

RESEARCH ARTICLE

Triglyceride–glucose index and liver fibrosis in chronic hepatitis C

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ABSTRACT

Background: Insulin resistance is associated with chronic hepatitis C virus (HCV) infection and liver fibrosis. The triglyceride–glucose (TyG) index is a low-cost surrogate derived from fasting triglyceride and glucose. This study aims to examine the association between TyG, fibrosis severity, glycemic status, and selected cardiometabolic variables. Methods: Cross-sectional study of 88 adults with chronic HCV at Binh Thanh District Hospital, Vietnam, from May 2020 to June 2021. Results: Mean TyG was 4.14 ± 0.52 and 47.7% met the study criterion for insulin resistance. TyG-defined insulin resistance increased from 18.2% in F0 to 79.3% in F4 ($p < 0.01$). TyG correlated with liver stiffness ($r = 0.35$, $p < 0.01$), HOMA-IR ($r = 0.23$, $p < 0.05$), systolic blood pressure ($r = 0.22$, $p < 0.05$), and inversely with HDL-C ($r = -0.24$, $p < 0.05$). Conclusion: Higher TyG was associated with more advanced fibrosis and an adverse metabolic profile.

KEYWORDS

Chronic hepatitis C; triglyceride–glucose index; insulin resistance; liver fibrosis; transient elastography; HOMA-IR.

ARTICLE INFORMATION

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1. Introduction

Chronic hepatitis C virus (HCV) infection is a systemic disease with metabolic consequences in addition to progressive liver injury. Insulin resistance, impaired glucose homeostasis, type 2 diabetes, and hepatic steatosis have all been described in chronic HCV (Bose & Ray, 2014; Romero-Gómez, 2006). Higher insulin resistance has also been associated with more advanced fibrosis (Hui et al., 2003; Moucari et al., 2008, 2013).

The euglycemic–hyperinsulinemic clamp is a reference method for insulin sensitivity, but it is impractical for routine use. HOMA-IR is widely used but requires fasting insulin (Wallace et al., 2004). The triglyceride–glucose (TyG) index uses routinely available fasting triglyceride and glucose and has been validated against the clamp in other populations (Guerrero-Romero et al., 2010; Simental-Mendía et al., 2008). Reported cutoffs vary by population and calculation method (Salazar et al., 2017).

Because insulin resistance is linked to fibrosis in HCV, a clinically relevant question is whether TyG also tracks with liver fibrosis severity. This study investigated the association between TyG and fibrosis stage, glycemic status, and selected cardiometabolic variables in Vietnamese adults with chronic HCV infection.

2. Literature Review

HCV-related inflammation and interference with insulin signaling may reduce hepatic and peripheral insulin action, while hyperinsulinemia and altered lipid metabolism may promote steatosis, oxidative stress, and profibrogenic pathways (Bose & Ray, 2014; Samuel & Shulman, 2012). Clinical studies using HOMA-IR have consistently reported associations between insulin resistance and significant or progressive fibrosis (Hui et al., 2003; Moucari et al., 2008, 2013).

Treatment studies support a virus–metabolism interaction: insulin resistance has improved after HCV clearance in both interferon-era and direct-acting antiviral studies (Adinolfi et al., 2018; Kawaguchi et al., 2007). TyG may therefore provide practical metabolic information when insulin assays are unavailable, but it should not be assumed interchangeable with HOMA-IR without disease-specific validation.

3. Methodology

3.1 Study design and participants

A cross-sectional study was conducted among 88 eligible adults with chronic HCV infection attending the Outpatient Department of Binh Thanh District Hospital, Ho Chi Minh City, Vietnam, from May 2020 to June 2021. Participants were aged 18 years or older and met the 2016 Vietnamese Ministry of Health diagnostic criteria for chronic HCV infection. Sampling was based on eligible patients presenting during the study period.

3.2 Eligibility criteria

Chronic infection was defined by an HCV infection duration greater than six months with positive anti-HCV and detectable HCV-RNA or positive HCV core antigen. Participants were excluded if they were receiving medications known to substantially alter insulin sensitivity or glucose metabolism; had severe hepatic failure, severe renal failure, or hepatic coma; had HIV or HBV coinfection or another cause of hepatitis; or had endocrine disorders that could materially affect glucose metabolism. All participants provided informed consent.

3.3 Clinical and laboratory measurements

Age, sex, smoking, physical activity, blood pressure, BMI, and waist circumference were recorded using standardized procedures. After an overnight fast, blood was collected for glucose, triglycerides, total cholesterol, HDL-C, LDL-C, and insulin. HOMA-IR was calculated as fasting insulin ($\mu\text{U/mL}$) $\times$ fasting glucose (mmol/L) / 22.5; HOMA-IR ≥ 2.6 indicated insulin resistance. TyG was calculated from fasting triglyceride and glucose using the original study formula, with TyG ≥ 4.11 as the study-specific threshold.

