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The predictive value of the triglyceride glucose index combined with body mass index in diagnosis of metabolic dysfunction associated steatotic liver disease in patients with type 2 diabetes mellitus

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Abstract
Background: A new index designed to detect insulin resistance in type 2 diabetes mellitus (T2DM) called the triglyceride glucose index paired with body mass index (TyG-BMI) has been developed. This work aimed to find out how well the TyG-BMI index predicts the development of metabolic dysfunction associated steatotic liver disease (MASLD) in T2DM individuals.
Methods: This cross-sectional study involved 320 patients aged >18 years old, both sexes, had the diagnostic criteria for T2DM which divided into group I (n=201): diagnosed as T2DM and have MASLD, group II (n=119): diagnosed as T2DM and don't have MASLD and 80 age-and sex-matched healthy individuals as a control group.
Results: Among the three groups, T2DM patients with MASLD had substantially higher BMI, lipid and glycemic profile, levels of insulin resistance estimated by the homeostasis model, TyG index, TyG-BMI index compared to patients without MASLD. According to area under ROC curve for each parameter, the TyG-BMI index was the most accurate parameter to predict MASLD in T2DM patients with the highest accuracy (88.8%), sensitivity (97.5%), and specificity (80%).
Conclusion: TyG-BMI has greater reliability, is easy to compute, and is inexpensive, making it a promising screening tool for the detection of MASLD in T2DM individuals compared to the other indices.

Keywords: Triglyceride glucose index, body mass index, metabolic dysfunction associated steatotic liver disease, type 2 diabetes mellitus

Introduction
Metabolism-Dysfunction-Associated Steatotic Liver Disease (MASLD), which was previously called non-alcoholic fatty liver disease (NAFLD), is defined by hepatic steatosis in individuals with a minimum of one metabolic risk factor, such as obesity, diabetes, dyslipidemia, or hypertension [1]. Metabolic dysfunction-associated steatohepatitis (MASH), hepatic fibrosis, cirrhosis, and hepatocellular carcinoma are advanced stages of this spectrum of liver illness [2]. Nearly all people who have obesity, 70% of people with type 2 diabetes, and 30% of the general population are affected by MASLD, making it the most common etiology of chronic liver disorders [3]. A growing number of people are suffering from metabolic syndrome and obesity, which has increased the likelihood of MASLD and is placing a financial burden on healthcare systems around the world [3]. Multiple and various studies have established a cross relations between MASLD and T2DM, dyslipidemia, cardiovascular disease, hypertension, and chronic kidney disease [4]. T2DM is a complex metabolic disorder characterized by insulin resistance, inadequate insulin release all of which lead to persistently high blood sugar levels and eventually complications affecting every cell in our human body [5]. The risk of developing MASLD is higher in T2DM individuals. Further, T2DM is associated with an increased risk of complications in MASLD patients, including cirrhosis, HCC, and MASH. Therefore, MASLD effects can be greatly reduced if diagnosed and treated early in T2DM patients [6].

T2DM and MASLD are both related to insulin resistance, which enhances the transport of free fatty acids to the liver parenchyma and increasing hepatic lipogenesis [7].

The triglyceride-glucose index (TyG index) which incorporates fasting triglycerides and plasma glucose levels, has been found to exhibit a strong correlation with insulin resistance [8]. There is a correlation between MASLD prevalence and severity with body mass index (BMI). Hence, integration of TyG index with BMI is more effective than utilizing the TyG index alone in predicting MASLD in T2DM [9]. This work aimed to determine whether the TyG-BMI is useful for predicting the presence of MASLD in T2DM individuals.

Patients and Methods

This cross-sectional study involved 320 patients aged >18 years, both sexes, had the diagnostic criteria for T2DM according to ADA criteria,201 patients of them (62.8%) had the diagnostic criteria of MASLD (no history of alcohol intake or a current alcohol intake each day was less than 30 grams for males and 20 grams for women, with at least one cardio-metabolic risk factor eg; obesity, hyperlipidemia, diabetes, or hypertension, and ultrasound imaging tests showed signs of diffuse fatty liver disease) and 80 healthy individuals matched age and sex as control group. The research ran from June 2023 to June 2024 after approval from the Tanta University Hospitals Ethical Committee in Tanta, Egypt. Written informed permission from patients was obtained.

