturation leading to development of neonatal RDS [7]. Maternal total serum cholesterol

Abbreviations:  
RDS  = Respiratory distress syndrome.  
HDL-C = High density lipoprotein cholesterol.  
LDL-C = Low density lipoprotein cholesterol.  
VLDL-C = Very low density lipoprotein cholesterol.  
TG = Triglycerides.

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less than 10th population percentile was strongly associated with premature delivery [8]. Maternal weight gain during pregnancy correlates significantly with newborn weight [9].

**The aim of this study was:**

To evaluate the level of lipid profiles in maternal and cord blood of preterm infants with respiratory distress syndrome and to assess the effect of maternal nutritional status and lipid profile on cord blood lipid profile of preterm infants.

**Patients and Methods**

30 patients admitted to NICU with diagnosis of RDS (diagnosed by cyanosis, retraction, diffuse alveolar atelectasis on X-ray, acidosis, hypoxemia, hypercarbia on blood gases and their mothers received antenatal care in Gynecological Obstetric Department (Banha University hospital in the period from October 2010 to January 2011).

**Inclusion criteria:** Gestational age less than or equal to 36 week.

**Exclusion criteria:** SGA, HIE, major congenital anomalies, mothers with complicated pregnancy with hypertension, pre-eclampsia, history of endocrinal diseases, drug intake that affect lipid metabolism.

**Control group:**

20 preterm newborn not suffering from any respiratory disease, age, and sex matched to the patient group and their mothers received ante-natal care at Gynecological Obstetric Department (Banha University Hospital).

**Methods:**

- Informed consent from parents before testing was obtained.
- Patients enrolled in the study were subjected to the following:
  - Full maternal history taking (including personal, menstrual, obstetric, past history).
  - Examination:
    - Newborn examination included (gestational age by Ballard score, and maternal data, Apgar score 1 and 5 minute, complete clinical examination).

**Anthropometric measurement:**

- For the mother: (Pre-pregnant weight, height, BMI, weight gain during pregnancy)
- For the newborn: (Body weight, crown heel length, ponderal index).

**c- Radiological & imaging investigations:**

  Obstetric ultrasound: (At beginning of pregnancy for dating, second trimester to exclude congenital anomalies, and last one within one week before delivery).

  Chest X-ray antero-posterior view for the neonates.

**d- Laboratory investigations:**

  Maternal blood sample (after period of fasting 12 hours, collected in plain tube), cord blood sample 4ml blood collected in plain tube.

**Samples were analyzed for:**

- Serum total cholesterol.
- Serum HDL-C.
- Serum LDL-C.
- Serum VLDL-C.
- Serum triglycerides.

**e- Statistical analysis:**

For parametric data: Student t-test was to compare between mean of two groups.

For continuous non parametric data: Pearson correlation coefficient test.

Inter group comparison of categorical data was performed by using chi square test with p-value <0.05 considered significant <0.0001 highly significant >0.05 non significant.

**Results**

The pre-gravid BMI, pre-gravid weight and weight gain during pregnancy were significantly lower in the mothers of RDS compared to non RDS groups. However, no significant difference of the ruptured membrane <24h, pre-gravid height, age, parity, and receiving dexamethasone between the mothers of RDS and mothers of the control.

Gestational age, birth weight, length, head circumference, Apgar scores at 5min were significantly lower in RDS compared to the control group and no significant difference of the ponderal index, sex and Apgar score at 1-min between RDS and control groups.

Total serum cholesterol, HDL-C, LDL-C levels, were significantly lower in neonates with RDS when compared to control group while their serum triglycerides and VLDL showed no significant difference.
Table (1): Comparison between demographic characteristics and anthropometric measurements of mothers of patient and control groups.

