

Review Article

Reduced Left Ventricular Global Longitudinal Strain in the Coronary Slow Flow Phenomenon: A Systematic Review and Meta-Analysis

Mahin Seifi Alan ^{1,2‡}, Kobra Hosseini ^{1,2‡}, Sanaz Seifi Alan ^{1,2}, Zahra Mirzaei ², Peyman Saeedi ^{1,2}, Soroush Bahrami ¹, Vahid Shahnavaaz ², Haniyeh Rashidi ¹, Mahnaz Seifi Alan ^{1,2}, Hadith Rastad ^{3*}

¹ Clinical Research and Development Center of the Kamali Hospital, Alborz University of Medical Sciences, Karaj, Iran.

² Cardiovascular Research Center, Alborz University of Medical Sciences, Karaj, Iran.

³ Imperial Clinical Trials Unit, Imperial College London, London, UK.

‡ Mahin Seifi Alan and Kobra Hosseini contributed equally as co-first authors.

Citation: Seifi Alan M, Hosseini K, Seifi Alan S, Mirzaei Z, Saeedi P, Bahrami S, et al. Reduced Left Ventricular Global Longitudinal Strain in the Coronary Slow Flow Phenomenon: A Systematic Review and Meta-Analysis. Res Heart Yield Transl Med 2025; 20(4):310-322.

Highlights

- CSFP is associated with a significant reduction in left ventricular global longitudinal strain (LVGLS) despite preserved left ventricular ejection fraction (LVEF).
- Layer-specific strain analysis reveals the most pronounced impairment in the endocardial layer.
- LVGLS demonstrates moderate-to-high diagnostic accuracy for detecting CSFP (pooled AUC: 0.80).
- These findings support LVGLS as a noninvasive marker of subclinical myocardial dysfunction in CSFP.

Article info:

Received: 3 Aug. 2025

Revised: 24 Aug. 2025

Accepted: 4 Sep. 2025

ABSTRACT

Background: The coronary slow flow phenomenon (CSFP) involves delayed coronary artery filling without obstruction and is associated with angina and myocardial ischemia. This meta-analysis assessed the link between CSFP and impaired left ventricular global longitudinal strain (LVGLS), a marker of subclinical myocardial dysfunction.

Methods: A systematic search (PubMed, Embase, Scopus up to January 2025) identified 18 observational studies comparing LVGLS and layer-specific strain in patients with CSFP vs controls with normal coronary flow.

Results: Patients with CSFP showed significantly reduced LVGLS vs controls (SMD, 1.22; 95% CI, 0.69 to 1.75). Layer-specific analysis revealed impairment across all myocardial layers, most pronounced in the endocardium (SMD, 0.79; 95% CI, 0.21 to 1.38). While left ventricular ejection fraction (LVEF) was preserved, LVGLS demonstrated moderate-to-high diagnostic accuracy for CSFP (AUC, 0.80; 95% CI, 0.66 to 0.95). Reduced LVGLS independently predicted CSFP (adjusted OR, 1.43; 95% CI, 1.19 to 1.46). Exercise stress effects on LVGLS were inconsistent.

Conclusion: CSFP is associated with impaired LVGLS, particularly in the endocardial layer, despite preserved LVEF. LVGLS may serve as a noninvasive marker for subclinical dysfunction in CSFP.

* Corresponding Author:

Hadith Rastad 
Imperial Clinical Trials Unit, Imperial College London, London, UK.
Email: h.rastad@imperial.ac.uk

Keywords: Coronary Slow Flow Phenomenon; Left Ventricle Global Longitudinal Strain; Speckle-Tracking Echocardiography

Introduction

The coronary slow flow phenomenon (CSFP) is an angiographic phenomenon characterized by delayed or slow distal coronary vessel filling in the absence of obstructive epicardial coronary artery disease.¹ First described in 1972 by Tambe et al,² CSFP is increasingly being recognized as a cause of recurrent angina, exercise tolerance reduction, and adverse cardiovascular outcomes, including myocardial ischemia, arrhythmias, and sudden death.^{1,3} With an incidence of 1% to 7% among patients undergoing coronary angiography, CSFP poses diagnostic and therapeutic challenges because of its elusive pathophysiology and lack of standardized management protocols.³

The underlying mechanisms of CSFP remain incompletely understood, but current evidence points to microvascular dysfunction, endothelial impairment, and low-grade inflammation as key contributors. Histopathological studies reveal microvascular remodeling, including fibromuscular hyperplasia and capillary rarefaction, which may impair coronary perfusion.⁴ Despite these insights, the absence of definitive diagnostic criteria beyond the Thrombolysis in Myocardial Infarction (TIMI) frame count underscores the need for additional tools to evaluate CSFP-related myocardial dysfunction.⁵

Conventional measures of left ventricular function, such as left ventricular ejection fraction (LVEF), are often preserved in patients with CSFP, masking subclinical systolic impairment because of their load dependence and limited sensitivity.⁶ In contrast, left ventricular global longitudinal strain (LVGSL), assessed via speckle-tracking echocardiography (STE), has emerged as a robust, angle-independent marker of myocardial deformation. LVGSL detects early LV dysfunction by quantifying longitudinal shortening, which is particularly vulnerable to microvascular ischemia.^{1,3}

Nonetheless, the available information on the reduction of LVGSL in CSFP is scattered in the literature, making it difficult to utilize this information for clinical decision-making.

Accordingly, the present systematic review and meta-analysis aimed to provide a cohesive evaluation of the association between LVGSL and CSFP.

Methods

Study Design

This systematic review and meta-analysis adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines⁷ to ensure methodological rigor and transparency. The study protocol was registered on PROSPERO (ID: CRD42025639236) before data extraction to minimize bias.

Search Strategy

A comprehensive literature search was performed across PubMed, Embase, and Scopus from inception until January 2025. The search strategy combined Medical Subject Headings (MeSH) terms and free-text keywords related to CSFP (eg, “coronary slow flow,” “CSFP,” and “TIMI frame count”) and LVGSL (eg, “global longitudinal strain” and “speckle tracking echocardiography”). Boolean operators (AND, OR) were used to refine the search, which was restricted to English-language studies. Manual screening of reference lists from included studies and relevant reviews was also conducted to identify additional eligible publications.

Study Selection

Studies were included if they met the following criteria: the population consisted of adults (aged ≥ 18 y) undergoing coronary angiography with LVGSL assessment by STE; the exposure was angiographically confirmed CSFP; the comparison group consisted of participants with normal coronary flow; and the outcome of interest was LVGSL. Eligible study designs included observational studies (case-control or cross-sectional, cohort) or randomized controlled trials reporting LVGSL values in patients with CSFP. Exclusion criteria were reviews, case reports, editorials, or conference abstracts; studies not reporting LVGSL or using nonstandard STE methods; and studies focusing

on obstructive coronary artery disease or other cardiac pathologies.

