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ABSTRACT

Coronary Slow Flow Phenomenon (CSFP) is characterized by the slow flow of contrast in one or more epicardial coronary vessels without evidence of coronary artery stenosis during coronary angiography procedures. CSFP is fairly common at the time of elective angiography with an incidence of around 7% and accounts for about 4% of hospitalized unstable angina cases. Coronary angiography is currently still the only effective way to detect CSFP, but this procedure is an invasive procedure with high costs, there is a risk of allergy to contrast. Electrocardiography (ECG), as a widely available, inexpensive, and simple modality is felt to be an attractive alternative in early detection of this abnormality. The ECG parameters on CSFP discussed in this study include; p-wave dispersion, QT interval dispersion, QRS intrinsic (Tpeak-Tenddeflection duration), and QRS fragmentation. Further studies are needed on the ECG image in CSFP so that in the future ECG can be a cheaper and non-invasive diagnostic modality for CSFP compared to coronary angiography.

1. Introduction

Coronary slow flow phenomenon (CSFP) was first described by Tambe in 1972,[1] characterized by the slow flow of contrast in one or more epicardial coronary vessels without evidence of coronary artery stenosis during coronary angiography procedures. CSFP has the same clinical symptoms as other coronary heart diseases, namely typical chest pain, both during activity and at rest. Up to 80% of patients experience recurrent complaints with a rehospitalization rate of up to 20% within a 2-year follow-up period.[3] Clinical manifestations of CSFP increase the rate of serious morbidity in the form of the acute coronary syndrome. CSFP is quite often found during elective angiography with incidence rates ranging from 7% and accounting for about 4% of cases of unstable angina in the hospital.[1,2]

The diagnosis is determined angiographically CSFP marked by a slow flow of contrast that can be calculated using TIMI (The Thrombolysis in Myocardial Infarction) Frame Count, TFC. The TFC correction

method was first introduced by Gibson in 1996.[4] To calculate TFC, the first frame starts when the contrast fills the coronary ostial and the last frame when the contrast reaches the end of the blood vessel. Patients are said to be CSFP if the number of frames is> 27 in one or more coronary arteries.[4]This Gibson method has been widely used in research for more than 20 years.

Coronary angiography is currently still the only effective way to detect CSFP, but this procedure is an invasive procedure with high costs, there is a risk of allergy to contrast. Therefore, an alternative test that is cheap, simple, and feasible is needed to diagnose CSFP at an early stage. Non-invasive examination using electrocardiography (ECG) has long been used to differentiate normal patients and patients with acute coronary syndromes such as stable angina pectoris, NSTEMI, and STEMI with clear characteristics. However, few assess the characteristics of the ECG in making the initial diagnosis of CSFP. So it is important to know the ECG characteristics of CSFP patients to diagnose CSFP early so that further coronary angiography is not required. Considering that EKG devices are widespread in first-level health facilities and all regional hospitals, there must be ECG criteria in establishing the ECG diagnosis of patients with CSFP to make it easier for primary care physicians to distinguish CSFP from normal patients and patients with obstructive coronary heart disease.

The exact pathogenesis of CSFP remains unclear but is thought to be multifactorial including vascular morphological abnormalities such as fibromuscular thickening, myofibril hypertrophy, hidden atherosclerosis, endothelial dysfunction, and compression of extravascular structures in the microvascular tissue of the myocardium.[5] Most of the evidence points to an important role of inflammation in the pathogenesis of this phenomenon.[6-9] Many mediators and markers of inflammation have been reported associated with this condition, suggesting the presence of a proinflammatory process as a cause as well as a consequence of the phenomenon.[10,11] Based on the above, the authors are interested in knowing the description of ECG characteristics such as P wave dispersion, QT interval dispersion, QRS intrinsic deflection, T peak T end interval, and fragmented QRS in the diagnosis of CSFP.