Exclusion criteria were excessive alcohol intake (>30 g/day for males and >20 g/day for females), viral hepatitis, haemochromatosis, Wilson's disease, autoimmune liver disease, hypothyroidism, Cushing's syndrome, inflammatory bowel disease, drugs causing liver steatosis (corticosteroids, amiodarone, estrogen and tamoxifen), pregnancy and breastfeeding.

Patients were divided into three groups: Group I (n=201): diagnosed as T2DM and have MASLD, group II (n=119): diagnosed as T2DM and don't have MASLD and group III (n=80): healthy matched age and sex individuals as a control group.

Every patient underwent a comprehensive medical history taking, anthropometric measurement, laboratory investigations [fasting plasma glucose (FBG), 2h postprandial plasma glucose (2hPP), HbA1C, lipid profile includes triglycerides, total cholesterol, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) levels, liver function tests (AST, ALT, bilirubin and albumin), hepatitis B surface antigen (HBsAg) and hepatitis C virus antibody (HCV Ab).

All biochemical parameters were estimated by THERMO FISHER SCIENTIFIC Konelab Prime 60i and virology assessment with Roche Cobas E601 Immunology Analyzer.

Plasma insulin level

The enzyme-linked immunosorbent assay (ELISA) method was utilized for measuring insulin, with a typical, healthy adult fasting insulin level less than 25 µlU/ml.

One way to quantify insulin resistance is with the help of the homeostasis model assessment of insulin resistance (HOMA-IR).

The HOMA-IR is determined as follows [8]: HOMA-IR = fasting insulin (µU/ml) × fasting blood glucose (mg/dL) / 405.

TyG index [8]: TyG = Ln (TG (mg/dL) × fasting blood glucose (mg/dL) / 2).

TyG-BMI [9]: TyG-BMI = TyG index × BMI.

Radiological assessment [10]

Abdominal ultrasonography was performed by expert radiologists, using Philips Affiniti 50 Ultrasound Machine equipped with a convex probe 2-6 MHz (C6-2), and the radiologist was not given information to the patients' medical records or test results. There were five criteria used to report hepatic steatosis: (1) parenchymal brightness (echogenicity), (2) contrast between the liver and either the kidneys or the spleen, (3) deep beam attenuation, (4) bright vessel walls, and (5) definition of the gallbladder wall and diaphragm.

Statistical analysis

SPSS v27 (IBM©, Chicago, IL, USA) was employed to conduct statistical analysis. The data was examined using histograms and the Shapiro-Wilks test to ascertain if it followed a normal distribution. To analyze quantitative parametric data, expressed as means and standard deviations (SD), we employed an ANOVA with a Tukey post hoc test. To compare the groups' quantitative non-parametric data, which was displayed as median and interquartile range (IQR), we utilized the Kruskal-Wallis test in conjunction with the Mann Whitney U test. The qualitative variables were analyzed with the Chi-square test and presented as percentages and frequencies. An analysis of the diagnostic performance's sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV). It was deemed statistically significant if the two-tailed P value was <0.05.

Results

Although there was no substantial difference in age or sex among the three groups, those had T2DM with MASLD had a considerably higher BMI than those without MASLD. In terms of SBP, the two groups of T2DM did not differ substantially from one another; however, the DBP of those had T2DM with MASLD was substantially greater than those without MASLD. Table 1.