Screening Process

Two independent reviewers screened titles and abstracts, followed by full-text assessment. Discrepancies were resolved via consensus or consultation with a third reviewer (Figure 1).

Data Extraction

Data extraction was performed using a standardized form to collect study characteristics (author, year, country, design, sample size, and demographics), LVGLS measurement details (STE method, vendor software, and strain parameters), and outcomes (diagnostic accuracy and correlation with LVEF or TIMI frame count). The methodological quality of included studies was assessed using the Newcastle-Ottawa Scale (NOS), which evaluates selection, comparability, and outcome domains. Studies were classified as high quality (NOS score ≥ 7), moderate quality (NOS score 5–6), or low quality (NOS score ≤ 4) (Table 1).

Statistical Analysis

Statistical analysis was performed using R software (meta package). The primary outcome was the standardized mean difference (SMD) in LVGLS values between patients with CSFP and controls with 95% confidence intervals (CIs). Heterogeneity was assessed using the I^2 statistic, with I^2 greater than 50% indicating high heterogeneity and warranting a random-effects model. Secondary analyses included pooled diagnostic accuracy (area under the curve [AUC] from receiver operating characteristic [ROC] analyses) and subgroup analyses (layer-specific strain).

Ethical Considerations

Because this study synthesized published data, ethical approval was not required.

Results

Study Selection and Characteristics

A total of 18 studies were included in this systematic review and meta-analysis, evaluating the predictive value of LVGLS for CSFP.^{1,3,8-23} The baseline characteristics of the included studies are summarized in (Table 2). Studies were conducted in Egypt, Turkey, Iran, China, and Serbia, with sample sizes ranging from 40 to 133 participants.^{8,9,13} The mean age in the CSFP group ranged from 44.6 to 60.0 years,^{3,10} while the control group ranged from 43.5 to 59.0 years.^{3,10} Male participants constituted 40% to 75% of the CSFP group^{8,9} and 33% to 80% of the control group.^{1,8} Common comorbidities such as diabetes mellitus, hypertension, and dyslipidemia were reported with varying prevalence.

LVGLS and CSFP

The association between LVGLS and CSFP was evaluated across multiple studies, as detailed in (Table 3). Most studies reported significantly lower LVGLS values in the CSFP group compared with the control group, indicating impaired myocardial function in patients with CSFP. For example, Shereef et al³ reported a mean LVGLS of -16.2 (1.3) in the CSFP group vs -19.3 (1.3) in the control group ($P<0.001$). Similarly, Liu et al¹³ found a mean LVGLS of -18.2 (2.8) in the CSFP group compared with -19.7 (2.2) in the control group ($P<0.001$). Multivariate logistic regression analysis in several studies, such as Shereef et al³ and Liu et al,¹³ demonstrated that LVGLS was a significant predictor of CSFP, with odds ratios (ORs) ranging from 1.2 to 2.2.^{3,12} The pooled analysis of LVGLS showed a significant difference between the CSFP and control groups, with an SMD of 1.22 (95% CI, 0.69 to 1.75; $P<0.001$; $I^2=93\%$), indicating reduced LVGLS in the CSFP group (Figure 2). Reduced LVGLS was independently associated with CSFP in the meta-analysis of adjusted ORs (pooled adjusted OR, 1.43 [95% CI, 1.19 to 1.46]; $P=0.018$; $I^2=70.2\%$) (Figure 3).

