



Insulin Resistance and Its Relation with Type 2 Diabetes Mellitus Patients

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ABSTRACT

One hundred samples were collected from the Al-Wafa Diabetes and Endocrinology Center in Mosul. The samples were divided into four groups: healthy, insulin-resistant, insulin resistance with diabetes and diabetic patients without insulin resistance. Individuals underwent additional age-based metabolic stratification into two age groups: 10-35 and 36-65 years. Blood samples were analyzed using biochemical tests to assess insulin levels, along with assessments of fasting blood glucose (FBS), glycated hemoglobin (HbA1c) and lipid profiles. Results showed that insulin-resistant and diabetic patients had higher insulin levels, FBS, and HbA1c compared to healthy group, suggesting that insulin resistance leads to metabolic problems. Insulin levels remained stable in young people but beta cells progressively lost function as their condition progressed with age. The research also investigates the relationship between insulin resistance and lipid metabolisms

Insulin-resistant and diabetic people had higher TC, TG, LDL and VLDL levels and lower HDL levels. Screening patients using early biomarkers can help assess insulin resistance (HOMA-IR) in patients with type 2 diabetes.

Keywords: Insulin resistance, Type 2 diabetes mellitus, HOMA-IR, Fasting blood sugar, HbA1c.

INTRODUCTION

Diabetes mellitus (DM) is an increasing issue, can be harmful and very costly (Mohammed and Abdul-Razzaq, 2020). The worldwide increase in metabolic disorders has made insulin resistance stand out as an important public health matter, Type2 diabetes mellitus (DM) is a metabolic condition characterized by persistently high blood sugar levels (AbdlWhab *et al.*, 2024).

Insulin resistance (IR) can be defined as an improper bodily response to insulin that affects targeted cells particularly within the liver, muscles together with various types and locations of adipose tissue. Insulin resistance creates problems with glucose release causing the pancreas to increase insulin secretion resulting hyperinsulinemia state within the whole body (Wolosowicz *et al.*, 2022). After eating blood glucose concentrations rise beta cells generate insulin for regulating sugar levels in the body. The regulation of homeostasis occurs because of insulin sensitivity in the body. Multiple body organs such as liver skeletal muscle together with adipose tissue maintain homeostatic control of glucose. Insulin clears a sequence of protein activations under precise regulation (Honka *et al.*, 2018). Multiple metabolic functions receive control through signaling including the reduction of liver-made glucose and the increase of glucose uptake by skeletal muscle and adipose tissue. The tissue functions of adipose tissue alongside skeletal muscle become better at absorbing glucose and they suppress the activation of free fatty acid release from adipocytes. The body controls insulin actions as an essential mechanism to achieve correct metabolic processes while maintaining energy equilibrium (Chandrasekaran and Weiskirchen, 2024). A wide range of pathogenic factors alongside their corresponding pathogenesis have been suggested to date in relation to insulin resistance including genetics, obesity, aging, exercise and diet. The development of insulin resistance in present times mostly depends on diet-induced metabolic dysfunctions (Yang *et al.*, 2024). An insulin resistance condition can result in hyperglycemia, elevated blood pressure, dyslipidemia, visceral adiposity, innate and chronic inflammatory responses, endothelial layer function impairment and dysregulation of the hemostasis balance (Wolosowicz *et al.*, 2022). Being overweight is the main reason for insulin resistance, but genetic factors are also a cause of this condition. It is still not clear how insulin resistance is clinically defined, since there is no standard insulin resistance test. Insulin resistance is diagnosed clinically based on the metabolic changes we see in metabolic syndrome and insulin resistance syndrome (Hossan *et al.*, 2019). The purpose of this study was to identify insulin resistance in individuals before appearance of diabetes by HOM-IR method across different age groups.

