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Analysis of Insulin Resistance Markers and Lipid Profile in Patients in Baghdad, Iraq

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ABSTRACT

Background: Insulin resistance (IR) is a detrimental common condition related to type 2 diabetes mellitus and different metabolic diseases. The common indices are the homeostasis model assessment of insulin resistance (HOMA-IR) and triglyceride glucose (TyG) index. This study analyzed IR markers and lipid profile in patients in Baghdad, Iraq.

Methods: A cross-sectional study from March 7 2024 to June 1st 2024 was performed at Nutrition and Obesity Center in Al-Kindy Hospital, Baghdad, Iraq enrolling 100 participants (81 females and 19 males) aged between 18-60 years. They were categorized into obese and non-obese groups based on their BMI values. BMI, fasting blood glucose, fasting insulin and lipid profile (total cholesterol, triglycerides, LDL-C, and HDL-C) and HbA1C were determined and analyzed.

Results: HOMA-IR index detected 38% of IR individuals, while TyG index verified 51%. BMI was shown to be correlated too (p=0.0001, p=0.033, respectively). For lipid profile, LDL-C and total cholesterol were inversely related to HOMA-IR index (p=0.048, p=0.163, respectively). HDL-C had inverse relationship with HOMA-IR (p=0.014), and triglyceride had a positive correlation with HOMA-IR (p=0.190). TyG revealed a positive correlation with total cholesterol and triglyceride (p=0.0006, p=0.0001, respectively). The correlation between TyG index and LDL was positive, and forTyG and HDL was negative (p=0.105, p=0.179, respectively).

Conclusion: TyG index was superior to HOMA-IR with a stronger positive correlation to the BMI marker too. Lipid profile revealed a weak relationship with HOMA-IR ant TyG indices.

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Introduction

The insulin resistance (IR) is defined as disrupted glucose metabolism that is attributed to defective insulin action, primarily in liver, fat, muscle tissues. IR is highly related to type 2 diabetes mellitus (T2DM) and other metabolic dysfunctions, where it might be the forerunner of these conditions too. Examples of these conditions might be obesity, dyslipidemia, and metabolic syndrome (1-3). IR status is provoked by both genetic and environmental factors. The prevalence of IR in healthy individuals was estimated 49.9% in Middle-East, particularly in Iraq (4). Globally, the prevalence was reported 15.5-46.5%; while Lebanon had a high rate in Middle East (44.6%) (5).

The American College of Endocrinology (AACE)

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demonstrated that IR is diagnosed by having at least one of the diagnostic criteria including (i) cardiovascular diseases (CVDs), polycystic ovary syndrome (PCOS), nonalcoholic fatty liver disease (NAFLD), hypertension, and impaired glucose tolerance or (ii) lack of physical activity, age >40 years, body mass index (BMI) >25, and waist circumference >102 cm in men and >88 cm in women. Additionally, at least fasting blood glucose between 110-125 mg/dL, triglycerides >150 mg/dL, and HDL-C <40 mg/dL in men and <50 mg/dL in women can be confirmatory (6). The Homeostasis model assessment index (HOMA-IR) was designed simply to assess degree of IR from direct measurements of normal ranges of glucose and insulin levels, through an equation that involves multiplying fating insulin by fasting glucose and dividing the result by a constant HOMA IR=[Fasting Insulin (μU/mL)×Fasting Plasma Glucose (mg/dL)]/405 (7).

The threshold value of HOMA-IR >2.5 was shown to be highly linked to the IR, and was also associated with obesity (8). The hyperinsulinemic euglycemic glucose clamp is the gold standard method for IR diagnosis (9). The latter is not applied nowadays, for being highly costly, laborious, and invasive. When the data that is obtained from hyperinsulinemic euglycemic glucose clamp test are compared to those with HOMA-IR, a highly correlation was noticed (10). Still, the trials in threshold values, particularly when applied in different races, various age groups or lifestyles, the most significant challenges in HOMA-IR were observed. Additionally, HOMA cannot specify the main cause of IR, whether peripheral or hepatic, which is also considered as another weakness point (11).

