

Evaluation of Serum Triglycerides as a Laboratory Biomarker of Dyslipidemia and Insulin Resistance in Patients with Type 2 Diabetes Mellitus

Hawraa Yousif Abed, Asaad Abbas Khalaf*, Fatimah Ali Kareem

Basic Sciences Branch, College of Nursing, University of Kerbala, Kerbala, Iraq.

ABSTRACT

Triglycerides are regarded as a primary contributor to metabolic syndrome and are being investigated as potential biomarkers for dyslipidemia that increase the risk of insulin resistance (IR). This study aimed to evaluate the reliability of triglyceride levels in diagnosing dyslipidemia and their correlation with IR in patients with type 2 diabetes mellitus (T2DM). The findings demonstrated a positive correlation between triglycerides and IR, as well as with other clinical and biochemical parameters. Linear regression analysis revealed that the risk of developing IR increased with advancing age, higher body mass index, longer disease duration, elevated total cholesterol, triglycerides, low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and HbA1c levels. Multiple logistic regression further showed that the risk of T2DM was higher in elderly individuals (≥ 60 years), followed by those aged 50–59 years, obese patients compared to normal-weight individuals, and those with longer duration (≥ 16 years). Elevated total cholesterol (≥ 240 mg/dL), triglycerides (200–499 mg/dL), and LDL (160–189 mg/dL) also contributed to higher risk, whereas increased high-density lipoprotein (≥ 60 mg/dL) was protective. In conclusion, elevated triglyceride levels are strongly indicative of dyslipidemia, and their association with insulin resistance highlights their role in obesity-, diabetes-, and cardiovascular-related complications. Emphasizing triglyceride monitoring in clinical practice is essential for better risk stratification and preventive healthcare strategies.

Keywords: Dyslipidemia, Insulin Resistance, Lipid Profile, Triglycerides, Type 2 Diabetes

INTRODUCTION

High blood sugar levels are a hallmark of diabetes, a metabolic syndrome caused by inadequate regulation over an extended period of time, either as a result of resistance to the action of insulin in target tissues or abnormalities in the secretion of insulin by pancreatic β -cells[1]. In addition to its role in glucose regulation, insulin also influences protein and fat metabolism, as well as the transport of amino acids and ions. Hyperlipidemia in diabetic patients has been proposed as a major contributing factor to the development of diabetic complications, particularly cardiovascular disease. Any abnormalities in fatty acid metabolism can negatively affect insulin receptors, glucose transporters, intracellular signaling, and the fluidity and permeability of cell membranes[2].

Cholesterol is a naturally occurring lipophilic molecule in the body, present in blood circulation together with other lipids. It plays a fundamental role in maintaining the structural composition of cell membranes, regulating fluidity, and contributing to the synthesis of steroid hormones, including testosterone, progesterone, and estrogen, as well as adrenal corticosteroids like aldosterone and cortisol[3]. Cholesterol is also essential for the synthesis of bile salts that aid in the absorption of fat-soluble vitamins. In the blood, cholesterol is transported within lipoprotein complexes composed of cholesterol esters, triglycerides, phospholipids, and free cholesterol. Based on their density and composition, three primary classes of lipoproteins are recognized: high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL)[4]. HDL, which accounts for approximately 25–30% of the proteins in circulation, is commonly referred to as “good cholesterol.” It functions as a key transporter of excess cholesterol, carrying it to the liver for excretion and thereby preventing the accumulation of atherosclerotic plaques in

arterial walls[5]. HDL also exerts anti-inflammatory and antioxidant properties, protecting endothelial function and reducing oxidative stress. Structurally, HDL consists of a core of cholesterol esters and triglycerides surrounded by phospholipids and proteins, particularly apolipoproteins ApoA-I, ApoA-II, and ApoC, which regulate HDL metabolism and receptor interactions[6]. The higher the protein-to-lipid ratio, the denser the lipoprotein particle, explaining HDL's characteristic high density. HDL also plays a critical role in reverse cholesterol transport and neutralization of oxidized lipids, mitigating the pro-atherogenic effects of LDL[7]. In contrast, LDL, or “bad cholesterol,” is the primary contributor to the development of atherosclerotic and cerebrovascular diseases. LDL particles transport cholesterol to peripheral tissues, but excess levels lead to arterial plaque formation, inflammation, and vascular obstruction[8]. LDL is composed mainly of triglycerides and cholesterol esters, surrounded by phospholipids and apolipoprotein B-100 (ApoB-100), a key determinant of LDL's metabolic activity. VLDL, synthesized in the liver, serves as a precursor of LDL and is strongly associated with triglyceride transport[9].

