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Clinical Association Between Triglyceride-to-HDL Cholesterol Ratio and C-Reactive Protein in Adults with Central Obesity

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Background: Central obesity contributes to insulin resistance, atherogenic dyslipidemia, and chronic low-grade inflammation, increasing cardiometabolic risk. The triglyceride-to-HDL cholesterol (TG/HDL-C) ratio is a simple marker of dyslipidemia and insulin resistance, while C-reactive protein (CRP) reflects systemic inflammation. However, evidence linking these indicators remains inconsistent, especially in Asian populations. This study aims to assess the association between TG/HDL-C ratio and CRP levels in adults with central obesity.

Materials and methods: A cross-sectional study was conducted among 80 adults with central obesity at Diponegoro National Hospital, Indonesia. Fasting lipid profiles, including triglycerides and HDL cholesterol, and CRP were measured using standard biochemical methods. The TG/HDL-C ratio was calculated, and its association with CRP levels was analyzed using the chi-square test (p<0.05).

Results: Among the participants, 57.5% had an elevated TG/HDL-C ratio (≥2.5), whereas elevated CRP levels (≥3 mg/L) were observed in only 6.25% of subjects. Statistical analysis showed no significant association between TG/HDL-C ratio and CRP levels (p=0.90).

Conclusion: Most adults with central obesity had an elevated TG/HDL-C ratio, while elevated CRP levels were relatively uncommon. However, no significant association was found between TG/HDL-C ratio and CRP. These findings suggest that dyslipidemia and systemic inflammation may occur at different stages of metabolic dysfunction. Further longitudinal studies with larger sample sizes are needed to clarify this relationship.

Keywords: *central obesity, TG/HDL-C ratio, c-reactive protein, inflammation*

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Introduction

Central obesity represents a major global health concern and is a key component of metabolic syndrome, a cluster of metabolic abnormalities that increase the risk of cardiovascular disease and type 2 diabetes.[1–3] It is characterized by excessive visceral fat accumulation around the abdomen, commonly assessed using waist circumference. Central obesity contributes to insulin resistance, dyslipidemia, and hypertension, thereby promoting adverse cardiometabolic outcomes.[4–7] Mechanistically, visceral adipose tissue releases increased amounts of free fatty acids, adipokines, and pro-inflammatory cytokines, which disrupt lipid and glucose metabolism, leading to elevated triglyceride levels and reduced HDL cholesterol.[8–14] These metabolic disturbances substantially increase cardiometabolic risk and highlight the importance of early identification and prevention strategies targeting visceral adiposity.[15–20]

In this context, the triglyceride-to-HDL cholesterol ratio (TG/HDL-C) serves as a simple yet reliable indicator of atherogenic dyslipidemia and insulin resistance, both hallmark features of metabolic syndrome.[21,22] This ratio, which increases with the number of metabolic syndrome components, correlates strongly with small, dense LDL particles and elevated cardiometabolic risk.[23,24] With cutoff values around 3.0 mg/dL or higher, TG/HDL-C is recognized as a practical, cost-effective biomarker for early detection and risk stratification, guiding preventive and therapeutic interventions in metabolic and cardiovascular disorders.[25–27]

Parallel to lipid abnormalities, excess visceral adiposity triggers chronic low-grade inflammation through the release of pro-inflammatory cytokines such as TNF-α and IL-6. Adipokines such as adiponectin also play key roles in obesity-related inflammation and metabolic dysregulation.[28] This "meta-inflammation" elevates biomarkers like hsCRP and NLR, linking visceral fat accumulation directly to systemic inflammation, dyslipidemia, and increased cardiovascular risk.[29–32] Chronic low-grade inflammation marked by elevated hs-CRP and other cytokines has been associated with cardiovascular risk and structural vascular changes, as demonstrated in patients with chronic inflammatory conditions.[33] Among these inflammatory markers, C-reactive protein (CRP), produced by the liver during inflammation, is a key biomarker of cardiometabolic risk. High-sensitivity CRP (hsCRP) detects low-grade inflammation from visceral fat and is strongly associated with metabolic syndrome, insulin resistance, and cardiovascular disease. Elevated CRP reflects endothelial dysfunction and atherogenesis, making it a valuable tool for early detection and risk stratification of cardiometabolic disorders.[34–37] Elevated CRP reflects endothelial dysfunction and atherogenesis, making it a valuable tool for early detection and risk stratification of cardiometabolic disorders.

