High-Sensitivity Cardiac Troponin T Elevation and Paroxysmal Supraventricular Tachycardia. Is it Always Acute Coronary Syndrome?

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ARTICLE INFO

Article history:
Submitted: 21. 5. 2024
Accepted: 14. 8. 2024
Available online: 18. 2. 2025

Klíčová slova:
Akutní koronární syndrom
Bolest na hrudi
Ischemie myokardu
Paroxysmální supraventrikulární tachykardie
Srdeční troponin měřený vysoce senzitivní metodou

SOUHRN

Kontext: Paroxysmální supraventrikulární tachykardie (PSVT) je běžnou formou arytmie, která se často projevuje symptomy připomínajícími akutní koronární syndrom (AKS).
Popis případu: Popisujeme případ 58letého muže s PSVT doprovázenou bolestí na hrudi a zvýšenými hodnotami troponinu naměřenými vysoce senzitivní metodou (high-sensitivity troponin, hs-Tn). Koronarografické vyšetření prokázalo pomalý průtok krve koronárními tepnami v nepřítomnosti obstrukčního postižení. Pacientovy symptomy vymizely spontánně a echokardiografické parametry měly při jeho propouštění normální hodnoty.
Závěry: Popisovaný případ ukazuje diagnostickou náročnost rozpoznávání AKS a upozorňuje na přechodné zvýšení hodnot troponinu při PSVT, možná v důsledku ischemie myokardu na podkladě rychlé srdeční frekvence. Při rozhodování o léčbě, zvláště v nepřítomnosti významné ischemické choroby, je nezbytné opřít se o klinické korelace a použít doplňkové diagnostické metody.

Keywords:
Acute coronary syndrome
Chest pain
High-sensitive cardiac troponin
Myocardial ischemia
Paroxysmal supraventricular tachycardia

ABSTRACT

Background: Paroxysmal supraventricular tachycardia (PSVT) is a common arrhythmia often presenting with symptoms mimicking acute coronary syndrome (ACS).
Case presentation: We present a case of a 58-year-old man with PSVT accompanied by chest pain and elevated high-sensitivity troponin (hs-Tn) levels. Coronary angiography revealed slow coronary flow in absence of obstructive disease. The patient's symptoms resolved spontaneously, and echocardiographic findings normalized upon discharge.
Conclusions: This case underscores the diagnostic challenge of individuating ACS and highlights the transient troponin elevation seen in PSVT, potentially due to myocardial ischemia secondary to rapid heart rates. Clinical correlation and adjunctive diagnostic modalities are crucial in guiding management decisions, especially in the absence of significant coronary artery disease.

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DOI: 10.33678/cor.2024.063

Please cite this article as: Miccichè S, Pelaggi G, Lo Nigro MC, et al. High-Sensitivity Cardiac Troponin T Elevation and Paroxysmal Supraventricular Tachycardia. Is it Always Acute Coronary Syndrome? Cor Vasa 2025;67:77–81.

Background

Paroxysmal supraventricular tachycardia (PSVT) is one of the most common tachyarrythmias and it encompasses a heterogeneous group of arrhythmias with different characteristics. It has a prevalence of 2.25/1000 and an incidence of 35/100000 people per year;[1–6] it is more common between the ages of 40 and 50 and predominantly affects females. PSVT refers to a rhythm disorder originating above the His bundle, which typically causes heart rates in excess of 150 per minute with regular and narrow QRS complex. The symptoms it presents with are often hardly distinguishable from acute coronary syndromes and include palpitations, chest pain, dyspnea, exertional intolerance, and syncope. The diagnosis is made through the recording of a 12-lead electrocardiogram during the arrhythmic episode.[7–11] It is generally a benign arrhythmia that resolves after vagal maneuvers or pharmacological therapy in most cases. The role of myocardial-specific marker measurement, particularly troponin, is not entirely clear. According to some studies, it has no correlation with coronary artery disease (CAD) in patients without cardiovascular risk factors, while it may indicate ischemic events in patients previously undergoing revascularization procedures.[12–21]

