Fasting and Postprandial Lipid Profile in Type 2 Diabetes Mellitus: A Comparative Study

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ABSTRACT

Introduction: Globally the incidence of type 2 DM is increasing due to various factors like a high level of insulin resistance, genetic predisposition, and environmental factors. Hyperglycemia characterizes type 2 DM due to insulin resistance or insulin deficiency with a group of metabolic abnormalities, which includes dyslipidemia, increase in macrovascular disease. So the present study aimed to assess the importance of fasting dyslipidemia concerning postprandial dyslipidemia, in the pathogenesis of atherosclerotic changes.

Material and Methods: The present study conducted in the Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, Puducherry, a tertiary health care center with an annual volume of 100,000 patients. The study approved by Institutional Human Ethical Committee for the year 2015-2016. The investigation was initiated with 100 cases (type 2 DM patients) and 100 controls (Non-diabetic patients) equally distributed in both genders. As per procedure, informed consent taken from the patients in prescribed formats before their participation in the study. The statistical analysis done by using the Students unpaired ‘t’-test.

Results: The fasting and postprandial lipid profile significantly altered in individuals with type 2 diabetes when compared with controls. The postprandial lipid parameters significantly increased in the type 2 DM subjects as compared to the fasting lipid parameters, and the postprandial HDL level significantly decreased as compared to the fasting HDL level.

Conclusion: Hence in the present study postprandial lipid profile significantly increased when compared with fasting lipid profile among type 2 DM patients. So we suggest to include the estimation of postprandial lipid profile, in addition to the fasting lipid profile to assess the risk for CVD among type 2 DM patients.

Keywords: Fasting and Postprandial Lipid Levels, Cardiovascular Risk Factor, Type 2 Diabetes Mellitus, Dyslipidemia, Metabolic Syndrome

INTRODUCTION

Diabetes mellitus (DM) referred as a group of metabolic disorders characterized by high blood sugar levels over an extended period. Hyperglycemia occurs due to increase in high blood sugar levels by a deficiency in insulin action or secretion or both. It may lead to disturbances in the metabolism of lipid, carbohydrates, and protein.¹,²,³,⁴ Worldwide, among DM the prevalence of type 2 or Non-Insulin dependent diabetes mellitus (NIDDM) increasing significantly in South Asian population, especially in developing country like India. The incidence is increasing due to several factors like high BMI, a top body fat percentage, high upper body adiposity, a high degree of genetic predisposition, high level of insulin resistance, and high susceptibility to an environmental factor. Globally, over 382 million people involving age group of 20-79 years have Type 2 DM making it a significant health disease. India has the highest prevalence (estimated 65.1 million) of this disease in the world and hence WHO considered India as the diabetic capital of the world.⁵,⁶,⁷ Deficiency of Insulin or resistance to Insulin may affect the vital enzymes and pathways in lipid metabolism resulting lipid abnormalities in DM.⁸ In a standard human diet, cholesterol and triglycerides are the significant fats that are taken up by the body. Insulin, the hormone that is primarily affected by diabetes has a role in esterification of fat in addition to assisting the glucose uptake and subsequent conversion into fat in the periphery and inhibition of hormone-sensitive lipase. Hence, in type 2 DM, the state of hyperinsulinemia hampers the entire downstream process that results in an abnormal increase of lipids especially triglycerides (TG) and cholesterol in the bloodstream, i.e., diabetic dyslipidemia. Also, it also reported that half-life of HDL reduced whereas, the half-life of LDL increased that exerts immunogenic effects on diabetic patients causing damage to arterial endothelium.⁹ It may introduce further risks for several macro-vascular complications that could affect a large proportion of the population, and around 80% of Indian diabetic patients are likely to have such
cardiovascular diseases (CVD). Hence, attempts for adequate control of dyslipidemia clinically could help to manage the DM and related complications. However, this aspect has been largely overlooked and remains underdiagnosed.  

Fasting dyslipidemia increases the risk of CVD as seen by a meta-analysis of six large prospective studies done by Hokanson J.E and Austin, which showed a significant rise of TGs associated with cardiovascular diseases. But in contrast, one of the earliest studies done on dyslipidemia showed a substantial increase of developing CVD with abnormal postprandial lipid levels. Additionally, there is high cardiovascular mortality associated with postprandial dyslipidemia in Type 2 DM. Regular exercise has been shown to give many benefits in controlling diabetes as well as a positive effect on lowering the lipid. Such training also increases the level of HDL cholesterol and improves insulin receptor sensitivity and reduces LDL-C levels. Based on various studies enumerated in the literature, it is clear that diabetes is now a significant lifestyle disease in India and coexists with dyslipidemia. However, countrywide data is lacking on the abundance and distribution of dyslipidemia and related control measures. Since it has established that attempts to lower the LDL-C level could be a useful treatment way, there is a need to generate sufficient data for meaningful analysis of the pattern of dyslipidemia to design adequate control measures of dyslipidemia among the Indian diabetic patients originating from various ethnicities. Hence the present study aimed to compare fasting lipid levels with post-prandial lipid levels in type 2 DM.

