

# **Noninvasive Predictors of Coronary Slow Flow Phenomenon in Patients Presenting with Chronic Coronary Syndrome**

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## **Abstract**

**Background:** Patients with coronary slow flow phenomenon (CSFP) exhibit the following characteristics: Predominantly middle-aged males, the majority have mixed pattern angina, persistent chest pain sensations after therapy, and many have had repeated invasive and non-invasive examinations.

**Objectives:** Our study aimed to determine the base of non-invasive predictors of coronary slow flow phenomenon in patients presenting with chronic coronary syndrome.

**Patients and Methods:** This a case-control study included 100 participants of suspected coronary artery disease were divided into two groups **matched in age and sex group I:** 50 patients with primary CSFP and **group II:** 50 patients with normal coronary angiography. Each patient was undergoing to demographic data taking, physical investigation, good hydration, restrict fasting hours requested for coronary angiography, 12 lead-Electrocardiogram (ECG)s were obtained for each patient at rest, laboratory parameters, coronary angiography, treadmill exercise ECG, transthoracic echocardiography to assess the thickness of the left ventricle's (LV) wall, its interior dimensions, as well as the LV's ejection fraction (EF) using M-mode method and the aortic propagation velocity.

**Results:** male sex and the Canadian Cardiovascular Society Angina grade's (CCSA) class 3 were significantly decreased in group I compared to group II and male sex, Diabetes mellitus

(DM), smoker and CCSA class 4 were substantially increase in group I compared to group II (P <0.05). P wave max, P Wave dispersion (PWd), corrected QT dispersion (QTcd) at resting ECG, T wave inversion and ST Waves segment depression at stress ECG were significantly increase in group I compared to group II. QTc min was significantly lower in group I compared to group II (P <0.05). LA diameter was significantly increased in group I than group II. Aortic propagation velocity was significantly decreased in group I than group II (P <0.05). Hematocrit, total leucocytic count, mean platelet volume and High-sensitivity C-reactive protein (hsCRP) were significantly increase group I compared to group II (P <0.05).

**Conclusions:** Patients suspected of having a coronary artery disease who are diagnosed with coronary slow flow, male sex, dilated LA, CCSA class 3 or 4, elevated hematocrit value, elevated total leucocytic count, increased mean platelet volume, increased HsCRP, P max, PWd, QTcd, T wave inversion, ST segment depression, and decreased aortic propagation were statistically higher in CSFP patient compared to controls.

**Keywords:** Coronary slow flow phenomenon, Chronic Coronary Syndrome, Aortic propagation velocity, Stress ECG.

## **Introduction:**

The coronary slow flow phenomenon (CSFP) is an extreme delay in vascular opacification in the absence of significant epicardial coronary stenosis is a clinical finding on angiography <sup>[1]</sup>. The overall prevalence of CSFP is 1% between participants undergoing coronary angiography, with the highest rate occurring in individuals presenting with acute coronary syndrome <sup>[2]</sup>. Patients with the CSFP are mostly middle-aged males; the majority have mixed pattern angina; many continue to have chest pain sensations after therapy; and many have repeated invasive and noninvasive examinations <sup>[3]</sup>. The majority of individuals with CSFP present with resting angina, however some may manifest as exercise-induced angina or mixed angina. <sup>[2]</sup>. Despite the favorable investigation for CSFP patients, following progression is typically marked by remitting, relapsing anginal episodes, resulting in significant degradation of quality of life <sup>[4]</sup>. Numerous hypotheses about its cause have been advanced, including an early stage of atherosclerosis, micro vessel dysfunction, an imbalance of vasoconstrictor and vasodilatory factors, and platelet function abnormality. <sup>[4]</sup>. This condition, which can damage any or all of the coronaries, was first reported by <sup>[5]</sup>. Since that time, it has been recognized as a distinct clinical unit known as 'CSFP' syndrome Y, or "primary" coronary slow flow. <sup>[1]</sup>. Numerous case series have now been published, consistently demonstrating that phenomena happen in a distinct demographic group <sup>[3]</sup>. Patients with CSFP may develop repeated angina, needing going back into to the coronary care unit or recurrence coronary angiography in the event of an acute exacerbation. However, people with CSFP have been observed to have several types of angina <sup>[2]</sup>. Therefore, we anticipated that patients with CSFP could exhibit atypical exercise stress electrocardiography data (ExECG). Additionally, it is unknown if individuals with CSFP have distinct exercise responses in terms of LV function. ExECG has been found to be valuable for risk stratification

