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Case Report

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Acute Severe Chest Pain with ST-Segment Elevation in Inferior Leads During Pharmacological Cardioversion of Paroxysmal Supraventricular Tachycardia: A Case Report

Pan Pan[1,2], Yi Qin Xia[1,2], Bin He[1,2], Yu Cao[1,2*]

[1]*Department of Emergency Medicine, West China Hospital, Sichuan University, Chengdu, Sichuan Province, 610041, China*

[2]*Emergency Department, West China Hospital, West China School of Medicine, Sichuan University, Chengdu, China*

Corresponding Author: **Yu Cao**

Address: Emergency Department, Sichuan University West China Hospital, No. 37, Guoxue Alley, Wuhou District, Chengdu, Sichuan Province, 610041 China; Email: caoyu@wchscu.cn

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Abstract

Paroxysmal supraventricular tachycardia (PSVT) is a frequently encountered arrhythmia in emergency departments, significantly affecting cardiac function and hemodynamics. The sudden onset of severe chest pain with accompanying ST-segment elevation on the electrocardiogram (ECG) during pharmacological cardioversion is rare but requires immediate intervention with coronary vasodilators, anticoagulants, and antiplatelet agents. Continuous ECG monitoring and assessment of cardiac biomarkers are essential. In facilities with appropriate resources, urgent coronary angiography is recommended to evaluate coronary anatomy, blood flow, and collateral circulation. Due to the rarity of this presentation, we report this case as a reference for clinical diagnosis and management.

Keywords

Paroxysmal Supraventricular Tachycardia; Diltiazem; Coronary Spasm-Induced Angina; Coronary Angiography

Case Report

A 38-year-old female patient was admitted on April 1, 2024, due to recurrent episodes of paroxysmal palpitations lasting over three years, with a recent episode accompanied by chest tightness that persisted for 20 hours. Three years ago, she experienced palpitations without identifiable triggers, along with fatigue and occasional chest tightness, but no chest pain, nausea, dyspnea, or syncope. Each episode lasted approximately one hour, and electrocardiography (ECG) performed at an external hospital indicated "paroxysmal supraventricular tachycardia" (PSVT), which resolved after pharmacological treatment. Throughout her condition, she experienced approximately 5 to 6 episodes, each successfully treated with medication to restore sinus rhythm. She presented to the emergency department for the recurrence of palpitations and chest tightness. Her medical history included a bladder tumor diagnosed three years ago, for which she underwent transurethral resection two and one year ago, as well as an ovarian cyst diagnosed three years ago, which was surgically removed two years ago.

Upon admission, her physical examination revealed the following vital signs: temperature 36.8 °C, respiratory rate 20 breaths per minute, pulse 176 beats per minute, blood pressure 96/73 mmHg (1 mmHg = 0.133 kPa), and oxygen saturation 100%. Lung

percussion was clear bilaterally, with normal breath sounds and no audible rales. Cardiac borders were within normal limits, exhibiting a regular rhythm with no murmurs auscultated across any valve areas. The abdomen was soft, with no shifting dullness, and limb muscle strength was graded as V, with no edema observed in the extremities.

Arterial blood gas analysis indicated a partial pressure of carbon dioxide ($PaCO_2$) of 27.2 mmHg, an oxygen saturation of 99.3%, a partial pressure of oxygen (PaO_2) of 113.6 mmHg, and a lactate level of 2.7 mmol/L (reference range: 0.5–1.6 mmol/L). Cardiac biomarkers were as follows: creatine kinase-MB (CK-MB) 9.65 ng/mL, myoglobin 31.90 ng/mL, pro-B-type natriuretic peptide (pro-BNP) 3566 ng/L, and troponin T 119.0 ng/L. Routine blood tests, a disseminated intravascular coagulation (DIC) panel, liver and renal function tests, and electrolyte levels showed no significant abnormalities. The admission ECG indicated PSVT (**Fig-1**).

