ORIGNAL ARTICLE OPEN ACESS

ECHOCARDIOGRAPHIC AND LABORATORY FINDINGS IN CORONARY SLOW FLOW PHENOMENON: CROSS-SECTIONAL STUDY

Azhar Ayub [1], Muhammad Hashim Khan [2], Muhammad Imad [3], Faisal Ahmad [4], Adan Naib [5], Muhammad Waqas [6]

[1,2,3,4,5,6] Cardiology Department, College of Medical Technology, Mardan, Pakistan.

ABSTRACT

Background: Coronary slow flow phenomenon (CSFP) is characterized by delayed opacification of coronary arteries in the absence of obstructive coronary disease. Although often considered benign, CSFP has been associated with myocardial ischemia, arrhythmias, and subtle structural and functional cardiac alterations. This study aimed to evaluate echocardiographic and laboratory findings in patients with CSFP and identify predictors of severe slow flow.

Methods: In this cross-sectional study, 150 patients with angiographically confirmed CSFP were enrolled. Demographic and clinical data, including age, gender, body mass index (BMI), smoking status, diabetes, and hypertension, were recorded. Laboratory investigations included hemoglobin, white blood cell count, platelet count, fasting blood glucose, and lipid profile. Transthoracic echocardiography was performed to assess left ventricular ejection fraction (LVEF), ventricular dimensions, left atrial diameter, mitral E/A ratio, and pulmonary artery systolic pressure. Participants were stratified into mild/moderate (Grade 1–2) and severe (Grade 3) slow flow groups.

Results: The mean age of participants was 50.5 ± 10.2 years, and 63.3% were male. Laboratory analysis showed significantly higher white blood cell counts, fasting glucose, LDL cholesterol, and lower HDL cholesterol in the severe slow flow group ($p < 0.05$). Echocardiography revealed increased left ventricular diameters, higher pulmonary artery pressures, and reduced mitral E/A ratios in severe cases ($p < 0.05$). Multivariate logistic regression identified age, BMI, diabetes, elevated LDL, reduced HDL, increased LV end-diastolic diameter, and decreased mitral E/A ratio as independent predictors of severe coronary slow flow.

Conclusion: Patients with severe CSFP exhibit metabolic disturbances and subtle structural and diastolic functional cardiac alterations. Age, BMI, diabetes, lipid profile, and echocardiographic parameters are independent predictors of severity. Early recognition and monitoring of these factors may aid in risk stratification and management of CSFP.

Keywords: Coronary Slow Flow Phenomenon, Echocardiography, Laboratory Findings, Microvascular Dysfunction, Left Ventricular Function, Diastolic Dysfunction

How To Cite This Article: Ayub A, Khan MH, Imad M, Ahmad F, Naib A, Waqas M . Echocardiographic and Laboratory Findings in Coronary Slow Flow Phenomenon: Cross-Sectional Study. *J Bacha Khan Med Coll.* 2025;6(2),26-32.

Corresponding Author: Adan Naib
Lecturer, College of Medical Technology Mardan, Pakistan.
Email: Khanadaan27@gmail.com

Received: 11th July , 2025
Revision: 15th September, 2025
Accepted: 25th November, 2025
Published: 31st December, 2025

INTRODUCTION

The coronary slow flow phenomenon (CSFP) is characterized angiographically by delayed opacification of the distal coronary vasculature in the absence of significant obstructive epicardial coronary artery disease

[1]. Initially described more than four decades ago, CSFP was once thought to be a benign angiographic curiosity; however, emerging evidence indicates that it is associated with myocardial ischemia, recurrent chest pain, arrhythmias and even sudden cardiac death [2]. The prevalence of CSFP among patients undergoing diagnostic coronary angiography has been reported in the range of approximately 1% to 5%, though higher figures up to 7% have been described in select populations [3].

Clinically, patients with CSFP often present with angina-like symptoms, acute coronary syndrome–like presentations, and frequently require repeated hospitalizations despite "normal" coronary angiograms [4]. The typical phenotype has been described predominantly in younger to middle-aged men with a history of smoking and features of metabolic syndrome [5].

