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Evaluation of the role of lipid profile and kidney function in men with type 2 diabetes in the city of Kirkuk

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Abstract

The aim of the current study is to Investigate the of lipid profiles and kidney function markers in the serum of patients with type 2 diabetes and to determine the correlation of these levels with various physiological parameters in patients with T2DM. The experiment was conducted from the beginning of September 2024 to the end of January 2025. Blood samples were collected from patients visiting Kirkuk General Hospital and private laboratories in Kirkuk. The study samples included 90 male participants with an average age of (59.46) years. The study samples were distributed into (60) participants with type 2 diabetes (T2DM) distributed into two groups: one group consisting of (30) T2DM patients with high weights (high body mass index, BMI=32 Kg/m2) and a second group consisting of (30) T2DM patients with normal weights (normal body mass index, BMI=24.18), in addition to (30) participants in the control group (BMI=22.95).

The study results showed a significant increase ($p \leq 0.05$) in the concentration of glycated hemoglobin and lipid profile indicators, including total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), kidney function indicators such as urea and creatinine, and body mass index (BMI) in the affected patients compared to the control group. Additionally, there was a significant decrease ($P \leq 0.05$) in the concentration of high-density lipoprotein (HDL) in patients with T2DM compared to the control group. The results also indicated, according to the groups of patients with high and normal weights, a significant increase ($P \leq 0.05$) in the concentration of lipid profile indicators, creatinine, and BMI in the group of patients with T2DM with high weights compared to the group of patients with T2DM with normal weights. In addition, there was a significant decrease ($P \leq 0.05$) in high-density lipoprotein (HDL) concentration among patients with T2DM who had high weights compared to the group of T2DM patients with normal weights. The results also showed no significant differences ($p \leq 0.05$) in the concentration of glycated hemoglobin and urea between the two groups of patients with high weights compared to the group of patients with normal weights.

Keywords: Lipid profile indicators (TC; TG; HDL; LDL; VLDL); Type 2 diabetes Mellitus (T2DM); BMI; Kidney function indicators (Urea; Creatinine)

1 Introduction

Type 2 diabetes mellitus (T2DM) is a common metabolic disorder characterized by chronic high blood sugar levels. The development of this disease generally occurs through the presence of two main factors: one being impaired insulin secretion in pancreatic beta cells and the other being impaired insulin response in insulin-sensitive tissues (1). The global prevalence of type 2 diabetes among adults reached 536.6 million people (10.5%) of the world's population in 2021 (2). Multiple estimates indicate that the number of affected individuals will rise to 642 million people by 2040 (3).

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The risk factors leading to the development of type 2 diabetes include a variety of factors such as high obesity rates, lifestyle and dietary changes, gestational diabetes, genetic and familial factors, environmental influences, and others, all of which contribute to the increased incidence of type 2 diabetes (4). Type 2 diabetes is clinically diagnosed through several tests, including the fasting blood glucose test and the glycated hemoglobin test. If the fasting blood glucose concentration is greater than (7.0) mmol/L and the glycated hemoglobin percentage is greater than 6.5%, this indicates the presence of type 2 diabetes (5). Early diagnosis is important to reduce the risk of infection and stop the progression of the disease and its complications. This requires finding numerous and reliable physiological indicators to detect the risk of developing type 2 diabetes (T2DM). Recently, various physiological indicators have been used worldwide to predict, diagnose, and even treat T2DM, including lipid profile indicators such as total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and kidney function indicators such as urea and creatinine, which are discussed in this study. The current study aimed to test various indicators as diagnostic markers to predict the development of type 2 diabetes, which included determining the concentrations of some physiological indicators such as total cholesterol and triglyceride levels. TC triglycerides TG, high-density lipoproteins (HDL), low-density lipoproteins (LDL), very low-density lipoproteins (VLDL), kidney function indicators such as urea and creatinine, as well as body mass index and hemoglobin A1c.

2 Material and methods

2.1 Study design

This study was conducted from the beginning of September 2024 to the end of January 2025, during which blood samples were collected from patients visiting Kirkuk General Hospital and private laboratories in Kirkuk. The study samples included 90 male participants with an average age of 59.46 years. The study samples were divided into 60 individuals with type 2 diabetes (T2DM), further divided into two groups: one group consisting of 30 T2DM patients with high body weight (BMI=32 Kg/m^2) and a second group consisting of 30 T2DM patients with normal body weight (BMI=24.18 Kg/m^2), in addition to 30 participants in the control group with the same weight as the second group.

