Scholars LITERATURE

Obesity and Diabetes Research

Research Article

DOI: https://doi.org/10.47275/2692-0964-131
Volume 6 Issue 1

Insulin Resistance Assessment in Relation to Obesity Levels Among Iraqi Type 2 Diabetics

Mohammad Abdul Ghafoor Mohammad[1]*, Rafid Abdulwahid Gatea[2] and Mohammed Sami Abdulhasan Al-Gharbawi[2]

[1]Department of Internal Medicine, Al-Sayab Teaching Hospital, Basrah Health Directorate, Basrah, Iraq

[2]Department of Internal Medicine, Basrah Teaching Hospital, Basrah Health Directorate, Basrah, Iraq

Abstract

Diabetes mellitus (DM) represents a metabolic syndrome characterized by dysregulation of carbohydrate metabolism. Obesity is recognized as a principal modifiable risk factor predisposing individuals to type 2 DM (T2DM) and is defined by excessive adiposity. In the obese state, adipose tissue exhibits increased secretion of non-esterified fatty acids, glycerol, hormonal mediators, pro-inflammatory cytokines, and additional bioactive substances that collectively contribute to the pathogenesis of insulin resistance (IR). A robust and well-documented association exists between body mass index (BMI), IR, and the subsequent development of T2DM. The present investigation aimed to elucidate the relationship between degrees of obesity and IR among Iraqi patients diagnosed with T2DM. Specifically, the study sought to quantify the impact of IR relative to obesity severity within this population group. A case-control study design was employed, encompassing 300 participants (163 females and 137 males), aged between 30 and 60 years, over the period extending from July to December 2024 study subjects, including both cases and controls, were stratified into four categories based on BMI: normal weight, overweight, obese, and markedly obese. All participants provided informed consent consistent with World Health Organization ethical guidelines. Biochemical and anthropometric assessments comprised the homeostatic model assessment of IR (HOMA-IR), glycated hemoglobin (HbA1c), fasting blood glucose (FBG), comprehensive lipid profile, and BMI determination. Comparative analysis demonstrated that the case group exhibited significantly elevated levels of FBG, HbA1c, total cholesterol, triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and HOMA-IR compared to the control group ($p = 0.001$). Conversely, no statistically significant difference was observed between groups regarding high-density lipoprotein cholesterol (HDL-C) concentrations. Emerging evidence underscores that incremental increases in BMI are associated with progressive reductions in insulin sensitivity and amplification of IR, ultimately facilitating the onset and progression of T2DM.

Keywords: Diabetes mellitus, Cholesterol, Triglycerides, Insulin resistance, Obesity

Correspondence to: Mohammad Abdul Ghafoor Mohammad, Department of Internal Medicine, Al-Sayab Teaching Hospital, Basrah Health Directorate, Basrah, Iraq.

Citation: Mohammad MAG, Gatea RA, Al-Gharbawi MSA (2025) Insulin Resistance Assessment in Relation to Obesity Levels Among Iraqi Type 2 Diabetics. Obes Diabetes Res, Volume 6:1. 131. DOI: https://doi.org/10.47275/2692-0964-131

Received: June 01, 2025; **Accepted:** July 28, 2025; **Published:** August 01, 2025

Introduction

T2DM is recognized as a chronic metabolic disorder that is fundamentally characterized by persistent hyperglycemia. This hyperglycemic state primarily arises due to an impaired responsiveness of insulin receptors to circulating insulin, a mechanism commonly referred to as IR [1]. In other words, IR represents a pathological condition in which normal physiological concentrations of insulin are insufficient to produce the expected biological responses. Specifically, this dysfunction manifests as an inadequate capacity of cells to uptake and utilize glucose effectively, alongside a failure to appropriately suppress circulating TG levels.

It is important to highlight that the emergence and progression of IR results from a multifaceted interaction between inherent genetic factors and modifiable lifestyle components. Among these, dietary habits and a sedentary lifestyle, characterized by reduced physical activity, play a particularly significant role [2].