Relationship between ECG and CSFP characteristics

Effect of ischemic conditions on depolarization and repolarization of the myocardium

ECG recordings describe the electrical activity of the myocardium, namely depolarization, and repolarization. At the cellular level, myocardial depolarization depends on the excitability of the myocardium which is regulated by multiple ion flows at different phases of the action potential cycle.[12] Under normal conditions, there is a difference in electrical potential between the intracellular and extracellular environments of the myocardium of -90 mV. This is called the resting membrane potential. This value is maintained by the role of the Na-K ATPase pump which continuously pumps potassium in and sodium out of the myocardium cells. In the action potential cycle, the phase of the resting membrane potential is known as phase 4.

The depolarization of a myocardial cell is triggered when a depolarizing current reaches the cell. It begins with the opening of the voltage-gated sodium channel, resulting in a rapid current of sodium entering the intracellular which causes the membrane potential difference to change up to 45 mV. This process in the action potential cycle is known as phase 0. The next stage is characterized by the closure of the voltage-gated sodium channel which terminates the inflow of sodium. When the membrane potential difference reaches +20 mV, potassium channels open, resulting in a passive extracellular flow of potassium. This stage is the beginning of the repolarization period and is known as phase 1. The next stage is marked by the 'plateau' phase of the repolarization process. Where the outflow of potassium is balanced by the inflow of calcium through calcium channels. This stage is known as phase 2. The final stage is characterized by the closure of the calcium channels. The extracellular flow of potassium occurs without any compensatory inflow of ions causing repolarization in this phase to occur rapidly. This stage is known as phase 3.

Under ischemic conditions, there are several cellular changes associated with the electrical activity

of the myocardium. Ringborn suggested several basic disorders that occur in ischemic myocardium, namely the function of the Na-K ATPase pump is disrupted which makes it difficult to maintain a normal resting membrane potential, lactate accumulation both intracellular and extracellular, and extracellular potassium accumulation. These things will have implications for the slow initial upstroke of depolarization (phase 0). This is because at a certain stage the voltage-gated Na channel *is* disturbed, its role in initiating depolarization is replaced by calcium influx. In addition, the amplitude of the action potential decreases, and the repolarization phase begins earlier.[13]

ECG on coronary slow flow phenomenon

Coronary slow flow phenomenon at a certain stage will result in an ischemic condition of the myocardium which will affect the process of depolarization and repolarization of the myocardium. These things can be detected by ECG examination.[14] The study of ECG findings in the coronary slow flow phenomenon is one of the active research areas. Several ECG markers representing atherosclerosis, impaired myocardial conduction, and abnormal repolarization of the myocardium were studied for their presence in the coronary slow flow phenomenon. The ECG markers include; P wave dispersion, QT interval dispersion, QRS intrinsic deflection, T peak T end interval, and fragmented QRS.

P-wave dispersion

P-wave dispersion is defined as the difference between the longest P wave duration and the shortest P wave duration on a 12 lead ECG. P wave dispersion is receiving more attention and has been studied in a wide range of clinical situations in both cardiovascular and non-cardiovascular diseases. Ocutucu et al. revealed that the normal limit for p-wave dispersion is ≤ 36 ms.[15]

P-wave dispersion was used to evaluate the inter and intra-atrial conduction times and the homogeneity of impulse propagation from the SA node. Disturbance of this is the electrophysiological character of atrial susceptibility to atrial fibrillation. From several studies, it was found that in the coronary slow flow phenomenon there is an increase in inflammatory markers, oxidative stress, increased sympathetic activity, and myocardial ischemia. They have an important role in the pathogenesis of atrial fibrillation.[16] Eshraghi et al. In his study, the P-wave dispersion was significantly longer in the CSFP group compared to the control group, 39.74 ± 17.48 m/s and 19.50 ± 8.54 m/s with $p < 0.001$, respectively.[17] In another study, Seyis also found that the P-wave dispersion was significantly longer in the CSFP group than the control group, namely 43.18±7.54 m/s and 20.71±3.94 m/s with $p<0.001$.[18]