2.2 Blood samples

Blood samples were drawn from the brachial vein using a 5 ml , in the early morning hours after a fasting period of no less than 12 hours. The samples were then placed in Gel tubes and left at room temperature for 30 minutes. Subsequently, the samples were placed in a centrifuge at a speed of 3000 rpm for 15 minutes to obtain the serum, which was stored at -20 °C until the required tests were conducted.

2.3 Physiological tests

A series of physiological tests were conducted on the study groups. These tests included the estimation of total cholesterol (TC), triglycerides (TG), and high-density lipoprotein (HDL-C) concentrations in the blood serum using ready-made analysis kits from the French company Biolabo, relying on the enzymatic methods specific to each kit (6) (7). Additionally, the concentrations of kidney function indicators, such as urea and creatinine, in the blood serum were determined using ready-made test kits from the French company Biolabo according to the colorimetric method (8).

2.4 Statistical analysis

The statistical analysis of the results was conducted using the SPSS program, where the T-test was used to compare the mean values between patients and healthy individuals at a significance level of $p<0.05$, and the values of the variables were expressed as mean ± standard deviation (9)

3 Results and discussion

3.1 Total cholesterol concentration (TC) in serum

The results in Table (1) showed a significant increase ($p \leq 0.05$) in the total cholesterol (TC) level in patients with type 2 diabetes, reaching approximately (205.37 ± 3.64) mg/dL, compared to the control group which had approximately (165.77 ± 6.22) mg/dL. These results are consistent with the findings of study (10), which indicated that total cholesterol levels in individuals with T2DM were significantly higher compared to healthy individuals, and this increase was considered a risk factor for developing T2DM. According to the groups of patients, the results in Table (2) showed a significant increase ($p \leq 0.05$) in total cholesterol (TC) concentration levels in patients with T2DM who had high weights, reaching approximately (225.96 ± 5.31) mg/dL, compared to the group of patients with T2DM who had normal weights, which reached approximately (182.36 ± 2.20) mg/dL. The increase in total cholesterol (TC) concentrations is

attributed to dyslipidemia, which is characterized by elevated total cholesterol (TC) levels in the serum. Half of individuals with type 2 diabetes mellitus (T2DM) suffer from dyslipidemia, and they are ten times more likely to be affected by this condition compared to non-diabetics. Males have a higher prevalence of all types of dyslipidemia compared to females, and the concentrations of dyslipidemia types increase with age, making them susceptible to cardiovascular diseases and their complications (11). The increase in cholesterol is explained by the increased production of cholesterol esters in patients with type 2 diabetes, where the level of ketone bodies is higher compared to the control group. Additionally, the free fatty acids (FFA) released due to insulin resistance or low insulin concentration increase in the blood plasma and then convert in the liver to phospholipids and Acetyl COA, causing an increase in cholesterol levels (hypercholesterolemia). Despite the high level of glucose in the blood, the cells are unable to use it as an energy source. Therefore, the body uses other energy sources, consuming stored fat, which leads to an increase in its level in the blood. This increase causes cholesterol deposition in the blood vessels, resulting in atherosclerosis, vascular complications, and heart diseases in T2DM patients (13).

3.2 The concentration of triglycerides (TG) in the serum

The results in Table (1) indicated a significant increase ($P \leq 0.05$) in the level of triglycerides (TG) in patients with type 2 diabetes, reaching approximately (183.36 ± 8.96) mg/dL, compared to the control group which was approximately (110.53 ± 3.88) mg/dL. These results are consistent with the findings of (14), which indicated that increased levels of triglycerides above the normal range are associated with a higher susceptibility to type 2 diabetes. The results showed that for every increase of 1 mmol/L in normal triglyceride levels, there was an 81% increase in the risk of developing T2DM. As for the two groups of T2DM patients, the results in Table (2) showed a significant increase ($P \leq 0.05$) in TG concentration among T2DM patients with high weights, reaching approximately (195.80 ± 6.33) mg/dL, compared to the group of T2DM patients with normal weights, which was approximately (170.63 ± 3.55) mg/dL.