in patients with supposed or established coronary heart disease who are able to exercise <sup>[6]</sup>. However, it is unknown if ExECG can assist in stratifying exercise capacity and LV function during exercise. Differentiating people with CSFP who respond differently to exercise may allow for differentiated therapy of these patients. In sight of the, we needed to examine the outcomes of ExECG and LV function during exercise using echocardiography, as well as the use of ExECG in stratifying exercise capacity and LV function in patients with CSFP <sup>[7]</sup>. The aim of study is to assess the value of non-invasive markers to predict individuals with main CSFP.

### **Patients and Methods:**

This a case-control study included 100 participants suspected coronary artery disease and referred for coronary angiography in Al-Azhar University Hospitals'' catheterization and noninvasive stress test laboratories. Coronary artery stenosis, coronary vasospasm. coronary ectasia, uncontrolled hypertension, severe LVH, atrial fibrillation and cardiac rhythm other than sinus as paced rhythm, angiography and stenting of heart failure, cardiomyopathy and valvular heart disease, acute myocardial infarction, renal and hepatic dysfunction, acute and chronic infection patients, and patient with contraindications of treadmill exercise test were excluded. Informed written permission was received. from all cases before participating in the study. Also, the study was done after approval by the local ethical committee of Faculty of Medicine, Al-Azhar University (Cairo). Patients divided equally into two groups matched in age and sex group I: 50 patients with primary (CSFP) and group II: 50 patients with normal coronary angiography. **All patients were underwent to the following:** demographic data taking and physical investigation, good hydration and restrict fasting hours, height, weight and BMI (Body mass index) were measured, 12 lead-ECGs were obtained for each patient at relaxation: one standard and the second with 10mm/mV amplitude and 25 mm/sec

rate with standard lead positions, ECGs were physically assessed, laboratory parameters including were composed from the patients after a 12 hours overnight fasting and were done as complete blood picture, lipid profile, Kidney function tests, coronary angiography, examination of the CSFP were made on the base of the TIMI flow grade or TIMI frame count. **Treadmill exercise ECG** using Bruce protocol. This procedure is separated into consecutive three-minute stages requiring the patient to walk faster and at a steeper incline. The testing regimen was tailored to a patient's tolerance, aiming for a period 6 to 12 minutes of exercise, followed by recovery phase which was taken 3 minutes or until return to his base line heart rate. **Transthoracic echocardiography and color Doppler:** the echocardiographic investigation was done at relaxation. Echocardiographic to determine the following parameters, fundamental measurements were included LV wall thickness, LV internal dimensions, LV EF by M-mode method. basic Doppler Echocardiography and aortic propagation velocity.

### **Statistical analysis**

Analysis was performed by SPSS v27 (IBM©, Chicago, IL, USA). Shapiro-Wilks's test and histograms were used to assess the normality of the distribution of data. Quantitative data were presented as mean and standard deviation (SD) and were analysed by unpaired student t-test. Qualitative variables were available in the form of frequency and percentage (%) and were analysed using the Chi-square test or, where appropriate, Fisher's exact test. If the P value is significant less than 0.05.

### **Results:**

In this study patients' demographics, risk factors and clinical data of both groups results in Age, BMI, HTN and CCSA class 2 were insignificantly different between both groups.