<table>
<thead>
<tr>
<th></th>
<th>Control (n=20)</th>
<th>Cases of RDS (n=30)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean S.D</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>25.825 3.0100</td>
<td>24.600 3.3718</td>
<td>1.3</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Gestational Weight</td>
<td>68.38 8.180</td>
<td>62.73 6.707</td>
<td>2.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Gestational Height</td>
<td>161.85 5.752</td>
<td>161.70 4.928</td>
<td>0.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>VLDL-C</td>
<td>14.726 6.493</td>
<td>30.28 5.758</td>
<td>3.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LDL-C</td>
<td>30.220 9.45</td>
<td>6.9291 1.6263</td>
<td>4.4601</td>
<td>0.3</td>
</tr>
<tr>
<td>HDL-C</td>
<td>26.04 9.45</td>
<td>30.73 18.437</td>
<td>1.5</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Sex</td>
<td>Male 80.0 4.00</td>
<td>23 16.7</td>
<td>2.9</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>Female 60.0 4.00</td>
<td>25 16.7</td>
<td>2.9</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

RDS: Respiratory distress syndrome. 
PROM: Premature rupture of membranes. 
BMI: Body mass index. 
VLDL: Very Low Density Lipoprotein. 
LDL: Low density lipoprotein. 
HDL: High Density Lipoprotein. 
Gestational: Gestational age. 

Maternal total cholesterol, HDL-C, LDL-C, triglycerides and VLDL-Cholesterol levels were significantly lower in mothers of patient when compared to mothers of control group.

No correlation between neonatal lipid profile and sex.

No correlation between neonatal lipid profile and mode of delivery.

The mean level of lipid profile between infants whose mothers had received dexamethasone and those whose mothers had not. There was no significant difference.

There is significant positive correlation between gestational age, length, ponderal index, head circumference and the total neonatal cholesterol.

Table (2): Comparison between demographic characteristics and anthropometric measurements of mothers of patient and control groups.

<table>
<thead>
<tr>
<th></th>
<th>Control (n=20)</th>
<th>Cases of RDS (n=30)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean S.D</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gestational Age</td>
<td>33.06 1.698</td>
<td>31.67 2.670</td>
<td>3.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Weight</td>
<td>2.016 0.47</td>
<td>1.54 0.56</td>
<td>3.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Length</td>
<td>45.35 2.739</td>
<td>42.03 2.463</td>
<td>3.3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Ponderal index</td>
<td>21.268 2.5616</td>
<td>20.428 2.9010</td>
<td>1.1</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>H.C</td>
<td>32.250 1.6263</td>
<td>30.250 2.1162</td>
<td>3.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>A PGar at 1min.</td>
<td>6.70 1.081 6.23</td>
<td>1.431 1.3 &gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A PGar at 3min.</td>
<td>9.05 0.686 8.33</td>
<td>1.028 2.9 &lt;0.05</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

No. % S.D % X^2 p

Table (3): Comparison between laboratory data of RDS and non RDS group.

<table>
<thead>
<tr>
<th></th>
<th>Control (n=20)</th>
<th>Cases of RDS (n=30)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean S.D</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>201.50 37.893</td>
<td>315.05 37.743</td>
<td>4.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TG</td>
<td>182.15 32.497</td>
<td>215.73 29.210</td>
<td>3.4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>HDL</td>
<td>48.98 6.940</td>
<td>38.61 6.658</td>
<td>3.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LDL</td>
<td>115.66 36.905</td>
<td>81.74 36.805</td>
<td>3.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>VLDL</td>
<td>36.41 6.493</td>
<td>30.28 5.758</td>
<td>3.5</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

Cholesterol = High Density Lipoprotein-Cholesterol. 
TG = Triglyceride. 
HDL-C = High Density Lipoprotein-Cholesterol. 
LDL-C = Low density lipoprotein cholesterol. 
RDS = Respiratory distress syndrome. 

Table (5): Effect of sex on the neonatal lipid profile.

<table>
<thead>
<tr>
<th></th>
<th>Male (n=27)</th>
<th>Female (n=23)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean S.D</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>69.70 15.532</td>
<td>77.05 18.437</td>
<td>1.5</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>TG</td>
<td>74.02 19.890</td>
<td>75.57 22.518</td>
<td>0.3</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>HDL</td>
<td>26.06 10.551</td>
<td>30.73 9.449</td>
<td>1.6</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>LDL</td>
<td>29.291 6.929</td>
<td>30.220 9.3727</td>
<td>0.4</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>VLDL</td>
<td>14.726 3.9716</td>
<td>15.093 4.4601</td>
<td>0.3</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Cholesterol = High Density Lipoprotein-Cholesterol. 
TG = Triglyceride. 
HDL-C = High Density Lipoprotein-Cholesterol. 
LDL-C = Low density lipoprotein cholesterol. 
RDS = Respiratory distress syndrome. 