3.4 Liver fibrosis assessment

Liver stiffness was measured using transient elastography (FibroScan). At least 10 successful measurements were obtained per participant, and fibrosis was categorized according to the study protocol as F0 (1–4.9 kPa), F1 (5–7 kPa), F2 (7.1–9.5 kPa), F3 (9.6–12.5 kPa), and F4 (> 12.5 kPa). These categories were used to evaluate whether the prevalence of TyG-defined insulin resistance increased with fibrosis severity.

3.5 Statistical analysis

Analyses were performed using SPSS version 20.0. Categorical variables were summarized as counts and percentages. Continuous variables were summarized as mean $\pm$ standard deviation or median and range, depending on distribution. Group differences were examined using chi-square tests and appropriate parametric or nonparametric tests. Pearson or Spearman correlation was used for continuous variables according to distributional assumptions. Statistical significance was defined as $p < 0.05$.

3.6 Ethical considerations

The study was approved by the Biomedical Research Ethics Committee of Hue University of Medicine and Pharmacy. Participants received information about the study and provided consent before enrollment. Personal information was kept confidential.

4. Results

The study population consisted of 88 adults with chronic HCV infection. As summarized in Table 1, mean age was 64.5 ± 9.1 years, 78.4% were aged 60 years or older, and 58.0% were women. Mean BMI was 23.2 ± 3.5 kg/m^2 ; 56.8% were overweight or obese and 55.7% had abdominal obesity. Hypertension was present in 36.4%. Two-thirds of the cohort had fibrosis stage F2 or higher, and 33.0% had F4 cirrhosis. Mean TyG was 4.14 ± 0.52 ; 47.7% met the study TyG criterion for insulin resistance, while 48.9% had HOMA-IR ≥ 2.6 .

Table 1. Selected characteristics of the study population

Characteristic	Value
Age, mean $\pm$ SD (years)	64.5 ± 9.1
Women, n (%)	51 (58.0)
BMI, mean $\pm$ SD (kg/m^2)	23.2 ± 3.5
Overweight/obesity, n (%)	50 (56.8)
Abdominal obesity, n (%)	49 (55.7)
Hypertension, n (%)	32 (36.4)
Fasting glucose, mean $\pm$ SD (mmol/L)	6.1 ± 2.1

Characteristic	Value
TyG, mean $\pm$ SD	4.14 $\pm$ 0.52
TyG-defined insulin resistance, n (%)	42 (47.7)
HOMA-IR $\geq$ 2.6, n (%)	43 (48.9)

A clear gradient was observed across fibrosis stages (Table 2). TyG-defined insulin resistance was present in 18.2% of patients at F0, 27.8% at F1, 31.6% at F2, 54.5% at F3, and 79.3% at F4. The distribution differed significantly across fibrosis categories ($p < 0.01$), with the greatest proportion observed among patients with cirrhosis.

Table 2. TyG-defined insulin resistance by liver fibrosis stage

Fibrosis stage	TyG $\geq$ 4.11 n (%)	TyG $<$ 4.11 n (%)
F0	2 (18.2)	9 (81.8)
F1	5 (27.8)	13 (72.2)
F2	6 (31.6)	13 (68.4)
F3	6 (54.5)	5 (45.5)
F4	23 (79.3)	6 (20.7)

TyG-defined insulin resistance also increased across fasting glucose categories (Table 3). It was present in 31.5% of participants with fasting glucose $<$ 5.6 mmol/L, 57.9% of those with glucose 5.6 to $<$ 7.0 mmol/L, and 93.3% of those with glucose $\geq$ 7.0 mmol/L ($p < 0.05$). In addition, all 24 participants with triglycerides $\geq$ 1.7 mmol/L were classified as insulin resistant by the study TyG threshold, compared with 28.1% among those with triglycerides $<$ 1.7 mmol/L ($p < 0.01$). These associations were expected in part because glucose and triglyceride are direct components of the TyG formula.

Table 3. TyG-defined insulin resistance across fasting glucose categories

Fasting glucose	TyG $\geq$ 4.11 n (%)	TyG $<$ 4.11 n (%)
$<$ 5.6 mmol/L	17 (31.5)	37 (68.5)
5.6– $<$ 7.0 mmol/L	11 (57.9)	8 (42.1)
$\geq$ 7.0 mmol/L	14 (93.3)	1 (6.7)

Correlations between continuous TyG values and clinical or metabolic variables are presented in Table 4. TyG correlated positively with liver stiffness ($r = 0.35$, $p < 0.01$), HOMA-IR ($r = 0.23$, $p < 0.05$), and systolic blood pressure ($r = 0.22$, $p < 0.05$), and inversely with HDL-C ($r = -0.24$, $p < 0.05$). Correlations with age, BMI, waist circumference, diastolic blood pressure, total cholesterol, LDL-C, and fasting insulin did not reach statistical significance.

