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# Evaluation of Some Biochemical Parameters in Iraqi Diabetic Patients

Khalidah Salih Merzah
Department of Medical laboratory Technology, Osol Aldeen University College, Baghdad, 11001, Iraq.
Corresponding Author email: drkhalida@ouc.edu.iq


## Abstract

High blood sugar occurs in diabetes mellitus when either the pancreas does not secrete enough insulin or cells do not respond to the insulin that is produced. This study aims to evaluate some biochemical variables in patients with type 2 diabetes and their relationship to exacerbating the disease condition. Seventy patients with diabetes mellitus and fifty healthy individuals were included as normal controls for this study. Blood analysis was carried out for all of them which include serum glucose, lipid profile, urea, creatinine and liver enzymes (ALT, AST, GGT) levels. This study included the verification of some biochemical variables by measuring their levels in diabetics and healthy people. As the levels of all the variables under study increased in diabetics except for high-density lipoproteins, its level decreased compared to the results obtained for the same variables in healthy people.

***Keywords:*** *Diabetes mellitus, Biochemical Parameters.*

## 1. Introduction

Hyperglycemia, caused by inadequate insulin synthesis, is a hallmark of diabetes mellitus, a metabolic disorder. Common microvascular disorders affecting the skin, kidneys, and nerves are linked to hyperglycemia in people with chronic diabetes, as is an increased risk of cardiovascular disease (CVD). Retinopathy, a complication of diabetes, is one of the microvascular complications highlighted in the diagnostic criteria for the illness [1].

Type 2 diabetes, which does not dependent on insulin, accounts for 90–95 percent of all cases of the disease. It usually affects people who have insulin resistance due to a defect in insulin receptors on cells or a lack of insulin secretion in proportions that do not make them need insulin therapy as cells Beta in the pancreas still secretes insulin [2]. Patients with type 2 diabetes often have insulin-resistant fat cells, liver cells, and muscle cells.

Diabetic type-II patients are tending to be overweight, with a Body Mass Index that is greater than 20. Because of insulin resistance, the pancreas has to work harder to create enough insulin for obese people. However, the insulin produced won't be enough to keep blood sugar levels steady [3].

Type 2 diabetes can be controlled by controlling the type and amount of food and exercise and thus controlling weight. As the disease progresses, diabetes medications are needed. To ensure control of the perfect diet and exercise, in addition to adhering to taking medications, it is advised to do the HbA1c test several times during the year that this test gives the average level of glucose during the life of red blood cells [3, 4, 5].

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When the blood sugar level rises to the point where it appears in the urine, it will cause excessive urination, thirst, hunger, and disruption of protein and fat metabolism [6]. Both emerging and developed nations face a serious challenge with the prevalence of Type 2 Diabetes. It's one of the major killers, coming up at number seven on the list of deadly diseases.

The diversity of diet systems and the lifestyles that differ in different countries and cultures have a great role in increasing the number of diabetics during the year and it is expected to reach about 300 million in the year of 2025 [7].

The complications caused by insulin deficiency are problems with metabolic disorders, which causes blood glucose to increase in addition to the high level of creatinine, cholesterol and transaminase enzymes accompanied by a decrease in the body proteins [8].

It was observed that secondary complications in diabetics are changes in the formation of the vascular basal membrane in addition to the accumulation of glucose and the products of the reactions that lead to an increase in the use of glucose in the insulin non-dependent tissues [9]. Increases in serum glycated proteins [10, 11, 12] and other changes in atherogenic risk factors have been validated by the vast majority of investigations. This study included an evaluation of some biochemical factors in type 2 diabetes patients and their comparison with the results obtained for healthy people.

## 2. Materials and Methods

In this section, the research materials and methods have been presented and it is summarized as in the following points:

- **Control Groups:** The study included 50 healthy adults who did not suffer from any disease (females and males) as a control group.
- **Patients:** The group of patients includes 70 patients with type 2 diabetes, their ages range from 20 to 70 years, and blood samples were taken from them after a 12-hour fasting period. Full history and general physical examination were obtained from the patient's file.
- **Sampling Procedures:** The patient's venous blood was drawn, deposited in EDTA tubes, and centrifuged at 2000 rpm for 10 minutes to separate the components. Prior to analysis, plasma samples were frozen at -20 degrees Celsius. The collected data was subjected to statistical analysis, with the findings being reported as a mean and standard deviation. The data from the two groups were compared using a T-test. Statistically significant level was kept at less than 0.05 (p-value).

The two group's distribution, depending on age and health status

| Groups | No. | Age range (Years) |
|---|---|---|
| Normal | **50** | **22-60** |
| Type 2 DM Patients | **70** | **20-70** |

- **Blood glucose measurement:** The blood glucose concentration was measured after a fasting period (FBG) using the commercially available enzymatic chromatography method [13].
- **Determination of Serum Total Cholesterol and Serum Triacylglycerol by** using an enzymatic method [14].
- **Measurement of Serum High-Density Lipoprotein- Cholesterol (S.HDL-C):** Using the Burstein et al. process, 1980[15], the serum HDL-C is determined by the HDL-C package.

