

**Review Article**

# Advances in the Value of Cardiac Troponin and ST Segment Depression in Predicting Coronary Artery Disease in Patients with Paroxysmal Supraventricular Tachycardia

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**Abstract:** Paroxysmal supraventricular tachycardia (PSVT) is similar to coronary artery disease (CAD) in that it is often accompanied by chest pain, ST segment depression or elevated cardiac troponin in the electrocardiogram (ECG). Studies have shown that only 20%-40% of these patients are diagnosed with CAD by coronary angiography and even lower in foreign countries. Therefore, a noninvasive and simple method is urgently needed in practice to determine the high-risk population of PSVT patients with CAD, so as to avoid excessive examination and waste of medical resources. Cardiac troponin and ECG are favored in clinic because of their simple, economical and non-invasive detection methods. By analyzing the mechanism, it is concluded that elevated troponin and ST segment depression are of low predictive value for CAD diagnosis in PSVT patients, and it is necessary to further evaluate the risk factors for cardiovascular disease in these patients to determine whether coronary angiography is required. However, it is worth noting that ST segment depression in PSVT patients may be associated with coronary microcirculation lesions. Future research should also start from the pathogenesis, symptoms and characteristics of coronary microcirculation lesions to find more similarities and differences with PSVT combined with ST segment depression. This paper may contribute to a better understanding of troponin elevation and ST segment depression in PSVT patients.

**Keywords:** Paroxysmal Supraventricular Tachycardia, Cardiac Troponin, ST Segment Depression, Coronary Artery Disease

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## 1. Introduction

Paroxysmal supraventricular tachycardia (PSVT) is a common arrhythmia [1]. Oreiarena retrospectively analyzed the cases diagnosed with PSVT from 1991 to 1993 in the Marshfield Epidemiology Study area in 1998 and concluded that Person-year incidence of PSVT was 36/100,000 [2, 3]. In addition, PSVT is more common in adolescents, and the incidence in females is about twice as high as that in males [3], which may be accompanied by chest pain or elevated cardiac troponin and/or depressed ST segment of ECG when seizure. Due to the rapid increase of ventricular rate and increase of myocardial oxygen consumption during the PSVT, the myocardial cells can present ST-segment

depression on the ECG and/or elevated cardiac troponin relative to ischemia and hypoxia. The above ECG and cardiac troponin are more obvious when combined with CAD. Therefore, most of these patients will choose to undergo coronary angiography to determine whether there is accompanied with CAD in clinical practice. Some reports have shown that the incidence of CAD in patients with PSVT combined with elevated cardiac troponin and/or depressed ST segment of ECG is 20%-40% in China, and even only 4% in foreign countries [4]. It is still controversial whether coronary angiography should be performed in patients with PSVT combined with elevated cardiac troponin and/or ST-segment depression. As one of the important cardiac examination methods, troponin and ECG are widely used in clinical because of their simplicity, economy and

non-invasive diagnosis. The paper discusses the predictive value of cardiac troponin and ST segment depression on CAD in patients with PSVT.

## 2. Predictors and Mechanisms

### 2.1. Cardiac Troponin

Cardiac troponin is an important serum marker and is the basis for diagnosis, risk assessment, prognosis, and determination of antithrombotic and vascular remodeling strategies. The European Heart Association proposed that for patients with normal baseline cTnI, an increase of cTnI exceeding 5 times of the 99th percentile of the upper reference value is defined as myocardial infarction [5].

