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ORIGINAL RESEARCH

Association Between the Aggregate Index of Systemic Inflammation and Slow Coronary Flow Phenomenon in Patients with Ischemia and No Obstructive Coronary Arteries

Hong-Yang Wang[1], Jiang Guo[2], Min-Yan Hong[3], Jia-Jia Li[4], Xue-Qiang Jin[3]

[1]Department of Cardiovascular Medicine, The First People's Hospital of Linhai, Taizhou, Zhejiang, 317000, People's Republic of China; [2]Department of Cardiovascular Medicine, Longquan People's Hospital Affiliated to Lishui University, LiShui, Zhejiang, 323700, People's Republic of China; [3]Department of General Surgery, The First People's Hospital of Linhai, Taizhou, Zhejiang, 317000, People's Republic of China; [4]Department of Emergency Medicine, The First People's Hospital of Linhai, Taizhou, Zhejiang, 317000, People's Republic of China

Correspondence: Xue-Qiang Jin, Email lhjinxueqiang@163.com

Background: Inflammation has been proposed as a potential pathogenic mechanism underlying the slow coronary flow phenomenon (SCFP). The aggregate index of systemic inflammation (AISI), a novel biomarker for evaluating inflammation, has been linked to various cardiovascular diseases. However, the relationship between AISI and the occurrence of SCFP in patients with ischemia and non - obstructive coronary arteries (INOCA) remains unclear.

Methods: In this study, 1328 consecutive patients with INOCA were recruited. Among them, 90 patients had SCFP (SCFP group). A total of 180 age - and sex - matched individuals with INOCA and normal blood flow were selected as controls at a ratio of 1:2. Clinical manifestations, laboratory parameters, and angiographic features were recorded to identify potential predictors of SCFP in INOCA patients.

Results: Compared with the control group, patients in the SCFP group had a higher prevalence of current smoking, as well as elevated white blood cell (WBC), neutrophil, monocyte, and platelet counts, and a higher AISI. The AISI value increased with the number of vessels affected by SCFP. Multivariate logistic regression analysis demonstrated that the WBC count and AISI were independent predictors of SCFP in INOCA patients. Additionally, when the AISI was > 264.1, the sensitivity and specificity were 64.4% and 64.4% respectively, and the area under the receiver operating characteristic curve (AUC) was 0.657 (95% CI: 0.590–0.723, $P < 0.001$). The AISI had a more favorable predictive value for the presence of SCFP than WBC, neutrophils, lymphocytes, monocytes, and platelets alone ($P < 0.05$).

Conclusion: Higher AISI scores are associated with an increased risk of SCFP in INOCA patients. As an easily - obtained biomarker for assessing the degree of inflammation, the AISI can serve as a promising tool for risk stratification and appropriate management in INOCA patients.

Keywords: aggregate index of systemic inflammation, slow coronary flow phenomenon, ischemia and no obstructive coronary arteries, predictors

Introduction

Slow coronary flow (SCFP) was first reported by Tambe et al in 1972, which is characterized by delayed coronary blood flow in the absence of obstructive coronary artery disease (CAD).[1] Initially, SCFP was considered to be a benign angiographic finding. With the deepening of understanding and research, SCFP has been suggested as an ominous phenomenon that predisposes individuals to atherosclerosis and obstructive CAD.[2] Moreover, in some case reports, SCFP is associated with sudden cardiac death due to ventricular arrhythmias[3] and ST-segment elevation myocardial infarction (STEMI).[4] As a subtype of ischemia and no obstructive coronary arteries (INOCA), SCFP had quite a complex pathogenesis. Although the pathogenic mechanism of SCFP is not fully elucidated, accumulating evidence has confirmed

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that early atherosclerosis,[2] microvascular disorder,[5] endothelial function,[6,7] oxidative stress,[7] and inflammation[8–12] are recognized as important pathogenesis in the initiation and development of SCFP.

Inflammation plays a pivotal role in the development of atherosclerosis and CAD,[13] the monocytes, neutrophils, and lymphocytes are intricately involved in this process.[14] Consequently, in recent years, a growing body of research has centered on routine blood tests and their derived indicators, which are easily acquired and calculated. The aggregate index of systemic inflammation (AISI), which represents the degree of immunity and inflammation through multiple blood cells, such as neutrophils, lymphocytes, monocytes, and platelets, was initially shown to be associated with prolonged hospital stay in open elective thoracic surgery.[15] Recently, AISI has been increasingly investigated in the context of CAD.[16–21]

Given the complex pathogenesis of SCFP, the diverse clinical manifestations, and the significant association between inflammation and SCFP, we aimed to investigate the potential role of the AISI in the occurrence of SCFP. We hypothesized that high AISI is associated with an increased risk of SCFP in patients with INOCA.

