Lipid Levels of Pregnant Women at Parirenyatwa Antenatal Clinic and Labour Ward, Zimbabwe

Simbarashe Lovemore Kadzere¹, Rudo Muswe², Danai Tavonga Zhou¹,3*¹
¹University of Zimbabwe, College of Health Sciences, Department of Medical Laboratory Sciences, P.O. Box AV 178, Avondale, Harare
²University of Zimbabwe, College of Health Sciences, Department of Chemical Pathology, P.O. Box AV 178, Avondale, Harare, Zimbabwe
³Africa University, College of Health, Agriculture and Natural Sciences, P.O.Box 1320, Mutare, Zimbabwe

*Corresponding author
Danai Tavonga Zhou

Abstract: Serum concentrations of total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL) have been shown to increase appreciably during pregnancy to meet maternal and fetal metabolic demands. However, this may expose the body to dangers of atherosclerotic plaques. The current study was a transversal study of 99 healthy pregnant women attending the Parirenyatwa Antenatal Clinic and Labour Ward in Zimbabwe and 24 age-matched, healthy, non-pregnant women as controls. Serum samples were collected for estimation of lipids: TC, LDL and HDL were measured and TC/HDL ratio was calculated. According to this Zimbabwean study, normal pregnant women were found to have significant increase in TC, LDL and HDL when compared with normal non-pregnant control group (P<0.001). TC/HDL changed with age of gestation but remained within the normal range. The study concludes that normal pregnancy does not predispose pregnant women to atherogenic lipid profiles most likely due to corresponding increase in HDL that appreciatively lowered TC/HDL ratio during gestation in our setting.

Keywords: total cholesterol (TC), high density lipoprotein (HDL), pregnancy, atherosclerotic plaques

INTRODUCTION

Increases in lipid metabolism have been shown to occur during pregnancy and these may expose the body to atherosclerotic plaques [1,2]. Increased lipid production during pregnancy is necessary as an energy store while hypertriglyceridemia that occurs towards late gestation has a crucial role in milk formation [3-5].

During normal pregnancy all lipid fractions increase in parallel to increase in pregnancy age and this increase may be secondary to increases in estrogen and progesterone levels during gestation [6]. Studies have shown elevated plasma lipids in preeclampsia [7] and such lipid changes play a role in endothelial damage leading to atherosclerosis [8,9].

Alterations of serum lipids have been associated with gestational age with reports that serum lipid levels increase steadily with age of gestation [10]. However, if serum HDL concentration also increases, this results in decreased cardiac risk factor, signifying possible protection from atherosclerotic cardiovascular disease [11]. Hence, it is very crucial to determine whether hypercholesterolemia of pregnancy is atherogenic or not in our setting [12]. This study was carried out to determine TC, LDL, HDL levels and TC/HDL ratio in three groups of Harare pregnant women at different ages of gestation at the Parirenyatwa Group of Hospitals.

MATERIALS AND METHODS

Study Design and Setting

A transversal study was carried out at the Parirenyatwa Group of Hospitals Antenatal Clinic and Labour Ward in Harare, Zimbabwe. Lipids were assayed at the University of Zimbabwe, in the Department of Medical Laboratory Sciences.

Participants

The study involved 99 apparently healthy pregnant women attending the Parirenyatwa Antenatal Clinic and Labour Ward in Harare, Zimbabwe, compared to 24 apparently healthy non-pregnant age matched controls.
Inclusion Criteria
Pregnant women between the ages of 18 and 45 years with normal uncomplicated pregnancy were included in this study.

Exclusion Criteria
Women with documented hereditary hyperlipidemia; complications such as hypertension, hypothyroidism, gestational diabetes, renal failure, nephrotic syndrome and obesity were excluded.

Sample Analysis
Analysis was carried out on a BS 120 Mindray® chemistry analyzer for TC, HDL and LDL.

Statistical Analysis
Data was analyzed using student’s t-tests and Pearson chi-square tests at 95% confidence level.

RESULTS AND DISCUSSION
Average age of the women was 28.7±8.2 years and there was no difference in ages of women by trimester of pregnancy (Table 1). However, as shown in Table 1 there were some differences in lipid levels (TC, LDL, HDL, TC/HDL ratio) when the pregnant women were compared by trimester.

The mean value of TC increased gradually with increase in gestational age (Table 2) which remained when mean values were tested for pairs of groups such as non-pregnant control and pregnant women in first trimester, control subjects and women in second trimester and between women in the second and third trimester, respectively (P=0.0003). The percentage increase of TC (Table 3) between the first and second trimester was 32.42% and that between the first and third trimester was 50.85%, P<0.0001. TC level began to exceed the upper reference range value (6.19mmol/L) in the second trimester.

