INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

CLINICAL PROFILE OF CORONARY SLOW FLOW PHENOMENON IN SOUTH INDIAN POPULATION

Cardiology

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ABSTRACT

BACKGROUND: Coronary slow-flow phenomenon (CSFP) is defined as delayed opacification of epicardial coronary arteries in the absence of obstructive coronary artery disease. CSFP is often underrecognized and presentation has wide spectrum.

MATERIALS & METHODS: A total of 75 patients with coronary slow flow phenomenon were compared with 75 control with normal angiogram over a period of 2 years from January 2017 - January 2019 in the Institute of Cardiology, Rajiv Gandhi Government General Hospital, Madras medical college, Chennai.

RESULTS: The most common age group was 41-60(64%), mean age was 49±10.5 years. Most common artery involved is LAD. CSFP had a wide spectrum ranging from CSA (48%), NSTEMI, UA (26.7%) and STEMI (25.3%). Male smokers had significant association with CSFP (p value <0.05).

CONCLUSION: CSFP is an infrequently reported diagnosis with a benign outcome with rare occurrence of sudden cardiac death.

KEYWORDS

CSFP, Coronary slow flow phenomenon, Coronary Angiogram

INTRODUCTION

Coronary slow-flow phenomenon (CSFP) is defined as delayed opacification of epicardial coronary arteries in the absence of obstructive coronary artery disease. Tambe et al first reported CSF phenomenon in 1972[1]. CSF phenomenon prevalence ranges between 1 to 5%[2,3]. Though exact pathophysiology is unclear, microvasculature abnormalities, endothelial dysfunction and elevated inflammatory markers are implicated. Patients with CSFP usually present with chest pain and ECG changes (abnormal QT dispersion and ventricular arrhythmias[4]) CSFP is common in young male smokers.

MATERIAL AND METHODS

Patients who underwent coronary angiogram between January 2017 and January 2019 in our institute were evaluated for CSFP. All angiograms were performed using Standard Judkin's left and right 6 French diagnostic catheters with manual low osmolar contrast (Iohexol) injection. Angiograms were evaluated, and TIMI frame counts were verified. Slow-flow was defined by a frame count greater than 27 for all vessels (for the LAD, the frame count was divided by 1.7 to correct for the longer vessel) from Gibson et al[5]. The first frame was considered as that at which a column of dye extended across the entire width of the origin of the artery touching both its borders with evidence of antegrade motion of the dye and the last frame was defined as the frame when the dye 1st entered a certain distal landmark branch in each vessel. For left anterior descending artery (LAD) the distal bifurcation, for left circumflex artery (LCX) the most distal bifurcation of the obtuse marginal branch farthest from the coronary ostium and for the right coronary artery (RCA) the first branch of the posterolateral segment were used as distal landmarks. In our institute cine fluoroscopy was acquired at 15 frames per second, and therefore, the recorded frame count was multiplied by 2. 75 consecutive patients with CSF phenomenon were studied and compared with 75 normal coronary angiogram patients in our study.

Inclusion criteria:
1. Age>18 years
2. CAG – CSFP with normal epicardial coronary arteries.

Exclusion criteria:
1. Coronary angiogram revealing ectasia, Bridging, Spasm and obstructive lesion
2. Refusal to give informed consent.

The data was analyzed by the statistical software SPSS version 20.

Results:
Table 1: Age wise distribution of CSFP in our study

Age	Number (Percentage)
21-40	23(30.6%)
41-60	48(64%)
>60	4(5.4%)

Table 1 reveals that most of the patients with CSF were under 41-60 were 64% and mean age of study was 49±10.5.

Table 2: Baseline characteristics

Characteristics	Number (75)	Control (n=75)	P value
Male	51	48	0.264
Female	24	27	0.267
DM	10	13	0.46
SHT	19	17	0.146
Smoker	29	15	0.06
Male smokers	29	15	0.002
Alcohol	10	8	0.3
Dyslipidemia	4	5	0.45
Obesity	11	2	0.009
Normal EF	47	75	
Mild LVSD	21	0	
Moderate LVSD	6	0	
Severe LVSD	1	0	

Table 2 reveals majority of the patients with coronary slow flow were males. Male smokers with CSFP were significant (p<0.05). Obesity was more common with CSF (p value<0.05).