Emerging evidence suggests that elevated TG/HDL-C ratios are positively associated with higher CRP levels, linking dyslipidemia with systemic inflammation. Both serve as complementary markers of insulin resistance and cardiometabolic risk, reflecting the intertwined roles of lipid imbalance and inflammation in metabolic syndrome.[38-41] However, evidence on this link remains inconsistent, especially in Asian populations. Although visceral fat–induced low-grade inflammation is key to metabolic dysfunction, variability in markers like ANC and NLR persists due to genetic and methodological differences.[42–49] Furthermore, studies in centrally obese populations have reported associations between pro-inflammatory biomarkers such as hs-CRP and adipokine imbalance.[50]

Notably, few studies have examined the association between the triglyceride-to-HDL cholesterol ratio and C-reactive protein levels in individuals with central obesity, particularly among Asian populations, who often display distinct metabolic profiles.[51,52] Thus, a clear understanding of this relationship in adults with central obesity remains limited. Understanding this relationship may halp identify simple and accessible clinical markers for inflammation and metabolic risk in obesity management. Therefore, this study aims to assess the association between the triglyceride to HDL cholesterol rasio and C-reactive protein levels in adults with central obesity.

Materials and methods

Study Population

This study employed a cross-sectional design to explore the association between metabolic parameters and inflammation in adults with central obesity. The target population comprised adult patients with central obesity who attended the Outpatient Clinic at Diponegoro National Hospital and agreed to participate in the study. A total of 80 participants with central obesity were included in this study, consisting of 31 males (38.75%) and 49 females (61.25%). Central obesity was defined based on waist circumference according to Asian criteria: ≥90 cm for men and ≥80 cm for

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women. Participants were eligible if they were adults aged 18–60 years and had a normal body temperature at the time of examination. Subjects were excluded if they had liver disease, kidney disease, pregnancy, malignancy, or were taking medications that could affect metabolic parameters. Laboratory analyses for triglycerides, fasting blood glucose (FBG), and high-density lipoprotein (HDL) levels were conducted at the RSND Laboratory. Meanwhile, the measurement of adiponectin and insulin levels was carried out at the Iodine Deficiency Disorders (GAKI) Laboratory, Faculty of Medicine, Diponegoro University. A consecutive sampling technique was used to recruit all eligible central obesity patients who met the inclusion and exclusion criteria and agreed to participate. Based on statistical considerations and expected sensitivity, a minimum of 70 subjects was required. To account for potential dropouts, a total of 80 participants were included in this study.

Blood Sampling and Laboratory Measurements

Venous blood samples were collected after an overnight fast of 8–12 hours. Serum triglycerides and HDL cholesterol were measured using standard enzymatic colorimetric method with Indiko™ Clinical Chemistry Analyzer (Thermo Fisher Scientific, Finland). The reagents used included Triglycerides reagent (Cat. No. 981786, Thermo Fisher Scientific Oy Clinical Diagnostics, Vantaa, Finland) and HDL Cholesterol Plus reagent (Cat. No. 981823, Thermo Fisher Scientific Oy Clinical Diagnostics, Vantaa, Finland). High-sensitivity C-reactive protein (hsCRP) levels were measured using a fluorescence immunoassay (FIA) with the ichroma™ II immunology analyzer (Boditech Med Inc., Chuncheon, Republic of Korea) according to the manufacturer's instructions.

The TG/HDL-C ratio was calculated by dividing triglyceride levels by HDL cholesterol levels and categorized using a cut-off value of 2.5, which has been suggested as a threshold for cardiometabolic risk in Asian populations. Triglyceride levels were categorized as normal (<150 mg/dL) and elevated (≥150 mg/dL), while HDL cholesterol was classified as low (<40 mg/dL in men and <50 mg/dL in women). CRP levels were categorized as normal (<3 mg/L) and elevated (≥3 mg/L) according to established cardiovascular risk thresholds.

Statistical Analysis

Data analysis was performed using JASP version 0.19.3 (Intel). Descriptive statistics were used to summarize participant characteristics, including mean and standard deviation for continuous variables and frequency distribution for categorical variables. The Chi-square test was applied to examine the association between categorical variables. A p-value of less than 0.05 was considered statistically significant.

Results

Lipid Profile, TGL/HDL Ratio, and CRP Distribution in Participants with Central Obesity

Most participants had normal triglyceride levels (71.25%) and normal HDL-C levels (87.5%). However, the mean TG/HDL ratio was 4.05 ± 2.87, and more than half of the participants (57.5%) were categorized as "at risk" (TG/HDL ≥ 2.5), suggesting the presence of atherogenic dyslipidemia despite overall normal lipid averages. Only 6.25% of participants showed elevated CRP (≥3 mg/L), indicating that most subjects did not exhibit systemic inflammation based on CRP levels (Table 1).