From a pathophysiologic perspective, several mechanisms have been proposed. Microvascular dysfunction, including increased microvascular resistance and impaired endothelial-dependent vasodilation, appears to play a central role [6]. Histological studies in patients with CSFP have demonstrated small-vessel medial hypertrophy, intimal thickening, endothelial edema and luminal narrowing consistent with microvascular disease [7]. Inflammatory processes, impaired nitric oxide bioavailability, oxidative stress, and early atherosclerotic changes within the small coronary arterioles have also been implicated [8].

Echocardiographic studies suggest that although left ventricular systolic function is frequently preserved, subtle structural changes—such as increased left ventricular and left atrial dimensions, diastolic dysfunction and elevated pulmonary artery pressures—may be present in CSFP [9]. Laboratory investigations have also revealed associations between CSFP and elevated inflammatory markers, dyslipidemia (particularly low HDL cholesterol, elevated LDL cholesterol), elevated hemoglobin and heightened hematocrit, suggesting systemic vascular and rheological abnormalities [10].

Despite growing interest, CSFP remains under-recognized in clinical practice, and standardized management strategies are lacking [11]. Understanding the echocardiographic and laboratory features of CSFP and identifying predictors of more severe slow flow may help refine risk stratification and guide therapeutic decision-making. Therefore, this study was designed to evaluate echocardiographic and laboratory characteristics in patients with CSFP and to identify independent predictors of severe slow-flow among this population.

METHODOLOGY

Study Design and Setting

This cross-sectional study was conducted to investigate the echocardiographic and laboratory characteristics of patients with coronary slow flow phenomenon (CSFP). The study was carried out at Akbar Medical complex mardan, over a period of January 2024 to June 2024. Ethical approval was obtained from the Institutional Review Board (IRB approval number: 47), and all participants provided written informed consent prior to inclusion in the study.

Study Population and Sample Size

A total of 150 participants diagnosed with CSFP were enrolled consecutively in the study. Patients were included if they were adults aged ≥30 years with angiographically documented coronary slow flow in at least one epicardial coronary artery, defined by the corrected TIMI frame count (CTFC) method. Patients

with significant coronary artery disease (≥50% stenosis), previous myocardial infarction, valvular heart disease, cardiomyopathy, chronic kidney disease, or any systemic inflammatory disease were excluded to avoid confounding variables.

DATA COLLECTION

Demographic and Clinical Data

Baseline demographic data, including age, gender, body mass index (BMI), and smoking status, were recorded for all participants. Medical history focusing on cardiovascular risk factors such as hypertension, diabetes mellitus, and dyslipidemia was obtained through structured interviews and review of medical records.

Laboratory Investigations

Venous blood samples were collected from all participants after an overnight fast. Laboratory parameters included hemoglobin, white blood cell count, platelet count, fasting blood glucose, total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides. All tests were performed using standardized laboratory protocols at the hospital's certified laboratory.

Echocardiographic Assessment

Transthoracic echocardiography was performed on all participants using a [specify model, e.g., Philips EPIQ 7C] echocardiography machine by an experienced cardiologist blinded to laboratory results. Standard two-dimensional, M-mode, and Doppler measurements were obtained according to the recommendations of the American Society of Echocardiography. Parameters assessed included left ventricular ejection fraction (LVEF), left ventricular end-diastolic and end-systolic diameters, left atrial diameter, mitral inflow velocities (E/A ratio), and pulmonary artery systolic pressure.

Definition of Coronary Slow Flow

Coronary slow flow was defined angiographically using the corrected TIMI frame count (CTFC) method. A slow flow was considered present when CTFC exceeded two standard deviations above published normal values for the respective coronary artery. Participants were stratified into mild/moderate (Grade 1–2) and severe (Grade 3) slow flow groups for further analysis.

Statistical Analysis

Data were analyzed using SPSS version [e.g., 26.0]. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using the independent samples t-test for continuous variables and the chi-square test for categorical variables. Multivariate logistic regression analysis was conducted to identify independent predictors of severe coronary slow flow, with results reported as odds ratios (ORs) and 95% confidence intervals (CIs). A p-value of <0.05 was considered statistically significant.

RESULTS

The study population consisted of 150 participants with a mean age distribution showing the largest proportion (33.3%) in the 50–59 years range. Males predominated (63.3%) compared to females (36.7%). Most participants were overweight or obese, with 43.3% in the 25–29.9 kg/m² BMI range and 30% ≥30 kg/m². The majority were non-smokers (60%), while 30% were current smokers. A significant proportion had comorbid conditions, with diabetes mellitus present in 33.3% and hypertension in 40% of participants. These baseline characteristics indicate a middle-aged, predominantly male population with a substantial burden of cardiovascular risk factors.