The increase in triglycerides (TG) in patients with type 2 diabetes is primarily associated with insulin resistance and the accompanying metabolic disturbances. When adipose cells lose their response to insulin, lipolysis increases, raising the level of free fatty acids in the blood. These acids are transported to the liver, where they are converted into triglycerides. The liver is also stimulated to produce more triglycerides through lipogenic pathways when large amounts of carbohydrates or glucose are consumed in the absence of an insulin response. Consequently, liver cells convert excess glucose into lipid pathways, exacerbating the accumulation of triglycerides in the body (15). Additionally, the body's reduced ability to break down triglycerides in the blood due to the decreased activity of the enzyme lipoprotein lipase (LPL), which relies on insulin for activation, leads to the accumulation of triglycerides in the plasma. Furthermore, the liver produces larger amounts of triglyceride-rich VLDL particles, which collectively increase the concentration of triglycerides in the blood.

3.3 The concentration of low-density lipoprotein cholesterol (LDL-C) in the serum

The results in Table (1) showed a significant increase ($P \leq 0.05$) in LDL-C levels among patients with type 2 diabetes, reaching approximately (127.86 ± 5.13) mg/dL, compared to the control group which had approximately (91.83 ± 6.12) mg/dL. These results are consistent with the findings of (17), which indicated that LDL-C levels were significantly higher in T2DM patients compared to the control group, and that this increase was positively associated with the risk of vascular complications and cardiovascular disease in the affected individuals. As for the two groups of T2DM patients with high and normal weights, the results in Table (2) showed a significant increase ($P \leq 0.05$) in LDL-C concentration among T2DM patients with high weights, reaching approximately (148.30±5.69) mg/dL, compared to the group of T2DM patients with normal weights, which was approximately (106.32±4.79) mg/dL.

The reason for the increased concentrations of LDL-C in T2DM patients is the presence of insulin resistance, which promotes the formation of small, dense LDL particles by inhibiting lipoprotein lipase (LPL) and increasing the activity of hepatic lipase and cholesteryl ester transfer protein (CETP), all of which raise LDL-C levels in the serum (18). IR also leads to impaired hepatic regulation and increased secretion of VLDL, which is loaded with triglycerides and cholesterol. When broken down in the bloodstream, part of it is converted into small, dense low-density lipoprotein (LDL) particles, which are more harmful to blood vessels and contribute to the increase in harmful cholesterol concentration (LDL-C). The rise in triglycerides (TG) exacerbates this process through the cholesterol ester transfer protein (CETP) enzyme, where triglycerides are transferred from VLDL to LDL, increasing LDL density and enhancing its atherogenic properties.

3.4 The concentration of high-density lipoprotein cholesterol (HDL-C) in the serum

The results in Table (1) showed a significant decrease ($P \leq 0.05$) in HDL-C concentration among patients with type 2 diabetes, as it reached approximately (40.93 ± 2.79) mg/dL, compared to the control group which was approximately (53.81 ± 5.80) mg/dL. These results are consistent with the findings of study (20), which indicated a decrease in HDL-

C concentrations among T2DM patients compared to the control group, and that the reduction in beneficial cholesterol HDL-C was inversely related to triglyceride (TG) levels in the serum of T2DM patients. As for the two groups of T2DM patients with high and normal weights, the results in Table (2) indicated a significant decrease ($P \leq 0.05$) in HDL-C concentrations among T2DM patients with high weights, which was approximately (38.68 ± 1.93) mg/dL, compared to the group of T2DM patients with normal weights, which was approximately (42.93 ± 3.81) mg/dL.

HDL-C plays an important role in removing harmful cholesterol (LDL) from tissues and returning it to the liver to be excreted from the body. Its low levels reflect a dyslipidemia, which is one of the causes of increased total cholesterol (TC) and harmful cholesterol (LDL) in the blood, raising the risk of vascular damage and accelerating microvascular complications associated with T2DM (21). The low concentration of HDL-C in T2DM patients is attributed to hyperglycemia, which alters the dynamics and composition of HDL-C through the glycation of the Apo lipoprotein APOA-I protein, the main component of HDL, leading to its removal from the blood.‹ The increase in TG concentrations also enriches HDL with triglycerides, making it more susceptible to hepatic lipase degradation, which breaks down HDL and removes APOA-I through the kidneys. Furthermore, the increased activity of endothelial lipase (EL) due to the reduced levels of angiopoietin-like protein 3 (ANGPTL3) leads to a decrease in the phospholipid content of HDL, contributing to its degradation and lowering its concentrations in the blood (22).