Table 4. Correlations between TyG and clinical/metabolic variables

Variable	r	p
Age	-0.07	$>$ 0.05
BMI	0.12	$>$ 0.05
Waist circumference	0.15	$>$ 0.05
Systolic blood pressure	0.22	$<$ 0.05
Diastolic blood pressure	0.20	$>$ 0.05
Total cholesterol	0.19	$>$ 0.05
HDL-C	-0.24	$<$ 0.05
LDL-C	0.05	$>$ 0.05
HOMA-IR	0.23	$<$ 0.05
Fasting insulin	0.16	$>$ 0.05

Variable	r	p
FibroScan liver stiffness	0.35	<0.01

5. Discussion

The main finding was a graded relationship between TyG and liver fibrosis. As shown in Table 2, TyG-defined insulin resistance increased from 18.2% at F0 to 79.3% at F4, and TyG correlated with liver stiffness in Table 4. This is consistent with previous HCV studies linking insulin resistance to significant fibrosis and fibrosis progression (Hui et al., 2003; Moucari et al., 2008, 2013).

HCV-related inflammation and disruption of insulin signaling may impair hepatic and peripheral insulin action, while altered glucose and lipid metabolism may promote steatosis, oxidative stress, and profibrogenic signaling (Bose & Ray, 2014; Romero-Gómez, 2006; Samuel & Shulman, 2012). Advanced liver disease may also worsen insulin clearance and glucose homeostasis. Because the study is cross-sectional, it cannot establish direction or causality, and multivariable analysis would be needed to determine whether TyG is independently associated with fibrosis.

Table 3 showed increasing TyG-defined insulin resistance across fasting glucose categories, and all participants with triglycerides ≥ 1.7 mmol/L met the TyG threshold. These associations are partly expected because glucose and triglyceride are direct components of TyG. More informative were the correlations with variables not included in the formula: TyG correlated positively with systolic blood pressure and HOMA-IR and inversely with HDL-C (Table 4), suggesting a broader adverse cardiometabolic phenotype.

The TyG–HOMA-IR correlation was significant but weak ($r=0.23$). HOMA-IR combines fasting insulin and glucose, whereas TyG combines triglyceride and glucose; they therefore capture related but different metabolic signals. Clamp studies support TyG as a practical marker in other populations (Guerrero-Romero et al., 2010), but the cutoff varies across populations (Salazar et al., 2017). TyG should therefore be considered a screening surrogate rather than an interchangeable substitute for HOMA-IR in chronic HCV.

The absence of significant associations with BMI and waist circumference, despite the high prevalence of overweight/obesity and abdominal obesity in Table 1, may reflect limited sample size, the older study population, HCV-specific mechanisms, or changes in body composition associated with chronic liver disease. Treatment studies showing improved insulin resistance after viral clearance (Adinolfi et al., 2018; Kawaguchi et al., 2007) further support the clinical relevance of metabolic assessment in HCV.

TyG is attractive because fasting glucose and triglycerides are inexpensive and widely available. Nevertheless, this study is limited by its cross-sectional design, convenience sampling at one hospital, modest sample size, lack of multivariable regression and ROC analysis, and absence of detailed genotype, antiviral-treatment, and steatosis data. Most importantly, the possible triglyceride conversion error in the source thesis requires verification of all TyG-derived values before submission. Prospective multicenter studies using standardized calculation and validated cutoffs are needed.

The study has several limitations. First, its cross-sectional design precludes causal inference. Second, convenience sampling from a single hospital limits generalizability. Third, the sample size was modest, particularly after stratification by fibrosis stage. Fourth, no multivariable regression was reported to determine whether TyG was independently associated with fibrosis after adjustment for age, sex, obesity, glycemia, or other confounders. Fifth, the original thesis document contains a possible error in triglyceride unit conversion used for TyG calculation; therefore, all TyG-derived results must be verified from the raw dataset before publication. Finally, HCV genotype, antiviral treatment status, and steatosis measures were not incorporated into the reported analysis.

Despite these limitations, the observed dose-response pattern across fibrosis stages is clinically interesting. If confirmed after recalculation and multivariable adjustment, TyG could offer a low-cost metabolic marker that complements liver-focused assessment in chronic HCV care, especially in resource-limited settings where insulin assays are not routinely available.

6. Conclusion

Among 88 Vietnamese adults with chronic HCV infection, higher TyG values were associated with more advanced liver fibrosis, higher systolic blood pressure, lower HDL-C, and higher HOMA-IR. The prevalence of TyG-defined insulin resistance rose progressively across fibrosis stages and was highest in F4 cirrhosis. TyG may offer a low-cost metabolic adjunct to routine HCV assessment, particularly where insulin assays are not readily available. However, it should not be considered a substitute for validated insulin-resistance methods or a direct marker of fibrosis until its calculation, cutoff, diagnostic performance, and independent association with liver outcomes are confirmed.

7. Statements and Declarations

Ethics approval and consent to participate: The study was approved by the Biomedical Research Ethics Committee of Hue University of Medicine and Pharmacy. All participants provided informed consent.

Consent for publication: Not applicable.

Availability of data and materials: The dataset is available from the corresponding author on reasonable request, subject to institutional and participant-confidentiality requirements.

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