Correlation of maternal lipid profile with newborn’s lipid profile

Anamika Gora, Deveshwar Dev*, Palak Gupta, M. L. Gupta

Background: Atherosclerotic cardiovascular disease (CVD) is a major cause for morbidity and mortality in the adult population. Altered lipid levels are the recognized factors. This process is considered to begin in early life and progress silently over decades. Maternal lipid concentrations may exert an influence on fetal lipid profile. The objective of the study was to find out the correlation of maternal lipid profile with newborn’s lipid profile.

Methods: This was a hospital based, cross sectional study. After applying inclusion and exclusion criteria, a total of 220 parturients and their respective newborns were enrolled. Out of 220 newborns, 110 were <2.5 kg (group A) and 110 were ≥2.5 kg (group B) babies. 2.5 ml of Cord blood sample from each of enrolled newborns was collected from the placental end of the cord just after the delivery. Blood samples from the parturients were collected right after delivery. The concentrations of total cholesterol, triglycerides and HDL-C were determined by an enzymatic colorimetric method and LDL-c was calculated by the Friedewald formula.

Results: In present study there was a statistically significant but poor negative correlation between maternal TG and babies’ TG level among low birth weight (<2.5 kg) babies and no other significant correlation was observed between maternal lipid profile and newborn’s lipid profile.

Conclusions: Change in maternal lipid profile is not significantly associated with the mean concentrations of total cholesterol, LDL-C, HDL-C and triglycerides in newborns. Hence it shows that neonatal lipogenesis may be independent of maternal lipogenesis.

Keywords: HDL-C, LDL-C, Total cholesterol, Triglycerides

INTRODUCTION

Lipids are of crucial importance for all cells in human body as a structural component for cell membranes. However in excess, cholesterol and/or triglycerides is strongly associated with an increased risk for the development of cardiovascular diseases in the future.

An increase in low-density lipoprotein cholesterol (LDL-C) and decrease in high-density lipoprotein cholesterol (HDL-C) levels are associated with the atherosclerotic process, with its prodromal stages starting in early life.1-3 Concentrations of the cord blood lipoprotein subtypes are influenced by fetal malnutrition and prematurity.4,6

High concentrations of triglyceride-rich lipoproteins, and apolipoprotein B in infants with a low gestational age; and increase of Apo c-1 rich in high-density lipoprotein (HDL) with low birth weight are potential risk factors for cardiovascular disease in the future.7,8

It is known that deviations in maternal hyperlipidemia, such as those caused by hypercholesterolemia, even when temporary and limited to pregnancy, trigger pathogenic
events in the fetal aorta and may lead to atherosclerosis later in life.

Hence, this study was planned to evaluate whether maternal lipid profile have an impact on lipid profile of the newborn.

**METHODS**

The present study has been conducted in Neonatal units of Department of Pediatrics, SMS Medical College, Jaipur from April 2015 to March 2016. It was a hospital based, cross sectional study. Prior permission from the institutional ethical committee was obtained. Written consent was obtained by parent(s) of all enrolled children. A Predesigned performa was used for history and data collection.

After applying inclusion and exclusion criteria, all newborns were divided in two subsets on the basis of birth weight; those having birth weight <2.5 kg considered as group A and those having birth weight ≥2.5 kg taken as group B. 2.5 ml of Cord blood sample from each of enrolled newborns was collected from the placental end of the cord just after the delivery of the baby in a plane dry test tube and lipid profile was done using enzymatic colorimetric method. Serum LDL was estimated using Friedewald’s formula.

**Inclusion criteria**

Healthy neonates with Gestational age of >28weeks and < 42 weeks with Apgar score >7 at 5 minutes.

**Exclusion criteria**

Neonates with maternal hypertension either before or during pregnancy; paternal or maternal Hyperlipidemia; maternal Cardiovascular disease and Diabetes mellitus or gestational diabetes; maternal history of smoking; and neonates with congenital malformations, Hypoxic ischemic encephalopathy, small for gestation age.

