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ORIGINAL ARTICLE

Predictors of Coronary Slow Flow Phenomena in Patients with Acute Coronary Syndrome; A Cross Sectional Study

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ABSTRACT

Background: Coronary slow flow phenomenon (CSPF) is a prevalent clinical syndrome that results in both chronic and acute coronary syndromes (ACS). Early prediction of CSFP in patients with ACS could change the treatment strategy from primary percutaneous coronary intervention (PCI) to pharmaco-invasive strategy with subsequent improvement of patient prognosis. **Methods:**A cross sectional study was performed in Zagazig university hospitals and Ahmed Maher teaching hospitals in the period between January 2023 and June 2023. We included all patients with ACS and checked for the prevalence of CSFP without obstructive coronary artery disease (CAD). Then, we studied the best non-invasive predictors of CSFP in patients with ACS.

Results: The study enrolled 100 patients with ACS in the period between January 2023 and June 2023. Coronary slow flow phenomenon without obstructive CAD was detected in 27 (27%) cases. P wave dispersion was the best independent predictor of coronary slow flow (Odds ratio: 1.302 (1.140 – 1.487), P <0.001). The best co-predictors were DM and LA diameter. **Conclusions:**Coronary slow flow phenomenon is not an uncommon finding among patients with ACS. Among non-invasive predictors, P wave dispersion was the best predictor CSFP in patients with ACS together with DM and LA diameter.

Key words: Coronary slow flow phenomenon**;** acute coronary syndromes**;** Patients..

INTRODUCTION

Coronary slow flow phenomenon (CSPF) is a prevalent clinical syndrome that results in both chronic and acute coronary syndromes (ACS). Its prevalence reaches 1-7% of all patients undergoing diagnostic coronary angiography [1].Coronary slow flow phenomenon is typically described as delayed opacification of epicardial coronary arteries during coronary angiography with subsequent

myocardial ischemia [2]. Furthermore, it increases the risk of ventricular arrhythmias and sudden cardiac death [3]. The true mechanism of CSFP is not known. However, many theories were suggested such as endothelial and microvascular dysfunction [3, 4].

As a common clinical outcome, the world's most common cause of cardiovascular death is acute myocardial infarction [5]. Early primary percutaneous coronary intervention (PPCI) saves myocardium and life. It is not uncommon to face a patient with CSFP without underlying obstructive coronary artery disease (CAD). In such scenarios, PPCI is not a treatment option [6]. Therefore, early prediction of CSFP in patients with ACS could change the treatment strategy from PPCI to pharmaco-invasive strategy. The early use of thrombolytic therapy or glycoprotein inhibitors (GPI) could improve the patient's prognosis.

Herein, we studied the incidence and predictors of CSFP in ACS patients who visit Ahmed Maher Teaching Hospital and Zagazig University Hospitals.

METHODS

We performed a cross sectional study in Zagazig university hospitals and Ahmed Maher teaching hospitals in the period between January 2023 and June 2023. Institutional research board (IRB) committee of faculty of medicine, Zagazig university, Egypt has reviewed and accepted the study protocol with reference number (ZU.IRB #10037/26-10-2022). We obtained informed consent from all participants. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Sample Size: The study enrolled 100 patients with acute coronary syndrome in the period between January 2023 and June 2023.

Patients:

All ACS patients, regardless of whether they had a non-ST elevation myocardial infarction (NSTEMI) or a ST elevation myocardial infarction (STEMI), were included. STEMI was defined as persistent acute chest pain more than 15 minutes with ST elevation equal or more than 1 mm in two contagious electrocardiogram (ECG) leads. In leads V2 and V3, we used a cut off value equal or more than 1.5 mm, 2 mm, and 2.5 mm in women, men older than 40 years, and men younger than 40 years respectively. Furthermore, we used 0.5 mm cut off value for ST segment elevation in right and posterior precordial leads in all patients. NSTEMI was defined as persistent acute chest pain more than 15 minutes with heart troponin levels rising or falling, with at least one reading exceeding the 99th percentile in absence of ST elevation in ECG [7]. We excluded patients with atrial fibrillation, history of prior obstructive CAD, history of coronary artery bypass graft (CABG) and percutaneous coronary intervention (PCI).