Male sex, DM, smoker and CCSA class 3 and 4 were significantly higher in group I than group II (P <0.05). in [Table 1]

**Table 1: Demographic data, risk factors, clinical data and Vital signs of both groups.**

		Group I (n = 50)	Group II (n = 50)	P value
Age (years)	<b>Mean ± SD</b>	44.34 ± 5.04	42.82 ± 4.76	0.124
	<b>Range</b>	33 – 52	35 - 55	
Sex	<b>Male</b>	38 (76.0%)	27 (54.0%)	<b>0.021*</b>
	<b>Female</b>	12 (24.0%)	23 (46.0%)	
BMI (kg/m <sup>2</sup> )	<b>Mean ± SD</b>	30.50 ± 3.83	29.90 ± 4.10	0.454
	<b>Range</b>	23.1 – 39.4	24.2 – 36.9	
Hypertension		20 (40.0%)	22 (44.0%)	0.685
DM		32 (64.0%)	19 (38.0%)	<b>0.009*</b>
Smoker		32 (64.0%)	16 (32.0%)	<b>0.001*</b>
CCSA	<b>Class 2</b>	18 (36.0%)	16 (32.0%)	0.673
	<b>Class 3</b>	23 (46.0%)	34 (68.0%)	<b>0.026</b>
	<b>Class 4</b>	9 (18.0%)	0 (0.0%)	<b>0.002</b>
Heart rate (beats/min)	<b>Mean ± SD</b>	44.34 ± 5.04	42.82 ± 4.76	0.124
	<b>Range</b>	33 – 52	35 - 55	
Systolic blood pressure (mmHg)	<b>Mean ± SD</b>	123.10 ± 9.25	121.80 ± 8.56	0.467
	<b>Range</b>	110 – 135	110 - 135	
Diastolic blood pressure (mmHg)	<b>Mean ± SD</b>	78.40 ± 6.18	77.10 ± 5.72	0.278
	<b>Range</b>	70 – 85	70 - 85	

BMI: body mass index, HTN: hypertension, DM: diabetes mellitus, CCSA: Canadian Cardiovascular Society grading of Angina pectoris, \*: significant as p value <0.05.

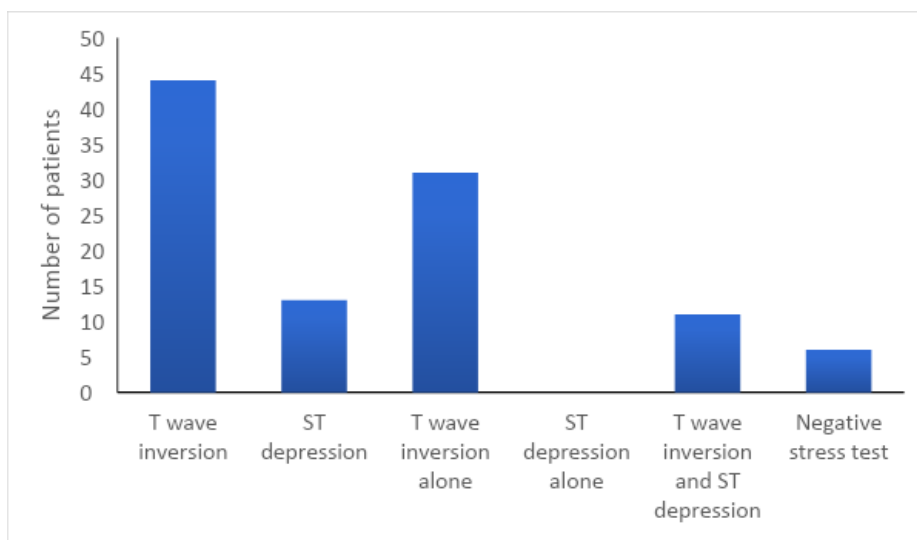
P max, PWd and QTcd were significantly increase in group I compared to group II. QTc min was significantly lower in group I than group II ( $P < 0.05$ ) in [Table 2]

