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# Biochemical Study of Obesity with Type 2 Diabetes Mellitus in Iraqi Patients

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## Abstract

Obesity causes diabetes to worsen faster. Here's what happens: Managing the level of glucose in your blood is the job of the pancreas. One hundred and ninety diabetic samples were collected from patients attended Alkaleeg Clinical Lab. In Wasit, from period 1<sup>st</sup> March 2023 to 1<sup>st</sup> November 2023. According the results, the mean and SD of age for patients and control groups were (43.86 ± 14.67) and (43.16 ± 13.77), respectively, with a non-significant difference (p > 0.05). In relation to gender, this table showed that the patient group and the control group were equal regarding the male: female ratio, with non-significant differences (p > 0.05). The results of smoking status showed that the majority of patients in group 112 (93.3%) were non-smokers, while 100% of the control group were non-smokers, with non-significant differences (p > 0.05). The non-significant differences in age and gender between the patient and control groups may be due to the study's recruitment strategy, which aimed to recruit participants of similar ages and genders in both groups to reduce the confounding factors that may influence the study's results. This finding is consistent with previous research studies that have emphasized the importance of controlling for demographic factors, such as age and gender, in clinical studies, the mean and (SD) of FBS for patients and control groups were (228.04 ± 110.77) and (100.52 ± 9.44), respectively, with highly significant differences (p < 0.001). while the mean and (SD) of HbA1c for patients and control groups were (9.07 ± 2.53) and (4.96 ± 0.54), respectively, with highly significant differences (p < 0.001).

Results in the same table also showed that the mean and (SD) of urea for patients and control groups were (27.48 ± 7.01) and (25.54 ± 5.67) respectively, with significant differences between them (P < 0.05). The mean and SD of creatinine for patients and controls was shown to be (0.75 ± 0.17) and (0.66 ± 0.15) respectively, with significant differences between the patients and controls (P < 0.05). In relation to cholesterol, table (4-3) revealed that the mean and (SD) of cholesterol for patients and control groups were (169.22 ± 29.04) and (173.28 ± 21.17) respectively, but with non-significant differences between them (P > 0.05), while the mean and SD of triglyceride for patients and controls was (131.30 ± 57.96) and (92.37 ± 26.09) respectively, with highly significant differences between them (P < 0.01). The same table showed that the mean (SD) of HDL for patients and control groups were (39.28 ± 6.006) and (45.22 ± 8.59), respectively, with highly significant differences (P < 0.01). The mean and (SD) of LDL for patients and control groups were (104.20 ± 27.86) and (109.59 ± 17.06), respectively, with significant differences between them (p < 0.05). The mean and SD of VLDL for patients and controls was (26.28 ± 11.56) and (18.47 ± 5.21), respectively, with highly significant differences between them (P < 0.01). The mean and SD of FBS for patients and controls was (228.04 ± 110.77) and (100.52 ± 9.44), respectively, with highly significant differences (P < 0.01). While the mean and (SD) of HbA1c for patients and control groups were (9.07 ± 2.53) and (4.96 ± 0.54), respectively, with highly significant differences (P < 0.01). Also the mean and (SD) of urea for patients and control groups were (27.48 ± 7.01) and (25.54 ± 5.67) respectively, with significant differences between the groups (P < 0.05). The mean and SD of creatinine for patients and controls was shown to be (0.75 ± 0.17) and (0.66 ± 0.15) respectively, with significant differences between both groups (P < 0.05). In relation to cholesterol, table (4-3) revealed that the mean and (SD) of cholesterol for patients and control groups were (169.22 ± 29.04) and (173.28 ± 21.17) respectively, but with non-significant differences between them (P > 0.05), while the mean and SD of triglyceride for patients and controls was (131.30 ± 57.96) and (92.37 ± 26.09) respectively, with highly significant differences between them (P < 0.01). The same table showed that the mean (SD) of HDL for patients and control groups were (39.28 ± 6.006) and (45.22 ± 8.59), respectively, with highly significant differences (P < 0.01). The mean and (SD) of LDL for patients and control groups were (104.20 ± 27.86) and (109.59 ± 17.06), respectively, with significant differences between them