Case report

A 58-year-old man presented with palpitations and chest pain for about two hours. Due to the persistence of symptoms, the patient contacted emergency services and was taken to the nearest emergency department. The patient's medical history revealed hypertension, smoking habit, and diabetes mellitus,[22–26] with no history of cardiovascular diseases or prior revascularization. On arrival, the patient's vital signs were as follows: heart rate 150 bpm, oxygen saturation 96% with no supplementation of oxygen, respiratory rate 21 breaths per minute. Physical examination showed an accelerated but mostly regular heart rhythm with no further abnormalities noted. A 12-lead electrocardiogram showed atrial flutter with a ventricular rate of 150 bpm and ventricular repolarization alterations secondary to the high heart rate (**Fig. 1**).

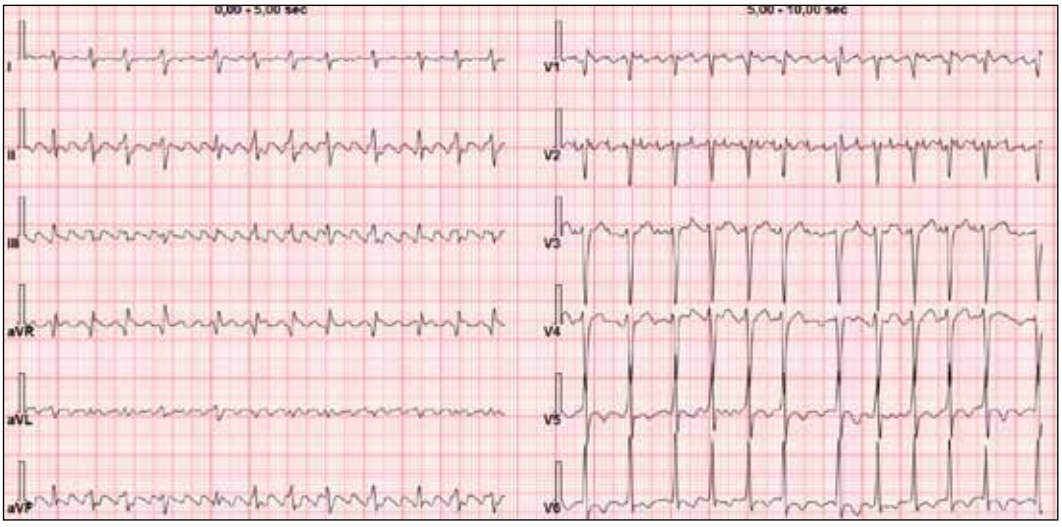

Fig. 1 – The ECG shows atrial flutter at a frequency of 150 bpm.