Triglycerides are the main form of stored fat in the body, serving as a critical source of energy. They consist of a glycerol backbone bound to three fatty acid chains of varying lengths and saturation levels. These variations influence multiple physiological processes, including energy production and lipid metabolism[10]. Triglyceride-rich adipose tissue provides mechanical protection to vital organs and serves as an insulator, maintaining thermal homeostasis under different environmental conditions. In addition to their structural role, triglycerides are vital in the transport of fat-soluble vitamins (A, D, E, and K) and essential fatty acids throughout the body. When dietary

energy exceeds immediate requirements, excess calories are converted into triglycerides and stored in adipose tissue[11]. During energy demand, triglycerides undergo lipolysis, releasing fatty acids for oxidation, which provides energy for cellular processes. These activities are tightly regulated by hormonal signals, including insulin, glucagon, and adrenaline, maintaining systemic energy balance[12]. Despite the well-established roles of cholesterol, HDL, LDL, and triglycerides in human physiology and pathology, their precise utility as reliable biomarkers in type 2 diabetes mellitus (T2DM) remains a subject of continued investigation[13]. In particular, triglycerides have been increasingly linked to insulin resistance, a central component of T2DM pathogenesis, yet their predictive value is still under debate[14]. Therefore, this study aims to evaluate triglyceride levels as a biomarker of dyslipidemia and their association with insulin resistance in patients with T2DM, thereby clarifying their potential diagnostic and prognostic significance in clinical practice [15-18].

MATERIALS AND METHODS

Seventy male and female patients with type 2 diabetes mellitus who visited outpatient clinics were included in the study. However, as a control group, 18 healthy people of both sexes were included. Once the patients gave their consent and provided information such as their name, age, length of diabetes, family history of the disease, and the medication they were taking to control their blood sugar levels, the samples were taken. Blood pressure, height, and weight were then measured. If necessary, the patients were examined for other illnesses. Patients with heart conditions, high blood pressure, insulin injections, thyroid conditions, and type 1 diabetes were not included. Using sterile medical syringes, five milliliters of venous blood were extracted from both patients and healthy individuals following a 12-hour fast. To do a hemoglobin HbA1c test, 1 milliliter of blood was put into EDTA tub anticoagulant tubes. A Jel container was then filled with 4 milliliters of blood. The samples were centrifuged at 3000 rpm for 15 minutes after the blood coagulated at room temperature. The serum was then separated using a micro pipette in order to perform the serological tests that were the subject of the investigation. The patients were categorized into five age groups: 30–39 years, 40–49 years, 50–59 years, and ≥60 years. They were also categorized by the length of time they had diabetes: 1–5 years, 6–10 years, 11–15 years, and ≥16 years. Additionally, their BMI was classified as normal (20–24.9 kg/m²),

overweight (25–29.9 kg/m²), and obese (≥30 kg/m²) [19]. Additionally, three groups of total cholesterol (TC) were created: normal (<200 mg/dl), high (200–239 mg/dl), and very high (>240 mg/dl)[20]. Additionally, blood fats were separated as follows: LDL is divided into five groups, and HDL is divided into three groups: low (<40 mg/dl), normal (40–59 mg/dl), and high (≥60 mg/dl). minimal level (<100 mg/dl), near ideal (100–129 mg/dl), somewhat elevated (130–159 mg/dl), elevated (160–189 mg/dl), and extremely elevated (≥190 mg/dl)[21]. Very low-density lipoprotein (VLDL) often has a proportion of less than 30 mg/dl. Triglycerides (TG) were separated into four groups: normal (less than 150 mg/dl), marginally elevated (between 150 and 199 mg/dl), high (≥200 mg/dl), and extremely high (more than 500 mg/dl)[22]. According to the working method included with the kit, the Spanish company Croma test Linear provided a kit for estimating the level of total cholesterol and high-density triglycerides, while Biolab, a French company, provided a kit for estimating triglycerides using the Roche Cobas device. By deducting the value of total cholesterol from the value of high-density lipoprotein, the lipid level was also determined and proved to be detrimental to the body. IBM SPSS 26 was used to statistically analyses all of the study's data (SPSS Inc., Chicago, IL). Frequencies and percentages from the normal data were also analyzed using the chi-square test. Additionally, the normal distribution was examined using the Kolmogorov-Smirnov test. The arithmetic mean and standard error were used to report the numerical data. The T test was used to compare two groups, while the ANOVA test was utilized to compare groups. Logistic regression and linear nominal regression analysis were used to forecast the rise in hazards.

