Original Research Article

Association between thyroid hormones & lipid profile in type 2 diabetes mellitus patients- A case control study in tertiary care hospital

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ABSTRACT

Background: Diabetes mellitus & thyroid dysfunction are the most common endocrinological disorders in the current era. Diabetes mellitus is a group of metabolic disorders usually associated with dyslipidemia which increases risk of cardiovascular disorders and resultant morbidity & mortality. Thyroid dysfunction particularly hypothyroidism is frequently encountered in diabetic patients. T2DM results in reduction in thyroid releasing hormone (TRH) synthesis & limits T3 & T4 production, which results in increased total cholesterol & LDL in circulation. By studying the association of thyroid hormones & lipid profile in diabetes patients, we can enhance the knowledge about the interrelation between diabetes, thyroid hormones & dyslipidemia.

Aim: To study the association between thyroid hormones & lipid profile in Type 2 Diabetes Mellitus patients.

Methodology: This is a case control study comprising of 50 cases (T2DM) & 50 age & sex matched healthy controls. Fasting blood sample was taken and analysed for blood sugar, lipid profile, Triiodothyronine(T3), Thyroxine(T4) & TSH.

Results: In this study, higher serum level of total cholesterol, LDL-cholesterol & triglycerides seen in T2DM patients. Also significantly lower T3&T4 and higher TSH are observed in T2DM. Moreover, there was a significant positive correlation between TSH and TC, LDL-C and TGL & significant negative correlation between T3&T4 and TC, LDL-C & TGL.

Conclusion: Thyroid dysfunction is associated with lipid dysregulation in T2DM patients. Early & routine screening of thyroid disorders in T2DM patients can reduce morbidity due to dyslipidemia.

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1. Introduction

Diabetes mellitus and thyroid dysfunction are the most common endocrinological disorders in the current era. Type 2 Diabetes mellitus is one of the most important global health issue as it affects more than 463 million people and it is expected to reach around 700 million by 2045.1 Type 2 Diabetes mellitus consists of an array of dysfunctions characterized by hyperglycemia, resulting from the combination of resistance to insulin action and/or inadequate insulin secretion. It is usually associated with dyslipidemia, which increases risk of cardiovascular events leading to morbidity & mortality. Thyroid dysfunction has been frequently encountered in diabetes patients with hypothyroidism being the most common type of dysfunction.2 Hypothyroidism, an important cause of secondary dyslipidemia, is often accompanied with increased levels of total cholesterol & low density lipoprotein-cholesterol (LDL-C).3 By studying the association of thyroid hormones & lipid profile in diabetes patients, we can enhance the knowledge about the interrelation between diabetes, thyroid hormones and lipid profile.

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2. Materials and Methods

This is a case control study conducted in a tertiary care hospital comprising of 50 Type 2 DM cases attending outpatient department and 50 age & sex matched healthy controls.

Patients with history of thyroid disorders, chronic liver disease, chronic kidney disease, cardiovascular disease, pregnant & lactating women and patients on drugs affecting thyroid hormones were excluded from the study.

2.1. Sample collection

After overnight fast, 5 ml of venous blood was collected in a plain tube and a tube containing EDTA. Serum and plasma are separated by centrifugation at 3000 rpm for 10 minutes. Fasting blood glucose and Postprandial blood glucose estimated by Glucose oxidase- peroxidase method in ERBA XL 640 auto analyser. Total cholesterol was estimated by cholesteryl oxidase phenol 4-aminoantipyrine peroxidase (CHOD PAP) method, Triglycerides by Glycerol phosphate oxidase phenol 4-aminoantipyrine peroxidase (GPO PAP) method, and HDL-C by PEG/CHOD-PAP method in ERBA XL 640 auto analyser.

2.2. Low-Density Lipoprotein cholesterol (LDL-C) was calculated using Friedewald’s equation

LDL- Cholesterol = total cholesterol - (HDL cholesterol + triglycerides/ 5) T3, T4, TSH (normal range : T3: 0.69 – 2.15 ng/ml, T4: 5.2 -12.7 µg/dl, TSH :0.3-4.5 µIU/ml) estimated by chemiluminescence immunoassay method in Maglumi 800.

2.3. Statistical analysis

Statistical analysis was done by IBM SPSS Statistics for Windows, Version 20.0. Mean and SD were used to summarize the continuous variables. Independent samples t test was used to test the significance in difference between the parameters for cases and controls. Pearson’s correlation coefficient was used to check the linear relation between parameters of lipid profile and thyroid profile. A P value of <0.05 was considered as statistically significant.

3. Result

Age and sex distribution is shown in Table 1 and Table 2. Cases include 28 males & 22 females whereas controls include 25 males & 25 females with mean age of 51.85±12.42 & 51.37±12.81 respectively.

The mean fasting blood glucose and post prandial blood glucose level among cases and controls which was found to be statistically significant.

Figure 1 shows the mean fasting blood glucose and post prandial blood glucose level among cases and controls which was found to be statistically significant.

