Association of Triglyceride-to-HDL Cholesterol ratio with Body Mass Index in Nepalese Adults with Non-Alcoholic Fatty Liver Disease

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ABSTRACT

Background

The triglyceride-to-high-density lipoprotein cholesterol (TG/HDL-C) ratio is an emerging marker of insulin resistance and metabolic dysfunction, even among non-obese individuals. Evidence from South Asia, particularly Nepal, remains limited.

Objective

To assess the association between TG/HDL-C ratio and BMI and to evaluate its distribution across BMI categories among adults with Non-Alcoholic Fatty Liver Disease (NAFLD).

Method

A cross-sectional study was conducted among 400 adults with ultrasonography-confirmed NAFLD attending a tertiary care hospital in Nepal. Participants were categorized into BMI groups, and fasting lipid profiles were obtained. The TG/HDL-C ratio was calculated, and values > 2.4 were considered elevated based on previously published evidence demonstrating its association with advanced non-alcoholic fatty liver disease and hepatic fibrosis. Associations were evaluated using correlation analysis, group comparisons across BMI categories, and multivariable linear regression.

Result

The median BMI and TG/HDL-C ratio of the participants were 30.04 (IQR: 26.06 – 32.84) kg/m² and 3.91 (IQR: 3.12 – 5.26), respectively. The TG/HDL-C ratio showed a weak positive correlation with BMI ($\rho = 0.302$, $p < 0.001$) and a very weak negative correlation with age ($\rho = -0.142$, $p = 0.004$). Significant differences in TG/HDL-C ratio were observed across BMI categories ($p < 0.001$), with the highest median values among obese individuals. Elevated TG/HDL-C ratio was present in 84% of participants and remained highly prevalent even among normal-weight individuals (75.8%). BMI remained an independent predictor of TG/HDL-C ratio, where each 5 kg/m2 increase in BMI was associated with a 0.705 unit increase in the ratio ($\beta=0.705$; $p <0.001$) after adjusting for age, sex, and LDL cholesterol.

Conclusion

TG/HDL-C ratio was significantly associated with BMI and was highly prevalent even among normal-weight individuals with NAFLD. This lipid ratio may serve as a simple and cost-effective marker for identifying metabolic risk in clinical settings.

KEY WORDS

Body mass Index, Dyslipidemia, Metabolic risk, Non-alcoholic fatty liver disease*, TG/HDL-C ratio*

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) has emerged as the most common chronic liver disease globally, with a prevalence exceeding 30%, particularly in Asian populations.[1,2] Traditionally, NAFLD has been closely associated with obesity, insulin resistance, and dyslipidemia. However, increasing evidence indicates that NAFLD can also occur in individuals with normal body mass index (BMI), a condition termed lean NAFLD.[3]

The South Asian population is particularly predisposed to this phenotype due to a distinct metabolic profile characterized by increased visceral adiposity, higher hepatic fat accumulation, and greater insulin resistance even at lower BMI levels.[4] This highlights the limitation of BMI as a sole marker of metabolic risk.

Among emerging lipid indices, the triglyceride-to-high-density lipoprotein cholesterol (TG/HDL-C) ratio has gained attention as a simple and reliable surrogate marker of insulin resistance and metabolic dysfunction.[5] Studies have demonstrated that TG/HDL-C ratio is strongly associated with NAFLD and may serve as a useful predictor of hepatic steatosis.[6,7]

Recent evidence further indicates that TG/HDL-C ratio is associated with metabolic dysfunction–associated fatty liver disease (MAFLD) and increased cardiovascular risk.[8,9] In addition, several studies have shown that TG/HDL-C ratio is independently associated with NAFLD across different BMI categories, including non-obese individuals.[10-13]

Despite these findings, data from Nepal remain limited, particularly regarding the role of TG/HDL-C ratio among non-obese individuals. Therefore, this study aims to assess the association between TG/HDL-C ratio and BMI, examine its relationship with lipid parameters, and determine whether BMI independently predicts TG/HDL-C ratio among Nepalese adults with NAFLD.