In the context of assessing obesity and its relationship to metabolic disturbances, BMI has emerged as a widely accepted and validated proxy indicator of body fatness. Notably, BMI offers a practical alternative to direct methods of body composition measurement. This is largely because the calculation of BMI is both precise and highly accessible, rendering it one of the most reliable approaches for evaluating overweight and obesity across diverse populations. Moreover, the simplicity and low cost of BMI estimation facilitate its widespread utilization by clinicians as well as the general public, given that only height and weight measurements are required [3].

Given this background, the present study was designed to explore the association between BMI and lipid profile parameters in Iraqi individuals diagnosed with T2DM. Understanding this relationship is crucial, as insulin performs essential regulatory functions within metabolic pathways. In particular, insulin inhibits lipolysis in adipose tissue and simultaneously promotes the activity of lipoprotein lipase, an enzyme that plays a central role in lipid metabolism.

Furthermore, in the setting of obesity, the progressive expansion of adipose tissue contributes to the increased mobilization of free fatty acids (FFAs) into systemic circulation. This process occurs through the activation of hormone-sensitive lipase, an enzyme regulated by cyclic AMP-dependent signaling pathways. In addition to this mechanism,

FFAs are also released within peripheral tissues as a result of lipoprotein lipase-mediated hydrolysis of TG-rich lipoproteins [4].

It is also worth noting that the prevalence of obesity, metabolic syndrome, and their associated metabolic derangements is shaped by a wide array of contributing factors. These include genetic susceptibility, age, sex, the quantity and composition of dietary intake, the degree of physical activity, and individual body habitus [5].

To objectively quantify IR in clinical and research settings, the HOMA-IR has been established as a reliable and widely used methodological tool for estimating β-cell function and the degree of IR [6].

Methods

In this study, a total of 300 participants were recruited. This study included 150 patients diagnosed with T2DM, comprising 84 females and 66 males, as well as 150 apparently healthy individuals who served as the control group, consisting of 71 males and 79 females. The age of subjects in both groups ranged from 30 - 72 years.

To facilitate further analysis, all participants-both cases and controls-were stratified based on their body weight into four categories: normal weight, overweight, obese, and markedly obese.

For the biochemical assessments, fasting blood sugar (FBS), HbA1c, total cholesterol, TG, HDL-C, LDL-C, and very-low-density lipoprotein cholesterol (VLDL) levels were measured. These measurements were performed using the COBAS INTEGRA® 400 plus automated biochemistry analyzer (Roche/Hitachi Diagnostics Ltd., Japan). Notably, this analytical system is capable of conducting colorimetric, immunoturbidimetric, and ion-selective assays with high precision and reliability.

IR was estimated to be using the HOMA method. The calculation was performed according to the following equation:

HOMA= {glucose [in mole/L] × insulin [in micro U/mL]}/22.5

Statistical Analysis

Regarding data processing and analysis, the dataset was evaluated utilizing the statistical package for the social sciences, version 30. Descriptive statistics, including frequency distributions, percentages, means, and standard deviations, were generated to summarize and present the data in a clear and interpretable manner.

Results

This study included a total of 300 participants. The baseline demographic and clinical characteristics of the study population are presented in table 1. Upon comparative analysis, the case group exhibited significantly elevated levels of FBS, HbA1c, total cholesterol, TG, LDL-C, and IR relative to the control group ($p < 0.001$). Conversely, no statistically significant difference was observed between the two groups regarding HDL-C concentrations and fasting insulin. These findings are comprehensively summarized in table 1.

The distributions of BMI categories differed markedly between the two groups, especially in marked obesity. Specifically, whereas 49.5% of participants in the control group were classified as obese and 0.0% as markedly obese, a substantially higher proportion of individuals in the case group were affected, with 50.5% categorized as obese and 100% as markedly obese (Table 2).

Normal-weight individuals demonstrated significant differences compared to their counterparts in the control group with respect to FBS, HbA1c, and IR. However, a significant difference in fasting insulin levels was observed between the two groups within this BMI category but with such low standard deviation that make the signification without importance. Unfortunately, in our study there were no participants of overweight in case group. In obese subjects, the case group exhibited significantly higher levels of FBS, HbA1c, fasting insulin, and IR compared to the control group. It seems to be that most cases of marked obesity have diabetes, so that we can't find any marked weighted participant without diabetes to consconsider as a control group. These findings are summarized in table 3.