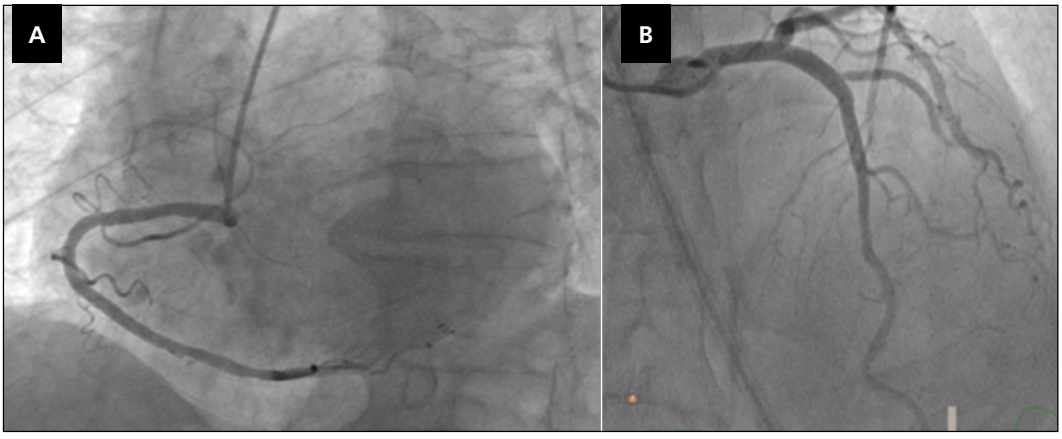

Fig. 2 – (A, B) The image depicts coronary angiography revealing sluggish and delayed flow of contrast material through the coronary arteries, characteristic of slow flow phenomenon. This phenomenon indicates impaired perfusion despite the absence of significant obstructive lesions in the arteries and is associated with conditions affecting coronary microcirculation.

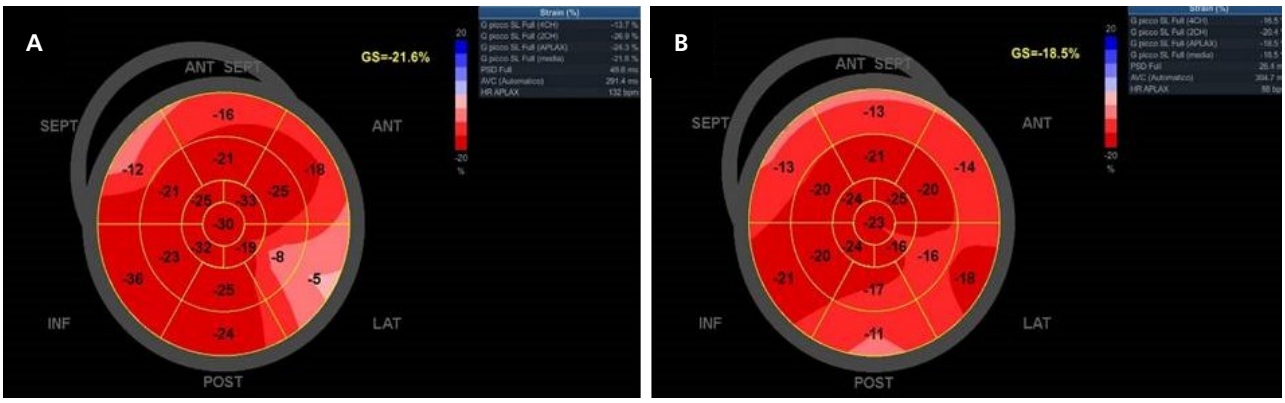

Fig. 3 – The figure illustrates the myocardial strain distribution represented as bull's eyes during and after the episode of paroxysmal supraventricular tachycardia (PSVT). (A) In the first image, the bull's eye plot of global longitudinal strain (GLS) during PSVT reveals regional abnormalities with decreased strain in the interventricular septum and lateral segments. (B) The bull's eye plot of GLS after resolution of PSVT, illustrates an improvement of strain in the interventricular septum and lateral segments. However, overall strain value remains preserved due to the hyperkinesia of the segments caused by supraventricular tachycardia.

Hematological tests including complete blood count, renal function, and myocardial-specific enzymes were performed. An echocardiogram revealed alterations in segmental kinetics and speckle tracking analysis showed normal global longitudinal strain (GLS) with hyperkinesis of apical segments and reduced GLS at the interventricular septum. The initial troponin Hs level was within normal limits (TropHS 10 ng/dl), but a subsequent control showed an approximately thirtyfold increase in the value (TropHS 300 ng/dl). The patient subsequently underwent coronary angiography, which showed no significant obstructive coronary artery disease but a condition of slow flow in all epicardial vessels (**Fig. 2**).

