INTRODUCTION

Diabetes mellitus DM is heredity, chronic and endocrine metabolic disorder which causes death worldwide. Type 2 diabetes is associated with cluster of interrelated plasma lipid and lipoprotein abnormalities, including reduced HDL cholesterol, predominance of small density LDL particles and elevated triglycerides.

There are probably 100 million people in the world with diabetes mellitus and incidences of diabetes are on the rise. As diabetes progress patients are at increased risk of developing coronary disease.

NIDDM is directly linked with dyslipidemia due to the lack of effect of insulin. Altered atherogenic lipoprotein pattern and elevation of some liver enzymes have been identified as independent risk factors for the development of cardiovascular disease along with prevalence of liver enzymes abnormality ranging from 7.2 to 22.9% in patients of NIDDM.

Infact, pre-diabetic individuals often exhibit an atherogenic pattern of risk factors that includes higher levels of total cholesterol, LDL cholesterol, triglycerides and lower levels of HDL cholesterol than individuals who do not develop diabetes.

In case where a disease such as diabetes disrupts metabolic function, the body’s electrolyte control system is broken down. The results of electrolyte imbalance can be severe.

In diabetic out patients, acid–base and electrolyte disorders occurred often even if the renal function is normal, and the most common disorders are metabolic alkalosis and
metabolic acidosis, in addition, the common electrolyte disorders are hypernatremia and hypokalemia.\textsuperscript{11}

**MATERIALS AND METHODS**

This study was conducted at the department of chemistry, Faculty of Science with collaboration of Central Medical Lab in Suliamania city, Kurdistan region/Iraq.

**The study group**

This study was conducted on 3 groups: group I, II and III. The patients were diagnosed on the basis of detailed clinical history, clinical examination and other relevant biochemical investigations. The patients suffering from other diseases, such as diabetes, inflammatory diseases, hepatic impairment, cardiac diseases and other systemic diseases. Fasting venous blood were drawn from all.

**Collection of blood samples**

After an overnight fasting of 10-12 hours, about 5 ml of whole blood was collected via vena puncture with the help of a disposable syringe in between 8.00-9.00 am.

Glucose detected by enzymatic reaction (glucose oxidase and peroxidase = GOD-POD).\textsuperscript{12}

Different lipid fractions were estimated along with fasting plasma glucose. Serum total cholesterol was determined by an enzymatic (CHOD-PAP) colorimetric method,\textsuperscript{13} triglycerides were determined by an enzymatic (GPO-PAP) method,\textsuperscript{14} HDL-cholesterol was estimated by a precipitant method,\textsuperscript{15} LDL-cholesterol was estimated by using Friedewald formula.\textsuperscript{16}

\[
\text{LDL-cholesterol} = \text{Total cholesterol} - (\text{HDL-cholesterol} + \text{triglycerides}/5)
\]

Serum analysis for fasting Na\textsuperscript{+}, K\textsuperscript{+} and Cl\textsuperscript{-} was performed by the automatic analyzer, ROCHE module Cobas 6000 (C-501 and C-601), and kits were procured by ROCHE.

Statistical analysis was carried out using standard deviation and chi-square test from which (P) value was derived. The P value <0.05 was considered to be significant.

**RESULTS**

The mean ± SD age of patients was 39.98±3.91 (range 25-55) while the mean±SD of control was 40.02±1.41 (23-57) years. Out of 85 patients 45 (55%) were male and 40 (45%) were females. Among control subjects 25 (50%) for male and females, with mean of duration of the disease (10.2±5.9) years (Table 1).

Descriptive statistics of all diagnostic parameters on group I, II and III presented in Tables 2-4.

Table 2 show the mean total cholesterol, triglycerides, HDL-Cholesterol, LDL-Cholesterol and fasting blood sugar levels (group I), the patients in this group have NIDDM syndrome with normal values of lipid profiles. The mean levels of serum Na\textsuperscript{+} (128.2±3.1 meq/L) were significantly lower in the sera of NIDDM in comparison to the control subjects (140.09±2.33 meq/L), the mean level of K\textsuperscript{+} (4.21±0.34 meq/L) show non significance change in this group, while serum Cl (110.16±5.54 meq/L) increased significantly in the sera of NIDDM patients in comparison to that of control subjects (95.08±0.08 meq/L).

Table 3 show the mean total cholesterol, triglycerides, HDL-Cholesterol, LDL-Cholesterol and fasting blood sugar levels (group II), the patients in this group have normal blood sugars with hyperlipidemia syndrome. The mean levels of serum Na\textsuperscript{+} (118.2±3.13 meq/L) were significantly lower in the sera of hyperlipidemia syndrome in comparison to the control subjects (140.09±2.33 meq/L), the mean level of K\textsuperscript{+}(4.41±0.74 meq/L) show non significance change in this group, while serum Cl (89.26±0.54 meq/L) decreased significantly in the sera of hyperlipidemic patients in comparison to that of control subjects (95.08±0.08 meq/L).

