CASE REPORT

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Can troponin-I be used as an independent predictor of cardiac dysfunction after supraventricular tachycardia in children with structurally normal heart?

Da li se troponin-I može koristiti kao nezavisan prediktor srčane disfunkcije posle supraventrikulske tahikardije kod dece sa strukturno zdravim srcem?

Aleksandra M. Simović*†, Jelena M. Tanasković-Nestorović*†, Sanja M. Knežević*†, Biljana P. Vuletić*†, Andjelka K. Stojković*†, Dejan M. Jeremić*, Jovanović Milan‡§, Maja Vulović*

University of Kragujevac, *Faculty of Medical Sciences, Kragujevac, Serbia; Clinical Centre of Kragujevac, †Pediatric Clinic, Kragujevac, Serbia; Military Medical Academy, ‡Clinic for General Surgery, Belgrade, Serbia; University of Defence, §Faculty of Medicine of the Military Medical Academy, Belgrade, Serbia

Abstract

Introduction. Elevated cardiac troponin gives excellent accuracy in the identification of myocardial damage in children, but it can also be elevated in a series of other diseases. **Case report.** We presented two children thirteen years of age with a high serum level of troponin-I after an acute episode of supraventricular tachycardia. We analyzed troponin-I levels in correlation with the maximum heart rate, duration of tachycardia and systolic left ventricular function (ejection fraction and fractional shortening). **Conclusion.** Abnormal troponin level can be seen in children with sustained supraventricular tachycardia and normal heart. Caution is advised in diagnosing cardiac dysfunction in children with supraventricular tachycardia and elevated troponin levels.

Key words:
tachycardia, supraventricular; arrhytmias, cardiac; troponin I; child; prognosis; ventricular dysfunction, left.

Apstrakt

Uvod. Povišen nivo srčanog troponina daje izuzetnu preciznost u identifikaciji oštećenja miokarda dece, ali može biti povišen i u nizu drugih bolesti. **Prikaz bolesnika.** Prikazano je dvoje dece uzrasta 13 godina sa visokim serumskim nivoom troponina-I nakon akutne epizode supraventrikulske tahikardije. Analiziran je nivo troponina-I u korelaciji sa maksimalnom srčanom frekvencijom, trajanjem tahikardije i sistolnom funkcijom leve komore (ejekciona frakcija i frakciono skraćenje). **Zaključak.** Izmenjen nivo troponina može se videti kod dece sa dugotrajnom supraventrikulskom tahikardijom i zdravim srcem. Savetuje se oprez u dijagnozi srčane disfunkcije kod dece sa supraventrikulaskom tahikardijom i povišenim nivoom troponina.

Ključne reči:
tahikardija, supraventrikulska; aritmija; troponin I; deca; prognoza; srce, disfunkcija leve komore.

Introduction

Cardiac troponin as a marker of myocardial necrosis is now commonly used in clinical practice in adults with coronary artery diseases. In children, it is a sensitive and specific biomarker consistent with cardiac damage (within severe acute and chronic heart failure, congenital heart disease and myocarditis, cardioversion, cateter ablation or trauma of myocardium, endomyocardial biopsy, drug- and toxin-induced cardiac toxicity) [1–3].

On the other hand, high level of cardiac troponin-I (cTnI) can be seen in sepsis, acute renal or respiratory dysfunction, "overtraining syndrome", pulmonary arterial hypertension or pulmonary embolism, amyloidosis or other infiltrative diseases, burns, as well after noncardiac surgery [4–9]. In neonatology, cTnI is analyzed as an early indicator of critically ill newborns with severe respiratory distress, hypoxic-ischemic encephalopathy, hemodynamically significant patent ductus arteriosus, or mortality risk. The reasons why the

Correspondence to: Stojković Andjelka, Clinical Centre "Kragujevac", Paediatric Clinic, Zmaj Jovina 30, 34 000 Kragujevac, Serbia. E-mail: andja410@mts.rs

poor prognosis is associated with increased cardiac troponin are still not fully understood [10–13].

Up to now, supraventricular tachycardia (SVT)-induced elevations in cTnI in children with normal heart was not investigated. The aim of this study was to determine the prognostic value of troponin assays in children presenting to the emergency department with tachycardia. We assessed the test characteristics for positive cTnI (defined as > 0.04 μg/L, the manufacturer's upper limit of normal) in correctly identifying children who had SVT.

