RESEARCH ARTICLE

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Comparative Assessment of High-sensitive CRP and Carotid Stiffness between Patients with and without Coronary Slow Flow

Remon Saleh Adly[1], Sally Magdy Teima[2], Shereen Philip Aziz[3], Ahmed Ibrahim Bedier[2]

[1]Department of Cardiology, Sohag University Faculty of Medicine, Sohag, Egypt
[2]Department of Cardiology, Mansoura University Faculty of Medicine, Dakahlia, Egypt
[3]Department of Clinical Pathology, Sohag University Faculty of Medicine, Sohag, Egypt

Abstract

Background and Aim: Coronary slow flow (CSF) phenomenon represents a distinct clinical entity characterized by delayed coronary opacification despite absence of significant epicardial stenosis. The pathophysiology involves microvascular dysfunction, endothelial impairment, and systemic inflammation. This study evaluated high-sensitivity C-reactive protein (hs-CRP) levels and carotid stiffness parameters in cases with CSF and in controls with normal coronary flow (CF).

Materials and Methods: This case-control study included 60 participants who underwent coronary angiography for chest pain evaluation. Participants (n=30) were divided into two groups: the CSF group, defined by corrected thrombolysis in myocardial infarction frame count >27, and the control group, with normal CF. Bilateral carotid artery ultrasonography and hs-CRP measurement were performed. Carotid stiffness parameters included β-stiffness index, Peterson's elastic modulus, distensibility coefficient, compliance coefficient, and arterial strain.

Results: CSF cases demonstrated significantly higher hs-CRP levels than controls (3.35±1.48 vs. 1.18±0.62 mg/L; $P < 0.001$). Carotid assessment revealed increased carotid intima-media thickness (0.82±0.24 vs. 0.68±0.19 mm, $P = 0.012$), elevated β-stiffness index (7.68±2.71 vs. 6.04±1.62, $P = 0.006$), and Peterson's elastic modulus (637.33±279.7 vs. 379.67±249.81 kPa, $P < 0.001$). Decreased distensibility and compliance coefficients indicated reduced arterial compliance.

Conclusion: CSF is associated with higher hs-CRP and adverse carotid stiffness profiles. These findings support an inflammatory-vascular dysfunction axis in the CSF and justify further studies assessing their prognostic utility.

Keywords: Coronary slow flow, high-sensitivity C-reactive protein, carotid stiffness, arterial compliance, microvascular dysfunction

INTRODUCTION

Coronary slow flow (CSF) is an angiographic phenomenon in which contrast advances slowly to distal coronary landmarks despite the absence of flow-limiting epicardial stenosis. Patients commonly present with angina-like chest pain and ischemic symptoms, yet coronary angiography (CAG) shows no obstructive coronary artery disease, leaving uncertainty about both the mechanism and the prognosis. Contemporary reviews increasingly interpret CSF within the spectrum of ischemia with non-obstructive coronary arteries, where coronary microvascular dysfunction and abnormal coronary vasomotor tone can exist even when the epicardial vessels appear angiographically normal.[1,2]

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Address for Correspondence: Sally Magdy Teima MD, Department of Cardiology, Mansoura University Faculty of Medicine, Dakahlia, Egypt
E-mail: sally88@mans.edu.eg
ORCID ID: orcid.org/0000-0001-7960-4816

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In daily practice, CSF is identified corrected thrombolysis in myocardial infarction (TIMI) frame count (cTFC), an accessible angiographic surrogate for delayed resting flow, although invasive physiological indices provide a more direct assessment of microvascular function.[2]

Several interrelated pathways may contribute to CSF. Endothelial dysfunction with reduced nitric oxide bioavailability, oxidative stress, and low-grade inflammation can promote microvascular constriction, adverse remodeling, and impaired perfusion, ultimately manifesting as delayed angiographic opacification.[2] High-sensitivity C-reactive protein (hs-CRP) is an accessible marker of low-grade vascular inflammation and residual inflammatory risk in atherosclerotic cardiovascular disease.[3] Prior work has reported higher hs-CRP levels in CSF compared with controls and linked inflammatory activation to endothelial dysfunction and microvascular impairment in this setting.[4,5]

More recently, broader inflammatory indices (e.g., the systemic immune-inflammation index) have also been explored in relation to CSF, reinforcing the concept that CSF may reflect a chronic inflammatory vascular milieu rather than an isolated coronary phenomenon.[6,7]