There was a significant difference in cholesterol, TG, LDL, VLDL and HDL levels between normal-weight participants in the case group and those in the control group. However, small standard deviation can easily give the same wrong signification. Obese participants in the case group exhibited significantly elevated levels of cholesterol, TG, LDL, and VLDL relative to their counterparts in the control group. These findings are summarized in table 4.

Discussion

This investigation revealed that HbA1c concentrations were substantially higher in patients with T2DM compared to healthy

Table 1: The distribution of the study markers according to the study groups.

Variable	Control group (Mean ± SD)	Case group (Mean ± SD)	P value
FBS (mmol/L)	5.3 ± 0.2	9.6 ± 0.7	<0.001
HbA1c	5.1 ± 0.2	8.5 ± 1.3	<0.001
Cholesterol (mg/dl)	228.0 ± 10.5	248.1 ± 21.6	<0.001
TG (mg/dl)	111.7 ± 14.4	207.1 ± 36.2	<0.001
LDL (mg/dl)	99.5 ± 6.6	174.1 ± 15.5	<0.001
HDL (mg/dl)	47.2 ± 4.7	40.6 ± 6.4	-
VLDL (mg/dl)	20.6 ± 2.6	50.8 ± 6.8	<0.001
Fasting insulin (mIU/L)	8.6 ± 0.3	10.0 ± 1.0	-
IR (HOMA-IR)	2.3 ± 0.1	5.3 ± 0.8	<0.001 (High significance)

Table 2: Distribution of BMI according to the study groups.

BMI	Control group N (%)	Case group N (%)	Total N (%)	P value
Normal	51 (50.5%)	50 (49.5%)	101 (33.7%)	<0.0001
Overweight	50 (100.0%)	0 (0.0%)	50 (16.7%)	<0.0001
Obesity	49 (49.5%)	50 (50.5%)	99 (33.0%)	<0.0001
Marked obesity	0 (0.0%)	50 (100.0%)	50 (16.7%)	<0.0001

Table 3: Distribution of FBS, HbA1c, insulin, and IR according to the BMI grade.

BMI (kg/m²)	Study groups	FBS (mmol/L) (Mean ± SD)	HbA1c (Mean ± SD)	Insulin (mIU/L) (Mean ± SD)	HOMA-IR (Mean ± SD)
Normal weight	Case N = 50	8.9 ± 0.1	7.1 ± 0.1	8.8 ± 0.0	4.3 ± 0.1
	Control N = 51	5.0 ± 0.1	4.8 ± 0.1	8.4 ± 0.0	2.2 ± 0.0
	P value	<0.001	<0.001	<0.001	<0.001
Overweight	Case N = 0	-	-	-	-
	Control N = 50	5.6 ± 0.1	5.1 ± 0.1	8.8 ± 0.0	2.3 ± 0.0
	P value	-	-	-	-
Obesity	Case N = 50	-	-	-	-
	Control N = 49	-	-	-	-
	P value	<0.001	<0.001	<0.001	<0.001
Marked obesity	Case N = 50	10.5 ± 0.2	10.2 ± 0.3	11.2 ± 0.2	6.3 ± 0.2
	Control N = 0	-	-	-	-
	P value	-	-	-	-

Table 4: Mean distribution of diabetic patients' lipid profile parameters among BMI groups.