Table 4 show the mean total cholesterol, triglycerides, HDL-Cholesterol, LDL-Cholesterol and fasting blood sugar levels (group III), the patients in this group have...
The potassium levels in IDDM and in NIDDM patients were reduced during periods of poor control of diabetes mellitus and increases when the blood glucose level are normal,\textsuperscript{21} the levels of potassium were reduced because of diuretics\textsuperscript{24} as well as due to diabetic keto acidosis (increased loss in urine). This explains the existence of hydrovolemia in these patients. We verified the presence of hyperkalemia and because of this metabolic acidosis can occur frequently in these patients.

Diabetic usually take medication that influences the electrolyte balance. Thus, loop diuretics and thiazides may cause hyponatremia, hypokalemia and deficiency of magnesium, disturbances in calcium handling (increased renal loss with loop diuretics and re absorption by thiazides) and hyperglycemia. The treated NIDDM patients may continue to have mild hypertriglycridemia, increased intermediate-density lipoprotein levels, small dense low-density lipoprotein LDL with increased apolipoprotein B, and decreased HDL cholesterol levels. The central and abdominal distribution of adipose tissue in IDDM (insulin dependent diabetic mellitus) is associated with insulin resistance, hypertension, and the above lipoprotein abnormalities. Improvement in glucose control, in the absence of weight gain, leads to lower triglyceride and higher HDL cholesterol levels. In addition, the diabetic patient is prone to develop other defects that, in themselves, leading to hyperlipidemia, such as proteinuria, hypothyroidism and hypertension.\textsuperscript{25}

**REFERENCES**


**Table 3: Mean±SD of serum lipids profile and electrolyte of non diabetics mellitus patients (group II)**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Case</th>
<th>Control</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bl.glucose mg/dl</td>
<td>97.54±13.29</td>
<td>95.21±8.91</td>
<td>NS</td>
</tr>
<tr>
<td>Triglyceride mg/dl</td>
<td>289.83±12.22</td>
<td>169.32±10.42</td>
<td>HS</td>
</tr>
<tr>
<td>Cholesterol mg/dl</td>
<td>395.52±16.65</td>
<td>160.4±14.64</td>
<td>HS</td>
</tr>
<tr>
<td>HDL-Chole mg/dl</td>
<td>61.12±0.54</td>
<td>38.22±7.10</td>
<td>HS</td>
</tr>
<tr>
<td>LDL-Chole mg/dl</td>
<td>195.55±7.46</td>
<td>112.65±4.65</td>
<td>HS</td>
</tr>
<tr>
<td>Na+ Meq/L</td>
<td>118.2±3.13</td>
<td>140.09±2.33</td>
<td>HS</td>
</tr>
<tr>
<td>K+ Meq/L</td>
<td>4.41±0.74</td>
<td>4.66±0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Cl- Meq/L</td>
<td>89.26±0.54</td>
<td>95.08±0.08</td>
<td>S</td>
</tr>
</tbody>
</table>

NS: no significance, HS: high significance, S: significance

**Table 4: Mean±SD of serum lipids profile and electrolyte of non dependent-diabetes mellitus NIDDM patients (group III) (NIDDM+Hyperlipidemia)**

<table>
<thead>
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<th>Parameters</th>
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</thead>
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<td>402.91±15.19</td>
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<td>Triglyceride mg/dl</td>
<td>327.03±7.11</td>
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<td>Cholesterol mg/dl</td>
<td>295.52±10.05</td>
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<td>HDL-Chole mg/dl</td>
<td>41.15±0.50</td>
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</tr>
<tr>
<td>LDL-Chole mg/dl</td>
<td>213.0±4.01</td>
<td>112.65±4.65</td>
<td>HS</td>
</tr>
<tr>
<td>Na+ Meq/L</td>
<td>127.52±3.21</td>
<td>140.09±2.33</td>
<td>HS</td>
</tr>
<tr>
<td>K+ Meq/L</td>
<td>4.21±0.34</td>
<td>4.66±0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Cl- Meq/L</td>
<td>85.26±4.14</td>
<td>95.08±0.08</td>
<td>HS</td>
</tr>
</tbody>
</table>

NS: no significance, HS: high significance, S: significance

NIDDM syndrome with hyperlipidemia. The mean levels of serum Na\textsuperscript{+} (127.52±3.21 meq/L) were significantly lower in comparison to the control subjects (140.09±2.33 meq/L), the mean level of K\textsuperscript{+} (4.21±0.34 meq/L) show non significant change in this group, while serum Cl\textsuperscript{−} (85.26±4.14 meq/L) decreased significantly in comparison to that of control subjects (95.08±0.08 meq/L).

**DISCUSSION**

The incidence of diabetes mellitus in the community is 5-10\%\textsuperscript{17,18} Diabetes mellitus damages every organ in the body, mainly the kidneys, leading to end-stage renal diseases.\textsuperscript{19,21} The patients suffering from diabetes mellitus have disturbances in the electrolytes and in the acid-base balance. These distribution are caused by the diabetes (glucose balance), renal diseases and medications (diuretics and calcium channel blockers).\textsuperscript{22}

Authors Contribution:
NT: Conceived, design of the study, collected and analyzed the data, manuscript preparation and review of the final edition of manuscript.

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