**Table 2: Resting ECG data of both groups.**

		Group I (n = 50)	Group II (n = 50)	P value
P max (msec)	<b>Mean <math>\pm</math> SD</b>	125.20 $\pm$ 25.17	110.80 $\pm$ 17.71	<b>0.001*</b>
	<b>Range</b>	80 – 160	80 - 140	
P min (msec)	<b>Mean <math>\pm</math> SD</b>	59.60 $\pm$ 14.84	61.20 $\pm$ 16.37	0.610
	<b>Range</b>	40 – 80	40 - 80	
PWd (msec)	<b>Mean <math>\pm</math> SD</b>	65.60 $\pm$ 20.62	49.60 $\pm$ 10.09	<b>&lt;0.001*</b>
	<b>Range</b>	40 – 100	40 - 60	
QTc max (msec)	<b>Mean <math>\pm</math> SD</b>	467.20 $\pm$ 55.37	469.20 $\pm$ 35.50	0.830
	<b>Range</b>	380 - 560	420 - 540	
QTc min (msec)	<b>Mean <math>\pm</math> SD</b>	365.20 $\pm$ 46.57	399.20 $\pm$ 36.80	<b>&lt;0.001*</b>
	<b>Range</b>	300 - 440	340 - 460	
QTcd (msec)	<b>Mean <math>\pm</math> SD</b>	102.0 $\pm$ 21.09	70.0 $\pm$ 10.10	<b>&lt;0.001*</b>
	<b>Range</b>	60 – 140	60 - 80	

PWd = P wave dispersion, QTc= corrected QT interval, QTcd = corrected QT dispersion, \*: significant as p value  $< 0.05$ .

Stress ECG test was negative in 6 (12%) patients. T wave inversion was in 44 (88%) patients and ST depression was in 13 (26%) patients. T wave inversion alone was in 31 (62%) patients and ST depression alone didn't occur in any patients. T wave inversion with ST depression was in 11 (22%) patients.



**Figure 1: Stress ECG data in group I.**

LA were significantly increased in group I compared to group II. Aortic propagation velocity was significantly decreased in group I than group II ( $P < 0.05$ ). [Table 3]

**Table 3: Echocardiographic data of both groups**

		Group I (n = 50)	Group II (n = 50)	P value
LVEDD (mm)	<b>Mean ± SD</b>	48.60 ± 5.12	47.94 ± 5.14	0.521
	<b>Range</b>	40 - 57	40 - 57	
LVESD (mm)	<b>Mean ± SD</b>	29.16 ± 6.02	27.96 ± 5.92	0.318
	<b>Range</b>	17 - 39	17 - 42	
EF (%)	<b>Mean ± SD</b>	65.26 ± 4.69	64.46 ± 4.34	0.378
	<b>Range</b>	57 - 72	57 - 72	
E/A ratio	<b>Mean ± SD</b>	0.77 ± 0.10	0.74 ± 0.11	0.164
	<b>Range</b>	0.6 - 0.9	0.6 - 0.9	
LA (mm)	<b>Mean ± SD</b>	36.68 ± 5.24	33.96 ± 3.50	<b>0.003*</b>
	<b>Range</b>	27 - 46	29 - 40	

Aortic propagation velocity (cm/s)	<b>Mean ± SD</b>	37.12 ± 5.48	46.62 ± 4.11	<b>&lt;0.001*</b>
	<b>Range</b>	28 - 45	40 - 53	

LVEDD: Left ventricular end diastolic dimension. LVESD: Left ventricular end systolic dimension, EF: Ejection fraction, \*: significant as p value <0.05.

TIMI frame count (frames/second) in LAD was significantly increased in 3 vessel involvements than 2 vessel involvements, in LCX was significantly increased in 3 vessel involvements than 2 vessel involvements, and in RCA was significantly increased in 3 vessel involvements than 1 vessel involvement **Table 4**].

**Table 4: Coronary angiographic data of coronary slow flow in group I.**

		Group I (n = 50)	TIMI frame count (frames/second)		
			LAD	LCX	RCA
Coronary angiography	<b>LAD alone</b>	0			
	<b>LCX alone</b>	0			
	<b>RCA alone</b>	6 (12%)			32.33 ± 2.74
	<b>LAD and LCX</b>	12 (24%)	40.5 ± 16.86	41.23 ± 17.4	
	<b>LAD and RCA</b>	0			
	<b>LCX and RCA</b>	0			
	<b>LAD, LCX and RCA</b>	32 (64%)	52.06 ± 15.98	52.06 ± 20.18	44.46 ± 13.83
<b>P value</b>			<b>0.041*</b>	<b>0.048*</b>	<b>0.041*</b>

Hematocrit, total leucocytic count, mean platelet volume and HsCRP were significantly increased in group I compared to group II (P <0.05).

