The correlation between disordered lipid profile (dyslipidemia) and progression of diabetes mellitus type 2

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Abstract

The study aims to assess the correlation between disordered lipid profile (dyslipidemia) and progression of diabetes mellitus type 2.

Materials and Methods: Fifty of types 2 DM patient diagnosed according to history and clinical examination, who attended the laboratory of 14 ramadan specialist analyses of immune and advanced hormones, and twenty-five apparently healthy(control group) and both group age ranged from 40-70 years in the period between 17/6/2016 until 25/8/2016.

Results: The results of triglycerides level in blood increase in patients with type2 diabetes (245.12 ± 21.98 mg/dl as compared with control group 134.60 ± 8.45 mg/dl ) , the level of cholesterol in blood also increase in patients with type2 diabetes (253.70 ±4.79 mg/dl as compared with control group 185.44 ± 7.85 mg/dl ) , and the level of HbA1c increase in patients with type2 diabetes (8.29 ± 0.21 mg/dl as compared with control 5.11 ± 0.08 mg/dl). Results showed a strong correlation between patients with type 2 diabetes and high level of triglycerides which gave Correlation coefficient-r 0.3 (P-value<0.01) and also triglyceride with HbA1c gave Correlation coefficient-r 0.31 (P-value<0.01) which increases the risk of coronary heart disease and macro and micro vascular complications which causes retinopathy, neuropathy and cardiovascular disease.

Conclusion: Blood levels of HbA1c have a significant correlation with levels of blood sugar and can give a clear idea about glycaemic control in the past three months and hence can be used as a preferred method to assess glycaemic control in diabetic.

Keywords: Dyslipedemia; diabetes mellitus type 2; cholesterol; HbAlc

Introduction

Whereas type 2DM is the most common of diabetes , its specific etiology is not yet known . Its frequency varies in different racial and ethnic subgroups and is often associated with a strong familial,likely genetic, predisposition more than autoimmune . The role for Human leukocyte antigen ( HLA ) system in the pathogenesis of type 2DM is less clear, and weak links between HLA class (I and II) and type 2 diabetes were reported for some ethnic groups however some alleles of class II consider as markers of susceptibility for type 2 diabetes in another ethnic groups. HLA is a kind of genetic marker of human beings that presents a component of the immune system, encoded by highly polymorphic genes that vary across racial/ethnic groups, has been suggested to be a biologically based risk factor for type 2diabetes and this may explain some of its variation by race/ethnicity [ 1,2].

Type 2 diabetes is associated with a cluster of interrelated plasma lipid and lipoprotein abnormalities, including reduced HDL cholesterol, a predominance of small dense LDL particles, and elevated triglycerides . These abnormalities occur in many patients despite normal LDL cholesterol levels. These changes are also a feature of the insulin resistance syndrome (also known as the metabolic syndrome), which underlies many cases of type 2 diabetes. The aims of present study is to assess the correlation between disordered lipid profile (dyslipidemia) and progression of diabetes mellitus type 2.

Material and Methods

Sample collection

This study include (50) patients were diagnosed with type-2 diabetes mellitus and (25) control. Blood samples were collected
from patients between (40-70 years old) with diabetes type 2, the samples were collected in the laboratory of 14 Ramadan specialist advanced immunological analyzes and hormones in the period between June and August.

Patients were fasting overnight for at least 10-12 hours, and the next morning 5 milliliters of venous blood samples were collected for the serum lipid profile and glycated hemoglobin (HbA1c).

**Material**

(Working methods depending on instructor of the manufacture company of kits).

**Methods**

**A-Glucose:** -Glucose kits (Linear- Spain).

<table>
<thead>
<tr>
<th>Tube</th>
<th>Blank</th>
<th>Sample</th>
<th>CAL Standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1. Monoreagent</td>
<td>1.0 ml</td>
<td>1.0 ml</td>
<td>1.0 ml</td>
</tr>
<tr>
<td>Sample (serum)</td>
<td>-</td>
<td>10 µl</td>
<td>-</td>
</tr>
<tr>
<td>CAL Standard</td>
<td>-</td>
<td>-</td>
<td>10 µl</td>
</tr>
</tbody>
</table>

- Incubate the sample and CAL standard for 5 minutes
- Photometer of measuring absorbance at 500 ± 10 nm.
- Calculation:
  A sample/ A standard * C standard = mg/dl total glucose
  C standard = 100mg/dl