Case report

Case 1

A 13 and a half years old female child was admitted to our hospital because of the chest pain and palpitation after a heavy meal. The symptoms lasted for at least 8 hours and were relieved when she arrived to the hospital. She had no personal or family history about congenital heart anomaly, or other diseases (complete blood count, sedimentation, C-reactive protein, procalcitonin, glycaemia, electrolytes, transaminase, urea, creatinine, thyroid hormones and native chest X-ray were within normal ranges).

The results of physical examination showed a maximum heart rate (maxHR) 224 per minute, without symptoms and signs of low cardiac output: symmetrical palpable pulses, well-filled, blood pressure (BP) = 100/70 mmHg, respiration (R) 23 per minute, percutaneous oxygen saturation (SaO_2) 90%–91%. Gas analysis showed mild respiratory acidosis (partial pressure of carbon dioxide (pCO_2) 6.3 kPa, partial pressure of oxygen (pO_2) 4.9 kPa).

The patient's electrocardiogram (ECG) at the admission to the intensive care unit (ICU) showed atrioventricular reentry tachycardia (AVRT); ST segment depression 1.5–2 mm in leads V5–6 and max HR = 217 per minute. Figure 1 shows intermittent preexcitation in the Wolff-Parkinson-White syndrome that was determined later.

The echocardiogram showed no abnormal changes [ejection fraction (EF) and fractional shortening (FS) showed in Table 1]. After attempting vagal maneuvers, she was treated with adenosine and then continued with oral therapy – tablets of metoprolol (2 × 25 mg, 5 days and then 50 mg + 25 mg to control).

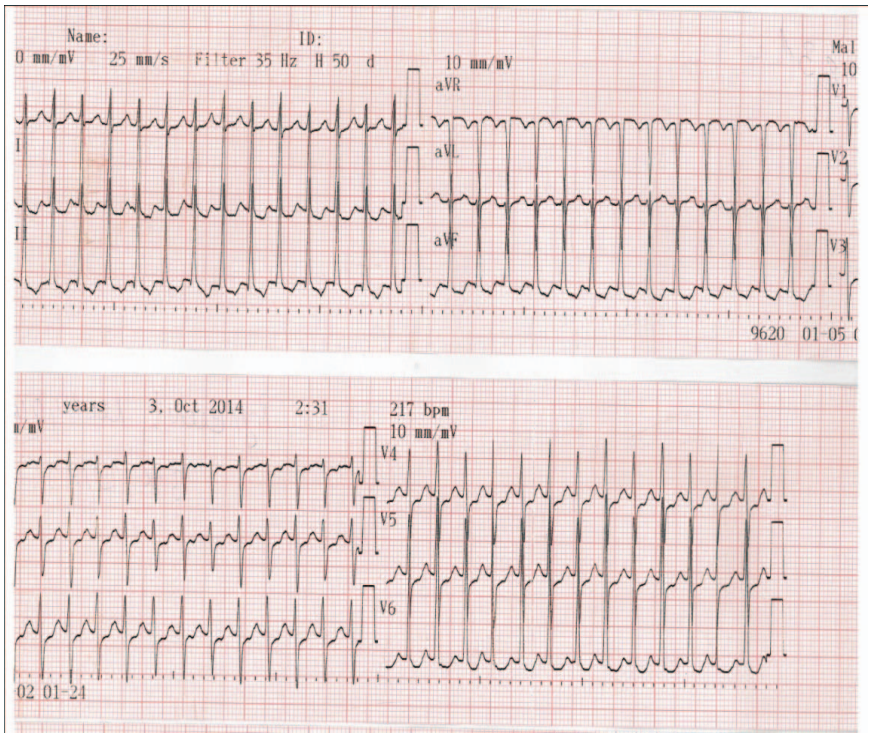

Fig. 1 – Electrocardiogram at admission **to the intensive care unit (ICU) showed atrioventricular reentry tachycardia (AVRT) in the patient.**

Table 1

Basic clinical characteristics of children presented to the emergency department with supraventricular tachycardia (SVT)

Patients	Duration of SVT (hours)	Max. HR (beats per minute)	EF (%)	FS (%)	Max. level of troponin-I (μg/L)
Case 1	8 h	217	66	37	0.115
Case 2	12 h	219	83	51	0.377

MaxHR – maximum heart rate; EF – ejection fraction; FS – fractional shortening.

A cardiac troponin I was determined at admission. The level of cTnI was 0.115 μg/L. On the fourth day, the value was lowered to normal (0.021 μg/L). In contrast, other cardiac markers simultaneously determined from the same blood sample were within normal ranges (creatine kinase 60 U/L, normal range 60–174 U/L; CK-MB fraction 10.3 U/L; lactate dehydrogenase 361 U/L, normal range 140–280 U/L), except for slightly elevated NT-Pro B-type natriuretic peptide (ProBNP): 391 pg/mL (normal range < 125 pg/mL, certain heart failure > 450 pg/mL).