Methods:

We collected basic characteristics and risk factors for all patients including age, gender, body built, and history of hypertension, diabetes mellitus, dyslipidemia, smoking, stroke, peripheral vascular disease, and chronic kidney disease (CKD). Additionally, we looked for a family history of premature cardiovascular disease, which is defined as cardiovascular illness that develops before the age of 45 for men and 55 for women. All patients underwent assessment of vital signs

and admission Killip class. We classified patients according to Killp class into; Killip class 1 defining patients with no evidence of heart failure, Killip class 2 defining patients with decompensated heart failure, Killip class 3 defining patients with acute pulmonary edema, and Killip class 4 defining patients with cardiogenic shock[8]. ECG was performed immediately within ten minutes of patient's presentation using Sonoscape ECG device at voltage of 10mm/mV and at speed of 25 mm/sec. From ECG, we obtained heart rate, site and P wave dispersion (PWd), which is defined as the difference between the longest and smallest P wave duration in the same surface ECG[9], and corrected QT interval (QTc) are examples of ischemia ECG abnormalities, including ST elevation, ST depression, and/or T wave inversion. We calculated QTc using Bazett formula (QT interval / √ R-R interval) [10]. The difference between the maximal and smallest QTc values in the same surface ECG was used to measure QTc dispersion [11].

We performed laboratory assessment for all patients without delayed invasive PPCI in patients with ACS. Laboratory assessment involved serum troponin, serum creatinine, hemoglobin A1C, and lipid profile including total cholesterol (TC), triglycerides (TG). high density lipoprotein (HDL), and low-density lipoprotein (LDL).

Echocardiographic assessment:

For all patients we measured the end diastolic posterior wall thickness (PWTD), end diastolic interventricular septal thickness (IVSTD), end diastolic end diastolic volume (LVEDV), and end diastolic systolic volume (LVESV), and LVEF as following [LVEDV – LVESV / LVEDV] in both apical 2 and apical 4 chambers views [12]. Using the M-mode cursor at the level of the aortic valve leaflets, the LA diameter was determined from the aortic root recordings [13]. Furthermore, we checked for mitral regurgitation and the territory of ischemic wall motion abnormality.

In compliance with the revascularization guidelines of the European Society of Cardiology, all patients received PPCI and coronary angiography. Through coronary angiography, CSFP was identified based on a decrease in thrombolysis in myocardial infarction (TIMI) flow grade of 2 or the increase in corrected TIMI frame count (TFC) of greater than 27 frames in one or more epicardial vessel using 30 frames/second frame rate. TIMI frame count stands for the bare minimum required through the cine-viewer frame counter for the first contrast edge to reach conventional distal coronary markers. The first frame to be counted is the one where the arterial ostium's diameter is at least 70% filled with contrast. The instant at which the contrast begins to fill the last landmark is represented by the final frame. The mustache segment, the distal bifurcation segment, and the first branch of the posterolateral artery are the markers for the left anterior descending (LAD), left circumflex (LCX), and right coronary artery (RCA), respectively, [14, 15]. LAD corrective TFC is calculated by dividing the TFC of the LAD by stand a factor of 1.7. We included patients with acute coronary syndrome with or without coronary artery ectasia.

The assessment of TIMI thrombus grade was performed as follows: grade 0 (G0) represents the absence of any angiographic characteristics associated with thrombus,

grade 1 (G1) denotes the presence of angiographic features that are indicative but not conclusive of thrombus, such as decreased contrast density, haziness, uneven lesion contour, or a smooth convex meniscus. Grade 2 (G2) indicates the presence of a thrombus with its greatest dimensions measuring 1/2 or less of the vessel diameter. Grade 3 (G3) indicates the presence of a definite thrombus with the greatest linear dimension being greater than 1/2 but less than 2 vessel diameters; grade 4 (G4) indicates a definite thrombus with the largest dimension measuring at least 2 vessel diameters; and grade 5 (G5) denotes thrombotic total occlusion [16].

Two interventional cardiologists independently assessed coronary angiography in each patient. A third observer resolved any disagreements when present.

Statistical analysis

While categorical variables are shown as numbers and percentages, continuous variables are shown as mean ± standard deviation (SD). To compare continuous variables, the independent-sample t-test was employed. To compare categorical variables, the chi-square test was employed.

RESULTS

Coronary slow flow phenomenon without obstructive CAD was detected in 27 (27%) cases. Patients were divided according to the presence of CSFP into two groups; group A CSFP without obstructive CAD and group B obstructive CAD.

Regarding basic characteristics (table1), demographic, risk factors, and baseline clinical data did not differ significantly in the two groups except for diabetes mellitus (DM) and dyslipidemia. Group A patients had higher prevalence of DM (77.8% vs 49.3%; P 0.011) and lower prevalence of dyslipidemia (41.1% vs 48.1%; P 0.028). Laboratory assessment did not show any significant difference between the two groups except for HDL that was lower in group A (39.69 ± 3.56 mg/dl vs 41.42 ± 3.11 mg/dl; P 0.019).

