ABSTRACT

Background: Diabetes Mellitus (DM) is a chronic metabolic disease characterized by hyperglycemia with type 2 DM accounting for more than 90% of adult diabetes cases worldwide. Hyperglycemia in type 2 DM is strongly linked with dyslipidemia which in turn increases risk for development of cardiovascular diseases. This study aimed to determine the correlation between glycemic parameters (fasting blood sugar-FBS, postprandial blood sugar-PPBS and glycated hemoglobin-HbA1c) and serum lipid profile in type 2 diabetic patients. Methods: Altogether 160 type 2 diabetic patients (Male=80 and Female=80) aged between 30-70 years visiting Chitwan Medical College Teaching Hospital (CMCTH) for their routine medical check-up were included in this study. Data were collected using preformed set of questionnaires and biochemical data were obtained from the laboratory analysis of the patient’s blood samples. Statistical analysis was done with SPSS version 20.0. Results: Hyperglycemia was significant as evident by elevated FBS, PPBS and HbA1c. Also, the serum lipid profile was deranged with elevated TC, TG, LDL, VLDL and lowered HDL levels suggestive of dyslipidemia. There was a Direct and significant correlation of glycemic parameters (FBS, PPBS and HbA1c) with total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C) while the correlation with high density lipoprotein cholesterol (HDL-C) was just reverse. Conclusion: Chronic hyperglycemia in type 2 DM is associated with dyslipidemia which further exposes risk for the development of cardiovascular diseases. HbA1c, being the gold standard for the assessment of glycaemia is also the better predictor of dyslipidemia in type 2 diabetic patients.

Key words: Dyslipidemia, Glycemic control, HbA1c, Type 2 Diabetes Mellitus.

INTRODUCTION

Diabetes mellitus (DM) is one of the most common chronic metabolic disorders that is characterized by hyperglycemia. It is caused by a complex interaction of genetics, environmental factors, and life-style choices. Many factors contribute to hyperglycemia in DM which may include reduced insulin secretion, decreased glucose utilization, and increased glucose production. Distinct genetic and metabolic defects in insulin action and/or secretion give rise to the common phenotype of hyperglycemia in type 2 DM. Various genetic loci contribute to susceptibility, and environmental factors (such as nutrition and physical activity) further modulate phenotypic expression of the disease. Individuals with this form of diabetes do not need insulin treatment for survival either initially, or often throughout their lifetime.

Type 2 DM frequently goes undiagnosed for many years because the hyperglycemia develops...
gradually and at earlier stages is often not severe enough for the patient to notice any of the classic symptoms of diabetes.\textsuperscript{1,4} Nevertheless, such patients are at increased risk of developing macrovascular and microvascular complications.\textsuperscript{5,6} Type 2 diabetic patients are more prone to develop cardiovascular disease (CVD) compared to the general population.\textsuperscript{7} Dyslipidemia, an established risk factor for CVD, is strikingly common in patients with type 2 diabetes, affecting almost 50% of this population.\textsuperscript{8}

The glycated haemoglobin (HbA1c) is still considered as the gold standard in the assessment and monitoring of glycemia in Diabetic patients.\textsuperscript{9} This study aimed to determine the correlation between glycaemic status and lipid profile of type 2 Nepalese diabetic patients.

**MATERIALS AND METHODS**

This is a hospital based case-control study conducted in the Department of Biochemistry, Chitwan Medical College Teaching Hospital (CMCTH), Bharatpur during February to July 2016 for a period of six months.

Study population: This study included a total of 160 patients (80 male and 80 female) with type 2 DM attending CMCTH for their routine medical check-up. World Health Organization (WHO) criteria were used for the diagnosis of patients with type 2 DM.\textsuperscript{10} All data were collected from personal interviews using a preformed set of questionnaires.

**Sample collection:** Five ml of the venous blood in fasting state was collected with the help of a sterile 5 ml syringe from the antecubital vein of each of the consenting subjects and kept in (EDTA) vacutainer.

