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Predictive significance of the triglyceride glucose index and associated indicators for hepatic steatosis in type 2 diabetes patients with diverse body mass index

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Abstract

Introduction: The objective was to explore the value of triglyceride glucose index and related indicators in predicting hepatic steatosis in type 2 diabetes patients (T2DM) with different body mass indexes (BMI).

Material and methods: Clinical data from 221 hospitalised patients withT2DM, diagnosed with hepatic steatosis via transient elastography, were collected and analysed. Patients with a BMI of 24 kg/m^2 or higher were designated as the high-BMI group, while those with a BMI below 24 kg/m^2 comprised the low-BMI group. Relevant indicators, including triglyceride glucose index (TyG), TyG-BMI index (TyG-BMI), and TyG-waist circumference (TyG-WC), were obtained.

Results: TyG, TyG-BMI, and TyG-WC were all significantly elevated in the high-BMI group relative to the low-BMI group ($p < 0.01$).Within both high- and low-BMI categories of T2DM patients, TyG, TyG-BMI, and TyG-WC were markedly higher in patients with hepatic steatosis than in those without the condition ($p < 0.01$ or $p < 0.05$). Multivariate logistic regression analysis identified TyG-BMI as an independent risk factor for hepatic steatosis in high-BMI T2DM patients ($p < 0.01$), while glycosylated haemoglobin (HbA_{1c}) and TyG-WC emerged as independent risk factors in low-BMI T2DM patients ($p < 0.01$ or $p < 0.05$). Receiver operating characteristic curve analysis for predicting hepatic steatosis in high-BMI T2DM patients demonstrated that TyG-BMI had an area under the curve of 0.84 ($p < 0.01$) with a cut-off value of 241.36, yielding a sensitivity of 79.80% and a specificity of 83.30%. For low-BMI T2DM patients, the TyG-WC index had an area under the curve of 0.80 ($p < 0.01$), a cut-off value of 824.63, a sensitivity of 68.30%, and a specificity of 84.90%

Conclusion: In T2DM patients, TyG-BMI can predict hepatic steatosis in high-BMI patients, and TyG-WC can predict hepatic steatosis in low-BMI patients. The latter is negatively correlated with HbA_{1c}.

Key words: *diabetes mellitus type 2; hepatic steatosis; body mass index; triglyceride glucose-body mass index; triglyceride glucose-waist circumference index*

Introduction

Non-alcoholic fatty liver disease (NAFLD), which refers to liver steatosis excluding alcohol consumption and other definite causes, if not detected and intervened in time, is prone to further develop into liver fibrosis, cirrhosis, and even liver cancer. The occurrence of NAFLD is closely related to metabolic diseases such as obesity, hyperlipidaemia, and diabetes. It has now been renamed metabolic associated fatty liver disease (MAFLD) [1]. In recent years, with the update of type 2 diabetes mellitus (T2DM) management concepts and the emergence of new hypoglycaemic drugs, such as glucagon-like peptide-1 receptor agonists (GLP-1RA), sodium glucose cotransporter 2 inhibitors (SGLT-2i), GLP-1, and glucose-dependent insulinotropic polypeptide (GIP) dual receptor agonists, as well as the development of metabolic surgeries, excellent methods have been provided for obese T2DM patients with fatty liver. However, it has also been found that a considerable number of non-obese patients have MAFLD, and these patients have not received sufficient attention. Recent studies have shown that the glucose triglyceride index (TyG) and its related indicators can be good indicators reflecting insulin resistance, and they are closely related to hepatic steatosis [2]. Therefore, the main purpose of this study was to observe the differences in risk factors for hepatic steatosis in T2DM patients with different BMIs and to further explore the predictive value of TyG and its related indicators for hepatic steatosis in these T2DM patients.