BMI (kg/m²)	Study groups	Cholesterol (mg/dl) (Mean ± SD)	TG (mg/dl) (Mean ± SD)	LDL (mg/dl) (Mean ± SD)	VLDL (mg/dl) (Mean ± SD)	HDL (mg/dl) (Mean ± SD)
Normal weight	Case N = 50	221.2 ± 1.5	176.8 ± 1.7	157.7 ± 1.2	43.6 ± 0.9	49.1 ± 0.2
	Control N = 51	215.4 ± 1.2	92.1 ± 1.4	91.5 ± 0.6	17.2 ± 0.8	52.8 ± 0.8
	P value	<0.001	<0.001	<0.001	<0.001	<0.001
Overweight	Case N = 50	-	-	-	-	-
	Control N = 50	229.0 ± 2.0	118.0 ± 2.5	100.0 ± 1.0	24.0 ± 1.2	48.0 ± 0.9
	P value	-	-	-	-	-
Obesity	Case N = 50	250.0 ± 3.0	190.0 ± 3.5	170.0 ± 2.0	50.0 ± 1.5	42.0 ± 1.0
	Control N = 49	226.0 ± 2.0	130.0 ± 2.5	110.0 ± 1.5	30.0 ± 1.2	45.0 ± 1.0
	P value	<0.001	<0.001	<0.001	<0.001	<0.001
Marked obesity	Case N = 50	275.0 ± 4.0)	260.0 ± 4.5)	195.0 ± 2.5)	60.0 ± 2.0)	34.0 ± 1.2)
	Control N = 0	-	-	-	-	-
	P value	-	-	-	-	-

controls ($p < 0.001$), indicating inadequate glycemic control over the observed period. These findings are consistent with the results reported by Atari-Hajipirloo et al. [7] who demonstrated that HbA1c levels in patients with T2DM were significantly elevated relative to non-diabetic individuals [7].

HbA1c formation is a physiological process reflecting chronic glycemic exposure, making HbA1c the most reliable indicator of long-term glucose regulation. Its levels strongly correlate with the risk of chronic diabetes complications and thus are widely regarded as the test of choice for monitoring and managing DM. Measurement of HbA1c provides an estimate of average blood glucose over the preceding 2 - 3 months, which aligns with the lifespan of circulating red blood cells.

Variability in HbA1c has been linked to the development of macrovascular complications in patients with T2DM, whereas short-term glucose fluctuations were associated with both macrovascular and microvascular disease [8]. The observed elevation in FBG among diabetic patients can be attributed to IR, which impairs cellular glucose uptake [9]. Indeed, IR is a hallmark feature of T2DM and plays a central role in disease progression [10].

Regarding lipid metabolism, TG concentrations were markedly increased in diabetic patients compared to controls, a finding in agreement with studies by Shabana et al. [11]. Dyslipidemia is a well-established component of T2DM, with IR contributing to the aberrant lipid profile by promoting FFAs release, reducing insulin-dependent muscle uptake of fatty acids, and stimulating hepatic lipid synthesis [12].

Consistent with prior research, patients with T2DM exhibited elevated levels of LDL cholesterol and triacylglycerol alongside decreased HDL cholesterol [13]. Wexler et al. [14] further demonstrated a strong positive correlation between HbA1c and lipid parameters including total cholesterol and LDL, corroborating the relationship between glycemic control and dyslipidemia [14].

Other studies have similarly shown significant associations between HbA1c and lipid abnormalities in T2DM patients [13]. The relationship between obesity and T2DM is of considerable importance. Obesity is recognized as a primary contributor to IR, manifesting early in the disease and frequently compensated for by hyperinsulinemia [15].

In the present study, T2DM patients with higher BMI values demonstrated significantly elevated TG and reduced HDL cholesterol compared to individuals with lower BMI. These findings support previous reports indicating that overweight and obesity represent major risk factors for the development of T2DM [16].

Recent changes in lifestyle activities and dietary patterns have noticeably contributed to the rising burden of T2DM in Iraq. This trend reflects observations reported both within the region and globally, highlighting increased BMI as a principal determinant in the progression of T2DM [17, 18]. For instance, Al-Fartosy et al. [19] demonstrated that individuals whose BMI exceeded 27 kg/m² bore a markedly higher relative risk of T2DM compared to those with a BMI closer to 23 kg/m².

Furthermore, accumulating evidence indicates that BMI is inversely related to glucose disposal rates and directly associated with hepatic glucose output in patients with T2DM [20].

Notably, BMI has been recognized as the most powerful predictor

of IR, even among non-obese individuals. Consistent with the findings of Gonzalez-Cantero et al. [21], the present results confirm that elevated BMI corresponds to diminished insulin sensitivity, thereby reinforcing the well-established connection between obesity and impaired insulin action.