Results

Comparison of Demographic Variables between Patients with Type 2 Diabetes and Healthy Individuals

The results in Table (1) indicated that there were no significant differences ($p = 0.099$) between the age groups(year) of the patient with type 2diabetes. They were 70 individuals, 32 men and 38 women (48.00 ± 1.00). The healthy group consisted of 18 individuals, 11 women and 7 men were chosen as a control group (41.00 ± 4.00), while significant increase appeared between the patient and the control groups ($p < 0.001$), 27.25 ± 0.65 22.95 ± 0.85 . For body mass index was (BMI kg/m²).

Table 1: Comparison of Demographic Variables between Patients with Type 2 Diabetes and Healthy Individuals.

Variables	Categories	Patients N= 70	Control N=18	P-Value
Sex	Men	32(45.7%)	11(61.1%)	0.099
	Women	38(54.3%)	7(38.9%)	
Age (Year)	Mean ± SE	48.00± 1.00	41.0±04.00	0.099
	30-39	15(21.4%)	10(55.6%)	
	40-49	26(37.1%)	2(11.1%)	
	50-59	14(20.0%)	2(11.1%)	
	≥ 60	15(21.4%)	4(22.2%)	
Duration (Year)	Mean ± SE	9.00 ± 1.00	0.00±0.00	NA
	1-5 year	22(31.4%)	0(0.0%)	
	6-10 year	22(31.4%)	0(0.0%)	
	11-15 year	16(22.9%)	0(0.0%)	
	≥ 16 years	10(14.3%)	0(0.0%)	
BMI kg/m ²	Mean ± SE	27.25 ± 0.65	22.95±0.85	<0.001*
	Normal weight	26(37.1%)	15(83.3%)	
	Overweight	19(27.1%)	2(11.1%)	
	Obesity	25(35.7%)	1(5.6%)	

X²: Chi-Square test; significant differences at p-value, * <0.05, **<0.01.

The Biochemical Characteristics of Healthy Individuals and Patients with Type 2 Diabetes are Compared

There were no significant differences between the two groups for HDL (mg/dl). But Table (2)'s analysis of the biochemical variables between the patients and control groups reveals a significant increase. ($P < 0.001$) in T.CH (mg/dl), TG (mg/dl), VLDL (mg/dl), LDL (mg/dl), F. B. SU. (mg/dl), HbA1C %, and HOMA IR sensitivity.

Correlations between Insulin Resistance and All Study Variables

The results in Table 3 indicate association between insulin resistance (IR) and all parameters in this study. A significant effect

($P = 0.041$) of age on insulin resistance was indicated in patients. There was a significant correlation ($p = 0.0001$) in BMI kg/m² (R.0.567), T.CH mg/dL (R.0.616), TG mg/dL (R.0.769), VLDL mg/dL (R. 0.570) with insulin resistance.

There is a negative relationship (R: -0.042) between HDL mg/dl and IR, that when the level of HDL in patients' blood increases, IR decreases; however, it was not significant ($p = 0.732$). Further, a significant increase ($p < 0.05$) was shown in F.B.SU. (mg/dL) (R.0.307), HbA1C% (R.0.271). Nevertheless, there is a negative relationship (R: -0.894; $p = 0.0001$) between insulin sensitivity and resistance in patients with type 2 diabetes.

Table 2: Comparison of Biochemical Variables between Patients with Type 2 Diabetes and Healthy Individuals.