MATERIAL AND METHODS

The present study was conducted in the Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, Puducherry, a tertiary health care center with an annual volume of 100,000 patients. The study was approved by Institutional Human Ethical Committee for the year 2015-2016. The study was initiated with 100 cases (type 2 DM patients) and 100 controls (non-diabetic patients) equally distributed in both genders. As per procedure, informed consent taken from the patients in prescribed formats before their participation in the study.

Inclusion criteria

- Type 2 DM patients aged between 30-60 years who were on oral hypoglycaemic drugs.
- Duration of diabetes of more than five years.

Exclusion criteria

As per study protocols patients excluded were those with

- Chronic Kidney Disease
- Acute Coronary Syndrome,
- Hypertension,
- Malignancy,
- Epilepsy,
- Stroke,
- Patients on Antiplatelets,
- Antiepilept Females.

Parameters measured

In the present study the following parameters were measured:

- FBS
- HbA1c
- PBS
- Total Cholesterol (TC)
- Triglycerides (TG)
- High Density Lipoprotein – Cholesterol (HDL-C)
- Very-low-density lipoprotein cholesterol (VLDL)
- Low-density lipoprotein cholesterol (LDL)
- LDL-Cholesterol = total cholesterol - (HDL-Cholesterol + tri glycerides/5)
- VLDL-C = triglycerides/5

Fasting blood sugar estimated by using GOD-POD method and HbA1c was estimated by using direct enzymatic assay method by using Ion exchange chromatography (Crest A Coral clinical system, USA.). Serum total cholesterol was measured by the CHOD-PAP method, triglycerides were measured by the GPO-Trinder method, HDL-cholesterol measured by the Phosphotungstic acid method, and the values of Low-density lipoproteins (LDL) and very-low-density lipoprotein cholesterol (VLDL) can be calculated using Friedewald’s equation as follows:

Reference range

The standard reference ranges according to the kits are as follows. FBS (normal range 70-110mg/dl), and HbA1c (normal range 4.2-6.5%). Normal values for lipid profile parameters are total cholesterol (TC < 200 mg/dl), triglycerides (TG < 150mg/ dl), HDL-Cholesterol (30-60 mg/dl), LDL-Cholesterol (<100 mg/ dl), and VLDL-Cholesterol (20-40 mg/dl).

STATISTICAL ANALYSIS

Various parameters listed in the protocol of study have been analysed statistically using tools implemented in Graph Pad Prism v3.0. To determine statistical significance (P < 0.05) between fasting and post-prandial lipid profiles, repeated measured ANOVA and additionally Newman-Keuls Multiple Comparison Test were used. Spearman coefficient was used for correlation tests and in addition where all the tests were two-tailed and a P value less than 0.05 were considered statistically significant.

RESULTS

In the present study, 200 subjects were included and divided into two groups, 100 controls (non-diabetic) and 100 cases (type 2 DM) with the age range of 30 – 60 years. Out of 100 non-diabetic controls, 58 were males and 42 females, and in...
Table-2: Demographic data of the study subjects

<table>
<thead>
<tr>
<th>Variables</th>
<th>Controls (n=100)</th>
<th>Cases (n=100)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender wise distribution</td>
<td>58M/42F</td>
<td>52M/48F</td>
</tr>
<tr>
<td>Family history of Diabetes (yes/no)</td>
<td>31/69</td>
<td>20/80</td>
</tr>
<tr>
<td>Age (years)</td>
<td>47.35 ± 8.38</td>
<td>47.6±8.2</td>
</tr>
<tr>
<td>Residence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tamilnadu</td>
<td>66</td>
<td>73</td>
</tr>
<tr>
<td>Puduchery</td>
<td>34</td>
<td>27</td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Farmer</td>
<td>26</td>
<td>42</td>
</tr>
<tr>
<td>Housewife</td>
<td>40</td>
<td>33</td>
</tr>
<tr>
<td>Labourer</td>
<td>34</td>
<td>25</td>
</tr>
</tbody>
</table>