S.D = Standard deviation.
Table (6): Effect of mode of delivery on the neonatal lipid profile.

<table>
<thead>
<tr>
<th>Lipid</th>
<th>C.S (n=25)</th>
<th>NVD (n=25)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>S.D</td>
<td>Mean</td>
<td>S.D</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>76.11</td>
<td>16.096</td>
<td>70.05</td>
<td>17.964</td>
</tr>
<tr>
<td>TG</td>
<td>78.67</td>
<td>18.864</td>
<td>70.80</td>
<td>22.507</td>
</tr>
<tr>
<td>HDL</td>
<td>29.04</td>
<td>10.810</td>
<td>27.38</td>
<td>9.769</td>
</tr>
<tr>
<td>LDL</td>
<td>30.162</td>
<td>7.9523</td>
<td>29.274</td>
<td>8.3269</td>
</tr>
<tr>
<td>VLDL</td>
<td>15.714</td>
<td>3.7286</td>
<td>14.076</td>
<td>4.4826</td>
</tr>
</tbody>
</table>

HDL-C : High Density Lipoprotein-Cholesterol.
LDL-C : Low density lipoprotein-cholesterol.
TG : Triglyceride.
VLDL-C : Very Low Density Lipoprotein-Cholesterol.
NVD : Normal vaginal delivery.
RDS : Respiratory distress syndrome.
SD : Standard deviation.
CS : Cesarean section.

Table (7): Effect of using dexamethasone as a drug of lung maturity on neonatal lipid profile.

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Negative (n=12)</th>
<th>Received dexamethasone (n=38)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>S.D</td>
<td>Mean</td>
<td>S.D</td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>72.20</td>
<td>18.430</td>
<td>75.71</td>
<td>13.750</td>
</tr>
<tr>
<td>TG</td>
<td>74.35</td>
<td>19.234</td>
<td>77.56</td>
<td>26.395</td>
</tr>
<tr>
<td>HDL</td>
<td>28.12</td>
<td>11.105</td>
<td>28.32</td>
<td>7.900</td>
</tr>
<tr>
<td>LDL</td>
<td>28.818</td>
<td>8.4480</td>
<td>23.222</td>
<td>6.7849</td>
</tr>
<tr>
<td>VLDL</td>
<td>14.814</td>
<td>3.8431</td>
<td>15.470</td>
<td>5.2079</td>
</tr>
</tbody>
</table>

HDL-C : High Density Lipoprotein-Cholesterol.
LDL-C : Low density lipoprotein-cholesterol.
TG : Triglyceride.
VLDL-C : Very Low Density Lipoprotein-Cholesterol.
SD : Standard deviation.

Table (8): Correlations between neonatal anthropometric measurements, Apgar score and their lipid profile in infants with RDS.

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Gestational Age</th>
<th>Weight</th>
<th>Length</th>
<th>Ponderal index</th>
<th>HC</th>
<th>Apgar 1 min</th>
<th>Apgar 5min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>p</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>0.720</td>
<td>&lt;0.001</td>
<td>-0.155</td>
<td>&gt;0.05</td>
<td>0.531</td>
<td>&lt;0.05</td>
<td>0.681</td>
</tr>
<tr>
<td>TG</td>
<td>0.255</td>
<td>&gt;0.05</td>
<td>-0.165</td>
<td>&gt;0.05</td>
<td>0.344</td>
<td>&gt;0.05</td>
<td>0.044</td>
</tr>
<tr>
<td>HDL</td>
<td>0.289</td>
<td>&gt;0.05</td>
<td>0.145</td>
<td>&gt;0.05</td>
<td>0.060</td>
<td>&gt;0.05</td>
<td>0.474</td>
</tr>
<tr>
<td>LDL</td>
<td>0.409</td>
<td>&lt;0.05</td>
<td>-0.363</td>
<td>&lt;0.05</td>
<td>0.257</td>
<td>&gt;0.05</td>
<td>0.428</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.256</td>
<td>&gt;0.05</td>
<td>-0.165</td>
<td>&gt;0.05</td>
<td>0.345</td>
<td>&gt;0.05</td>
<td>-0.044</td>
</tr>
</tbody>
</table>

p : Probability.
HDL-C : High Density Lipoprotein-Cholesterol.
TG : Triglyceride.
VLDL-C : Very Low Density Lipoprotein-Cholesterol.
LDL-C : Low density lipoprotein-cholesterol.