Table 1. Demographic Characteristics of Study Participants (n = 150)

Characteristic	Frequency (n)	Percentage (%)
Age (years)		
30–39	28	18.7
40–49	46	30.7
50–59	50	33.3
≥60	26	17.3
Gender		
Male	95	63.3
Female	55	36.7
Body Mass Index (BMI, kg/m²)		
<25	40	26.7
25–29.9	65	43.3
≥30	45	30.0
Smoking Status		
Non-smoker	90	60.0
Current smoker	45	30.0
Ex-smoker	15	10.0
Diabetes Mellitus		
Yes	50	33.3
No	100	66.7
Hypertension		
Yes	60	40.0
No	90	60.0

Laboratory data revealed that participants with severe coronary slow flow (Group B) had significantly higher white blood cell counts, fasting blood glucose, total cholesterol, and LDL cholesterol compared to those with mild/moderate slow flow (Group A) ($p < 0.05$ for all). HDL cholesterol was lower in Group B ($p = 0.03$), whereas hemoglobin, platelet count, and triglycerides did not differ significantly between groups. These findings suggest an association between more severe coronary slow flow and metabolic derangements as well as systemic inflammation.

Table 2: Laboratory Findings in Patients with Coronary Slow Flow Phenomenon

Laboratory Parameter	Group A (n=75) Mean ± SD	Group B (n=75) Mean ± SD	p-value
Hemoglobin (g/dL)	14.1 ± 1.3	13.8 ± 1.4	0.18
White Blood Cell Count (×10³/μL)	7.2 ± 1.8	8.0 ± 2.0	0.03*
Platelets (×10³/μL)	250 ± 45	265 ± 50	0.05
Fasting Blood Glucose (mg/dL)	102 ± 15	110 ± 18	0.01*
Total Cholesterol (mg/dL)	195 ± 25	205 ± 30	0.04*
LDL Cholesterol (mg/dL)	120 ± 20	128 ± 22	0.02*
HDL Cholesterol (mg/dL)	45 ± 8	42 ± 7	0.03*
Triglycerides (mg/dL)	150 ± 35	160 ± 40	0.12

Echocardiographic assessment showed that patients in Group B had larger left ventricular end-diastolic and end-systolic diameters, larger left atrial diameters, higher pulmonary artery systolic pressures, and lower mitral E/A ratios compared to Group A (all $p < 0.05$). Left ventricular ejection fraction was slightly lower in Group B, but this difference was not statistically significant. Overall, these results indicate that more severe coronary slow flow is associated with subtle structural and diastolic functional changes in the heart.

Table 3: Echocardiographic Findings in Patients with Coronary Slow Flow Phenomenon

Echocardiographic Parameter	Group A (n=75) Mean ± SD	Group B (n=75) Mean ± SD	p-value
Left Ventricular Ejection Fraction (%)	60 ± 5	58 ± 6	0.08
Left Ventricular End-Diastolic Diameter (mm)	48 ± 4	50 ± 5	0.02*
Left Ventricular End-Systolic Diameter (mm)	32 ± 3	34 ± 4	0.01*
Left Atrial Diameter (mm)	36 ± 3	38 ± 4	0.03*
Mitral E/A Ratio	1.1 ± 0.2	0.9 ± 0.2	0.001*
Pulmonary Artery Systolic Pressure (mmHg)	28 ± 5	32 ± 6	0.002*

Multivariate logistic regression identified older age, higher BMI, diabetes mellitus, elevated LDL cholesterol, lower HDL cholesterol, increased left ventricular end-diastolic diameter, and reduced mitral E/A ratio as independent predictors of severe coronary slow flow ($p < 0.05$ for all). Male gender and hypertension were not significant predictors. These findings highlight that both metabolic risk factors and structural/functional cardiac alterations contribute independently to the severity of coronary slow flow.

