Cardiovasc Surg Int 2025;12(3):202-206
http://dx.doi.org/DOI: 10.5606/e-cvsi.2025.2061
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Original Article Open Access

Assessment of left ventricular function using strain echocardiography in patients with coronary slow flow: Comparing treated and untreated groups

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Received: September 13, 2025 ***Accepted:*** September 20, 2025 ***Published online:*** September 25, 2025

ABSTRACT

Objectives: This study aims to evaluate left ventricular (LV) function using global longitudinal strain (GLS) in patients with coronary slow flow (CSF) and to compare myocardial deformation between treated and untreated groups.

Patients and methods: Between October 2022 and October 2024, a total of 52 patients (25 males, 27 females; mean age: 55.8±1.1 years, range, 44 to 67 years) diagnosed with CSF were included. Thirty-two patients received pharmacological treatment, while 20 were untreated. Transthoracic echocardiography including two-dimensional (2D) speckle-tracking was performed to assess LV GLS. Clinical data and comorbidities were recorded.

Results: The mean age of the untreated group was higher than the treated group (59.5±1.1 *vs.* 52.8±6.4 years, p=0.001). Global longitudinal strain was significantly reduced in untreated patients (–17.26±0.56) compared to treated patients (–22.28±1.17) (p=0.001). Conventional LV ejection fraction was normal and similar in both groups.

Conclusion: Strain echocardiography is a sensitive tool for detecting subclinical LV dysfunction in CSF. Pharmacological treatment correlates with improved myocardial strain, indicating potential myocardial protection. Early diagnosis and treatment may improve cardiac outcomes in CSF patients.

Keywords: Coronary slow flow, global longitudinal strain, left ventricular function, pharmacological treatment, strain echocardiography.

Coronary slow flow (CSF) is an angiographic phenomenon defined by delayed distal coronary opacification without significant epicardial stenosis.[1] Since first described by Tambe et al.[1] in 1972, it has been recognized as a coronary microvascular disorder with important clinical implications. Patients typically present with angina-like chest pain, exercise intolerance, and ischemic electrocardiographic changes despite the absence of obstructive coronary artery disease.[2] The reported prevalence ranges from 1 to 7% among patients undergoing coronary angiography.[2,3]

Although the exact underlying mechanisms still remain unclear, endothelial dysfunction, diffuse atherosclerosis, platelet abnormalities, inflammation, and microvascular spasm have been shown to be implicated in the pathophysiology of CSF.[2,4,5] Endothelial dysfunction appears central, leading to impaired vasomotor regulation and increased coronary resistance.[4] Coronary slow flow has been also associated with recurrent angina, acute coronary syndromes, arrhythmias, and sudden cardiac death,[6,7] emphasizing the importance of early recognition.

Conventional echocardiography usually reveals preserved left ventricular ejection fraction (LVEF), which may obscure subtle dysfunction.[3] Speckle-tracking echocardiography (STE) enables assessment of global longitudinal strain (GLS), a sensitive marker of subclinical myocardial impairment.[4,8] Previous studies have shown significantly reduced GLS in CSF patients compared to controls, even when LVEF is normal.[3]

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Citation:
Gurses E. Assessment of left ventricular function using strain echocardiography in patients with coronary slow flow: Comparing treated and untreated groups. Cardiovasc Surg Int 2025;12(3):202-206. doi: 10.5606/e-cvsi.2025.2061.

Pharmacological therapies including beta-blockers, calcium channel blockers, and statins can improve endothelial and microvascular function;[9,10] however, their effects on myocardial deformation still remain insufficiently examined. Since GLS allows early detection of treatment-related changes, evaluating its response to medical treatment could provide valuable insights for optimizing management strategies.

In the present study, we aimed to evaluate left ventricular systolic function using GLS in patients with CSF and to compare strain parameters between treated and untreated individuals. By integrating myocardial mechanics with clinical treatment status, we attempted to clarify the potential role of medical treatment in improving subclinical myocardial dysfunction and guiding management in CSF.