ECG and echocardiographic assessment (table 2) showed that QTc min was significantly lower in group A (359.48 ± 17.22ms vs 398.85ms ± 43.62; P <0.001). However, QTc max did not differ significantly in the two groups. Group A patients had higher P wave dispersion (63.04 ± 6.46ms vs 42.45 ± 10.35ms; P <0.001) and larger left atrial diameter (38.78 ± 2.81mm vs 33.34 ± 3.48mm; P <0.001). The two groups did not differ regarding all other ECG and echocardiographic variables.

The two groups were matched regarding culprit vessel (table 2). Patients in the group B showed different grades of thrombus (grade 0 (35.6%), grade 1 (19.2%), grade 2 (11%), grade 3 (19.2%), grade 4 (2.7%), and grade 5 (12.3%).

Stepwise regression analysis revealed that P wave dispersion is the best independent predictor of coronary slow flow (Odds ratio: 1.302 (1.140 – 1.487), P <0.001). The best co-predictors were DM and LA diameter. Details of univariate and multivariate analysis are illustrated in (table 3). Receiver operating characteristics (ROC) curve (figure 1) was performed to P wave dispersion showing that the best cut off value predicting CSFP was 57.5 msec with sensitivity 85.2% and specificity 83.6%.

Table 1: Baseline characteristics of the study groups.

		Group A N = 27	Group B N= 73	Test-value	P-value
Age		54.93 ± 10.71	56.64 ± 10.58	0.718	0.474
Gender	Males	10 (37.0%)	28 (38.4%)	0.015	0.904
	Female	17 (63.0%)	45 (61.6%)		
Diabetes Mellitus		21 (77.8%)	36 (49.3%)	6.515	0.011
Hypertension		13 (48.1%)	30 (41.1%)	0.400	0.527
Dyslipidemia		15 (55.6%)	23 (31.5%)	4.838	0.028
Smoker		15 (55.6%)	41 (56.2%)	0.003	0.957
Obesity		13 (48.1%)	23 (31.5%)	2.369	0.124
Chronic kidney disease		11 (40.7%)	20 (27.4%)	1.641	0.200
Peripheral vascular disease		4 (14.8%)	14 (19.2%)	0.254	0.614
Stroke		3 (11.1%)	8 (11.0%)	0.000	0.983
Family history of premature CVD		6 (22.2%)	14 (19.2%)	0.114	0.735
Admission heart rate		84.78 ± 10.61	85.93 ± 11.84	0.444	0.658
Admission systolic BP		121.11 ± 18.03	127.78 ± 19.28	1.562	0.121
Admission diastolic BP		81.56 ± 11.2	76.88 ± 12.19	-1.741	0.085
Killip class	1	16 (59.3%)	50 (68.5%)	2.886	0.410
	2	9 (33.3%)	14 (19.2%)		
	3	1 (3.7%)	7 (9.6%)		
	4	1 (3.7%)	2 (2.7%)		
Acute coronary syndrome type	UA	14 (51.9%)	30 (41.1%)	0.926	0.629
	NSTEMI	6 (22.2%)	20 (27.4%)		
	STEMI	7 (25.9%)	23 (31.5%)		
Troponin Median (IQR)		40 (32 - 200)	62 (35 - 199)	-0.369	0.712
CK MB Median (IQR)		27 (21 - 37)	29 (21 - 45)	-0.699	0.484
Creatinine		1.33 ± 0.49	1.18 ± 0.46	-1.338	0.184
Low density lipoprotein		118.7 ± 27.2	111.37 ± 35.74	-0.966	0.336
High density lipoprotein		39.69 ± 3.56	41.42 ± 3.11	2.376	0.019
Total cholesterol		226.93 ± 25.31	224.78 ± 25.85	-0.370	0.712
Triglycerides		265.44 ± 84.91	258.96 ± 90.29	-0.324	0.747
Hemoglobin A1C		8.97 ± 1.36	8.62 ± 1.41	-1.114	0.268

Group A: patients with coronary slow flow phenomenon, group B: patients with obstructive coronary artery disease, CVD: cardiovascular disease, BP: blood pressure, CK MB: creatine kinase myocardial band, IQR: interquartile range.

Table 2:ECG, echocardiographic, and angiographic findings in the study population.

