Original Research Article

Lipid Profiles of Diabetic and Hypertensive Pregnant Subjects in some Referral Hospitals in Enugu State, Nigeria

P. Ikeyi Adachukwu*, O. Ogbonna Ann and C. Ndubuisi Sunday

Department of Science Laboratory Technology, Institute of Management and Technology (IMT), Enugu, Nigeria

*Corresponding author

ABSTRACT

The level of lipid profile in the serum of hypertensive and diabetic pregnant women were determined in order to establish relationships between lipid, hypertension and diabetes in pregnancy. Twenty pregnant women who were hypertensive, diabetic and diabetic-hypertensive were recruited for the study. The study was aimed at analyzing serum lipid profile for triglyceride (TG), High density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and total cholesterol (TC) in hypertensive and diabetic pregnant women attending UNTH as out-patients. Data were analyzed using ANOVA and (p<0.05) was considered statistically significant. Finally, there was no significant difference in the mean serum concentration of the four groups, that is total Mean ±SD for hypertensive pregnant women 2.6530 ± 1.78, diabetic pregnant women 2.2145 ± 1.39, both condition 2.1485 ± 1.13 and non condition 1.930 ± 1.45. These changes may have important implications for the development of atherosclerosis and the long term cardiovascular health of women with diabetes and hypertension.

Introduction

Diabetes and hypertension in pregnant women as associated with an increased incidence of congenital abnormalities when compared with normal pregnancy (Hagay et al., 2005). Frequency of diabetic and hypertensive mothers are estimated to be 6 -10% currently (Hagay and Reece, 2006). Alteration of lipid profile is known to occur in diabetic and hypertensive pregnant women (Caro and Klos, 2000). In reference to diabetes, pregnant women experience physiological changes to support foetal growth and development. Pregnancy is associated with significant changes in the functions of the normal liver. Although the precise mechanism underlying these various alterations is not clear in every case, their recognition is essential to a proper clinical evaluation (Angel, 2006). (Brizzi et al., 2008), explains that natural rising of plasma lipid is seen in normal pregnancy, but this event is not atherogenic and it is believed that this process is under hormonal control.
(Rovinsky and Gaffin, 2010). But in complicated pregnancy, there is a possible defect in the mechanism of adjusting physiological hyperlipidemia. Plasma lipid profile in the first trimester of pregnancy may predict the incidence and severity of preeclampsia. The anabolic phase of early pregnancy encourages lipogenses and fat storage in preparation for rapid foetal growth in late pregnancy. Lipolysis is increased as a result of insulin resistance, leading to increased flux of fatty acids to the liver thereby promoting the synthesis of very low-density lipoprotein (VLDL) and increased triglyceride (TG) concentration (Ross, 2007). Because of a decrease in the activity of lipoprotein lipase, very –LDL remains in the plasma and could lead to the accumulation of LDL.

An increase LDL is associated with the development of atherosclerosis (Ross, 2007). Abnormal lipid metabolism also seems important in the pathogenesis of pregnancy-induced hypertension (PIH). Pregnancy induce hypertension is characterized by elevated blood pressure, proteinuria and edema (Dutta, 2001). Although considered to be relatively rare in the United States, PIH occurs worldwide in from 2 to 35 percent of pregnancies, depending on diagnostic criteria and study population. PIH is also called preeclampsia and it occurs most often in young women with first pregnancy. It is more common in twin pregnancies, in which chronic hypertension, pre-existing diabetes and in women who had PIH in their previous pregnancy. Hypertensive disorders in pregnancy, contribute significantly to serious complications for both the foetus and mother (Hagay et al., 2005).

PIH occurs more frequently in women with pre-existing hypertension than in women who are normotensive prior to pregnancy. The hypertensive disorders of pregnancy collectively represent a significant public health problem in the United States and throughout the world. The cause and nature of this disorder is only partially understood (Angel, 2006). Therefore, this study was carried out to evaluate the plasma lipid concentrations in normal and hypertensive pregnancies in order to establish the link between hypertension, diabetes and pregnancy.

**Objectives of the study**

To determine the biochemical parameters (lipid profile) of some diabetic and hypertensive pregnant women that attends three referral Hospitals in Enugu State, Nigeria.

**Statement of the problem**

This research work based mainly on the examination of differences in lipid profile among diabetic, hypertensive, non-hypertensive and non diabetic pregnant women, so to avoid future complications which may lead to atherosclerosis, myocardial infection and otherwise congenital abnormalities.

