Case report

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Recurrent ST-Segment Elevation Myocardial Infarction with Coexisting Coronary Slow Flow Phenomenon and Right Coronary Artery Occlusion: A Case Report

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Abstract: Background: The Coronary Slow Flow Phenomenon (CSFP) defined as a distinct clinical condition marked by delayed opacification of the distal vasculature during coronary angiography, occurring even in the absence of obstructive lesions or significant stenosis in the epicardial coronary arteries. Its acute coronary syndrome-like presentation can complicate causal interpretation when obstructive coronary disease is also present. **Case Presentation:** A 49-year-old male with two previous ST-segment elevation myocardial infarctions presented with acute chest pain, ST-segment elevation in leads V1-V4, and troponin concenctration of 0.966 ng/dL. Coronary angiography demonstrated a dominant RCA with total proximal occlusion. The LM artery was patent; the LAD and LCx arteries had no significant focal stenosis but were markedly tortuous and showed delayed contrast transit. A proximal LAD artery aneurysm was also observed. **Discussion:** The findings support coexistence of obstructive coronary disease and an angiographic slow-flow pattern. Because corrected TIMI frame counts and adjunctive coronary imaging were unavailable, he ST-segment elevation cannot be attributed to coronary slow flow alone**. Conclusion:** When slow flow and an occlusive lesion coexist, complete angiographic assessment and cautious causal attribution are essential. Recognition of slow flow should complement, not delay or replace, guideline-directed evaluation and treatment of acute coronary occlusion.

Keywords: coronary slow flow phenomenon, acute coronary syndrome, ST-segment elevation myocardial infarction, coronary artery occlusion

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Introduction

Initially documented by Tambe et al. in 1972, the Coronary Slow Flow Phenomenon (CSFP) is a unique clinical condition characterized by delayed opacification of the distal vasculature during coronary angiography, occurring even in the absence of obstructive lesions or significant stenosis in the epicardial coronary arteries. It can be associated with recurrent angina, acute coronary syndrome-like presentations, ventricular arrhythmias, and adverse cardiovascular outcomes. (Tambe et al., 1972; Aparicio, et al., 2022; Zhu et al., 2024; Martin, et al., 2025). Quantitative assessment with the corrected Thrombolysis in Myocardial Infarction frame count (cTFC) is preferred to visual estimation alone, moreover, angiographic slow flow should not automatically be equated with invasive evidence of coronary

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microvascular dysfunction (Dutta, et al. 2023). In this case report, we describe a patient with recurrent ST-segment elevation myocardial infarction (STEMI), total proximal right coronary artery (RCA) occlusion, delayed flow in coronary tortuosity, and a proximal left anterior descending artery (LAD) aneurysm. This case illustrates the diagnostic challenge of assigning causality when obstructive and non-obstructive angiographic abnormalities coexist.

Case Presentation

A 49-year-old male was admitted to our hospital with chest pain 4 hours prior to admission. The pain was described like being stabbed on his left chest and lasted up to 2 hours. The pain not radiated to left arm nor jaw but accompanied with epigastric pain. He also had risk factors for smoking, hypertension and dyslipidemia. His initial blood pressure in emergency room was 134/101 mmHg and heart rate was 83 beats per minute. From physical examination, no cardiac murmur was found nor jugular vein engorgement, breathing sound was normal and no oedema on lower extremities. Previously, he had history of inferior STEMI 2 years ago. The coronary angiography showed normal left main, slow flow coronary in LAD, LCx, and RCA with dominant RCA. A year later, he had inferoposterior STEMI. He underwent primary percutaneous intervention (PCI) which showed acute total occlusion in mid RCA. Thrombus aspiration was performed with thrombuster but the thrombus shifted to distal RCA.