		Group A N = 27	Group B N= 73	Test-value	P-value
Site of ischemia in ECG	No	5 (18.5%)	10 (13.7%)	5.050	0.168
	Lateral	5 (18.5%)	22 (30.1%)		
	Inferior	12 (44.4%)	18 (24.7%)		
	Anterior	5 (18.5%)	23 (31.5%)		
QTc maximal value (msec)		471.41 ± 13.95	475.67 ± 117.12	0.188	0.851
QTc minimal value (msec)		359.48 ± 17.22	398.85 ± 43.62	4.548	0.000
P wave dispersion (msec)		63.04 ± 6.46	42.45 ± 10.35	-9.647	0.000
Left ventricular ejection fraction (%)		61.28 ± 7.69	62.5 ± 9	0.623	0.535
Left atrial diameter (mm)		38.78 ± 2.81	33.34 ± 3.48	-7.272	0.000
Significant MR		4 (14.8%)	12 (16.4%)	0.039	0.844
LVDD grade	Grade 1	16 (59.3%)	46 (63.0%)	0.475	0.789
	Grade 2	9 (33.3%)	24 (32.9%)		
	Grade 3	2 (7.4%)	3 (4.1%)		
Culprit vessel in CA	No	5 (18.5%)	10 (13.7%)	5.050	0.168
	LCX	5 (18.5%)	22 (30.1%)		
	RCA	12 (44.4%)	18 (24.7%)		
	LAD	5 (18.5%)	23 (31.5%)		

Group A: patients with coronary slow flow phenomenon, group B: patients with obstructive coronary artery disease, ECG: electrocardiogram, LVDD: left ventricular ejection fraction, CA: coronary angiography, MR: mitral regurgitation

Table 3: Univariate and multivariate regression analysis for the best predictors of coronary slow flow phenomenon.

	Univariate regression analysis			Multivariate regression analysis		
	Odds ratio	**95% confidence interval**	**P value**	**Odds ratio**	**95% confidence interval**	**P value**
DM	3.597	1.301 – 9.943	0.001	45.379	1.928 – 1068.263	0.018
QTc min	0.962	(0.943 – 0.980)	<0.001	0.986	0.971 – 1.002	0.087
PWd	1.302	(1.140 – 1.487)	<0.001	1.305	1.095 – 1.555	0.003
LA diameter	1.574	(1.304 – 1.899)	<0.001	2.112	1.283 – 3.476	0.003

DM: diabetes mellitus, PWd: P wave dispersion, LA: left atrium.