**Statistical analysis**

Data was entered on excel sheet and analyzed statistically using SPSS, trial version 20 for Windows statistical software package (SPSS inc., Chicago, il, USA). The test of normality was done by Kolmogorov-Smirnov test. The Categorical data were presented as numbers (percent) and were compared among groups using Chi square test.

Normally distributed variables were summarized using the mean±SD, and non-normally distributed variables by the median and range Groups were analyzed using the student T Test for parametric data and Mann-Whitney U test for the non-parametric. Correlation analyses were performed using Spearman’s rho correlation coefficient. Probability P value <0.05 was considered statistically significant.

**RESULTS**

A total of 220 newborns (94 female and 126 male subjects), along with their mothers, were enrolled. Out of them 110 were <2.5 kg (group A) and 110 were ≥2.5kg (group B) babies.

**Table 1: Mean (SD) of lipid profile in maternal and umbilical cord blood (mg/dl).**

<table>
<thead>
<tr>
<th>Serum lipids</th>
<th>Mothers (n=220)</th>
<th>Neonates (&lt;2.5kg) (n=110)</th>
<th>Neonates (≥2.5kg) (n=110)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG (mg/dl)</td>
<td>160.14±37.20</td>
<td>60.44±36.63</td>
<td>41.61±18.05</td>
</tr>
<tr>
<td>TC (mg/dl)</td>
<td>188.5±27.54</td>
<td>103.30±33.65</td>
<td>96.04±24.33</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>108.67±24.86</td>
<td>67.22±30.93</td>
<td>64.71±21.02</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>47.23±12.94</td>
<td>23.51±7.78</td>
<td>23.05±7.04</td>
</tr>
<tr>
<td>VLDL-C (mg/dl)</td>
<td>32.58±7.49</td>
<td>12.72±8.95</td>
<td>8.27±3.59</td>
</tr>
</tbody>
</table>

**Table 2: Correlation between maternal lipid profile and babies’ lipid profile in low birth weight babies (Group A).**

<table>
<thead>
<tr>
<th></th>
<th>B TG</th>
<th>B TC</th>
<th>B VLDL</th>
<th>B LDL</th>
<th>B HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>M TG</td>
<td>Pearson correlation</td>
<td>-0.194</td>
<td>Sig. (2-tailed)</td>
<td>0.042</td>
<td></td>
</tr>
<tr>
<td>M TC</td>
<td>Pearson correlation</td>
<td>0.023</td>
<td>Sig. (2-tailed)</td>
<td>0.811</td>
<td></td>
</tr>
<tr>
<td>M VLDL</td>
<td>Pearson correlation</td>
<td>0.042</td>
<td>Sig. (2-tailed)</td>
<td>0.662</td>
<td></td>
</tr>
<tr>
<td>M LDL</td>
<td>Pearson correlation</td>
<td>0.117</td>
<td>Sig. (2-tailed)</td>
<td>0.222</td>
<td></td>
</tr>
<tr>
<td>M HDL</td>
<td>Pearson correlation</td>
<td>0.075</td>
<td>Sig. (2-tailed)</td>
<td>0.436</td>
<td></td>
</tr>
</tbody>
</table>
Maternal and neonatal serum lipid levels are shown in Table 1 and correlation coefficients between the maternal serum and umbilical cord blood lipid levels are presented in Table 2 and 3.

Table 3: Correlation between maternal lipid profile and babies’ lipid profile in normal birth weight babies (Group B).

<table>
<thead>
<tr>
<th></th>
<th>B TG</th>
<th>B TC</th>
<th>B VLDL</th>
<th>B LDL</th>
<th>B HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>M TG</td>
<td>Pearson correlation 0.111</td>
<td>Sig. (2-tailed) 0.248</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M TC</td>
<td>Pearson correlation -0.103</td>
<td>Sig. (2-tailed) 0.286</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M VLDL</td>
<td>Pearson correlation 0.114</td>
<td>Sig. (2-tailed) 0.237</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>M LDL</td>
<td>Pearson correlation -0.063</td>
<td>Sig. (2-tailed) 0.515</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M HDL</td>
<td>Pearson correlation 0.064</td>
<td>Sig. (2-tailed) 0.506</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

There was statistically significant but poor negative correlation observed between maternal TG and babies TG level \((r = -0.194, P = 0.04)\). There was a poor positive correlation between maternal TC and babies TC \((r = 0.023, P = 0.8)\), maternal VLDL and babies VLDL \((r = 0.042, P = 0.662)\), maternal LDL and babies LDL \((r = 0.117, P = 0.222)\) and maternal HDL and babies HDL \((r = 0.075, P = 0.436)\) in LBW babies which was not significant (Table 2).