MATERIALS AND METHODS

One hundred blood samples were gathered from patients at Al Wafaa Center for Diabetes and Endocrine Gland Diseases in Mosul. The evaluations of patients were carried out by physicians, approval for research project numbered 2024182 has been given on 10/10/2024, and samples were collected under the supervision of a field supervisor. Collection of samples took a three-month period from October 2024 to January 2025. Samples were divided into four distinct groups consisting of 30 healthy individuals and 30 patients with insulin resistance together with another group of 30 patients who had both insulin resistance and diabetes, and 10 patients with type 2 diabetes without insulin resistance, and each group underwent additional age-based categorization for metabolic assessment into two age groups (10-35) years and (36-65) years.

Blood sample collection and preservation

Five millimeters of venous blood obtained from participants following a fasting period of 8-12 hours, 3 ml of the blood sample put into gel tubes for serum obtain while 2 ml put into EDTA tubes.

The gel tubes left for 20-minute rest before centrifugation at 3000 xg for 10 minutes to obtain serum, then divided into Eppendorf tubes and stored at -20 °C. The study utilized Mindray CL1000i for insulin measurement, Mindray BS230 for lipid profile, Indika Thermo Scientific for glucose estimation and i-sense A1Care Analyzer for HbA1c measurement, while Body mass index (BMI) was calculated in this study by measuring participants' weight (in kilograms) and height (in centimeters),

then dividing weight by the square of height in meters (Weigley, 1994). Insulin resistance was determined by homeostatic models' assessment (HOMA-IR) process, $HOMA-IR = [Glu. (mg/dl) \times insulin (\mu Unit/ml)] \div 405$ (Matthews, *et al.*, 1985).

Statistical analysis

The data were analyzed according to the system of simple and universal experiments using a completely randomized design. The different information was significantly distinguished by different letters of the alphabet by Duncan's test, one-way ANOVA at value of $P \leq 0.05$ by SPSS version 26 (Kirkwood, 1988).

RESULTS AND DISCUSSION

The data presented in (Table 1) demonstrates that Insulin, FBS, HOMA-IR and HbA1c levels increased significantly ($P \leq 0.05$) for all patient groups compared to the control group.

Table (1): levels of some biochemical parameters in T2DM and insulin resistance

Groups Parameter	Control Mean $\pm$ SD	Patients		
		IR Mean $\pm$ SD	Diabetes Mean $\pm$ SD	IRand Diabetes Mean $\pm$ SD
Insulin (μ Unit/ml)	8.89 $\pm$ 2.1 ^b	25.32 $\pm$ 6.34 ^a	7.11 $\pm$ 1.2 ^b	28.7 $\pm$ 9.9 ^a
FBS (mg/dl)	85.08 $\pm$ 9.44 ^c	95.39 $\pm$ 9.88 ^c	170.3 $\pm$ 27.78 ^b	255.5 $\pm$ 43.1 ^a
HOMA-IR	1.86 $\pm$ 0.46 ^{cd}	6.00 $\pm$ 1.60 ^b	2.98 $\pm$ 0.42 ^c	15.90 $\pm$ 5.47 ^a
HbA1c (mmol/mol)	5.1 $\pm$ 0.3 ^b	5.35 $\pm$ 0.45 ^b	8.22 $\pm$ 2.76 ^a	9.83 $\pm$ 2.59 ^a

*Different letters mean there is a significant difference between values according to Duncan test.

** The values are means $\pm$ standard deviation (SD)

Scientific evidence reveals a direct link between the number of parameters and diabetes development and insulin resistance progression since metabolic disorder progresses with age as shown in (Table 2). The IR and Diabetes group patients experienced the highest significant change in HOMA-IR levels during the study. The development of insulin resistance together with its effects on glucose metabolic processes became evident through elevated HOMA-IR levels in patients from the IR and Diabetes group.