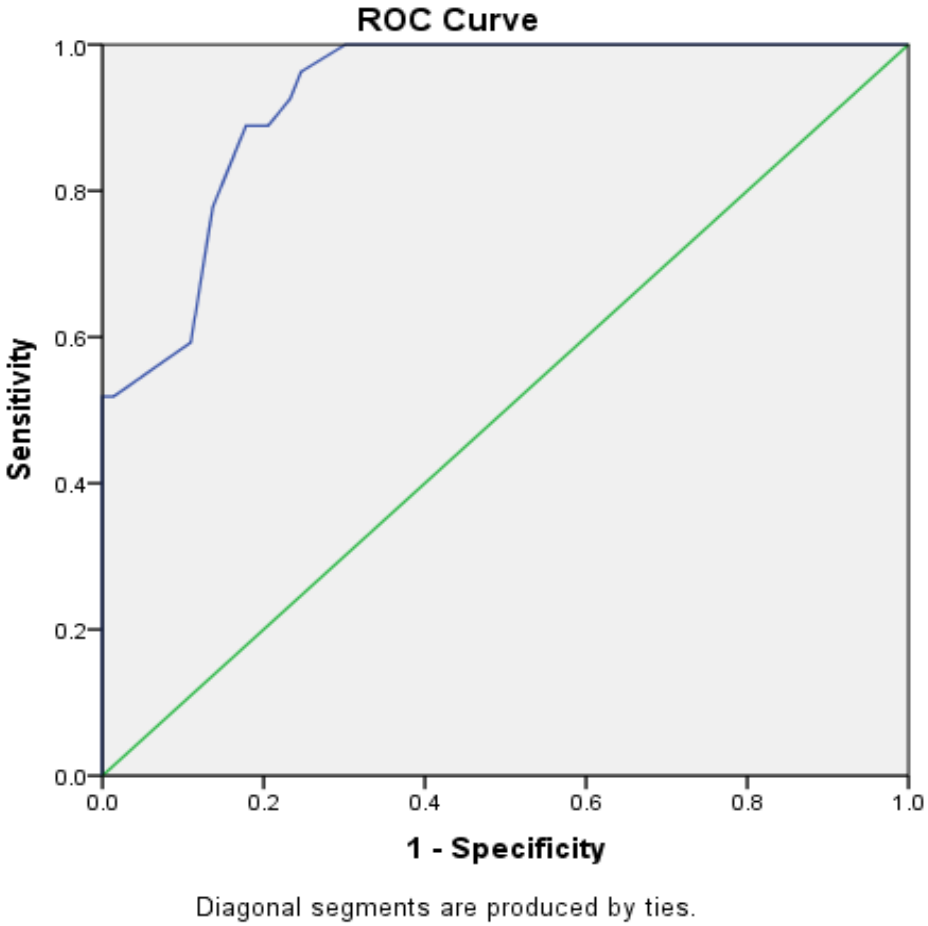

Figure 1: Receiver operation characteristics curve for P wave dispersion for prediction of coronary slow flow phenomenon in patients with acute coronary syndrome.

DISCUSSION

Coronary slow flow phenomenon (CSPF) is a prevalent clinical syndrome that results in both chronic and acute coronary syndromes (ACS). Its prevalence reaches 1-7% of all patients undergoing diagnostic coronary angiography [1]. CSPF is typically described as delayed opacification of epicardial coronary arteries during coronary angiography with subsequent myocardial ischemia [2].

It is not uncommon to face a patient with CSFP without underlying obstructive CAD. In such scenarios, PPCI is not a treatment option [6]. Therefore, early prediction of CSFP in patients with ACS could change the treatment strategy from PPCI to pharmaco-invasive strategy. The early use of thrombolytic therapy or glycoprotein inhibitors (GPI) could improve the patient's prognosis.

The study enrolled 100 patients with acute coronary syndrome in the period between January 2023 and June 2023. Coronary slow flow phenomenon without obstructive CAD was detected in 27 (27%) cases. Compared with patients with obstructive CAD, patients with CSFP had higher prevalence of DM, lower prevalence of dyslipidemia, lower levels of HDL, greater P wave dispersion,

lower QTc minimal values, and larger LA diameter. According to stepwise regression analysis, the most effective independent predictor of CSFP is P wave dispersion and the best co-predictors were DM and LA diameter.

The prevalence of CSFP without obstructive CAD observed in the study (27%) is high in comparison with the range reported in previous research. Studies have reported varying prevalence rates of CSFP, ranging from around 1% to 7% in patients with chest pain or ACS without significant CAD [1]. Selection of patients with ACS could be the cause of such increased burden.

The association between DM and CSFP has been explored in previous studies as well. Diabetes Mellitus has been identified as a potential risk factor for the development of CSFP. Numerous studies have revealed that patients have a greater prevalence of DM with CSFP compared to those with obstructive CAD[17]. The findings from our study, showing a higher prevalence of DM in CSFP patients, are in line with these previous observations. Guo and colleagues reported 66% prevalence of DM in patients with CSFP[18]. Furthermore, poor glycemic control was suggested by Elsanan and colleagues as strong predictor of CSFP in diabetic patients[19]. However, Xiaogang and colleagues reported only 16.2% prevalence of DM in patients with CSFP compared to 77% reported prevalence in our study [20]. We could explain such difference by the type of study population. Our study enrolled ACS patients not all elective patients undergoing coronary angiography as Xiaogang and colleagues' study.

The relationship between dyslipidemia and CSFP is less clear in the literature. While dyslipidemia is a well-established risk factor for CAD, its association with CSFP is not consistently reported. Xiaogang and colleagues reported about 55% prevalence of dyslipidemia in patients with CSFP which is in line with our study [20]. Furthermore, Sanghvi and colleagues reported that dyslipidemia was independent predictor of CSFP[21]. Some studies did not find significant difference in the prevalence of dyslipidemia between CSFP and other patients [22, 23]. In our study, the higher prevalence of dyslipidemia in CSFP patients suggests that lipid abnormalities may be a major contributor to CSFP.The lower levels of HDL observed in CSFP patients in our study are consistent with previous research. In line with our results, Yuksel and his colleague reported a significant negative correlation between HDL levels and TMF in patients with CSFP [24]. Patients with CSFP have been found to have low HDL levels, which are linked to poor lipid metabolism and reduced cardioprotective effects[24, 25]. In terms of clinical outcome, Aksoy and colleagues reported low HDL levels as a good predictor of cardiovascular mortality in patients with CSFP [26]. However, other studies did not find HDL as a predictor of CSFP[27, 28]. Therefore, it's important to note that the relationship between HDL levels and CSFP is complex, and more investigation is required to completely comprehend their correlation.

