Original Article

Study of a Selection of Biochemical Parameters for Type II Diabetes Mellitus Patients in Iraqi Baghdad Hospitals

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Abstract

Background: Diabetes mellitus is characterized by hyperglycemia caused by full or partial insulin insufficiency. This study examines type 2 diabetics' lipid profile, malondialdehyde levels, antioxidant levels, and electrolyte levels.

Methods: The levels of glucose, total cholesterol, triglycerides, high density lipoprotein, very low density lipoprotein, low density lipoprotein, malondialdehyde (MDA), ceruloplasmin, uric acid, sodium, potassium, and chloride in the serum of 75 patients with type II diabetes mellitus and 25 healthy volunteers were measured.

Blood glucose levels, total cholesterol levels, triglyceride levels, very low-density lipoprotein levels, low-density lipoprotein levels, malondialdehyde levels, ceruloplasmin levels, and sodium, potassium, and chloride levels are all higher in people with type II diabetes compared to healthy controls. Type II diabetics exhibited lower HDL and uric acid levels than healthy controls. In this study, low HDL and high triglyceride values indicated typical diabetic dyslipidaemia. MDA levels may indicate oxidative stress. Increased oxidative stress may cause humans to consume more ceruloplasmin. Diabetes type II reduces blood uric acid levels. This study suggests that disturbed glucose homeostasis and electrolyte imbalance are related to higher sodium and potassium chloride levels.

Keywords: Lipid Profile, Antioxidants, Diabetes Mellitus, Serum Electrolytes, Malondialdehyde.

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INTRODUCTION

Diabetes mellitus (DM) is a complex disorder that can be caused by a number of different metabolic issues and is characterized by a variety of complexities. Hyperglycemia is the manifestation of this condition, which occurs when there is either an absolute or relative shortage of insulin [1]. The digestion of carbohydrates, proteins, and lipids, as well as water and electrolytes, are all affected when insulin is either completely or somewhat absent [2]. Insulin regulates several different destinations along the path of lipid breakdown in mammalian cells. In the liver, the fat tissue, and the digestive system, it stimulates the union of unsaturated fats. It has also been suggested that insulin is responsible for the increase in cholesterol combination. In addition to this, increased lipoprotein lipase activity has been seen in white fat tissue [3]. Patients with diabetes who seek treatment at diabetic centers report experiencing significant gastrointestinal side effects in a high percentage [4]. From the present estimate of 415 million people, it is anticipated that by the year 2040, That would have resulted in 642 million extra cases of diabetes if it had occurred in the past. The prevalence of Type 2 Diabetes Mellitus (T2DM) is on the rise worldwide, with the majority (75%) of the world's diabetic population residing in agricultural nations [5]. Rising prevalence of diabetes mellitus (DM) means it will likely overtake smoking and obesity as a primary cause of mortality and disability in the near future. That's because the disease's incidence is on the rise. It is estimated that between 10 and 73% of people with type 2 diabetes also suffer from dyslipidaemia [6, 7], making it a major risk factor for widespread vascular problems. It is also a known risk factor for an elevated mortality rate. Increased levels of low-density lipoprotein cholesterol (LDL-C), decreased levels of high-density lipoprotein cholesterol (HDL-C), or increased amounts of fatty material are the most common types of dyslipidemia seen in people with diabetes (TG). In addition, the United Kingdom Prospective Diabetes Study found that both low HDL-C and high LDL-C levels were predictors of cardiovascular risk in people with diabetes. [Footnote required] It's important to cite this phrase. All applicable local, national, and international regulations necessitate the use of coercive methods when feeding this population lipids [7].

