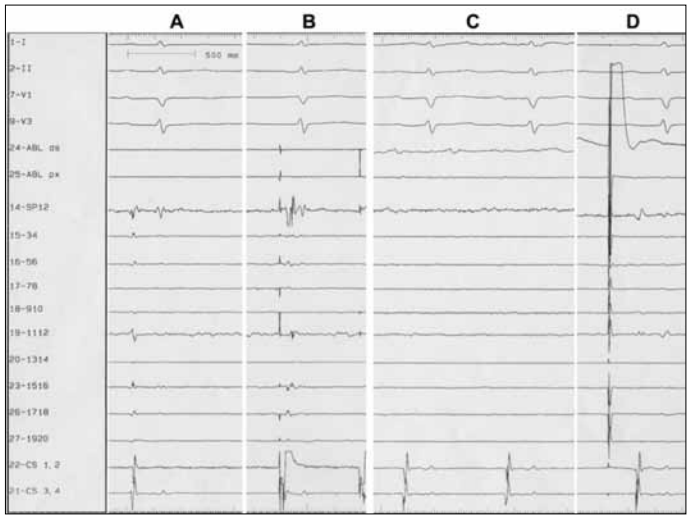


**Figure 2 . Intracardiac tracings acquired by circular mapping and ablation catheter placed in the right top PV prior to ablation reveals that it as an active focus as evident by PV potentials during sinus rhythm (Column A) where the PV potentials becomes more apparent (especially in channel SP 12) by coronary sinus pacing (Column B). After circumferential ablation, right top pulmonary vein was electrically inert as evident by abolished potentials in sinus rhythm (Column C) and exit block was confirmed by noncapture during pacing from ablation catheter inside the vein (Column D)**

ABL-ablation catheter, CS-coronary sinus catheter, SP 12 and other channels remarked with consecutive numbers represents individual poles of the circular mapping catheter

## Conclusion

By means of the presented case, we emphasize the importance of pre-procedural imaging of PV anatomy and suggest electrical disconnection of all PV variants as far as possible. If available, utilization of an image integration system combining electro-anatomic mapping images with reconstructed 3-dimensional CT scans can offer ease and safety when performing similar complex ablation procedures.

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# Changes of high-sensitive troponin level in a patient with paroxysmal supraventricular tachycardia

*Paroksismal supraventriküler taşikardili bir hastada yüksek duyarlı troponin düzeyindeki değişiklikler*

## Introduction

High sensitive troponin (hsTn) assays offer great opportunities to diagnose acute myocardial infarction (AMI). The demonstration of rise and/or fall of troponin (Tn) with at least one value about the 99$^{th}$ percentile upper than the reference limit as well as the evidence of myocardial ischemia are necessary to diagnose AMI. These days the frequency of Tn positivity in the non-acute coronary syndrome has increased resulting in more overcrowding of emergency departments. It is evident that AMI is a clinical rather than a biological diagnosis and a physician should consider the clinical content. In this study, the specific pattern of hsTn change in a patient admitted with chest pain, paroxysmal supra-ventricular tachycardia (PSVT) and ST segment depression in electrocardiogram (ECG) without coronary artery disease (CAD) would be introduced.

## Case Report

A 64-year-old woman has been recently referred to our emergency department due to chest pain and narrow complex tachycardia before performing exercise tolerance test (ETT). She experienced an episodic epigastric pain intensified by physical exercise. On admission, she had substernal and epigastric pains and palpitation without any associated symptoms. On physical examination, she had no pathological finding except for tachycardia with a rate of 140 beats/min. The patient had no history of CAD and her only risk factor within past 10 years was hypertension. ECG revealed narrow complex tachycardia, ST segment depression in inferior leads, V5-V6 and ST segment elevation in aVr lead (Fig. 1) Tachyarrhythmia was cardioverted with administration of 5 mg verapamil intravenously (Fig. 2). Considering the patient's suspicious symptoms and ECG findings, hsTn was checked on admission which it showed a normal level of 3.28 ng/L [reference range <14ng/L, Coefficient of Variation (CV) <10%]. Six hours later the pronounced rising level of hsTn in the second sample (39.56 ng/L) followed by a falling pattern to 22.13ng/L in 16 hours of admission was observed (Fig. 3). The serial CK-MB mass levels were 2.2, 2.86 and 2.3 ng/ml (reference value for females < 3.77 ng/ml). Because