METHODS

This cross-sectional analytical study was performed among 400 adults with NAFLD attending a tertiary hospital in Nepal. The diagnosis of NAFLD was based on abdominal ultrasonography interpreted by an experienced radiologist using standard sonographic criteria for hepatic steatosis. The participants were consecutively enrolled over eight months (July 2024 - February 2025) post ethical approval from the Institutional Review Committee, Kathmandu University School of Medical Sciences (approval no. 186/24). Individuals with significant alcohol consumption (> 4 drinks per day), or on lipid-lowering, anti-hypertensives, or anti-diabetic medications were excluded. Informed written consent was obtained from all participants.

Demographic data were recorded using a structured Proforma. Weight and height were measured to calculate BMI (kg/m^2). BMI was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Participants were categorized according to the WHO Asia-Pacific BMI classification as underweight (< 18.5 kg/m^2), normal weight (18.5–22.9 kg/m^2), overweight (23.0–24.9 kg/m^2), obese class I (25.0–29.9 kg/m^2), and obese class II (≥ 30 kg/m^2).[14]

Fasting lipids (TG, HDL, LDL) were obtained from the Biochemistry department of the central hospital laboratory. TG/HDL-C ratio was calculated by dividing triglyceride (mg/dl) by HDL-cholesterol (mg/dl). A TG/HDL-C ratio > 2.4 was considered elevated based on previously published evidence demonstrating its association with advanced non-alcoholic fatty liver disease and hepatic fibrosis.[9]

All data obtained were entered in the logbook as well as in the computer daily. Data analysis was performed using IBM SPSS Statistics for Windows, Version 20.0. Normality of the continuous variables was assessed using the Shapiro-Wilk test, which showed significant non-normal distribution for all parameters. Spearman correlation analysis was performed to evaluate the association of TG/HDL-C ratio with age and BMI. TG/HDL-C ratio was compared across the BMI categories using the Kruskal-Wallis H test. Odds ratios (OR) with 95% confidence intervals (CI) were calculated using cross-tabulation with normal-weight individuals as the reference category to quantify the odds of having elevated TG/HDL-C in other BMI groups. Finally, a multiple linear regression was done with TG/HDL-C ratio as the dependent variable and BMI, age, sex, and LDL levels as predictors. Model assumptions were assessed prior to analysis. The significance level used was $p < 0.05$.

RESULTS

A total of 400 participants were taken for the study. Among them, 236 (59%) were female, and 164 (41%) were male. The study population had a median age of 53 (IQR: 44.25 - 62) years and a median BMI of 30.04 (IQR: 26.06 – 32.84) kg/m^2. The median TG/HDL-C ratio was 3.91. The distribution of age, BMI, and laboratory parameters is shown in table 1.

Table 1. Descriptive statistics of age, BMI and laboratory parameters

Variable (n=400)	Mean ± SD	Median (IQR)
Age (years)	54.07 ±10.51	53.00 (44.25 – 62.00)
Triglyceride (mg/dl)	172.21 ± 88.72	146.00 (117.00 – 200.00)
HDL-C (mg/dl)	38.17 ± 8.95	37.00 (32.25 – 44.00)
LDL-C(mg/dL)	90.50 ± 33.27	90.00 (70.00 – 108.00)
TG/HDL-C Ratio	4.82 ± 2.85	3.91 (3.12 – 5.26)
BMI (kg/m^2)	29.36 ± 4.91	30.04 (26.06 – 32.84)

TG/HDL-C ratio showed a weak positive correlation with BMI ($\rho = 0.302$, $p < 0.001$) and a very weak negative correlation with age ($\rho = – 0.142$, $p = 0.004$) as shown in table 2.

Table 2. Correlation of TG/HDL-C ratio with BMI and age

TG: HDL/C	BMI	Age
Correlation coefficient	0.302	- 0.142
P value	< 0.001	0.004

P-values from Spearman correlation analysis. A p of < 0.05 was considered statistically signifi-cant.