Association Between Lipid Profile Indicators and CRP Levels

The chi-square test revealed no significant association between triglyceride levels and CRP (p = 0.65), HDL-C levels and CRP (p = 0.60), or TG/HDL ratio and CRP (p =

Table 1. Descriptive characteristics of triglyceride, HDL-C, TG/HDL ratio, and CRP among participants (n=80).

Variable	Mean ± SD	n (%)
Triglyceride/TG (mg/dL)	171.41± 87.50	
Normal		57 (71.25)
At risk		23 (28.75)
HDL-C (mg/dL)	48.77 ± 12.63	
Normal		70 (87.50)
At risk		10 (12.50)
TG/HDL ratio	4.05 ± 2.87	
Normal (<2.5)		34 (42.50)
At risk (≥2.5)		46 (57.50)
CRP (mg/L)		
Normal (<3 mg/L)		75 (93.75)
At risk (≥3 mg/L)		5 (6.25)

0.90). Although a higher proportion of participants in the "at risk" TG/HDL category had elevated CRP (60%) compared to those in the normal category (40%), the difference was not statistically significant. These results suggest that, within this sample, the TG/HDL ratio did not show a significant relationship with systemic inflammation as indicated by CRP levels (Table 2).

Table 1. Association between lipid profile indicators and CRP levels (Chi-square test).

Variable	CRP Category n (%)		P (X^2)
	Normal	At risk	
Triglyceride/TG			
Normal	53 (70.67)	4 (80)	0.65
At risk	22 (29.33)	1 (20)	
HDL-C			
Normal	66 (88)	4 (80)	0.60
At risk	9 (12)	1 (20)	
TG/HDL			
Normal	32 (42.67)	2 (40)	0.90
At risk	43 (57.33)	3 (60)	

Discussion

In this study, 80 patients with central obesity, we observed that while mean triglyceride (TG) levels (171.41 ± 87.50 mg/dL) and mean HDL-C levels (48.77 ± 12.63 mg/dL) fell within ranges seen in many obese populations (Table 1), a substantial portion of participants (57.50%) exhibited a TG/HDL ratio ≥2.5 (mean ratio 4.05 ± 2.87). Meanwhile, only 6.25% of subjects had elevated C-reactive protein (CRP) in the "at risk" category (≥3 mg/L) (Table 1). However, the bivariate chi-square analyses did not demonstrate a statistically significant association between TG/HDL category and elevated CRP (p = 0.90), nor between TG or HDL categories individually and CRP (p=0.65 and p=0.60, respectively) (Table 2).

The high prevalence of elevated TG/HDL-C ratio in our sample reflects a common pattern of atherogenic dyslipidemia in individuals with central obesity. Excess visceral adiposity promotes increased free fatty acid flux to the liver, which stimulates hepatic triglyceride synthesis and reduces HDL-C levels, resulting in an elevated TG/HDL-C ratio. Therefore, the TG/HDL ratio has been widely proposed as a simple surrogate marker for insulin resistance and cardiovascular risk.[53] Several studies have reported that individuals with central obesity tend to exhibit higher TG/HDL-C ratios compared with those without abdominal adiposity, reflecting underlying metabolic disturbances.[54] Previous studies have also shown that higher TG/HDL-C ratios are associated with increased hs-CRP concentrations in individuals at cardiometabolic risk.[55] In our study, however, the proportion of subjects with elevated CRP was relatively low, suggesting that overt systemic inflammation may not yet be prominent in many centrally obese individuals. Alternatively, the CRP cut-off used (≥3 mg/L) may primarily capture more advanced inflammatory states.[56]

Despite the expected relationship, our chi-square analyses revealed no statistically significant association between TG/HDL ratio and CRP levels. This finding may be explained by several factors. First, the low event rate for elevated CRP could have limited the statistical power to detect an association, as only 5 out of 80 participants (6.25%) were classified with elevated CRP levels, resulting in insufficient variability between groups. Second, the cross-sectional study design prevents determination of causality or temporal sequence between lipid dysregulation and systemic inflammation. It is possible that lipid abnormalities precede measurable increases in CRP or that other metabolic mediators, such as adipokines or cytokines, mediate this relationship. In addition, hsCRP levels can be influenced by various factors including age, sex, smoking status, physical activity, and the presence of acute or chronic inflammatory conditions, which may contribute to variability in CRP measurements.[57] Third, metabolic heterogenity among individuals with central obesity might contribute to the nonsignificant results. Not all centrally obese individuals display equal degrees of insulin resistance, dyslipidemia, or inflammation some may retain relatively preserved metabolic profiles despite increased visceral adiposity. Lastly, methodological considerations such as the cut-off thresholds may also influence the findings. The TG/HDL ratio cut-off of ≥2.5 and CRP threshold of ≥3 mg/L may not optimally identify early subclinical inflammation in this cohort. Some studies have suggested higher TG/HDL ratio