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- **Measurement of Serum Low-Density Lipoprotein- Cholesterol (S.LDL-C):** The LDL amount is most frequently extracted from the formula of friedwalds as follows [16]. LDL-cholesterol = Total cholesterol – [HDL- cholesterol + TG/5]
- **Measurement of Serum Urea:** Urease enzyme hydrolysis urea by-product ammonium by (urease –modified Berthelot) reaction [17], by this method urea concentration, was measured.

### 2.1 Determination of Serum Creatinine

Serum creatinine was determined using the Jaffes reaction, which involves the development of an orange color with picric acid in alkaline conditions. This color reaction occurs quantitatively and is measured at 520 nm after an incubation time of 15 minutes at room temperature [17].

### 2.2 Determination of Serum ALT, AST & GGT

Serum levels of Alanine aminotransferase (ALT) and Aspartate aminotransferase (AST) enzymes were assessed using the Reitman and Frankel method [18]. For Gamma Glutamyl Transferase (GGT), the SZASZ method was employed [19].

## 3. Results and discussion

The study included seventy diabetics and fifty healthy people as a control group.The results obtained for diabetes patients were divided into two groups, females and males, due to the presence of some differences in biochemical parameters, and then compared with the control group.

Tables 1 & 2 shows the levels of glucose of fasting control and diabetes patients. The results showed a high level of blood glucose in male fasting patients (175.4 ± 1.46) compared to the control group under the same conditions (88 ± 4.0) and this also applies to female results, where a high level of blood glucose was recorded (170.4 ± 1.65) compared to the control group of females who recorded (84 ± 4.2).

**Table 1:** Average fasting blood glucose level (FBG) (mg/dl) in the diabetic and control group.

| Gender / FBG | Normal | Diabetics | p-value* |
|---|---|---|---|
| Mail | 88±4.0 | 175.4 ±1.46 | <0.001 |
| Femaile | 84±4.2 | 170.4 ±1.65 | <0.001 |

* Significantly different at P<0.01 from the corresponding control value

The routine method for screening for diabetes is measuring blood glucose concentration; however, there are differences in blood glucose concentrations if measured randomly. In the work of Andrade-Cetto et al. [20], this is in addition to glucose intolerance by diabetics. This study showed an increase in the level of blood glucose in diabetics compared to the healthy group as agreed in all studies.

Tables 2 and 3 shows the concentrations of lipid profile in diabetics and in healthy subjects, where total cholesterol showed an increase of 183.6 ± 35.9 mg/dl in addition to low-density lipoprotein LDL-C (155 ± 4.75 mg/dl) and triglycerides (39 ± 11.42 mg/dl) On the other hand, the results showed a decrease in the level of high-density lipoprotein HDL-C (21 ± 3.0 mg/dl). The findings were compared to those from the healthy control group. Their levels were as follows cholesterol (**182.8 ± 34.1**), LDL-C (**155** ±4.75 mg/dl), triglycerides (**39** ±11.42 mg/dl) and HDL-C (24 ± 3.77).

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**Table 2:** The mean of serum Total Cholesterol, HDL C, Triglyceride and LDL- Cholesterol levels (mg/dl) in diabetic and healthy groups (Males).

| Biochemical Parameters(mg/dl) | Normal | Diabetics | p-value* |
|---|---|---|---|
| **Total cholesterol** | 182.8 ± 34.1 | 183.6 ± 35.9 | 0.658 |
| **HDL cholesterol** | 24 ±3.77 | 21 ±3.0 | <0.001 |
| **Triglycerides** | 30 ±13.42 | 39 ±11.42 | 0.006 |
| **LDL cholesterol** | 151 ±3.77 | 155 ±4.75 | 0.001 |

**Table 3:** The mean of serum Total Cholesterol, HDL C, Triglyceride and LDL- Cholesterol levels (mg/dl) in diabetic and healthy groups (Females).

| Biochemical Parameters(mg/dl) | Normal | Diabetics | p-value* |
|---|---|---|---|
| **Total cholesterol** | 192.9 ± 36.7 | 200.2 ± 36.2 | 0.001 |
| **HDL cholesterol** | 48.6 ± 11.7 | 45.8 ± 10.9 | <0.001 |
| **Triglycerides** | 123 ± 86 | 161 ± (113–243.5) | <0.001 |
| **LDL cholesterol** | 119.7±3.27 | 122.2±4.65 | 0.001 |

*Significantly different from corresponding control value at P<0.01

Metabolism pathways are generally controlled by many enzymes. In the case of diabetes, these pathways will be affected, including fat metabolism, through changes in the activity of these enzymes [21]. The increased incidence of death for patients with diabetes type 2 coincides with the myocardial infarction; the hyperlipidemia must be treated severely.

The presence of high levels of low-density lipoprotein cholesterol (LDL-C) is a major predictor of atherosclerosis and CHD. The severity of the disease and the likelihood of mortality can be mitigated by reducing it. This study agrees with the findings of Sharma (1970) and Jain (1980), which show that treatment with drugs that regulate lipid levels to normal levels causes a rise in LDL-C and a fall in HDL-C, as compared to the group that was treated with no drugs [22, 23].