Table (2): Some biochemical parameters in T2DM and insulin resistance according to age

Groups Parameters	Age(year)	Control Mean $\pm$ SD	Patients		
			IR Mean $\pm$ SD	Diabetes Mean $\pm$ SD	IRand Diabetes Mean $\pm$ SD
Insulin (μ Unit/ml)	(10-35)	9.30 $\pm$ 1.75 ^b	27.65 $\pm$ 6.57 ^a	7.46 $\pm$ 1.12 ^b	29.39 $\pm$ 2.61 ^a
	(36-65)	8.47 $\pm$ 1.11 ^b	22.7 $\pm$ 4.27 ^a	6.80 $\pm$ 0.93 ^b	28.11 $\pm$ 7.72 ^a
FBS (mg/dl)	(10-35)	85.47 $\pm$ 7.41 ^d	94.73 $\pm$ 7.97 ^d	160.1 $\pm$ 8.60 ^c	248.6 $\pm$ 13.6 ^a
	(36-65)	84.78 $\pm$ 13.76 ^d	97.85 $\pm$ 10.0 ^d	180.1 $\pm$ 33.1 ^{bc}	201.6 $\pm$ 25.7 ^b
HOMA-IR	(10-35)	1.96 $\pm$ 0.40 ^{cd}	6.58 $\pm$ 2.20 ^c	2.94 $\pm$ 0.29 ^{cd}	17.98 $\pm$ 2.41 ^a
	(36-65)	1.76 $\pm$ 0.49 ^d	5.47 $\pm$ 1.81 ^{bc}	3.00 $\pm$ 0.37 ^{cd}	13.74 $\pm$ 3.81 ^b
HbA1c mmol/mol	(10-35)	5.16 $\pm$ 0.26 ^c	5.29 $\pm$ 0.51 ^c	8.22 $\pm$ 2.29 ^b	11.16 $\pm$ 0.84 ^a
	(36-65)	5.04 $\pm$ 0.5 ^c	5.44 $\pm$ 0.36 ^c	9.5 $\pm$ 2.86 ^b	8.56 $\pm$ 1.56 ^b

*Different letters mean there is a significant difference between values according to Duncan test.

** The values are means $\pm$ standard deviation (SD)

The results show that there is a metabolic disorder in patients, through the high levels of insulin in groups, which in turn reflects that there is defect in the function of β -cells of pancreas the study results agree with a study for Moon and BIÉU demonstrate that fasting serum insulin testing matters for type 2 diabetes clinical practice (Moon and BIÉU, 2023). The significant increase in insulin levels

in insulin-resistant (IR) and IR and Diabetes is a response to maintain appropriate blood glucose levels. β -cells show signs of wear and tear when insulin resistance occurs for extended periods thus resulting in a decrease of insulin secretion (Lee *et al.*, 2023). Regarding diabetes group, there were no difference in insulin levels in compare to control group. Gerich, (2000) mentioned in his study that insulin resistance is not necessarily an essential component of type 2 diabetes this may be due to β -cell function in type 2 diabetes patients' glucose-tolerant exhibited impairments with normal insulin sensitivity. Which means that β -cell dysfunction represents the principal defect for these patients and it aligns with García-Estevez *et al.*, (2002) who pointed out that the heterogeneity of type 2 diabetes becomes clear because patients can develop the condition through insulin secretion failure.

As for FBS levels a significant increase in patients with diabetes and diabetes with insulin resistance is shown this agrees with Sati *et al.*, (2021) study on insulin resistance in type 2 diabetes mellitus and its effect on FBS levels, their results agreed with the fact that T2DM was considered the syndrome of insulin deficit and resistance, and this in turn will affect the levels of sugar in the blood by which it decreases the ability of insulin to facilitate glucose uptake thereby leading to a high FBS level (Saxena *et al.*, 2024). Failure of FBS to increase substantially in insulin resistance could be accredited to various mechanisms that underlie the condition affecting glucose metabolism and this aligns with the study of Wysham and Shubbrook, (2020) on beta-cell failure in type 2 diabetes.

