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The Relationship between Triglyceride/High-Density Lipoprotein Cholesterol Ratio and Coronary Slow-Flow Phenomenon

Ahmed F. Tamara [a], Hisham K. Rasheed [b], Mohamed F. Abd Ulqader [c], Reham H. Amin [d], Ahmed Y. Nammour [b], Ahmed I. Elbarbary [c]

[a] Cardiovascular Medicine Department, Faculty of Medicine Ain Shams University, Egypt.

[b] Cardiovascular Medicine Department, Faculty of Medicine Benha University, Egypt.

[c] Cardiovascular Medicine Department, Faculty of Medicine Misr University for Science and Technology (MUST), Egypt.

[d] Pediatric Department, Faculty of Medicine Benha University, Egypt.

Corresponding to:
Dr. Mohamed F. Abd Ulqader. Cardiovascular Medicine Department, Faculty of Medicine Misr University for Science and Technology (MUST), Egypt.

Email:
fmohamedfarouk86@gmail.com

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Abstract:

Background: Coronary Slow-Flow Phenomenon (CSFP) poses a diagnostic challenge in the context of coronary artery disease (CAD), and the search for reliable biomarkers continues. The Triglyceride/High-density Lipoprotein Cholesterol (TG/HDL-C) ratio has emerged as a potential candidate, given its established role in atherogenesis. This study aimed to investigate the relationship between the TG/HDL-C ratio and CSFP, assessing its potential as a diagnostic marker. **Methods:** This prospective interventional study included 100 patients who underwent elective coronary angiography for suspected CAD. The patients were divided into two groups; 50 patients has CSFP and 50 controls. On admission to the ER, all patients were subjected to physical examination including; vital data and local examination, and 12 lead ECG, laboratory investigations including; cardiac enzyme, lipid profile random blood sugar, complete blood picture and kidney function tests. **Results:** The study revealed a significantly higher TG/HDL-C ratio in the CSFP group compared to controls ($p < 0.001$). ROC analysis indicated the TG/HDL-C ratio as a moderately accurate diagnostic tool for CSFP (AUC = 0.822). Significant correlations were observed between the TG/HDL-C ratio and key parameters, including non-HDL-C, triglycerides, ECG features (P max, PWd, and QTcd), while negatively correlating with HDL-C, WBC, and QTc. Logistic regression affirmed the TG/HDL-C ratio as the sole parameter associated with CSFP risk. **Conclusion:** Among patients with suspected CAD, the TG/HDL-C ratio strongly was associated with CSFP and serves as a noteworthy independent predictor of CSFP. The TG/HDL-C ratio demonstrated moderate accuracy as a diagnostic tool for CSFP, with an optimal cutoff value exceeding 2.75. Thus, it holds the potential to be a valuable biomarker in predicting the occurrence and severity of CSFP.

Keywords: Coronary Slow-Flow Phenomenon; Triglyceride/High-Density Lipoprotein Cholesterol Ratio; Coronary Angiography; Diagnostic Biomarker.

Introduction

The coronary slow-flow phenomenon (CSFP) is a coronary artery disease, characterized by delayed vessel opacification in the absence of obstructive coronary artery disease. It is defined angiographically, and its incidence varies from 1 to 7% among the patients undergoing coronary angiography for suspected coronary artery disease (CAD) [(1)].

Although the exact mechanisms of CSFP are still unclear, small vessel disease, endothelial dysfunction, diffuse atherosclerosis, microvascular vasomotor dysfunction, and Inflammations- are implicated in its pathophysiology [(2)]. In addition, CSFP may cause transient myocardial hypoperfusion in patients with both normal coronary arteries and angina, thereby, leading to increasing the risk of CAD and worsening of the prognosis [(3)].

There is no definitive treatment of CSFP and traditional anti-anginal drugs are often used in the treatment. A particular attention has been paid to control hypertension and dyslipidemia in CSFP cases [(4)].

In some studies, statins are used to regulate cholesterol levels and take vascular inflammation under control. In recent years, intimal thickening in the coronary arteries, diffuse calcification, lumen irregularity, and non-occlusive atheroma plaques have been identified in the majority of cases with CSFP. In the light of recent findings, CSFP should be evaluated as CAD, which was earlier considered a subgroup of cardiac syndrome X [(1)].

Several studies have suggested that established cardiovascular risk factors may play a role in the pathogenesis of microvascular angina in healthy individuals, result in coronary microvascular dysfunction [(4)].

