Do Elevated Troponins during Supraventricular Tachycardia (SVT) Predict the Presence of Coronary Artery Disease?

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More than one third of patients will have chest pain associated with Supraventricular Tachycardia (SVT). It is unclear if Troponin markers in this setting are useful in predicting which patients will have Coronary Artery Disease (CAD). Elevated Troponins are not pathognomonic for CAD or acute coronary syndrome. In fact, modest Troponin elevation is well documented 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. Routine ordering of Troponin in patients with SVT who convert easily to sinus rhythm could potentially result in unnecessary testing and admission. Given the high incidence of Troponin elevation in patients with SVT, we sought to evaluate the literature and determine the prognostic value of Troponin assays in patients presenting to the emergency department with SVT.

The current literature on this topic is limited to case reports, case series, and retrospective reviews. Twelve to 48% of patients will have elevated Troponins after SVT [2,3,6]. There was no significant difference between the groups with a positive Troponin and negative Troponin in having coronary artery disease. One retrospective review of 104 patients demonstrated that more than two thirds of patients with evidence of myocardial ischemia/ injury by ST segment depression and Troponin I increase during SVT did not have obstructive CAD [7]. This population was noted to be heterogeneous, have a wide variety of age ranges, and high incidence of CAD. There did not appear to be any correlation with degree of Troponin elevation, substantiating what had previously been reported in a number of case series (Table 1). Based on these limited studies, there is no difference in the diagnosis of CAD based on Troponin elevation.

However, in a retrospective study by Chow et al, the authors concluded that elevated Troponin levels with SVT were associated with increased risk of mortality, defined by the primary endpoints of death, myocardial infarction, or cardiovascular rehospitalisation [8]. It should be noted though that none of their primary endpoints (death, myocardial infarction, or cardiovascular rehospitalisation) were independently noted to reach a significant value of p<0.05. On close scrutiny of this study, the only variable that was even close to reaching statistical significance was hospital re-admission rates and they were secondary to atrial fibrillation/ flutter and congestive heart failure. Furthermore, only 30% of patients in this study with SVT and elevated Troponins underwent angiography, and of those, all of them again had normal coronaries. The study was limited by its retrospective design, loss to follow-up and limited number of patients who had definitive testing and angiography.

In conclusion, further prospective study needs to be performed to determine the predictive value of elevated Troponins in patients with SVT. Based on current evidence, the routine ordering of Troponin in patients with SVT is not supported. Further testing and admission should be reserved for patients who have persistent symptoms after conversion to normal sinus rhythm, abnormal electrocardiograms after conversion to normal sinus rhythm, or those who have significant risk and pre-test probability for underlying coronary artery disease. Other patients may be appropriate for outpatient evaluation and electrophysiology studies.

References


Table 1: Studies published in patients with SVT, elevated troponin and angiography

<table>
<thead>
<tr>
<th>Author and Date</th>
<th>Study Type</th>
<th>Key Results (Angiography)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kanjwal et al. [1]</td>
<td>Case Series (7 patients)</td>
<td>All with NORMAL coronary arteries</td>
</tr>
<tr>
<td>Miranda et al. [2]</td>
<td>Case Study</td>
<td>NORMAL coronary arteries</td>
</tr>
<tr>
<td>Yeo et al. [3]</td>
<td>Case series (3 patients)</td>
<td>All with NORMAL coronary arteries</td>
</tr>
<tr>
<td>Zellweger et al. [4]</td>
<td>Case Series (4 patients)</td>
<td>2 patients with NORMAL coronary arteries on angiography and 2 patients with normal myocardial perfusion scans</td>
</tr>
<tr>
<td>Bakshi et al. [5]</td>
<td>Prospective Cohort (3 patients)</td>
<td>All with NORMAL coronary arteries</td>
</tr>
<tr>
<td>Redfearn et al. [6]</td>
<td>Retrospective Cohort (7 patients)</td>
<td>All with NORMAL coronary arteries</td>
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</tbody>
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