The increased P wave dispersion observed in CSFP patients in our study aligns with findings from previous investigations. P wave dispersion has been suggested as a marker of atrial electrical inhomogeneity and has been associated with arrhythmias, including atrial fibrillation. Studies have consistently reported increased P wave dispersion in CSFP patients supporting its

potential role as a predictor of CSFP[29, 30]. Furthermore, P wave dispersion was correlated with TFC in patients with CSFP [23].

The lower minimal values of QTc in CSFP patients in our study are in line with the commonly reported increased QTc dispersion in CSFP which may be due to inflammatory activity [31, 32]. Saya and colleagues reported a case of CSFP with recurrent arrhythmic syncopal attacks and increased QTc dispersion [33]. Prolongation of QTc has been associated with an increased risk of ventricular arrhythmias [34]. However, conflicting results have been reported, and some studies did not find significant change in QTc intervals in patients with CSFP [35, 36]. Further research is needed to clarify the relationship between QTc intervals and CSFP.

The larger LA diameter observed in CSFP patients in our study is consistent with previous findings. LA enlargement has been reported in CSFP patients and is associated with increased pressure or volume overload on the left side of the heart[29].Left Atrium enlargement is a common finding in various cardiovascular conditions, including heart failure and atrial fibrillation. However, Fallah and colleagues found that LA diameter, volume, and functions did not differ between patients with CSFP and other patients[37].

While the stepwise regression analysis in our study identified P wave dispersion as the best independent predictor of CSFP, previous research highlighted several other potential predictors as well. These predictors include inflammatory markers, endothelial dysfunction, platelet indices, microvascular dysfunction, and genetic factors. Among recent predictors, CSFP was recently predicted with soluble vascular cell adhesion molecule-1[38], systemic immune-inflammation index (SII, platelet × neutrophil/lymphocyte ratio)[39], tumor necrosis factor-like weak apoptosis inducer [40], neutrophil-to-lymphocyteratio[41], and eosinophil cationic protein[42]. Integrating these potential predictors with the findings from our study could provide a more comprehensive understanding of CSFP.

From the pathophysiological point of view, P wave dispersion and QTc dispersion were linked to inflammatory process mediated by interleukin-6 (IL-6) that could lead to fibrosis induceddisturbance of the intracardial conduction. CSFP is strongly associated with such inflammatory process [43].

Limitations

This study had some limitations being observational study, with low sample size, and incomplete laboratory assessment of inflammatory markers, endothelial dysfunction markers, and genetic factors. We recommend further larger studies to verify the ability of non-invasive parameters to accurately predict CSFP in patients with ACS.

CONCLUSIONS

Coronary slow flow phenomenon is not an uncommon finding among patients with ACS. Among non-invasive predictors, P wave dispersion was the best predictor CSFP in patients with ACS together with DM and LA diameter.

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REFERENCES

1. **Goel PK, Gupta SK, Agarwal A, Kapoor A**. Slow coronary flow: a distinct angiographic subgroup in syndrome X. Angiology. 2001; 52 (8): 507-14.
2. **Gibson CM, Cannon CP, Daley WL**, Dodge Jr JT, Alexander B, Marble SJ et al. TIMI frame

count: a quantitative method of assessing coronary artery flow. Circulation. 1996; 1;93(5):879-88.