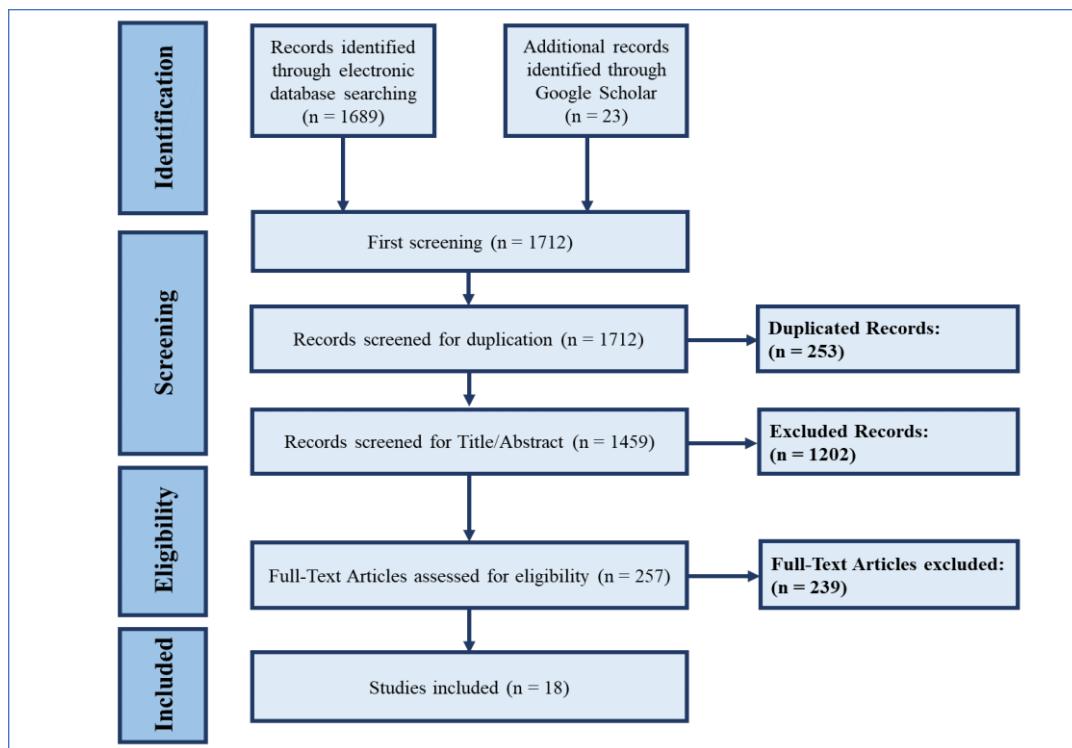


Figure 1. PRISMA flowchart of the literature search and selection process

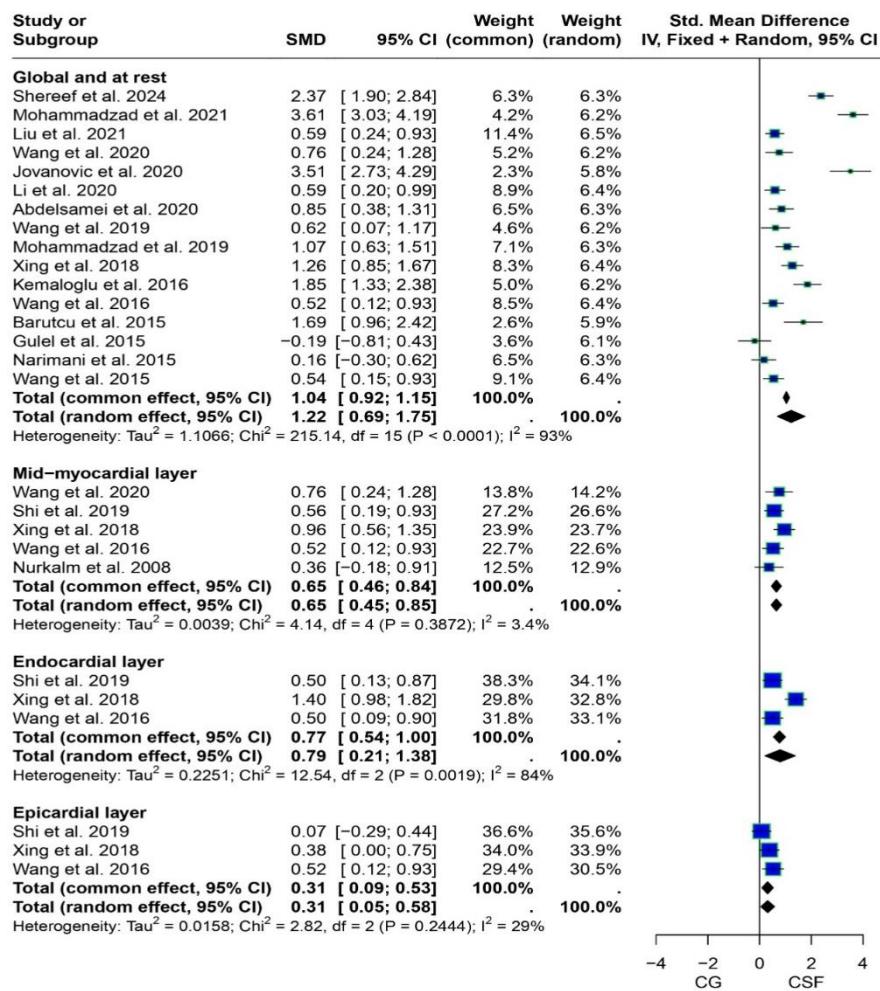


Figure 2: Forest plot of standardized mean difference (SMD) with 95% CI and weights for studies comparing Left ventricle global longitudinal strain (LVGLS) and layer-specific strain (mid-myocardial, endocardial, epicardial) in the CSF versus CG. CG: Control Group; CI: Confidence Interval; CSF: Coronary Slow Flow; SD: Standard Deviation

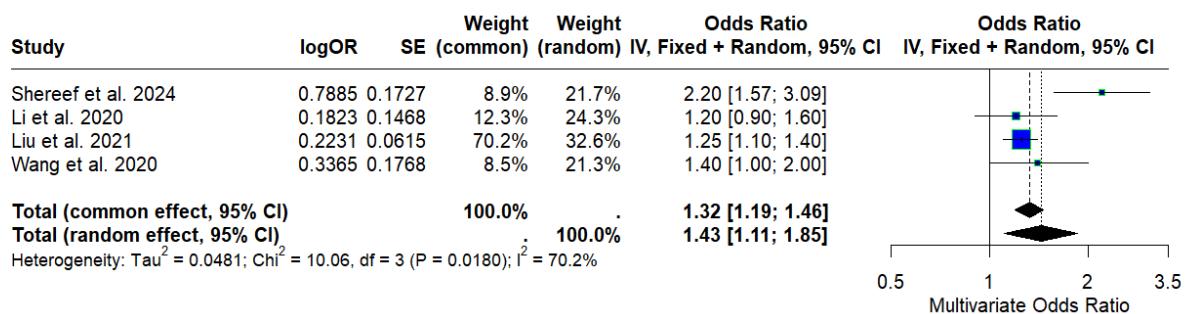


Figure 3. Pooled analysis of adjusted odds ratios (OR) and 95% confidence intervals (95% CIs) for LVGLS in CSF.

Diagnostic Accuracy of LVGLS

The diagnostic accuracy of LVGLS for predicting CSFP was assessed using ROC curve analysis. The AUC values ranged from 0.66 to 0.96,^{3,12} indicating moderate to high diagnostic accuracy. For example, Shereef et al³ reported

an AUC of 0.96 (95% CI, 0.92 to 0.99; $P < 0.001$) at a cutoff of 17.8%. The pooled AUC for global LVGLS in predicting CSFP was 0.80 (95% CI, 0.66 to 0.95; $P < 0.001$; $I^2 = 93.5\%$) (Figure 4). Notably, reported cutoffs varied across studies, ranging from 15.85% to 33.4%.^{13,14}

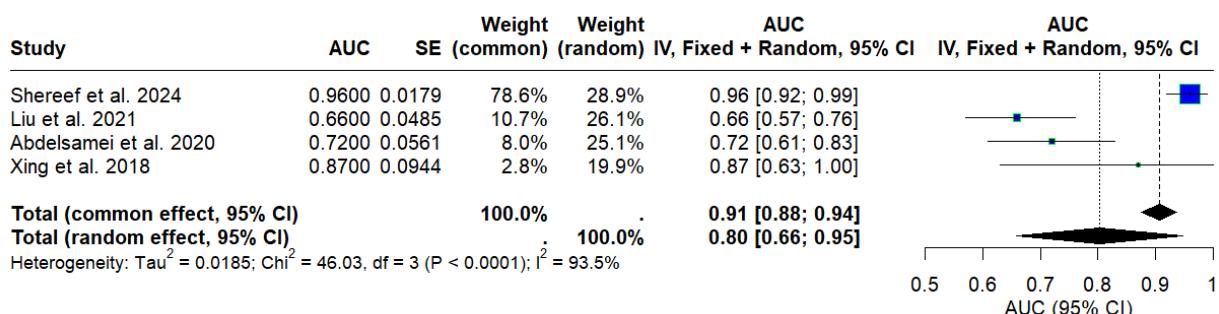


Figure 4. Forest plot of Area Under Curve (AUC) with standard error (SE) and weights for studies evaluating diagnostic accuracy. CI: Confidence Interval; SD: Standard Deviation.

Layer-Specific Strain Analysis

The meta-analysis results for layer-specific strain demonstrated significant reductions in the CSFP group compared with the control group across all myocardial layers. For the mid-myocardial layer, the pooled analysis under the common-effect model showed an SMD of 0.65 (95% CI, 0.46 to 0.84; $P = 0.387$; $I^2 = 3.4\%$). Similarly, the epicardial layer exhibited a smaller but statistically significant reduction under the common-effect model, with an SMD of 0.31 (95% CI, 0.09 to 0.53; $P = 0.244$; $I^2 = 29\%$). These findings align with the most pronounced reduction observed in the endocardial layer, where the random-effects model was applied because of high heterogeneity, yielding an SMD of 0.79 (95%

CI, 0.21 to 1.38; $P = 0.002$; $I^2 = 84\%$) (Figure 2).