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( $P < 0.05$ ). The mean and SD of VLDL for patients and controls was ( $26.28 \pm 11.56$ ) and ( $18.47 \pm 5.21$ ), respectively, with highly significant differences between them ( $P < 0.01$ ). The correlation coefficients between C-peptide and FBS in both the patient and control groups are very small, indicating a weak or no relationship between these variables. In the patient group, the correlation coefficient is slightly negative, while in the control group, it is positive but small, with a non-significant correlation between the two groups. There are several possible reasons why there may not be a significant relationship between C-peptide and FBS in this study. The correlation coefficients between C-peptide and HbA1c in both the patient and control groups are small, indicating a weak or no relationship between these variables. In the patient group, the correlation coefficient is slightly negative, while in the control group, it is also negative but smaller. However, only the correlation coefficient in the patient group is statistically significant ( $p = 0.041$ ).

**Keywords:** Biochemical, Type 2, Diabetes mellitus, Iraqi patients

## 1. Introduction

Type 2 diabetes mellitus risk is strongly associated with excess body weight. It has been reported that IR increases with increasing BMI, waist circumference, and waist-hip ratio. In addition to being overweight or obese, risk factors for developing T2DM include increasing age, lifestyle factors such as physical inactivity and an unhealthy diet, a family history of T2DM, and a history of GDM or polycystic ovarian syndrome (PCOS) (Sparks *et al.*, 2022). As a result glucose production increases, which leads to diabetes, Likewise The pancreas loses its ability to insulin production gradually (Zhang *et al.*, 2019). Obesity arises and develops in 1 or 2 out of every 5 people (due to malnutrition: eating a lot of fatty meat, a lot of starches and drinking Soft and sweetened drinks, Obesity also increases with age and lack of movement. Metabolic syndrome is known as; insulin resistance syndrome (Foretz *et al.*, 2019). Serious global health issue that has developed in response to quick cultural, economic, and social change, older people, rapid and unplanned urbanization, dietary changes such as an increase in the consumption of highly processed foods and sugar-sweetened beverages, obesity, decreased physical activity, unhealthy lifestyle and behavioral patterns, fetal malnutrition, and an increase in the exposure of fetuses to hyperglycemia during pregnancy (Adler *et al.*, 2021). T2DM affects more adults than children or adolescents, however, this figure is rising (Wong *et al.*, 2022). In the Arabic countries like other developing countries, the prevalence of T2DM is increasing (Milibari *et al.*, 2020). The World Health Organization reports that some 415 million people worldwide live with diabetes. Global trends suggest a steady rise of the prevalence rate by about 2.5% a year (Kotwas *et al.*, 2021). Connecting peptide (C-peptide) is short polypeptide chain consist from 31 amino acid, when the blood glucose level increased; insulin release from B-cell and also the equal amounts of C-peptide are produced at the same time of insulin secretion (Bidlingmaier *et al.*, 2022). The amount of c-peptide reflects the amount

of insulin produced. The low C-peptide production indicates low insulin production, and vice versa, but the half-life of C-peptide more than insulin (Böyük *et al.*, 2018). As the c-peptide test is used to determine the cause of high or low blood glucose, and it is also used to differentiate between type 1 and type 2 diabetes (Huang *et al.*, 2020). The aim of our study to evaluation of biochemical aspect in patients with type 2 Diabetes mellitus.