Dilaveris et al. reported an increase in P wave duration and P wave dispersion in patients with angina episodes. Yilmaz et al. reported that P wave dispersion was greater in CAD patients with >50% stenosis than in the control group.[15] Turkmen et al. found an association between prolonged P wave duration and increased P wave dispersion in patients. with Coronary Slow Flow Phenomenon. This finding is thought to be due to microvascular ischemia and/or autonomic disturbance in Coronary Slow Flow Phenomenon patients.[19] Khaled Mahmoud found that there is a correlation between P-wave dispersion and TIMI Frame Count of the left coronary artery, but this correlation was not found in TIMI.frame count Right coronary artery.[16] Gunes et al. reported a decrease in P wave duration and P wave dispersion in Coronary Slow Flow Phenomenon patients receiving nebivolol therapy.[20]

QT interval dispersion

The QT interval on ECG images has long been known to represent ventricular electrical activity, both depolarizing and repolarizing. QT interval analysis is often used to assess a patient's ventricular arrhythmogenic risk, where QT prolongation is known to be associated with an increased risk of sudden cardiac arrest. QT intervals. A prolongation of the QT interval can also be an early marker of transmural ischemia. Kenigsberg in his study of patients undergoing elective balloon angioplasty found that inflating the intracoronary balloon for 20 seconds found a corrected QT prolongation interval in all patients.[21,22]

One of the new findings in QT interval observations began in 1985, where Mirvis reported the presence of significant spatial variation of the QT interval in normal individuals and patients with acute myocardial infarction. In 1990, Campbell et al. suggested that differences in the duration of the QT interval between leads on a 12-lead ECG may reflect differences in refractory periods between regions of the myocardium, and are associated with an increased risk of arrhythmias. This difference in the duration of the QT interval is known as the dispersion of the QT interval.[18] Eshraghi et al. In his study, the dispersion of the QT interval was significantly longer in the CSFP group compared to the control group, 76.17±35.23 m/s, and 39.25±19.26 m/s, respectively, with $p<0.001$.[17]

Wave dispersion QT was defined as the difference between the longest QT duration and the shortest QT duration on the 12 lead ECG. The measurement of the QT interval starts from the beginning of the QRS wave to the end of the T wave. If there is a difference in heart rate when recording the ECG between leads, it is necessary to correct the QT interval according to the heart rate so that the dispersion of the QT interval is corrected. The QTc dispersion value $\leq$ 50 ms is considered a normal value. The QT interval correction is generally performed using Bazzet's formula, namely $QT/\sqrt{RR}$.[21]

QT wave dispersion in conditions of myocardial infarction or ischemia has been studied extensively, both as a marker of myocardial ischemia or infarction or as a predictor of post-ischemic ventricular arrhythmias or myocardial infarction. Higham and colleagues found that there was an increase in the dispersion of QT waves in patients with acute myocardial infarction. Moreno and colleagues found that there was a decrease in the value of QT wave dispersion in STEMI patients who were successfully reperfused with thrombolytics. Meanwhile, Schneider and colleagues in their study using positron emission tomography (PET) found that the value of QT wave dispersion after myocardial infarction was determined from the number of post-infarction myocardium.[22,23]

QT wave dispersion was also studied in Coronary Slow Flow Phenomenon. Eshraghi et al. reported that the QT wave dispersion in patients with coronary slow flow phenomenon was significantly higher than in patients with normal coronary arteries without coronary slow flow. Khaled Mahmoud reported that the corrected QT dispersion value has a strong positive correlation with the TIMIframe count (TFC) LAD, LCx and TFC mean, but has no significant correlation with TFC RCA.[16,17]