Hyperglycemia, a metabolic disease that is associated with the growing free-extremist movement, is a hallmark of the type 2 diabetes that causes mellitus. Mellitus diabetes, or Type 1 diabetes, is a common name for this condition. Some examples of chemical events that might lead to the formation of free radicals include glucose auto-oxidation, protein glycation, the production of subsequent glycated end products, and the initiation of the polyol pathway. Ultimately, these events cause oxidative pressure to build up in many different organs [8]. A redox imbalance is created as a result of an inadequate supply of acceptable compensating components derived from endogenous cell reinforcement frameworks, which then leads to the beginning of stress-sensitive intracellular flagging pathways [9]. Therefore, there is a possibility that oxidative stress is involved in the development of diabetes.

[10] Ceruloplasmin is an enzyme with the molecular formula E.C. 1.16.3.1. It is a protein that goes through a vigorous stage of synthesis in the liver and is a separate entity from blue multicopper oxidase. However, ceruloplasmin does have a role in copper transport, coagulation, angiogenesis, protection against oxidative pressure, and iron homeostasis [10,11]. Although the physiological components of ceruloplasmin are debatable, ceruloplasmin does play a role in these processes. In metabolic disease and type 2 diabetes, there has been material that contradicts itself about serum ceruloplasmin. In metabolic disease and DM, a few studies found significantly elevated levels of ceruloplasmin in the blood [12,13]. The available information mostly clarifies the connection between elevated levels of ceruloplasmin and cardiovascular diseases [14,15]. There have only been a few studies that have showed a reduction in plasma ceruloplasmin in type 2 diabetes [16.17].

However, uric acid may also have a restorative role as a cell reinforcement [18]. Uric acid can act as a favorable to oxidant, and it may thus be a sign of oxidative pressure. In addition, uric acid may be a marker of oxidative pressure. Urate, which is a dissolvable form of uric corrosive, has the ability to search for superoxide and hydroxyl revolutionaries, in addition to having the ability to chelate changing metals [19]. In addition, hyperuricaemia has been included to the collection of metabolic abnormalities that are associated with insulin resistance as well as hyperinsulinaemia in the metabolic state [20]. While some studies found that pre-diabetes and diabetes were associated with an increase in uric acid levels, other studies found that rising blood glucose levels were associated with a decreasing pattern of serum uric acid levels [21]. While some studies found that uric acid levels increased in pre-diabetes and diabetes, others found that uric acid levels decreased with rising blood glucose levels.

In individuals with diabetes, an uneven concentration of electrolytes is typical. This might be the result of an altered appropriation of electrolytes, and it is related with hyperglycemia-actuated osmotic liquid movements or absolute body deficits generated by osmotic diuresis [22]. However, the glucose entrance into cells is reduced when insulin is present, whereas the potassium intake into cells remains unaffected. Patients who have type 2 diabetes mellitus often have hyperkalemia because of an expansion in the composition of their plasma. This enlargement is due to a shift of potassium from the intercellular space to the extracellular region, which has caused this expansion. In addition to this, DM is able to induce dysnatremias by the utilization of a couple of hidden gadgets [23, 24]. In light of this, the objectives of this study were to evaluate the blood electrolytes (sodium, potassium, and chloride), take note of the lipid profile, malondialdehyde, ceruloplasmin, and uric corrosive levels in people who had type 2 diabetes, and make a note of the uric corrosive level.

Materials and Methods

This investigation will take place in the Laboratories of Biochemistry on the Faculty of Sciences at the University of Thi-Qar, Iraq. This institution is focused on diabetes and the endocrine glands. 125 participants took part in the study; 50 healthy persons served as controls, while 75 people diagnosed with Type 2 Diabetes Mellitus did the research (T2DM). Every participant had a sample of their blood taken that was 5 milliliters in volume. After allowing the samples in the single-use tubes, they were centrifuged for ten minutes at a speed of 3000 revolutions per minute to allow the blood to coagulate at room temperature. After the serum samples were separated, they were kept at a temperature of -20 degrees Celsius until the time of the test.