Dyslipidemia is one of the risk factors of cardiovascular disease (CVD) [(5)]. It is defined as a disorder of lipoprotein metabolism, rather than elevations in total cholesterol (TC) or low-density lipoprotein cholesterol (LDL-C) alone. Triglyceride (TG), LDL-C, and high-density lipoprotein cholesterol (HDL-C)- are important components of the lipid fraction of the human body [(6)].

However, epidemiological researches have demonstrated that lipid-related ratios such as TC/HDL-C, TG/HDL-C, or LDL-C/HDL-C may be probably better predictors of CVD risk than any other single lipid marker [(7)]. Among these, the TG/HDL-C ratio was first proposed by Gaziano et al. as an atherogenic index. Several studies have shown that TG/HDL-C ratio is a strong predictor of CAD such as myocardial infarction (MI), LDL phenotype B, and atherogenic risk [(8)].

In addition, the TG/HDL-C ratio appears to be more valuable than any other single lipid marker, since it has an ability to reflect the complex interactions between the lipoprotein metabolisms and to better predict plasma atherogenicity [(8)]. Besides its simplicity and practicality, there is a growing number of evidences supporting the predictive value of TG/HDL-C ratio in cardiovascular events and may be of clinical relevance [(9)].

The aim of this study was to investigate the relationship between the TG/HDL-C ratio and CSFP, in patients undergoing elective coronary angiography for suspected CAD and to identify whether TG/HDL-C ratio was a feasible biomarker in distinguishing CSFP cases from healthy individuals.

Patients and methods

This was a prospective interventional study conducted on patients undergoing elective coronary angiography for suspected CAD, at Cardiology department in Ain Shams University Hospitals and Misr University for Science and Technology (MUST) University Hospital over a period of one year from April 2022 till fulfillment of the required sample number.

An informed written consent was obtained from the patients. Every patient received an explanation of the purpose of the study and had a secret code number. The study was done after being approved by the Research Ethics Committee, Faculty of Medicine, Benha University {M.S.24.5.2022}.

The study included 100 subjects, 50 subjects diagnosed as CSFP "coronary slow flow phenomenon" and 50 controls. Informed consent was taken from all patients for the study participation.

Inclusion criteria were both sexes, risk factors for CAD as HTN, Diabetes, smoking, family history of coronary artery disease and dyslipidemia, symptoms consistent with myocardial ischemia in the form of anginal pain and patients without hemodynamics or electrical instability.

Exclusion criteria were ventricular dysfunction (ejection fraction < 35%), those underwent surgery or mechanical revascularization for significant CAD, chronic renal or hepatic insufficiency, acute and chronic inflammatory diseases, or autoimmune disease, those receiving drugs affecting the lipid metabolism, hemodynamic impairment or electrical un-instability and pregnancy.

Methodology:

All patients were subjected to:

Complete history taking: Demographic data including name, age, sex, race. **Comorbidities**: smoking status, diabetes, hypertension, renal impairment. History of coronary artery disease and previous coronary intervention. Drug history included CCBs, Beta-blockers, ACE inhibitors, ARBX, anti-platelets, oral hypoglycemics and statins. **Physical examination:** including vital data and local examination. **Vital data** as body temperature, blood pressure, pulse (heart rate), and breathing rate (respiratory rate). **Laboratory investigations** included cardiac enzyme, lipid profile, random blood sugar, complete blood picture and kidney function tests (Urea, Creatinine and uric acid).

Biochemical analyses: Venous blood samples were drawn from each participant after an overnight (12-h) fasting. Hematological analyses were carried out using the BC-6800 Hematology Analyzer. All biochemical parameters were analyzed on the day of sample collection using a commercial autoanalyzer (c8000i; Abbott Diagnostics GmbH, Germany). The LDL-C was calculated using the Friedewald formula [(LDL-C = TC – HDL-C – (TG/5)], Non-HDL-C was calculated as: TC-HDL-C. TG/HDL-C ratio was calculated dividing absolute TG levels by absolute HDL-C levels in peripheral blood.

Coronary angiography was percutaneously performed using the Judkins technique (1). The coronary arteries were visualized at right and left oblique and caudal planes at 30 frames per second (fps). A contrast agent was injected manually during coronary angiography (6 to 10 mL of contrast agent at each position using right and left, and cranial and caudal angulations).

Angiographic images were stored in 34 runs on a CD in accordance with the Digital Imaging and Communications in Medicine (DICOM) standards and the flow velocity was measured. All angiographic measurements were carried out by two cardiologists who were blind to the clinical status of the patients.