The results in Table 4 showed urea and creatinine concentrations in normal and diabetic patients. In normal subjects, the concentration of urea (30.1±3.20) and creatinine (0.95±0.06) obviously, it is higher in diabetics (58.9±1.195), (1.96±0.109) than normal subjects.

**Table 4:** The mean level of serum Urea & Creatinine (mg/dl) in DM groups and normal groups

| Biochemical Parameters(mg/dl) | Normal | Diabetics | p-value* |
|---|---|---|---|
| **Urea** | 30.1±3.200 | 58.9±1.195 | 0.001 |
| **Creatinine** | 0.96±0.067 | 1.96±0.109 | <0.001 |

Kidney function measured by measuring the levels of creatinine and urea in the blood, it is necessary to monitor their levels in diabetics because the kidneys can be exposed to failure due to diabetes and as shown, the level of creatinine and urea is higher in patients with diabetes compared to healthy people, so these results show the strong relationship between the blood sugar and urea levels.

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High blood urea level is observed when the blood sugar level increases. High blood sugar level is the leading cause of kidney failure [24]. From these data, it is possible to confirm the relationship between high blood sugar level and high urea level, as shown in the results.

A study was conducted on diabetic mice by Anjaneyulu *et al.,* 2004 and it was found that the high level of urea and creatinine in mice serum caused kidney damage gradually [25]. Urea and creatinine levels decreased after treating diabetic patients.

Tables 5&6 show the concentration of ALT, AST, and GGT in normal groups and diabetic patients. In normal subjects the serum AST (**25 ±3.43**), ALT (**21 ±3.54**) and GGT (**30 ±4.02**) appeared to be higher in diabetic patients (**26 ±4.45**), (**24 ±4.43**) and (**39 ±4.50**) than normal subjects.

**Table 5:** The mean of serum ALT, AST & GGT levels (mg/dl) in Diabetes groups and normal groups (males)

| Biochemical Parameters(units/l) | Normal | Diabetics | p-value* |
|---|---|---|---|
| **AST** | 25 ±3.43 | 26 ±4.45 | 0.089 |
| **ALT** | 21 ±3.54 | 24 ±4.43 | <0.001 |
| **GGT** | 30 ±4.02 | 39 ±4.50 | 0.006 |

*Significantly different from corresponding control value at P<0.01

**Table 6:** The mean of serum ALT, AST & GGT levels (mg/dl) in Diabetes groups and normal groups (females)

| Biochemical Parameters(units/l) | Normal | Diabetics | p-value* |
|---|---|---|---|
| **AST** | 22 ±3.46 | 23± 3.64 | 0.076 |
| **ALT** | 16 ±3.64 | 20 ± 3.34 | 0.001 |
| **GGT** | 15 ±4.32 | 20 ± 4.45 | 0.001 |

*Significantly different from corresponding control value at P<0.01

This study showed the relationship between liver enzymes present in blood serum and type 2 diabetes. As the results showed that serum levels of ALT and GGT positive association with increased risk of type 2 diabetes and for both sexes in comparison to healthy subjects.

One of the major functions of the liver is to regulate metabolic processes and maintain glucose concentrations in all cases. When insulin is lost, its effect on the liver will lead to the activation of glycogenolysis to produce glucose from the liver and an increase in the process of lipolysis that leads to increased production of fatty acids.

In situations classified as insulin resistance, lipolysis and abnormalities of triglyceride accumulation in insulin-sensitive organs like the liver can be observed prior to fasting hyperglycemia The results regard to liver enzymes are agree with the results of previous studies [26, 27], where they have proven high levels of AST, ALT, and GGT in diabetics compared to healthy people. The reason is that the liver has been affected by an increase in fatty acid production that may lead to toxic effects on the liver.

Mechanisms may include disturbance of the cell membrane at high concentration, imbalance of mitochondria, the formation of toxins, And the activation and inhibition of major steps in metabolism

regulation. The oxidative stress of reactive lipid peroxide, peroxide beta-oxidation, and recombinant inflammatory cells are other potential reasons for the increase in transaminases in insulin resistance states.

Elevated pro-inflammatory cytokines, such as -tumor necrosis factor (TNF-), may contribute to hepatocyte injury in people with insulin resistance. An increase in ALT is thought to reflect disruption of the insulin signal rather than harm to isolated hepatocytes [28], as ALT is a gluconeogenic enzyme whose gene transcription is inhibited by insulin. Gaining weight is another factor that might raise ALT levels [29].

## 4. Conclusion

The most common metabolic disease is diabetes mellitus, and one of the five leading causes of death. It is characterized by a lack of insulin secretions in the pancreas from the beta cells, or insufficiency of insulin receptors on the cells, chronic hyperglycemia and metabolic disorders of carbohydrates, fats and proteins. In our research, different biochemical parameters for normal and diabetic patients have been examined. In addition to, TG, LDL, urea, creatinine, total cholesterol, AST, ALT, and GGT, a high level of fasting glucose was observed and the amount of high-density lipoprotein in diabetic patients decreased relative to the normal control group.

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