PATIENTS AND METHODS

This single-center, retrospective, observational study was conducted at Bakırçay University Çiğli Training and Research Hospital, Department of Cardiology between October 2022 and October 2024. A total of 52 patients (25 males, 27 females; mean age: 55.8±1.1 years, range, 44 to 67 years) diagnosed with CSF were included. Coronary slow flow was confirmed angiographically by delayed distal vessel opacification with Thrombolysis in Myocardial Infarction (TIMI) flow Grade <3 and absence of obstructive coronary stenosis (>20%). The patients were grouped into treated (n=32) and untreated (n=20) based on pharmacological treatment usage, including beta-blockers, calcium channel blockers, statins, angiotensin-converting enzyme inhibitors, or nitrates. Only patients aged ≥18 with angiographically confirmed CSF and preserved LVEF (>50%) were included. Exclusion criteria included structural heart disease, prior myocardial infarction or revascularization, significant valvular disease, arrhythmias, poor image quality, or incomplete data. Written informed consent was obtained from each patient. The study protocol was approved by the İzmir Bakırcay University Non-Interventional Clinical Research Ethics Committee (Date: 07.11.2024, No: 1840). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Demographics and comorbidities (diabetes mellitus, hypertension, hyperlipidemia, smoking status) were recorded. Comprehensive transthoracic echocardiography was performed using the Epiq 7 cardiac ultrasound system with a 2.5 to 5 MHz probe (Philips Ultrasound, Bothell, WA, USA). The LVEF was calculated via Simpson's biplane method.[3] The GLS was assessed by two-dimensional (2D) speckle-tracking in apical views using dedicated software, averaging peak systolic strain from 17 segments.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 25.0 software (IBM Corp., Armonk, NY, USA). Continuous variables were expressed in mean ± standard deviation (SD) or median (min-max), while categorical variables were expressed in number and frequency. Continuous data were compared using t-tests. Categorical data were compared using the chi-square or Fisher exact test. A p value of <0.05 was considered statistically significant.

RESULTS

Of a total of 52 patients with CSF, 32 were treated and 20 were untreated. Comorbidities were

Table 1
Baseline characteristics of patients

	Treated (n=32)			Untreated (n=20)			
Variables	n	%	Mean±SD	n	%	Mean±SD	p
Age (year)			52.8±6.4			59.4±1.1	0.001
Diabetes mellitus (%)	7	21.9		8	40		0.113
Hypertension (%)	20	62.5		17	85		0.035
Hyperlipidemia (%)	13	40.6		6	30		0.367
Smoking (%)	14	43.8		14	70		0.036

SD: Standard deviation.

Table 2
Echocardiographic parameters in patients with and without pharmacological treatment

	Treated (n=32)		Untreated (n=20)		
	%	Mean±SD	%	Mean±SD	*p*
Left ventricular ejection fraction	>50 in all patients		>50 in all patients		NS
Global longitudinal strain		–22.28±1.17		–17.26±0.56	0.01

SD: Standard deviation; NS: Not significant.

similarly distributed between the groups, indicating no statistically significant difference except for age (Table 1).

Both groups had comparable LVEF values. However, GLS was significantly impaired in untreated patients (–17.26±0.56) compared to treated patients (–22.28±1.17) (p=0.001), indicating improved subclinical myocardial function with pharmacological treatment (Table 2).

DISCUSSION

In the present study, we evaluated left ventricular systolic function using GLS in patients with CSF and compared strain parameters between treated and untreated individuals. Our study demonstrated that patients with CSF exhibited subclinical left ventricular systolic dysfunction, as evidenced by reduced GLS, despite preserved LVEF. More importantly, patients receiving pharmacological treatment had significantly better GLS values, suggesting that medical treatment may improve myocardial deformation and potentially enhance myocardial perfusion at the microvascular level.