TIMI Frame Count and LVEF

Supplementary Table 1 summarizes the TIMI frame count and LVEF in the CSFP and control groups. The mean TIMI frame count in the CSFP group ranged from 22.5 to 45.2,^{8,16} significantly higher than in the control group (range, 15.1–22.7)^{18,19} across all studies ($P < 0.001$), indicating slower coronary flow in patients with CSFP. LVEF in both groups was comparable, with the CSFP group ranging from 54.5% to 66.0%^{1,10} and the control group ranging from 54.8% to 68.5%.^{1,10} The percentages indicated no significant differences between the groups.

LVGLS After Exercise Stress

The analysis of exercise stress LVGLS in patients with CSFP revealed conflicting findings between the two included studies. Jovanovic et al¹⁰ reported a substantially lower LVGLS in the

CSFP group than in the controls (SMD, 4.74 [95% CI, 3.78 to 5.71]), whereas Wang et al¹⁹ found no significant difference (SMD, -0.26 [95% CI, -0.80 to 0.28]). The pooled effect size was an SMD of 2.23 (95% CI, -2.68 to 7.13; P<0.001; I²=98.7%) (Figure 5).

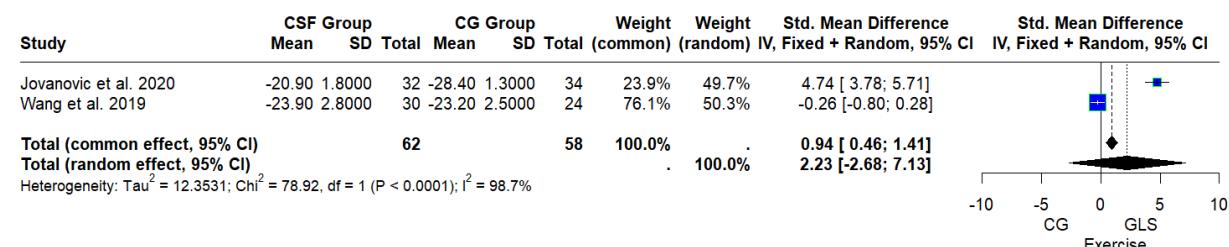


Figure 5. Forest plot of standardized mean difference (SMD) with 95% CI and weights for studies comparing Left ventricle global longitudinal strain (LVGLS) and in the CSF versus CG after exercise.

CG: Control Group; CI: Confidence Interval; CSF: Coronary Slow Flow; SD: Standard Deviation.

Meta-analysis of LVGLS in CSFP: 2D and 3D Studies

The pooled analysis of 16 studies using 2D imaging demonstrated a significant reduction in LVGLS among patients with CSFP (SMD, 1.12;

95% CI, 0.59 to 1.66; P<0.001; I²=93%), with individual study SMDs ranging from -0.19 to 3.61.^{9,1} Two studies utilizing 3D imaging also showed impaired LVGLS in patients with CSFP, with an SMD of 1.18 (95% CI, -0.12 to 2.47; P<0.001; I²=94%) (Figure 6).

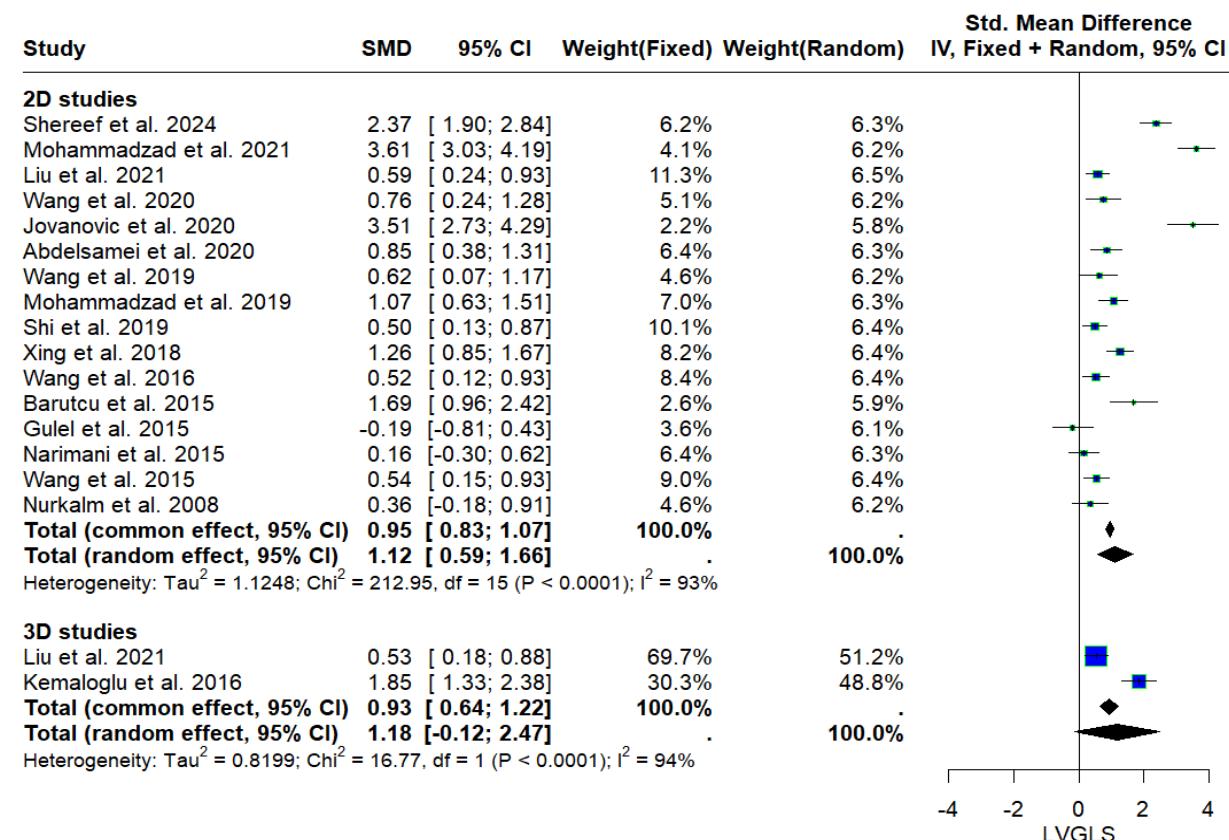


Figure 6. Forest plot comparing left ventricular global longitudinal strain (LVGLS) between the coronary slow flow phenomenon (CSFP) group and the control group (CG), stratified by imaging modality. Standardized mean differences (SMDs) with 95% confidence intervals (CIs) are shown for 2D speckle-tracking echocardiography studies and 3D speckle-tracking echocardiography studies.

CG: Control Group; CI: Confidence Interval; CSF: Coronary Slow Flow; SD: Standard Deviation.

Funnel Plot Analysis

Visual inspection of the funnel plot revealed symmetrical distribution of effect sizes around the pooled SMD (Figure 7), suggesting no evidence of publication bias. This was corroborated by the nonsignificant Q test for heterogeneity ($P=0.62$).