The coronary slow-flow phenomenon (CSFP) is a coronary artery disease, characterized by delayed vessel opacification in the absence of obstructive coronary artery disease [(1)].

Statistical analysis

The collected data was processed using IBM SPSS (SPSS Inc., Chicago, Illinois, USA) Statistics for Windows (Version 25.0), with normality checked using the Shapiro-Wilk test. Descriptive statistics, including mean and standard deviation for numerical data and frequency/percentage for non-numerical data- were employed. Analytical statistics involved the Student's T Test for comparing means, Mann-

Whitney Test (U test) for non-parametric variables, and Chi-Square test to explore relationships between qualitative variables. Correlation analysis assessed the strength of association between quantitative variables. The Receiver Operating Characteristic (ROC) Curve determined the optimal cutoff point for diagnostic measures, with an area under the curve (AUC) indicating accuracy levels. Logistic regression analyses were performed to predict risk factors when the dependent variable is categorical, with odds ratios (OR) and 95% confidence intervals (CI) used to measure associations. Significance was set at $p < 0.05$ with a 95% confidence interval.

Results

Demographic data, cardiovascular risk factors and Medication were illustrated in **Table (1)**.

The TG/HDL-C ratio showed several significant correlations with various parameters:

- Positive correlations:
  - Non-HDL-C ($p = 0.042$)
  - Triglycerides ($p < 0.001$)
  - P max ($p < 0.001$)
  - PWd ($p < 0.001$)
  - QTc max ($p < 0.001$)
  - QTcd ($p = 0.018$)
- Negative correlations:
  - HDL-C ($p < 0.001$)
  - WBC ($p < 0.001$)
  - QTc min ($p < 0.001$)

Table 1: Demographic data, cardiovascular risk factors and Medication in the studied groups

		CSFP group N=50	Control group N=50	Test	p
Age, years	M±SD	55.31 ± 2.73	55.76 ± 2.7	t=0.821	0.413
Gender, n (%)	Male	30(60%)	29(58%)	X2=0.041	0.839
	Female	20(40%)	21(42%)		
Comorbidities, n (%)					
Smoking		13(26%)	10(20%)	0.508	0.476
Hypertension		24(48%)	23(46%)	0.040	0.841
Diabetes mellitus		13(26%)	11(22%)	0.219	0.64
Medication, n (%)					
Calcium channel blockers		10(20%)	6(12%)	1.190	0.275
Beta-blocker		11(22%)	16(32%)	1.268	0.260
ACEI/ARB		9(18%)	16(32%)	2.613	0.106
Antiplatelet		11(22%)	18(36%)	2.38	0.123
Oral hypoglycemic medications		9(18%)	9(18%)	0.000	1.000
Statin		7(14%)	10(20%)	0.638	0.424

t= T student test, X^2= Chi-Square. CSFP: Coronary Slow Flow Phenomenon. ACEI/ARB: Angiotensin-Converting Enzyme Inhibitors/Angiotensin II Receptor Blockers

No significant correlations were observed between the TG/HDL-C ratio and the other studied parameters, as detailed in **Table (2).**

Logistic regression analysis was conducted for prediction of CSFP using Fasting glucose, ALT, AST, Uric acid, Non-HDL-C, Triglyceride, TG/HDL-C ratio, P max, PWd, QTc min and QTcd. All parameters associated with risk factors for CSFP in uni variate analysis. TG/HDL-C ratio was the only parameters associated with the risk of CSFP in uni- and multivariate analyses, **Table (3).**

Table 2: Correlation analysis between TG/HDL-C ratio and other studied parameters among CSFP subjects

	rs	p
Age	-0.129	0.200
Gender	0.102	0.314
Smoking	0.107	0.291
Hypertension	0.184	0.066
Diabetes mellitus	0.213	0.124
Fasting glucose (mg/dL)	0.163	0.105
Urea (mg/dL)	0.060	0.554
Creatinine (mg/dL)	0.629	0.352
ALT (IU/L)	0.013	0.899
AST (IU/L)	0.104	0.303
Uric acid (mg/dL)	0.177	0.078
TC (mg/dL)	-0.005	0.961
LDL-C (mg/dL)	0.183	0.069
HDL-C (mg/dL)	-.661**	**<0.001***
Non-HDL-C	.204*	**0.042**
Triglyceride (mg/dL)	.731**	**<0.001***
WBC (103/mm3)	-0.059	0.558
Hemoglobin (mg/dL)	0.122	0.226
Platelet (103/mm3)	0.105	0.300
P max msec	.337**	**<0.001***
P min msec	-0.068	0.503
PWd msec	.386**	**<0.001***
QTc max msec	.298**	**<0.001***
QTc min msec	-.336**	**<0.001***
QTcd msec	.236*	**0.018**