Material and methods

Subjects

A total of 221 hospitalised T2DM patients were selected. Inclusion criteria: 18 to 70 years old; agreed to measure liver fat attenuation parameters by iLivTouch and completed the hospital admission questionnaire survey; and the subject (or their legal guardian) understood the requirements of this clinical trial and signed

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the informed consent form. Exclusion criteria: patients diagnosed with viral hepatitis and liver schistosomiasis; patients with severe diseases, liver area surgery, ascites, or cardiac pacemaker implantation; pregnant, or lactating women; and history of alcohol abuse/drug abuse. The diagnosis of T2DM conformed to the 1999 diagnostic and classification criteria of T2DM by the World Health Organisation. Patients with body mass index (BMI) ≥ 24 kg/m^2 were called the high-BMI group, and patients with BMI < 24 kg/m^2 were called the low-BMI group. This study was approved by the Ethics Committee of our hospital, and all subjects signed the informed consent form.

Methods

At admission, the patients' gender, age, duration of diabetes, BMI, waist circumference (WC), hip circumference (HC), fasting blood glucose (FBG), 2-hour postprandial blood glucose (2hPG), glycosylated haemoglobin (HbA_{1c}), fasting C-peptide (FCP), 2-hour postprandial C-peptide (2hCP), white blood cell count (WBC), alanine aminotransferase (ALT), aspartate aminotransferase (AST), blood uric acid (UA), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), small dense low-density lipoprotein cholesterol (sdLDL-C), etc. were recorded. Islet function was determined using a 100 g steamed bread meal test, and C-peptide detection was performed using the Cobas e601 chemiluminescence analyser from Roche. The modified homeostasis model assessment method was applied to calculate the C-peptide resistance index (HOMA-CR) and islet function (HOMA-β). HOMA-CR = 1.5 + FPG × FCP/(2.8 × 0.333), HOMA-β = 270 × FCP/[0.333 × (FBG – 3.5)], where the unit of FCP is ng/mL and the unit of FPG is mmol/L [3]. WC/HC (WHR) = the ratio of WC to HC. TyG = Ln [TG (mg/dL) × FBG (mg/dL)/2], TyG-BMI = TyG × BMI (kg/m^2), TyG-WC = TyG × WC (cm) [2, 4]. The controlled attenuation parameter (CAP) of the liver for assessing the severity of hepatic steatosis was measured by the transient elastography technique iLivTouch (Haisiker, China). CAP ≥ 244 dB/m indicated the presence of hepatic steatosis in patients [5]. The specific method was in accordance with the user manual and was measured by the same doctor who received professional training.

Statistical analysis

Statistical analysis was performed using SPSS 22.0 software. Normal distribution measurement data were expressed as mean ± standard deviation, and non-normal distribution data were expressed as median (quartile range). T-tests or rank sum tests were conducted, and chi-square tests were performed for count data. Correlations were analysed using Spearman's correlation analysis. Multivariate Logistic regression analysis was conducted with high-BMI or low-BMI T2DM combined with hepatic steatosis as the dependent variables, respectively. The cut-off values of TyG and related indicators for predicting the occurrence of hepatic steatosis in T2DM patients with different BMIs were determined by the receiver operating characteristic (ROC) curve. $P < 0.05$ was considered statistically significant.

Results

Among 221 T2DM patients, the rate of hepatic steatosis was 85.83% in the high-BMI group and 43.62% in the low-BMI group. WC, WHR, WBC, UA, ALT, AST, TG, sdLDL-C, HOMA-CR, HOMA-β, TyG, TyG-BMI, and TyG-WC were significantly higher in the former than in the latter. Age, disease duration, and HDL-C were lower in the high-BMI group than in the low-BMI group ($p < 0.01$ or $p < 0.05$) (Tab. 1).