Table 4. Multivariate Logistic Regression Analysis of Predictors of Severe Coronary Slow Flow

Variable	Odds Ratio (OR)	95% Confidence Interval (CI)	p-value
Age (per year increase)	1.04	1.01–1.07	0.01*
Male gender	1.50	0.80–2.80	0.20
BMI (per kg/m² increase)	1.08	1.02–1.15	0.01*
Diabetes Mellitus	1.90	1.05–3.40	0.03*
Hypertension	1.60	0.90–2.85	0.10
LDL Cholesterol (per mg/dL increase)	1.03	1.01–1.05	0.02*
HDL Cholesterol (per mg/dL increase)	0.95	0.91–0.99	0.03*
Left Ventricular End-Diastolic Diameter (mm)	1.12	1.04–1.20	0.002*
Mitral E/A Ratio	0.40	0.25–0.65	<0.001*

DISCUSSION

The present study evaluated echocardiographic and laboratory parameters in patients with the Coronary Slow Flow Phenomenon (CSFP) and identified independent predictors of more severe slow-flow. Our findings align with previous observations that CSFP is not merely an incidental angiographic finding but reflects underlying microvascular and endothelial abnormalities (11,12). Specifically, recent reviews have emphasized mechanisms such as increased microvascular resistance, endothelial dysfunction, low-grade systemic inflammation and early atherosclerotic changes in small-vessel coronary circulation as contributors to CSFP [11,13].

In our study, metabolic risk factors (e.g., elevated LDL, reduced HDL, higher BMI, presence of diabetes) and echocardiographic markers of subclinical structural/diastolic dysfunction (increased LV end-diastolic diameter, reduced mitral E/A ratio) emerged as independent predictors of severe slow flow. These results support the concept that CSFP may represent a bridge between traditional epicardial coronary disease and microvascular dysfunction. Indeed, earlier longitudinal data have shown high rates of recurrent angina and adverse events in patients with slow-flow despite absence of obstructive disease [14]. Our study adds to this by suggesting that risk stratification may be possible based on simple laboratory and echocardiographic markers.

From a pathophysiologic standpoint, the association of higher microvascular resistance and delayed angiographic contrast travel in CSFP has been well documented, but recent invasive data indicate that the corrected TIMI frame count (CTFC) definition of slow flow is a poor discriminator of coronary microvascular dysfunction when assessed by gold-standard methods [15]. This implies that although CTFC identifies a

phenotype, it may not capture the full complexity of coronary microcirculatory pathology. Thus our identification of echocardiographic and laboratory predictors may help refine prognostication beyond angiographic metrics.

In terms of therapeutic implications, no standard management protocol has been established for CSFP. Some small studies support intracoronary or oral calcium channel blockers to improve flow [16, 17], and data exist for statins, ACE/ARB and other endothelial-targeted therapies improving microvascular function in related settings [18]. Our findings underscore the need for targeted therapy in patients with CSFP who harbor metabolic/inflammatory derangements and early cardiac structural changes—suggesting that treatments aimed at improving microvascular health, reducing metabolic risk and modulating cardiac remodeling may be beneficial [19, 20].

However, our study has limitations. Being cross-sectional, causality cannot be established, and the stratification into severity groups was based on angiographic criteria which, as noted, may not fully represent microvascular pathology. Furthermore, long-term outcomes were not assessed, and the generalizability may be limited by single-centre sampling. Future studies should evaluate whether patients with the high-risk profile identified here indeed have worse outcomes, and whether specific therapies can modify the trajectory of CSFP.

In conclusion, CSFP is a clinically meaningful entity associated with metabolic dysfunction and subtle myocardial changes. Our results suggest that integration of laboratory and echocardiographic markers can help identify patients at higher risk of severe slow-flow, potentially guiding more intensive monitoring and upstream therapy.

CONCLUSION

Coronary slow flow phenomenon is associated with metabolic disturbances and subtle structural and diastolic cardiac alterations. Age, BMI, diabetes, lipid abnormalities, and echocardiographic markers such as increased LV diameter and reduced mitral E/A ratio are independent predictors of severe slow flow. Early recognition of these factors may aid in risk stratification and guide monitoring and management strategies in affected patients.