Variables	Categories	Patients N= 70	Control N=18	P-Value
T.CH (mg/dl)	Mean $\pm$ SE	214.50 $\pm$ 4.60	136.10 $\pm$ 4.40	<0.001*
	< 200 mg/dl	26 (37.1%)	18 (100.0%)	
	200-239 mg/dl	25 (35.7%)	0 (0.0%)	
	$\geq$ 240 mg/dl	19 (27.1%)	0 (0.0%)	
TG (mg/dl)	Mean $\pm$ SE	269.60 $\pm$ 8.60	122.70 $\pm$ 9.60	<0.001*
	< 150 mg/dl	0 (0.0%)	15 (83.3%)	
	150-199 mg/dl	5 (7.1%)	3 (16.7%)	
	200-499 mg/dl	65 (92.9%)	0 (0.0%)	
VLDL (mg/dl)	Mean $\pm$ SE	48.78 $\pm$ 2.32	25.7 $\pm$ 92.42	<0.001*
	< 30 mg/dl	11 (15.7%)	14 (77.8%)	
	$\geq$ 30 mg/dl	59 (84.3%)	4 (22.2%)	
LDL (mg/dl)	Mean $\pm$ SE	113.07 $\pm$ 4.05	72.04 $\pm$ 3.48	<0.001*
	< 100 mg/dl	25 (35.7%)		
	100-129 mg/dl	23 (32.9%)		
	129-159 mg/dl	15 (21.4%)		
HDL (mg/dl)	Mean $\pm$ SE	50.00 $\pm$ 2.00	43.80 $\pm$ 2.90	0.142
	> 40 mg/dl	20 (28.6%)	7 (38.9%)	
	40-59 mg/dl	34 (48.6%)	10 (55.6%)	
	$\geq$ 60 mg/dl	16 (22.9%)	1 (5.6%)	
F. B. SU. (mg/dl)	Mean $\pm$ SE	182.92 $\pm$ 7.04	71.45 $\pm$ 2.64	<0.001*
HbA1C %	Mean $\pm$ SE	7.81 $\pm$ 0.21	4.9 $\pm$ 30.08	<0.001*
HOMA IR	Mean $\pm$ SE	3.11 $\pm$ 0.13	1.70 $\pm$ 0.03	<0.001*
Sensitivity	Mean $\pm$ SE	35.00 $\pm$ 1.10	50.4 $\pm$ 02.30	<0.001*

X²: Chi-Square test; significant differences at p-value, * <0.05, ** <0.01.

Table 3: Correlations between Insulin Resistance and All Study Variables

Variables	HOMA IR R	P-Value
Age (year)	0.269*	0.041
BMI kg/m ²	0.567**	0.0001
Duration	0.318**	0.007
T.CH (mg/dl)	0.616**	0.0001
TG (mg/dl)	0.769**	0.0001
VLDL (mg/dl)	0.570**	0.0001
LDL (mg/dl)	0.396**	0.001
HDL (mg/dl)	-0.042	0.732
F. B. SU. (mg/dl)	0.307**	0.010
HbA1C %	0.271*	0.023
Sensitivity	-0.894**	0.0001

Disturbances at the P-value, * <0.05, ** <0.01.

Predicting the Independent Risk Factors Linked to Insulin Resistance Using Linear Regression

From Table (4) it can be predicted that insulin has a significant relationship ($p < 0.05$) with age and body mass index in diabetic

patients, and whenever age exceeds ($B = 0.022$) the resistance increases one unit. Likewise, the body mass index ($B = 0.194$) drives the main stream of insulin action. The duration of disease was highly predictive ($p = 0.0001$; $B = 0.104$) in increasing the number of diabetic patients.

Table 4: The Independent Risk Factors (Demographic Variables) Linked to Insulin Resistance are Predicted Using Linear Regression.

Variables	B	SE	p-value	95.0% CI
Age (year)	0.022	0.011	0.046 *	0.044-0.0001
BMI (kg/m ²)	0.194	0.033	0.0001 **	0.128-0.260
Duration (Year)	0.104	0.034	0.003 **	0.037-0.171

B: Unstandardized Coefficients (effect size). SE: standard error. CI: Confidence Interval.

Significant differences at p-value * <0.05, ** < 0.001. Dependent Variable: HOMA IR

The results of Table (5) show that the lipid profile parameters T.CH(B.0.006;P=0.297),LDL(B.0.002;P=0.737),VLDL(B.0.003;P=0.697) are markers with a statistically significant predictive value of increasing insulin resistance, It also appeared that TG (B.0.009;

P=0.0001) has an important diagnostic relationship in increasing insulin resistance in patients with type 2 diabetes, while HDL level did not have (B.-0.003 ;P=0.693) significant effect on increasing insulin resistance.

Table 5: Linear Regression to Predict the Independent Risk Factors (Lipid Profile Variables) Associated with the Insulin Resistance.

Variables	B	SE	P-value	95.0% CI
T.CH (mg/dl)	0.006	0.006	0.029 *	0.006-0.029
TG (mg/dl)	0.009	0.001	0.0001 **	0.006-0.012
LDL (mg/dl)	0.007	0.006	0.037 *	0.001-0.041
VLDL (mg/dl)	0.006	0.008	0.049 *	0.003-0.019
HDL (mg/dl)	-0.003	0.009	0.693	-0.003-0.014

B: Effect size, or unstandardized coefficients. SE stands for standard error. Confidence Interval, or CI
Significant differences at p-value **<0.001. Dependent Variable: HOMA IR.