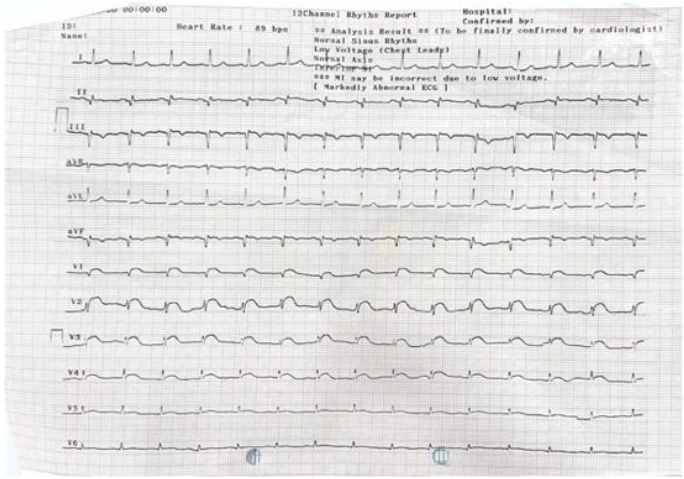

Figure 1. Admission ECG showing ST-elevation in lead V1-V4

When admitted to our hospital, the ECG showed ST-elevation on lead V1-V4 with pathological q waves on lead II, III, aVF (Figure 1). The troponin level was mildly elevated, 0.966 ng/dL. This patient immediately went under PCI. The coronary angiography showed RCA was dominant with total occlusion in proximal RCA. The left main was patent. The LAD showed slow flow coronary with very tortuous vessel and aneurysm in proximal LAD (TIMI flow 2-3). The LCx also showed slow flow coronary with very tortuous vessel (TIMI flow 2-3). Medical therapy included aspirin, clopidogrel, and nitroglycerin.

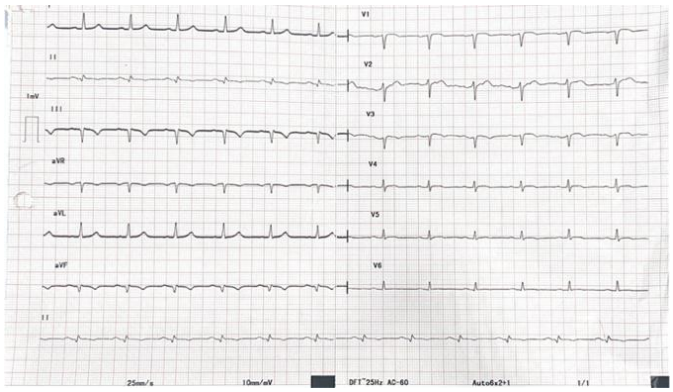

Figure 2. Post PCI ECG

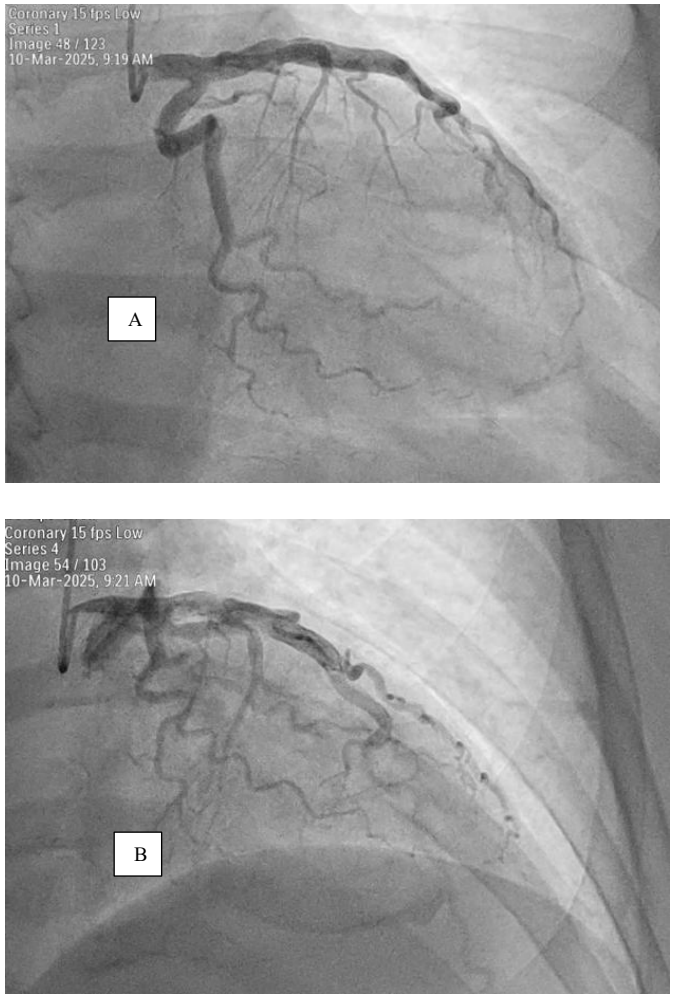

Figure 3. Coronary angiogram showing patent left-sided coronary arteries with no obstructive stenosis but demonstrating delayed contrast runoff (coronary slow flow) in the LAD. Views shown: (A) RAO caudal, and (B) LAO cranial.