Mechanisms, markers, and clinical implications, they mentioned that normoglycemia is effectively maintained through compensatory production of an increased amount of insulin in most individuals. Compensatory hyperinsulinemia during early insulin resistance might be the reason for maintaining normal FBS levels in IR patient (Hunt and DeBellis, 2022). Insulin-resistant patients with diabetes showed the highest HOMA-IR levels. HOMA-IR has proven reliable in assessing the severity of insulin resistance according to medical research demonstrates type 2 diabetes mellitus patients possess elevated insulin resistance levels determined through HOMA-IR. Research shows T2DM patients have markedly higher HOMA-IR results than healthy controls according to (Purohit and Tiwari, 2015; Jayagopal *et al.*, 2002). High HOMA-IR values make pancreatic insulin production increase as blood glucose rises which intensifies insulin resistance (Khalili *et al.*, 2023). Regarding to the patient with diabetes only illustrate normal HOMA-IR levels, which supports that β -cell dysfunction stands as the main factor causing their condition, these patients primarily exhibit β -cell dysfunction rather than insulin resistance (García-Estevez *et al.*, 2002). Treatment strategy differentiation demands this understanding because patients with insulin-sensitive diabetes need therapeutic support of their β -cells instead of agents to increase insulin sensitivity (Gerich, 2000).

Regarding HbA1c the elevated levels in diabetes and diabetes with insulin resistance is predicted because this increase is attributed to reduced glucose uptake, based on the idea of fasting blood glucose and glycated hemoglobin A1c (HbA1c) as intended typical assumptions of chronic glycemic control (Yuan *et al.*, 2020). The reason behind this elevation is that over time HbA1c measures a representation of average blood glucose in the ~3 months. Both diabetes and IR are associated with intractable postprandial and fasting hyperglycemia which delivers an increment in the glycation of hemoglobin (Yuan *et al.*, 2020). The process of elevated HbA1c develops when glucose attaches itself to erythrocytic hemoglobin to create stable glycated hemoglobin molecules which exist in blood circulation (Freeman, 2014).

As for HbA1c levels in IR group there was no significant increase in compare to control group insulin resistance and glycemic control research evidence of the research is mixed some researches indicates an incensement in HbA1c levels like Asmasary *et al.*, (2023) study on (HbA1c) Level and HOMA-IR value in obese children. Whereas other study of Nguyen *et al.*, (2023) conducted no significant difference in HbA1c level in IR patients which aligns with our results and they mentioned that the reason behind it is that the rise in insulin resistance, hyperinsulinemia can lead to a reduction in HbA1c due to non-glycemic effects on erythrocytosis. Greater Hb is correlated to reduce HbA1c, Increase in IR was also found to increase Hb and the number of red cells in observational studies as well (Woo and Hawkins, 2014).

As for the effect of age, (Table 2) demonstrate that insulin levels decrease with aging in all groups as it has a negative effect on insulin levels and this decline in insulin levels is due to a reduction in the mass of the islet, as by an age-related functional impairment of the beta cells (Kurauti *et al.*, 2021). Our results align with the study of Aguayo-Mazzucato, (2020) on functional changes in beta cells during ageing and senescence, whom mentioned that the process of aging is linked to decreased β cells mass and defective glucose stimulated insulin secretion. This contributes to reduced insulin in the blood and in reaction to glucose. Also FBS levels showed a significant increase in patients with only diabetes with aging and this is due to a that aging is a major risk factor of type 2 diabetes which modulates the functional capacity of the β cells and glucose homeostasis (Zhu *et al.*, 2021).

And this is in agreement with Yohannes *et al.*, (2021) and their study on the factors affecting FBS levels and aging was one of these factors they recommended that diabetic patients had an increased rate of development of blood glucose level with patients with advanced age. However FBS levels in diabetes and insulin resistance group showed a significant decrease with aging and the reason for this declining in FBS is that in diabetes and insulin resistance patients, fasting blood glucose level might show age dependent decrease because of enhanced basal insulin secretion, compensatory effects, as well as the proliferation of senescence of β cells, making them less effective in release of insulin and control glucose levels with time (Aguayo-Mazzucato, 2020). This findings agreed with what Mittendorfer *et al.*, (2022) found by their study on β cell function the study suggests that a worsening of the β cell functional state, but not the insulin sensitivity state underlies a decline in FBS level, especially, in subjects with obesity and various glycemic conditions. On the other hand, the results also demonstrate that older patients who were insulin-resistant displayed a significant decrease in HOMA-IR levels.