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thresholds (≥3.0 or ≥3.5) to better reflect metabolic risk in certain populations, while CRP cut-offs might require adjustment in younger or metabolically healthy obese populations.[58]

Numerous mechanistic pathways link dyslipidemia, insulin resistance, and inflammation. The elevated TG/HDL-C ratio reflects increased very-low-density lipoprotein (VLDL) production, decreased HDL-C-mediated reverse cholesterol transport, and lipid-rich remnant particles all of which can promote endothelial dysfunction and macrophage activation. Activated macrophages subsequently release pro-inflammatory cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), which stimulate hepatic CRP production.[59] In parallel, chronic inflammation can further impair HDL functionality and enhance the atherogenicity of TG-rich lipoproteins, creating a vicious cycle between dyslipidemia and inflammation.[60] Based on these mechanisms, a positive correlation between TG/HDL-C ratio and CRP levels would be expected. Indeed, several studies in other populations have demonstrated this association. For instance, previous study found that elevated TG/HDL-C ratio was significantly associated with higher hs-CRP concentrations, reflecting systemic low-grade inflammation.[61] However, their study included a larger population with broader metabolic characteristics, whereas our study specifically focused on individuals with central obesity and a relatively smaller sample size. These differences in population characteristics and sample size may partly explain the discrepancy between their findings and ours.

The absence of a significant association between TG/HDL ratio and CRP levels in our study contrasts with a number of prior studies reporting significant positive correlations between TG/HDL ratio and inflammatory markers. For example, previous study demonstrated that TG/HDL-C ratio was a superior predictor of metabolic syndrome compared to CRP/HDL-C ratio in a large NHANES cohort.[62] Similarly, observed a significant association between TG/HDL-C ratio and hs-CRP levels in individuals with impaired fasting glucose, suggesting that lipid imbalance and subclinical inflammation are intertwined in early metabolic dysregulation. Reported that components of metabolic syndrome, including TG/HDL ratio, were linked with low-grade inflammation in an Indonesian population. The discrepancy between our results and those of previous studies may be attributed to differences in population characteristics (such as ethnicity, dietary habits, and genetic background), variations in inflammation thresholds, or the more homogeneous nature of our sample, which consisted solely of centrally obese adults rather than a broader cardiometabolic risk group.

Despite the lack of statistical association in our sample, the high prevalence of elevated TG/HDL ratio (57.5%) remains clinically relevant. A high TG/HDL ratio may still serve as an early warning signal of metabolic dysregulation even before detectable systemic inflammation by CRP. Clinicians should consider screening for dyslipidemia and perhaps monitor lipid ratios even in the absence of elevated CRP, as part of risk stratification in central obesity. Furthermore, interventions aimed at reducing TG/HDL ratio (via diet, physical activity, weight loss) may still be justified given the known links to insulin resistance and cardiovascular risk.

Key limitations of our study include the cross-sectional design (precluding causal inference), the relatively small sample size (n=80) and low number of elevated CRP cases, and the absence of adjustment for potentially confounding variables such as age, sex, BMI, diet and medication use. Moreover, the dichotomous categorization of TG/HDL ratio and CRP may have reduced analytic sensitivity. Future studies should incorporate larger cohorts, stratify by metabolic phenotype, include continuous variable analyses and examine longitudinal changes in lipid ratios and inflammation over time. Inclusion of additional inflammatory markers (e.g., IL-6, TNF-α, adiponectin) may enhance mechanistic insight.

Conclusion

This study found that although a high proportion of adults with central obesity exhibited an elevated TG/HDL-C ratio, only a small percentage had elevated CRP levels. No significant association was observed between TG/HDL-C ratio and CRP concentration. These findings suggest that atherogenic dyslipidemia may not directly correspond to systemic inflammation in centrally obese individuals. Further longitudinal studies are needed to clarify this relationship.

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Authors' Contributions

MH as the corresponding author, conceptualized and designed the study, performed data analysis, drafted the manuscript, and critically revised the manuscript. EK contributed to data analysis and interpretation of the results. VSC and BRH conducted data acquisition and collection. AKN was responsible for data processing and table design. All authors reviewed the manuscript critically for important intellectual content and approved the final version.

Ethical Statement

The Ethical clearance was obtained from the Health Research Ethics Committee of the Faculty of Medicine, Universitas Diponegoro (No.095/EC/KEPK/FK-UNDIP/IV/2025). All participants provided written informed consent prior to data collection.

Conflict of Interest

The authors declare no conflicts of interest.

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