The TG/HDL-C ratio differed significantly across BMI categories, with the highest median values observed in obese participants (Table 3). Pairwise comparisons showed significantly higher TG/HDL-C ratios in obese individuals compared with normal-weight, overweight, and underweight groups (Table 3).

Table 3. Association of TG: HDL-C with BMI categories

BMI Category	Overall comparison	
	TG/HDL-C ratio Median (IQR)	p-value
Underweight (n=11)	3.68 (2.27–3.68)	< 0.001
Normal weight (n=66)	3.23 (2.47–3.80)	
Overweight (n=123)	3.13 (2.49–4.14)	
Obese (n=200)	4.50 (3.83–7.81)	

P- value for overall comparison from the Kruskal-Wallis H test.
Pairwise comparisons for obese group showed:
*Significantly different from the Underweight group (p = 0.006)
#Significantly different from the Normal weight group (p < 0.001)
@Significantly different from the Overweight group (p < 0.001)
Pairwise comparison Note: Pairwise comparisons among Underweight, Normal weight, and Overweight groups were not statistically signifi-cant (p > 0.05)

Table 4 shows that the prevalence of elevated TG/HDL-C ratio increased progressively across BMI categories, from 63.6% in underweight individuals to 91.5% among obese participants. However, a substantial proportion of normal-weight individuals (75.8%) also demonstrated an elevated TG/HDL-C ratio, indicating metabolic risk even in non-obese participants. The association between BMI category and elevated TG/HDL-C ratio was statistically significant (χ^2 = 18.34, p < 0.001). The odds for elevated TG: HDL-C were significant in obese patients compared to the normal individuals with an OR of 3.44 (95% CI: 1.63 -7.30).

Table 4. Elevated TG/HDL-C across BMI categories with odds ratios (reference = normal weight)

BMI Category	Elevated TG/HDL-C n (%)	Normal TG/HDL-C n (%)	OR (95% CI)	Pairwise p-value
Underweight (n=11)	7 (63.6)	4(36.4)	0.56(0.14 - 2.16)	0.400
Normal (n=66)	50(75.8)	16(24.2)	Reference	Refer-ence
Overweight (n=123)	96(78.0)	27(22.0)	1.14(0.56 -2.30)	0.720
Obese (n=200)	183(91.5)	17(8.5)	3.44(1.63 -7.30)	< 0.001
Total (n=400)	336(84.0)	64(16.0)	-	-

Note: Overall chi-square value (χ^2) for association of BMI category with elevated TG/HDL-C = 18.34, p < 0.001.

Higher BMI was independently associated with increased TG/HDL-C ratio (Table 5). In a multiple linear regression model adjusted for age, sex, and LDL cholesterol, each 5 kg/m^2 increase in BMI corresponded to a 0.705-unit rise in TG/HDL-C ratio (95% CI: 0.415–0.995, p < 0.001).

Table 5. Multiple linear regression of TG/HDL-C ratio with BMI and adjusted covariates

	Beta Co-efficient (adjusted)	Unad-justed P-value	Ad-justed P-value	95% CI for Beta (Adjusted)	
				Lower	Upper
BMI (5kg/m^2)	0.705	< 0.001	< 0.001	0.415	0.995
Age	- 0.017	0.039	0.229	-0.043	0.015
Sex (male)	1.020	0.026	0.001	0.441	1.600
LDL	0.006	0.062	0.145	-0.002	0.014

Unadjusted p-value from simple linear regression. Adjusted P-value from multiple linear regression. P < 0.05 considered statistically significant.

DISCUSSIONS

This study evaluated the association between TG/HDL-C ratio and BMI among Nepalese adults with NAFLD. The findings demonstrate that TG/HDL-C ratio is significantly associated with BMI and varies across BMI categories, supporting the role of adiposity in lipid dysregulation.