Large-artery structure and biomechanics offer another window into the systemic vascular milieu in which CSF occurs. Carotid intima-media thickness (CIMT) reflects arterial remodeling, while stiffness indices—including the β-stiffness index, Peterson's elastic modulus, distensibility, compliance, and strain—capture complementary aspects of arterial wall mechanics and have prognostic relevance across cardiovascular conditions.[8,9]

Echo-tracking and ultrasonographic studies have suggested that carotid stiffness is higher in CSF and that stiffness may be related to inflammatory activity.[10] Notably, measured stiffness is strongly influenced by blood pressure, including hypertension, making careful interpretation and appropriate adjustments essential when comparing stiffness parameters between non-randomized groups.[8,9]

Although pooled evidence (e.g., Moawad et al.[11]) supports associations between inflammatory biomarkers and impaired coronary flow patterns, many reports emphasize inflammation alone and do not consistently combine it with detailed carotid biomechanical phenotyping or address blood pressure-related confounding.

Accordingly, the present study, conducted in a rigorously selected cohort that excluded overt inflammatory conditions, was designed to compare hs-CRP levels and a comprehensive panel of carotid stiffness parameters between patients with CSF and controls with normal coronary flow and to contextualize vascular stiffness findings in light of blood pressure, a key determinant of arterial biomechanics.

METHODS

This case-control study included 60 participants (both sexes, aged 18 years or older) who underwent CAG for evaluation of chest pain or suspected myocardial ischemia at Sohag University Hospitals, Egypt, from May to December 2025. The Institutional Review Board/Ethics Committee of Sohag University accepted the study protocol (approval no: Soh-Med-25-5-6PD, date: 12.05.2025). Written informed consent was obtained from all participants prior to enrollment, and the study was conducted in accordance with the Declaration of Helsinki.

Patients referred for diagnostic CAG because of chest discomfort or suspected ischemia were recruited consecutively. The recruitment period was sufficient to achieve the planned sample size.

Based on angiographic findings, participants were divided into two equal groups (n=30): the CSF group, defined by cTFC >27, and the control group, characterized by normal coronary flow (CF) and absence of obstructive coronary artery disease.

Participants were excluded if they presented with any of the following conditions: pregnancy or lactation, a documented history of cardiovascular system (CVS) events, including myocardial infarction or cerebrovascular accident; diagnosed autoimmune disorders; chronic hepatic or renal dysfunction; current or recent infections; a history of malignancy; or concurrent use of medications known to influence inflammatory markers, such as corticosteroids or immunosuppressive agents.

Clinical Assessment and Data Collection

Comprehensive clinical evaluation was performed for all participants, encompassing detailed medical history, including demographic characteristics and smoking habits, anthropometric measurements with calculation of body mass index (BMI), blood pressure measurements, and assessment of comorbid conditions, including diabetes mellitus and hypertension. A complete physical examination was conducted according to standard clinical protocols.

Laboratory Investigations

Venous blood samples were collected after an overnight fast of 8-12 hours for biochemical analysis. hs-CRP concentrations were determined using a high-sensitivity enzyme-linked immunosorbent assay kit, with levels exceeding 2 mg/L were considered elevated according to the American College of Cardiology/American Heart Association guidelines. Additional laboratory parameters included fasting blood glucose, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, serum creatinine levels, total cholesterol, and triglycerides.

Cardiac Imaging and Angiographic Assessment

Transthoracic echocardiography was performed using standard protocols to evaluate cardiac structure and function. CAG was conducted via the femoral or radial approach, and CF velocity was quantified using the cTFC method to identify cases with the CSF phenomenon.

cTFC was assessed offline by two experienced interventional cardiologists who were blinded to participants' clinical data, laboratory results, and carotid ultrasound measurements. The TIMI frame count was defined as the number of cine frames required for the contrast to reach a predefined distal landmark in each major epicardial artery. For the left anterior descending, the raw frame count was corrected by dividing by 1.7 to yield the cTFC. CSF was defined as cTFC >27 in at least one major coronary artery, in the absence of obstructive epicardial coronary artery disease.

Carotid Artery Evaluation

Bilateral carotid artery ultrasonography was performed using a high-resolution B-mode ultrasound system equipped with a 7.5-MHz linear array transducer. Comprehensive measurements included CIMT assessment, determination of systolic and diastolic arterial diameters, and calculation of multiple arterial stiffness parameters, including the β-stiffness index, Peterson's elastic modulus, the distensibility coefficient, the compliance coefficient, and the arterial strain values.