It is clear from Table (6) that increasing the value of the fasting blood sugar test gives a non-significant predictive relationship (p > 0.05) in increasing insulin resistance in diabetics. Also, the negative predictive relationship of insulin sensitivity was non-significant (p=888; R.-0.102)

With insulin resistance, and this indicates that it is a weak predictive indicator. On the other hand, it can be predicted that the amount of resistance in the body will increase whenever the value of the cumulative blood sugar test increases by (B.0.123) at (P=0.028) in diabetic patients.

Table 6: Linear Regression to Predict the Independent Risk Factors (Blood Variables) Associated with the Insulin Resistance.

Variables	B	SE	P-Value	95.0% CI
F. B. SU. (mg/dl)	0.002	0.002	0.094	0.005-0.001
HbA1C %	0.123	0.048	0.028 *	0.027-0.220
Sensitivity	-0.102	0.006	-0.888	-0.114--0.089

B: Non-standard coefficients (effect size). SE: standardized error. Confirmation Interval (CI).
Dependent Variable: Significant differences at p-value* <0.05 IR HOMA

Multiple Logistic Regression Tabblies

The logistic regression Table (7) showed that the increased risk of developing diabetes is higher among older people within the age group ≥ 60 (OR.1.733, P = 0.05), While there was no significant predictive effect for the age group 50-59 (OR = 1.000), and less than that in the

age group 40-49 (OR = 0.933, P = 0.853) compared to the reference group (30-39). The regression table (8) indicates that the prediction of an increased risk of diabetes for males (OR = 0.842, P = 0.474) was not significant compared to females, which was considered a reference category.

Table 7: To Forecast which Age Groups' Risk Variables Are Linked to Type 2 Diabetes, Multiple Logistic Regression Analysis is Used.

Variables	B	SE	P-Value	OR	95.0% CI
Age Groups ^b	40-49	0.069	0.372	0.853	0.451-1.934
	50-59	0.0001	0.365	0.100	0.489-2.046
	≥ 60	0.550	0.324	0.050	0.918-3.272

B: Unstandardized Coefficients (Effect Size). SE: Standard Error. CI: Confidence Interval for OR.
The reference category is: 30-39.

Table 8: The Categories of Risk Factors for Sex Groups Linked to Type 2 Diabetes are Predicted Using Multiple Logistic Regression Analysis.

Variables	B	SE	P-Value	OR	95.0% CI
Sex ^b	Men	0.172	0.240	0.474	0.526 -1.348

B: Unstandardized Coefficients (effect size). SE: standard error. CI: Confidence Interval for OR.
b: The reference category is: Women

Table (9) indicates that obesity (OR = 0.562) is considered an indicator with a significant value P = 0.059 for the possibility of exposure to the risk of developing diabetes, but as for overweight (OR

= 0.731), the result was not significant when compared with the reference category normal weight. Significance was not significant when compared with the reference category Normal weight.

Table 9: Multiple Logistic Regression Analysis to Predict the Categories of Risk Factors of Body Mass Index Groups Associated with Type 2 Diabetes.

Variables	B	SE	P-Value	OR	95.0% CI
BMI Groups ^b	Overweight	0.314	0.302	0.299	0.404 -1.320
	Obesity	0.039	0.280	0.059	0.555 -1.665

B: Unstandardized Coefficients (effect size). SE: Standard Error. CI: Confidence Interval for OR.
The reference category is: Normal weight.

It is shown in Table (10) that the duration of infection has an effect on predicting the risk of developing diabetes. When the duration is longer, the effect becomes stronger. Hence, in the period ≥16 the

value was (OR = 1.000), and less than that in the period 11-15 (OR = 0.727), then the period 6-10 (OR=0.455) compared to the reference category 1-5 years.

Table 10: Multiple Logistic Regression Analysis to Predict the Categories of Risk Factors of Duration Groups Associated with Type 2 Diabetes

Variables	B	SE	P-Value	OR	95.0% CI	
Duration Groups ^b	6-10	0.788	0.381	1.000	0.455	0.215-0.960
	11-15	0.318	0.329	0.332	0.727	0.382 -1.385
	≥16	0.0001	0.302	0.039	1.000	0.554 -1.806

B: Unstandardized Coefficients (Effect Size). SE: Standard Error. CI: Confidence Interval for OR.

The reference category is: 1-5.

The results of the statistical analysis of Table (11) indicates that the risk of developing type 2 diabetes does not show any significance for the level of the overall effect. Thus, there was no significant value

200-239: P = 0.299; ≥ 240 mg L: P = 0.889. However, the most influential category is ≥ 240 mg L (OR = 0.962), not allowing the reference category < 200 mg L.