HOMA-IR tends to decrease over time (Kant *et al.*, 2024). The long-term decline of HOMA-IR corresponds with β -cell exhaustion which causes insulin-producing capacity to decrease as insulin resistance persists (Mezza *et al.*, 2019).

Finally the results indicate that there is a significant decrease in HbA1c levels in IR and Diabetes group in compared to younger age group, this results are in agreement with the study of Wang *et al.*, (2010) about elderly patients with elderly-onset type 2 diabetes mellitus which found that elder diabetes patients tend to have lower levels of glycosylated hemoglobin and fasting plasma glucose and fasting insulin compared to those who develop diabetes before reaching older age, as for other aged groups there was no significant difference in advanced age.

Results also revealed a significant increase in the levels of TC, TG, LDL, and VLDL in both IR and Diabetes patients in comparison to the control group. The levels of high-density lipoprotein (HDL) dropped significantly as healthcare participants compare to those found in controls. Patients who had metabolic conditions displayed a significant increase in BMI, as shown in (Table 3) and (Table 4).

Table (3): Effect of lipid profile, BMI in T2DM and insulin resistance

Parameters \ Groups	Control	Patients		
		IR	Diabetes	IRand Diabetes
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD
TC (mg/dl)	156.57 $\pm$ 20.94 ^b	175.61 $\pm$ 34.31 ^{ab}	179.95 $\pm$ 22.13 ^a	186.88 $\pm$ 40.24 ^a
TG (mg/dl)	69.70 $\pm$ 16.00 ^b	143.4 $\pm$ 23.14 ^a	172.3 $\pm$ 46.72 ^a	183.89 $\pm$ 47.83 ^a
HDL (mg/dl)	50.97 $\pm$ 8.77 ^a	39.29 $\pm$ 7.51 ^b	37.55 $\pm$ 6.38 ^b	41.37 $\pm$ 7.72 ^b
LDL (mg/dl)	90.1 $\pm$ 18.17 ^a	103.71 $\pm$ 33.29 ^a	96.76 $\pm$ 17.83 ^a	108.1 $\pm$ 33.55 ^a
VLDL (mg/dl)	15.49 $\pm$ 5.17 ^b	30.34 $\pm$ 5.77 ^a	35.36 $\pm$ 15.88 ^a	35.71 $\pm$ 7.04 ^a
BMI	25.31 $\pm$ 2.7 ^{ab}	31.45 $\pm$ 4.79 ^a	27.66 $\pm$ 2.03 ^{ab}	26.93 $\pm$ 3.55 ^{ab}

*Different letters mean there is a significant difference between values according to Duncan test.

** The values are means $\pm$ standard deviation (SD)

Table (4): Effect of lipid profile and BMI in T2DM and insulin resistance according to age

