Comparison Study of insulin level and lipid profile in diabetes mellitus in Ramady city

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ABSTRACT

The aim of this study was to determine of insulin and complete lipid profile in patients of diabetes mellitus (type 1 and type 2). The investigation was carried out in a group of 80 patients (45 samples of type 2 and 35 samples of type 1). Blood serum was used to determine enzymatically the concentration of lipid: total cholesterol, High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL), triglycerides (Tri), Very Low Density Lipoprotein (VLDL) and insulin. The results revealed that serum cholesterol, LDL, Tri and VLDL were significantly (p≤0.05) higher in type 1 DM and also in type 2 DM (p≤0.05). serum insulin and HDL were significantly (p≤0.05) lower in type 1 DM and type 2 DM.

Keywords: insulin, lipid profile, diabetes mellitus, Ramady.

Introduction

Diabetes mellitus is associated with the development of premature arteriosclerosis and a higher cardiovascular morbidity and mortality (1, 2). Diabetic dyslipidaemia is believed to play an important role in the pathogenesis of accelerated atherosclerosis in this condition (3). The predominant lipid abnormalities seen in diabetes mellitus are an elevated serum triglyceride (Tg) level and a low HDL-C level. While several studies have found a significant association of fasting hypertriglyceridaemia (4) and coronary artery disease (CAD) in diabetes mellitus.

Type 1 diabetes is an autoimmune disease that occurs when the insulin-producing beta cells within the pancreas are gradually destroyed and eventually fail to produce insulin. Insulin is a hormone that helps the body's cells use glucose for energy.

The causes of type 1 diabetes are complex and still not completely understood. People with type 1 diabetes are thought to have an inherited, or genetic, predisposition to the disease. Researchers believe that this genetic predisposition may remain dormant until it is activated by an environmental trigger or triggers such as a virus or a chemical. This starts an attack of the immune system that results in the eventual destruction of the beta cells of the pancreas (5).

Patients with type 1 diabetes present when progressive β-cell destruction has crossed a threshold at which adequate insulin secretion and normal blood glucose levels can no longer be sustained (6,7).

Type 2 diabetes is more common in adults aged 50 and older, although it can occur in children and adolescents as well (8). Type 2 Unlike type 1 diabetes, where destruction of the pancreas reduces and eventually stops the supply of the hormone insulin, most people with type 2 diabetes are still able to produce insulin at diagnosis. However, the insulin they produce is unable to perform its primary job, which is helping the body's cells use glucose for energy. Usually this is due to a problem with the body’s insulin receptors, the location on cells where insulin binds so that glucose can enter (although less frequently there may be a problem with the chemical makeup of the insulin itself). This condition is called insulin resistance (5). Type 2 is more complex condition than type 1 diabetes because there is combination of resistance to the action insulin in liver and muscle together with impaired pancreatic β-cell function leading to "relative" insulin deficiency (6).

Materials and methods

The study location is in the Ramadi, Iraq. A total of 80 samples were taken to assess the lipid profile and insulin in diabetes mellitus group (45 samples of type 2, 35 samples of type 1). Samples of normal individuals which served as controls were evaluated for lipid profile and insulin. Age group in both diabetes and controls were taken from 25-65 years. Venous blood samples
were taken from all the subjects in the morning of the fasting overnight. Serum total cholesterol levels was determined by enzymatic colorimetric method (9), triglycerides by the enzymatic method (10), HDL-cholesterol was estimated using precipitant method (11), LDL-cholesterol by Friedewald formula (12) and VLDL was calculated indirectly by the method of Wilson (1998) as shown below (13):

\[ \text{VLDL-cholesterol (mg/100ml) = Triglycerides (mg/dl) / 5} \]

Insulin measured by DRG insulin enzyme immunoassay kit (ELISA) based on the sandwich principle (14).

All values were expressed as mean ± S.D statistical significance of differences between control and study groups were evaluated by L.S.D test. The acceptable level of significance was \( p \leq 0.05 \).

### Results and Discussion

Comparison of mean value of lipid profile between type 1, type 2 and control group was shown in (table 1).

Mean serum cholesterol, LDL, TRI and VLDL were significantly higher in type 1 DM (\( p \leq 0.05 \)) and also in type 2 DM (\( p \leq 0.05 \)).

Mean serum insulin and HDL were significantly lower in type 1 DM (\( p \leq 0.05 \)) and also in type 2 DM (\( p \leq 0.05 \)).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Type 1 diabetes</th>
<th>Type 2 diabetes</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>*192.60 ±10.88</td>
<td>*191.66 ±11.76</td>
<td>141.76 ±8.58</td>
</tr>
<tr>
<td>HDL (mg/dl)</td>
<td>*30.382.0 ±9</td>
<td>*33.33 ±3.03</td>
<td>40.60 ±1.62</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>*122.49 ±12.21</td>
<td>*112.35 ±10.23</td>
<td>66.22 ±12.09</td>
</tr>
<tr>
<td>TRI (mg/dl)</td>
<td>*184.71 ±12.07</td>
<td>*196.35 ±14.72</td>
<td>121.37 ±19.24</td>
</tr>
<tr>
<td>VLDL (mg/dl)</td>
<td>*39.71 ±3.91</td>
<td>*39.89 ±2.75</td>
<td>24.26 ±3.84</td>
</tr>
<tr>
<td>INSULINE(µ/m)</td>
<td>*3.58 ±0.16</td>
<td>*4.50 ±0.12</td>
<td>5.01 ±0.14</td>
</tr>
</tbody>
</table>

*indicates (\( p \leq 0.05 \))

The results suggest that the over production of LDL is one of the possible explanation of hypercholesterolemia was loss of affinity for Apo B receptors of the glycated LDL may contribute to the increase in serum cholesterol level in diabetes.

Other cause which could explain the high serum cholesterol in diabetic patients is that, the intake of saturated fatty acid is positively associated with high serum cholesterol (15).

It is a well known fact that low HDL-C is common in type 2 and may be a strong factor for coronary heart disease (CHD) (16,17). Which is in agreement with our result that low HDL-C is evident in both types compared with the control group (table 1).

High serum LDL-C is noted in both types. It could be explained that patients have an increased level of small dense LDL-C and reduced clearance due to glycosylation of ligand proteins (18).

Serum TRI and VLDL-C have always been increased in diabetic patients. The most common lipid abnormality in DM is the hypertriglyceridemia which is known to be an independent risk factor of CHD. It is due to increase in VLDL-C synthesis and an impaired VLDL-C catabolism (19), however this phenomenon is less evident in type 1 with different mechanism which is based on the fact that in type 1 DM the reduced chylomicron and VLDL catabolism occur as a direct consequence of reduced lipoprotein lipase activity due to insulin deficiency. Since the enzyme activity requires insulin for activation (20).

Low serum insulin is shown in both types. Such decreasing is more in type 1 then type 2, because of destruction of insulin producing beta cells in the pancreas resulting in absolute insulin deficiency (6,7).

### References


