



CLINICAL SIGNIFICANCE OF SLOW FLOW PHENOMENON OF CORONARIES DURING ANGIOGRAM IN PATIENTS WITH MYOCARDIAL INFARCTION AT A TERTIARY CARE HOSPITAL

Cardiology

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ABSTRACT

Objectives: The goal of this study was to assess the clinical significance of slow flow phenomenon of coronaries during angiogram in patients with acute myocardial infarction at Stanley medical college, Chennai. **Background:** Coronary slow-flow phenomenon (CSFP), also known as cardiac syndrome Y, is characterised angiographically by delayed distal vessel opacification in the absence of obstructive coronary artery disease and represents a pathology related to underlying dysfunction of microvascular resistance. The diagnosis of CSFP is made via coronary angiography based on either a reduced thrombolysis in Myocardial Infarction (TIMI) flow grade of 2 or increased corrected TIMI frame count of greater than 27 frames in one or more epicardial vessel. **Materials And Methods:** This was an observational study based on a single tertiary cardiac center in stanley medical college which includes 40 consecutive patients who were diagnosed with CSFP from August 2022 to April 2023. Angiograms were evaluated and reviewed, and TIMI frame counts were verified. Slow-flow was defined by a frame count greater than 27 for all vessels Furthermore, all subjects had TIMI-2 flow, which is defined by ≥ 3 beats to opacify prespecified branch points in the distal vasculature. **Result:** Majority of the patients were males (75%). Diagnosis of CSFP was established in 40 patients by an initial corrected TIMI frame count >27 frames (mean of 105 frames in vessels affected by CSFP), with improvement to <27 frames after administration of IC nitroglycerin average dose = 200 μ g). Of the 40 patients studied, the mean age was 55.5 years (range = 39-71 years;) all had left ventricular ejection fraction $\geq 45\%$. Hypertension was present in 55% patients and diabetes in 32% of patients.

KEYWORDS

Coronary Angiography, Coronary Artery Disease, Slow Flow Phenomenon

INTRODUCTION:

The coronary slow flow phenomenon (CSFP) is an angiographic clinical entity, characterized by delayed distal vessel opacification in the absence of significant epicardial coronary stenosis. Although it is well-known to interventional cardiologists for approximately four decades, the pathogenic mechanisms are incompletely understood. Rather than representing a simple angiographic curiosity, CSFP has direct clinical implications, as it has been linked to clinical manifestations of myocardial ischemia, life-threatening arrhythmias, sudden cardiac death, and recurrent acute coronary syndromes. However, current clinical practice tends to underestimate the impact of CSFP due to the yet unknown mechanisms, its relative rarity, and the subsequent difficulties in conducting randomized trials to evaluate different treatment options. In this article, we will summarize the characteristics, possible mechanisms, and clinical implications of this entity to provide further insight into its clinical significance and management strategies.

DEFINITION :

Coronary slow-flow phenomenon (CSFP), also known as cardiac syndrome Y, is characterized angiographically by delayed distal vessel opacification in the absence of obstructive coronary artery disease and represents a pathology related to underlying dysfunction of microvascular resistance. The diagnosis of CSFP is made via coronary angiography based on either a reduced thrombolysis in Myocardial Infarction (TIMI) flow grade of 2 or increased corrected TIMI frame count of greater than 27 frames in one or more epicardial vessel.

MATERIALS & METHODS:

Setting:

Critical Care Unit Cardiology Department at a Tertiary Center in Chennai.

Design Of Study:

This was an observational study based on a single tertiary cardiac center in our medical college which includes 40 patients (10 females and 30 males)

Period Of Study:

9 Months from August 2022 to April 2023.

Inclusion Criteria:

All patients with myocardial infarction with angiographic severity

with less than significant critical stenosis of 70% were included in this study. Patients of STEMI & NSTEMI/UA were included.No age limitation

Diagnosis And Evaluation:

Angiograms were evaluated and reviewed, and TIMI frame counts were verified. Slow-flow was defined by a frame count greater than 27 for all vessels Furthermore, all subjects had TIMI-2 flow, which is defined by ≥ 3 beats to opacify pre-specified branch points in the distal vasculature. The left anterior descending coronary artery (LAD) frame counts were divided by 1.7 for correction of the longer length, and all the films should be corrected at 30 frames per second (fps). The CTFC above 27 frames for at least one among three major vessels was defined to CSFP, only if the qualitative results from clinical center and angiographic core laboratory were consistent.