Among high-BMI T2DM patients, BMI, WC, WHR, FBG, 2hPG, AST, TG, sdLDL-C, HOMA-CR, TyG, TyG-BMI, and TyG-WC were significantly higher in the hepatic steatosis group than in the non-hepatic steatosis group ($p < 0.01$ or $p < 0.05$). Among low-BMI T2DM patients with hepatic steatosis, BMI, WC, UA, TG, sdLDL-C, HOMA-β, TyG, TyG-BMI, and TyG-WC were higher than those in the non-hepatic steatosis group, while HbA_{1c} and HDL-C were lower in the former than in the latter ($p < 0.01$ or $p < 0.05$) (Tab. 2).

Spearman correlation analysis revealed that CAP had positive correlations with BMI, WC, WHR, FBG, 2hPG, AST, TG, sdLDL-C, HOMA-CR, TyG, TyG-BMI, and TyG-WC in the high BMI group ($p < 0.01$ or $p < 0.05$). In the low-BMI group, BMI, WC, TC, TG, sdLDL-C, HOMA-β, TyG, TyG-BMI, and TyG-WC were positively correlated with CAP, while HbA_{1c} was negatively correlated ($p < 0.01$ or $p < 0.05$).

A multifactorial Logistic regression analysis was performed. Taking the occurrence of hepatic steatosis in high-BMI T2DM patients as the dependent variable, and BMI, WC, WHR, FBG, 2hPG, AST, TG, sdLDL-C, HOMA-CR, TyG, TyG-BMI, and TyG-WC as independent variables, TyG-BMI was an independent risk factor for hepatic steatosis in high-BMI T2DM patients ($p < 0.01$) (Tab. 3). Taking the occurrence of hepatic steatosis in low-BMI T2DM patients as the dependent variable, and BMI, WC, TC, TG, sdLDL-C, HOMA-β, TyG, TyG-BMI, and TyG-WC as independent variables, HbA_{1c} and TyG-WC were independent risk factors for hepatic steatosis in low-BMI T2DM patients ($p < 0.01$ or $p < 0.05$) (Tab. 3).

The ROC curve for predicting the occurrence of hepatic steatosis in high-BMI T2DM patients showed that the area under the curve of TyG-BMI was 0.84 ($p < 0.01$), the cut-off value was 241.36, the sensitivity was 79.80%, and the specificity was 83.30% (Fig. 1). The ROC curve for predicting the occurrence of hepatic steatosis in low-BMI T2DM patients showed that the area under the TyG-WC curve was 0.80 ($p < 0.01$), the cut-off value was 824.63, the sensitivity was 68.30%, and the specificity was 84.90% (Fig. 2).

Discussion

Most T2DM patients are overweight or obese, often combined with multiple metabolic disorders such as hypertension, hyperlipidaemia, and hyperuricaemia. The incidence of MAFLD in overweight or obese T2DM patients also increases significantly, reportedly reaching more than 50% [6], suggesting that there may be a common pathogenesis – insulin resistance – in such patients. Through comprehensive management of diabetes, including weight management and the emer-

Table 1. *Comparison of clinical data of type 2 diabetes mellitus (T2DM) patients in different body mass index (BMI) groups [± s, or median (interquartile range)]*