Parameters	Age(year)	Control Mean $\pm$ SD	patients		
			IR Mean $\pm$ SD	Diabetes Mean $\pm$ SD	IR and Diabetes Mean $\pm$ SD
TC (mg/dl)	(10-35)	148.46 $\pm$ 17.81 ^b	166.7 $\pm$ 34.21 ^{ab}	187.4 $\pm$ 10.26 ^a	163.7 $\pm$ 16.99 ^{ab}
	(36-65)	174.4 $\pm$ 16.02 ^{ab}	186.31 $\pm$ 32.31 ^a	172.5 $\pm$ 29.82 ^{ab}	209.5 $\pm$ 34.83 ^a
TG (mg/dl)	(10-35)	666 $\pm$ 12.5 ^c	153.5 $\pm$ 34.5 ^{ab}	183.05 $\pm$ 10.32 ^a	182.6 $\pm$ 8.91 ^a
	(36-65)	72.8 $\pm$ 9.88 ^c	133.93 $\pm$ 16.3 ^{ab}	161.55 $\pm$ 34.12 ^a	184.15 $\pm$ 31.5 ^a
HDL (mg/dl)	(10-35)	50.63 $\pm$ 9.82 ^a	38.77 $\pm$ 6.23 ^b	36.32 $\pm$ 5.5 ^b	42.38 $\pm$ 2.59 ^b
	(36-65)	51.71 $\pm$ 6.27 ^a	39.92 $\pm$ 8.98 ^b	38.77 $\pm$ 7.79 ^b	41.17 $\pm$ 8.4 ^b
LDL (mg/dl)	(10-35)	83.3 $\pm$ 13.65 ^b	98.8 $\pm$ 20.3 ^b	100.12 $\pm$ 35.67 ^b	82.8 $\pm$ 12.7 ^b
	(36-65)	96.9 $\pm$ 10.33 ^b	107.0 $\pm$ 15.5 ^b	93.41 $\pm$ 2.33 ^b	134.4 $\pm$ 16.25 ^a
VLDL (mg/dl)	(10-35)	14.39 $\pm$ 4.31 ^c	29.67 $\pm$ 7.28 ^{ab}	38.41 $\pm$ 5.92 ^a	32.60 $\pm$ 6.06 ^a
	(36-65)	17.9 $\pm$ 6.28 ^{bc}	30.53 $\pm$ 4.44 ^a	32.31 $\pm$ 11.69 ^a	39.58 $\pm$ 6.74 ^a
BMI	(10-35)	24.41 $\pm$ 2.49 ^c	30.95 $\pm$ 5.01 ^a	27.02 $\pm$ 1.66 ^{bc}	25.60 $\pm$ 2.91 ^{bc}
	(36-65)	26.64 $\pm$ 2.55 ^{bc}	32.05 $\pm$ 4.61 ^a	28.10 $\pm$ 2.11 ^b	28.60 $\pm$ 3.33 ^b

*Different letters mean there is a significant difference between values according to Duncan test.

** The values are means $\pm$ standard deviation (SD)

A significant increase in TC noticed in our result this is due to dyslipidemia which plays its role in insulin resistance by a loop of lipid accretion, oxidative stress with interfering with insulin signaling (Wang and Ahmadizar, 2021). This high concentrations of total cholesterol had a very close relationship with the risk of T2DM and that cholesterol is highly dependent on physical activity and food intake (Ahmad *et al.*, 2017). As for having high triglycerides is due to the fact that in diabetes, insulin resistance will make glucose unutilized in body tissues which will result in excessive levels of blood sugar. Thus, fatty acids in fatty tissues are broken to derive energy, and, hence, excess fat gathers in the liver and gets converted into triglycerides (Sabahelkhier *et al.*, 2016). A decrease in HDL cholesterol is seen ever since diabetic patients and those with insulin resistance developed both conditions their bodies' exhibit markedly diminished levels of HDL-c, which aligns with the study on the link between insulin resistance and dyslipidemia because these conditions lead to future cardiovascular complications (Alidu *et al.*, 2023).

Considering LDL Almwala *et al.*, (2022) as they pointed out the direct impact of T2DM alongside insulin resistance does not affect small dense LDL levels which agreed with our results, the reason for this is that in particular, insulin resistance leads to excessive production of VLDL and hypertriglyceridemia, which, together with the activity of cholesterol ester transfer protein CETP and hepatic lipase reconstitute LDL to produce smaller, denser and more atherogenic particles, but does not increase the overall level of LDL. Regarding VLDL the significant increase in its levels is due to Insulin resistance that comes with increased inflow of free fat acids consequently triggers hepatic VLDL- C production. This causes production of VLDL-C, which is required in the transport of these triglycerides (Hirano, 2018).

These results in agreement with the results obtained by Almwala *et al.*, (2022) and their study on the relationship between insulin hormone and insulin resistance (IR) with Lipid profile in people with diabetes, as they pointed out the direct impact of T2DM alongside insulin resistance does not affect small dense LDL levels but these conditions indirectly cause their rise through a decrease in HDL cholesterol and elevated triglycerides ever since diabetic patients and those with insulin resistance developed both conditions their bodies' exhibit markedly diminished levels of HDL-c, which aligns with the study on the link between insulin resistance and dyslipidemia because these conditions lead to future cardiovascular complications (Alidu *et al.*, 2023). Our results regarding TC, TG and VLDL also aligns with Ahmed and Hatam (2023), as they pointed in their study on adropin, insulin, insulin resistance and lipid profile levels in diabetes mellitus type2 patients that there was a significant increase in total cholesterol, triglycerides and VLDL.