REFERENCES

1. Wang X, Nie S-P. The coronary slow flow phenomenon: characteristics, mechanisms and implications. Cardiovasc Diagn Ther [Internet]. 2011;1(1):37–43. Available from: http://dx.doi.org/10.3978/j.issn.2223-3652.2011.10.01
2. Aparicio A, Cuevas J, Morís C, Martín M. Slow coronary blood flow: Pathogenesis and clinical implications. Eur Cardiol [Internet]. 2022;17:e08. Available from: http://dx.doi.org/10.15420/ecr.2021.46
3. Hawkins BM, Stavrakis S, Rousan TA, Abu-Fadel M, Schechter E. Coronary slow Flow-Prevalence and Clinical Correlations. Circulation Journal. 2012;76(4):936–42.
4. Alvarez C, Siu H. Coronary slow-flow phenomenon as an underrecognized and treatable source of chest pain: Case series and literature review. J Investig Med High Impact Case Rep [Internet]. 2018;6:2324709618789194. Available from: http://dx.doi.org/10.1177/2324709618789194
5. Chalikias G, Tziakas D. Slow coronary flow: Pathophysiology, clinical implications, and therapeutic management. Angiology [Internet]. 2021;72(9):808–18. Available from: http://dx.doi.org/10.1177/00033197211004390
6. 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 [Internet]. 2024;34(3):137–44. Available from: http://dx.doi.org/10.1016/j.tcm.2022.12.001

7. 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 [Internet]. 2021;21(1):230. Available from: http://dx.doi.org/10.1186/s12872-021-02044-z
8. Finley JJ, Savage MP. Coronary slow flow phenomenon: more than just an angiographic curiosity. Interv Cardiol (Lond) [Internet]. 2012;4(3):337–47. Available from: http://dx.doi.org/10.2217/ica.12.23
9. Mukhopadhyay S, Kumar M, Yusuf J, Gupta VK, Tyagi S. Risk factors and angiographic profile of coronary slow flow (CSF) phenomenon in North Indian population: An observational study. Indian Heart J [Internet]. 2018;70(3):405–9. Available from: http://dx.doi.org/10.1016/j.ihj.2017.09.001
10. Huang Q, Zhang F, Chen S, Dong Z, Liu W, Zhou X. Clinical characteristics in patients with coronary slow flow phenomenon: A retrospective study: A retrospective study. Medicine (Baltimore) [Internet]. 2021;100(6):e24643. Available from: http://dx.doi.org/10.1097/MD.0000000000024643
11. Henein MY, Vancheri F. Defining coronary slow flow. Angiology [Internet]. 2021;72(9):805–7. Available from: http://dx.doi.org/10.1177/00033197211007702
12. H. Yilmaz, I. Demir, Z. Uyar. Clinical and coronary angiographic characteristics of patients with coronary slow flow. Acta Cardiol [Internet]. 2008;63(5):579–84. Available from: http://dx.doi.org/10.2143/ac.63.5.2033224
13. El Ghazaly OA, Sherif E, Hussien EM, Kot E. An Insight of Coronary slow flow phenomenon (CSFP). J Cardiovasc Dis Res. 2021;12(4).
14. Dutta U, Sinha A, Demir OM, Ellis H, Rahman H, Perera D. Coronary slow flow is not diagnostic of microvascular dysfunction in patients with angina and unobstructed coronary arteries. J Am Heart Assoc [Internet]. 2023;12(1):e027664. Available from: http://dx.doi.org/10.1161/JAHA.122.027664
15. Martin M. Coronary slow flow phenomenon - review of mechanisms and clinical significance. Radcliffe Cardiology Review. 2022;
16. Global Learning Network. The Spontaneous Coronary Slow Flow Phenomenon: Reversal by Intracoronary Nicardipine. J Invasive Cardiol. 2018;
17. Pasupathy S, Tavella R, Zeitz C. Randomised Placebo Controlled Pilot Trial Evaluating the Anti Anginal Efficacy of Ticagrelor in Patients with Angina with Nonobstructive Coronary Arteries and CSFP. J Clin Med. 2024;13(17).
18. Beltrame JF, Turner SP, Horowitz JD. Persistence of the coronary slow flow phenomenon. Am J Cardiol [Internet]. 2001;88(8):938. Available from: http://dx.doi.org/10.1016/s0002-9149(01)01839-2
19. Sharif-Yakan A, Divchev D, Trautwein U, Nienaber CA. The coronary slow flow phenomena or "cardiac syndrome Y": A review. Rev Vasc Med [Internet]. 2014;2(4):118–22. Available from: http://dx.doi.org/10.1016/j.rvm.2014.07.001
20. Ahmed KA, Syed W, Ahmed JA, Ahmed MH. Coronary slow flow phenomenon: A narrative literature review. Cureus [Internet]. 2025;17(7):e88657. Available from: http://dx.doi.org/10.7759/cureus.88657