Table-3: Comparison of fasting lipid profile among the cases and controls.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Cases (type 2 Diabetic) (n=100) Mean ± SD</th>
<th>Controls (Non-Diabetic) (n=100) Mean ± SD</th>
<th>t-test</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBS</td>
<td>147.79 ± 42.08</td>
<td>84.80 ± 6.37</td>
<td>14.79</td>
<td>&lt; 0.0001*</td>
</tr>
<tr>
<td>HbA1c</td>
<td>9.121 ± 1.824</td>
<td>4.806 ± 0.189</td>
<td>23.52</td>
<td>&lt; 0.0001*</td>
</tr>
<tr>
<td>TC</td>
<td>214.44 ± 15.16</td>
<td>180.13 ± 10.87</td>
<td>18.38</td>
<td>&lt; 0.0001*</td>
</tr>
<tr>
<td>TG</td>
<td>182.88 ± 19.58</td>
<td>149.65 ± 9.46</td>
<td>15.28</td>
<td>&lt; 0.0001*</td>
</tr>
<tr>
<td>HDL-C</td>
<td>43.41 ± 7.46</td>
<td>57.32 ± 4.91</td>
<td>15.57</td>
<td>&lt; 0.0001*</td>
</tr>
<tr>
<td>VLDL</td>
<td>36.57 ± 3.91</td>
<td>29.92 ± 1.90</td>
<td>15.29</td>
<td>&lt; 0.0001*</td>
</tr>
<tr>
<td>LDL</td>
<td>169.84 ± 16.00</td>
<td>86.97 ± 9.82</td>
<td>44.14</td>
<td>&lt; 0.0001*</td>
</tr>
</tbody>
</table>

< 0.0001* = extremely statistically significant.

Table-4: Comparison of Postprandial lipid profile among the cases and controls.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Fasting Mean ± SD</th>
<th>Postprandial Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>214.44 ± 15.16</td>
<td>238.13 ± 22.09</td>
</tr>
<tr>
<td>TG</td>
<td>182.88 ± 19.58</td>
<td>189.87 ± 14.70</td>
</tr>
<tr>
<td>HDL-C</td>
<td>43.41 ± 7.46</td>
<td>36.69 ± 7.86</td>
</tr>
<tr>
<td>VLDL</td>
<td>36.57 ± 3.91</td>
<td>37.87 ± 5.27</td>
</tr>
<tr>
<td>LDL</td>
<td>169.84 ± 16.00</td>
<td>175.89 ± 18.25</td>
</tr>
</tbody>
</table>

Table-5: Comparison of fasting and postprandial lipid profile of type 2 diabetes mellitus.

DISCUSSION

Diabetes Mellitus (DM) and lipid profile together are related to being the essential predictors and also increase the risk factors for dyslipidemia, cardiovascular diseases (CVD), Hypertension and metabolic syndrome. Among

100 diabetic cases, 52 were males and 48 women as shown in the Table – 1, Figure - 1 and Figure – 2. Table 2 explains the demographic status of the study subjects.
metabolic abnormalities, dyslipidemia is most common abnormality associated with DM.

In the present study, we compared the fasting and postprandial lipid profile between controls (non-diabetic subjects) and type 2 DM patients, i.e., TC, TG, LDL and VLDL levels were significantly increased in the type 2 DM patients as compared to those in the control subjects both in fasting and also postprandial. The HDL levels are decreased considerably in the type 2 DM patients when compared with the controls in both fasting and even in postprandial as shown in table 3 and 4.

In the present study, we compared the fasting lipid profile with postprandial lipid profile among type 2 DM subjects. TC, TG, LDL, and VLDL, significantly increased in the type 2 DM as compared to the fasting lipid profile. The postprandial HDL levels significantly decreased to the fasting HDL levels as shown in table 5. Similar studies reported by Haffner SM (1998), Idogun ES et al., 2007, and Albrki WM et al., 2007. According to Krauss RM, 2004, dyslipidemia is a most common metabolic abnormality and frequently associated with DM. Among DM patients’ defect in lipid metabolism have been reported and significantly increases the risk of cardiovascular atherosclerosis. Several studies conducted on fasting lipid levels in type 2 DM, very few studies done on postprandial lipid levels in type 2 DM. According to studies reported by Madhu SV et al., 2005, Axelsen M et al., 1999 postprandial dyslipidemia is more critical in atherosclerosis and the pathogenesis of the vascular changes and increases the significant risk factors for the CVD. According to Suryabhan L L et al., 2013, asymptomatic and symptomatic macrovascular diseases are linked with postprandial hypertriglyceridemia among type 2 DM patients. Oxidative stress and postprandial dysmetabolism related to the insulin resistance. Therefore it increases the prevalence of cardiovascular disease among type2 DM. Prolonged and exaggerated postprandial lipid profile linked with mortality and morbidity of CVD.

CONCLUSION

Hence in the present study postprandial lipid profile significantly increased when compared with fasting lipid profile among type 2 DM patients. So we suggest to include the estimation of postprandial lipid profile, in addition to the fasting lipid profile to assess the risk for CVD among type 2 DM patients.

REFERENCE


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