The patient was immediately placed on ECG monitoring and received oxygen supplementation, followed by a slow intravenous injection of 10 mg of diltiazem diluted in 0.9% normal saline. After administering approximately 7 mL, her heart rate decreased to 70-75 beats per minute, while her blood pressure dropped to 79/54 mmHg. The patient then reported severe, persistent chest pain described as a crushing sensation in the precordial region, accompanied by chest tightness, profuse sweating, and nausea, but without vomiting, dizziness, headache, or abdominal pain. An urgent ECG indicated sinus rhythm with ST-segment elevation in leads II, III, and aVF, as well as ST-segment depression in leads V1-V6 (**Fig-2**). Rapid bedside testing for cardiac biomarkers returned positive results. Acute Coronary Syndrome (ACS) was suspected, and the patient was immediately treated with 300 mg of aspirin, 300 mg of clopidogrel, 20 mg of atorvastatin, and low-molecular-weight heparin for anticoagulation. Emergency coronary angiography (**Fig-3**) revealed no structural abnormalities or coronary artery stenosis. Following treatment, the patient's condition improved significantly, and she was discharged. A follow-up call one-month post-discharge indicated no recurrence of tachycardia or symptoms of angina.

Discussion

PSVT is one of the most common arrhythmias encountered in clinical practice, characterized by sudden, brief episodes of tachycardia. The pathophysiology of PSVT is typically associated with reentrant activity in the atrioventricular (AV) node. Calcium channel blockers (CCBs), such as diltiazem, are frequently used for cardioversion due to their efficacy and favorable safety profiles [1,2]. CCBs function by

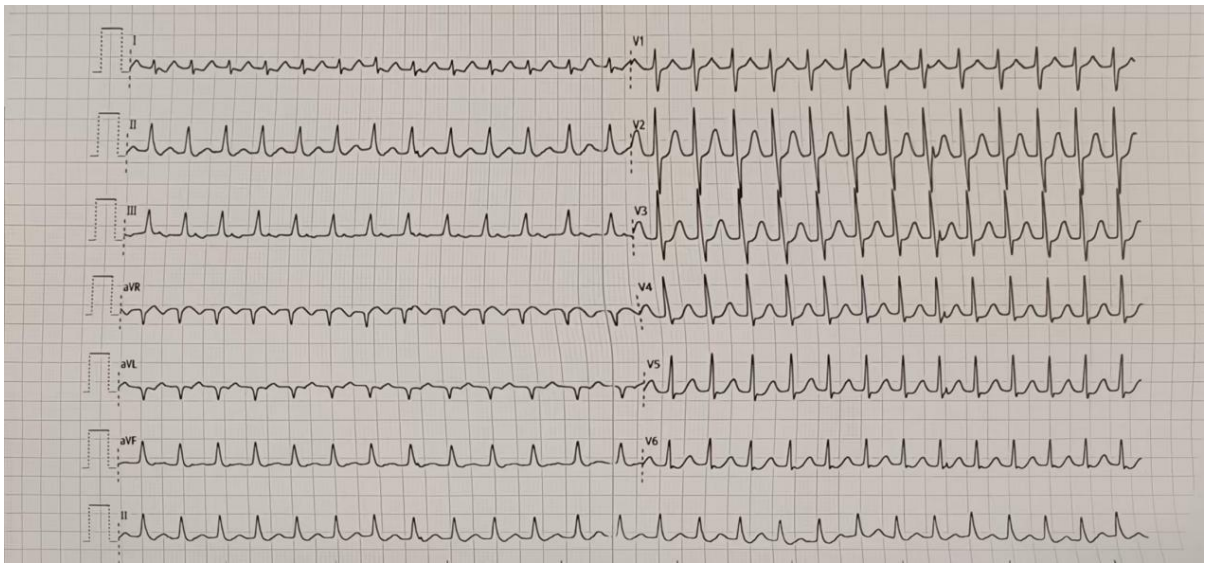

Fig-1: Admission Electrocardiogram (ECG)

(Retrograde P' waves visible following QRS complexes, with RP' < P'R, RP' < 1/2 RR interval, and RP' > 70ms. Regular R-R intervals, ST-segment depression in leads V1-V6, and ST-segment elevation in lead aVR.)