Methods

Study Population

We consecutively recruited 1328 patients with INOCA in the present study between March 2019 and February 2024. The flowchart of the study is presented in Figure 1. 90 patients had SCFP (SCFP group), accounting for 6.8% of individuals with INOCA. A total of 180 age - and sex - matched individuals with INOCA and normal blood flow were selected as controls at a ratio of 1:2 using a simple random sampling method (n=180). In total, 270 individuals were included in the study. The exclusion criteria are shown in Figure 1. This study was conducted in accordance with the ethical standards of the Helsinki Declaration. Informed consent was obtained before participation.

Coronary Angiography

Coronary angiography was performed by experienced cardiologists using the standard Judkins technique, and the Thrombolysis in Myocardial Infarction (TIMI) frame count (TFC) was used to quantitatively evaluate the coronary blood flow.[22] The first frame was defined as antegrade contrast filling in 70% of the ostium of left anterior descending artery (LAD), left circumflex (LCX) and right coronary artery (RCA). The last frame was defined as the contrast filling the mustache area for the LAD, bifurcation segment with the farthest distance for the LCX, and first branch of the posterolateral artery for the RCA.[22] TFC was calculated as the last frame count minus the first frame count.[22] Considering the longer distance of the LAD, the corrected TFC (TFC divided by 1.7) was calculated to assess the coronary blood flow of the LAD.[22] The cut off value of TFC for SCFP were 36.2±2.6 for the LAD (21.1±1.5 cTFC), 22.2 ±4.1 for the LCX, and 20.4±3.1 for the RCA.[16] If the TFC of any of the three main coronary vessels exceeded the

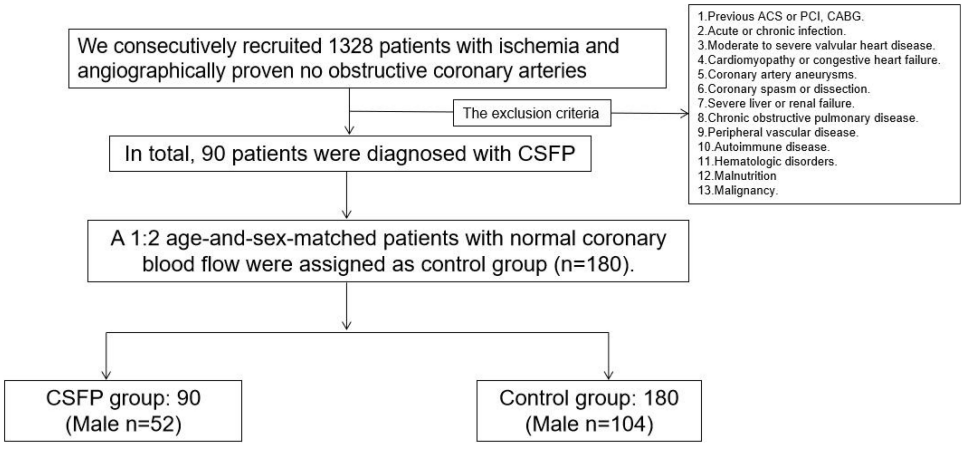

Figure 1 The study flowchart.

diagnostic criteria, SCFP was diagnosed. The mean TFC (mTFC) was calculated by dividing the sum of the TFC of the three main vessels by three.[8–11]

Laboratory Measurements

Routine blood biochemical examinations were conducted prior to the procedure. Laboratory parameters, including routine blood testing, were performed at the core laboratory of our hospital. AISI was calculated as follows: neutrophil count × platelet count × monocyte count platelet/lymphocyte count.[15]

Statistical Analysis

SPSS software (version 20.0) was used for data processing. Categorical variables are shown as rates or percentages and were analyzed using chi-square or Fisher's exact test. Continuous variables were displayed as mean±standard deviation or median and 25th-75th percentile values, which were compared using the *t*-test or Mann–Whitney *U*-test, as applicable. Univariate logistic regression analysis was used to investigate indicators associated with the presence of SCFP. Factors with a p value <0.1 were added in the multivariate logistic regression analysis to determine the independent predictors for the presence of SCFP in patients with INOCA. Smoking status, WBC count, AISI, hypertension, and diabetes mellitus were included in the univariate analysis. The receiver operating characteristic (ROC) curve was used to determine the predictive value of the factors associated with CSFP. The 2-sided P<0.05 was considered statistical significance.