Figure 2 shows that TSH is significantly and positively correlated with LDL-C, TG and TC and negatively correlated with that of HDL-C.

T3 & T4 levels are significantly and negatively correlated with LDL-C, TG and TC and positively correlated with that of HDL-C as shown in Figures 3 and 4.

4. Discussion

In this present study, higher serum level of Total cholesterol, triglycerides & LDL –C seen in T2DM patients. Dyslipidemia in diabetes is due to low activity of lipoprotein lipase or limited lipoprotein clearance.

Result of the present study showed that the levels of serum T3, T4 were significantly lower in diabetes while serum TSH was significantly higher in diabetes when compared to that of controls. These findings were consistent.
Table 1: Age distribution of cases and controls

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Yrs)</td>
<td>Cases</td>
<td>51.85</td>
<td>12.42</td>
<td>0.92</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>51.37</td>
<td>12.81</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Gender distribution of cases and controls

<table>
<thead>
<tr>
<th>Gender</th>
<th>N (Cases)</th>
<th>%</th>
<th>N (Controls)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>28</td>
<td>56.0</td>
<td>25</td>
<td>50.0</td>
</tr>
<tr>
<td>Female</td>
<td>22</td>
<td>44.0</td>
<td>25</td>
<td>50.0</td>
</tr>
</tbody>
</table>

Table 3: Biochemical parameters of cases and controls

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Mean</th>
<th>SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBS (mg/dL)</td>
<td>Cases</td>
<td>199.30</td>
<td>29.61</td>
<td>0.0008</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>92.26</td>
<td>13.99</td>
<td></td>
</tr>
<tr>
<td>PPBS (mg/dL)</td>
<td>Cases</td>
<td>250.25</td>
<td>28.29</td>
<td>0.0005</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>128.37</td>
<td>20.61</td>
<td></td>
</tr>
<tr>
<td>TC (mg/dL)</td>
<td>Cases</td>
<td>208.50</td>
<td>29.67</td>
<td>0.007</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>174.21</td>
<td>17.41</td>
<td></td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>Cases</td>
<td>162.50</td>
<td>23.55</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>99.79</td>
<td>17.56</td>
<td></td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>Cases</td>
<td>141.80</td>
<td>18.39</td>
<td>0.0004</td>
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<tr>
<td></td>
<td>Controls</td>
<td>87.47</td>
<td>11.65</td>
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<tr>
<td>HDL-C (mg/dL)</td>
<td>Cases</td>
<td>27.9</td>
<td>4.3</td>
<td>0.0005</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>50.5</td>
<td>8.7</td>
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</tr>
<tr>
<td>T3 (ng/mL)</td>
<td>Cases</td>
<td>0.59</td>
<td>0.08</td>
<td>0.029</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>1.79</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>T4 (µg/dL)</td>
<td>Cases</td>
<td>4.5</td>
<td>1.90</td>
<td>0.005</td>
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<tr>
<td></td>
<td>Controls</td>
<td>8.84</td>
<td>1.17</td>
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<tr>
<td>TSH (µIU/mL)</td>
<td>Cases</td>
<td>4.98</td>
<td>1.32</td>
<td>0.0002</td>
</tr>
<tr>
<td></td>
<td>Controls</td>
<td>1.97</td>
<td>0.98</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 3: Correlation between T3 and lipid profile

with that found by Sarala Devi Tenepalli et al., Jiffri EH et al., Demitrost et al., In diabetic patients, the nocturnal TSH peak is blunted and the TSH response to TRH is impaired, resulting in reduced iodide uptake by the thyroid gland that limits T3 and T4 production.

Also this study shows TSH is significantly and positively correlated with LDL-C, TG and TC and negatively correlated with that of HDL-C. T3 & T4 levels are significantly and negatively correlated with LDL-C, TG and TC and positively correlated with that of HDL-C. Our findings are in harmony with that of Palacios et al., Asvold et al., Saeed W et al., Decreased levels of thyroid hormones attenuate activity of lipoprotein lipase (LPL), the enzyme responsible for clearance of TG-rich lipoproteins and thus lead to increased levels of TG in the serum. Thyroid hormones such as T3 have been demonstrated

Fig. 4: Correlation between T4 and lipid profile
to regulate LDL receptors by directly binding to thyroid hormone responsive elements (TREs) and controlling sterol regulatory element-binding protein. In hypothyroidism, decreased thyroid hormones lead to reduced expression of LDL receptors, which may attenuate cellular uptake of LDL-C from circulation and catabolism of LDL-C and finally result in increased levels of circulating TC.\textsuperscript{12,13}

5. Conclusion
Thyroid dysfunction is associated with lipid dysregulation in T2DM patients. By maintaining good glycemic control in T2DM, we can prevent the development of thyroid dysfunction and its complications. Also, screening for thyroid disorders and early intervention in T2DM patients can reduce morbidity due to dyslipidemia. Hence thyroid function tests may be included as part of routine investigations in patients with Type 2 DM.

6. Source of Funding
None.

7. Conflict of Interest
The authors declare that there is no conflict of interest.

References

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