The metabolic path of lipids gets disrupted by insulin resistance because it blocks adipose tissue insulin functions which leads to increased free fatty acid levels in circulation hepatic lipid accumulation together with elevated triglyceride levels intensifies the metabolic issues associated with patients who have IR and diabetes (Papakonstantinou *et al.*, 2022).

As for body mass index BMI results show a significant increase in patients with insulin resistance while BMI remains lower for control subjects. Research findings validate obesity as the main cause of insulin resistance (Al-Juwayr and Al-Hayali, 2025). Through the expansion of visceral fat tissue and the development of ectopic fat accumulation which align with the study of Fazeli *et al.*, (2020) that indicates aging as a powerful risk factor for type 2 diabetes mellitus independent of body mass index. Obesity-related tissue dysfunction in patients results in elevated adipokine secretion including leptin and resisting but decreased adiponectin production. Insulin signaling undergoes impairment because of these changes that enable the development of systemic insulin resistance (Ayman *et al.*, 2019). The BMI measurements throughout different age groups show similar results to metabolic changes in lipid metabolism functions. The lipid profiles of younger age-group IR patients show no significant difference in compared to those of older age-group IR patients according to (Table 4) findings. Research demonstrates that insulin sensitivity decreases together with lipid dysregulation when metabolic flexibility deteriorates because of age despite BMI remaining stable (Kant *et al.*, 2024).

CONCLUSION

The research study revealed that certain type 2 diabetic patients lack insulin resistance yet have diabetes which demonstrates that beta-cell dysfunction serves as the primary diabetes mechanism for these patients. Different management strategies need to be developed specifically for type 2 diabetics with insulin resistance because this subgroup requires distinct treatment approaches from insulin-sensitive patients.

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مقاومة الأنسولين وعلاقته بمرض السكري من النوع الثاني

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الملخص

جمعت 100 عينة وتم تقسيمها الى أربع مجاميع وهي: أفراد أصحاء، ومقاومون للأنسولين، ومصابون بمقاومة الأنسولين والسكري معاً، بالإضافة إلى مرضى السكري الذين لم يعانون من مقاومة الأنسولين من مركز الوفاء للسكري وأمراض الغدد الصماء في الموصل. خضع الأفراد لتصنيف إضافي قائم على العمر لتقييم الأيض في فئتين عمريتين: 10-35 عاماً و 36-65 عاماً. تم تحليل عينات الدم باستخدام اختبارات بيوكيميائية لتقييم مستويات الأنسولين مع إجراء تقييمات لسكر الدم الصائم (FBS)، الهيموغلوبين السكري (HbA1c) ومستوى الدهون. أظهرت النتائج أن المرضى المقاومين للأنسولين ومرضى السكري لديهم مستويات أنسولين أعلى مع FBS و HbA1c مقارنةً بالبالغين الأصحاء، مما يشير إلى أن مقاومة الأنسولين تؤدي إلى مشاكل أيضية. ظلت مستويات الأنسولين لدى الشباب مستقرة، إلا أن خلايا بيتا فقدت وظيفتها بشكل مطرد مع تطور حالتهم الصحية مع التقدم في السن. كما تناولت الدراسة علاقة مقاومة الأنسولين وايض الدهون إذ وجد أن الأشخاص الذين يعانون من مقاومة الأنسولين كانت قيم كل من TC, TG, LDL, VLDL أعلى من القيم التي سجلت لمجموعة السيطرة بينما كانت قيم HDL اقل مقارنة بمجموعة السيطرة. يساعد فحص المرضى باستخدام المؤشرات الحيوية المبكرة في تقييم مقاومة الأنسولين HOMA-IR لدى مرضى داء السكري من النوع الثاني.

الكلمات المفتاحية: مقاومة الأنسولين، داء السكري من النوع الثاني، HOMA-IR، سكر الدم الصائم، HbA1c.