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Unusual Troponin Level in Atrioventricular Nodal Reentrant Tachycardia Despite Normal Coronary Arteries

Authors' Contribution:

Study Design A

Data Collection B

Statistical Analysis C

Data Interpretation D

Manuscript Preparation E

Literature Search F

Funds Collection G

ABCDEF 1 **Muhammed Atere**E 2 **Sean Galligan**

1 Department of Internal Medicine, Richmond University Medical Center, Staten Island, NY, U.S.A.

2 Department of Cardiology, Richmond University Medical Center, Staten Island, NY, U.S.A.

Corresponding Author: Muhammed Atere, e-mail: MAtere@Rumcsi.org**Conflict of interest:** None declared

Patient: Female, 36-year-old
Final Diagnosis: Atrioventricular nodal reentrant tachycardia
Symptoms: Palpitation
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course**Background:** Elevation of troponin in atrioventricular nodal reentrant tachycardia may occur but it is usually mild. Although there are often no identifiable etiologies, stimulants and excessive activities have been implicated.**Case Report:** We present the case of a 36-year-old female with palpitations. Her laboratory investigation was positive for a very high level of troponin despite insignificant illicit drug use, unremarkable inflammatory markers, unremarkable coronary arteries after a coronary angiogram, and normal biventricular function without gadolinium enhancement on cardiac magnetic resonance imaging. The only attributable culprit was atrioventricular nodal reentrant tachycardia during electrophysiology studies but radiofrequency ablation was unsuccessful. We believe it is important that physicians should be aware that a very high troponin does not always reflect an infarction or structural damage to the heart.**Conclusions:** It has been documented that tachyarrhythmias cause a mild increase of troponin levels and severe elevations of troponin are often attributed to myocardial infarction. Physicians should be aware that troponin may increase to over 200 times above the normal limit in a patient with atrioventricular nodal reentrant tachycardia, normal coronary arteries, and no structural heart disease.**MeSH Keywords:** Arrhythmias, Cardiac • Cardiac Electrophysiology • Cardiology • Tachycardia, Atrioventricular Nodal Reentry**Full-text PDF:** <https://www.amjcaserep.com/abstract/index/idArt/922831>

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Background

Atrioventricular nodal reentrant tachycardia (AVNRT) is known to cause mild elevation of troponin but not significant enough to mimic the level seen in acute myocardial infarction. It usually presents in paroxysms and about 67% occur in women [1]. There are usually no identifiable provoking factors, but the use of stimulants and excessive activities have been implicated.

Case Report

A 36-year-old female with a past medical history of palpitations since her 20s (with no prior investigations) and depression on paroxetine, presented to the Emergency Department (ED) because of worsening of persistent palpitations. Her symptoms had been ongoing for the past three months before she came to the ED. She was consuming caffeinated beverages during that time. Her palpitations improved but got worse again after consuming caffeine on the day of presentation. Her palpitations lasted about one hour but had resolved before she was seen. As described by the patient, it was associated with shortness of breath and mild left-sided chest pain, which also resolved before coming to the ED for evaluation. At presentation, vital signs and physical examination did not reveal any positive findings. Her laboratory findings were pertinent for hemoglobin of 11.8 g/dL, and an initial troponin I was 8.16 ng/mL approximately seven hours after palpitations started with a peak of 10.50 ng/mL and a lowest level of 6.86 ng/mL at the time of discharge. Urine beta-human chorionic gonadotropin was

negative for pregnancy and a urine drug screen was also negative for phencyclidine, amphetamine, and cocaine. Thyroid-stimulating hormone (TSH), C-reactive protein, and erythrocyte sedimentation rate were essential within normal limits. An electrocardiogram (Figure 1) was not evident for acute coronary syndrome and a chest x-ray was normal. An echocardiogram was unremarkable with an ejection fraction of 70%. She was hospitalized in the coronary care unit (CCU). Telemetry revealed no documented evidence of an arrhythmia while in the CCU. The patient underwent a cardiac catheterization, which revealed normal coronary arteries. Electrophysiology studies were performed. At baseline, the patient was in sinus rhythm: sinus cycle length was 820 milliseconds, AH interval was 78 milliseconds, HV was 46 milliseconds, QRS duration was 80 milliseconds, and PR interval was 140 milliseconds. Pacing from the high right atrium using the right atrial catheter was performed, which showed AV Wenckebach cycle lengths at 360 milliseconds. Block was at the AV nodal level. There was evidence of slow pathway conduction. AVNRT was easily and reproducibly induced at any pacing cycle length. Programmed atrial stimulation showed AV node effective refractory period of 280 milliseconds at drive cycle lengths of 600 milliseconds. The longest A2H2 was 362 milliseconds. AVNRT was reproducibly induced. Programmed ventricular stimulation showed concentric retrograde atrial activation sequence with decremental conduction and no bypass tract. The ventricular atrial block cycle length was 310 milliseconds. Unfortunately, radiofrequency energy ablation was unsuccessful. The patient remained asymptomatic after electrophysiology studies, and hence, a troponin was not done afterward. The patient was discharged on metoprolol