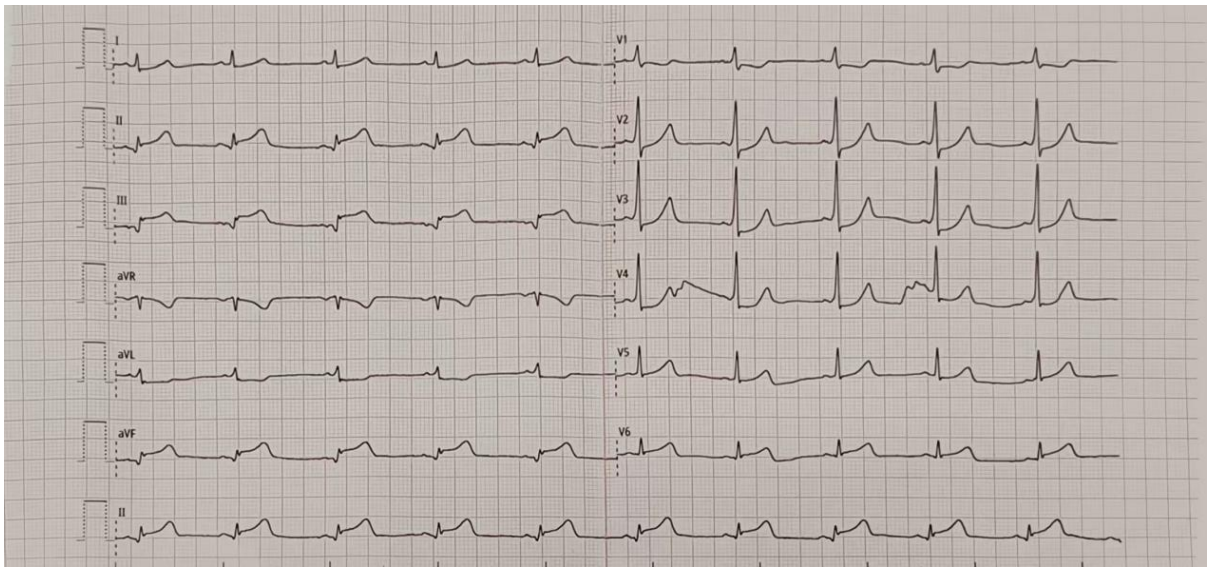

Fig-2: Electrocardiogram During Patient's Chest Pain

(Repeat ECG showing PR interval < 120ms; presence of pre-excitation wave (Delta wave) at QRS onset; ST-T changes (ST-segment depression in leads I, aVL, V1-V4, ST-segment elevation in leads II, III, aVF), and low voltage in limb leads.)