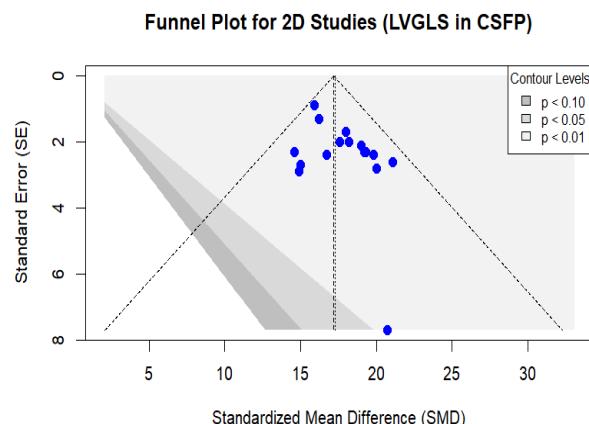


Figure 7. Funnel plot assessing publication bias in studies evaluating left ventricular global longitudinal strain (LVGLS) in the coronary slow flow phenomenon (CSFP)

Discussion

According to the present systematic review and meta-analysis, the CSFP group exhibited a significantly lower mean LVGLS (SMD, 1.22) than controls, whereas mean LVEF was comparable. Layer-specific strain analysis further revealed reduced deformation across all myocardial layers in patients with CSFP, with the most pronounced impairment in the endocardial layer. LVGLS showed moderate to high diagnostic accuracy for the presence of CSFP as reported by four studies (AUC range, 0.66–0.96; pooled AUC, 0.80).

Given the preserved LVEF, the reduced LVGLS highlights the value of this parameter in detecting subclinical myocardial dysfunction in CSFP. These findings align with a systematic review of evidence from observational studies, which demonstrated that LVGLS has superior prognostic value compared with LVEF for predicting major adverse cardiac events, including all-cause mortality, in patients with various cardiac conditions. The investigators concluded that LVGLS is essential for risk stratification and early intervention, whereas LVEF may miss significant impairment.^{24,25}

Although LVEF is a key systolic function marker, its limitations (load dependence, geometric assumptions, and insensitivity to early injury) reduce its reliability for subtle impairment. In contrast, LVGLS is more reproducible, geometry independent, and sensitive to longitudinal deformation abnormalities, particularly in the endocardial layer.^{24,25}

CSFP is not merely an angiographic curiosity but carries serious clinical consequences, including recurrent ischemia, life-threatening arrhythmias, and sudden cardiac death. Traditional tools such as the TIMI frame count are invasive and impractical for longitudinal monitoring, whereas transthoracic Doppler echocardiography remains underused. Given its significant predictive value for CSFP, LVGLS could serve as a noninvasive, reproducible metric to improve risk stratification, particularly in patients with preserved LVEF.^{24,25}

Limitations and Future Directions

The current study has several limitations, including small sample sizes that may affect generalizability, a lack of reported abnormal LVGLS proportions, and technical heterogeneity in measurement protocols, which could introduce bias. However, this study represents a systematic review and meta-analysis that comprehensively evaluates the reduction of LVGLS in CSFP.

Conclusion

The findings of our systematic review and meta-analysis suggest that CSFP may reduce LVGLS, particularly in the endocardial layer, whereas LVEF may remain unaffected. The observed reduction in LVGLS could serve as a potential diagnostic marker for CSFP, given its moderate to high diagnostic accuracy.

Declarations:

Ethical Approval

This study synthesized published data; thus, ethical approval was not required.

Funding

No funding was received for conducting this study.

Conflict of Interest

The authors declare no conflicts of interest.

Acknowledgment

The researchers appreciate the Clinical Research Development Units of Kamali and Rajaei Hospitals at Alborz University of Medical Sciences.

References

1. Seyyed Mohammadzad MH, Khademvatani K, Gardeshkhah S, Sedokani A. Echocardiographic and laboratory findings in coronary slow flow phenomenon: cross-sectional study and review. *BMC Cardiovasc Disord.* 2021;21(1):230.
2. Tambe A, Demany M, Zimmerman HA, Mascarenhas E. Angina pectoris and slow flow velocity of dye in coronary arteries—a new angiographic finding. *Am Heart J.* 1972;84(1):66-71.
3. Shereef AS, Abdelmajeed MG, Alshair MH, El-Dosouky II, Khalil WA, Wageeh S, et al. Coronary slow flow and its correlation with reduced left ventricle global longitudinal strain: a case-control study. *Echo Res Pract.* 2024;11(1):2.
4. Zhu Q, Wang S, Huang X, Zhao C, Wang Y, Li X, et al. Understanding the pathogenesis of coronary slow flow: recent advances. *Trends Cardiovasc Med.* 2024;34(3):137-44.
5. Barutçu A, Akşit E, Arslan M, Tok O, Kucück U. Evaluation of the effect of coronary slow flow phenomenon on cardiac functions. *Ann Clin Anal Med.* 2022;13(10):1098-1102.
6. Dong T, Zhu Q, Wang S, Liu S, Wang S, Wang Y, et al. Evaluation of left ventricular function in patients with coronary slow flow: A systematic review and meta-analysis. *Cardiol J.* 2023;30(6):929-37.
7. Vrabel M. Preferred Reporting Items for Systematic Reviews and Meta-Analyses. *Oncol Nurs Forum.* 2015;42(5):552-4.
8. Barutçu A, Bekler A, Temiz A, Kırılmaz B, Yener AÜ, Tan YZ, et al. Left ventricular twist mechanics are impaired in patients with coronary slow flow. *Echocardiography.* 2015;32(11):1647-54.
9. Gulel O, Akçay M, Soylu K, Aksan G, Yuksel S, Zengin H, et al. Left ventricular myocardial deformation parameters are affected by coronary slow flow phenomenon: a study of speckle tracking echocardiography. *Echocardiography.* 2016;33(5):714-23.
10. Jovanovic I, Tesic M, Giga V, Dobric M, Boskovic N, Vrtonjic J, et al. Impairment of coronary flow velocity reserve and global longitudinal strain in women with cardiac syndrome X and slow coronary flow. *J Cardiol.* 2020;76(1):1-8.
11. Kemaloğlu Öz T, Eren M, Atasoy I, Gürol T, Soylu Ö, Dağdeviren B. Are biventricular systolic functions impaired in patient with coronary slow flow? A prospective study with three-dimensional speckle tracking. *Int J Cardiovasc Imaging.* 2017;33(5):675-81.
12. Li J, Wang Y, Zhao C, Zhu Q, Li G, Yang J, et al. Incremental value of three-dimensional echocardiography for evaluating left atrial function in patients with coronary slow flow phenomenon: a case control study. *Cardiovascular Ultrasound.* 2020; 18:1-10.
13. Liu S, Wang Y, Li J, Li G, Mu L, Jia D, et al. Incremental value of three-dimensional speckle-tracking echocardiography for evaluating left ventricular systolic function in patients with coronary slow flow. *Curr Probl Cardiol.* 2022;47(9):100928.
14. MOHAMED MG, MAGDY MA, AHMED SE, ISLAM MA. Coronary Slow Flow Phenomenon: The Role of New Echo cardiographic Indices *Med J Cairo Univ.* 2020;88(9):1783-92.
15. Narimani S, Hosseinsabet A, Pourhosseini H. Effect of coronary slow flow on the longitudinal left ventricular function assessed by 2-dimensional speckle-tracking echocardiography. *J Ultrasound Med.* 2016;35(4):723-9.
16. Nurkalem Z, Gorgulu S, Uslu N, Orhan AL, Alper AT, Erer B, et al. Longitudinal left ventricular systolic function is impaired in patients with coronary slow flow. *Int J Cardiovasc Imaging.* 2009;25(1):25-32.