Elevated TG concentrations frequently observed in T2DM can be largely attributed to insulin deficiency, which perpetuates hyperglycemia and enhances the mobilization of FFAs from adipose stores. These fatty acids are subsequently delivered to the liver, where they undergo conversion to TG, contributing to increased VLDL production. In addition, hyperinsulinemia has been implicated in the upregulation of hepatic synthesis of TG, LDL, and VLDL particles [22].

It is also vital to note that insulin exerts regulatory control over LDL receptor expression. Chronic insulin deficiency, a hallmark of T2DM, may lead to a reduction in LDL receptor density on hepatocyte surfaces, thereby resulting in elevated circulating LDL cholesterol concentrations [23].

Interestingly, lipid profile parameters tended to be comparatively lower among lean diabetic patients relative to other BMI categories. Nevertheless, this finding contrasts with the report of Al-Fartosy et al. [19], who found no significant differences in lipid profiles among lean diabetic individuals, irrespective of glycemic control status. Taking together, these results further corroborate the established link between excess adiposity and T2DM, echoing prior studies that have consistently associated overweight and obesity with the onset and progression of T2D [24].

Conclusion

This study demonstrates a clear and statistically significant association between increasing degrees of obesity and heightened IR among Iraqi patients with T2DM. As BMI increased, markers of IR-such as HOMA-IR, FBG, and HbA1c also rose, underscoring the pivotal role of obesity in the pathophysiology of T2DM. These findings reinforce the importance of early intervention strategies targeting weight reduction and metabolic control as critical components in the prevention and management of T2DM. Public health efforts should prioritize obesity prevention and treatment to mitigate the growing burden of IR and related metabolic disorders in the Iraqi population.

Acknowledgements

None.

Conflict of Interest

None.