Table 1: Demographic data, blood pressure of the participants

		T2DM with MASLD (n=201)	T2DM without MASLD (n=119)	Control group (n=80)	Test of sig.	p
Age (years)		53.1±9.08	53.5±8.13	52.0±8.19	F=0.207	0.813
Sex	Male	91(45.3%)	66(555%)	40(50.0%)	χ^2=3.200	0.202
	Female	110(54.7%)	53(44.5%)	40(50.0%)		
BMI (kg/m²)		32.8±3.09	27.5±1.72	21.3±1.83	F=159.754	<0.001*
		P1<0.001*, P2<0.001*, P3<0.001*				
SBP (mmHg)		122.8±17.67	121.5±14.42	116.0±9.40	8.627	<0.001*
		P1 = 1.000, P2 = <0.001*, P3 = <0.001*				
DBP (mmHg)		89.9 ± 6.55	80.0 ± 9.87	74.0 ± 6.81	29.495	<0.001*
		P1<0.001*, P2<0.001*, P3 = 0.024*				

Data are presented as mean ± SD or frequency (%). * Significant P value <0.05, $\chi 2$: Chi square test, F: ANOVA, P1: between T2DM with MASLD and T2DM without MASLD, P2: between Control and T2DM with MASLD, P3: between Control and T2DM without MASLD, BMI: Body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure. The duration of diabetes, usage of oral antidiabetic medications, use of statins, and smoking were not substantially different between T2DM patients with or without MASLD, while there was significant difference as regards insulin use and antihypertensive drugs. Table 2.

Table 2: Clinical characteristics of diabetic patients

		T2DM with MASLD (n=201)	T2DM without MASLD (n=119)	Test of sig.	P
Duration of T2DM (years)	< 10	131(65.2%)	68(57.1 %)	$\chi 2$=1.896	0.168
	≥ 10	70(34.8%)	51(42.8 %)		
Insulin use		53(26.3%)	14(11.8%)	$\chi 2$=11.815	<0.001*
Oral antidiabetic drugs		143(71.1%)	91(76.4%)	$\chi 2$=1.033	0.309
Statin use		84(41.8%)	56(47%)	$\chi 2$=0.810	0.368
Anti-hypertensive drugs		139(69.2%)	61(51.3%)	$\chi 2$=11.719	<0.001*
Smoking		45(22.4%)	33(27.7%)	$\chi 2$=1.067	0.302

Data are presented as mean ± SD or frequency (%). * Significant P value <0.05, $\chi 2$: Chi square test. FBG, 2hPP, HbA1C levels, total cholesterol, LDL-C, triglycerides, AST, and ALT were substantially higher in T2DM patients with MASLD than patients without MASLD. HDL-C level was considerably lower in patients with MASLD. Table 3.

Table 3: Laboratory data of the participants

		T2DM with MASLD (n=201)	T2DM without MASLD (n=119)	Control group (n=80)	F	P
Blood glucose levels	FBG (mg/dl)	195.1±57.95	133.8±15.64	85.5±6.57	161.694	<0.001*
		P1<0.001* P2<0.001* P3<0.001*				
	2hr. PP (mg/dl)	250.2±80.97	200.7±36.98	122.9±9.87	84.050	<0.001*
		P1<0.001*, P2<0.001*, P3<0.001*				
	HbA1C (%)	9.2±1.65	8.3±0.93	5.1±0.26	106.230	<0.001*
		P1<0.001*, P2<0.001*, P3<0.001*				
Lipid profile	Total cholesterol (mg/dl)	219.9±38.37	183.9±14.89	170.9±10.78	42.742	<0.001*
		P1<0.001*, P2<0.001*, P3=0.440				
	TG (mg/dl)	195.2±49.16	144.9±14.12	123.2±8.92	39.588	<0.001*
		P1<0.001*, P2<0.001*, P3 = 0.130				
	LDL (mg/dl)	132.4±33.35	90.6±17.71	82.5±11.60	55.503	<0.001*
		P1<0.001*, P2<0.001*, P3= 0.986				
	HDL (mg/dl)	46.8±7.70	64.1±4.48	64.6±4.30	102.925	<0.001*
		P1<0.001*, P2<0.001*, P3 = 1.000				
Liver function tests	ALT (U/L)	47.9±19.78	24.3±3.46	23.5±3.03	42.113	<0.001*
		P1 <0.001*, P2<0.001*, P3 = 1.000				
	AST (U/L)	50.9±22.58	24.1±4.97	20.8±2.95	43.711	<0.001*
		P1<0.001*, P2 <0.001*, P3 = 1.000				
	Albumin (g/dl)	4.1±0.27	4.2±0.33	4.6±0.29	20.315	<0.001*
		P1=0.629, P2<0.001*, P3 <0.001*				
	Bilirubin (mg/dl)	0.9±0.23	0.8±0.20	0.8±0.13	1.805	0.170