3.5 The concentration of very low-density lipoprotein cholesterol (VLDL-C) in the serum

The results in Table (1) showed a significant increase ($P \leq 0.05$) in VLDL-C levels among patients with type 2 diabetes, reaching approximately (36.64 ±2.91) mg/dL, compared to the control group which had approximately (22.11 ± 6.17) mg/dL. These results are consistent with the findings of (23), which indicated a significant increase in VLDL-C concentrations among T2DM patients, with the highest levels found in men with T2DM compared to the healthy control group.

According to my groups of T2DM patients with high and normal weights, the results in Table (2) showed no significant increase ($P \leq 0.05$) in VLDL-C concentration among T2DM patients with high weights, which was approximately (39.17 ±3.83) mg/dL, compared to the group of T2DM patients with normal weights, which was approximately (34.12 ± 2.00) mg/dL. The increase in VLDL-C levels is attributed to the hydrolysis of triglycerides rich in VLDL-C by the enzyme lipoprotein lipase (LPL), which is found in the endothelial lining of blood vessels in adipose tissue and muscles. During this process, fatty acids are released, which subsequently undergo oxidation processes either to reform and store triglycerides or to be used as an energy source. These fatty acids are absorbed by various body tissues according to their metabolic needs, or the increase in VLDL-C in the blood occurs. Insulin resistance and high concentrations reduce the activity of the enzyme Lipoprotein Lipase (LPL), which is responsible for breaking down triglycerides in VLDL particles to supply energy to muscle and heart tissues. This leads to decreased LPL activity, accumulation of VLDL in the blood, and increased concentrations. Additionally, elevated levels of apolipoprotein ApoC-III, a protein that inhibits LPL activity, hinder the breakdown of VLDL and reduce its clearance from the blood, increasing the risk of hypertriglyceridemia and cardiovascular diseases (CVD) (25). The increase in glucose and insulin levels activates the De Novo Lipogenesis (DNL) pathway, which is a metabolic pathway that converts excess carbohydrates into fatty acids in the liver. The fatty acids are then esterified and converted into triglycerides within the liver. Since triglycerides cannot be stored in large quantities within the liver, they are packaged into VLDL particles for transport through the blood, leading to elevated levels of VLDL-C in the blood (26).

3.6 Creatinine concentration in blood serum

The results in Table (1) showed a significant increase ($P \leq 0.05$) in serum creatinine concentration in patients with type 2 diabetes, reaching approximately (1.19 ± 0.34) mg/dL, compared to the control group which had approximately (0.71 ± 0.11) mg/dL. These results were consistent with those of study (27), which showed elevated creatinine levels in individuals with T2DM compared to the control group, and this increase was considered a confirmed indicator of kidney function deterioration and the occurrence of DKD complications in T2DM patients. According to the groups of patients, the results in Table (2) indicated a significant increase ($P \leq 0.05$) in the creatinine concentration level among patients with T2DM and high body weight, which was approximately (1.25 ± 0.37) mg/dL, compared to the group of patients with T2DM and normal body weight, which was approximately (1.07 ± 0.35) mg/dL.

Serum creatinine is a more effective indicator for assessing kidney function compared to serum urea levels. Elevated blood sugar levels in T2DM patients lead to damage and blockage of the microvessels in the kidneys, causing damage to millions of nephrons in the renal glomeruli, which weakens their ability to filter waste from the blood. Since creatinine filtration occurs through these glomeruli, serum creatinine levels are considered an indicator of the Glomerular Filtration Rate (GFR). The values of creatinine and the GFR are inversely proportional, so a decrease in the GFR leads to an increase in serum creatinine concentrations in the plasma, causing kidney disease.

Furthermore, many patients with T2DM suffer from hypertension, which is a major risk factor for kidney damage. Hypertension exacerbates kidney damage and increases the likelihood of cardiovascular complications, leading to a decrease in the glomerular filtration rate (GFR), consequently resulting in elevated serum creatinine levels (29).

3.7 Urea concentration in blood serum

The results in Table (1) showed a significant increase ($P \leq 0.05$) in urea concentration in patients with type 2 diabetes, reaching approximately (40.36 ± 3.18) mg/dL, compared to the control group which had approximately (26.17 ± 1.58) mg/dL. These results were consistent with the findings of (30), which showed elevated urea levels in individuals with T2DM compared to the control group. This increase in urea levels, along with creatinine, is considered a clinical indicator of diabetic nephropathy in uncontrolled diabetic patients with hyperglycemia. Therefore, these levels can predict the progression of kidney disease in its final stage. As for the groups of patients, the results in Table (2) showed no significant increase ($P \leq 0.05$) in the urea level among patients with T2DM who have high weights, as it was approximately (42.57 ± 3.18) mg/dL, compared to the group of patients with T2DM who have normal weights, which was approximately (37.87 ± 3.18) mg/dL.