As shown in Table 2, there was also a gradual increase in the mean value of LDL with increasing gestation age though difference was not apparent when control subjects were compared with women in first trimester of pregnancy (P=0.1746). Percentage increase of mean LDL between those in first and second trimester was 35.51% whilst 62.32% was the increase when subjects in first trimester were compared to those in third trimester. Mean LDL reached the upper reference value (4.12mmol/L) for women in the third trimester.

There was steady increase in the mean value of HDL with increase in gestation age. We hypothesise that the increase in HDL was responsible for the gradual decrease in cardiovascular risk (which can be approximated by TC/HDL ratio) with increase in the gestation age (Table 2). There was no difference in mean TC/HDL ratio between women in the various groups except for women in the first and second trimesters and women in the first and third trimesters, respectively. In spite of differences between some groups all TC/HDL ratio values were below the upper reference value of 4.5.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control group n=24</th>
<th>1st trimester n=25</th>
<th>2nd trimester N=27</th>
<th>3rd Trimester n=48</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (N=124)</td>
<td>29.25±7.01</td>
<td>28.04±8.72</td>
<td>28.29±9.65</td>
<td>28.96±7.67</td>
<td>0.411</td>
</tr>
<tr>
<td>TC (N=124)</td>
<td>4.08±0.38</td>
<td>4.69±0.31</td>
<td>6.30±0.48</td>
<td>7.25±0.47</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LDL (N=124)</td>
<td>2.63±0.18</td>
<td>2.74±0.34</td>
<td>3.8±0.47</td>
<td>4.56±0.32</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL (N=124)</td>
<td>1.08±0.09</td>
<td>1.20±0.09</td>
<td>1.68±0.11</td>
<td>1.90±0.31</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TC/HDL ratio (N=124)</td>
<td>3.79±0.25</td>
<td>3.91±0.25</td>
<td>3.78±0.18</td>
<td>3.60±0.40</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

SD=standard deviation, P* for Bartlett’s test for equal variances, TC, total cholesterol; LDL, low density lipoprotein, HDL, high density lipoprotein; Comparison of means for all groups using one way ANOVA with Bonferroni adjustments for multiple testing
Table 2: Comparison of demographic and biochemical statistics of controls and women in 1st, 2nd and 3rd trimester

<table>
<thead>
<tr>
<th>Statistic (Mean±SD)</th>
<th>Comparison groups</th>
<th>P</th>
<th>Comparison groups</th>
<th>P</th>
<th>Comparison groups</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group</td>
<td>1st trimester</td>
<td></td>
<td>Control group</td>
<td>2nd trimester</td>
<td></td>
<td>Control group</td>
</tr>
<tr>
<td>Age</td>
<td>29.25±7.01</td>
<td>0.5958</td>
<td>29.25±7.01</td>
<td>0.8013</td>
<td>29.25±7.01</td>
<td>0.8761</td>
</tr>
<tr>
<td>TC</td>
<td>4.08±0.38</td>
<td>&lt;0.001</td>
<td>4.08±0.08</td>
<td>&lt;0.001</td>
<td>4.08±0.38</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LDL</td>
<td>2.63±0.18</td>
<td>0.1746</td>
<td>1.08±0.09</td>
<td>&lt;0.001</td>
<td>1.08±0.09</td>
<td>0.001</td>
</tr>
<tr>
<td>HDL</td>
<td>1.08±0.09</td>
<td>0.0915</td>
<td>3.79±0.25</td>
<td>0.9106</td>
<td>3.79±0.25</td>
<td>0.0453</td>
</tr>
<tr>
<td>TC/HDL ratio</td>
<td>3.79±0.25</td>
<td>0.0152</td>
<td>3.79±0.25</td>
<td>0.9106</td>
<td>3.79±0.25</td>
<td>0.0453</td>
</tr>
</tbody>
</table>