3. **Tambe AA, Demany MA, Zimmerman HA, Mascarenhas E**. Angina pectoris and slow flow velocity of dye in coronary arteries—a new angiographic finding. Am Heart J. 1972; 1;84(1):66-71.
4. **Sezgin AT, Sgrc A, Barutcu I, Topal E, Sezgin N, Ozdemir R et al**. Vascular endothelial function in patients with slow coronary flow. Coron Artery Dis. 2003; 1;14(2):155-61.
5. **Nascimento BR, Brant BC, Marino LG, Passaglia AL**. Ribeiro, Implementing myocardial infarction systems of care in low/middle-income countries. Heart. 2019; 105 (1): 20-6.
6. **Claeys MJ, Coussement P, Pasquet A, De Backer T, De Pauw M.** Summary of 2018 ESC Guidelines on definition of myocardial infarction, myocardial revascularisation, cardiovascular disease during pregnancy and on arterial hypertension.ActaCardiol .2020; 75 (3): 179-85.
7. **Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H et al.** ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation. Rev EspCardiol (Engl Ed). 2017; 70 (12): 1082.
8. **Killip T, Kimball JT**. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. Am J Cardiol. 1967; 20 (4): 457-64.
9. **Prajapat L, Ariyarajah V, Frisella ME, Apiyasawat S, Spodick DH**. Association of P-wave duration, dispersion, and terminal force in relation to P-wave axis among outpatients. Ann Noninvasive Electrocardiol. 2007; 12 (3): 210-5.
10. **Indik JH, Pearson EC, Fried K, Woosley RL. Bazett, Fridericia QT.** correction formulas interfere with measurement of drug-induced changes in QT interval. Heart Rhythm. 2006; 3 (9): 1003-7.
11. **Demirtas AO, Urgun OD.** Can QT interval prolongation or dispersion detected in a positive exercise ECG test predict critical coronary artery disease? Arch Med SciAtheroscler Dis. 2019; 4: e7-e12.
12. **Wilson DJ, North N, Wilson RA**. Comparison of Left Ventricular Ejection Fraction Calculation Methods.Echocardiogr. 1998; 15 (8 Pt 1): 709-12.
13. **Dittrich HC, Pearce LA, Asinger RW, McBride R, Webel R, Zabalgoitia M et al.** Left atrial diameter in nonvalvular atrial fibrillation: An echocardiographic study. Stroke Prevention in Atrial Fibrillation Investigators. Am Heart J. 1999; 137 (3): 494-9.
14. **Yetkin E, Turhan H, Erbay AR, Aksoy Y, Senen K**. Increased thrombolysis in myocardial infarction frame count in patients with myocardial infarction and normal coronary arteriogram: a possible link between slow coronary flow and myocardial infarction. Atherosclerosis. 2005; 181 (1): 193-9.
15. **Vakili H, Sadeghi R, Tabkhi M, Safi M.** Corrected thrombolysis in myocardial infarction frame count and ejection fraction in patients undergoing primary percutaneous coronary intervention for myocardial infarction. ARYA Atheroscler. 2013; 9 (2): 134-9.
16. **Abdallah AA, Elrhman MA, Elshazly A, Bastawy I**, Relationship of serum vitamin D levels with coronary thrombus grade, TIMI flow, and myocardial blush grade in patients with acute ST-segment elevation myocardial infarction. Egypt Heart J. 2020; 72 (1): 84.
17. **Chang SM, Yu YT, Luan B, Hou AJ, Wang Y,** Predictive Value of Soluble Growth Stimulator Gene 2 Protein for Coronary Slow Flow/No-Reflow in ST-Elevation Myocardial Infarction Patients Receiving Percutaneous Coronary Intervention. J IntervCardiol. 2022; 2022: 9322460.
18. **Guo F, Dong M, Ren F, Zhang C, Li J, Tao Z et al.,** Association between local interleukin-6

levels and slow flow/microvascular dysfunction. J Thromb Thrombolysis. 2014; 37 (4): 475-82.