## 2. Materials and methods

One hundred and ninety diabetic samples were collected from patients attended Alkaleeg Clinical Lab. In Wasit, from period 1st March 2023 to 1st November 2023. In the current study, eight milliliters (8 ml) of venous blood were collected from each subject between 9 and 11 a.m. after an overnight fast of 8 to 12 hrs. Samples were divided into 2 aliquot: 2 ml of blood was put in the EDTA tubes for measurement of glycated hemoglobin (HbA1c), while 6 ml of the blood was placed in a clean and dry gel tube and left to clot at room temperature for 15 minutes, then centrifuged at 3000 rpm for 15 minutes to obtain serum samples, which were put in two Eppendorf tubes. The first Eppendorf tubes contain 4 mL of separated serum to measure the level of fasting blood glucose (FBS) and lipid profiles: cholesterol (CHO), triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL), and renal function as urea and creatinine. The second Eppendorf tube contain 2 mL of separated serum stored in the freezer at  $-20^{\circ}\text{C}$  until used for assays (dopamine 2 receptors, insulin receptors, and C-peptides). HbA1c were detected by minividas devise, while FBS, Lipid profile, Urea and Critinine were tested by Spectrophotometer. C-Peptide ELISA Test Principle: The essential reagents required for an immunoenzymometric assay include high affinity and specificity antibodies (Ab). (enzyme conjugated and immobilized).

Calculation of Body Mass Index (BMI) is by a simple calculation using a person's height and weight, which

Table 1. Relationship of age, gender and smoking in the study group.

Demographical data			Patients (n = 120)	Controls (n = 60)	P-values
Age	20-29	NO	28	16	.759 N.S
		%	23.3%	26.7%	
	30-39	NO	10	6	
		%	8.3%	10.0%	
	40-49	NO	26	16	
		%	21.7%	26.7%	
	50-59	NO	38	16	
%		31.7%	26.7%		
60 and more	NO	18	6		
	%	15.0%	10.0%		
Total	NO	120	60		
	%	100.0%	100.0%		
	Mean ± SD	43.86 ± 14.67	43.16 ± 13.77		
	Min-Max	16-65	16-65		
Gender	Male		60	30	.400 N.S
		%	50.0%	50.0%	
	Female		60	30	
%		50.0%	50.0%		
Total		120	60		
		100.0%	100.0%		
Smoking	No		112	60	.051 N.S
		%	93.3%	100.0%	
	Yes		8	0	
%		6.7%	0.0%		
Total		120	60		
	%	100.0%	100.0%		

\*Mann-Whitney t-test, p-value ≤ 0.05, N.S. = no significance, H.S. = highly significance.

is a reliable indicator of body fatness for most people, according to the equation:  $BMI = \text{kg}/\text{m}^2$ , where kg refers to the weight in kilograms and  $\text{m}^2$  refers to height in square meter ( $\text{kg}/\text{m}^2$ ). The results of BMI are commonly utilized to classify underweight, normal weight, overweight and obese in adults.

### 2.1. Statistical analysis

Continuous (quantitative) variables are given as means, minimum and maximum standard deviations, whereas categorical (qualitative) variables are given as frequency and percentage. Kolmogorov-Smirnov test was used to test distribution normality.

## 3. Results

Mean and SD of age for patients and controls were  $(43.86 \pm 14.67)$  and  $(43.16 \pm 13.77)$ , respectively, with a non-significant difference ( $p > 0.05$ ). In relation to gender, this table showed that the patient group and the control group were equal regarding the male:female ratio, with non-significant differences ( $p > 0.05$ ). The results of smoking status showed that the majority of patients in group 112 (93.3%) were non-smokers, while 100% of the control group were non-smokers, with non-significant differences ( $p > 0.05$ ). The non-significant differences in age and

gender between the patient and control groups may be due to the study's recruitment strategy, which aimed to recruit participants of similar ages and genders in both groups to reduce the confounding factors that may influence the study's results. This finding is consistent with previous research studies that have emphasized the importance of controlling for demographic factors, such as age and gender, in clinical studies, as shown in Table 1.

Results in Table 2 show that the mean and (SD) of FBS for patients and control groups were  $(228.04 \pm 110.77)$  and  $(100.52 \pm 9.44)$ , respectively, with highly significant differences ( $p < 0.01$ ). While the mean and (SD) of HbA1c for patients and control groups were  $(9.07 \pm 2.53)$  and  $(4.96 \pm 0.54)$ , respectively, with highly significant differences ( $P < 0.01$ ).