Urea or Blood Urea Nitrogen (BUN) is a nitrogen-containing compound that is formed in the liver as the final product of protein metabolism and the urea cycle. About 85% of urea is excreted by the kidneys, while the remaining portion is excreted by the gastrointestinal tract. The increase in blood urea levels is attributed to three main causes: Pre-renal, renal, and post-renal causes: Pre-renal causes arise from increased production of urea in the liver due to a protein-rich diet, leading to uremia. Additionally, low blood pressure, shock, or dehydration can reduce the rate of urea clearance, contributing to its accumulation and increased concentration in the blood. The renal causes include a decrease in kidney function due to certain diseases such as acute or chronic renal failure, tubular necrosis, and others. Post-renal causes occur due to obstruction in the urinary tract caused by prostate enlargement or urinary tract infections (32). Moreover, insulin resistance in T2DM causes an increase in protein breakdown and reduces the cells' ability to inhibit proteolysis, leading to increased degradation of muscle proteins and the release of amino acids. These amino acids then enter the urea cycle in the liver, increasing urea production and consequently raising its concentrations in the serum (33).

Table 1 Concentration of Total cholesterol (TC) , Triglycerides (TG) , low-density lipoprotein cholesterol (LDL-C) , high-density lipoprotein cholesterol (HDL-C) , very low-density lipoprotein cholesterol (VLDL-C) , Creatinine , and Urea in Diabetic Patients and the Control Group

Variables	Diabetic Group	Control Group	Significance Level
Total cholesterol (TC)	205.37 ± 3.64	165.77 ± 6.22	$P \leq 0.05$
Triglycerides (TG)	183.36 ± 8.96	110.53 ± 3.88	$P \leq 0.05$
LDL-C	127.86 ± 5.13	91.83 ± 6.12	$P \leq 0.05$
HDL-C	40.93 ± 2.79	53.81 ± 5.80	$P \leq 0.05$
VLDL-C	36.64 ±2.91	22.11 ± 6.17	$P \leq 0.05$
Creatinine	1.19 ± 0.34	0.71 ± 0.11	$P \leq 0.05$
Urea	40.36 ± 3.18	26.17 ± 1.58	$P \leq 0.05$

Table 2 Concentration of Total cholesterol (TC) , Triglycerides (TG) , low-density lipoprotein cholesterol (LDL-C) , high-density lipoprotein cholesterol (HDL-C) , very low-density lipoprotein cholesterol (VLDL-C) , Creatinine , and Urea According to Subgroups of Patients with High and Normal Body Weight

Variables	Diabetic (High Weight)	Diabetic (Normal weight)	Significance Level
Total cholesterol (TC)	225.96 ± 5.31	182.36 ± 2.20	$P \leq 0.05$
Triglycerides (TG)	195.80 ± 6.33	170.63 ± 3.55	$P \leq 0.05$
LDL-C	148.30±5.69	106.32±4.79	$P \leq 0.05$
HDL-C	38.68 ± 1.93	42.93 ± 3.81	$P \leq 0.05$
VLDL-C	39.17 ±3.83	34.12 ± 2.00	$P \leq 0.05$

Creatinine	1.25 ± 0.37	1.07 ± 0.35	P ≤ 0.05
Urea	42.57 ± 3.18	37.87 ± 3.18	P ≤ 0.05

4 Conclusion

The present study demonstrated that patients with type 2 diabetes mellitus exhibit significant disturbances in lipid profile and kidney function parameters compared to healthy individuals, including elevated levels of HbA1c, TC, TG, LDL, VLDL, creatinine, urea, and BMI, along with decreased HDL levels. Moreover, diabetic patients with higher BMI showed more pronounced dyslipidemia and renal burden than those with normal BMI, except for HbA1c and urea, which showed no significant differences. These findings underscore the combined metabolic impact of diabetes and obesity, highlighting the need for integrated weight management and biochemical monitoring in clinical practice. This study contributes to a better understanding of obesity-related complications in T2DM and supports future research toward developing targeted preventive strategies to reduce disease burden in the community.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that there is no conflict of interest to disclose."

Statement of ethical approval

The study was conducted in accordance with ethical guidelines.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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