The glucose concentration in the serum was measured using this sample. It was approximated by the method that Barham and colleagues used. [25], the pre-owned reagents were given by (Randox, UK), and the technique for Allan and Dawson was used to estimate cholesterol (TC). [26], the pre-owned reagents were given by (Biolabo, France), and the procedure for Tietz et al. was used to assess the amount of fatty oils. [27], the pre-owned reagents were given by (Biolabo, France). The procedure for Lopes-Virella was used to assess high-density lipoprotein (HDL). [28], the pre-owned reagents were given by (Biolabo, France), and the approach for Friedwald et al. was used to estimate extremely low-thickness lipoprotein (VLDL) and low-thickness lipoprotein (LDL). [29], an estimation of malondialdehyde was carried out using the method developed by Fong et al. [32], the pre-owned reagents was donated by (Biolabo, France),, Sodium and Potassium were calculated by Retiman and Frankel. [30], ceruloplasmin was evaluated using the approach for Menden et al. [31], uric corrosive was estimated using the technique for Fossati et al. [32] [33], the pre-owned reagents were given by (Randox, UK), and chloride was calculated by Florence and Farrar [34], the pre-owned reagents was provided by [33]; [35], the pre-owned reagents was provided by [33]; [36], the pre-owned reagents was provided by [(Biolabo, France).

RESULTS

The results showed that the concentrations of blood glucose, total cholesterol (TC), fatty substances (TG), exceptionally low thickness lipoprotein (VLDL), and low thickness lipoprotein (LDL) all increased significantly (P 0.05) in the DM group as compared to the control group. In a similar manner, the concentration of serum high-density lipoprotein (HDL) was shown to be significantly lower (P 0.05) in the diabetes group as compared to the control group. (Table 1).

In addition, the results of the current investigation indicated a significant rise (P 0.05) in the concentrations of serum malondialdehyde (MDA) and ceruloplasmin (Cp) in the diabetics with metabolic syndrome (DM) group in comparison with the control group. In addition to this, there was a statistically significant decrease (P 0.05) in the concentration of serum uric acid in the DM group in comparison to the group that served as the control (Table 2). Also, the studies that are still being done have found that the amounts of serum sodium (Na+), potassium (K+), and chloride (Cl-) were statistically significantly higher (P 0.05) in the DM group than in the control group (Table 3).

DISCUSSION

The phones rely heavily on glucose as their major energy source. Nonetheless, glucose cannot enter the cell in the absence of insulin. This must be done immediately. Glucose can enter cells if the pancreas is healthy and producing enough insulin. The dysfunctional pancreas either fails to pump enough insulin into the circulation or the body's cells become resistant to the insulin that is secreted. In turn, this leads to a rise in blood glucose levels, a condition known as diabetes mellitus [35].

A greater concentration of glucose was seen in the blood of those with type 2 diabetes compared to the controls. This illness, which often appears after the age of 40, may have several reasons, such as a lack of beta cells, a limited ability to produce insulin, and a growing resistance to the effects of insulin [36,37]. The absolute cholesterol levels of diabetes patients were found to be higher than those of the controls by a statistically significant margin. Cholesterol catabolism may be less active than previously thought [38], or other variables may be at play. Conversely, it might be attributed to an increase in plasma grouping of VLDL-C and LDL [1], which could be the consequence of hepatic synthesis of VLDL or a reduction in the evacuation of VLDL-C and LDL from the circulation. These two scenarios are equally plausible. Patients with diabetes were shown to have significantly greater amounts of fatty oils compared to healthy controls. The buildup of unsaturated lipids in adipose tissue and subsequent hyperglycemia may be the cause of this [39]. The liver is in charge of storing any extra unsaturated fats, which are subsequently completely transformed into fatty oil [39]. Unsaturated fats in adipose tissue are activated to generate energy. VLDL cholesterol levels are considerably higher in diabetic individuals compared to controls. Hyperinsulinemia, which would produce a rise in triglycerides and LDL and VLDL cholesterol, is a possible explanation for this phenomenon. New evidence suggests that insulin and the development chemical have a role in promoting VLDL cholesterol synthesis. They achieve this by increasing lipolysis in the liver's fatty tissues and fatty oils and by encouraging the development of Apo-E and Apo-B 48 [40].