Lastly, it is important to keep in mind that the HOMA depends primarily on insulin hormone in IR diagnosis, the latter might be subjected to many different impacting factors, that include being secreted in pulsatile rhythm, or having a short half-life, in addition to the main significantly important factors which are the decreased insulin hormone amount at the later stages of IR and T2DM as the beta cells storage might be depleted (11). Meanwhile the triglyceride glucose (TyG) index which is another surrogate marker for IR is considerably related with the euglycemic glucose clamp test and is yet simpler, non-invasive, and easy to compute. High TyG index results were highly linked to the advanced stages of IR and CVD $\mathrm{TyG} = \mathrm{Ln}\left(\mathrm{TG}\left[\frac{\mathrm{mg}}{\mathrm{dL}}\right] \times \mathrm{glucose}\left[\frac{\mathrm{mg}}{\mathrm{dL}}\right] / 2\right)$ (12).

TyG index was also shown to be effective as a screening tool for T2DM in the third-world countries (13). A TyG index threshold value of 4.5 was proven to be better in diagnosis of IR, and individuals with an index of 4.5 or above illustrated more advanced IR symptoms (14). Many studies have shown that TyG index is even superior to HOMA-IR in diagnosis of IR, specifically when associated with obesity or other metabolic dysfunctions, as this index has better reliable components which are fasting glucose and fasting triglyceride, while both are highly associated with the IR status. The triglyceride might be highly elevated as an early manifestation of the condition, since the elevated glucose level is predominantly transformed to the triglyceride (15-17).

Additionally, dyslipidemia has been claimed to be a major component in the diagnosis of IR, where many different studies showed the link between IR and abnormal lipid profile (18-20). Nonetheless, it should be kept in mind that abnormal lipid profile might not be essentially found in all IR cases, as some studies have demonstrated some weak relationship between abnormal lipid profile components and IR markers, and that BMI or other obesity markers were proven to be more related to the direct IR diagnosis (21, 22).

Materials and Methods

A cross-sectional study was performed at the Nutrition and Obesity Center in Al-Kindy Hospital, Baghdad, Iraq from March 7 2024 to June 1st 2024. The study was approved by the Ethical Committee of the Al-Kindy Hospital, Baghdad, Iraq; while consent form was taken from the participants, with fully detailed explanation. A total of 100 participants (81 females and 19 males), aged between 18-60 years, were enrolled and categorized into obese and non-obese groups based on their BMI values. Individuals with HbA1c above 6.5, hypertension, CVD, or other chronic endocrine diseases were excluded from the study to avoid any interference with insulin resistance. Full history was taken with physical examination and measurement of height and weight to calculate BMI. In addition, fasting blood glucose, fasting insulin and lipid profile [total cholesterol, triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C)] and HbA1C, were determined, through blood sample drawn from vein puncture between 8.00 and 10:00 a.m. (5 mL). Blood sample was then put in disposable syringes, and divided into two halves. The first half of the blood was put in the jell tube, and clotted at 37°C, then was centrifuged to get serum for 10 min at 3000 round per minute (rpm), and then stored at -20°C for lab analysis. The second half was placed in a tube that contained ethylene diamine tetra acetic acid (EDTA) for HbA1c. The collected data were analyzed by computer using the software statistical data of IBM SPSS-29 (IBM Statistical Packages for Social Sciences, version 29, Chicago, IL, USA). Results were applied in simple measures of frequency, percentage, mean, standard deviation (SD), and range (minimum and maximum values). The significance of difference of means was tested using Students t-test between the two independent means. The significance of difference of percentages was calculated using Pearson Chi-square test (c^{2}-test) by applying Yate's correction or Fisher Exact test whenever applicable. Statistical significance was considered whenever the *p* value was equal or less than 0.05 (23).

Results

Table 1 demonstrates the IR indices among individuals, where HOMA-IR threshold value was considered ≥2.5 and TyG index as ≥4.5. The mean HOMA was 2.850±1.896, with a range of 0.468-6.740. The mean of TyG index was 4.526 ± 0.249 with a range of 4.003-4.946. Table 2 shows relationships between HOMA-IR index and lipid parameters based on the threshold value of ≥2.5 of HOMA-IR index. The values of HOMA-IR≥2.5 were significantly related to fasting blood glucose (*p*=0.0001), fasting insulin (*p*=0.0001), and HDL (*p*=0.014). The LDL level had an inverse

Table 1: Distribution of homeostasis model assessment of insulin resistance (HOMA-IR) and triglyceride-glucose (TYG) indices in IR.