Data are presented as mean ± SD. * Significant P value <0.05, F: ANOVA, p1: Between T2DM with MASLD and T2DM without MASLD, P2: Between Control & T2DM with MASLD, P3: Between Control and T2DM without MASLD, FBG: fasting blood glucose, 2hPP: 2 hours post prandial, HbA1C: glycated hemoglobin, TG: triglycerides, LDL: low-density lipoprotein, HDL: high-density lipoprotein, CBC: complete blood count, Hb: hemoglobin, PLT: platelets, TLC: total leucocytic count, AST: aspartate transaminase, ALT: alanine transaminase. There was a substantial difference among T2DM individuals with and without MASLD, as indicated by the considerably higher HOMA-IR, TyG index, TyG-BMI index. Table 4.

Table 4: HOMA-IR, TyG-index, TyG-BMI of the participants

	T2DM with MASLD (n=201)	T2DM without MASLD (n=119)	Control group (n=80)	Test of sig.	p
HOMA-IR	9.1±3.47	5.1±1.20	1.3±0.14	F= 79.888	<0.001*
	P1<0.001* P2<0.001* P3<0.001*				
TyG-index	5.4±0.27	4.9±0.12	4.6±0.06	F= 108.882	<0.001*
	P1<0.001* P2<0.001* P3<0.001*				
TyG-BMI	186.1±22.71	138.26±9.70	98.4±8.56	F= 213.477	<0.001*
	P1<0.001* P2<0.001* P3<0.001*				
	P1<0.001* P2<0.001* P3=0.003*				

Data are presented as mean ± SD or median (IQR). * Significant P value <0.05, F: ANOVA, p1: Between T2DM with MASLD and T2DM without MASLD, P2: Between Control & T2DM with MASLD, P3: Between Control and T2DM without MASLD, HOMA-IR: homeostasis model assessment-estimated insulin resistance, TyG: triglyceride glucose, BMI: body mass index. According to area under ROC curve for each parameter in diagnosis of MASLD in all patients, the TyG-BMI index was the most accurate parameter to predict MASLD in T2DM patients with the highest accuracy (88.8%), sensitivity (97.5%), and specificity (80%). Figure 1.

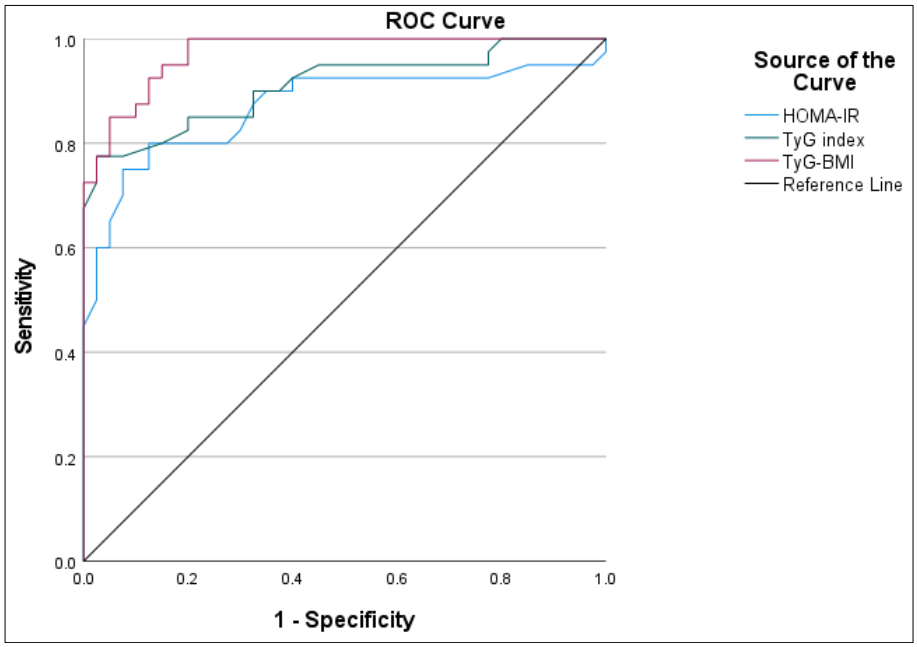

Fig 1: ROC curve for each parameter in diagnosis of metabolic dysfunction-associated steatotic liver disease in the participants

Discussion

Due to the global obesity and T2DM epidemics, the incidence of MASLD has increased dramatically, making it a prominent etiology of liver-related mortality and complications [2, 3].