Table 3: Descriptive statistics for TC, LDL, HDL by trimester of pregnancy

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Population</th>
<th>Mean (mmol/L) ±SD</th>
<th>Median (mmol/L)</th>
<th>% increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>1st trimester</td>
<td>24</td>
<td>4.69±0.31</td>
<td>4.67</td>
</tr>
<tr>
<td></td>
<td>2nd trimester</td>
<td>24</td>
<td>6.30±0.48</td>
<td>6.35</td>
</tr>
<tr>
<td></td>
<td>3rd trimester</td>
<td>41</td>
<td>7.25±0.47</td>
<td>7.42</td>
</tr>
<tr>
<td>LDL</td>
<td>1st trimester</td>
<td>24</td>
<td>2.76±0.33</td>
<td>2.79</td>
</tr>
<tr>
<td></td>
<td>2nd trimester</td>
<td>24</td>
<td>3.74±0.47</td>
<td>3.81</td>
</tr>
<tr>
<td></td>
<td>3rd trimester</td>
<td>41</td>
<td>4.48±0.67</td>
<td>4.64</td>
</tr>
<tr>
<td>HDL</td>
<td>1st trimester</td>
<td>25</td>
<td>1.20±0.09</td>
<td>1.2 (0.11)</td>
</tr>
<tr>
<td></td>
<td>2nd trimester</td>
<td>27</td>
<td>1.68±0.11</td>
<td>1.67 (0.12)</td>
</tr>
<tr>
<td></td>
<td>3rd trimester</td>
<td>48</td>
<td>1.97±0.31</td>
<td>2.1 (0.23)</td>
</tr>
</tbody>
</table>

DISCUSSION

Comparison between mean age of control women and pregnant women showed no significant difference P=0.411 (Table 1). This possibly indicates less likelihood of age-dependent effects on lipid metabolism during pregnancy for this cohort. The concentrations of TC, LDL and HDL increased appreciably during gestation (Table 2). Percentage increase between first and second trimester was for: TC: 34.33%, LDL: 35.51%, HDL: 40%; and the percentage increased further by the time women reached third trimester (Table 3). Increases in TC, LDL and HDL levels may be a physiological change to ensure a continuous supply of energy and nutrients to the developing fetus, despite the intermittent maternal food intake [13]. Other studies reported that the TC, LDL, HDL, were affected by maternal hormonal changes (elevated insulin, progesterone, 17-beta estradiol and human placental lactogen) [14, 15], although hormone levels were not measured in the current study. The elevation in maternal cholesterol during late pregnancy is also attributed to the independent high capacity of the fetal tissues to synthesize cholesterol leading to lesser importance of maternal cholesterol for fetal growth [16]. This theory was not explored further in our current study.

A similar outcome was obtained by Okojie et al who determined serum lipid profiles in Nigerian pregnant women and discovered that TC level was significantly high during the first, second and third trimester when compared...
with that of the control subjects [17]. In contrast, a study contacted in India by Shalini et al showed no significant increase in TC during gestation [18]. Similar results were also obtained by Abdehai et al who assessed lipid profiles in Sudanese pregnant women and observed no significant increase in TC compared with controls, P=0.7534 [19].

Our study reports a significant increase in LDL level with increase in gestation age and this agrees with findings from the study by Mankuta et al and his colleagues who produced similar results with an average increase of about 23% in the third trimester [21]. Similar results were also produced by Okojie et al which showed no significant increase in LDL during the first trimester with elevations in the second and third trimester compared to controls [17]. Interestingly, in the current study a significant increase in HDL levels in pregnant women (1.58±0.40) was observed compared to controls (1.08±0.09), P<0.0001. HDL concentration showed gradual increase with gestation age and this finding is in agreement with a previous study by Okojie et al in which there was a significant increase in HDL level in normal pregnant women with increase in gestation age [17]. It was also in line with the results from Neboh et al who studied the relationship between lipid and lipoprotein metabolism in trimesters of pregnancy in Nigerian women and reported a significant increase in HDL with increase in gestation age [20]. In contrast, Mankuta et al study reported no such change in HDL concentration during the first trimester; the second trimester was characterized by elevation in HDL level followed by a decline in the third trimester. [21]. The results of the current study were also dissimilar to the outcome of the study performed by Nepoh et al in which a decrease in HDL levels during second trimester was reported [22].

CONCLUSION

The cardiovascular ratio (TC/HDL) was calculated in each trimester as a predictor of coronary heart disease in pregnant women. According to our results (Table 2), though TC/HDL ratio changed with age of gestation, levels remained within normal range (<4.5). The corresponding increase in HDL with TC and LDL significantly reduced the cardiovascular ratio with increasing gestational age. This finding was in line with similar studies that reported decreases in TCL/HDL ratio with gestation age [20, 23]. In conclusion, according to data obtained from this study, the pregnant women were found to have significant increase in pro-atherogenic TC, LDL together with increases in anti-atherogenic HDL when compared with non-pregnant control group. Thus, normal pregnancy does not predispose pregnant women to atherogenic lipid profile as previously thought because the corresponding increase in HDL alongside TC and LDL significantly lowers the TC/HDL ratio during normal pregnancy. Though data was not available, decrease in TC/HDL ratio during pregnancy could be attributed to the traditional Zimbabwean diet and less sedentary life style of women due to local activities such as farming and vending, which might possibly protect them from atherogenic lipid profile in our setting.

REFERENCES