Parameters	High BMI	Low BMI	t/ ^{2} value	p-value
No. of patients	127	94		
Male/female	77/50	54/40	0.23	0.63
Age [year]	52.39 ± 11.66	58.33 ± 8.56	–4.18	0.00
Diabetes duration [year]	7.77 ± 7.36	10.53 ± 7.23	–2.78	0.01
WC [cm]	97.17 ± 8.43	84.84 ± 7.59	11.22	0.00
WHR	0.96 ± 0.08	0.91 ± 0.06	5.48	0.00
Hepatic steatosis n (%)	109 (85.83)	41 (43.62)	44.14	0.00
CAP [dB/m]	278.58 ± 31.77	235.70 ± 32.19	9.86	0.00
WBC (×109/L)	7.33 ± 1.97	6.62 ± 1.96	2.64	0.01
FBG (mmol/L)	10.86 ± 4.53	10.32 ± 3.66	0.94	0.35
2hPG (mmol/L)	18.16 ± 4.92	17.32 ± 5.08	1.23	0.22
HbA_{1c} (%)	9.31 ± 1.88	9.64 ± 1.93	–1.28	0.20
UA [umol/L]	326.18 ± 106.71	284.73 ± 83.96	3.12	0.00
ALT [U/L]	35.85 (16, 41)	20.83 (13.75, 24.25)	–4.51	0.00
AST [U/L]	29.23 (16, 32)	19.58 (14.75, 21.25)	–3.84	0.00
TC [mmol/L]	4.89 ± 4.10	4.49 ± 1.27	0.91	0.37
TG [mmol/L]	2.80 ± 2.24	2.04 ± 2.16	2.53	0.01
HDL-C [mmol/L]	1.13 ± 0.41	1.24 ± 0.38	–2.03	0.04
sdLDL-C [mmol/L]	1.34 ± 0.71	1.06 ± 0.73	2.87	0.00
HOMA-CR	29.73 (13.32, 38.06)	19.59 (10.26, 25.73)	–4.42	0.00
HOMA-b	349.14 (167.28, 474.34)	258.69 (111.71, 340.27)	–3.24	0.00
TyG	9.77 ± 0.96	9.40 ± 0.78	3.08	0.00
TyG-BMI	265.69 ± 40.37	205.99 ± 24.15	12.74	0.00
TyG-WC	951.71 ± 140.05	798.21 ± 100.49	9.04	0.00

BMI — body mass index; WC — waist circumference; WHR — waist-to-hip ratio; CAP — controlled attenuation parameter; WBC — white blood cells count; FBG — fasting blood glucose; 2hPG — 2-hour postprandial blood glucose; HbA_{1c} — glycated haemoglobin; UA — uric acid; ALT — alanine aminotransferase; AST — aspartate aminotransferase; TC — total cholesterol; TG — triglycerides; HDL-C — high-density lipoprotein cholesterol; sdLDL-C — small dense low-density lipoprotein cholesterol; HOMA-CR — C-peptide resistance index; HOMA-β — inlet function; TyG — triglyceride glucose index; TyG-BMI — triglyceride glucose-BMI index; TyG-WC — triglyceride glucose-WC

gence of new hypoglycaemic drugs, various metabolic disorder factors can be controlled or even reversed [7]. This study showed that WC, WHR, WBC, UA, ALT, AST, TG, sdLDL-C, HOMA-CR, and HOMA-β in high-BMI T2DM patients were significantly higher than those in normal-weight or lean patients, suggesting that overweight or obese T2DM patients were more prone to metabolic disorders, insulin resistance, and mild inflammation in the body. Correspondingly, overweight or obese T2DM patients were younger and had a shorter duration of diabetes, and they were more likely to achieve diabetes remission.

The pathophysiological change of MAFLD is marked by the long-term deposition of lipids in the liver leading to hepatic steatosis. Liver biopsy is the gold standard, but this examination is an invasive test and is difficult to popularise in clinical practice. In recent years, studies have shown that transient elastography can effectively evaluate hepatic steatosis [8]. In this study, CAP was determined by transient elastography to evaluate hepatic steatosis. The influencing factors of hepatic steatosis in T2DM patients are complex. Studies have shown that it is related to obesity, abnormal blood glucose, dyslipidaemia, visceral fat, inflammatory indicators, etc. [9, 10]. The study showed that in high-BMI T2DM patients, the incidence of hepatic steatosis was high, reaching 85.83%. In this group, BMI, WC, WHR, FBG, 2hPG, AST, TG, sdLDL-C, and HOMA-CR were significantly higher than those in the non-hepatic steatosis group, and the above indicators were positively correlated with the CAP value. There was also a certain proportion of hepatic steatosis in low-BMI T2DM patients, reaching 43.26%. BMI, WC, UA, TG, sdLDL-C, and HOMA-β in patients with hepatic steatosis were higher than those in the non-hepatic steatosis group, and the above