**A-Cholesterol:** -Cholesterol kits (Linear- Spain).

<table>
<thead>
<tr>
<th>Tube</th>
<th>Blank</th>
<th>Sample</th>
<th>CAL Standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1. Monoreagent</td>
<td>1.0 ml</td>
<td>1.0 ml</td>
<td>1.0 ml</td>
</tr>
<tr>
<td>Sample (serum)</td>
<td>-</td>
<td>10 µl</td>
<td>-</td>
</tr>
<tr>
<td>CAL Standard</td>
<td>-</td>
<td>-</td>
<td>10 µl</td>
</tr>
</tbody>
</table>

- Incubate the sample and CAL standard for 5 minutes
- Photometer of measuring absorbance at 500 ± 10 nm.
- Calculation:
  A sample/ A standard * C standard = mg/dl total cholesterol
  C standard = 200mg/dl

**B-Triglyceride:** -Triglyceride kits (Linear- Spain).

<table>
<thead>
<tr>
<th>Tube</th>
<th>Blank</th>
<th>Sample</th>
<th>CAL Standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1. Monoreagent</td>
<td>1.0 ml</td>
<td>1.0 ml</td>
<td>1.0 ml</td>
</tr>
<tr>
<td>Sample (serum)</td>
<td>-</td>
<td>10 µl</td>
<td>-</td>
</tr>
<tr>
<td>CAL Standard</td>
<td>-</td>
<td>-</td>
<td>10 µl</td>
</tr>
</tbody>
</table>

- Incubate the sample and CAL standard for 5 minutes
- Photometer of measuring absorbance at 500 ± 10 nm.
- Calculation:
  A sample/ A standard * C standard = mg/dl total triglycerid
  C standard = 200mg/dl

**D-HbA1c:** -HbA1c kits (STANBIO-Texas).

<table>
<thead>
<tr>
<th>Tube labeled</th>
<th>Unknown (U)</th>
<th>Standard (S)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lysing reagent</td>
<td>5.0 ml</td>
<td>5.0 ml</td>
</tr>
<tr>
<td>Sample (blood)</td>
<td>100 µl</td>
<td>-</td>
</tr>
<tr>
<td>CAL Standard</td>
<td>-</td>
<td>100 µl</td>
</tr>
</tbody>
</table>

- Allow to stand for 5 minutes at room temperature to complete hemolysis.
- Pipette 100 µl of the prepared hemolysate into appropriately labeled resin tube.
- Position a resin separator in the pre-Fil tube so rubber sleeve is approximately 1-2 cm above liquid level.
- Mix tubes on a hematology rocker for 5 minutes.
- At the end of 5 minutes mixing, push resin separator into tube until resin is firmly packed in bottom of the 13mm tube.
- Pour each supernate directly into separate cuvettes for absorbance measurement, read absorbance (A) of standard and unknown at 415 nm within 60 minutes.
- Calculation:
  Glycohemoglobin (%) = A unknown/A standard*C Standard (%)
  C Standard (%) = 10

**Results and Discussion**

This study was designed to determine the relationship between the levels of cholesterol, triglyceride and HbA1c in diabetes mellitus type 2 patients. Fasting blood glucose, cholesterol, triglyceride and HbA1c was determined enzymatically by using kits.

Figure (4.1) and appendix showed the result a significant increase in the level of fasting blood glucose in diabetic patients compared to control groups (186.98 ± 8.60 mg/dl vs. 95.24 ± 1.61 mg/dl).

This result is similar to other studies were given the same indicator higher glucose levels in patients compared to control group such as the study of Suryawanshi et al., [3] and Barua et al.,[4]. The high levels will be for many reasons, according to research and studies. One of these reasons is defective islet cell function is the primary event which may be due to an autoimmune reaction producing hyperglycemia in type 2 DM [5].

In diabetes, the body either fails to properly respond to its own insulin or does not make enough insulin, or both. This causes glucose to accumulate in the blood, often leading to various complications [6].

This result from absolute or relative impairment in insulin secretion or insulin action or both [7], also have disorder in fat
metabolism as a result of obesity and abnormal insulin action, hypertension, elevated cholesterol (combined hyperlipidemia), and with the condition often termed metabolic syndrome [8].

There is also a strong inheritable genetic connection in type 2 diabetes having relatives (especially first degree) with type 2 is a considerable risk factor for developing type 2 diabetes [9]. The level of cholesterol was significantly increased in diabetic patients compared to control groups (253.70 ±4.79 mg/dl vs. 185.44 ± 7.85 mg/dl) as it is shown in Figure (4.2).