17. Seyyed-Mohammadzad MH, Rashtchizadeh S, Khademvatani K, Afsargharehbagh R, Nasiri A, Sepehrvand N. Ventricular dysfunction in patients with coronary slow-flow phenomenon: A single-center case-control study. *Heart Views*. 2020;21(2):60-4.
18. Shi J, Xing Y, Qian J, Yang H, Yan Y, Li Q, et al. Early assessment of left ventricular function by layer-specific strain and its relationship to pulsatile arterial load in patients with coronary slow flow. *Int Heart J*. 2019;60(3):586-92.
19. Wang Y, Li J, Liu S, Mu L, Li G, Yu H, et al. Value of exercise stress electrocardiography for stratification of exercise capacity and left ventricular systolic and diastolic function on coronary slow flow: case-control study. *BMC Cardiovasc Disord*. 2019;19(1):288
20. Wang Y, Ma C, Zhang Y, Guan Z, Liu S, Li Y, et al. Assessment of left and right ventricular diastolic and systolic functions using two-dimensional speckle-tracking echocardiography in patients with coronary slow-flow phenomenon. *PLoS One*. 2015;10(2):e0117979.
21. Wang Y, Ma C, Zhang Y, Guan Z, Liu S, Li Y, et al. Layer-specific analysis of left ventricular myocardial contractility in patients with coronary slow-flow phenomenon. *J Clin Ultrasound*. 2016;44(7):429-36.
22. Wang Y, Yu H, Zhao C, Li G, Yang J, Jia D, et al. Usefulness of soluble endothelial protein C receptor combined with left ventricular global longitudinal strain for predicting slow coronary flow: A case-control study. *Cardiol J*. 2022;29(4):619-26.
23. Xing Y, Shi J, Yan Y, Liu Y, Chen Y, Kong D, et al. Subclinical myocardial dysfunction in coronary slow flow phenomenon: identification by speckle tracking echocardiography. *Microcirculation*. 2019;26(1): e12509.
24. Kalam K, Otahal P, Marwick TH. Prognostic implications of global LV dysfunction: a systematic review and meta-analysis of global longitudinal strain and ejection fraction. *Heart*. 2014;100(21):1673-80.
25. Wang X, Nie S-P. The coronary slow flow phenomenon: characteristics, mechanisms and implications. *Cardiovasc Diagn Ther*. 2011;1(1):37-43.

Table 1: Quality Assessment of the included studies.

Author/Year	Representativeness of the sample:	Selection		Ascertainment of the exposure (risk factor):	Comparability		Outcome		Total score of 7 scores
		Non-respondents:	Representativeness of the sample:		Assessment of the outcome:	Statistical test:			
Shereef et al. 2024	*	*	*	*	**	*	*	*	7
Mohammadzad et al. 2021	*	*	*	*	*	*	*	*	6
Liu et al. 2021	*	*	*	*	**	*	*	*	7
Wang et al. 2020	*	*	*	*	**	*	*	*	7
Jovanovic et al. 2020	*	*	*	*	**	*	*	*	7
Li et al. 2020	*	*	*	*	**	*	*	*	7
Abdelsamei et al. 2020	*	*	*	*	**	*	*	*	7
Wang et al. 2019	*	*	*	*	*	*	*	*	6
Mohammadzad et al. 2019	*	*	*	*	*	*	*	*	6
Shi et al. 2019	*	*	*	*	**	*	*	*	7
Xing et al. 2018	*	*	*	*	**	*	*	*	7
Kemaloglu et al. 2016	*	*	*	*	**	*	*	*	7
Wang et al. 2016	*	*	*	*	*	*	*	*	6
Barutcu et al. 2015	*	*	*	*	*	*	*	*	6
Gulel et al. 2015	*	*	*	*	*	*	*	*	6
Narimani et al. 2015	*	*	*	*	*	*	*	*	6
Wang et al. 2015	*	*	*	*	*	*	*	*	6
Nurkalm et al. 2008	*	*	*	*	*	*	*	*	6

A study can be awarded a maximum of one star for each numbered item within the Selection and Exposure categories. A maximum of two stars can be given for Comparability.