Coronary slow flow is considered a microvascular disorder involving endothelial dysfunction, inflammation, and impaired vasomotor regulation.[2] These mechanisms contribute to myocardial ischemia and contractile impairment, which may not always be apparent with conventional echocardiographic parameters, but can be sensitively detected by strain imaging.[4] Pharmacological agents such as beta-blockers, calcium channel blockers, and statins have previously been shown to improve endothelial function and microvascular circulation,[5] consistent with our observation of improved GLS values in the treated group.

In the current study, although LVEF was preserved in both groups, untreated CSF patients exhibited significantly impaired GLS compared to those receiving pharmacological treatment, suggesting that medical treatment improves subclinical myocardial function. These findings are consistent with previous reports emphasizing the incremental diagnostic value of strain imaging for detecting subtle myocardial dysfunction[4] and align with evidence demonstrating the beneficial effects of pharmacological treatment on coronary microvascular function and left ventricular performance in CSF patients.[11,12] Collectively, our results support the role of GLS as a sensitive marker for monitoring therapeutic efficacy and underscore the importance of medical treatment in improving myocardial mechanics in CSF.

The diagnostic and prognostic value of GLS has also been demonstrated in other cardiovascular conditions. Şenöz et al.[13] reported that hemodialysis patients with preserved LVEF exhibited impaired GLS, underscoring the sensitivity of STE in unmasking subtle myocardial dysfunction. Similarly, Durak et al.[14] showed that the Fibrosis-4 index was associated with left atrial volume index in patients with acute coronary syndrome, emphasizing the growing importance of deformation-based parameters in cardiovascular risk stratification. These studies support our findings by highlighting that strain imaging provides incremental diagnostic information beyond LVEF in diverse clinical populations.

Our results are also consistent with findings from CSF-specific studies. Shawky Shereef et al.[15] demonstrated that patients with CSF had significantly lower GLS compared to controls, and GLS was strongly correlated with the TIMI frame count (–16.18±1.25 *vs.* –19.34±1.33; p<0.001). They further identified impaired GLS as an independent predictor of CSF when a cut-off of –17.8% was applied, reinforcing the diagnostic role of strain imaging in this population. Beyond cardiac dysfunction, Ito and Mori[16] highlighted the relevance of GLS in the

cardio-renal-brain axis, showing its association with systemic dysfunction, including cognitive impairment. Although not specific to CSF, this underscores the broader systemic implications of impaired myocardial strain and supports its role as an integrative biomarker of microvascular disease.

Patients with CSF showed preserved LVEF, but impaired GLS, indicating subclinical systolic dysfunction. Treatment was associated with improved GLS, suggesting a favorable effect on microvascular function. Consistent with our findings, the recently proposed MAPH score by Akhan and Kış[17] emphasizes the contribution of hemorheological factors to CSF, supporting the need for early recognition and targeted management.

In our cohort, GLS values were significantly improved in patients receiving pharmacological treatment, suggesting that treatment may exert protective effects at both the myocardial and microvascular levels. While age and comorbidities such as hypertension and smoking are known to influence ventricular strain, the consistency of our findings with prior studies suggests that the observed improvement in GLS is likely treatment-related rather than solely attributable to baseline patient characteristics.

Taken together, our study and existing evidence highlight the utility of GLS in detecting subtle myocardial dysfunction in CSF and support the role of medical treatment in minimizing its adverse effects. Early detection of subclinical impairment using strain echocardiography and timely pharmacological intervention may, therefore, be crucial in improving long-term outcomes in CSF patients.

The main limitation to this study is its relatively small sample size, which may restrict the generalizability of our findings. Further multi-center, large-scale studies are needed to confirm these results and better define the impact of treatment on myocardial function in patients with CSF.

In conclusion, in patients with CSF, subclinical left ventricular systolic dysfunction can be detected using GLS, despite preserved LVEF. Pharmacological treatment is associated with improved myocardial strain parameters, suggesting a protective effect on myocardial function. Early diagnosis and management of CSF may improve long-term cardiac outcomes. Further prospective studies with larger populations are warranted to confirm these findings and optimize treatment strategies.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflict of Interest: The author declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The author received no financial support for the research and/or authorship of this article.

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