References

1. Nascè A, Gariani K, Jornayvaz FR, Szanto I (2022). NADPH oxidases connecting fatty liver disease, insulin resistance and type 2 diabetes: current knowledge and therapeutic outlook. Antioxidants 11: 1131. https://doi.org/10.3390/antiox11061131
2. Rasmussen L, Knorr S, Antoniussen CS, Bruun JM, Ovesen PG, et al. (2021) The impact of lifestyle, diet and physical activity on epigenetic changes in the offspring-a systematic review. Nutrients 13: 2821. https://doi.org/10.3390/nu13082821
3. Kullo IJ, Lewis CM, Inouye M, Martin AR, Ripatti S, et al. (2022) Polygenic scores in biomedical research. Nat Rev Genet 23: 524-532. https://doi.org/10.1038/s41576-022-00470-z
4. Li Y, Li Z, Ngandiri DA, Perez ML, Wolf A, et al. (2022) The molecular brakes of adipose tissue lipolysis. Front Physiol 13: 1-19. https://doi.org/10.3389/fphys.2022.826314
5. Harada MD (2022) The role of health and diet in the development of metabolic syndrome stratified by race, sex, and age. Walden University. (Doctoral Dissertation)
6. Tung Y-T, Pan C-H, Chien Y-W, Huang H-Y (2020) Edible mushrooms: novel medicinal agents to combat metabolic syndrome and associated diseases. Curr Pharm Des 26: 4970-4981. https://doi.org/10.2174/1381612826666200831151316
7. Atari-Hajipirloo S, Valizadeh N, Khadem-Ansari M-H, Rasmi Y, Kheradmand F (2016) Altered concentrations of copper, zinc, and iron are associated with increased levels of glycated hemoglobin in patients with type 2 diabetes mellitus and their first-degree relatives. Int J Endocrinol Metab 14: e33273. https://doi.org/10.5812/ijem.33273
8. Mulani MR, Gawali PS (2016) Effect of iron deficiency on glycation of hemoglobin in non diabetics. Annals Clin Chem Lab Med 2: 4–7. https://doi.org/10.3126/acclm.v2i2.14824
9. Prasad M, Jayaraman S, Eladl MA, El-sherbiny M, Abdelrahman MAE, et al. (2022) A comprehensive review on therapeutic perspectives of phytosterols in insulin resistance: a mechanistic approach. Molecules 27: 1595. https://doi.org/10.3390/molecules27051595
10. Hashim HA, Al-Shammaa NMJ (2022) Study of irisin level hormone and some biochemical parameters in Iraqi diabetic type 2 without hypothyroidism. Pak J Med Health Sci 16: 403. https://doi.org/10.53350/pjmhs22166403
11. Shabana, Shahid SU, Sarwar S (2020) The abnormal lipid profile in obesity and coronary heart disease (CHD) in Pakistani subjects. Lipids Health Dis 19: 1–7. https://doi.org/10.1186/s12944-020-01248-0
12. Meex RCR, Blaak EE, van Loon LJC (2019) Lipotoxicity plays a key role in the development of both insulin resistance and muscle atrophy in patients with type 2 diabetes. Obes Rev 20: 1205–1217. https://doi.org/10.1111/obr.12862
13. Alzahrani SH, Baig M, Aashi MM, Al-Shaibi FK, Alqarni DA, et al. (2019) Association between glycated hemoglobin (HbA1c) and the lipid profile in patients with type 2 diabetes mellitus at a tertiary care hospital: a retrospective study. Diabetes Metab Syndr Obes 12: 1639-1644. https://doi.org/10.2147/dmso.s222271
14. Wexler DJ, Grant RW, Meigs JB, Nathan DM, Cagliero E (2005) Sex disparities in treatment of cardiac risk factors in patients with type 2 diabetes. Diabetes Care 28: 514–520. https://doi.org/10.2337/diacare.28.3.514
15. Deeb A (2017) Insulin resistance, impaired fasting, glucose intolerance and type II diabetes mellitus in overweight and obese children in Abu Dhabi. J Di Obes 4: 1-8. https://doi.org/10.15436/2376-0494.17.1389
16. Barb D, Repetto EM, Stokes ME, Shankar SS, Cusi K (2021) Type 2 diabetes mellitus increases the risk of hepatic fibrosis in individuals with obesity and nonalcoholic fatty liver disease. Obesity 29: 1950–1960. https://doi.org/10.1002/oby.23263
17. Abid SM, Alaaraji SF, Alrawi KF (2019). Study of Interleukins 13, 18, 27, 32 and 38 levels in Iraqi type 2 diabetes with insulin resistance. J Educ Sci Stud 4: 14.
18. Al-Bachir M, Bakir MA (2017). Predictive value of body mass index to metabolic syndrome risk factors in Syrian adolescents. J Med Case Rep 11: 1–7. https://doi.org/10.1186/s13256-017-1315-2
19. Al-Fartosy AJM, Awad NA, Abdalemam DJ (2017). Biochemical study of the effect of insulin resistance on adiponectin, lipid profile and some antioxidants elements with relation to obesity in type 2 diabetic patients/Basrah-Iraq. Am J Biochem 7: 73–82.
20. Taniguchi A, Nakai Y, Sakai M, Yoshii S, Hamanaka D, et al. (2002). Relationship of regional adiposity to insulin resistance and serum triglyceride levels in nonobese Japanese type 2 diabetic patients. Metab Exp 51: 544-548. https://doi.org/10.1053/meta.2002.31984
21. Gonzalez-Cantero J, Martin-Rodriguez JL, Gonzalez-Cantero A, Arrebola JP, Gonzalez-Calvin JL (2018). Insulin resistance in lean and overweight non-diabetic Caucasian adults: Study of its relationship with liver triglyceride content, waist circumference and BMI. PLoS One 13: 1-13. https://doi.org/10.1371/journal.pone.0192663
22. Zhang J, Yu H, Zhong H, Wang Q, Tang J, et al. (2022). Dietary emulsifier glycerol monodecanoate affects the gut microbiota contributing to regulating lipid metabolism, insulin sensitivity and inflammation. Food Funct 13: 8804-8817. https://doi.org/10.1039/d2fo01689c
23. Majid A, Sayer SA, Farhood HB (2018). Study of some biochemical parameters for patients with type II diabetes mellitus in Thi-Qar Governorate, Iraq. J Pharm Sci Res 10: 2938-2941.
24. Klein S, Gastaldelli A, Yki-Järvinen H, Scherer PE (2022). Why does obesity cause diabetes? Cell Metab 34: 11–20. https://doi.org/10.1016/j.cmet.2021.12.012