Table 2: Baseline Characteristics of Included Studies

Author/ Year	Country	Sample size			Age		Male %		DM %		HTN%		Smoking %		Dyslipidemia %		Alcohol consumption %		BMI		Vendor
		CSF	CG	Total	CSF Mean (SD)	CG Mean (SD)	CSF	CG	CSF	CG	CSF	CG	CSF	CG	CSF	CG	CSF Mean (SD)	CG Mean (SD)			
Shereef et al. 2024	Egypt	60	60	120	44.6 (4.9)	43.5 (5.0)	73 %	45 %	37 %	30 %	57 %	32 %	73 %	30 %	52 %	35 %	NR	NR	27.3 (2.6)	25.7	GE Vivid E95 Philips
Mohammadzad et al. 2021	Iran	53	71	124	52.2 (12.6)	51.8 (10.4)	68 %	39 %	NR	NR	59 %	25 %	62 %	18 %	NR	NR	NR	NR	28.1 (2.3)	24.6 (1.7)	NR
Liu et al. 2021	China	73	60	133	56.4 (9.1)	55.6 (8.2)	62 %	37 %	6 %	7 %	38 %	43 %	NR	NR	NR	NR	NR	NR	NR	NR	Philips TomTec GE Vivid E9
Wang et al. 2020	China	28	34	62	58.1 (6.6)	56.2 (6.8)	64 %	47 %	14 %	9 %	18 %	32 %	39 %	24 %	NR	NR	NR	NR	24.6 (3.1)	25.3 (3.7)	Innova 3100 EchoPAC
Jovanovic et al. 2020	Serbia	32	34	66	60.0 (6.0)	59.0 (7.0)	NR	NR	19 %	9 %	91 %	76 %	44 %	41 %	84 %	62 %	NR	NR	NR	NR	GE Vivid E9 EchoPAC
Li et al. 2020	China	60	45	105	56.5 (8.8)	55.5 (8.4)	60 %	47 %	8 %	4 %	43 %	44 %	NR	NR	NR	NR	NR	NR	NR	NR	Philips QLAB
Abdelsamei et al. 2020	Egypt	31	52	83	53.1 (8.9)	53.2 (7.4)	71 %	50 %	39 %	19 %	52 %	64 %	58 %	25 %	NR	NR	NR	NR	31.6 (6.3)	30.0 (3.1)	Vivid E9 GE Innova
Wang et al. 2019	China	30	24	54	56.3 (7.6)	54.0 (8.1)	67 %	50 %	7 %	13 %	33 %	46 %	37 %	21 %	NR	NR	NR	NR	24.1 (3.2)	24.7 (3.5)	Innova 3100 EchoPAC
Mohammadzad et al. 2019	Iran	45	45	90	52.6 (9.7)	53.2 (8.3)	62 %	33 %	13 %	8 %	20 %	34 %	43 %	18 %	13 %	24 %	NR	NR	29.2 (4.5)	28.4 (4.5)	GE Vivid 6
Shi et al. 2019	China	70	50	120	56.9 (7.8)	52.6 (9.3)	57 %	54 %	6 %	4 %	31 %	36 %	52 %	46 %	NR	NR	NR	NR	24.1 (2.5)	23.9 (3.1)	GE Vivid E9 EchoPAC
Xing et al. 2018	China	60	51	111	56.8 (8.0)	52.6 (9.3)	72 %	53 %	NR	NR	55 %	45 %	38 %	35 %	NR	NR	NR	NR	25.3 (2.1)	24.0 (3.1)	GE Vivid E9 EchoPAC
Kemaloglu et al. 2016	Turkey	40	40	80	53.4 (11.7)	54.1 (10.8)	65 %	55 %	33 %	28 %	13 %	10 %	53 %	48 %	NR	NR	NR	NR	28.7 (3.1)	27.2 (4.0)	Philips TomTec
Wang et al. 2016	China	54	44	98	54.6 (8.3)	55.5 (8.4)	63 %	50 %	6 %	2 %	31 %	36 %	NR	NR	NR	NR	NR	NR	NR	NR	GE Vivid 7 EchoPAC
Barutcu et al. 2015	Turkey	20	20	40	47.0 (8.0)	44.0 (10.0)	75 %	80 %	20 %	15 %	25 %	20 %	30 %	30 %	NR	NR	NR	NR	25.1 (0.7)	25.2 (1.3)	GE Vivid 7
Gulel et al. 2015	Turkey	20	20	40	59.2 (9.6)	54.1 (9.0)	40 %	55 %	25 %	15 %	65 %	55 %	10 %	15 %	35 %	45 %	NR	NR	30.2 (3.2)	31.0 (6.6)	Innova 2100 EchoPAC
Narimani et al. 2015	Iran	36	36	72	53.9 (8.3)	54.5 (9.4)	69 %	69 %	19 %	19 %	42 %	42 %	42 %	33 %	44 %	42 %	NR	NR	30.6 (4.7)	28.5 (4.0)	GE
Wang et al. 2015	China	64	44	108	56.7 (8.6)	55.5 (8.2)	58 %	43 %	6 %	2 %	44 %	41 %	NR	NR	NR	NR	NR	NR	NR	NR	GE Vivid 7 EchoPAC
Nurkalm et al. 2008	Turkey	35	21	56	48.0 (7.0)	50.0 (7.0)	71 %	76 %	17 %	15 %	43 %	40 %	44 %	43 %	NR	NR	NR	NR	28.3 (4.1)	28.0 (3.3)	GE Vivid 7

BM: Body Mass Index; CG: Control Group; CMR: Cardiac Magnetic Resonance; CS: Cross-Sectional Study; CSF: Case Subject Group; DM: Diabetes Mellitus; EchoPAC: Echocardiography Analysis Software; GE: General Electric; HTN: Hypertension; NR: Not Reported; PC: Prospective Cohort Study; QLAB: Philips QLAB Ultrasound Software; SD: Standard Deviation; TomTec: TomTec Imaging Systems.

Table 3: Association Between Left Ventricle Global Longitudinal Strain (LVGLS) and Coronary Slow Flow (CSF)

Author/Year	Imaging	Layer	Condition (Rest/Stress)	LVGLS			Multivariate Logistic Regression of LVGLS		ROC Analysis of LVGLS			LVGLS vs. TFC Correlation	
				CSF Mean (SD)	CG Mean (SD)	P-value	OR (95% CI)	p-value	cutoff	AUC (95% CI)	p-value	R	P-value
Shereef et al. 2024	2D	Global	At rest	- 16.2 (1.3)	- 19.3 (1.3)	0.001	2.2 (1.57 to 3.09)	0.001	17.8%	0.96 (0.92 to 0.99)	0.001	0.49	< 0.001
Mohammadzad et al. 2021	2D	Global	At rest	- 15.9 (0.9)	- 18.6 (0.6)	0.010	NR	NR	NR	NR	NR	NR	NR
Liu et al. 2021	2D	Global	At rest	- 18.2 (2.8)	- 19.7 (2.2)	0.001	1.3 (1.1 to 1.5)	0.004	33.4%	0.66 (0.57 to 0.76)	0.002	0.19	0.040
Wang et al. 2020	2D	Mid	At rest	- 14.9 (2.9)	- 17.0 (2.6)	0.004	1.4 (1.0 to 2.0)	0.04	NR	NR	NR	NR	NR
Jovanovic et al. 2020	2D	Global	At rest	- 18.0 (1.7)	- 23.2 (1.2)	0.001	NR	NR	NR	NR	NR	NR	NR
	2D	Global	exercise stress	- 20.9 (1.8)	- 28.4 (1.3)	0.001	NR	NR	NR	NR	NR	NR	NR
Li et al. 2020	NR	Global	NR	- 19.2 (2.3)	- 20.6 (2.4)	0.004	1.2 (0.9 to 1.6)	0.006	NR	NR	NR	NR	NR
Abdelsamei et al. 2020	2D	Global	At rest	- 15.0 (2.7)	- 17.2 (2.5)	0.001	1.5 (NR)	0.03	15.85%	0.72 (0.61 to 0.83)	> 0.05	0.33	0.002
Wang et al. 2019	2D	Global	At rest	- 19.8 (2.4)	- 21.3 (2.4)	0.050	NR	NR	NR	NR	NR	NR	NR
	2D	Global	exercise stress	- 23.9 (2.8)	- 23.2 (2.5)	< 0.001	NR	NR	NR	NR	NR	NR	NR
Mohammadzad et al. 2019	2D	Global	At rest	- 16.7 (2.4)	- 18.9 (1.6)	0.001	NR	NR	NR	NR	NR	NR	NR
	2D	Endo	At rest	- 23.0 (3.1)	- 24.6 (3.3)	NR	NR	NR	NR	NR	NR	0.31	0.010
Shi et al. 2019	2D	Mid	At rest	- 20.0 (2.8)	- 21.6 (2.9)	< 0.010	NR	NR	NR	NR	NR	0.29	0.043
	2D	Epi	At rest	- 18.9 (2.8)	- 19.1 (2.5)	< 0.05	NR	NR	NR	NR	NR	0.45	0.032
	2D	Global	At rest	- 19.0 (2.1)	- 21.4 (1.6)	0.001	NR	NR	22.5%	0.87 (0.63 to 1)	0.001	0.46	< 0.05
Xing et al. 2018	2D	Endo	At rest	- 21.0 (3.0)	- 24.7 (2.1)	< 0.001	NR	NR	NR	NR	NR	NR	NR
	2D	Mid	At rest	- 18.6 (3.0)	- 21.2 (2.3)	< 0.001	NR	NR	NR	NR	NR	NR	NR
	2D	Epi	At rest	- 17.6 (2.4)	- 18.4 (1.7)	0.074	NR	NR	NR	NR	NR	NR	NR
Kemaloglu et al. 2016	3D	Global	At rest	- 15.9 (3.1)	- 21.7 (3.1)	0.001	NR	NR	NR	NR	NR	0.69	0.001
	2D	Endo	At rest	- 19.6 (2.3)	- 20.8 (2.5)	0.009	NR	NR	NR	NR	NR	NR	NR
Wang et al. 2016	2D	Mid	At rest	- 17.6 (2.0)	- 18.7 (2.2)	0.010	NR	NR	NR	NR	NR	NR	NR
	2D	Epi	At rest	- 15.9 (1.8)	- 16.9 (2.0)	0.010	NR	NR	NR	NR	NR	NR	NR
Barutcu et al. 2015	2D	Global	At rest	- 18.2 (2.0)	- 22.1 (2.5)	0.001	NR	0.001	NR	NR	NR	NR	NR
Gulel et al. 2015	2D	Global	At rest	- 21.1 (2.6)	- 20.5 (3.5)	0.580	NR	NR	NR	NR	NR	NR	NR
Narimani et al. 2015	2D	Global	At rest	- 14.6 (2.3)	- 15.0 (2.6)	0.510	NR	NR	NR	NR	NR	NR	NR
Wang et al. 2015	2D	Global	At rest	- 19.3 (2.3)	- 20.6 (2.5)	0.004	NR	NR	NR	NR	NR	NR	NR
Nurkalm et al. 2008	2D	Mid	At rest	- 20.7 (7.7)	- 23.7 (8.8)	0.140	NR	NR	NR	NR	NR	0.80*	0.0001