Table 2. *Comparison of clinical data between the hepatic steatosis group and the non-hepatic steatosis group in type 2 diabetes mellitus (T2DM) patients with different body mass indexes (BMIs) [±s, or median (interquartile range)]*

Parameters	High BMI				Low BMI			
	Hepatic steatosis	Non-hepatic steatosis	t/X² value	p-value	Hepatic steatosis	Non-hepatic steatosis	t/X² value	p-value
No. of patients	109	18			41	53		
Male/female	67/ 42	10/ 8	0.23	0.63	21/ 20	33/ 20	1.15	0.28
Age [years]	52.34 ± 11.92	52.72 ± 10.16	–0.13	0.90	59.00 ± 8.79	57.81 ± 8.42	0.67	0.51
Diabetes duration [years]	7.49 ± 7.31	9.46 ± 7.64	–1.05	0.29	10.27 ± 6.82	10.73 ± 7.59	–0.30	0.76
WC [cm]	97.99 ± 8.67	92.22 ± 4.67	2.76	0.01	88.12 ± 6.74	82.30 ± 7.27	3.97	0.00
WHR	0.97 ± 0.07	0.92 ± 0.11	2.56	0.01	0.92 ± 0.08	0.90 ± 0.06	1.25	0.22
BMI [kg/m²]	27.40 ± 2.65	25.52 ± 1.24	2.95	0.00	22.54 ± 1.08	21.36 ± 1.45	4.34	0.00
WBC [×10⁹/L]	7.39 ± 2.05	7.00 ± 1.36	0.77	0.44	6.99 ± 2.28	6.35 ± 1.65	1.58	0.12
FBG [mmol/L]	11.34 ± 4.59	7.91 ± 2.76	3.08	0.00	10.42 ± 3.73	10.25 ± 3.64	0.22	0.83
2hPG [mmol/L]	18.62 ± 4.89	15.35 ± 4.19	2.68	0.01	16.71 ± 4.09	17.80 ± 5.73	–1.04	0.30
HbA_{1c} (%)	9.41 ± 1.92	8.74 ± 1.55	1.41	0.16	9.12 ± 1.72	10.01 ± 2.01	–2.13	0.04
UA [umol/L]	333.69 ± 110.42	280.71 ± 66.22	1.97	0.05	333.69 ± 110.42	280.71 ± 66.22	2.71	0.01
ALT [U/L]	38.06 ± 33.10	22.50 ± 14.90	1.96	0.05	21.10±11.13	20.62 ± 13.12	0.19	0.85
AST [U/L]	30.20 (16.5, 32)	23.33 (13.75, 24)	–2.12	0.03	18.63 ± 5.08	20.31 ± 12.57	–0.81	0.42
TC [mmol/L]	5.01 ± 4.39	4.19 ± 1.12	0.79	0.43	4.78 ± 1.15	4.27 ± 1.32	1.20	0.05
TG [mmol/L]	3.00 ± 2.31	1.59 ± 1.21	2.52	0.01	2.63 (1.35, 2.78)	1.59 (0.95, 1.66)	–3.49	0.00
HDL-C [mmol/L]	1.12 ± 0.43	1.20 ± 0.27	–0.75	0.45	1.15 ± 0.22	1.32 ± 0.45	–2.19	0.03
SdLDL-C [mmol/L]	1.41 ± 0.72	0.90 ± 0.44	2.89	0.01	1.26 ± 0.80	0.88 ± 0.63	2.48	0.02
HOMA-CR	31.52 (19.01, 28)	18.90 (7.48, 23.51)	–3.18	0.00	23.81 (11.19, 27.75)	16.33 (9.23, 22.35)	–1.66	0.10
HOMA-b	348.02 (152.62, 483.62)	355.92 (242.45, 410.19)	–1.29	0.19	303.73 (151.45, 456.95)	223.84 (85.51, 302.89)	–2.19	0.03
TyG	9.91 ± 0.91	8.91 ± 0.88	4.09	0.00	9.65 ± 0.84	9.21 ± 0.67	2.87	0.01
TyG-BMI	271.79 ± 39.27	228.76 ± 24.46	4.50	0.00	217.88 ± 24.05	196.79 ± 20.01	5.06	0.00
TyG-WC	972.38 ± 136.54	826.55 ± 87.27	4.38	0.00	851.22 ± 100.86	757.20 ± 79.41	4.64	0.00