rs: Spearman correlation coefficient, *: Significant ≤0.05
TG/HDL-C: Triglyceride to High-Density Lipoprotein Cholesterol ratio. CSFP: Coronary Slow Flow Phenomenon ALT: Alanine Aminotransferase .AST: Aspartate Aminotransferase ,TC: Total Cholesterol.LDL-C: Low-Density Lipoprotein Cholesterol. HDL-C: High-Density Lipoprotein Cholesterol. WBC: White Blood Cells .P max: Maximum P-wave duration. P min: Minimum P-wave duration. PWd: P-wave dispersion .QTc max: Corrected QT maximum interval. QTc min: Corrected QT minimum interval. QTcd: Corrected QT dispersion

Table 3: Logistic regression analysis for prediction of CSFP among studied subjects.

	Univariable			Multivariable		
	P	**OR**	**95% C.I**	**p**	**OR**	**95% C.I**
Fasting glucose (mg/dL)	<0.001*	1.034	1.015-1.054	0.125	1.041	0.989-1.095
ALT (IU/L)	0.007	1.085	1.023-1.152	0.068	1.162	0.989-1.364
AST (IU/L)	0.172	1.035	0.985-1.087			
Uric acid (mg/dL)	0.007	1.605	1.139-2.261	0.883	0.922	0.313-2.713
Non-HDL-C	0.003	1.019	1.006-1.031	0.556	1.009	0.979-1.04
Triglyceride (mg/dL)	<0.001*	1.079	1.044-1.115	0.064	1.092	0.995-1.199
TG/HDL-C ratio	<0.001*	8.111	3.272-20.105	0.033*	0.800	0.035-18.289
P max msec	<0.001*	1.056	1.027-1.086	0.238	1.041	0.974-1.112
PWd msec	<0.001*	1.101	1.055-1.149	0.019	1.133	1.021-1.257
QTc min msec	0.013	0.981	0.966-0.996	0.642	0.992	0.957-1.028
QTcd msec	<0.001*	1.031	1.014-1.049	0.051	1.053	1-1.109

OR, odds ratio; CI, confidence interval, *: Significant ≤0.05..CI: Confidence Interval
ALT: Alanine Aminotransferase. AST: Aspartate Aminotransferase. Non-HDL-C: Non-High-Density Lipoprotein Cholesterol. TG/HDL-C ratio: Triglyceride to High-Density Lipoprotein Cholesterol ratio. P max msec: Maximum P-wave duration in milliseconds. PWd msec: P-wave dispersion in milliseconds. QTc min msec: Minimum corrected QT interval in milliseconds. QTcd msec: QT interval dispersion in milliseconds

According to laboratory test, the results showed statistically significant differences between the CSFP and control groups for fasting glucose, ALT, AST, uric acid, non-HDL-C, and triglycerides, with p-values less than 0.05. There were no statistically significant differences for urea, creatinine, TC, LDL-C, HDL-C, WBC, hemoglobin, or platelet count, **Figure (1).**

The mean value of the TG/HDL-C ratio in the CSFP group was 3.33 with a standard deviation of 0.92, while in the control group the mean value was 2.39 with a standard deviation of 0.47. The statistical test comparing the two groups resulted in a p-value of less than 0.001, which is highly significant, **Figure (2).**

Receiver operating characteristic (ROC) curve of TG/HDL-C ratio was conducted for discrimination between CSFP subjects and controls. TG/HDL-C ratio showed moderate accuracy AUC (=0.822) as a diagnostic ability for CSFP with best cut off value >2.75, **Figure (3).**