Previous studies have consistently shown that TG/HDL-C ratio is a reliable surrogate marker of insulin resistance and metabolic syndrome.[5,15,16] Furthermore, several studies have reported that TG/HDL-C ratio is significantly associated with NAFLD and metabolic abnormalities across diverse populations.[6,7,10-13] These findings support its utility as a simple indicator of underlying metabolic dysfunction.

A key observation in this study is the high prevalence of elevated TG/HDL-C ratio among normal-weight individuals, indicating metabolic risk despite normal BMI.[17] This supports the concept of the metabolically obese normal-weight (MONW) phenotype, widely described in South Asian populations.[4] Individuals with this phenotype often exhibit increased visceral adiposity and insulin resistance despite normal body weight, predisposing them to NAFLD.

Consistent with this, previous studies have demonstrated that TG/HDL-C ratio remains significantly associated with NAFLD even in non-obese individuals and serves as an independent predictor after adjusting for BMI and metabolic factors.[10-12,18] Additionally, large population-based studies have shown that NAFLD prevalence increases progressively across TG/HDL-C ratio quartiles, highlighting its role as an early metabolic marker.[13]

The present study also demonstrated that BMI independently predicts TG/HDL-C ratio, indicating that increasing adiposity contributes to worsening lipid imbalance. This is consistent with earlier studies showing

that TG/HDL-C ratio increases with BMI and correlates with insulin resistance and metabolic abnormalities.[5,6,15] However, the moderate strength of association suggests that factors beyond BMI, such as visceral adiposity and insulin resistance, also contribute significantly.

Although NAFLD prevalence generally increases with age, age was not independently associated with TG/HDL-C ratio after adjustment in the present study. This suggests that TG/HDL-C may primarily reflect metabolic dysfunction and insulin resistance rather than aging itself. Similar findings from previous studies have reported TG/HDL-C as an independent marker of NAFLD and insulin resistance across different age groups, including pediatric and non-obese populations.[10,12] Notably, the global burden of NAFLD among young adults is rising, underscoring the importance of early metabolic risk identification.[19]

From a clinical perspective, TG/HDL-C ratio has important implications as a practical screening tool. Studies have shown that it is associated not only with NAFLD but also with cardiovascular risk and liver disease severity.[20-22] Its simplicity and availability from routine lipid profiles make it especially valuable in resource-limited settings such as Nepal.

This study included a relatively large sample size (n = 400) and specifically evaluated both obese and non-obese individuals with NAFLD, allowing assessment of metabolic risk across BMI categories. In addition, rigorous statistical analyses, including correlation, non-parametric comparisons, and multivariable regression, were performed to evaluate the independent association between TG/HDL-C ratio and BMI.

However, the cross-sectional design limits the ability to establish causal relationships between TG/HDL-C ratio and NAFLD. The direct measures of insulin resistance such as HOMA-IR were not assessed. Along with this, hepatic steatosis was diagnosed using ultrasonography rather than more sensitive imaging modalities such as MRI or liver biopsy. Additionally, data on waist circumference or waist-to-hip ratio were not available, which may have provided better assessment of central obesity.

Future prospective studies are needed to determine TG/HDL-C ratio thresholds specific to the Nepalese population and to evaluate its predictive value for disease progression, metabolic complications, and cardiovascular outcomes in NAFLD. Incorporating additional markers of insulin resistance and measures of central obesity may further improve risk stratification.

CONCLUSION

In conclusion, the present study demonstrates a significant association between TG/HDL-C ratio and BMI among Nepalese adults with NAFLD. Elevated TG/HDL-C ratio was highly prevalent across BMI categories and was particularly pronounced among obese individuals. Importantly, a substantial proportion of normal-weight individuals also exhibited elevated TG/HDL-C ratios, suggesting the presence of underlying metabolic risk even in the absence of overt obesity. These findings highlight the potential utility of TG/HDL-C ratio as a simple and cost-effective marker for identifying metabolic dysfunction in patients with NAFLD.

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