Results in the same table also showed that the mean and (SD) of urea for patients and control groups were  $(27.48 \pm 7.01)$  and  $(25.54 \pm 5.67)$  respectively, with significant differences between the groups ( $P < 0.05$ ). The mean and SD of creatinine for patients and controls was shown to be  $(0.75 \pm 0.17)$  and  $(0.66 \pm 0.15)$  respectively, with significant differences between patients and controls ( $P < 0.05$ ). In relation to cholesterol, table (4-3) revealed that the mean and (SD) of cholesterol for patients and control groups were  $(169.22 \pm 29.04)$  and  $(173.28 \pm 21.17)$  respectively, but with non-significant differences between

Table 2. Distribution of biochemical parameters among the study groups.

		N	Mean $\pm$ SD	p-value	p-value
FBS	Patients	120	228.04 $\pm$ 110.77	.000*	.000*H.S
	Control	60	100.52 $\pm$ 9.44		
HbA1c	Patients	120	9.07 $\pm$ 2.53	.000*	.000*H.S
	Control	60	4.96 $\pm$ .54		
Urea	Patients	120	27.48 $\pm$ 7.01	.048**	.048**Sig
	Control	60	25.54 $\pm$ 5.67		
Creatinine	Patients	120	.75 $\pm$ .17	.001*	.001H*Sig
	Control	60	.66 $\pm$ .157		
Cholesterol	Patients	120	169.22 $\pm$ 29.04	.077	.077*N.S
	Control	60	173.28 $\pm$ 21.17		
Tri	Patients	120	131.30 $\pm$ 57.96	.000*	.000*H.S
	Control	60	92.37 $\pm$ 26.09		
H D L	Patients	120	39.28 $\pm$ 6.006	.000*	.000*H.S
	Control	60	45.22 $\pm$ 8.59		
L D L	Patients	120	104.2 $\pm$ 27.86	.100	.100*N.S
	Control	60	109.59 $\pm$ 17.06		
V L D L	Patients	120	26.28 $\pm$ 11.56	.000*	.000*H.S
	Control	60	18.47 $\pm$ 5.21		

\*Mann-Whitney test, \*\*Independent sample t-test, p-value  $\leq$  0.05.

Table 3. Correlations between C. peptide level and biochemical parameters among the study groups.

C. peptide	Pearson correlation	p-value	
FBS	Patients	-.059	.525N.S
	Control	.101	.441N.S
HbA1c	Patients	-.187*	.041 sig
	Control	-.128	.331N.S
Urea	Patients	-.258*	.004Sig
	Control	.113	.388N.S
Creatinine	Patients	-.219*	.016N.S
	Control	.164	.212Sig
Chol	Patients	.130	.156N.S
	Control	.062	.637N.S
Tri	Patients	-.069	.453N.S
	Control	-.090	.492N.S
H D L	Patients	-.152	.097N.S
	Control	-.280	.030Sig
L D L	Patients	.188*	.040N.S
	Control	.240	.065N.S
V L D L	Patients	-.067	.469N.S
	Control	-.089	.497N.S

them ( $P > 0.05$ ), while mean and SD of triglyceride for patients and controls was (131.30  $\pm$  57.96) and (92.37  $\pm$  26.09) respectively, with highly significant differences between both groups ( $p < 0.01$ ). The same table showed that the mean (SD) of HDL for patients and control groups were (39.28  $\pm$  6.006) and (45.22  $\pm$  8.59), respectively, with highly significant differences ( $p < 0.01$ ). The mean and (SD) of LDL for patients and control groups were (104.20  $\pm$  27.86) and (109.59  $\pm$  17.06), respectively, with significant differences between them ( $P < 0.05$ ). The mean and SD of VLDL for patients and controls was (26.28  $\pm$  11.56) and (18.47  $\pm$  5.21), respectively, with a highly significant difference between them ( $p < 0.001$ ).