Results

Baseline and Clinical Characteristics

The baseline characteristics and medication histories are displayed in Table 1. Variables including age, sex, body mass index (BMI), heart rate, comorbidities (hypertension and diabetes mellitus), and medication history did not show significant difference between the two groups (p>0.05) (Table 1). However, compared to controls, patients with SCFP had a higher proportion of current smokers (p < 0.05) (Table 1).

Laboratory Parameters of the Two Groups

The laboratory indicators are listed in Table 2. An increased white blood cell (WBC) count, neutrophil count, monocyte count, platelets count, and AISI were observed in patients with CSFP compared to controls (p< 0.05) (Table 2). For the remaining parameters, including lymphocyte count, fasting blood glucose (FBG), creatinine (Cr), albumin (ALB), and lipid indicators, there were no significant differences between the two groups (p>0.05) (Table 2).

Table 1 Baseline Characteristics and Medication of the Two Groups

	SCFP Group (n=90)	Control Group (n=180)	P value
Age, years	62.6±12.4	62.6±12.4	1
Male sex, n (%)	52(57.8)	104(57.8)	1
BMI	25.1±3.5	25.4±3.2	0.517
Heart rate	71.1±13.7	73.1±13.6	0.815
Current smoking, n (%)	26(28.9)	26(14.4)	0.006
Hypertension, n (%)	49(54.4)	77(42.8)	0.092
Diabetes mellitus, n (%)	19(21.1)	22(12.2)	0.071
ACEI/ARB/ARNI, n (%)	22(24.4)	39(21.7)	0.644
Beta-blocker, n (%)	25(27.8)	56(31.1)	0.673
Calcium canal blocker, n (%)	18(20.0)	39(21.7)	0.874
Antiplatelet, n (%)	24(26.7)	44(24.4)	0.766
Statin, n (%)	21(23.3)	48(26.7)	0.657
Nitrates, n (%)	26(28.9)	48(26.7)	0.772

Abbreviations: BMI, body mass index; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; ARNI, angiotensin receptor enkephalinase inhibitor.

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Table 2 Laboratory Parameters of the Two Groups

	SCFP Group (n=90)	Control Group (n=180)	P value
WBC count, 10^9/L	7.4±2.4	6.6±1.7	0.002
Neutrophils count, 10^9/L	4.8±1.6	4.3±1.6	0.020
Lymphocytes count, 10^9/L	1.8±0.7	1.7±0.6	0.444
Monocytes count, 10^9/L	0.5±0.2	0.5±0.1	<0.001
Platelets count, 10^9/L	243.3±56.2	222.4±53.2	0.003
AISI	396.2±275.4	287.0±218.9	<0.001
FBG, mmol/l	6.5±2.5	6.3±2.6	0.526
Cr, mmol/l	69.5±17.6	67.6±18.9	0.438
Uric acid, umol/L	340.9±105.2	335.7±92.4	0.679
ALB, g/L	40.3±3.7	40.5±3.7	0.698
TC, mmol/l	4.4±1.0	4.4±1.1	0.561
TG, mmol/l	1.5±1.1	1.6±1.5	0.518
HDL-C, mmol/l	1.1±0.4	1.1±0.3	0.290
LDL-C, mmol/l	2.7±0.9	2.8±0.7	0.343

Abbreviations: WBC, white blood cell; AISI, aggregate index of systemic inflammation; FBG, fasting blood glucose; Cr, creatinine; ALB, albumin; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

Table 3 Angiographic Findings of the Two Groups

	SCFP Group (n=90)	Control Group (n=180)	P value
TIMI frame count			<0.001
LAD	35.2 ±6.4	23.6 ± 2.3	
LCX	29.4 ± 3.5	21.1 ± 1.8	
RCA	28.3 ± 3.9	20.8 ± 2.0	
Mean TFC	31.0 ± 11.0	23.0 ± 0.9	
Distribution of SCFP			
LAD, n (%)	57 (63.3)		
LCX, n (%)	55 (61.1)		
RCA, n (%)	72 (80.0)		
Numbers of vessels involved in SCFP			
1, n (%)	29 (32.2)		
2, n (%)	28 (31.1)		
3, n (%)	33 (36.7)		

Abbreviations: TIMI, thrombolysis in myocardial infarction; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; TFC, TIMI frame count; SCFP, slow coronary flow phenomenon.