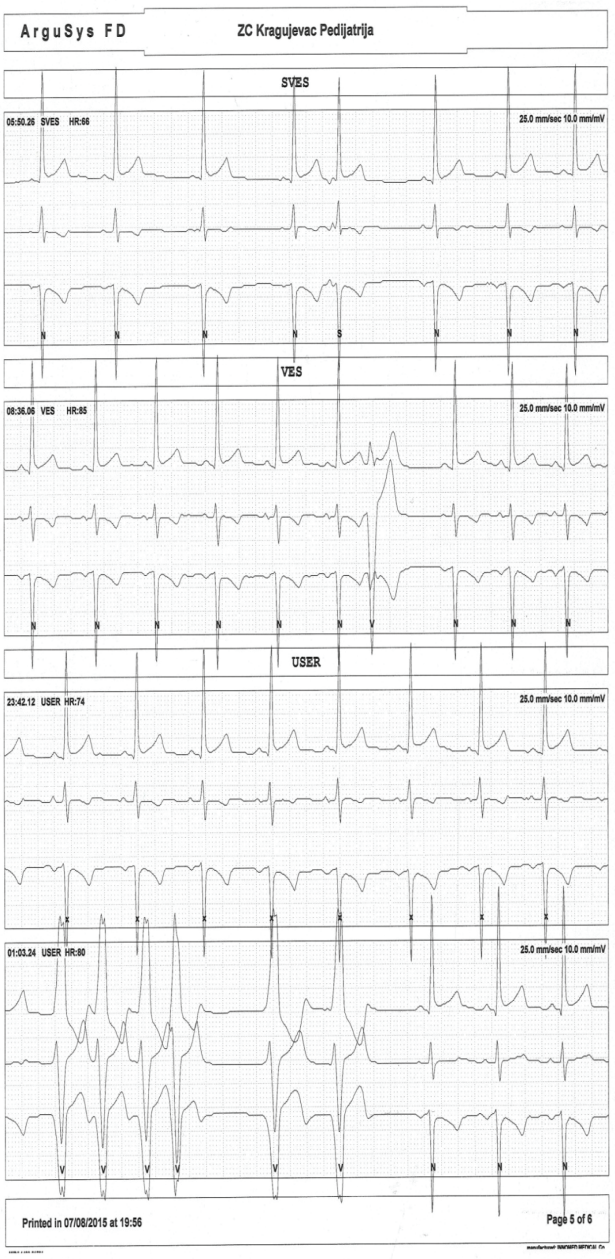

Fig. 2 – Accelerated idioventricular rhythm and supraventricular extrasystoles (SVES) after atrioventricular reentry tachycardia (AVRT) attacks.

Accelerated idioventricular rhythm [6 beats; 80 beat per minuta (bpm); 01:03.24h)], 25 supraventricular and 12 ventricular extrasystoles [or aberrantly conducted supraventricular extrasystoles (SVES)] were found during the 24-hour ambulatory ECG monitoring (Figure 2). ECG (without attack of SVT) and the test load were normal (MaxHR 139 per minute reached at the 5th to a degree; Blood pressure (BP) prior to the test load was 120/70 mmHg, at the maximum load BP was 140/50 mmHg, and after staying BP was 120/60 mmHg).

Case 2

A 13 years and 9 months old female child was hospitalized with complaint of the chest pain, palpitation and shortness of breath during the previous 12 hours, after emotional stress. A family history was as follows: her father suffers from high blood pressure, and grandfather had acute myocardial infarction. Physical examination showed maxHR 217 per minute, R 12 per minute, BP 91/51 mmHg, SaO_2 94%, (after SVT R 19 per minute, BP 108/50 mmHg, SaO_2 97%). Biochemistry and thyroid hormones showed normal values.

She was diagnosed as atrioventricular nodal reentry tachycardia (AVNRT): maxHR 219 per minute; ST segment depression 2 mm in leads V4–V6 in the surface ECG (Figure 3) and no abnormal changes in echocardiogram (Table 1). She received the same medicine as the first patient described.