Murer [6] studied 473 PSVT who treated with radiofrequency ablation, including 326 patients underwent coronary angiography examination at the same time. The prevalence of significant CAD was 14%. Among 67 patients who accepted the troponin test there were 36 patients with elevated cardiac troponin, the ratio was 54% (36/67 patients). Cardiac troponin was elevated in 83% (10/12 patients) with significant CAD and in 47% (26/55 patients) without CAD. He argues that the low prevalence of CAD in PSVT questions the role of routine invasive coronary angiography during radiofrequency ablation. He thinks that cardiac troponin cannot accurately exclude or confirm CAD in these patients. Multiple studies [6-12] have concluded that cardiac troponin does not predict CAD, while is more likely to add additional cardiac tests, which may lead to unnecessary medical costs and invasive tests. Chow [13] analyzed a study of 78 patients with PSVT, and 29 patients (37.2%) had an elevated cardiac troponin level of  $\geq 0.06$  ng/ml. After multivariable adjustment, the presence of elevated cardiac troponin with PSVT was associated with increased risk of the primary endpoint of death, myocardial infarction, or cardiovascular rehospitalization ([HR] 3.67,  $P=0.02$ ). He indicated that a mild increase in troponin in patients with PSVT is common and associated with an increased risk of future cardiovascular events. The reversal of most previous findings may be due to the fact that the average age of the study population was older than 60 years, whereas previous studies focused on younger people with fewer risk factors for coronary heart disease.

There are many causes of elevated troponin, including non-cardiac factors such as chronic kidney failure, cerebrovascular accident, acute pulmonary embolism, chronic obstructive pulmonary disease, acute non-cardiac critical illness, and vigorous exercise. Cardiac factors in addition to acute coronary syndromes also include acute pericarditis, acute myocarditis, and tachycardia [14]. So it should be noted that elevated troponin does not mean apoptosis of cardiomyocytes [15], such as intense exercise can cause a rise in troponin, the rise usually drops to normal within 24 to 48 hours. Previous studies have shown increased troponin levels in STEMI on day 1 and peak on day 1 of STEMI, while peak troponin levels in PSVT patients

occurred within 8 to 16 hours [16]. To find the reason for PSVT associated with increased troponin, from the pathophysiological point of view, troponin is composed of three subunits, cTnC, cTnI and cTnT, which interact with tropomyosin to form a tropomyosin-troponin complex. This complex forms the skeleton of the striated muscle and regulates the excitation-contraction coupling of the heart. If myocardial cells are damaged by acute ischemia or other mechanisms, these proteins are released into the blood stream, known as troponin leakage. PSVT or any other tachycardia can lead to an increase in troponin by increasing the oxygen consumption of the myocardium, a phenomenon that may be caused by temporary myocardial cell damage due to hemodynamic damage, resulting in the release of troponin into the bloodstream [14]. Duration of PSVT, mean arterial blood pressure, and end-diastolic left ventricular pressure all had effects on coronary perfusion, which may lead to troponin leakage [17].

Part of scholars consider the troponin will be higher, owing to the doesn't match between the oxygen supply and demand, patients without apparent restrictive epicardial narrow, and the increasing demand of oxygen when PSVT, atrial fibrillation and other type of quick attacks occurred, which eventually lead to an elevated troponin [18].

It has also been reported that the potential factors of elevated troponin in PSVT may be related to occulted epicardial CAD, microvascular coronary artery disease and endothelial function damage, as well as myocardial cell damage caused by local and systemic catecholamine release [19].

Elevated troponin levels are common in PSVT and cannot be used to diagnose CAD, but may point to mild myocardial injury, so it is necessary to monitor troponin levels clinically. In addition, the time interval of troponin measurement should be shortened to further clarify the characteristics and mechanism of troponin elevation in PSVT.

### 2.2. ST Segment Depression

Traditionally, ST segment depression in patients with CAD is due to coronary atherosclerosis, stenosis and obstruction, which leads to myocardial ischemia and hypoxia, resulting in myocardial cell damage, and thus manifesting as ST depression on ECG. However, the current study [8, 20] unanimously believes that the incidence of CAD in patients with ST segment depression during PSVT is low, and the predictive value of ST segment depression in the diagnosis of CAD is low. The idea was further confirmed by Dorenkamp [4]. He studied 114 patients with PSVT, of whom ST segment depression accounted for 61%, and the positive rate of CAD after coronary angiography was only 4%. He believed that ST segment depression was not a specific indicator for predicting CAD in patients with PSVT. The reason may be that the ST segment depression mechanism in CAD is different from that in PSVT. At present, the mechanism of ST segment depression in PSVT patients has not been clarified, which may be related to the following reasons.