Angiographic Findings of the Two Groups

The angiographic findings are shown in Table 3. We found that 90 individuals developed SCFP in at least one main coronary vessel. The TFCs in patients with SCFP were significantly higher than those in controls ($p < 0.001$) (Table 3). A total of 29 individuals (32.2%) were affected by SCFP in one coronary arteries, 28 (3.1%) in two coronary arteries, and 33 (36.7) in three coronary arteries. In terms of the vessels involved, the RCA was the most frequently affected, followed by LAD and LCX (Table 3).

Predictors of SCFP

Smoking, WBC count, AISI, hypertension, and diabetes mellitus were included in the univariate analysis to investigate the potential risk factors for the presence of SCFP. As shown in Table 4, the WBC count and AISI were associated with SCFP. Multivariate logistic analysis showed that WBC count and AISI were independent predictors of SCFP in patients with INOCA (Table 4). In addition, we found that the AISI increased as the number of vessels affected by SCFP increased (Figure 2). The ROC curve was used to assess the predictive value of indicators for the presence of SCFP.

Table 4 Univariate and Multivariate Logistic Regression Analysis for Presence of SCFP

	Univariate Analysis			Multivariate Analysis		
	OR	95% CI	P value	OR	95% CI	P value
Current smoking	0.418	0.512–1.187	0.418			
WBC	1.223	1.069–1.339	0.003	1.155	1.005–1.327	0.042
AISI/100	1.196	1.075–1.331	0.001	1.154	1.032–1.290	0.012
Hypertension	0.684	0.411–1.137	0.143			
Diabetes mellitus	0.591	0.309–1.129	0.111			

Abbreviations: WBC, white blood cell; AISI, aggregate index of systemic inflammation.

When the AISI was > 264.1, the sensitivity and specificity were 64.4% and 64.4%, respectively, and the area under the ROC curve (AUC) was 0.657 (95% CI: 0.590–0.723, P < 0.001). AISI had a better predictive value for the presence of SCFP than WBC, neutrophils, lymphocytes, monocytes, and platelets alone (Table 5 and Figure 3).

Discussion

In the current study, we first explored the relationship between the AISI and the presence of SCFP in patients with INOCA. We found that a higher AISI score was associated with an increased risk of SCFP among INOCA patients. Additionally, the AISI increased with the number of vessels affected by SCFP. AISI could serve as a promising indicator for risk assessment as well as for the appropriate management of INOCA.

As a matter of fact, SCFP is not merely an angiographic finding. This study had several important clinical implications. Unlike cardiac X syndrome (CXS), patients with SCFP are more prone to suffer angina attacks at rest rather than exertional angina in CXS.[6,9–11] Moreover, nearly 20% of SCFP patients had repeated emergency visits or