BMI — body mass index; WC — waist circumference; WHR — waist-to-hip ratio; CAP — controlled attenuation parameter; WBC — white blood cells count; FBG — fasting blood glucose; 2hPG — 2-hour postprandial blood glucose; HbAlc — glycated haemoglobin; UA — uric acid; ALT — alanine aminotransferase; AST — aspartate aminotransferase; TC — total cholesterol; TG — triglycerides; HDL-C — high-density lipoprotein cholesterol; sdLDL-C — small dense low-density lipoprotein cholesterol; HOMA-CR — C-peptide resistance index; HOMA-β — inlet function; TyG — triglyceride glucose index; TyG-BMI — triglyceride glucose-BMI index; TyG-WC — triglyceride glucose-WC

Table 3. *Logistic regression analysis of hepatic steatosis in type 2 diabetics with different body mass indexes (BMIs)*

	β	Standard error	Wald statistic	p-value	Odds ratio	95% confidence interval
High BMI						
TyG-BMI	0.048	0.012	15.383	0.000	1.049	1.024–1.075
Low BMI						
HbA_{1c}	–0.315	0.145	4.742	0.029	0.730	0.549–0.969
TyG-WC	0.013	0.003	16.396	0.000	1.013	1.007–1.020

BMI — body mass index; WC — waist circumference; HbAlc — glycated haemoglobin; TyG — triglyceride glucose index; TyG-BMI — triglyceride glucose-BMI index; TyG-WC — triglyceride glucose-WC

indicators were positively correlated with the CAP value, suggesting that there were certain differences in metabolic indicators of hepatic steatosis in different BMI T2DM populations. The occurrence of hepatic

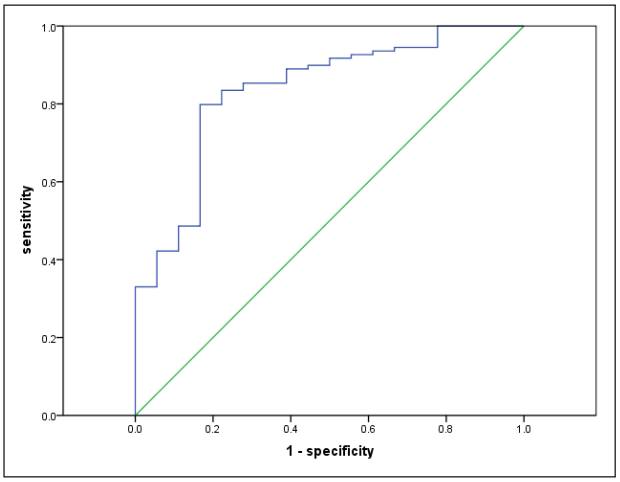

Figure 1. *Receiver operating characteristic (ROC) curves of triglyceride glucose-BMI index (TyG-BMI) for predicting hepatic steatosis in type 2 diabetics with high body mass index (BMI)*