Carotid systolic diameter (Ds) was measured at peak systole and diastolic diameter (Dd) at end-diastole (electrocardiography-gated when available), and values were averaged over three consecutive cardiac cycles. Stiffness indices were calculated as follows:

- **Arterial strain (%)**: (Ds-Dd)/Dd×100
- **β-stiffness index**: ln [systolic blood pressure (SBP)/diastolic blood pressure (DBP)]/[(Ds-Dd)/Dd]
- **Peterson's elastic modulus**: (SBP-DBP)/[(Ds-Dd)/Dd]
- **Distensibility coefficient**: 2×[(Ds-Dd)/Dd]/(SBP-DBP)
- **Compliance coefficient**: [π (Ds^2-Dd^2)/4]/(SBP-DBP)

where SBP and DBP denote systolic and diastolic blood pressures, respectively.

Sample Size Calculation

G*Power 3.1.9.2 (Universitat Kiel, Germany) was employed to calculate the sample size. We carried out a pilot study (five cases in each group), and we found that the mean [± standard deviation (SD)] of hs-CRP was 3.39±1.66 in the case group and 1.374±0.50 in the control group. The sample size was determined using factors such as an effect size of 0.829, a 95% confidence level, 80% study power, a 1:1 group ratio, and the addition of six cases per group to account for dropout. Therefore, we recruited 30 participants per group.

Statistical Analysis

Data were analyzed using SPSS v29 (IBM©, Armonk, NY, USA). To assess the normality of the data distribution, both the Shapiro-Wilk test and histograms were used. Quantitative data conforming to a parametric distribution were reported as mean and SD and analyzed using an unpaired Student's t-test, whereas non-parametric data were reported as median and interquartile range and analyzed using the Mann-Whitney U test. Conversely, qualitative measures were summarized as frequencies and percentages, and analyzed using the chi-square test or Fisher's exact test, as appropriate for the dataset. Univariate regression was used to estimate the relationship between a dependent variable and one independent variable. Multivariate regression was also used to estimate the relationship between a dependent variable and multiple independent variables. Statistical significance was defined as a two-tailed $P \leq 0.05$.

RESULTS

No significant differences were observed in age, sex, weight, height, BMI, diabetes, and smoking. Hypertension and systolic and DBPs were considerably higher in the CSF group than in the control group ($P < 0.05$) (Table 1 and Figure 1).

Both groups had similar levels of fasting blood glucose, total cholesterol, HDL, LDL, triglycerides, and serum creatinine. hs-CRP was significantly increased in the CSF group compared with the control group ($P < 0.001$) (Table 2 and Figure 2).

Both groups had similar systolic and diastolic diameters. The CSF group had significantly higher CIMT, β-stiffness index, and Peterson's elastic modulus than the control group ($P < 0.05$). Distensibility, compliance, and strain were considerably reduced in CSF group compared to control group ($P < 0.05$) (Table 3 and Figure 3).

In univariate regression analysis, hypertension, systolic and DBP, hs-CRP, CIMT, β-stiffness index, and Peterson's elastic modulus were independent predictors of the occurrence of CSF ($P < 0.05$). In multivariate regression analysis, DBP, hs-CRP, CIMT, β-stiffness index, and Peterson's elastic modulus were independent predictors of the occurrence of CSF ($P < 0.05$), while hypertension and SBP were not independent predictors (Table 4).

Table 1. Demographic data and blood pressure of the studied groups

		CSF group (n=30)	Control group (n=30)	*P*-value	MD/OR (95% CI)
Age (years)		53.73±14.44	55.13±16.54	0.728	-1.4 (-9.43 to 6.63)
Sex	Male	19 (63.33%)	13 (43.33%)	0.121	2.26 (0.8 to 6.36)
	Female	11 (36.67%)	17 (56.67%)		
Weight (kg)		80.03±12.19	82.3±8.73	0.411	-2.27 (-7.75 to 3.21)
Height (cm)		171.07±6.6	168.83±6.49	0.191	2.23 (-1.15 to 5.61)
Body mass index (kg/m^2)		27.36±3.98	29.05±4.26	0.118	-1.69 (-3.82 to 0.44)
Comorbidities	Hypertension	17 (56.67%)	9 (30%)	**0.037***	3.05 (1.05 to 8.84)
	Diabetes mellitus	7 (23.33%)	10 (33.33%)	0.390	0.61 (0.2 to 1.9)
	Smoking	11 (36.67%)	7 (23.33%)	0.260	1.9 (0.62 to 5.86)
Systolic blood pressure (mmHg)		132.07±15	123.5±10.34	**0.013***	8.57 (1.91 to 15.22)
Diastolic blood pressure (mmHg)		88.43±10.32	81.1±8.62	**0.004***	7.33 (2.42 to 12.25)