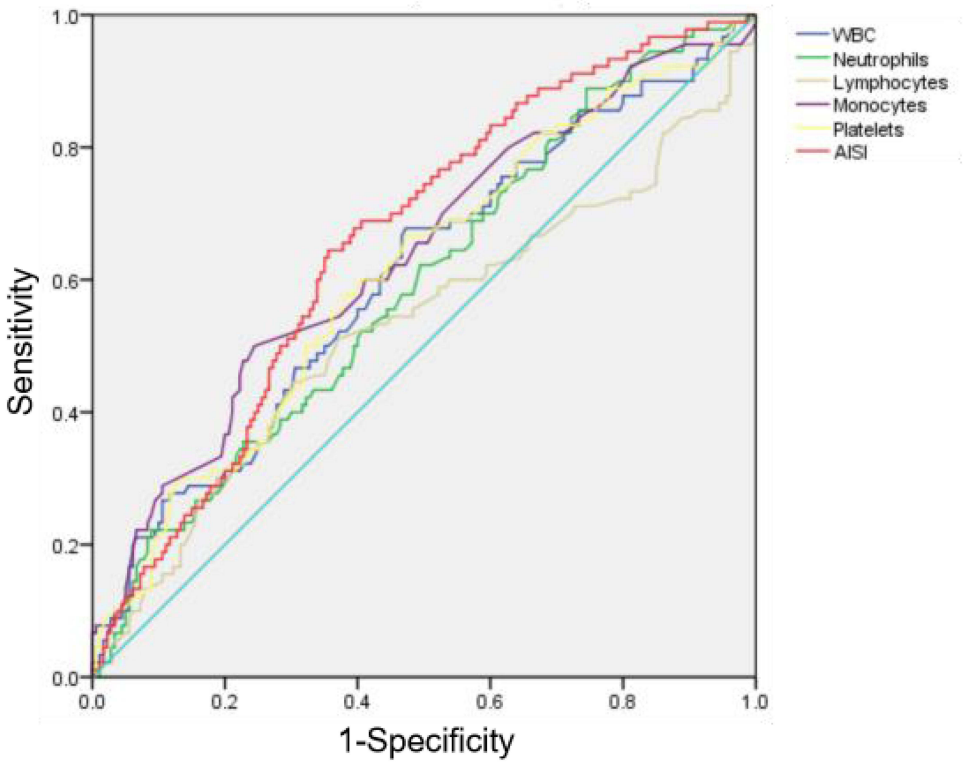

Figure 2 Correlation between the number of vessels affected by SCFP and AISI.

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Table 5 ROC Analysis of the Related Parameters

	AUC	95% CI	P value
WBC	0.604	0.532–0.677	0.005
Neutrophils	0.590	0.519–0.660	0.016
Lymphocytes	0.530	0.452–0.608	0.419
Monocytes	0.809	0.756–0.863	<0.001
Platelets	0.609	0.537–0.681	0.003
AISI	0.657	0.590–0.723	<0.001

Abbreviations: WBC, white blood cell; AISI, aggregate index of systemic inflammation.

admissions to the CCU due to recurrent angina attacks resembling ACS.[5,6,9–11] Some patients may even experience sudden cardiac death[3] and STEMI.[4] Therefore, precise prediction of SCFP in INOCA is important for the optimal management of these specific populations. However, the available indicators for the predicting SCFP in patients with INOCA are still limited. Hence, in the present study, we aimed to explore the easily acquired and calculated indicators for the presence of SCFP in patients with INOCA to improve the management of these patients.

However, the relationship between the traditional CVD risk factors and SCFP remains unclear. Some studies have suggested that hypertension and diabetes are the risk factors for SCFP.[23,24] In other studies, no significant associations were found.[6,8–11,25] In the present study, there was no significant difference in the incidence of hypertension and diabetes between the groups. However, the p-value was less than 0.1. Therefore, hypertension and diabetes were included in univariate analysis. We found that hypertension and diabetes were not associated with SCFP. We speculate that SCFP is a unique clinical entity with complex pathogenesis and diverse clinical manifestations. Different studies had different comorbidities, medications, sample sizes, and races; therefore, different results.[9–11] Nonetheless, optimal management of these risk factors is still important.