Spontaneous cardioversion occurred, and chest pain slowly subsided. Considering a CHA_2DS_2-VASc score >2, long-term anticoagulant therapy was initiated. A follow-up echocardiogram before discharge showed complete resolution of segmental kinetic abnormalities, and GLS analysis showed improved values in previously affected segments but with slightly lower global strain values compared to those during tachycardia (**Fig. 3**). The remaining anti-ischemic therapy was intensified, a calcium channel blocker and ranolazine were added for suspected microvascular disease, and lipid-lowering therapy was adjusted to achieve LDL targets. The patient experienced further episodes of tachycardia, albeit at a reduced frequency, and no further episodes of chest pain.[17,18,27–36]

Discussion

Paroxysmal supraventricular tachycardias are extremely common arrhythmias; the clinical presentation may vary from asymptomatic or perception of palpitation to severe chest pain or syncope. In the literature, there is widespread consensus on the close correlation between supraventricular tachycardia (SVT) and increased serum troponin levels.[37,38] However, the mechanism underlying troponin elevation in PSVT is not yet fully understood, but there are several hypotheses.[39] The most widely accepted mechanism is as follows: the increase in heart rate, even exceeding 150 bpm, on one hand, leads to an increased demand for oxygen, while on the other hand, it reduces the diastolic phase of the cardiac cycle considerably and coronary perfusion consequently. The mismatch between increased oxygen demand and reduced nutrient supply leads to a transient condition of ischemia that can result in oxidative stress, free radical release, cellular damage, and subsequent troponin release. In fact, troponin positivity has been reported in up to 30% patients with PSVT.[40–43] The transient increase in circulating myocardial necrosis enzymes is often unrelated to evidence of significant stenosis on coronary angiography and resolves once sinus rhythm is restored. Unnecessary coronary angiography carries potential risks and may add costs.[44–48] In fact, in the retrospective study by Dorenkamp, troponin levels were elevated in 14 out of 114 patients with PSVT. Out of these, 13 underwent coronary angiography, with none demonstrating significant coronary stenosis. According to this study, a positive exercise stress test performed in sinus rhythm was the best predictor of coronary artery disease and subsequent revascularization.[47] However, pre-existing pathological conditions such as intermediate stenosis or microvascular disease, which do not generate symptoms at rest, can manifest during tachycardia.[48] In a study conducted by Ben Yedder et al. on 73 patients with SPVT, one-third exhibited an elevation in troponin levels. Among these patients, only one had a significant coronary stenosis requiring revascularization intervention.[44] In another retrospective five-year study conducted by Huseyin Ede et al. involving 85 hospitalized patients with supraventricular tachycardia (SVT) associated with elevated high-sensitivity troponin, only two patients had obstructive coronary artery disease. Both patients were over 60 years old and had a high pretest probability of coronary artery disease.[49] Therefore, although significant coronary stenosis is uncommon in patients with PSVT, a comprehensive history (including cardiac risk factors), a physical examination and an interpretation of serial electrocardiograms (with particular attention to markers of myocardial ischemia, such as ST-segment depressions) are required to detect such cases. While the correlation

between troponin elevation and acute coronary syndrome remains unproven, several studies concur on the association between elevated troponin levels during supraventricular tachycardia and worsened prognosis, with an increased risk of future mortality, myocardial infarction, or hospital readmission.[50]

Conclusions

Troponin is a sensitive and specific biomarker of cardiac damage and plays an important role in the diagnosis of patients with acute coronary syndromes (ACS). A high troponin level however is not synonymous to ACS.[51–55] It is known that troponin elevation can occur in various situations such as myocarditis, chronic kidney disease, anemia, pulmonary embolism, stroke, sepsis, and not rarely, even during high heart rates.[44,45] Therefore, clinical correlation, history, ST-segment changes on ECG, and segmental kinetics on echocardiography are useful.

Some authors argue that it is also useful to subject the patient to coronary CT after arrhythmia resolution to avoid invasive procedures,[56] but the definitive test for diagnosing acute coronary artery disease remains coronary angiography. Improvements in non-invasive diagnostic techniques in the future will certainly lead to a more rapid and less invasive diagnosis.

Conflict of interest
None.

Funding body
None.

Ethical statement
We declare that the case report has been conducted in accordance with applied ethical standards and guidelines; the Declaration of Helsinki.

Informed consent
Appropriate permissions including written informed consent were obtained.

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