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DA LI MOŽE TROPONIN-I DA SE KORISTI KAO NEZAVISAN PREDIKTOR SRČANE DISFUNKCIJE NAKON SUPRAVENTRIKULARNE TAHIKARDIJE KOD DECE SA STRUKTURNOM ZDRAVIM SRCEM?


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Can troponin-I be used as an independent predictor of cardiac dysfunction after supraventricular tachycardia in children with structural normal heart?
Da li može Troponin-I da se koristi kao nezavisan prediktor srčane disfunkcije nakon supraventrikularne taikardije kod dece sa strukturnom zdravim srcem?

*Faculty of Medical Sciences, University of Kragujevac, Svetozara Markovica 69, Kragujevac, Serbia,
†Pediatric Clinic, Clinical Centre of Kragujevac, Zmaj Jovina 30, Kragujevac, Serbia
‡Clinic for General Surgery, Military Medical Academy, Belgrade, Serbia,
*Faculty of Medicine of the Military Medical Academy, University of Defence, Belgrade, Serbia

Corresponding author: Stojkovic Andjelka*,† MD, PhD, Assoc.Prof. Mail address: 
†Paediatric Clinic, Clinical Centre "Kragujevac", Zmaj Jovina 30, 34000 Kragujevac and 
*Faculty of Medical Science, University of Kragujevac, Serbia, Phone: +38134505175; E-mail: andja410@mts.rs
First author: Simovic M. Aleksandra*,†, Assoc.Prof.MD, PhD, Mail address: ¹Paediatric Clinic, Clinical Centre "Kragujevac", Zmaj Jovina 30, 34000 Kragujevac and ²Faculty of Medical Science, University of Kragujevac, Serbia, Phone: +38134505173; Mobile: +381631623191, E-mail: aleksandra.simovic@yahoo.com
Co-authors email:
Tanaskovic-Nestorovic M. Jelena*,† MD, MS, Assist. E-mail: jelenanestorovickg@gmail.com
Knezevic M. Sanja*,† MD, PhD, Assist. E-mail: sanjaknez1980@yahoo.com
Vuletic P. Biljana*,† MD, PhD, Assoc.Prof. E-mail: sibil@ptt.rs
Jeremic M. Dejan*, MD, PhD, Assist.Prof. E-mail: dejananatom@yahoo.com
Jovanovic Milan‡*, MD, Assist. E-mail: hsekci@gmail.com
Vulovic Maja* MD, PhD, Assoc.Prof. E-mail: maja@medf.kg.ac.rs
Short title: Troponin in supraventricular tachycardia

Contributions: Aleksandra Simovic, Jelena Tanaskovic-Nestorovic and Sanja Knezevic analyzed and interpreted the patients’ data and was a major contributor in writing the manuscript. Biljana Vuletic, Andjelka Stojkovic, Dejan Jeremic and Maja Vulovic evaluated the draft and suggested revisions. All authors read and approved the final manuscript.

Abbreviations:
CTnI: Cardiac troponin I, BP-blood pressure, R-respiration, ECG-electrocardiogram, NT-Pro B-Type Natriuretic Peptide (proBNP), SVES-supraventricular extrasystoles, SVT: supraventricular tachycardia; AVRT: Atrioventricular Reentry Tachycardia; AVNRT: Atrioventricular Nodal Reentry Tachycardia

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 showed two children aged thirteen years with a high serum troponin-I after an acute episode of supraventricular tachycardia. We analyzed troponin-I levels in correlated 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 presenting with sustained supraventricular tachycardia and normal heart. Caution is advised in the diagnosis cardiac dysfunction in children with supraventricular tachycardia and elevated troponin levels.
Keywords: hearts, troponin-I, child, tachycardia, supraventricular
Sažetak

**Uvod** Povišen nivo srčanog troponina daje izuzetnu preciznost u identifikaciji oštećenja miokarda kod dece, ali može biti povišen i u nizu drugih bolesti. **Prikaz slučaja** Prikazali smo dvoje dece uzrasta trinaest godina sa visokim serumskim troponina-I nakon akutne epizode supraventrikularne tahikardije. Analizirali smo nivo troponina-I u korelaciji sa maksimalnom srčanom frekventom, trajanjem tahikardije i sistolnom funkcijom leve komore (ejekcionalna frakcija i frakcionalno skraćenje). **Zaključak** Izmenjen nivo troponina se može videti u dece sa dugotrajnom supraventrikularnom tahikardijom i zdravim srcem. Savetuje se oprez u dijagnozi srčane disfunkcije u dece sa supraventrikularnom tahikardijom i povišenim nivoom troponina.