RESULTS:

The mean age was 55.5 years. Majority of the patients were males (75%). (table1)The maximum age at presentation was 71 years and the minimum age was 39.

Table:1 Demographics	
Age(mean years)	55.5
Male%	75
Female%	25

All patients had left ventricular ejection fraction $>45\%$. Hypertension was present in 55% patients and diabetes in 32% of patients. Hyperlipidemia present in 82.5% patients. Mean BMI was 31.1%. (table2)Majority of the patients were unstable angina (58%). Majority of the study patients had slow flow in the LAD territory (80%).(Table3) Majority of the patients prior to angiogram were started on aspirin, atorvastatin and beta blocker by primary care physicians. Of the 6 patients with STEMI 5 patients had AWMi with recanalised LAD and slow flow. 1 patient had IWMI with recanalised RCA with slow flow. Patients who were found with coronary slow flow during angiogram were given intracoronary NTG and IC adenosine in some patients for flow improvement. Patients were started on calcium channel blocker diltiazem and nitrates in some patients for relieving angina. Patients were followed up and reviewed every two weeks and observed for symptoms of chest pain and ECG changes.

Table:2 Comorbidities prior to angiogram	
Hypertension%	55

Diabetes%	32.5
Hyperlipidemia%	82.5
Body mass index (mean)	31.1
Tobacco use%	65
Alcohol%	40
Substance abuse%	2.5
Table:3 Angiogram findings	
LVEF%(average)	45%
ACS on presentation	
CSF in LAD%	80.2
CSF in LCX%	30.3
CSF in RCA%	50
CSF in 2 or more epicardial vessels%	45.5
Table:4 Medications prior to diagnosis	
Calcium channel blocker	0
Betablocker	64
Statin	67.5
Aspirin	70.1
ACE inhibitor	23.1
Long acting nitroglycerin	0

936–942, 2012.

DISCUSSION:

The exact etiology and pathogenesis of CSFP is not definitively established; The possible mechanisms are 1)Microvascular dysfunction; 2)Small vessel disease, cell edema, capillary damage, subclinical atherosclerosis, inflammation, fibromuscular hypertrophy, and degeneration of endothelial cells with resultant microvascular luminal narrowing.Blood flow patterns in epicardial coronary arteries depend on the geometry and motion of these vessels. Disturbed laminar blood flow occurs in arterial segments with geometric irregularities such as curvatures, branches, and bifurcations. It is in these complex regions that low blood velocity rates tend to occur. The results showed that the presence of CSFP was associated with higher tortuosity and more distal branches in coronary arteries.

Box 1. Proposed mechanisms of the coronary slow flow phenomenon.

Structural

- Endothelial edema, fibromuscular hypertrophy and degeneration of endothelial cells with resultant microvascular luminal narrowing
- Early, borderline atherosclerosis in epicardial coronary vessels (intravascular ultrasound detection)
- Abnormal angulations of the main coronary arteries

Physiological/dynamic

- Microvascular vasomotor dysfunction
- Endothelial dysfunction
- Increased resting coronary microvascular resistance
- Preserved coronary flow reserve
- Resistance improves with microvascular vasodilators (papaverine, adenosine)

Molecular/genetic

- Inappropriate high release of neuropeptide Y, endothelin-1, thromboxane A2 (vasoconstrictor autacoids)
- Low nitric oxide plasma concentrations and reduced bioactivity
- Raised levels of plasma homocysteine and asymmetric dimethylarginine (nitric oxide synthase inhibitors)
- Inflammatory/oxidative stress
- Elevated high-sensitivity CRP
- Decreased adiponectin concentrations and paraoxonase activity
- Elevated levels of paraoxonase, α-1-antichymotrypsin (particularly in acute presentation)

CONCLUSION:

CSFP is an important, angiographic finding typically observed in patients presenting with acute coronary syndrome, in particular unstable angina. Previous studies have shown that small vessel disease, endothelial dysfunction, subclinical atherosclerosis, inflammation, and anatomic properties of coronary arteries are related to the occurrence of CSF. Currently, by using CTFC as a quantitative index of coronary flow, coronary angiography is the only tool for the diagnosis and assessment of CSFP. Yet, owing to its invasiveness, this method does not permit long-term clinical follow-up and dynamic treatment evaluation.

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