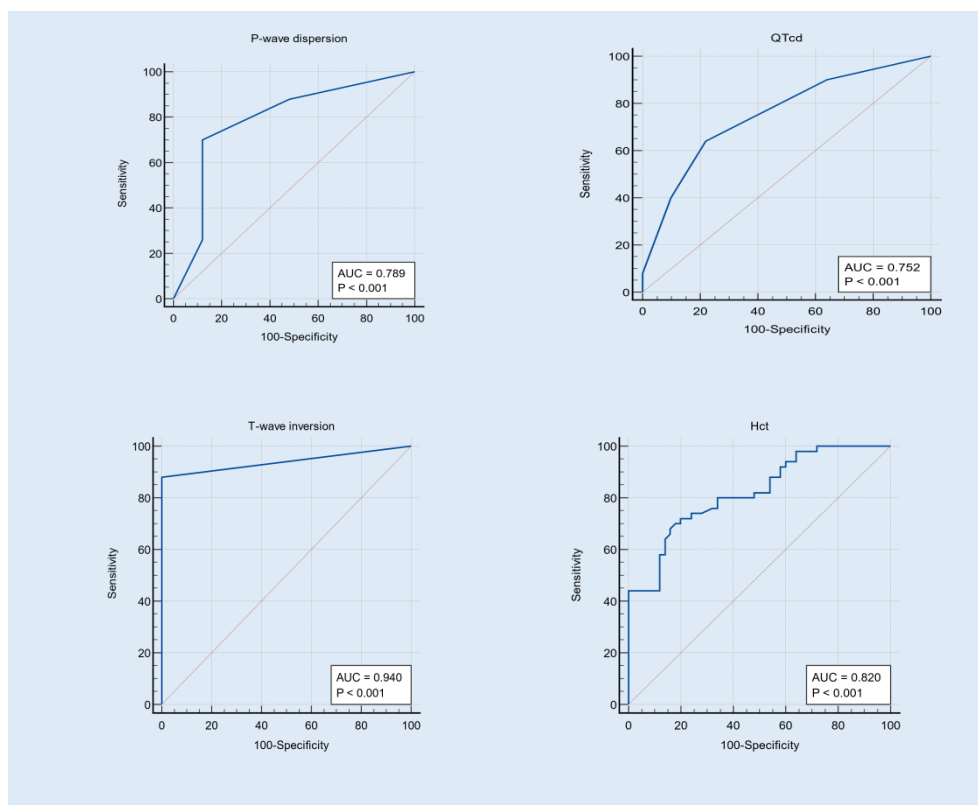
**Table 5: Laboratory data of both groups**