Table 11: To Forecast the Types of Risk Variables of Duration Groups Linked to Type 2 Diabetes, Multiple Logistic Regression Analysis is Used.

Variables	B	SE	P-Value	OR	95.0% CI
T.Ch.	200-239 mg/dl	0.314	0.302	0.731	.404 -1.320
Groups ^b	≥ 240 mg/dl	0.039	0.280	0.889	0.555 -1.6650

B: Unstandardized Coefficients (Effect Size). SE: Standard Error. CI: Confidence Interval for OR.

The reference category is: < 200 mg/dl.

The logistic regression of Table (12) confirms that a high level of triglycerides has a high predictive effect, P = 0.0001, on the risk of developing type 2 diabetes. Thus, the level of triglycerides within the

category 200-499 mg/dl (OR = 13,000) is 13 times more predictive than the reference category. 150-199 mg/dl in predicting the risk of infection.

Table 12: Multiple Logistic Regression Analysis to Predict the Categories of Risk Factors of Triglyceride Groups Associated with Type 2 Diabetes.

Variables		B	SE	P-Value	OR	95.0% CI
TG groups ^b	200-499 mg/dl	2.565	0.464	0.0001	13.000	5.235-32.284

B: Unstandardized Coefficients (effect size). SE: Standard Error. CI: Confidence Interval for OR.

The reference category is: 150-199 mg/dl

It is clear from the results of Table (13) that a high level of low-density lipoprotein within the category 160-189 mg/dl (OR=0.920) has a high predictive value for the risk of developing type 2 diabetes, P=0.003.

Meanwhile, the category 130-159 mg/dl (OR=0.600) is significant in prediction, and less appeared in the category 100-129 mg/dl (OR = 0.280) compared to the reference category < 100 mg/dl.

Table 13: Using Multiple Logistic Regression Analysis, the Risk Variables for Low Density Lipoprotein Groups Linked to Type 2 Diabetes can be Predicted.

Variables	B	SE	P-Value	OR	95.0% CI	
LDL. Groups ^b	100-129 mg/dl	1.273	0.428	0.773	0.280	0.121 -0.647
	130-159 mg/dl	0.511	0.327	0.118	0.600	0.316 -1.138
	160-189 mg/dl	0.083	0.289	0.003	0.920	0.522 -1.621

B: Coefficients without standardization (effect size). SE: standard error. OR Confidence Interval (CI).

The reference category is: < 100 mg/dl.

Regression analysis in Table (14) indicates that there is a significant negative relationship (P=0.05) between good cholesterol values and the risk of developing diabetes. Hence, the level of good

cholesterol increased within the category ≥ 60 mg/dl (OR=1.700), followed by 40-59 mg/dl. (OR = 0.800) compared to the reference category <40 mg/dl, where the risk of developing disease decreased.

Table 14: Using Multiple Logistic Regression Analysis to Predict the Risk Factor Categories of High-Density Lipoprotein Groups Associated with Type 2 Diabetes.

Variables	B	SE	p-value	OR	95.0% CI	
HDL.groups ^b	40-59 mg/dl	-0.531	0.335	0.056	0.800	0.415 -1.544
	≥ 60 mg/dl	-0.223	0.282	0.050	1.700	0.979 -2.953

B: Unstandardized Coefficients (effect size). SE: Standard Error. CI: Confidence Interval for OR. The reference category is: < 40 mg/dl.

Discussion

In the current study, comparing the demographic data of the sick group and the healthy control group revealed no statistically significant changes in the age distribution. One possible explanation for this result is the deliberate selection of control participants' ages, which was done to minimize age-related confounding variables and enhance the precision of the comparative analysis.

However, the body mass index (BMI) of each group varied significantly. Because it is hard to identify volunteers who are not fat and do not also have metabolic issues, the control group was mainly composed of people who were normal weight or overweight. Common examples of these effects include dyslipidemia, cardiovascular disorders, chronic pain, insulin resistance, and hypertension. Importantly, the observed variations in BMI draw attention to the

potential connection between weight gain, increased insulin resistance, and the etiology of diabetes mellitus. These variations are particularly apparent in older age groups. The results of this examination also revealed significant changes in all of the biochemical variables between the two sick and healthy groups, with the exception of high-density lipoprotein, which did not clearly differ between the two research groups. In the diabetic patients, the higher lipid profile values were consistent with the findings of [23], with the exception of HDL. These results were consistent with those of [24], which concluded that persons with diabetes mellitus 2 who had elevated total cholesterol levels also have elevated blood levels of both LDL and VLDL. As a result, the liver slows down the removal of VLDL and LDL from the bloodstream by producing more VLDL. LDL transports cholesterol to all other tissues from the liver, which is the body's main source. As a result, cells—especially those that need cholesterol—have LDL receptors on their surface, allowing them to