**Scope of the study**

Scope of the study was limited to the lipid profile of diabetes and hypertensive pregnant women

**Materials and Methods**

**Experimental Design**

A total of 20 subjects were used from three referral Hospitals / Clinics

Group 1: Normal pregnant subjects (5 subjects) that were non-diabetic and non-hypertensive
Group 2: Diabetic pregnant women (5 subjects) without hypertension.

Group 3: Hypertensive pregnant women (5 subject) without diabetes

Group 4: Pregnant women (5 subjects) that are both hypertensive and diabetic

Sample collection and storage

A total of (2.5mls) of venous blood were collected. The serum was separated from the cells using Pasteur pipette, transferred into cryogenic vial and screwed tightly. The samples were stored in ultra freezer.

Lipid profile determination

Determination of Total Cholesterol Concentration

Total cholesterol concentration was determined by the method of Allain et al. (1976) using Randox kit.

Determination of High-Density Lipoproteins (HDL) – Cholesterol Concentration

High density lipoprotein (HDL) concentration was determined by the method of Albers et al. (1978) using Randox kit.

Determination of Triacylglycerol Concentration

Triacylglycerol (TAG) concentration was determined by the method of Allain et al. (1976) using Randox kit.

Determination of Low Density Lipoprotein-Cholesterol Concentration

Low density lipoprotein (LDL) concentration was determined by the method of Assmann et al. (1984) using Randox kit.

Results and Discussion

The results obtained from this study show that in hypertensive and diabetic pregnant women the plasma level for the four parameter namely TC, HDL, LDL and TG were not-significant by (p>0.005). This pattern of hyperlipidaemia is not in agreement with the report of (Ross, 2007) which state that lipolysis is increased as a result of insulin resistance and changes in lipid metabolism during pregnancy, leading to increase flux of fatty acids to the liver thereby promoting the synthesis of very low-density lipoprotein and increased triglyceride concentration, because of a decrease in the activity of lipoprotein lipase, very LDL remains in the plasma for longer and leads to the accumulation of LDL.

Fig.1, 2, 3, 4 and 5 showed that the lipid profile of hypertensive pregnant women, diabetic pregnant women, both diabetic and hypertensive pregnant women and non diabetic and hypertensive pregnant women were not significant when compared.

It can be therefore concluded that the high lipid concentrations in hypertension and diabetic pregnant woman obtained from this study seems not to be directly involved with hypertension and diabetes in pregnancy and appears to be a reflection of the metabolic condition of pregnant women.
Table 1: Lipid Profile of Pregnant Women

<table>
<thead>
<tr>
<th>Group</th>
<th>Hypertensive Pregnant Women</th>
<th>Diabetic Pregnant Women</th>
<th>Both Hypertensive and Diabetic Women</th>
<th>Non Hypertensive and Diabetic Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>5.1040</td>
<td>4.1920</td>
<td>3.9580</td>
<td>3.6160</td>
</tr>
<tr>
<td>N</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Std Deviation</td>
<td>1.20649</td>
<td>78570</td>
<td>23274</td>
<td>1.54686</td>
</tr>
<tr>
<td>HDL-C</td>
<td>1.5840</td>
<td>1.0840</td>
<td>1.2800</td>
<td>0.8480</td>
</tr>
<tr>
<td>N</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Std Deviation</td>
<td>47987</td>
<td>18716</td>
<td>22068</td>
<td>46677</td>
</tr>
<tr>
<td>LDL-C</td>
<td>2.9540</td>
<td>2.4800</td>
<td>2.0160</td>
<td>2.0900</td>
</tr>
<tr>
<td>N</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Std Deviation</td>
<td>69745</td>
<td>47265</td>
<td>35360</td>
<td>1.27736</td>
</tr>
<tr>
<td>TG</td>
<td>0.9900</td>
<td>1.1020</td>
<td>1.3400</td>
<td>1.2580</td>
</tr>
<tr>
<td>N</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Std Deviation</td>
<td>0.63400</td>
<td>0.47151</td>
<td>0.20482</td>
<td>0.48792</td>
</tr>
<tr>
<td>Total Lipid</td>
<td>2.6580</td>
<td>2.2145</td>
<td>2.1485</td>
<td>1.9530</td>
</tr>
<tr>
<td>N</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Std Deviation</td>
<td>1.78190</td>
<td>1.39314</td>
<td>1.13737</td>
<td>1.45753</td>
</tr>
</tbody>
</table>

![Fig.1: Total Cholesterol (mmol/L)](image)

![Fig.2: High Density Lipoprotein Cholesterol (mmol/L)](image)
**Fig. 3: Low Density Lipoprotein Cholesterol (mmol/L)**

**Fig. 4: Triacylglycerol (mmol/L)**

**Fig. 5: Total Lipids (mmol/L)**
References