Duration of QRS Intrinsic deflection

Intrinsicoid deflection (ID) QRS time or also known as R wave peak time (RWPT) is the time of the beginning of the QRS wave to the peak of the R or R' wave. This parameter describes the spread of the depolarization wave from the endocardium to the epicardium. Riera revealed the normal value for the right ventricular intrinsicoid duration was $\leq$ 35 ms, measured at v1 and v2. While the normal value for the left ventricle is $\leq$ 45 ms measured at v5 and v6.[24] In his research, Seyis found that the duration of the QRS intrinsic deflection in lead V1 was significantly longer in the CSFP group compared to the control group, which was 45.85, respectively. ±5.75 ms and 27.44±4.38 ms, with $p<0.001$. Likewise for lead V6 65.61±5.86 ms and 38.58±28.89 ms with $p<0.001$.[25]

The duration of QRS intrinsic deflection is known to increase in conditions of diastolic dysfunction where there is an increase in wall pressure. ventricle during diastole. It is known from several studies studying diastolic dysfunction with tissue Doppler imaging (TDI) that there was a significant relationship between the duration of the intrinsicoid deflection and the parameters of diastolic dysfunction.[18]

It was found that the QRS ID duration can also be a predictor of the incidence of no-reflow in STEMI cases. undergoing primary percutaneous intervention (primary PCI). Bendary and colleagues in their study of STEMI patients undergoing primary PCI found that QRS ID duration values >46 ms could predict the incidence of no-reflow. with a sensitivity of 79.5% and a specificity of 86.9%.[26] Seyis studied the effect of slow coronary flow on the duration of QRS intrinsicoid deflection. It is suspected that diastolic dysfunction is one of the structural factors that cause slow coronary flow. Seyis found a positive linear correlation between the duration of the deflection intrinsicoid V1 and V6

with TFC LAD, Cx, RCA, and TFC mean.[18]

The duration of T_{peak}-T_{end}

Contrary to the direction of the wave of depolarization, the ventricular repolarization wave stems from the epicardium toward the endocardium, where the last cells to depolarize are the *M cells* in the endocardium. The peak of the T wave represents the end of repolarization of the cells located in the epicardium, while the end of the T wave represents the end of repolarization of the M cells. Length T_{peak}-T_{end} describes transmural repolarization of the myocardium. Length T_{peak}-T_{end} of the T wave is calculated from the peak to the end of the T wave.[27]

Length T_{peak}-T_{end} has been widely studied and found to have an association with the incidence of ventricular arrhythmias in various conditions. Antzelevitch found a link between long-duration T_{peak}-T_{end} with the incidence of sudden cardiac arrest in patients with Brugada ECG patterns. Dinshaw found an association between duration T_{peak}-T_{end} with the incidence of ventricular arrhythmia in cardiomyopathy hypertrophy. Chua and colleagues found $Tvalue_{peak}$-T_{end} corrected independent predictive factors on the risk of the incidence of sudden cardiac arrest, in which the value of> 90 ms had a significant correlation with the incidence of sudden cardiac arrest.[28-30]

Some studies also studied duration $T_{peak}T_{late}$ in conditions of myocardial infarction or ischemia. Conlon and colleagues found an increase in the duration of$T_{thepeak}T_{final}$ in patients with coronary artery ataxia. Kaneko and colleagues found an inverse correlation between the duration of moderate strength T_{peak}-T_{end} with the degree of myocardial blush in patients with STEMI undergoing primary PCI.[31,32]

Length T_{peak}-T_{end} also studied the condition of the coronary slow flow phenomenon. Zehir and friends, as well as Tenekecioglu and colleagues, found the lengthening duration T_{peak}-T_{end} and T_{peak}-T_{end} / QT in patients with Coronary Slow Flow Phenomenon compared to patients with normal coronary arteries.[29,33]