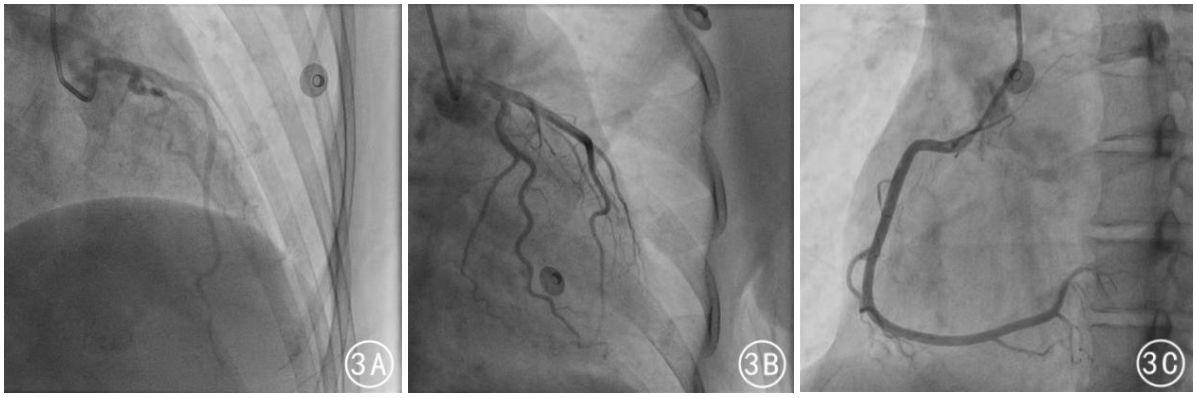

Fig-3: Normal Coronary Angiography Findings

3A: Left main coronary artery: No significant stenosis at ostium, body, or distal segment. TIMI flow grade 3; Left anterior descending artery (LAD): No significant stenosis at ostium, proximal, mid, or distal segments. TIMI flow grade 3.

3B: Circumflex artery: No significant stenosis at ostium, proximal, or distal segments. TIMI flow grade 3.

3C: Right coronary artery: No significant stenosis at ostium, proximal, mid, or distal segments. TIMI flow grade 3.

by inhibiting L-type calcium channels, which reduces calcium influx into myocardial and coronary artery smooth muscle cells. This action decreases myocardial excitability, contractility, and conduction velocity, effectively controlling heart rate. However, in this case, the patient developed sudden chest pain with significant ST-segment elevation in leads II, III, and aVF during cardioversion with diltiazem, indicating acute myocardial ischemia. Coronary angiography revealed no significant coronary artery obstruction, suggesting a more complex underlying mechanism [3]. Given the patient's clinical context, medication history, and drug response characteristics, we explore potential pathophysiological mechanisms from multiple perspectives.

In this case, the patient's heart rate during the PSVT episode reached 180 beats per minute, indicating significant sympathetic nervous system activation. Tachycardia-induced sympathetic excitation stabilizes cardiac hemodynamics by promoting the release of catecholamines, such as norepinephrine and epinephrine [4]. However, excessive sympathetic excitation increases myocardial oxygen demand and may also trigger abnormal contraction of coronary artery smooth muscle via α1-receptor activation.

Studies indicate that CCBs may induce abnormal coronary responses under certain circumstances, particularly when sympathetic nervous system activity is heightened, potentially exacerbating this response [5]. During cardioversion, as diltiazem rapidly reduced heart rate, a reflex coronary spasm may have occurred via a vagal reflex. The literature suggests that the acute use of CCBs can provoke vasoconstrictive reactions in some patients, especially those with underlying coronary flow insufficiency, which may relate to the pharmacological mechanisms of the drug and individual patient variability [6]. In this case, the patient's heart rate dropped abruptly from 180 to 70 beats per minute during cardioversion, suggesting that vagal reflex hyperactivity may have significantly contributed to the observed coronary spasm.

Previous studies have reported occasional cases of acute coronary obstruction or stenosis during the cardioversion of PSVT and atrial fibrillation, suggesting significant fluctuations in autonomic regulation during this process. HASDEMIR et al. [3] found that during radiofrequency ablation, the close anatomical proximity between the endocardium and coronary arteries may induce reflexive coronary spasms. Although no invasive procedures were performed in this case, the sudden drop in heart rate during cardioversion could have triggered excessive contraction of coronary smooth muscle through vagal reflex excitation, resulting in chest pain and ST-segment elevation. The vagal reflex plays a crucial role in regulating baseline coronary artery tone, and when the sympathetic-vagal balance is disturbed, a sudden decrease in heart rate can elicit a pronounced response in coronary smooth muscle, potentially leading to acute coronary spasm. This pathophysiological mechanism is particularly common in patients without significant coronary obstruction and is regarded as neuroregulation-driven coronary dysfunction [7].

Additionally, the pharmacological effects of CCBs may lead to paradoxical hemodynamic responses under certain pathological conditions. Diltiazem, by inhibiting L-type calcium channels, reduces calcium influx, thereby decreasing the contractility of myocardial and vascular smooth muscle, which lowers heart rate and controls rhythm. However, this inhibition may cause a "calcium imbalance" effect at the level of the coronary microvasculature. Recent studies suggest that CCBs may induce localized vasoconstrictive reactions by altering calcium dynamics within microvascular smooth muscle cells [8]. Calcium ions are crucial for maintaining vascular smooth muscle tone; when their influx through cell membranes is inhibited, coronary smooth muscle may exhibit abnormally high reactivity, potentially leading to localized coronary spasm. The sudden chest pain experienced by this patient during diltiazem administration indicates a strong coronary response, possibly due to local endothelial dysfunction and abnormal calcium influx triggered by the drug.