19. **Elsanan M, Tahoon I, Mohamed GI, ZeinElabdeen SG, Shehata IE.** Relationship between inflammatory markers and coronary slow flow in type 2 diabetic patients. BMC CardiovascDisord. 2023; 23 (1): 244.
20. **Zhu X, Shen H, Gao F, Wu S, Ma Q, Jia S et al.** Clinical Profile and Outcome in Patients with Coronary Slow Flow Phenomenon.Cardiol Res Pract. 2019; 2019: 9168153.
21. **Sanghvi S, Mathur R, Baroopal A, Kumar A,** Clinical, demographic, risk factor and angiographic profile of coronary slow flow phenomenon: A single centre experience. Indian Heart J. 2018; 70 Suppl 3 (Suppl 3): S290-S4.
22. **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. 2018; 70 (3): 405-9.
23. **Dogan SM, Yildirim N, Gursurer M, Aydin M, Kalaycioglu E, Cam F**. P-wave duration and dispersion in patients with coronary slow flow and its relationship with Thrombolysis in Myocardial Infarction frame count. J Electrocardiol. 2008; 41 (1): 55-9.
24. **Yuksel Y, Yildiz C.** [Evaluation of triglyceride-glucose index in coronary slow flow patients].Kardiologiia. 2023; 63 (5): 62-67.
25. **Kaplangoray MK, Toprak F, Basanalan A, Palice C, Aydin A, Demirkiran et al**. Investigation of the Relationship Between Triglycerides-Glucose Index and Coronary Slow Flow: A Retrospective Case-Control Study.Arq Bras Cardiol. 2023; 120 (6): e20220679.
26. **Aksoy SD, Oz M, Agirbasli M.** Predictors of Long-Term Mortality in Patients with Stable Angina Pectoris and Coronary Slow Flow.Medicina (Kaunas) 2023; 59 (4),5.
27. **Faramarzzadeh R, Fekrat F, Haghtalab A,** Evaluation of the relationship between clinical and laboratory risk factors in atherosclerosis patients with coronary slow flow: a case-control analysis. Egypt Heart J. 2023; 75 (1), 61.
28. **Ipek BU, Rukiye NA, Derya KA, Hande SE, Koray AM, Gokay NA**. Assessment of triglyceride/glucose index with respect to coronary slow flow.BratislLekListy. 2022; 123 (8): 585-8.
29. **Seyis S.** Effect of Coronary Slow Flow on Intrinsicoid Deflection of QRS Complex.Cardiol Res Pract. 2018; 2018: 2451581.
30. **Zhuang X, Peng Y, Bardeesi AS, Bardisi ES, Liao X, Luo C.** Vessel heterogeneity of TIMI frame count and its relation to P-wave dispersion in patients with coronary slow flow. J Thorac Dis. 2016; 8 (3): 476-81.
31. **Karahan MZ, Aktan A, Güzel T, Günlü S, KılıçR**.The effect of coronary slow flow on ventricular repolarization parameters. J Electrocardiol. 2023; 78: 39-43.
32. **Askin L, Tanrıverdi O.** Evaluation of index of cardio-electrophysiological balance in patients with coronary slow flow.ActaCardiol. 2022; 77 (4): 337-41.
33. **Saya S, Hennebry TA, Lozano P, Lazzara R, Schechter E**. Coronary slow flow phenomenon and risk for sudden cardiac death due to ventricular arrhythmias: a case report and review of literature.ClinCardiol. 2008; 31 (8): 352-5.
34. **Roden DM.** Keep the QT interval: it is a reliable predictor of ventricular arrhythmias. Heart Rhythm. 2008; 5 (8): 1213-5.
35. **Sucu M, Ucaman B, Ozer O, Altas Y, Polat E**. Novel Ventricular Repolarization Indices in Patients with Coronary Slow Flow. J Atr Fibrillation. 2016; 9 (3): 1446.
36. **Karaagac K, Yontar OC, Tenekecioglu E, Vatansever F, Ozluk OA, Tutuncu A et al.** Evaluation of Tp-Te Interval and Tp-Te/QT Ratio in Patients with Coronary Slow Flow Tp-Te/QT Ratio and Coronary Slow Flow. Eurasian J Med 2015; 47 (2): 104-8.

37. **Fallah F, Narimani S, Yarmohammadi S, Hosseinsabet A, Jalali A.** Is Left Atrial Function Affected by Coronary Slow Flow? A Two-dimensional Speckle-Tracking Echocardio-graphic Study. J CardiovascEchogr. 2017; 27 (4): 121-5.
38. **Zhu Q, Zhao C, Wang Y, Mu L, Li X, Qi Y et al.** Soluble Vascular Cell Adhesion Molecule-1 as an Inflammation-Related Biomarker of Coronary Slow Flow. J Clin Med 2023; 12 (2), 543.
39. **Dai XT, Kong TZ, Zhang XJ, Luan B, Wang Y, Hou AJ**. Relationship between increased systemic immune-inflammation index and coronary slow flow phenomenon. BMC CardiovascDisord. 2022; 22 (1): 362.
40. **Tatlisu MA, Atici A, Ozcan FB, Kirac E, Baycan OF, Caliskan M.** Can plasma TWEAK levels predict coronary slow flow in patients with chronic kidney disease? Am J Med Sci. 2022; 364 (5): 595-600.
41. **Yang Z, Yuan J, Cui J, Guan H, Qiao S.** Association of the lymphocyte-to-monocyte ratio, mean diameter of coronary arteries, and uric acid level with coronary slow flow in isolated coronary artery ectasia. BMC CardiovascDisord. 2021; 21 (1),1-7.
42. **Soylu K, Akcay M, Aksan G, Gedikli O, Gokay N**. Eosinophil cationic protein: A new diagnostic biomarker in coronary slow flow phenomenon.BratislLekListy. 2021; 122 (3): 212-6.
43. **Okutucu SK, Aytemir A, Oto.** P-wave dispersion: What we know till now? JRSM Cardiovasc Dis. 2016; 5: 2048004016639443.

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