In terms of smoking, duration of diabetes, usage of oral antidiabetic medications, and statin use, there was no considerable difference between the two groups of T2DM with and without MASLD. Our findings are supported by Mantovani *et al*. [11] results.

Insulin usage and antihypertensive medication usage were substantially different among T2DM individuals with and without MASLD in agreement with Targher. G, *et al*. [12] at which patients using insulin or antihypertensive drugs were significantly higher among patients with MASLD.

In line with Chen. K, *et al*. [13], there was no discernible difference in SBP between MASLD and non-MASLD patients. However, MASLD patients exhibited considerably greater DBP.

The body mass index was considerably greater in those with MASLD group consistent with Portillo-Sanchez, *et al*. [14] findings.

This study revealed that compared to those without MASLD, people with T2DM who it had also had considerably higher levels of FBG, 2hPP and HbA1C in agreement with Ding. X, *et al*. [15].

The results indicated that patients with MASLD had considerably lower levels of HDL-C and substantially higher levels of total cholesterol, LDL-C, and TG compared to those without the disease in consistent with the Chinese cross-sectional study conducted by Guo. K, *et al*. [16]. The same results were in Almobarak. A, *et al*. [17]. There is a strong link between dyslipidemia and MASLD as the increased level of cholesterol and triglycerides will increase the level of circulating adipokines and cytokines as well as associated lipotoxicity, mitochondrial dysfunction, oxidative stress, and endoplasmic reticulum stress which are all involved in pathogenesis of liver steatosis [18].

The results of this study corroborate those of Ajmera. V. *et al*. [19], who found that MASLD patients had substantially greater AST and ALT values than non-MASLD patients. The same was proved in the study conducted in Pakistan by Ali. A, *et al*. [20]. The mechanism of the development of a hepatic steatosis is attributed mainly to insulin resistance that activates lipolysis which enhance free fatty acids accumulation in the liver parenchyma which results in increase in ALT and AST levels [18].

The HOMA-IR was considerably greater in individuals who had MASLD than non-MASLD patients in line with Kim. B, *et al*. [21] results.

Patients with MASLD had a considerably higher TyG index than non-MASLD patients matched with Li. W. *et al*. [22] results.

Patients with MASLD had a considerably higher TyG-BMI index than non-MASLD patients, the same results obtained by Chen, Q., *et al*. [23].

This study confirmed the findings of Li. Y, *et al*. [9] and Wang. M, *et al*. [24] that TyG-BMI was the best parameter to predict MASLD in type 2 diabetes patients. Although the Korean study by Song *et al*. [25] TyG-WC was superior in predicting MASLD, but the majority of their patients did not have diabetes.

Results from this study demonstrate that TyG-BMI significantly predicts MASLD in T2DM patients. Furthermore, this finding provides more evidence that MASLD can be effectively reduced in T2DM individuals with weight loss, increased physical activity, and strict control of diabetes and lipid profiles.

The study has certain limitations, such as the fact that it was conducted in just one center. Ultrasonography was used to diagnose MASLD, but it is not very sensitive and does not consistently identify liver fat infiltration below 30%, but it is suggested as the initial imaging method for screening patients for MASLD in a clinical setting due to the high cost of computed tomography (CT) and magnetic resonance spectroscopy (MRS), and the invasiveness of liver biopsy. Further research is needed to determine whether TyG-BMI is effective in identifying MASH and advanced fibrosis.

Conclusion

TyG-BMI has greater reliability, is easy to compute, and is inexpensive, making it a promising screening tool for the identification of MASLD in T2DM patients compared to the other indices.

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Conflict of Interest: Nil

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