Table (9): Correlation between maternal characteristics and their neonates lipid profile in infants with RDS.

<table>
<thead>
<tr>
<th>Lipid</th>
<th>Pregravid WT</th>
<th>Pregnant Ht</th>
<th>BMI</th>
<th>Wt. gain during pregnancy</th>
<th>Cholesterol</th>
<th>TG</th>
<th>HDL</th>
<th>LDL</th>
<th>VLDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>p</td>
</tr>
<tr>
<td>Pregravid WT</td>
<td>0.721</td>
<td>&lt;0.001</td>
<td>0.197</td>
<td>&gt;0.05</td>
<td>0.405</td>
<td>&lt;0.05</td>
<td>0.42</td>
<td>&lt;0.05</td>
<td>0.196</td>
</tr>
<tr>
<td>Pregnant Ht</td>
<td>0.522</td>
<td>&lt;0.01</td>
<td>0.22</td>
<td>&gt;0.05</td>
<td>0.38</td>
<td>&lt;0.05</td>
<td>0.194</td>
<td>&gt;0.05</td>
<td>0.22</td>
</tr>
<tr>
<td>BMI</td>
<td>0.541</td>
<td>&lt;0.01</td>
<td>0.12</td>
<td>&gt;0.05</td>
<td>0.18</td>
<td>&gt;0.05</td>
<td>0.439</td>
<td>&lt;0.05</td>
<td>0.121</td>
</tr>
<tr>
<td>Wt. gain during pregnancy</td>
<td>-0.09</td>
<td>&gt;0.05</td>
<td>0.213</td>
<td>&gt;0.05</td>
<td>0.166</td>
<td>&gt;0.05</td>
<td>0.02</td>
<td>&gt;0.05</td>
<td>0.214</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>0.44</td>
<td>&lt;0.05</td>
<td>-0.339</td>
<td>&lt;0.05</td>
<td>0.568</td>
<td>&lt;0.01</td>
<td>0.138</td>
<td>&gt;0.05</td>
<td>-0.338</td>
</tr>
<tr>
<td>TG</td>
<td>0.51</td>
<td>&lt;0.01</td>
<td>0.452</td>
<td>&lt;0.05</td>
<td>0.136</td>
<td>&gt;0.05</td>
<td>0.467</td>
<td>&lt;0.01</td>
<td>0.452</td>
</tr>
<tr>
<td>HDL</td>
<td>-0.18</td>
<td>&gt;0.05</td>
<td>0.181</td>
<td>&gt;0.05</td>
<td>0.178</td>
<td>&gt;0.05</td>
<td>-0.071</td>
<td>&gt;0.05</td>
<td>0.181</td>
</tr>
<tr>
<td>LDL</td>
<td>0.439</td>
<td>&lt;0.01</td>
<td>-0.419</td>
<td>&lt;0.05</td>
<td>0.604</td>
<td>&lt;0.001</td>
<td>0.089</td>
<td>&gt;0.05</td>
<td>0.418</td>
</tr>
<tr>
<td>VLDL</td>
<td>0.489</td>
<td>&lt;0.01</td>
<td>0.435</td>
<td>&lt;0.05</td>
<td>0.135</td>
<td>&gt;0.05</td>
<td>0.448</td>
<td>&lt;0.05</td>
<td>0.35</td>
</tr>
</tbody>
</table>

TG : Triglyceride.
VLDL-C : Very Low Density Lipoprotein-Cholesterol.
SD : Standard deviation.
BMI : Body mass index.

p : Probability.
• There is significant positive correlation between gestational age, birth weight, ponderal index, and the low density lipoprotein cholesterol (LDL-C).

• There is a positive correlation between maternal cholesterol and neonatal cholesterol.

• There is a positive correlation between maternal total cholesterol and neonatal HDL-C.

• There is a positive correlation between maternal and neonatal triglycerides.

• There is a positive correlation between maternal triglyceride level and neonatal total cholesterol, neonatal LDL-C, and neonatal VLDL-C.

• There is a positive correlation between maternal low density lipoprotein cholesterol, LDL-C and neonatal Total cholesterol, neonatal TG, neonatal HDL-C and neonatal VLDL-C.

Discussion

There is a positive correlation between maternal very low density lipoprotein cholesterol, VLDL-C and neonatal TG, neonatal cholesterol, neonatal HDL-C, neonatal VLDL-C.