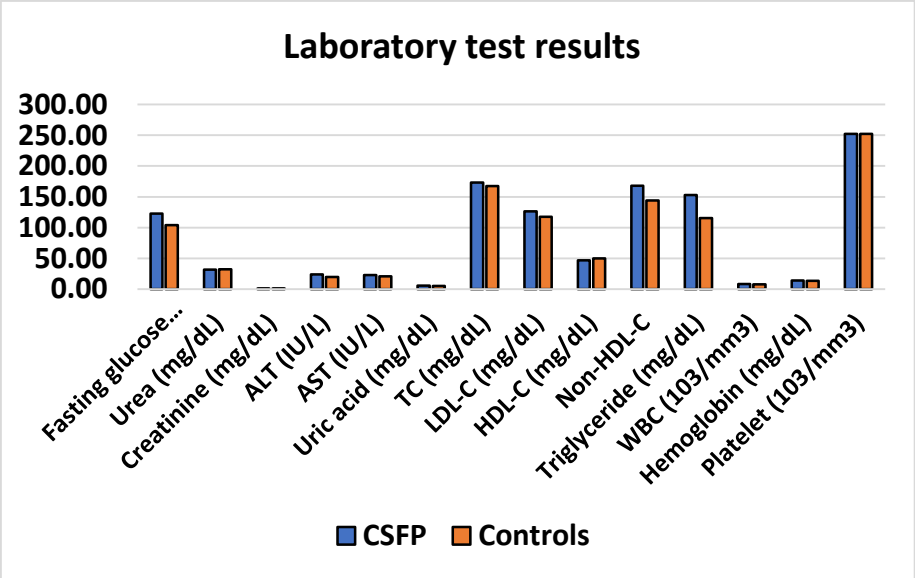

Figure 1: Mean laboratory test results in the studied groups

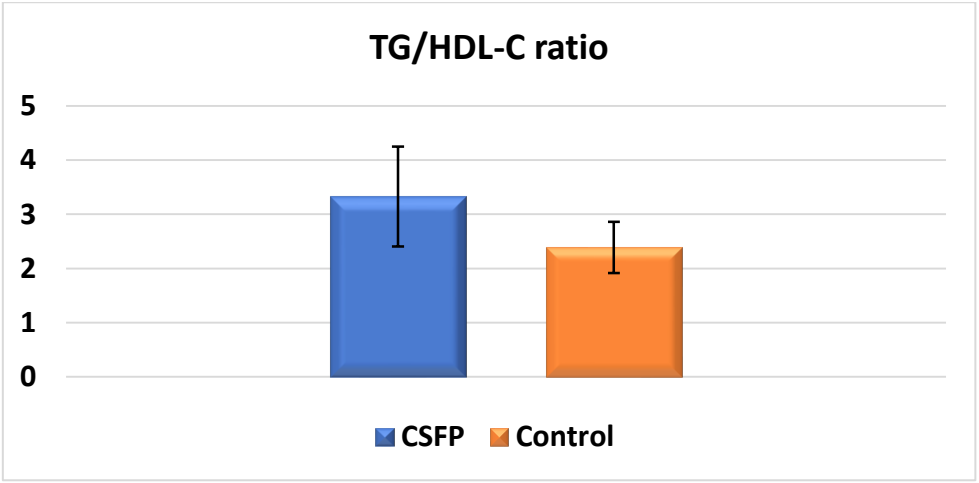

Figure 2: Mean TG/HDL-C ratio in the studied groups

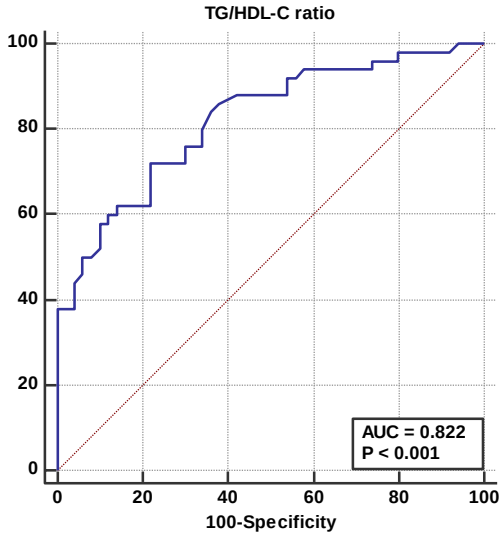

Figure 3: ROC curve analysis of TG/HDL-C ratio for discrimination between CSFP and healthy subjects

Discussion

In our study, according to laboratory test, the results showed statistically significant differences between the CSFP and control groups for fasting glucose, ALT, AST, uric acid, non-HDL-C, and triglycerides, with p-values less than 0.05. There were no statistically significant differences for urea, creatinine, TC, LDL-C, HDL-C, WBC, hemoglobin, or platelet count.