Variable		No.	%
HOMA-IR (≥2.5)	≥2.5	38	38.0
	<2.5	62	62.0
	Mean±SD (Range)	2.850±1.896 (0.468-6.740)	
TYG index	≥4.5	51	51.0
	<4.5	49	49.0
	Mean±SD (Range)	4.526±0.249 (4.003-4.946)	

Table 2: Relationship between homeostasis model assessment of insulin resistance (HOMA-IR) index and biological analysis.

Variable		Homa-IR (>2.5)				*P* value
		≥2.5		<2.5		
		No.	%	No.	%	
Fasting blood glucose (mg/dL)		106.29±5.03		101.03±6.81		0.0001#
Fasting Insulin (mIU/L)		18.91±4.84		6.27±2.20		0.0001#
Cholesterol (mg/dL)		158.89±24.37		167.45±32.23		0.163
Triglyceride (mg/dL)		100.74±45.28		88.90±42.49		0.190
LDL (mg/dL)		78.96±19.98		88.56±25.10		0.048#
HDL (mg/dL)		36.97±5.10		40.15±6.78		0.014#
Triglyceride-Glucose Index (TYG)	≥4.5	21	55.3	30	48.4	0.504
	<4.5	17	44.7	32	51.6	
	Mean±SD	4.586±0.243		4.490±0.247		0.060

Data were presented as Mean±SD. *Significant difference between percentages using Pearson Chi-square test (c^{2}-test) at 0.05 level. #Significant difference between two independent means using Students-t-test at 0.05 level. Low-density lipoprotein cholesterol (LDL-C). High-density lipoprotein cholesterol (HDL-C).

Table 3: The association between triglyceride-glucose (TyG) index and biochemical parameters of IR.

Variable	TYG				*P* value
	≥4.5		<4.5		
	No.	%	No.	%	
Fasting blood glucose (mg/dL)	104.88±6.83		101.10±6.00		0.004#
Fasting Insulin (mIU/L)	10.27±4.77		11.91±8.80		0.245
Cholesterol (mg/dL)	172.07±29.40		156.00±27.91		0.006#
Triglyceride (mg/dL)	129.51±31.34		55.81±9.63		0.0001#
LDL (mg/dL)	88.68±20.79		80.99±25.94		0.105
HDL (mg/dL)	38.10±6.58		39.81±6.07		0.179

Data were presented as Mean±SD. *Significant difference between percentages using Pearson Chi-square test (c^{2}-test) at 0.05 level. #Significant difference between two independent means using Students-t-test at 0.05 level. Low-density lipoprotein cholesterol (LDL-C). High-density lipoprotein cholesterol (HDL-C).

Table 4: Association of homeostasis model assessment of insulin resistance (HOMA-IR) index and body mass index (BMI) marker.

Variable		Homa-IR (>2.5)				*P* value
		=>2.5		<2.5		
		No.	%	No.	%	
BMI (Kg/m²)	Normal (18.5-24.9)	7	18.4	24	38.7	0.004*
	Overweight (25-29.9)	-	-	8	12.9	
	Obese I (30-34.9)	8	21.1	8	12.9	
	Obese II (35-39.9)	21	55.3	16	25.8	
	Morbid obesity (≥40)	2	5.3	6	9.7	
	Mean±SD	34.12±7.39		29.73±7.58		0.006#

Table 5: The association between triglyceride-glucose (TYG) index and body mass index (BMI).