There is a positive correlation between maternal pre-gravid weight and (neonatal Total cholesterol, neonatal HDL-C and neonatal LDL-C).

There is a positive correlation between maternal height and (neonatal Total cholesterol and neonatal LDL-C).

There is a positive correlation between maternal BMI and (neonatal Total cholesterol and neonatal LDL-C).

The maternal High Density Lipoprotein-Cholesterol HDL-C is the most significant in predicting RDS.

Bi-parietal diameter, abdominal circumference, femoral length, ultrasonographic weight and birth weight were significantly lower in RDS neonates compared to non RDS group.

Table (11): Comparison between control preterm & preterm cases according to (US) data which was done within 1 week before delivery.

<table>
<thead>
<tr>
<th></th>
<th>Control (n=20)</th>
<th>Cases of RD (N=30)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>Mean</td>
<td>Std. Deviation</td>
<td>Std. Deviation</td>
<td></td>
</tr>
<tr>
<td>WT</td>
<td>2.0160</td>
<td>.46590</td>
<td>5.76651</td>
<td>6.65385</td>
</tr>
<tr>
<td>FL</td>
<td>61.9000</td>
<td>5.76651</td>
<td>54.8333</td>
<td>21.24395</td>
</tr>
<tr>
<td>BPD</td>
<td>80.8000</td>
<td>6.65385</td>
<td>73.0333</td>
<td>8.30240</td>
</tr>
<tr>
<td>AC</td>
<td>286.4000</td>
<td>21.24395</td>
<td>265.8333</td>
<td>24.00443</td>
</tr>
<tr>
<td>USWT</td>
<td>2.0407</td>
<td>.48185</td>
<td>1.5643</td>
<td>2.9</td>
</tr>
</tbody>
</table>

\( t \)-test : Student’s \( t \)-test.

BPD : Bi-parietal diameter.

US WT : Ultrasongraphic weight.

FL : Femoral length.

AC : Abdominal circumference.

WT : Birth Weight.

p-value : Probability.

SD : Standard deviation.

In the present study the mothers of RDS were characterized by having a significant less pre-gravid weight, BMI, weight and weight gain during pregnancy when compared to mothers of preterm infants without RDS. However, no statistical significant difference of the ruptured membrane >24 hours, pre-gravid height, age, parity, and receiving dexamethasone between the mothers of RDS and mothers of the control group.

The previous observations are consistent with the study of Gunes et al. [10], Bansal et al. [11], who reported a significant decrease in pre gravid weight, BMI, and weight gain during pregnancy among mothers of infants with RDS. Boskbaday et al. [12] observed that ruptured membrane >24 hours was significantly higher in mothers of infants with RDS than control. Sollid et al. [13] observed that low weight, BMI, at conception or delivery, as well as poor weight gain during pregnancy are associated with LBW, prematurity and maternal delivery complications. Nematollahzadeh et al. [14] found that overweight and obese mothers with high BMI have a great risk of preterm birth and low birth weight infants, however Khashan and Kenny [15] observed that the risk of preterm birth was reduced in overweight and obese women and was increased in underweight women.

As regard the clinical characteristics of the studied infants, the gestational age, birth weight, length, head circumference, Apgar score at 5 minutes were significantly lower in RDS compared to the control group and no significant difference of

Table (10): Maternal lipid profile in predicting RDS.

<table>
<thead>
<tr>
<th></th>
<th>Beta</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>0.803</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>TG</td>
<td>0.116</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>HDL</td>
<td>−0.676</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LDL</td>
<td>−1.008</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>VLDL</td>
<td>−0.450</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

HDL-C : High Density Lipoprotein-Cholesterol.

LDL-C : Low density lipoprotein-cholesterol.

TG : Triglyceride.

VLDL-C : Very Low Density Lipoprotein-Cholesterol.

p : Probability.
the ponderal index, sex, and Apgar score at 1 minute between RDS patients and control group.

The previous observations are consistent with the study of Gunes et al. [10] except that ponderal index was also significantly lower in RDS compared to the control group.

Similar trend was observed by Fiona et al. [16] who reported that there was a highly significant association between birth weight, RDS incidence and severity, and there was highly significant fall in incidence of RDS with longer gestational age. Miller and Futrekull. [17] found no significant difference in incidence of RDS between the two sex. Loughrey et al. [18] found no significant difference in infants with RDS when compared to infant without RDS regarding gestational age group from 30–42 week as regard sex and mode of delivery.