		Group I (n = 50)	Group II (n = 50)	P value
Total cholesterol (mg/dl)	<b>Mean ± SD</b>	203.9 ± 40.05	199.66 ± 36.55	0.582
	<b>Range</b>	141 – 300	148 - 258	
Triglycerides (mg/dl)	<b>Mean ± SD</b>	101.84 ± 21.21	96.60 ± 20.06	0.207
	<b>Range</b>	66 – 134	67 - 133	
LDL (mg/dl)	<b>Mean ± SD</b>	128.76 ± 27.47	131.88 ± 22.07	0.533
	<b>Range</b>	93 – 169	90 - 169	
HDL (mg/dl)	<b>Mean ± SD</b>	42.86 ± 4.69	41.90 ± 4.93	0.321
	<b>Range</b>	35 – 50	35 - 50	
Hemoglobin (gm/dl)	<b>Mean ± SD</b>	12.46 ± 0.84	12.34 ± 0.84	0.462
	<b>Range</b>	11 - 14.1	11 - 13.5	
Hematocrit (%)	<b>Mean ± SD</b>	39.38 ± 2.53	37.01 ± 2.51	<b>&lt;0.001*</b>
	<b>Range</b>	35 - 44.3	33 - 40.5	
Total leucocytic count (*10 <sup>3</sup> cells/dl)	<b>Mean ± SD</b>	9.76 ± 2.07	7.33 ± 1.97	<b>&lt;0.001*</b>
	<b>Range</b>	5.4 – 13	4.2 - 10.8	
Platelets	<b>Mean ± SD</b>	260.28 ± 60.12	250.40 ± 67.98	0.443
	<b>Range</b>	156 – 368	152 - 370	
Mean platelet volume (fl)	<b>Mean ± SD</b>	10.30 ± 1.52	7.98 ± 0.87	<b>&lt;0.001*</b>
	<b>Range</b>	8 – 13	7 - 9	
HsCRP (mg/l)	<b>Mean ± SD</b>	7.50 ± 1.90	3.02 ± 1.96	<b>&lt;0.001*</b>
	<b>Range</b>	0 – 10	0 - 6	
Serum creatinine (mg/dL)	<b>Mean ± SD</b>	0.93 ± 0.20	0.99 ± 0.24	0.194
	<b>Range</b>	0.6 - 1.3	0.6 - 1.3	
HbA1c (mmol/l)	<b>Mean ± SD</b>	6.09 ± 0.65	6.32 ± 0.75	0.118

	Range	5 - 7.2	5 - 7.8	
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LDL: Low density lipoprotein, HDL: High density lipoprotein, HsCRP: High sensitivity C reactive protein  
HbA1C: Hemoglobin A1C \*: significant as p value <0.05.

T-wave inversion is a good predictor for CSFP (AUC =0.940, 95% CI =0.874-0.978, p value <0.001). When present, it has 88% sensitivity, 100% specificity, 100% PPV, and 89.3% NPV. P-wave dispersion is a good predictor for CSFP (AUC =0.789, 95% CI =0.696-0.864, p value <0.001). At a cut off value of >60, it has 70% sensitivity, 88% specificity, 85.4% PPV, and 74.6% NPV. QTcd is a good predictor for CSFP (AUC =0.752, 95% CI =0.655-0.833, p value <0.001). At a cut off value of >80, it has 64% sensitivity, 78% specificity, 74.4% PPV, and 68.4% NPV. Hct is a good predictor for CSFP (AUC =0.820, 95% CI =0.730-0.889, p value <0.001). At a cut off value of >38.5, it has 72% sensitivity, 80% specificity, 78.3% PPV, and 74.1% NPV. HsCRP is a good predictor for CSFP (AUC =0.950, 95% CI =0.887-0.984, p value <0.001). At a cut off value of >5, it has 90% sensitivity, 86% specificity, 86.5% PPV, and 89.6% NPV.



**Figure 3: ROC curve of (a) T-wave inversion, (b) P-wave (c) Qtcd,(d) Hematocrit**

### **Discussion**

CSFP is defined by the disappear of significant coronary artery lesions identified during coronary angiography but with delayed blood perfusion, excluding thrombolytic therapy, coronary angioplasty, coronary spasm, coronary dilatation, coronary stenosis, cardiomyopathy, significant valvular heart disease, decompensated heart failure, and certain connective tissue disorders of the coronary microvasculature. Tambe et al. described it for the first time in 1972. CSFP manifests clinically similarly to coronary atherosclerotic heart disease.<sup>[8-10]</sup> In agreement with our study, Mahfouz et al. (2014)<sup>[11]</sup> conducted a case control study, taking patients admitted to cardiac catheterization for suspected coronary artery disease. They took two groups 50 patients each. Group I: primary (CSF). Group II: normal coronary angiography. All patients were subjected to thorough clinical investigation and total lab including lipid panel, hsCRP and Troponin, ECG where PWD and QTc dispersion were measured, as well as assessment of TIMI frame counts there. The study reported an insignificance difference between the two groups regarding the age, the gender, the BMI, and hypertension. In contrast to our findings, Huang et al. (2021)<sup>[12]</sup> study included patients with normal epicardial arteries angiographically (145 patients with CSFP and 145 normal controls). Clinical data and laboratory indices were collected and analyzed prior to coronary angiography using peripheral venous blood samples. Logistic regression analysis was performed for statistical analysis. They demonstrated that the BMI was higher in the CSFP group than the control group (P=0.01). this may be linked to the larger used sample size and the different nature of Chinese people compared to the Egyptian. However, Gunes et al. (2011)<sup>[13]</sup> reported that transthoracic echocardiography and brachial artery ultrasonography