Data was presented as mean ± standard deviation or frequency (%)

CSF: Coronary slow flow, MD: Mean difference, OR: Odds ratio, CI: Confidence interval, *: Statistically significant $P < 0.05$

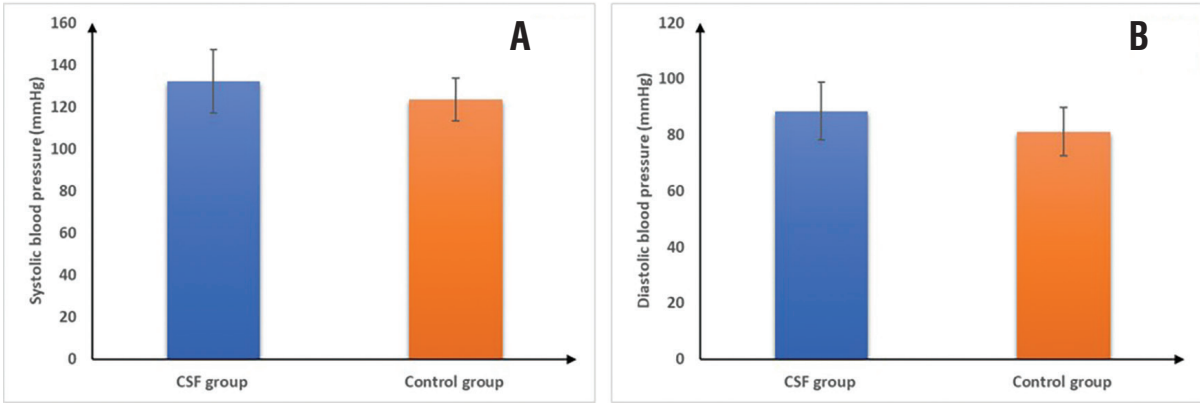

Figure 1. (A) Systolic blood pressure (B) diastolic blood pressure of the studied groups

CSF: Coronary slow flow

Table 2. Laboratory investigation of the studied groups

	CSF group (n=30)	Control group (n=30)	*P*-value	MD (95% CI)
Fasting blood glucose (mg/dL)	108.57±35.62	106.9±27.57	0.840	1.67 (-14.8 to 18.13)
Total cholesterol (mg/dL)	137.57±40.4	127.83±60.83	0.468	9.73 (-16.95 to 36.42)
High density lipoprotein (mg/dL)	40.1±4.87	41.73±6.73	0.286	-1.63 (-4.67 to 1.4)
Low density lipoprotein (mg/dL)	101.17±39.28	120.87±64.44	0.158	-19.7 (-47.28 to 7.88)
Triglycerides (mg/dL)	164.13±31.73	166.43±32.7	0.783	-2.3 (-18.95 to 14.35)
Serum creatinine (mg/dL)	1.04±0.42	0.98±0.31	0.561	0.06 (-0.13 to 0.25)
High-sensitive C-reactive protein (mg/L)	3.655 (2.38-4.28)	1.035(0.78-1.73)	**<0.001***	-2.3 (-2.94 to -1.59)

Data are presented as mean ± standard deviation or median (interquartile range), as appropriate

CSF: Coronary slow flow, MD: Mean or median difference, CI: Confidence interval, *: Statistically significant $P < 0.05$

DISCUSSION

The CSF phenomenon represents a complex clinical entity characterized by delayed opacification of coronary arteries in the absence of significant epicardial stenosis, with emerging evidence suggesting that systemic inflammatory and vascular processes play fundamental roles in its pathogenesis.[12,13]

The present study demonstrates a pronounced elevation of hs-CRP levels in cases with CSF compared with controls, a 2.8-fold increase that underscores the significant inflammatory burden associated with CSF. This finding is consistent with previous investigations: Barutcu et al.[4] reported elevated hs-CRP concentrations in CSF cases and demonstrated positive correlations between inflammatory markers and TFC across all major coronary arteries. Similarly, Ardahanli and Özmen[14] found significantly elevated hs-CRP levels in CSF samples and identified hs-CRP as a sensitive indicator of inflammatory severity with notable diagnostic value.