We found that total serum cholesterol, HDL-C, LDL-C levels, were significantly lower in neonates with RDS when compared to control group while there serum triglycerides and VLDL-C showed no significant difference. As regard mothers there were significantly lower total cholesterol, HDL-C, LDL-C, triglycerides and VLDL-C, levels in mothers of patients when compared to mothers of control group.

On the attempt to compare lipid profile of infants with RDS and there mothers with those of control group it was found that the mean total cholesterol, HDL-C, and LDL-C were significantly lower in infants RDS and their mother than in the controls. However, the differences in serum triglycerides and VLDL-C, and triglycerides levels were found to be significant in the mothers group only. These results suggest that lower lipid parameters in RDS infants are evidence of reduced maternal supply, which could delay lung maturation.

The same observations had been found by Gunes et al. [10] and are consistent with the earlier reports of McConathy and Lane [19] who found that preterm infants with RDS had significantly lower levels of cord serum lipid and apolipoproteins compared to control infants of equivalent gestational age and birth weight. Later on, Lane et al. [7] stated that preterm infant developing RDS had significantly lower cord serum lipid levels than those of normal term infants or preterm infants free of respiratory distress. Moreover, Saleh et al. [20] documented that lipid levels in cord blood, particularly triglycerides were markedly decreased in infant with RDS when compared to control group.

However Donega et al. [21] observed that cord lipid profile of appropriate for gestational age preterm neonates were higher than those of term group, and Pardo et al. [22] demonstrated that the cholesterol level of premature group were substantially higher than those of term group, in agreement with the report of Diaz et al. [23].

Yonezawa et al. [5] found no difference in lipid profile between preterm neonates with RDS and preterm neonates without RDS however none of the neonates born after 34 week of gestation in his study developed RDS.

Upon study the effect of sex on the measured neonatal lipid profile no significant difference was found between male and females which is consistent with the result of Singh et al. [24] and Descamps et al. [25]. While Kelishadi et al. [26] Bansal et al. [11] and Kharbt et al. [27] reported that cord blood of female newborn had higher total cholesterol, HLD-C, LDL-C, Apo A-1, Apo B and A-1 as compared to male newborn, whereas triglycerides and VLDL-C were higher in male newborn.

We found no significant difference in neonatal lipid profile regarding to mode of delivery. This is consistent with observation of Gunes et al. [10] However Bansal et al. [11] and Rodie et al. [28] stated that methods of delivery associated with higher level of fetal distress like vaginal delivery and emergency caesarean section are associated with higher cord lipid level when compared to elective caesarean section.

Kelishadi et al. [26] reported that there is a significant difference in cord lipid profile in neonates regarding difference in gestational age as well as gestational age.

Upon studying the association between the neonatal anthropometric characteristics and the cord blood lipid profile, significant positive correlations were found between birth weight, length, ponderal index and head circumference and total neonatal cholesterol level, significant positive correlation were found between gestational age, birth weight, ponderal index, and the LDL-C, and significant positive correlation were found between ponderal index, and HDL-C.

In agreement with our finding Lane et al. [7] reported that cord blood lipid profile in infants with RDS depends on their birth weight. Huxley et al. [29] documented a retrospective association between birth weight and lipid level, and suggested that birth weight and body size have a great effect on blood cholesterol level which may be explained.
by report of Dutta-Roy [30] who demonstrated that fetal supply of free fatty acid is directly related to infant body weight and length at birth.

In our study there is a significant positive correlation between gestational age, and the total cholesterol of the infant. These results supported the presented data of Johnson et al. [31] and Bastida et al. [32] who documented that cholesterol (total and free) and apo E, and triglycerides increase progressively with gestational age, reflecting increases in total body fat during the later stages of gestation although Pardo et al. [22] demonstrated that the cholesterol level of the premature group were substantially higher than those of the term group, in agreements with the report of Diaz M et al. [23].

In our study there was a positive correlation between maternal cholesterol and neonatal cholesterol this in agreement with reports by Ophir et al. [33] that these influenced by environmental factor mainly maternal diets. These observation explained by the fact that maternal cholesterol can cross the placenta Bansal et al. [11], and essential fatty acids must be ultimately derived from the mother along maternal fetal concentration gradient [34].