Variable		TYG				*P* value
		=>4.5		<4.5		
		No.	%	No.	%	
	Mean±SD	159.7±7.7		161.2±5.4		0.274
BMI (Kg/m²)	<25 Kg/m²	6	11.8	25	51.0	0.0001*
	≥25 Kg/m²	45	88.2	24	49.0	
BMI (Kg/m²)	Normal (18.5-24.9)	6	11.8	25	51.0	0.001*
	Overweight (25-29.9)	4	7.8	4	8.2	
	Obese I (30-34.9)	10	19.6	6	12.2	
	Obese II (35-39.9)	25	49.0	12	24.5	
	Morbid obesity (≥40)	6	11.8	2	4.1	
	Mean±SD	34.93±5.43		27.72±8.17		0.0001#

relationship with HOMA values above 2.5 in a statistically significant ratio (p=0.048), and the total cholesterol (p=0.163). Triglyceride was positively correlated to HOMA (p=0.190). The TyG index showed insignificant difference (p=0.504) when compared to HOMA in IR diagnosis.

Table 3 demonstrates the analysis of the TyG and IR parameters, with TyG threshold values equal or above 4.5. TyG index >4.5 was significantly related with high fasting blood glucose (p=0.004), and cholesterol (p=0.006), and triglycerides (p=0.0001). Meanwhile, statistically insignificant difference of the fasting insulin levels (p=0.245), and LDL (p=0.105), and HDL (p=0.179) in relation with TyG index were noticed. Table 4 illustrates the association between BMI and HOMA-IR index to be significant (p=0.006). Table 5 shows the association between TyG index and BMI to be more strongly related with higher P value (p=0.0001).

Discussion

T2DM can lead to hyperglycemia, IR and different metabolic diseases affecting weight, liver function parameters and lipid profile (24-26). This study presented a prevalence of 38% for IR detected by HOMA index; while TyG index for IR was 51%. Although the difference was shown statistically non-significant, the TyG has detected few more cases than HOMA-IR index. This indicates that the TyG index might have more powerful accuracy than HOMA-IR in the early detection of the condition. Many studies have shown the superiority of TyG index in comparison to HOMA in diagnosis of many metabolic conditions including IR, T2DM and metabolic syndrome (15, 27).

Our study depicted that individuals with HOMA-IR values (>2.5) had higher percentage of high BMI values, as well as fasting insulin and blood glucose. Previous studies are in agreement with our results. Boyer *et al.* in US with 3127 participants demonstrated a high link between HOMA-IR and these parameters indicating that IR was highly correlated to obesity (28). In comparison, the TyG index with a threshold value of equal or more than 4.5 revealed relationship with BMI. This was also confirmed by another study revealing the strength of TyG and the relationship with BMI (29). Similarly in our study, the TyG index had a positive correlation with the fasting triglyceride and fasting glucose in a statistically significant ratio. Nonetheless, TyG index in relation to fasting insulin was shown to be positively and insignificantly related yet.

HOMA-IR index with lipid parameters were presented unexpectedly, where LDL-C and total cholesterol were inversely correlated to HOMA values, which is very much opposite to the common belief that the dyslipidemia is associated with IR. Also, LDL-C with HOMA-IR were specifically inversely correlated in a significant statistical ratio. This might indicate that the LDL-C was less reliable as a parameter for IR detection, or it might be explained by the fact that LDL-C had large and small size particles, while the latter was known to be strongly atherogenic and more related to the IR, unlike the large particle LDL-C that was shown to be found in healthier individuals even with abnormal lipid profile (30).

Meanwhile, HOMA had positive correlation with triglyceride, though non-significantly, while negative correlation was significantly visible with HDL. Alternatively, TyG index demonstrated positive correlation with total cholesterol, and LDL-C, though the latter was insignificantly correlated. Also, HDL-C was inversely and insignificantly correlated with TyG index. Some studies have described the weak relationship between lipid parameters and IR markers (31, 32). This may indicate that both indices had weak relationship with lipid profile in general, and that IR might not be directly related to dyslipidemia and should not be considered an essential criterion in its diagnosis. More importantly, lipid profile should not be utilized as a parameter for IR diagnosis too.

Conclusion

TyG index was shown to be superior to HOMA index in IR diagnosis, and with a stronger positive correlation to BMI. Lipid parameters have also demonstrated weak relationship with both IR markers, HOMA, ant TyG indices.

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Authors' Contribution

All authors contributed equally to this work.

Conflict of Interest

The authors declare no conflict of interest.

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