were used to investigate patients with CSF and 23 persons with normal epicardial coronary arteries. Two months following therapy with aspirin or aspirin with nebivolol, the patients were reevaluated. The results highlighted that patient with CSF had higher body mass index ( $26.5 \pm 3.3$  vs.  $23.8 \pm 2.8$ ,  $p < 0.001$ ). This difference may be linked to relatively recruited small sample size and the higher incidence of obesity in the Egyptians. In the present study, CCSA class 2 were insignificantly different between both groups. Our findings were in line with Mahfouz et al. (2014)<sup>[11]</sup> study results which showed that patients with CSFP and control groups showed insignificant difference regarding CCSA class 2. In our study, male sex, DM, and smoking, were significantly increased in group I than group II. In line with our findings, Mahfouz et al. (2014)<sup>[11]</sup> noted the number of smokers was higher in the PCSF group compared to the control group ( $p \text{ value} < 0.0001$ ) and a significant difference between both groups as regards to DM ( $p \text{ value} = 0.025$ ), but an insignificant difference was found in sex between the two studied groups. This was confirmed by Madak et al. (2010)<sup>[14]</sup> who found that smoking rate was significantly increased in the CSFP compared to the normal flow group ( $p < 0.001$ ), yet insignificant difference was found in DM and sex. In contrast, Sadr-Ameli (2015) [21] in a prospective cross-sectional investigation, 217 consecutive patients who underwent coronary angiography and had signs of CSFP were examined at baseline and after therapy for demographic and coronary risk factor profiles, as well as clinical outcomes and found that 76% of the patients were male, so male gender may act as a predictor of CSFP. CCSA class 3 were significantly decreased in group I than group II CCSA class 4 were significantly higher in group I than group II. Our results agreed with Mahfouz et al. (2014)<sup>[11]</sup> findings; CCSA class 3 were significantly lower in CSFP group than control group, and CCAS class 4 were significantly increased in CSFP group than control group. In the present study, P max, PWd, QTcd, T wave inversion and ST depression were significantly increased

in group I than group II. QTc min was significantly lower in group I than group II ( $P < 0.05$ ). Our findings were in line with Mahfouz et al. (2014)<sup>[11]</sup> reported that the PCSF group had higher P max, P wave dispersion, QTcd compared to the control group with significant difference, but patients with PCSF had higher QTc min than the control group with significant statistical difference. Comparable to our results, Mahmoud et al. (2013)<sup>[15]</sup> CSF patients had more P max, P dispersion, QTc min and QTc dispersion In our study, stress ECG was negative in 6 (12%) of patients in group 1. T wave inversion was in 44 (88%) of patients, while ST depression was in 13 (26%) of patients. T wave inversion alone was in 31 (62%) ST depression alone didn't occur in any patient. Similarly, Wang et al. (2019)<sup>[16]</sup> enrolled 30 patients with CSFP and 24 controls in a case-control study. Investigation of CSFP was made by TIMT frame count (TFC). Exercise stress electrocardiography (ExECG) and LV function evaluated by echocardiography at rest, during exercise and recovery phase. It was noted that in total, positive ExECG was found in 6 (20%) CSFP patients, negative ExECG was found in 24 (80%) CSFP patients and ST-segment depression was in 5 (17%). In our study, LVEDD, LVESD, EF and E/A ratio were insignificantly different between both groups. Similarly, Mahfouz et al. (2014) found that in terms of the echo data; there was no statistically significant difference concerning LVEDD, LVESD, EF, E/A ratio. Nevertheless, Güneş et al. (2009)<sup>[17]</sup> reported that E/A ratio ( $0.89 \pm 0.27$  vs.  $1.27 \pm 0.27$ ,  $p < 0.001$ ) was decreased in patients with CSF as compared with control group. This might be explained that most of our patients showed impaired diastolic function may be due to the prevalent obesity, hypertension among both groups. In the present study, LA were significantly increased in group I compared to group II. Aortic propagation velocity was significantly decreased in group I than group II ( $P < 0.05$ ). In line with our results, Ozdemir et al. (2021)<sup>[18]</sup> enrolled 86 CSF patients and 43 subjects with normal coronary flow. They utilized the TIMI frame count (TFC)