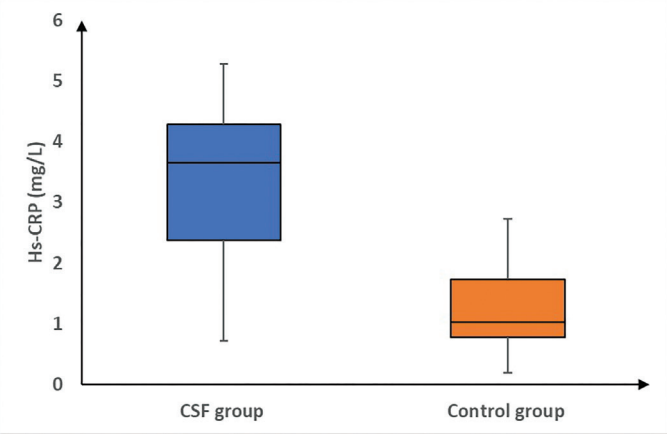

Figure 2. hs-CRP of the studied groups

CSF: Coronary slow flow, hs-CRP: High-sensitive C-reactive protein

Moawad et al.[11] conducted a comprehensive meta-analysis confirming that elevated hs-CRP levels are significant predictors of CSF, no-reflow, and high thrombus burden, thereby supporting inflammation as a fundamental pathophysiological mechanism.

The lack of significant differences in metabolic parameters between CSF cases and controls suggests that traditional risk factors may not be primary contributors, whereas Wen et al.[7] identified the inflammatory burden index as an independent predictor, highlighting the metabolic-inflammatory interface in the condition.

The observed increase in CIMT in CSF cases reflects a broader pattern of systemic vascular pathology that extends beyond the coronary circulation. This finding suggests that CSF may represent a manifestation of generalized arterial disease rather than an isolated coronary microvascular disorder. The increased CIMT observed in our study population indicates structural vascular changes that are characteristic of early atherosclerotic processes, which supports the hypothesis that CSF cases exhibit accelerated vascular aging and an increased CVS risk profile.

Significantly elevated arterial stiffness parameters in CSF cases, as demonstrated by Yang et al.[12] using echo-tracking technology, indicate impaired vascular elasticity and are positively correlated with hs-CRP levels, suggesting that inflammation contributes to vascular structural changes. Similarly, Yang et al.[13] demonstrated elevated carotid artery stiffness parameters in CSF cases and found that these measures independently predicted the condition.

The reduced arterial compliance, as evidenced by decreased distensibility and compliance coefficients in our study population, reflects functional impairment of vascular elastic properties. This pattern is consistent with Wang et al.[14] who reported significantly higher elastic modulus values in CSF cases compared with healthy controls, with differences persisting after adjustment for age and smoking status.

Table 3. Carotid artery ultrasound and stiffness measurement of the studied groups

	CSF group (n=30)	Control group (n=30)	*P*-value	MD (95% CI)
Carotid intima-media thickness (mm)	0.82±0.24	0.68±0.19	**0.012***	0.15 (0.03 to 0.26)
Systolic diameter (mm)	6.12±0.65	5.81±0.83	0.115	0.31 (-0.08 to 0.69)
Diastolic diameter (mm)	5.54±0.69	5.37±0.7	0.368	0.16 (-0.2 to 0.52)
β-stiffness index	7.68±2.71	6.04±1.62	**0.006***	1.64 (0.48 to 2.8)
Peterson's elastic modulus (kPa)	637.33±279.7	379.67±249.81	**<0.001***	257.7 (120.6 to 394.7)
Distensibility coefficient (10^{-3} kPa)	19.8±1.07	23.04±7.35	**0.02***	-3.24 (-5.95 to -0.52)
Compliance coefficient (mm^2 kPa^{-1})	5.24±4.69	7.19±1.82	**0.038***	-1.95 (-3.79 to -0.11)
Strain (%)	7.33±1.86	8.58±2.24	**0.022***	-1.25 (-2.31 to -0.18)

Data are presented as mean ± standard deviation

CSF: Coronary slow flow, MD: Mean difference, CI: Confidence interval, *: Statistically significant $P < 0.05$

This association between hypertension and CSF is supported by the broader understanding of hypertension as a systemic vascular disorder that affects both large and small vessels. Shroff et al.[15] demonstrated relationships between arterial stiffness and CVS biomarkers, including hs-CRP, suggesting that arterial stiffness, inflammation, and cardiac function are interconnected processes.