When PSVT attacks, a sudden increase in the heart's work causes an increase in metabolic demand, which in turn leads to impaired function of abnormal small blood vessels and thus impaired coronary blood flow reserve. Leaving tissue in a state of relative ischemia, especially in the myocardium's subendocardial layer, may exacerbate the ischemia with shortened diastolic period, resulting in reduced coronary blood flow [21].

Shorter cardiac cycles were associated with more pronounced ST segment depression, and faster ventricular rate showed more pronounced ST segment depression than slower in PSVT [22], which may be related to the repolarization process rather than coronary involvement. For example, Lin [23] believes that the repolarization process in PSVT is mainly caused by hemodynamic changes, which leads to ST segment depression.

In patients with atrioventricular reentrant tachycardia (AVRT) [24, 25], atrial retrograde activation takes place through the bypass, in a very short time conduction, retrograde atrial depolarization of the negative on the ECG P wave, and ventricular bipolar overlap, it might give a person with ST segment depress impression, but it could not be used to explain the atrioventricular nodal reentrant tachycardia (AVNRT). Because the atrium-ventricular interphase is short, atrial depolarization occurs simultaneously with ventricular depolarization, rather than simultaneously with ventricular repolarization.

In addition, Gulec suggested that ST segment depression may be associated with cardiac autonomic nerve dysfunction and the change in the order and duration of atrial inverse transmission [25].

I believe that in patients with PSVT combined with ST segment depress, there may be "coronary microcirculation dysfunction" which refers to the decreased coronary blood flow reserve caused by functional microvascular lesions in the absence of obstructive epicardial disease. "Coronary microcirculatory dysfunction" was first proposed by Cannon and Epstein in 1988. International diagnostic criteria [26] including symptoms of myocardial ischemia, no obstructive CAD (coronary angiography or CTA prompt extent of coronary stenosis  $< 50\%$  or fractional flow reserve  $> 0.80$ ), the objective evidence of myocardial ischemia (stress caused by chest pain and/or ischemia ECG changes), evidence of coronary microvascular dysfunction (fractional flow reserve acuities were between 2.0 and 2.5 or less). Patients with PSVT combined with ST segment depression had both negative results of coronary angiography and changes in ischemic ECG, and had myocardial ischemia symptoms such as chest pain and palpitations. Coronary microcirculatory dysfunction is often neglected because of their complex etiology and diverse clinical manifestations. One of the reasons may be related to the structural and functional changes of coronary microcirculation caused by endothelial dysfunction and smooth muscle dysfunction [27], which is also consistent with previous studies.

Therefore, ST segment depression in PSVT is not only related to myocardial ischemia and injury. Thus ST segment

depression in PSVT is not a reliable predictor of coronary heart disease. However, it is worth noting that ST segment depression in PSVT patients may be associated with coronary microcirculation lesions. Future research should also start from the pathogenesis, symptoms and characteristics of coronary microcirculation lesions to find more similarities and differences with PSVT combined with ST segment depression. In addition, previous studies did not indicate whether the specific type of ST depression was related to the prediction of CAD in PSVT patients. For example, the predictive value of the extent and duration of ST segment depression on PSVT complicated with CAD still needs to be further explored in future studies.

### 3. Conclusion and Prospect

In summary, elevated troponin and ST segment depression are of low predictive value for the diagnosis of CAD in PSVT patients, and it is necessary to further evaluate the risk factors for cardiovascular disease in these patients to determine whether coronary angiography is required. At present, the mechanism of troponin elevation in PSVT patients is not yet clear, while the relationship between CAD in PSVT patients and the extent and duration of ST segment depression still needs further study, especially whether ST segment depression in PSVT patients is related to coronary microcirculation lesions needs more theoretical and experimental demonstration.

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