These results are supported by other studies such as Hashim, Shankarpras et al, Suryawanshi et al, Panditsingh et al, and Zeqollari et al.

Several reasons lead to the increase, according research and studies. In diabetes, many factors may affect blood lipid level, this is because carbohydrate and lipid metabolisms are interrelated to each other if there is any disorder in carbohydrate metabolism, it also leads to lipid metabolism disorder, so the results revealed that there was a significant increase in lipid profile in diabetic patient, high concentration of cholesterol in type 2 diabetics [14].

The possible reason for high serum cholesterol in diabetes may be due to decrease muscular exercise or inhibition of cholesterol catabolism

In type 2 diabetes mellitus enhanced lipolysis leads to high free fatty acid levels in plasma and consequent accumulation of fat in liver. Due to this, more Acetyl-COA is now available which cannot be efficiently oxidized by TCA cycle because the availability of oxaloacetate is limited. The stimulation of gluconeogenesis is responsible for the depletion of oxaloacetate. The excess of Acetyl-COA therefore is diverted to cholesterol leading to hypercholesterolemia [15,16].

When person of type 2 diabetes eats meal, insulin cannot stop the manufacture of glucose in the liver, but it can stop the burning of fat stores “fatty acids accumulate from food and from the liver” [17].

Diabetic state appears to be associated with increased synthesis of cholesterol. It has been hypothesized that hyperphagia of diabetes induces increased activity of HMG-CoA reductase of the intestine resulting in increased synthesis of cholesterol leading to raised levels in plasma. Dietary cholesterol also adds up to total cholesterol by increased absorption [18-19].

The results showed the level of triglyceride (TG) was significantly higher in patients suffering from diabetes compared to control groups (245.12 ± 21.98 mg/dl vs. 134.60 ± 8.45 mg/dl) as it is shown in Figure (4.3).

Several reasons lead to the increase, according research and studies. The hypertriglyceridemia may be due to higher rates of production of triglyceride and VLDL particles have been the most commonly identified metabolic abnormalities [12]. Also Lipoprotein Lipase enzyme, which removes TG from VLDL is activated leading increased level of VLDL, LDL and TG in diabetes mellitus type2 [19]. Many hyper-triglyceridemic type2 patients also appear to have defect in the clearance of triglyceride with lipoproteins [20].
There is hyperlipidemia, especially an increase in non-esterified free fatty acids, triglycerides and cholesterol. Other factors which are responsible for hypercholesterolemia are low fiber diet, lack of exercise, sedentary and inactive life style, high energy intake tends to obesity, stress etc. Lipid abnormalities that occurs in this situations are hypertriglyceridemia hypercholesterolemia [15,16].

The results also showed high level of HbA1c in diabetic patients compared to control groups (8.29 ± 0.21 mg/dl vs. 5.11 ± 0.08 mg/dl) as it is shown in Figure (4).

Similar type of result obtained by [4,21]. In type2 diabetes fasting blood glucose and HbA1c correlate very reliably with each other , although establishing precise relationship between the two is difficult.[4].

**Correlation coefficient between diabetes, cholesterol, triglyceride and HbA1c**

Table (1) results showed the differences moral between parameters in patients, through this relationship and studies we found that the relationship between diabetes type 2 and triglyceride gave the highest probability at the level compare to other parameter, and triglyceride with HbA1c also gave highest probability at the level.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Correlation coefficient-r</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBG&amp;Cholesterol</td>
<td>0.43 **</td>
<td>0.0001</td>
</tr>
<tr>
<td>FBG&amp;Triglyceride</td>
<td>0.30 **</td>
<td>0.0099</td>
</tr>
<tr>
<td>FBG&amp;HbA1c</td>
<td>0.69 **</td>
<td>0.0001</td>
</tr>
<tr>
<td>Cholesterol &amp; Triglyceride</td>
<td>0.52 **</td>
<td>0.0001</td>
</tr>
<tr>
<td>Cholesterol &amp; HbA1c</td>
<td>0.51 **</td>
<td>0.0001</td>
</tr>
<tr>
<td>Triglyceride &amp; HbA1c</td>
<td>0.31 **</td>
<td>0.0072</td>
</tr>
</tbody>
</table>

**Table 1:** Correlation coefficient between parameters in patients

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