The plasma LDL cholesterol levels of persons with type 2 diabetes were significantly greater than those of the healthy controls. Chronic insulin shortage, such as that found in type 2 diabetes, may be linked to a lower level of LDL receptors and, thus, a rise in plasma levels of LDL cholesterol [41]. This is because insulin induces an increase in the number of LDL receptors. Potentially at play here is the fact that insulin upregulates the expression of genes encoding for LDL receptors.

Table 1: Diabetic patients' serum glucose levels and lipid profiles, compared to those of their controls

Diabetic patients' versus healthy controls' serum glucose and lipid profiles. Groups	**No.**	**Glucose (mg/dL)**	**TC (mg/dL)**	**TG (mg/dL)**	**HDL (mg/dL)**	**VLDL (mg/dL)**	**LDL (mg/dL)**
Control,	50	99.33 ± 5.24 b	176.39± 7.00 b	124.22± 5.72 b	47.00 ± 3.52 a	24.84 ± 1.15 b	104.64± 8.60 b
DM	75	296.66± 38.16 a	233.61± 8.46 a	160.89± 6.96 a	34.22 ± 3.48 b	32.18 ± 1.39 a	166.79± 8.48 a
LSD		15.51	4.49	3.59	2.00	1.18	4.93

The numbers with non-identical superscripts (a, b, or c...etc.) were determined to be substantially different from one another (P 0.05). Each value indicates the Mean and Standard Deviation.

Table 2: MDA and antioxidant levels in the serum of diabetic patients and their respective controls

Serum levels of MDA and antioxidants in diabetic patients and the control groups for these patients	**No.**	**MDA (µmol/L)**	**Cp (mg/dL)**	**Uric acid (g/L)**
control,	50	3.83±0.24 a	3.03±0.30 b	7.24±0.48 a
DM	75	2.06±0.27 b	4.12±0.32 a	4.82±0.28 b
LSD		0.15	0.18	0.32

Each value represents Mean±SD values with non-identical superscript (a, b or c …etc.) were considered significantly different ($P \leq 0.05$).

Table 3: Serum electrolytes of diabetic patients as well as their healthy counterparts

Serum electrolytes of DM patients and their controls Groups	**No.**	**Na+(mmol/L)**	**K+(mmol/L)**	**Cl-(mmol/L)**
control	50	138.72±4.40 b	4.15±0.72 b	98.77±2.35 b
DM	75	164.14±10.77 a	5.41±0.88 a	117.22±5.80 a
LSD		4.79	0.47	2.57

Each value indicates the results for mean and standard deviation that did not have the same superscript (a, b, or c...etc.) were determined to be substantially different (P 0.05).

Patients with type 2 diabetes had considerably decreased plasma HDL cholesterol levels compared to healthy controls. This finding could be a result of the HDL-intervened uptake of excess cholesterol from other hepatic tissues reaching a breaking point due to the loss of lecithin: cholesterol acytransferase (LCAT) in the urine [42]. When the number of HDL-C receptors in the liver drops dramatically, the problem becomes much more severe. Low serum HDL-C levels are associated with primary and secondary modifications that promote arterial stiffening.

An increased MDA blood level might be a valuable diagnostic indicator of oxidative stress. Increased lipid peroxidation is associated with a wider range of free-revolutionary activity in those with type 2 diabetes. When this last tactic is combined with insulin blockade, it has the potential to activate stress-sensitive pathways, which may play a crucial part in the complexity of diabetes [43].