method for determining each enrolled patient's coronary flow rate. APV values were determined via color M-mode Doppler echocardiography. Differences in NLR and APV values were determined in CSF and control groups. They observed that patients with CSF had lower APV values ( $39.9 \pm 11.4$  vs  $48.0 \pm 10.6$ ,  $p < 0.01$ ). However, Güneş et al. (2009)<sup>[17]</sup> found insignificantly different concerning LA diameter between CSF and control subjects ( $p$  value = 0.140). In contrast, Mahmoud et al. (2013) results showed significant statistical difference concerning LA diameter between CSF patients and controls. Our results found that total cholesterol, triglycerides, LDL, HDL, hemoglobin, platelets, serum creatinine and HbA1c were insignificantly different between both groups. This is confirmed by Mahfouz et al. (2014)<sup>[11]</sup> who reported that there was an insignificance difference in total cholesterol, triglycerides, LDL, HDL, hemoglobin, platelets, serum creatinine and HbA1c. In line with our findings, Hawkins et al. (2011)<sup>[19]</sup> found insignificant difference between subjects with CSFP and controls total cholesterol, triglycerides, LDL, HDL, and HbA1c. Additionally, Huang et al. (2021)<sup>[20]</sup> findings demonstrated an insignificant difference in total cholesterol, triglycerides, LDL, HDL, and platelets between both groups, yet hemoglobin and creatinine were higher in CSFP group. Further, Mahmoud et al. (2013)<sup>[15]</sup> observed that total cholesterol, triglycerides, LDL, and HDL levels were not statistically different, but serum creatinine was higher in CSF group. In our present study, Hematocrit (HCT), total leucocytic count (TLC), mean platelet volume (MPV) and HsCRP were significantly increased in group I compared to group II ( $P < 0.05$ ). Mahfouz et al. (2014) [4] findings are in agreement with our results. The patients with CSFP had higher levels of WBCs, HCT, HsCRP and MPV compared to patients in the control group. P-wave dispersion was a good predictor for CSFP with 70% sensitivity and 88% specificity. Similarly, QTcd was a good predictor for CSFP with 64% sensitivity and 78% specificity as well Hct with 72% sensitivity and 80% specificity. HsCRP was good

predicator for CS 90% sensitivity and 86% specificity. In line with our findings, Mahfouz et al. (2014) found that P-wave dispersion showing sensitivity of 78 % and specificity of 70 %, QTcd showing sensitivity of 76 % and specificity of 64%, HCT showing sensitivity of 82 % and specificity of 46%, and HsCRP showing sensitivity of 90 % and specificity of 70%. Our study was limited with a low number of participants and was performed at a single hospital. Further, the APV was only measured once for each patient (at the time of admission), so we could not determine whether medical treatment had any effect on these values. Using of a single blood sample can't predict the persistence of laboratory parameters over time. Follow up values of these parameters and their relation to clinical prognosis were not evaluated. The patients didn't undergo IVUS to detect atherosclerotic changes in the current study despite IVUS is a more sensitive tool for identifying coronary atherosclerosis than coronary angiography.

### **Conclusions:**

Patients with suspected coronary artery disease and diagnosed as coronary slow flow, male sex, smoker, dilated LA, CCSA class 3, increased hematocrit value, increased total leucocytic count, higher mean platelet volume, increased HsCRP, P max, PWd, QTcd, T wave inversion, ST segment depression, and aortic propagation were statistically higher in CSFP patient compared to controls. Consent: Informed written permission was received. from all cases before participating in the study.

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**Conflict of Interest:** Nil

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