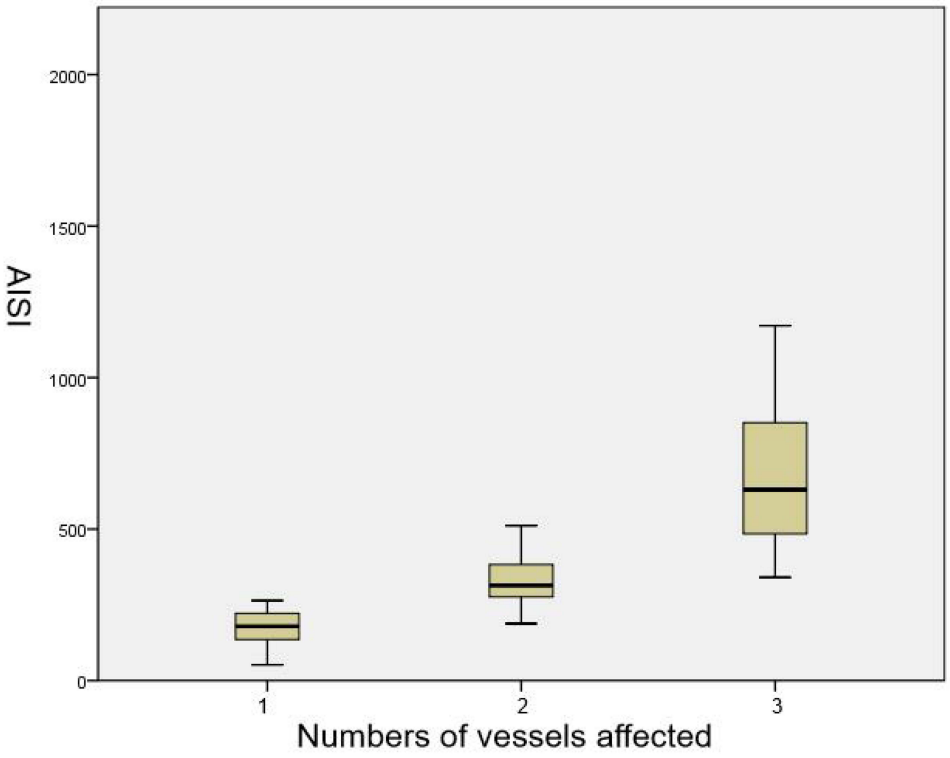

Figure 3 ROC curve showing the predicting value of risk factors for the presence of SCFP.

Although the pathogenesis of SCFP is not fully understood, substantial evidence supports the relationship between inflammation and SCFP.[8–12] As a newly developed indicator for assessing inflammation, the AISI integrates various blood cells, including neutrophils, lymphocytes, monocytes, and platelets.[15] It is well established that an increased inflammatory reaction is a significant pathological change during the initiation and development of CAD.[13] Systemic inflammation can increase platelet reactivity, resulting in localized ischemia, hypoxia, and microthrombus formation, ultimately causing tissue necrosis.[26] A decreased lymphocyte count suggests increased cell death and subsequent immune system dysfunction.[20] Lymphocyte apoptosis, along with an increase in monocytes and neutrophils, collectively aggravates the development of atherosclerosis,[20] resulting in a higher risk of SCFP. A high AISI indicates an increased inflammatory response and disrupted immune function, which contribute to the initiation and development of SCFP in patients with INOCA. A recent study demonstrated that patients with SCFP had higher neutrophil counts and that the neutrophil percentage to albumin ratio was an independent predictor of the presence of SCFP.[9] Moreover, in recent years, indicators derived from blood cells, including pan-immune-inflammation,[12] systemic immune-inflammation index (SII),[8] and systemic inflammation response index (SIRI),[10] have been proven to be associated with the presence of SCFP in patients with INOCA. Nonetheless, the relationship between the AISI and SCFP remains undetermined. Therefore, we aimed to investigate the potential role of the AISI in the presence of SCFP in patients with INOCA. We discovered that patients with SCFP had increased AISI, which could serve as a valuable predictor of SCFP occurrence in INOCA. Initially, AISI was demonstrated as a predictor of an increased risk of prolonged hospital stay in patients who underwent open chest surgery.[15] Recently, the AISI has been widely discussed in CAD. AISI is associated with in-stent restenosis after PCI,[16] major adverse cardiovascular events (MACE) in patients with acute coronary syndrome,[17] myocardial infarction with non-obstructive coronary arteries,[18] cardiovascular death in acute myocardial infarction,[20] and in-hospital mortality in patients with aortic stenosis after mechanical aortic valve replacement.[21] It is speculated that this association between AISI and CVD may be caused by the combined effects of different inflammatory blood cells. Considering the contributions of these inflammatory blood cells to the pathogenesis of SCFP, the combined indicator of AISI has a biological rationale and a reliable clinical indication.

Limitations

This study had some limitations. First, it was a single-center study with a relatively small sample size, which may have limited the analysis. Second, although we tried to include all the indicators associated with SCFP, some may have escaped from this study, which may have affected prediction efficiency. Third, the gold standard index of inflammation, C-reactive protein (CRP), was not included in the present study. Finally, large-scale multicenter studies are required to validate our conclusions.

Conclusion

We discovered that increased AISI may provide a valuable and reliable method for distinguishing SCFP from INOCA. AISI provided a better predictive value for the presence of SCFP than indicators alone.

Data Sharing Statement

The datasets generated and analyzed during the current study are not publicly available because of further study in this area but are available from the corresponding author upon reasonable request.

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Disclosure

The authors declare that they have no conflicts of interest in this work.

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