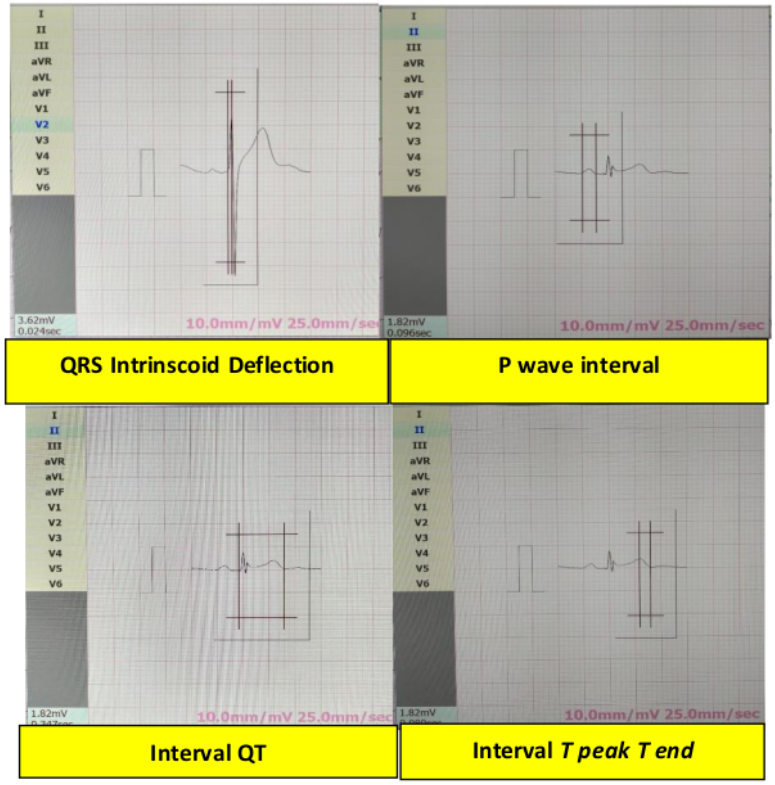

Figure 1: Intrinsic QRS deflection, P wave interval, QT interval, and interval T peak T end.

Fragmented QRS complex

Fragmented QRS is a new ECG sign that reflects impaired ventricular conduction around the scar tissue of the myocardium. A fragmented QRS was defined (fQRS) by the presence of an additional R wave (R'), or 'notching' in the R or S wave, or the presence of more than one R' in at least two leads corresponding to one of the major coronary arteries. To be classified as a fragmented QRS, the QRS width must be < 80ms and not meet the classic LBBB or RBBB picture.[12,34]

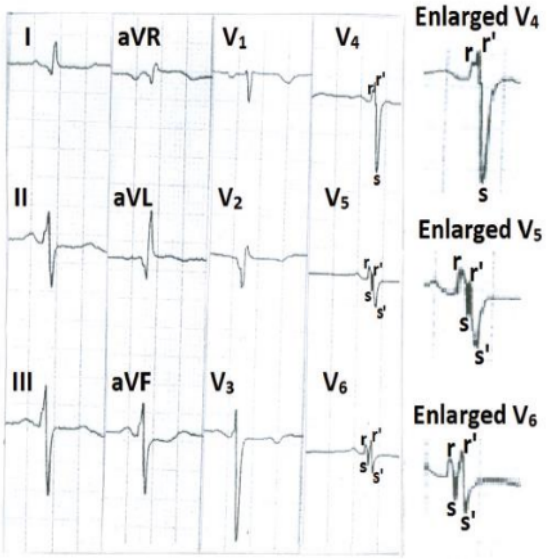

Figure 2: QRS fragmented at V4-V6[34]

Fragmented QRS has long been a marker of long-standing myocardial infarction and a predictor of arrhythmogenicity. in patients with Brugada syndrome and nonischemic cardiomyopathy. In the condition of acute myocardial infarction, the presence of fQRS is a strong predictor of cardiovascular mortality.[12,34]

Yilmaz and colleagues in their study found that the presence of fQRS in patients with Coronary Slow Flow Phenomenon was significantly higher (31.7%) compared to patients with normal angiography and no coronary slow flow (6.8%).[34]

2. Conclusion

Recognition of signs of coronary slow flow phenomenon An EKG may save a person from invasive tests such as coronary angiography. However, at this time further studies are needed regarding the ECG picture in the coronary slow flow phenomenon so that an ECG scoring system can be produced for this disorder that has good reliability.

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