Table 3: Clinical spectrum of CSFP

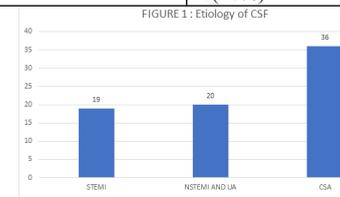| STEMI	19(25.3%)
NSTEMI AND UA	20(26.7%)
CSA	36(48%)

FIGURE 1 : Etiology of CSF

Figure 1 and table 3 reveals that 19 patients had STEMI (25.3%), 20 had NSTEMI and UA (26.7%) and 36 had CSA (48%). Majority of the patients had CSA.

Table 4: TIMI frame count in subjects of slow flow versus controls.

Artery involved	Normal	CSF	P value
LAD	17.43.32	39.62±7.42	<0.05
LCX	16.512.32	33.62±5.42	<0.05
RCA	18.422.82	37.52±6.45	<0.05

TIMI frame count correlated with all arteries in CSF and were significant as shown in table 4.
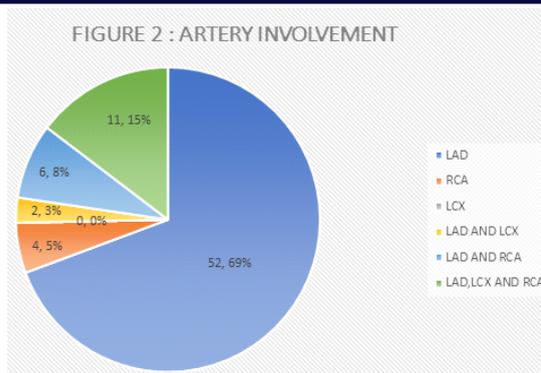
Figure 2: Artery involvement in coronary slow flow

Figure 2 reveals LAD involvement alone in 52 patients (69%), RCA in 4 patients (5%), LCX alone in 0 patients, LAD and LCX in 2 patients (3%), LAD and RCA in 6 patients (8%) and all the three vessels involvement in 11 patients (15%).

DISCUSSION:

This is a case control study. CSFP has been associated with variable risk factors. In our study, CSFP was more common in males with smoking (p value <0.05). This association was similar to the study conducted by Beltrame et al[5] in an Australian population. LAD was the most common artery affected (69%). CSFP presentation is diverse ranging from atypical chest pain, stable or unstable angina (USA), non-ST-elevation myocardial infarction (NSTEMI) to ST-elevation myocardial infarction (STEMI)[6,7]. In our study, 48 % of patients with CSFP presented with CSA and remaining 52% with ACS similar to Iranian population study[8]. In our study, prevalence of CSF phenomenon was 1.7% of all coronary angiograms which is similar to that reported by Beltrame et al of 1% in the Australian population. Most common clinical diagnosis in our study is CSA. 6 patients with CSFP had recurrent CAD. Interestingly we also found that 11 patients had CSFP in all the three coronary arteries and six of them had STEMI-IWMI indicating that diffuse pathology (endothelial dysfunction, inflammation) rather than focal pathology (plaque rupture) could be the possible mechanism.

CSFP usually has a benign long term outcome but occasional ventricular arrhythmias and even sudden cardiac death have been reported.

CONCLUSION:

CSFP is infrequently diagnosed and has a benign course and can rarely cause sudden death. CSFP has a varying clinical spectrum ranging from CSA to STEMI, more common in male smoker and LAD is the most commonly involved artery. The possible mechanism is diffuse endothelial dysfunction, microvascular abnormalities or inflammation rather than focal plaque rupture. Regular follow up is advised as patient can present with recurrences.

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