**Ključne reči:** srce, troponin-I, dete, tahikardija, supraventrikularna

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**CASE REPORT**

**Introduction**

Cardiac troponin as 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 or myocarditis, cardioversion, cateter ablation or trauma of myocardium, endoyocardial 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 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 poor prognosis associated with increased cardiac troponin are still not fully understood\(^10\)-\(^13\).
Up to now, was not researched supraventricular tachycardia (SVT) - induced elevations in cTnI in children with normal heart. The aim of our 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 years and 6 months-old female child was admitted to our hospital because of chest pain and palpitation after a heavy meal. The symptoms lasted for at least 8 hours and were relieved when she arrived at 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 maximum heart rate (MaxHR) 224 per minute, without symptoms and signs of low cardiac output: symmetrical palpable pulses, well-filled, blood pressure (BP) =100/70mmHg, respiration (R) 23 per minute, percutaneous oxygen saturation (SaO2) 90-91%. Gas analysis showed mild respiratory acidosis (partial pressure of carbon dioxide (pCO2) 6,3kPa, partial pressure of oxygen (pO2) 4,9kPa).

Her electrocardiogram (ECG) at admision to 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.

Figure 1: Electrocardiogram at admision to ICU showed AVRT in case 1
ICU: intensive care unit; AVRT: Atrioventricular Reentry Tachycardia

The echocardiogram showed no abnormal changes (EF and FS showed in Table 1). After attempt vagal maneuvers, she was treated with Adenosine and then continued with oral therapy - tablets Presolol (2x25mg 5 days and then 50+25mg to control).

Table I: Basic clinical characteristics of children presenting to the emergency department with SVT

<table>
<thead>
<tr>
<th>Clinical characteristics</th>
<th>Duration of SVT (in hours)</th>
<th>Max. HR (beats per minute)</th>
<th>EF (%)</th>
<th>FS (%)</th>
<th>Max. level of Troponin-I (µg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>8h</td>
<td>217</td>
<td>66</td>
<td>37</td>
<td>0.115</td>
</tr>
<tr>
<td>Case 2</td>
<td>12h</td>
<td>219</td>
<td>83</td>
<td>51</td>
<td>0.377</td>
</tr>
</tbody>
</table>
A cardiac troponin I was drawn at admission. The level of cTnI was 0.115 µg/l. On the fourth day the value was down to normal (0.021 µg/l). In contrast, other cardiac markers simultaneously determined from the same blood sample were within normal ranges (creatinine kinase 60 IU/l; CK-MB fraction 10.3 IU/l; Lactate dehydrogenase test 361 IU/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).

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

Figure 2: Accelerated Idioventricular Rhythm and SVES after AVRT attacks
SVES: supraventricular extrasystoles; AVRT: Atrioventricular Reentry Tachycardia
Case 2

A 13 years and 9 months-old female child was hospitalized with complaint of chest pain, palpitation and shortness of breath the last 12 hours, after emotional stress. A family history: 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=91/51mmHg, SaO2 94% , (after SVT R=19 per minute, BP=108/50mmHg, SaO2 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 electrocardiogram (Figure 3) and no abnormal changes in echocardiogram (Table I). She received the same medicine as the first case.

Figure 3: Electrocardiogram at admission to ICU showed AVNR in case 2
ICU: intensive care unit; AVNR: Atrioventricular Nodal Reentry Tachycardia

The level of cTnI was 0.377 µg/l at admission and fourth day later the value was up to 0.038 µg/l (Table I, Figure 2). Also, ProBNP was clearly elevated in the serum (1617 pg/ml) and the value was up to 286 pg/ml. During 24-hour ambulatory electrocardiogram monitoring 6 supraventricular extrasystoles were found. Also, ECG (without attack of SVT) and load test were normal.
Discussion

Cardiac troponin T and I in serum is commonly used as standard biomarker for the diagnosis of acute coronary syndrome or myocardial infarction. In adult patients with SVT, most authors posed the question are troponin levels useful for evaluating the presence of coronary artery disease\textsuperscript{14-24}? Published reports (limited case series: 1-7 patients ages 18-72) illustrate that troponins can be released because of tachycardia alone in the absence of myodepressive factors, inflammatory mediators, or coronary artery disease\textsuperscript{20-24}. The current literature on this topic shows that 12 to 48\% of adult patients will have elevated troponins after SVT. Schmitz G et al.\textsuperscript{14} in their research find troponin elevation in patients with SVT with normal coronary angiography and is 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. Causes of SVT include: lung disease, abnormal heart structure or an abnormal extra electrical 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)\textsuperscript{25,26}. The severity of SVT can vary greatly. It can last for < 30 sec. (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 showed persisted symptoms or abnormal electrocardiograms 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 was series of two hemodynamically stable patients with various troponin elevation in proportion to the duration of tachycardia. Therefore the troponin rise in our series 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. Routine testing can result in false positives 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 the best performed selectively based on presenting symptoms and risk factors for cardiac dysfunction.
Conclusion

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

References


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