Low-grade inflammation may facilitate carotid remodeling via endothelial activation, diminished nitric oxide bioavailability, oxidative stress, and upregulated pathways that enhance vascular smooth muscle proliferation and extracellular matrix turnover (e.g., elevated collagen levels and compromised elastin integrity), which collectively result in increased arterial stiffness. Discrepancies among published research may indicate changes in inclusion criteria, comorbidities (notably hypertension), and the exact vascular measures employed.

In our cohort, traditional metabolic parameters were similar between groups. However, because blood pressure/hypertension burden was higher in the CSF group, we performed adjusted analyses to avoid attributing stiffness differences solely to CSF status. The observational design also cannot determine temporality; inflammation and stiffness may precede CSF, arise concurrently from shared vascular pathology, or represent downstream consequences of microvascular dysfunction.

From a clinical standpoint, carotid stiffness indices may complement hs-CRP by reflecting vascular target-organ

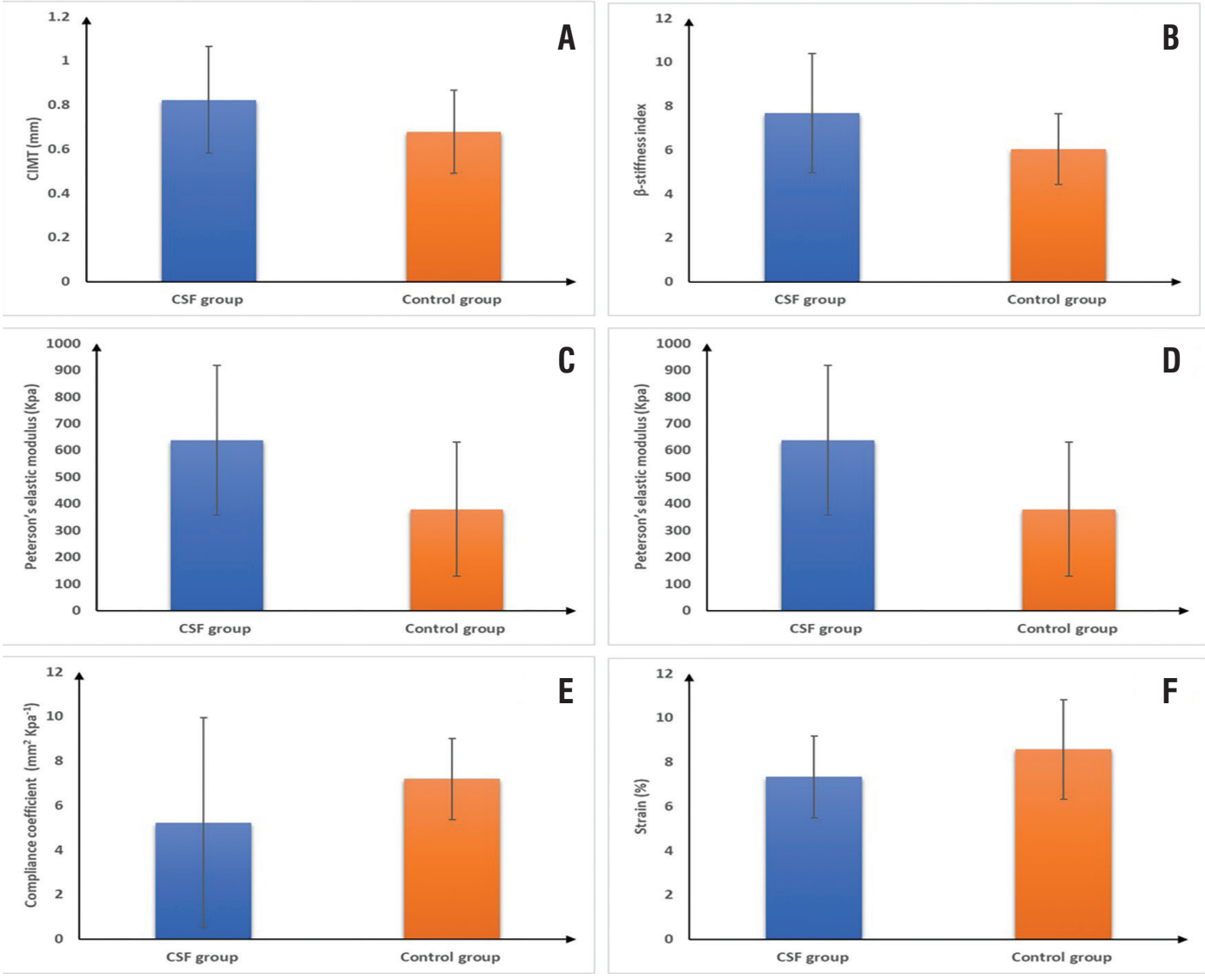

Figure 3. (A) CIMT (B) β-stiffness index (C) Peterson's elastic modulus (D) distensibility coefficient (E) compliance coefficient (F) strain of the studied groups

CIMT: Carotid intima-media thickness, CSF: Coronary slow flow

Table 4. Univariate and multivariate regression of different variables versus occurrence of coronary slow flow

	Univariate			Multivariate		
	OR	**95% CI**	***P*-value**	**OR**	**95% CI**	***P*-value**
Body mass index	0.903	0.794 to 1.03	0.121	-	-	-
Hypertension	3.051	1.05 to 8.84	**0.039***	0.014	0 to 2.63	0.109
Systolic blood pressure	1.052	1.01 to 1.09	**0.015***	1.49	0.992 to 2.25	0.055
Diastolic blood pressure	1.081	1.022 to 1.14	**0.006***	1.49	1.04 to 2.11	**0.028***
High-sensitive C-reactive protein	5.65	2.36 to 13.57	**<0.001***	14.90	2.56 to 86.87	**0.003***
Carotid intima-media thickness	22.48	1.77 to 284.79	**0.016***	38.78	1.6637 to 903.96	**0.023***
β-stiffness index	1.39	1.08 to 1.79	**0.010***	1.58	1.11 to 2.23	**0.01***
Peterson's elastic modulus	1.004	1.001 to 1.01	**0.002***	1.01	1.002 to 1.01	**0.001***