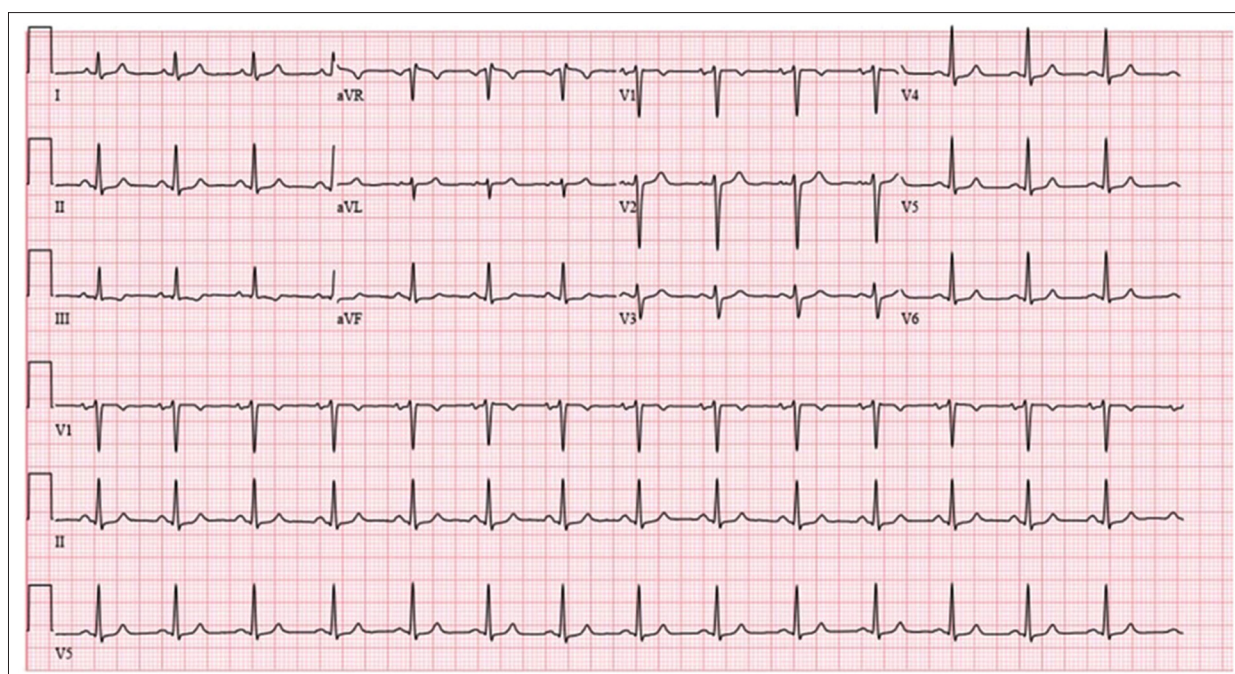


Figure 1. Electrocardiogram showing no obvious evidence of acute coronary syndrome.

tartrate 25 mg twice daily and was instructed to have a cardiac magnetic resonance imaging as an outpatient, which demonstrated normal biventricular function with no late gadolinium enhancement. She has remained symptom-free since her discharge at the time of writing this case report.

Discussion

Troponin is a marker that can be found in cardiac muscles. It is often released into the bloodstream during myocardial injury. There are three forms of troponin – troponin C, troponin I and troponin T. They can be found in both cardiac and skeletal muscles, but many laboratories can detect specific cardiac troponins, for example, troponin I (cTnI). When an injury occurs to the myocardium, proteins are released into the circulatory system including troponin, creatine kinase, alanine transaminase, aspartate transaminase, and lactate dehydrogenase. cTnI can be detected as early as six hours post-injury and usually peaks at 24 hours after the cardiac injury. It remains elevated after several days but in a declining manner. Many diseases can cause an elevation in cTnI, the commonest is acute myocardial infarction. Others include myocarditis, tachyarrhythmia, cardiac traumas, pulmonary embolism and cardiopulmonary resuscitation. Severe and critical elevation of cTnI is usually found in myocardial infarction but with other causes often causing mild elevation. In a retrospective study conducted in Australia, peaked cTnI was a strong indicator of coronary artery disease [2]. In another article the peaked cTnI in patients with AVNRT was 0.89 ng/mL [3]. Studies have shown that AVNRT is mainly associated with a mild increase of cTnI but there are no levels specific for AVNRT. In clinical practice,

many physicians will not hesitate to perform a coronary angiogram in a patient with cTnI more than 230 times above the normal upper limit with a presentation of palpitations associated with shortness of breath and chest pain. The initial differential diagnosis was acute coronary syndrome because of the severe elevation of troponin level. Acute myocarditis was also considered but was less likely because the history and examination did not fully support this diagnosis. A tachyarrhythmia was also considered, and it was deemed the culprit for her presentation and laboratory findings. A very high cTnI is often associated with significant myocardial injury; however, the level of troponin seen in our patient without evidence of coronary artery disease, illicit drug use, pregnancy or other structural heart disease makes our case unusual and unique. Furthermore, we are not aware of any published case report demonstrating such a level of troponin in AVNRT without structural heart disease.

Conclusions

It has been documented that tachyarrhythmias cause a mild increase of troponin levels and severe elevations of troponin are often attributed to myocardial infarction. Physicians should be aware that troponin may increase to over 200 times above the normal limit in a patient with AVNRT and normal coronary arteries.

Conflict of interest

None.

References:

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