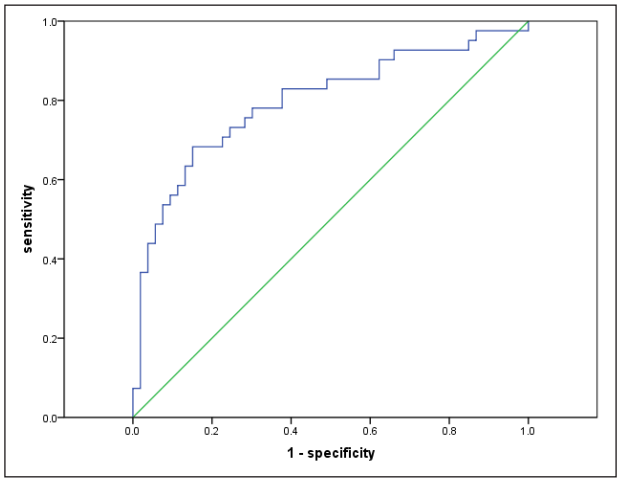

Figure 2. *Receiver operating characteristic (ROC) curves of triglyceride glucose-waist circumference (TyG-WC) for predicting hepatic steatosis in type 2 diabetics with low body mass index (BMI)*

steatosis in overweight or obese patients was more closely related to disorders of glucose metabolism, liver function, and insulin resistance, while normal-sized or lean patients were more closely related to blood uric acid and islet function, and both were related to BMI and dyslipidaemia; in particular, TG and sdLDL-C were significantly correlated with hepatic steatosis in different BMI populations. HbA_{lc} was negatively correlated with the occurrence of hepatic steatosis in low-BMI T2DM patients, and the reason was difficult to explain. It may be related to the deposition of visceral fat in patients, including the pancreas, affecting islet function and large fluctuations in blood glucose [11].

The TyG index contains TG and FBG and can be used as one of the important indicators for predicting insulin resistance, and it is related to cardiovascular diseases. The addition of obesity indicators such as BMI and WC to TyG gives rise to the TyG-BMI and TyG-WC indices, which can predict liver fat lesions [2, 12]. The latest studies have shown that high levels of TyG-BMI and TyG-WC are associated with an increased mortality rate in patients with diabetes and hepatic steatosis [13]. There are no studies on the differences in liver steatosis occurrence at different BMIs regarding TyG-BMI and TyG-WC. This study showed that regardless of BMI, TyG, TyG-BMI, and TyG-WC were significantly higher in the hepatic steatosis group than in the non-hepatic steatosis group, and all 3 were significantly correlated with CAP. Regression analysis showed that TyG-BMI was closely related to the occurrence of hepatic steatosis in high-BMI T2DM patients, and HbAlc and TyG-WC were independent risk factors for hepatic steatosis in low-BMI T2DM patients. The cut-off value of TyG-BMI for predicting the occurrence of hepatic steatosis in high-BMI T2DM patients was 241.36, and the cut-off value of TyG-WC for predicting the occurrence of hepatic steatosis in low-BMI T2DM patients was 824.63, both with good sensitivity and specificity. BMI and WC are important indicators reflecting obesity; in particular, WC can better reflect visceral fat deposition and metabolic disorders, and thus WC is one of the diagnostic indicators of metabolic syndrome. For low-BMI patients, excluding the influence of BMI, WC can more accurately reflect the obese state of patients. Therefore, TyG-WC can better predict the occurrence of hepatic steatosis in low-BMI T2DM patients.

Conclusions

In T2DM patients, regardless of BMI, the occurrence of hepatic steatosis is closely related to BMI, WC, and lipid metabolism disorders. TyG-BMI can predict the occurrence of hepatic steatosis in high-BMI patients, and TyG-WC can predict the occurrence of hepatic steatosis in low-BMI patients, while the latter is negatively correlated with HbA_{lc}, showing certain differences. This study is a single-centre study with a small sample size, and it requires multi-centre large samples for further verification.

Data availability statement

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Ethics statement

This study was approved by the Ethics Committee of Jiangdu People's Hospital Affiliated to Yangzhou University on 26 December 2022 (Approval Number: YJRY-2022-K-033).

Author contributions

CM: writing the original draft, interpretation of data, and discussion of results. YC: conception, study design, and acquisition of data. PD: proofreading and final approval of the manuscript. JL: drafting and critical revision of the manuscript. XS: data analysis and statistical evaluation. YZ: literature review and acquisition of data.

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Conflict of interest

The authors declare that they have no competing interests.

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