More young red platelets and the compensatory components following increased oxidative pressure [4] may account for the increased clustering of serum ceruloplasmin in diabetes patients compared to normal participants. This is supported by the observation that diabetes individuals tend to have a higher percentage of younger red blood cells. In addition, a rise in serum copper level [45] as a disobedient capacity may be at play, as may an increase in hepatic catalysis of Cp union in opposition to iron over-burden condition [44]. In contrast, purines are metabolized to uric acid (UA) in the human body. Uric acid is formed when hypoxanthine is transformed to xanthine and xanthine is converted xanthine oxido-reductase is the enzyme responsible for converting xanthine to uric acid. This latter process makes use of molecular oxygen as an electron acceptor in order to generate superoxide anion and several other reactive oxygen species (ROS) [46,47,48]. The antioxidant capacity of plasma is greatly enhanced by UA because of its role as a physiological scavenger of free radicals [49]. Therefore, UA has a dual role, since it both promotes oxidation and functions as an antioxidant [50,51]. Because type 2 diabetes promotes the formation of free radicals and makes the body's natural antioxidant defense less efficient [52, 53, 54], oxidative stress is associated with the disease. Having low amounts of uric acid has been linked to type 2 diabetes in the past [55.56]. Scientists [57] discovered a positive connection between glucosuria and uricosuria. Increases in hyperglycemia were also associated with increased uric acid excretion and decreased uric acid levels in the blood plasma [57]. Hypouricaemia and uric acid tubular transport are both extensively studied in [58]. Improper tubular urate management has been linked to increased glomerular hyperfiltration in type 2 diabetes, leading to greater urate clearance [59]. Researchers who carried out the investigation came to this conclusion. An imbalance in serum electrolytes, especially sodium and potassium, can result from excessive urination because of the loss of electrolytes and water. Studies have shown that uncontrolled diabetes can produce osmotic diuresis, which can lead to hypovolemic hyponatraemia. Additionally, the loss of electrolytes through urine contributes to the loss of sodium through the kidneys in the presence of diabetic ketoacidosis [60,61]. The current study found a negative association between glucose levels and sodium levels, with diabetic patients having higher salt levels than the control group. In the setting of hyperglycemia, increased or normal plasma sodium concentrations are indicative of a clinically severe shortage in total body water. [Case in point:] Take into consideration the following: [Example:] This is only one illustration: This is only one illustration: Because the diabetes wasn't being appropriately treated, hypernatraemia developed in a few of these patients. As a consequence of this, persons who have diabetes that is not under control have a diverse range of Na+ concentrations in their blood. This represents a balance between glucosuria-induced osmotic diuresis, which tends to boost [Na+, and hyperglycemia-induced water transport out of the cells,

which tends to reduce [Na+. As a result, hypernatraemia and hyperosmolarity can be identified as risk factors for the onset of diabetes [62]. The results of this study indicated that those with diabetes were more likely to experience mild hyperkalemia than controls.

Previous studies have demonstrated that exogenous insulin can cause a mild type of hyperkalemia. This is because it increases Na+ and K+ ATPase pump activity, which in turn increases potassium input into skeletal muscles and hepatic cells. Hyperkalemia is linked to reduced insulin production as well as poor peripheral glucose utilization, both of which are further consequences of hyperkalemia. Carbohydrate intolerance and hyperglycemia [63] are two of these other repercussions. The finding that diabetic individuals have higher levels of Cl- in their blood has been connected to the development of diabetic ketoacidosis. The latter contributes to a decrease in the pH of the blood, which further disrupts the acid-base equilibrium and leads to an increase in serum chloride levels.

Conclusion

Low HDL, high TG, and high levels of very little dense LDL are the hallmarks of diabetic dyslipidaemia. It is anticipated that the early screening of diabetic individuals for dyslipidaemia and the early intervention of these patients may minimize the risk of future cardiovascular death. There is an increase in the formation of free radicals in patients with type 2 diabetes, which is associated with insulin resistance. Consequently, in order to postpone the development of common auxiliary difficulties, it would be necessary to develop methods to standardize the production of free extremists. In addition, the assessment of serum electrolytes is an important component of monitoring the condition of individuals who have type 2 diabetes.

Validation of Conscience: The Research Ethics Committee of the College of Sciences and University of Thi-Qar in Iraq was the source of this information.

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