In normal birth weight babies (group B) there was a poor positive correlation between maternal TG and babies TG \((r = 0.111, P = 0.248)\), maternal VLDL and babies VLDL \((r = 0.114, P = 0.237)\) and maternal HDL and babies HDL \((r = 0.064, P = 0.506)\) which was not significant. There was a poor negative correlation between maternal TC and babies TC \((r = -0.103, P = 0.286)\) and maternal LDL and babies LDL \((r = -0.063, P = 0.515)\) in normal birth weight babies which was not significant (Table 3).

DISCUSSION

In present study we observed higher lipid levels in preterm babies than term babies. Since cholesterol is utilized as substrate for fetal adrenal steroidogenesis, it is suggested that the increasing rate of growth and steroid production by the fetal adrenals near term is causally related to the significant decline in the concentration of both LDL-cholesterol and TC in fetal plasma during normal human development near term gestation.9

In present study, there was a poor negative correlation between maternal TG and babies TG levels which was significant \((r = -0.194, P = 0.04)\) in LBW babies and no other significant correlation was observed between any type of maternal and neonatal lipid profile among low birth weight babies (Table 2).

No statistically significant correlation was found between maternal and neonatal lipid profile among normal birth weight babies (Table 3). Poor positive correlation was found between maternal TG and babies TG \((r = 0.111, P = 0.248)\), maternal VLDL and babies VLDL \((r = 0.114, P = 0.237)\) and maternal HDL and babies HDL \((r = 0.064, P = 0.506)\). There was a poor negative correlation between maternal TC and babies TC \((r = -0.103, P = 0.286)\) and maternal LDL and babies LDL \((r = -0.063, P = 0.515)\).

The relationship between maternal cholesterol and cord blood cholesterol was independent of participants’ dietary, anthropometric and personal data.

Ghiasi A et al also found that there was a significant positive correlation between maternal TC and neonatal serum TC levels \((r = 0.23, P = 0.042)\) which was similar to our results in the LBW group, however in present study the correlation was not significant \((r = 0.023, P = 0.8)\).10

Choo KE et al found significant positive correlations between maternal and neonatal serum total \((P = 0.038)\) and especially HDL-cholesterol \((P < 0.001)\).11 This is in concordance with present study in the LBW group, however the correlation was not significant.

Ortega R et al found a significant correlation between maternal cholesterol concentrations and those of newborn infants \((r = 0.3298)\).12 A correlation was also found between maternal cholesterol levels and infant HDL-cholesterol \((r = 0.2575)\) and LDL-cholesterol \((r = 0.3053)\) levels. Further, a positive correlation was seen between maternal LDL-cholesterol and infant cholesterol \((r = 0.3204)\) and LDL-cholesterol \((r = 0.3507)\). In the present study, we also observed a positive correlation between maternal TC, LDL-C, HDL-C level and neonatal TC, LDL-C, HDL-C, TC, levels in low birth weight babies.
Since maternal serum TG does not seem to pass the placental barrier, we could not suggest a mechanism for the significant negative relationship between maternal TG and neonatal TG in low birth weight newborns.\textsuperscript{13}

**CONCLUSION**

In present study there was a statistically significant but poor negative correlation between maternal TG and babies’ TG level among low birth weight (<2.5kg) babies and no other significant correlation was observed between maternal lipid profile and newborn’s lipid profile. Hence it shows that neonatal lipogenesis may be independent of maternal lipogenesis. However more studies with larger number of subjects for a prolong follow up are recommended to clarify this issue further and to see the impact of further development of cardiac disease in later life.

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**Conflict of interest: None declared**  
**Ethical approval: The study was approved by the Institutional Ethics Committee**

**REFERENCES**


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