**Biochemical analysis:** Fasting blood sugar (FBS) was measured by glucose oxidase-peroxidase (GOD-POD) method.\textsuperscript{11} Glycated hemoglobin (HbA1c) was estimated by NycoCard Reader.\textsuperscript{12} Blood collected in plain test tube was allowed to clot at room temperature and the serum was carefully separated. Serum lipids (triglyceride-TG, total cholesterol-TC, and high density lipoprotein cholesterol-HDL-C) were directly measured and the value of low density lipoprotein cholesterol LDL-C was calculated using the Friedewald’s formula.\textsuperscript{13} All these parameters were analyzed using a fully automated chemistry analyzer (Siemens Advia Centaur 1800) and ready-to-use reagent kits according to the manufacturer’s instructions (Siemens Diagnostics, Germany).

**Ethical issues:** The study was approved by the institutional ethical committee and informed consent was obtained from all the patients.

**Data analysis:** The obtained data were analyzed using Statistical Package for Social Sciences (SPSS) version 20. Quantitative data were expressed as mean and standard deviation (SD). Pearson's bivariate correlation analysis was used to show the correlation between blood sugar parameters and lipid profile. p value less than 0.05 was considered to be statistically significant.

**RESULTS**

Table 1. shows the baseline characteristics of the study subjects. The result showed no significant difference between age, BMI, WHR, SBP and DBP between male and female patients included in this study.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Male</th>
<th>Female</th>
<th>Mean values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>51.8 ± 9.0</td>
<td>52.55 ± 9.6</td>
<td>52.17 ± 9.2</td>
</tr>
<tr>
<td>BMI (kg/m\textsuperscript{2})</td>
<td>25.9 ± 3.0</td>
<td>25.8 ± 3.7</td>
<td>25.9 ± 3.3</td>
</tr>
<tr>
<td>WHR</td>
<td>0.91 ± 0.07</td>
<td>0.90 ± 0.06</td>
<td>0.90 ± 0.07</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>131.0 ± 8.5</td>
<td>130.1 ± 6.4</td>
<td>130.5 ± 7.6</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>85.0 ± 6.6</td>
<td>86.79 ± 4.8</td>
<td>85.9 ± 6.0</td>
</tr>
</tbody>
</table>

Abbreviations: BMI= Body Mass Index, WHR= Waist Hip Ratio, SBP= Systolic Blood Pressure, DBP= Diastolic Blood Pressure

Table 2. shows the mean values of blood glucose parameters (FBS, PPBS and HbA1c) and lipid profiles. With the exception of TC, no statistically significant difference between male and female biochemical parameters was seen in this study.
Table 2. Biochemical parameters of diabetic patients

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Male</th>
<th>Female</th>
<th>Mean values</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBS (mg/dl)</td>
<td>139.7 ± 43.2</td>
<td>134.8 ± 36.4</td>
<td>137.25 ± 39.9</td>
</tr>
<tr>
<td>PPBS (mg/dl)</td>
<td>215.4 ± 57.2</td>
<td>212.2 ± 63.0</td>
<td>213.8 ± 59.6</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>7.27 ± 1.2</td>
<td>7.21 ± 1.0</td>
<td>7.24 ± 1.1</td>
</tr>
<tr>
<td>TC* (mg/dl)</td>
<td>214.1 ± 28.5</td>
<td>197.0 ± 19.7</td>
<td>206.0 ± 26.0</td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>188.9 ± 53.6</td>
<td>174.1 ± 31.9</td>
<td>181.9 ± 45.0</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>46.57 ± 6.6</td>
<td>45.63 ± 4.8</td>
<td>46.1 ± 5.8</td>
</tr>
<tr>
<td>LDL-C* (mg/dl)</td>
<td>129.4 ± 27.7</td>
<td>116.6 ± 19.1</td>
<td>123.3 ± 24.7</td>
</tr>
<tr>
<td>VLDL-C (mg/dl)</td>
<td>38.0 ± 10.7</td>
<td>34.73 ± 6.3</td>
<td>36.5 ± 9.0</td>
</tr>
</tbody>
</table>