2D: Two-Dimensional Imaging; 3D: Three-Dimensional Imaging; AUC: Area Under the Curve; CG: Control Group; CI: Confidence Interval; CMR: Cardiac Magnetic Resonance; CSF: Coronary slow flow Group; Endo: Endocardial Layer; Epi: Epicardial Layer; LVGLS: left ventricle Global Longitudinal Strain; LVEF: Left Ventricular Ejection Fraction; Mid: Mid-myocardial Layer; NR: Not Reported; OR: Odds Ratio; P-value: Probability value; ROC: Receiver Operating Characteristic; SD: Standard Deviation.* Global peak Longitudinal Strain Rate (GLSR) vs. TFC Correlation

Supplementary Table 1: Thrombolysis in Myocardial Infarction Frame Count (TFC) and Left Ventricular Ejection Fraction (LVEF) in Coronary Slow Flow (CSF) and Control Groups

Author/Year	Condition (Rest/Stress)	Mean TFC			LVEF %		
		CSF, Mean (SD)	CG, Mean (SD)	P-value	CSF, Mean (SD)	CG, Mean (SD)	P-value
Shereef et al. 2024	At rest	39.9 (3.9)	19.3 (1.4)	0.001	61.1 (5.3)	61.5 (4.9)	0.668
Mohammadzad et al. 2021	At rest	NR	NR	NR	54.5 (1.5)	54.8 (1.0)	0.020
Liu et al. 2021	At rest	35.6 (13.5)	19.9 (3.7)	0.001	63.0 (3.8)	63.7 (3.7)	0.240
Wang et al. 2020	At rest	40.3 (4.9)	22.7 (3.3)	0.001	64.0 (4.1)	64.9 (4.2)	0.430
Jovanovic et al. 2020	At rest	NR	NR	NR	66.0 (5.7)	68.5 (4.2)	NR
	exercise stress	NR	NR	NR	69.2 (3.9)	71.7 (2.9)	NR
Li et al. 2020	NR	38.3 (13.6)	21.5 (2.3)	0.001	63.3 (4.0)	64.3 (4.4)	0.140
Abdelsamei et al. 2020	At rest	36.4 (3.7)	19.7 (0.9)	0.001	57.8 (5.7)	59.3 (3.3)	0.180
Wang et al. 2019	At rest	39.8 (12.4)	21.8 (2.5)	< 0.001	63.0 (4.0)	63.7 (2.4)	0.110
	exercise stress	NR	NR	NR	68.5 (3.3)	68.0 (2.9)	0.370
Mohammadzad et al. 2019	At rest	NR	NR	NR	NR	NR	NR
Shi et al. 2019	At rest	43.3 (9.6)	15.1 (2.7)	0.001	62.1 (4.3)	63.2 (2.8)	NR
Xing et al. 2018	At rest	39.8 (7.3)	19.9 (1.3)	0.001	62.9 (3.2)	64.3 (3.8)	0.109
Kemaloglu et al. 2016	At rest	40.6 (7.5)	22.4 (1.3)	0.001	60.3 (5.2)	62.0 (4.1)	0.052
Wang et al. 2016	At rest	38.6 (14.0)	21.4 (2.3)	0.001	63.0 (4.0)	64.5 (4.4)	0.100
Barutcu et al. 2015	At rest	22.5 (5.8)	NR	NR	65.3 (4.5)	64.4 (3.0)	0.467
Gulel et al. 2015	At rest	39.1 (10.9)	20.6 (3.0)	0.001	62.5 (3.5)	64.5 (5.4)	0.180
Narimani et al. 2015	At rest	40.0 (10.7)	15.2 (3.5)	0.001	60.7 (3.9)	62.1 (4.3)	NR
Wang et al. 2015	At rest	38.6 (13.5)	21.5 (2.3)	0.001	62.9 (3.8)	64.3 (4.4)	0.100
Nurkalm et al. 2008	At rest	45.2 (14.3)	20.8 (2.4)	0.001	60.9 (3.8)	62.0 (3.4)	0.890

2D: Two-Dimensional Imaging; 3D: Three-Dimensional Imaging; CG: Control Group; CMR: Cardiac Magnetic Resonance; CSF: Coronary slow flow Group; Endo: Endocardial Layer; Epi: Epicardial Layer; GLS: Global Longitudinal Strain; GLSR: Global peak Longitudinal Strain Rate; Mid: Mid-myocardial Layer; NR: Not Reported; R: Correlation Coefficient; SD: Standard Deviation; TFC: Thrombolysis in myocardial infarction frame count