The data in Table 3 showed that the correlation coefficients between C-peptide and FBS in both the patient and control groups are very small, indicating a weak or no relationship between these variables. In the patient group, the correlation coefficient is slightly negative, while in the control group, it is positive but small, with a non-significant correlation between the two groups. There are several possible reasons why there may not be a significant relationship between C-peptide and FBS in this study. The correlation coefficients between C-peptide and HbA1c in both the patient and control groups are small, indicating a weak or no relationship between these variables. In the patient group, the correlation coefficient is slightly negative, while in the control group, it is also negative but smaller. However, only the correlation coefficient in the patient group is statistically significant ( $p = 0.041$ ).

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Table 4. Distribution of biochemical parameters among the study groups.

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	Control	60	100.52 $\pm$ 9.44		
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	Control	60	4.96 $\pm$ .54		
Urea	Patients	120	27.48 $\pm$ 7.01	.048**	.048**Sig
	Control	60	25.54 $\pm$ 5.67		
Creatinine	Patients	120	.75 $\pm$ .17	.001*	.001H*Sig
	Control	60	.66 $\pm$ .157		
Cholesterol	Patients	120	169.22 $\pm$ 29.04	.077	.077*N.S
	Control	60	173.28 $\pm$ 21.17		
Tri	Patients	120	131.30 $\pm$ 57.96	.000*	.000*H.S
	Control	60	92.37 $\pm$ 26.09		
H D L	Patients	120	39.28 $\pm$ 6.006	.000*	.000*H.S
	Control	60	45.22 $\pm$ 8.59		
L D L	Patients	120	104.2 $\pm$ 27.86	.100	.100*N.S
	Control	60	109.59 $\pm$ 17.06		
V L D L	Patients	120	26.28 $\pm$ 11.56	.000*	.000*H.S
	Control	60	18.47 $\pm$ 5.21		

\*Mann-Whitney test, \*\*Independent sample t-test, p-value  $\leq$  0.05.

and (173.28  $\pm$  21.17) respectively, but with non-significant differences between them ( $P > 0.05$ ), while the mean and SD of triglyceride for patients and controls was (131.30  $\pm$  57.96) and (92.37  $\pm$  26.09) respectively, with highly significant differences between them ( $P < 0.01$ ). The same table showed that the mean (SD) of HDL for patients and control groups were (39.28  $\pm$  6.006) and (45.22  $\pm$  8.59), respectively, with highly significant differences ( $P < 0.01$ ).

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#### 4. Discussion

According to the results the mean and SD of age for patients and control groups with no significant differences. A study conducted by (Campagna *et al.*, 2019), found that cigarette smoking was significantly associated with an increased risk of T2DM. Also, a study done by (Wang *et al.*, 2020) reported that smoking was associated with an increased risk of T2DM, with a stronger association observed in women, while a study by (Gallucci *et al.*, 2020) found that smoking cessation was associated with a significant reduction in the risk of developing T2DM. A study by (Huang *et al.*, 2020) showed that exposure to secondhand smoke was associated with an increased risk of T2DM. A review by (Stewart *et al.*, 2020) [15] found consistent evidence linking smoking to an increased risk of T2DM and recommended smoking cessation as a

primary prevention strategy for T2DM. Smoking may raise the risk of diabetes development or deteriorate the disease in individuals who are already affected with diabetes, which is attributed to insulin resistance caused by smoking, which means the body's cells become less responsive to insulin. The hormone Insulin can help the human to utilize and store sugar from food we consume, and when cells resist insulin, the levels of blood sugar can increase, resulting in type-2 DM. As well as insulin resistance increase, smoking may also increase inflammations in the human's body, which may also deteriorate insulin resistance and contribute to diabetic development. Moreover, smoking increases other complication risks of which are related to DM, like cardiovascular diseases, renal diseases and nerve damages (Campagna *et al.*, 2019). Regarding the FBS and HbA1c results, several studies by (Godman *et al.*, 2020; Omazi *et al.*, 2021; and Morieri *et al.*, 2020) found significantly higher FBS and HbA1c levels in the patient group compared to the control group, indicating impaired glucose metabolism and poor long-term glucose control in the patient group. The findings of urea and creatinine agreed with a study done by (Salem *et al.*, 2022), who found significantly higher urea and creatinine levels in the patient group compared to the control group, indicating impaired kidney function in the patient group.