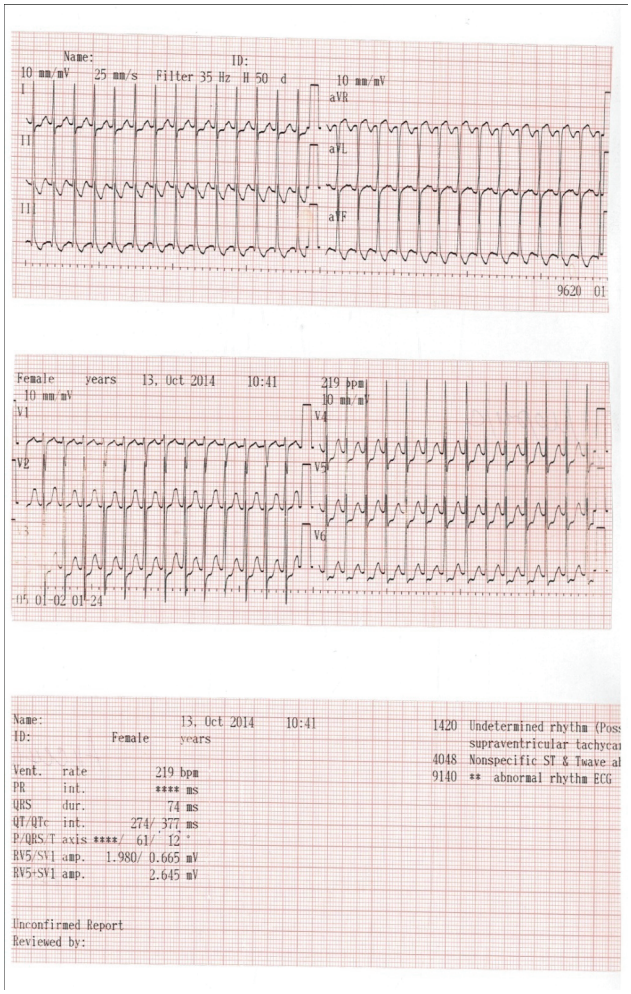

Fig. 3 – Electrocardiogram at admission to the intensive care unit (ICU) showed atrioventricular nodal reentry tachycardia (AVNR) in the case 2.

The level of cTnI was 0.377 μg/L at admission, and four days afterwards the value was up to 0.038 μg/L (Table 1, Figure 2). Also, ProBNP was clearly elevated in the serum (1,617 pg/mL) aut adission and after four days and the value was up to 286 pg/mL. During the 24-hour ambulatory ECG monitoring 6 SVES were found. Also, ECG (without attack of SVT) and the load test were normal.

Discussion

Cardiac troponin T (cTnT) and cTnI in serum are commonly used as standard biomarkers for the diagnosis of an acute coronary syndrome or myocardial infarction. In adult patients with SVT, most authors posed the question if troponin levels were useful for evaluating the presence of coronary artery disease [14–24]? Published reports (limited case series: 1–7 patients ages 18–72) presented that troponins could be released because of tachycardia alone in the absence of myodepressive factors, inflammatory mediators, or coronary artery disease [20–24]. The current literature on this topic shows that 12% to 48% of adult patients will have elevated troponins after SVT. Schmitz and Rezaic [14] in their research found troponin elevation in patients with SVT with normal coronary angiography and it was thought to be due to cardiac stretch, poor diastolic perfusion and/or coronary artery vasospasm.

In children, SVT is a common and generally benign arrhythmia. The causes of SVT include: lung disease, abnormal heart structure, or an abnormal extraelectrical pathway of the heart and use of certain medications (in the asthmatic status diastolic hypotension and tachycardia are dose-dependent side effects of high-dose albuterol) [25, 26]. The severity of SVT can vary greatly. It can last for < 30 s. (nonsustained SVT) and cause little or no symptoms or it can last for hours (sustained SVT) and cause palpitations, chest pain, shortness of breath and even fainting in rare cases.

Left ventricular dysfunction can show persisted symptoms or abnormal ECGs after conversion to normal sinus rhythm. While you treat the children's heart rate, you wonder if a troponin level would be useful in evaluating the presence of cardiac dysfunction. Our case report presented hemodynamically stable patients with various troponin elevation in proportion to the duration of tachycardia. Therefore, the troponin rise in our patients was a direct result of sustained SVT.

There has not been enough research to date to support the routine use of troponin in the evaluation of SVT in children. A routine testing can result in false positive findings (shortness of breath, persistent infection, chronic anemia, hemolysis and other reasons). Consequently, in children with various duration tachycardias, the use of troponin testing would be best performed selectively according to presented symptoms and risk factors for cardiac dysfunction.

Conclusion

Having the evidence, we do not recommend that troponin levels are determined in uncomplicated SVT in children. Future research in cardiology could be determination of the peak cardiac troponin levels that indicate a risk for left ventricular dysfunction after SVT. Moreover, it should be looked into whether these patients have increased cardiac rehospitalization over the next year.

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