Table 3. Correlation between glycemic parameters and lipid profile

<table>
<thead>
<tr>
<th>Correlation between</th>
<th>TC</th>
<th>TG</th>
<th>HDL-C</th>
<th>LDL-C</th>
<th>VLDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBS</td>
<td>0.218**</td>
<td>0.316**</td>
<td>-0.140*</td>
<td>0.206**</td>
<td>0.316**</td>
</tr>
<tr>
<td>PPBS</td>
<td>0.230**</td>
<td>0.332**</td>
<td>-0.173*</td>
<td>0.266**</td>
<td>0.332**</td>
</tr>
<tr>
<td>HbA1c</td>
<td>0.270**</td>
<td>0.381**</td>
<td>-0.185*</td>
<td>0.291**</td>
<td>0.381**</td>
</tr>
</tbody>
</table>

* significant difference between male and female, at the level of p<0.05

**Correlation is significant at the 0.01 level.

The relationship between blood glucose parameters and lipid profile is shown in table 3. The result showed statistically significant positive correlation between FBS, PPBS and HbA1c with TC, TG, LDL-C and VLDL-C. In addition, there was an inverse relationship between HDL-C and all glucose parameters (FBS, PPBS and HbA1c). This showed that hyperglycemia is strongly associated with derangement of lipid metabolism.

DISCUSSION

Diabetes Mellitus is characterized by chronic hyperglycemia. Constantly elevated blood glucose level results in glycation of common proteins including Hemoglobin. HbA1c is still considered as the gold standard for the assessment of glycemic control in diabetic patients. Measurement of HbA1c provides the mean blood glucose of the past 8-12 weeks. In this study, we found significantly raised blood glucose parameters including HbA1c. We also found significant positive correlation of glycemic parameters with TC, TG, LDL-C and VLDL-C while the correlation with HDL-C was just reverse. Also, the serum lipid profile was deranged with elevated TC, TG, LDL-C, VLDL-C and lowered HDL-C levels suggestive of dyslipidemia.

Majority of people with type 2 DM have insulin resistance as primary defect. Many studies have shown that insulin affects the liver apolipoprotein production and regulates the enzymatic activity of lipoprotein lipase and cholesterol ester transport protein, which causes dyslipidemia in diabetes mellitus.14-16 Hence insulin resistance is a key factor for the pathogenesis of type 2 DM and contributes to dyslipidemia.17 Dyslipidemia as a metabolic abnormality is strongly linked with type 2 DM. Its prevalence is variable, depending on the type and severity of diabetes, glycaemic control, nutritional status, age and other factors.

Our findings in this study are concomitant with Khan et al. who also reported a direct correlation between FBS and HbA1c with TC, TG and LDL-C and inverse correlation with HDL-C.18 Mahato et al. also observed significant correlations between HbA1c with TC, LDL-C and LDL-C/HDL-C ratio.19 Ramona et al. reported direct and significant correlation between HbA1c with TC, TG and LDL-C, and reverse correlation with HDL-C.20 In our study, correlation of HbA1c with the lipid...
profile was even more compared to FBS and PPBS. This finding supports HbA1c is even better predictor of dyslipidemia and can be used as a potential biomarker for predicting dyslipidemia in type 2 diabetic patients.

The most common pattern of dyslipidemia in type 2 diabetic patients is elevated TG levels and decreased HDL-C levels, which is also prevalent in our study. Chronic hyperglycemia in type 2 DM associated with dyslipidemia, further increases two-four fold excess risk for the development of coronary heart disease. The highest priority for diabetic individuals who have poor glycaemic control should be to achieve near normal blood glucose levels, in the expectation that this approach will also improve dyslipidemia.21-23 Our study clearly depicts that lipid fractions are abnormal in type 2 DM. Realizing that most of the diabetics have a high probability of developing cardiovascular and cerebrovascular disease, it is essential that an individual who is diabetic should take care of dyslipidemia.

CONCLUSIONS

Our study confirms, type 2 DM is strongly linked with dyslipidemia. For the optimal care of type 2 diabetic patients, frequent monitoring of lipid profile along with blood sugar is equally important to lessen the risk for development of CHD. HbA1c, being gold standard in the assessment of glycemic control together with its strong correlation with the lipid profile, makes it an ideal marker for predicting dyslipidemia in type 2 DM.

REFERENCES


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