In our study there is positive correlation between maternal and fetal triglycerides which agrees with the result of Berghaus et al. [35].

In our study there is reported positive correlation between maternal total cholesterol and newborn HDL-C. This agrees with the result of Rodie et al. [28].

In our study there is reported positive correlation between maternal LDL-C and newborn HDL-C this in agreement with Rodie et al. [28], and neonatal total cholesterol this in agreement with Ortega et al. [36]. In contrast to all previous observations Lane et al. [7], found no association between maternal and neonatal lipid profile and suggest that abnormalities in placental transport of lipid component in preterm with RDS may result in change in cord serum lipid and apo lipoprotein levels.

In our study we found that there is positive correlation between maternal pre-gravid weight, neonatal total cholesterol, HDL-C, LDL-C. A positive correlation between maternal height and neonatal total cholesterol, HDL-C, Merzouk et al. [37] reported that maternal obesity may affect neonatal lipid profile only when associated with macrosomia. Kelishadi et a. [26] observed that maternal weight may affect fetal growth and maturation. While their impact on serum cords lipid profile remain controversial. Catalon et al. [38] concluded that various genetic, and environmental factor may modify fetal growth by differentially affecting the growth of the fetal fat and fat free mass.

We found also a positive correlation between maternal BMI and neonatal total cholesterol and LDL-C. This is in agreement with Gunes et al. [10].

In our study we found that maternal HDL-C is the most significant in prediction of RDS. This is in agreement with Pac-Kazuchowska E. [39] who found that RDS infants and their mothers have reduced serum level of LDL-C suggesting that it may have an important role on regulation of surfactant synthesis. Also Voyno Yasenetskaya et al. [40] stated that LDL and HDL stimulate alveolar cell type II to secrete phosphatidylcholine, thus reduced fetal supply through placenta thought to predispose to RDS. Also Davidson et al. [41] reported that inhibition of cholesterol synthesis leads to impaired surfactant synthesis Ryan et al. [6] demonstrated that maternal VLDL loading regulates surfactant synthesis in fetal lung. Also Sosenko et al. [42] observed that surfactant deficiency in RDS can be ameliorated by lipid loading.

Importantly Wojcicka-Jagodzinska et al. [43] speculated that a reduction in cholesterol concentration of amniotic fluid of hypertensive mothers, in whose children developed RDS, might have a prognostic significance in predicting respiratory distress in early neonatal period.

In preliminary analysis Legras et al. [44] found no significant difference in lipid profiles among mothers of infants with RDS compared with those without RDS, while Hardell Li., [45] and Okazoki et al. [46] reported no difference in lipoprotein profile in preterm neonates (28 to 34 gestation weeks) with RDS when compared with preterm without RDS.

Surprisingly Karagiorga et al. [47] reported an increase of cholesterol in mothers of RDS infants over control, also Hummel et al. [48] stated that total cholesterol and triglycerides were higher in infants with RDS than the control.

Yonezawa et al. [5] found that preterm neonates had lower VLDL-TG concentration than term neonates and 34 week of gestation is a critical period of triglycerides metabolism in neonates because cord blood VLDL-TG increase dramatically from 32 to 34 week and the development of RDS seemed to be inhibited after this period.
These contradictory results might be due to the fact that there are many factors that determine the concentration and composition of maternal and cord lipid profile.

We use measurements established by ACOG Committee on Practice Bulletins [49] as a reference for ultrasonographic measurement but we could not find a possible explanation of the reason why ultrasonographic fetal measurement such as Biparietal diameter, abdominal circumference, femoral length, and ultrasonographic weight are significantly lower in RDS neonates when compared with non RDS neonates.

Conclusion:
- Low plasma lipid during gestation appears to have negative effects on the fetal lung development, mean total HDL-C, LDL-C were significantly lower in infants with RDS and their mothers than in control.
- There is a strong relation between lipid profile in maternal blood and cord blood, and lipid levels in cord blood depend on gestational age.
- Measurement of lipid profile in maternal blood has an important role in predicting the occurrence of RDS in preterm infants, and subsequently use of precautions to prevent the occurrence of RDS.
- Weight of the mother during pregnancy, and body mass index may have a role in predicting the occurrence of RDS.

References