take up cholesterol and use it to make membranes or perform other vital functions. LDL is an effective carrier of cholesterol, but high cholesterol causes it to accumulate in the arterial walls over time, leading to restricted atherosclerotic plaque. This leads to cardiovascular disease, a significant consequence of diabetes [25]. Furthermore, a disturbance in the cholesterol balance of the blood may affect the B cells in the pancreas, which may subsequently reduce the release of insulin [26]. An imbalance in the metabolic processes of the patient group is responsible for the raised triglycerides, and the glucose metabolism issue is the source of the body's fat balance [27]. There are significant differences in the percentage of fasting sugar and cumulative sugar in the test results of diabetes patients and the control group because these tests are used to monitor blood sugar levels. As blood sugar levels rise, the outcome frequently improves for those with diabetes. A readout of the blood sugar level immediately after an eight-hour fast is provided by the blood sugar test for fasting patients, whereas the cumulative sugar test shows the average blood sugar level over the preceding three months. It can therefore diagnose diabetes more accurately since it is less affected by other factors that could lead to inaccurate results [28]. Significant levels of the lipid profile, all demographic parameters, and the results of diabetes diagnostic tests are all associated with insulin resistance, as shown by the results in Tables (3,4). Age-related genetic alterations in the DNA of pancreatic beta cells may be linked to increased insulin resistance because aging-related complications, elevated oxidative stress, and impaired autophagy activity lead stressed cells to progress towards senescence and ultimately reduce pancreatic function [29]. Age-related cellular damages include increased free radical production, decreased mitochondrial adenosine triphosphate production, disruption of cell repair mechanisms, accumulation of certain harmful molecules, such as oxidized fats and specific proteins, and decreased autophagy of accumulated harmful molecules. As a result, cells age and lose some or all of their functional efficacy [29]. It was also shown that the likelihood of acquiring insulin resistance is strongly correlated with age. This conclusion was validated by the findings of the linear regression in Table (4) of the current study. The results also corroborated the study of [30], which used a battery of tests to evaluate a group of young and old people and discovered that the elderly had compromised pancreatic beta cell activity and decreased efficiency as a result of insulin resistance. Additionally, it has been shown that body mass index has a major impact on insulin resistance. This measure of total fat is one of the factors contributing to the increase in insulin resistance. According to a study [31], the obese group exhibited notable levels of insulin resistance, which was consistent with this finding. According to another study, increased adipose tissue has been associated with increased pro-inflammatory responses, such as elevated cytokine and particular interleukin levels [32]. The body's expanding fat cells may cause inflammatory factors that lead to insulin resistance by decreasing the sensitivity of insulin-responsive body cells and increasing the release of fat molecules into the bloodstream from adipose tissue [33]. This explains why insulin sensitivity and insulin resistance were shown to be significantly correlated negatively. Predicting the development of insulin resistance as a consequence of growing body mass was also consistent with [34], which showed a strong relationship between the rise in body mass index and the development of insulin resistance. Obesity is linked to specific factors that lead to insulin resistance because obese people consume a lot of saturated fats, which releases pro-inflammatory cytokines that promote inflammation. By inhibiting the insulin