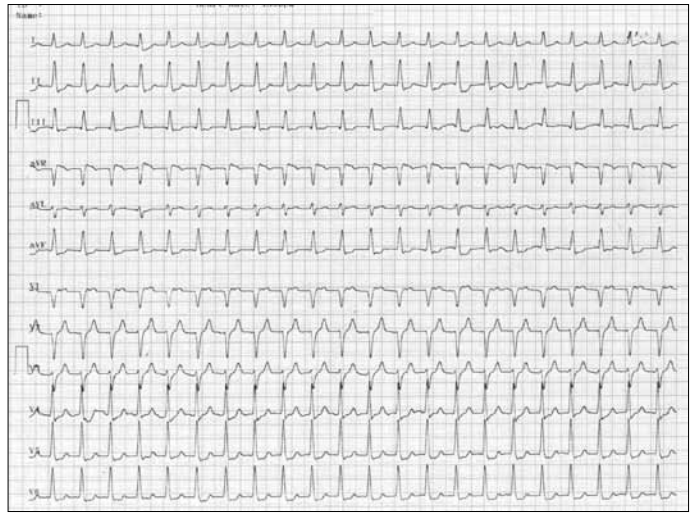

**Figure 1. ECG at presentation with chest discomfort**

ECG - electrocardiogram

of this atypical pattern for AMI and atypical chest pain, 2-D echocardiography and symptom limited ETT under close supervision was performed before discharge, suggesting no evidence of ischemia. After 112 hours of the patient's first admission, another hsTn was and it decreased from 22.13 ng/L to normal level of 3.34 ng/L. Due to patient's previous chest discomfort and tachycardia with high level of hsTn, concomitant electrophysiological study (EPS) and ablation and coronary artery angiography (CAG) were electively performed for the patient in the next few days. CAG and EPS showed normal epicardial coronary arteries and atrioventricular node reentry (AVNRT), respectively; and slow pathway ablation was performed successfully.

## Discussion

Troponin elevation shows the presence of myocardial damage. However myocardial injury can happen due to a variety of mechanisms rather than acute ischemia. Therefore, the other causes of myocardial injury should be considered when troponin elevation is inconsistent with AMI (1). In a recent published study, by 51 PSVT cases, 11 of them had a positive Tn test. Furthermore one month follow up showed no PSVT recurrence or death for these patients (2). This study confirms our opinion, considering further evaluation of troponin in SVT patients. In another study, Bukkapatnam et al. (3) showed that ST segment depression and the increase of troponin were not significant predictors of CAD. Dorenkamp et al. (4) also showed that there was no relation between troponin increase or ST-segment depression and CAD. In both studies, the association between isolated troponin levels with CAD was assessed without considering the changes in the pattern of troponin. But cardiac troponin elevation has also been conserved after strenuous exercise and it has been shown that its increase is typically transient and troponin usually reaches normal level within 24-48 hours (5). The most probable mechanism for post tachycardia troponin rising might be the shortening of diastole with subsequent subendocardial ischemia (6). Furthermore, it has been hypothesized that the troponin source degenerates cytosolic troponin or increases permeability of the cell membranes of myocytes under stress (7). Considering in this case, it seems that the pattern of rising and falling of troponin in tachyarrhythmia without AMI is similar to strenuous exercise but not to AMI and cytosolic troponin is responsible for transient serum troponin rise. This rise in troponin can be marked as type 2 MI which is defined as MI secondary to ischemia due to either increased oxygen demand or reduced supply (8). However, discrimination between type 1 (typical) MI and type 2 MI is paramount in order to provide timely and proper treatment.

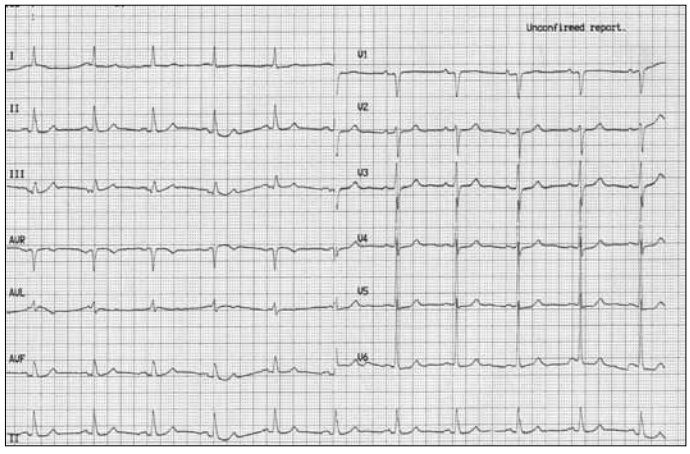


**Figure 2. ECG after paroxysmal supraventricular tachycardia cardioversion at emergency department**
ECG - electrocardiogram

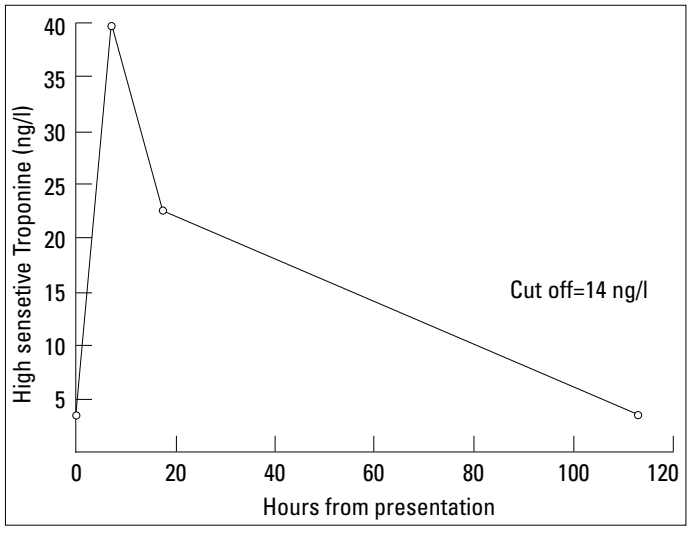


**Figure 3. High sensitive troponin changes in supraventricular tachycardia**

## Conclusion

In patients with chest pain and tachyarrhythmia, reliance only on two sets of troponin for diagnosis of AMI can be misleading and rising and falling within 2-4 days establishes type 2 MI and excludes type 1 MI. The significance of this troponin elevation has not been established until to date.

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