In regard to lipid results, they agreed with the findings of (Chen *et al.*, 2020) who also found non-significant differences in cholesterol levels between patient and controls, but significantly higher triglyceride levels and significantly lower HDL levels in the patient group, indicating dyslipidemia and poor lipid profile in the patient group.

Finally, in relation to LDL and VLDL results, studies conducted by (Saber *et al.*, 2020), also found non-significant differences in LDL levels between the patient and controls but significantly higher VLDL levels in the patient group, indicating poor lipid metabolism in the patient group. Diabetes can lead to an increase in the lipid profile, which includes the levels of triglycerides, LDL cholesterol, and total cholesterol in the blood. There are several possible reasons why there may be a weak negative relationship between C-peptide and HbA1c: Impaired insulin secretion: C-peptide is byproduct of insulin production, so a decrease in C-peptide levels may indicate impaired insulin secretion. This may lead to higher HbA1c levels due to poor glucose control. FBG (Fasting Blood Glucose), HbA1c (Glycated Hemoglobin), and C-peptide are all measures that can provide insight into an individual's glucose control and diabetes management (Cefalu *et al.*, 2022).

FBG is a measure of the amount of glucose in the blood after an individual has fasted for at least 8 hours, while HbA1c is a measure of the average blood glucose level over the past 2-3 months, and C-peptide is a marker of insulin secretion. Higher FBG levels are generally associated with higher HbA1c levels, as the average glucose level over time will be higher if the individual's glucose level is consistently high. However, the relationship between FBG and C-peptide is more complex, as C-peptide is a marker of insulin secretion rather than glucose control. In general, higher C-peptide levels are associated with better insulin secretion and glucose control, while lower C-peptide levels may indicate impaired insulin secretion (Sherwani *et al.*, 2016). The correlations between C-peptide and Urea, as well as C-peptide and creatinine, suggest that there may be some relationship between these variables in patients with diabetes. Renal function can be assessed by measuring urea and creatinine levels in the blood, which may be affected by changes in glucose control and insulin secretion. In individuals with diabetes, high levels of glucose can damage the kidneys over time, leading to impaired kidney function and changes in the levels of urea and creatinine in the blood (Campbell *et al.*, 2011). Higher C-peptide levels may indicate better insulin secretion, which in turn may help to protect the kidneys and maintain kidney function (D'elia *et al.*, 2019).

Higher levels of urea and creatinine, which are renal function markers, may be an indication of decreased renal functions or renal damages. In diabetic people, renal damage is a regular complication called diabetic nephropathy. There are many reasons, such as glucose toxicity, that means high levels of glucose in blood over for a long time causing blood vessel damage in both kidneys, impairing their capacity for filtering

waste products in blood, leading to urea and creatinine accumulations in blood (Kawahit *et al.*, 2009). In regard to lipid results, they agreed with the findings of (Chen *et al.*, 2020), who also found non-significant differences in cholesterol levels between the patient and controls, but significantly higher triglyceride levels and significantly lower HDL levels in the patient group, indicating dyslipidemia and poor lipid profile in the patient group.

Finally, in relation to LDL and VLDL results, studies conducted by (Saber *et al.*, 2020), also found non-significant differences in LDL levels between the patient and control groups but significantly higher VLDL levels in the patient group, indicating poor lipid metabolism in the patient group. Diabetes can lead to an increase in the lipid profile, which includes the levels of triglycerides, LDL cholesterol, and total cholesterol in the blood.

## 5. Conclusions

According to the results there was a highly levels in the concentration of Lipid profile and HbA1c diabetes mellitus and they are effected by obesity.

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