receptor in muscle and adipose tissue, these viruses produce resistance to changes in the body's insulin levels. The concentration of macrophages caused by inflammation causes the production of adipokine and kinase, which leads to insulin resistance. Furthermore, there is evidence linking insulin resistance to the linearly increasing duration of diabetes. Long-term exposure to glucose-regulating treatments may have caused this, as they decrease beta cell mass and impair their functionality [35]. Glucotoxicity, one of the primary factors causing elevated apoptosis, may produce aberrant beta cell proliferation [36]. Therefore, if diabetes is present for a long time and blood sugar control is inadequate, insulin resistance may be an expected outcome. According to the findings of the present study, there was a statistically significant increase in the lipid profile levels. Triglycerides were the most important indicator of insulin resistance among the variables. These results are in line with another study's findings [37]. This result suggests that increased insulin resistance and cholesterol absorption are negatively correlated. Since the body's capacity to synthesize more cholesterol depends on the extent of decreased intestinal absorption, increased insulin resistance in type 2 diabetics is likely to lead to raised cholesterol levels [38]. The development of dyslipidemia is primarily caused by insulin resistance, which increases the release of free fatty acids from adipose tissue, their absorption by the liver, and the synthesis of very low-density lipoprotein (VLDL), which is high in triglycerides [39]. Triglyceride and cholesterol transit, as well as LDL metabolism, are all facilitated by low-density lipoproteins such as VLDL. Complex interactions between enzymes, receptors, and transport proteins are involved in the metabolism of VLDL, which is produced in the liver [40]. The breakdown is followed by the removal of the associated triglycerides. In muscle and adipose tissue endothelial cells, liver lipase (HL) and lipoprotein lipase (LPL) help transform the residual VLDL into intermediate-density lipoproteins (IDL). Triglycerides are broken down and fatty acids are released. It is taken up by cells for energy and then transformed from IDL to LDL, which has a higher cholesterol content. Hepatocytes and other cells' surface LDL receptors are ultimately in charge of this cholesterol's endocytosis [41]. As free fatty acid levels grow, the pancreatic beta cells will store less, which will result in less insulin being released. Moreover, oxidative processes are employed by free lipids to increase insulin resistance [42]. As triglyceride levels rise, more free fats are transported from adipose tissue to the liver. This leads to increased release of blood glucose and increased hepatic glycogenolysis. [43] Found that a high triglyceride level can affect how glucose is metabolized in target tissues, such muscles, and increase insulin resistance and type 2 diabetes risk. Assessing insulin sensitivity and fasting blood sugar did not statistically significantly alter the prediction of elevated insulin resistance, as demonstrated by the results of the blood parameters under study. A different study came to the same conclusion [44]. It might be because fasting blood sugar measurement isn't able to properly identify insulin resistance in those who have it. Diabetes can be caused by biological, psychological, and environmental causes, and it fluctuates every day. The cumulative blood sugar test, however, had a major impact on the prediction of the presence of insulin resistance because this value is the result of a high blood sugar level throughout the previous three months [28]. Muscles cannot use glucose as an energy source, which leads to a variety of metabolic issues throughout the carbohydrate metabolism process. Another explanation is the overproduction of glucose caused by the breakdown of glycogen in the liver due to a failure in the insulin hormone. Another factor is the resistance of the

target tissues to insulin, which results from their decreased sensitivity to the actions of insulin. The results of the current study indicate that this leads to high insulin resistance, which is a sign of type 2 diabetes [45]. Those over 60 and those between the ages of 50 and 59 had a higher risk of type 2 diabetes than those in younger age groups, according to the data. The results of another investigation were in agreement with this one [46]. It could be explained by the fact that aging is associated with several variables that can cause some human organs to work less well, including a decrease in the effectiveness of B-cell function, programmed death, and a decrease in the ability of the pancreas to regenerate. As a result, pancreatic B-cells lose mass and functioning. It is a major contributing factor to the development of type 2 diabetes. An imbalance in HDL levels, especially in those over 60, is a normal result of aging's issues.

Normal HDL levels protect B cells from damage brought on by oxidation of low-density lipoprotein. According to the current study, older people are more likely to get the illness. Because diabetes and B cells naturally replenish in young persons, younger age groups are less affected than older ones [47]. Gender differences and the risk of type 2 diabetes did not correlate, according to the results of logistic regression. Previous studies that found no positive correlations between gender and diabetes risk [48] were in line with this finding. Additionally, the results did not match those of several other studies, such as [49]. A variety of factors, including differences in the study design, the selection of the study group, regional or societal disparities, or other factors influencing readiness, may make the findings of the current study inconsistent with those of other studies. The results of the present investigation indicate that obesity is the body mass index category that most correctly predicts the risk of type 2 diabetes. This result aligned

with the findings of another study [50], which found that the risk of acquiring diabetes is seven times higher for those who are obese or morbidly obese than for people who are normal weight. Obesity affects the body's ability to use insulin in the target tissues efficiently. As a result, blood sugar levels become abnormal, increasing the risk of diabetes [51].

Conclusion

The researchers of this study concluded that the link between diabetes and dyslipidemia is bidirectional. Glucose concentrations that are elevated beyond the norm have an effect on fat metabolism. This can disturb cholesterol levels across the board. Conversely, alterations in the levels of LDL, HDL, VLDL, and triglycerides influence insulin resistance and the likelihood of developing type 2 diabetes. Due to the strong link between elevated triglyceride levels and the danger of type 2 diabetes onset, this factor serves as a significant biomarker for evaluating disease risk. Consequently, diabetes management should encompass efforts to regulate blood fat levels and lower triglycerides through lifestyle changes such as adopting a low-fat diet, engaging in physical activity for weight loss, and implementing necessary treatments to reduce fats and control blood sugar levels.

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