In line with our study, a study documented that ALT, Uric acid were significant differences between the CSFP and control groups. There were no statistically significant differences for TC, LDL-C, HDL-C, WBC, or platelet count [(10)].

In addition, a study showed there was no significant difference between the CSF group and the control group in white blood cell count, red blood cell count, average red blood cell volume, hemoglobin concentration, platelet count, urea nitrogen, fasting blood glucose, creatinine, total triglycerides, and high-density lipoprotein [(11)].

In disagreement with our study, a study included 124 patients. The eligible patients based on the inclusion and exclusion criteria were divided into two groups: the study group, including the patients with CSFP, and the control group, including the patients with normal epicardial coronary arteries (NECA). They noted that Absolute Neutrophil Count (ANC), Absolute Lymphocyte Count (ALC), Hct, Plt, MPV, RDW, Cr, TG, total cholesterol, and LDL were significantly higher in the study group than in the control group [(12)].

In the current study, the mean value of the TG/HDL-C ratio in the CSFP group was 3.33 with a standard deviation of 0.92, while in the control group the mean value was 2.39 with a standard deviation of 0.47. The statistical test comparing the two groups resulted in a p-value of less than 0.001, which is highly significant.

In agreement with our study, a study found that CSFP patients had higher TG/HDL-C ratio ($p < 0.001$) than control group [(13)].

Also, a study enrolled 93 stable patients randomly who had undergone coronary angiography and had near-normal coronary arteries and coronary slow flow. Their study observed that TG/HDL-c ratio was significantly higher in CSF group ($p=0.007$) [(14)].

According to the current study, receiver operating characteristic (ROC) curve of TG/HDL-C ratio was conducted for discrimination between CSFP subjects and controls. TG/HDL-C ratio showed

moderate accuracy AUC (=0.822) as a diagnostic ability for CSFP with best cut off value >2.75.

The ROC curve was used to evaluate the ability of TG, HDL-C, and TG/HDL-C ratio to discriminate CSFP and healthy individuals (Fig. 4). The ROC curve showed an area under the curve (AUC) value of 0.72 (%95 CI 0.65–0.81; $p < 0.001$). The sensitivity and specificity of TG/HDL-C ratio for predicting CSFP were 72% and 71%, respectively [(13)].

Values of the area under the curve (AUC) were greater for the TG/HDL-C ratio (AUC=0.932) and TC/HDL-C ratio (AUC=0.923) than for individual lipid profiles including TG (AUC=0.909) and HOMA-IR. The optimal cutoff values of TG/HLD-c ratio and TC/HDL-c ratio for validating individuals with MetS were 3.3 and 3.8 in both boys and girls [(15)].

In our study, TG/HDL-C ratio showed a significant positive correlation with non-HDL-C, triglycerides, P max, PWd and QTcd. TG/HDL-C ratio showed a significant negative correlation with HDL-C, WBC and QT c, while no significant correlation with other studied parameters.

In the current study, Logistic regression analysis was conducted for prediction of CSFP using Fasting glucose, ALT, AST, Uric acid, Non-HDL-C, Triglyceride, TG/HDL-C ratio, P max, PWd, QTc min and QTcd. All parameters associated with risk factors for CSFP in uni variate analysis. TG/HDL-C ratio was the only parameters associated with the risk of CSFP in uni- and multivariate analyses.

To determine the possible confounding factors for CSFP, logistic regression analysis was used. It was selected variables with P < .2 in the univariate analysis for the multivariable analysis, and those with multicollinearity were not included in this regression model. The monocyte count (OR: 14.151; 95% CI: 1.663–120.394, P = .02), hemoglobin (OR:1.025, 95% CI: 1.004–1.045, P = .02), creatinine (OR: 1.026, 95% CI: 1.007–1.045, P = .01) and globulin (OR: 1.125, 95% CI: 1.041–1.215, P = .01) levels were all independent predictors of CSFP. The red blood cell count, mean cell volume, platelet count, platelet-large cell ratio, total protein, direct bilirubin, and indirect bilirubin levels were not included in the multivariate analysis because of multicollinearity [(10)]

Conclusion

Among patients with suspected CAD, the TG/HDL-C ratio strongly was associated with CSFP and serves as a noteworthy independent predictor of CSFP. The TG/HDL-C ratio demonstrated moderate accuracy as a diagnostic tool for CSFP, with an optimal cutoff value exceeding 2.75. Thus, it holds the potential to be a valuable biomarker in predicting the occurrence and severity of CSFP.

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