Moreover, the coronary spasm observed in this patient may be related to coronary microvascular dysfunction (CMD). CMD is a syndrome characterized by myocardial ischemia in the absence of significant coronary stenosis, resulting from dysregulation within the coronary microvasculature. Its pathogenesis includes endothelial dysfunction, microvascular spasm, hyperreactivity of smooth muscle cells, and microvascular embolism-factors that become particularly pronounced during episodes of tachycardia and cardioversion [9]. Research has shown that under such conditions, coronary microvessels often exhibit marked hyperreactivity [10,11]. In this case, although coronary angiography revealed no significant stenosis, the pronounced ST-segment elevation suggested localized myocardial ischemia. Given the patient's history of no coronary artery disease or other atherosclerotic conditions, CMD is suspected. CMD is often undetectable via standard coronary angiography, so further diagnostic tools, such as myocardial perfusion imaging or coronary functional tests (e.g., acetylcholine provocation test), should be considered to elucidate the underlying mechanisms.

This patient, a 38-year-old with no history of diabetes, hypertension, or other common cardiovascular diseases, likely experienced coronary spasm influenced by complex sympathetic-vagal interactions, microvascular dysfunction, and the pharmacological effects of CCBs. Studies have shown that coronary spasms are more frequent in young patients, particularly those with a history of smoking, a family history of cardiovascular conditions, or other

vascular dysfunctions [12,13]. Although this patient exhibited no typical risk factors, the ST-segment elevation observed during cardioversion suggests underlying coronary hyperreactivity, potentially related to endothelial dysfunction and excessive responsiveness of smooth muscle cells [14]. Vascular smooth muscle cells in patients with coronary endothelial dysfunction often display abnormal responses to both vasoconstrictive and vasodilatory factors, leading to pronounced vasospastic reactions [15]. This mechanism is especially prevalent in patients with coronary spasm without atherosclerotic lesions and is commonly associated with myocardial ischemia in the presence of non-obstructive coronary arteries (MINOCA) [16].

Clinically, it is crucial to consider individual patient characteristics and the potential for coronary hyperreactivity, especially when administering CCBs. To reduce the risk of coronary spasm, an individualized treatment approach should be employed, which may include monitoring heart rate variations, adjusting medication dosages, or selecting alternative drug combinations. For high-risk patients, a comprehensive evaluation using cardiac imaging and functional assessments can enhance the understanding of coronary function, facilitating the development of safer and more effective treatment strategies.

In conclusion, effective monitoring and intervention strategies for patients with PSVT are vital in clinical practice. During acute cardioversion, close observation of heart rate and ST-segment changes is essential for promptly identifying the risk of coronary spasm. Clinicians should perform individualized assessments for patients receiving CCBs and implement ECG monitoring during cardioversion to enable rapid responses to any abnormalities. By optimizing monitoring and intervention strategies, both patient safety and therapeutic outcomes can be significantly enhanced. This case highlights the necessity for vigilance regarding heart rate fluctuations and dynamic changes in coronary blood flow during arrhythmia cardioversion, especially in patients exhibiting significant heart rate variability, to prevent coronary spasm. Future research should explore the effects of CCBs on coronary blood flow across various arrhythmia types and aim to develop more personalized treatment protocols.

Availability of Data and Materials

The data presented in this study are available on reasonable request from the corresponding author.

Ethical approval and consent to participate

This study was approved by the Sichuan University Medical Ethics Review Board of Tianfu West China Hospital, 2024 No. 029. Participants agreed to participate in the study.

Conflict of Interest

The authors have read and approved the final version of the manuscript. The authors have no conflicts of interest to declare.

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