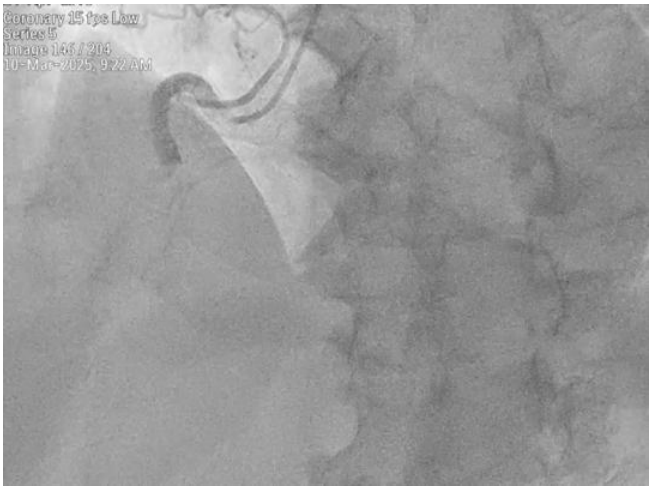

Figure 4. Coronary angiography showing total occlusion at proximal RCA

Discussion

This case should not be interpreted as evidence that CSFP alone caused the index STEMI. Total proximal RCA occlusion provides an obstructive ischemic mechanism and required urgent invasive evaluation. At the same time, the anterior distribution of ST-segment elevation, delayed filling in the non-obstructed LAD and LCx, marked tortuosity, and a proximal LAD aneurysm indicate a more complex coronary phenotype. The relative contributions of the RCA occlusion and left-sided slow flow cannot be separated with certainty from the available data.

CSFP is conventionally defined in a vessel without a significant epicardial stenosis, commonly using a cTFC greater than 27 frames (Gibson et al., 1996; Aparicio et al., 2022). Visual TIMI grading is semiquantitative, and recent invasive physiology studies have shown that angiographic slow flow has limited diagnostic accuracy for coronary microvascular dysfunction or endothelial dysfunction (Sen et al., 2013; Beltrame et al, 2019; Dutta et al., 2023). Accordingly, the present report uses the more cautious term angiographic slow-flow pattern for the LAD and LCx because cTFC was unavailable. The differential diagnosis of ST-segment elevation includes acute atherothrombotic occlusion, coronary embolism, epicardial vasospasm, spontaneous coronary artery dissection, myocarditis, pericarditis, takotsubo syndrome, and non-ischemic repolarization variants. In this patient, the angiographic RCA occlusion and troponin elevation support an acute coronary syndrome, while the discordance between ECG territory and angiographic findings warrants cautious interpretation rather than exclusive attribution to slow flow (Byrne et al., 2023, Mareai et al., 2024).

Coronary tortuosity may alter local shear stress and contrast transit, while an aneurysmal segment can promote disturbed flow and thrombus formation. These anatomical findings may therefore contribute to recurrent ischemic presentations, but they do not establish causality. Their coexistence with atherosclerotic occlusion reinforces the need to describe each angiographic abnormality separately and to individualize surveillance and antithrombotic decisions. Contemporary reviews describe CSFP as a heterogeneous, multifactorial condition involving vascular tone, endothelial and inflammatory pathways, subclinical atherosclerosis, metabolic factors, and coronary anatomy (Zhu et al., 2024; Martin et al., 2025).

Management during an ST-elevation presentation must prioritize rapid angiography, identification of an occlusive culprit, reperfusion when technically appropriate, antiplatelet therapy, intensive lipid lowering, and risk-factor control (Saya, et al., 2019; Byrne et al., 2023). No universally accepted disease-specific treatment for CSFP has been established; calcium-channel blockers and nitrates may improve symptoms or angiographic flow in selected patients, but supporting evidence remains limited (Simsek et al., 2011; Aparicio et al., 2022; Li et al., 2023; Martin et al., 2025).

Conclusion

This case demonstrates recurrent STEMI in a patient with coexisting total proximal RCA occlusion and an angiographic slow-flow pattern in non-obstructed left coronary arteries, together with coronary tortuosity and a proximal LAD aneurysm. The available findings do not support attributing the ST-segment elevation to CSFP alone. Complete angiographic evaluation, quantitative flow assessment when feasible, and adjunctive testing when the ECG and angiographic territories are discordant are essential for accurate diagnosis. Recognition of slow flow should complement, not delay or replace, guideline-directed evaluation and treatment of obstructive acute coronary disease.

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