*: Statistically significant $P < 0.05$, CI: Confidence interval, OR: Odds ratio

involvement; however, whether stiffness provides incremental prognostic value beyond hs-CRP in CSF patients requires larger prospective studies with hard clinical endpoints.

Study Limitations

First, it was a single-center case-control study with a modest sample size; the pilot-based sample size estimation (n=5 per group) may have inflated the expected effect size, potentially reducing power to detect smaller true differences and widening confidence intervals. Second, CSF was defined using cTFC, an angiographic surrogate that does not directly quantify coronary microvascular function; the absence of invasive physiological assessment (e.g., coronary flow reserve or index of microcirculatory resistance) may have introduced misclassification and diluted associations. Finally, the cross-sectional design precludes causal inference and does not establish whether inflammation, arterial stiffness, or both precede CSF or occur concomitantly.

CONCLUSION

CSF cases showed significantly higher hs-CRP levels, increased carotid stiffness (elevated CIMT, β-stiffness index, and Peterson's modulus), reduced distensibility and compliance, and a higher prevalence of hypertension, highlighting systemic inflammation and vascular dysfunction that may serve as biomarkers of vascular pathology and guide risk stratification and management in CSF.

Ethics

Ethics Committee Approval: The Institutional Review Board/Ethics Committee of Sohag University accepted the study protocol (approval no: Soh-Med-25-5-6PD, date: 12.05.2025).

Informed Consent: Written informed consent was obtained from all participants prior to enrollment, and the study was conducted in accordance with the Declaration of Helsinki.

Footnotes

Authorship Contributions

Surgical and Medical Practices: R.S.A., S.P.A., Concept: R.S.A., S.P.A., Design: S.M.T., A.I.B., Data Collection or Processing: R.S.A., S.M.T., A.I.B., Analysis or Interpretation: S.M.T., A.I.B., Literature Search: R.S.A., A.I.B., Writing: S.M.T.

Conflict of Interest: No conflict of interest was declared by the authors.

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