Causes of Increased Cardiac Troponin I Levels in Hospitalized Patients Without Coronary Artery Disease

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Keywords: cardiac troponin I, sepsis, coronary artery disease, heart failure, stroke

ABSTRACT

We reviewed the charts of 3,979 hospitalized patients who had troponin I levels measured to determine the causes of increased cardiac troponin I levels in patients without coronary artery disease (CAD) and their in-hospital mortality. An increased troponin I level was >0.4 ng/ml. Troponin I levels were increased in 581 of 3,979 hospitalized patients (14.6%) and in 199 of 3,398 patients (5.9%) without CAD. The 199 patients included 122 men and 77 women, mean age 79 years. Of the 199 patients with increased troponin I levels and no CAD, 59 (29.6%) had sepsis, 49 (24.6%) had congestive heart failure, 22 (11%) had a stroke, 18 (9%) had hypotension, 13 (6.5%) had respiratory failure, 13 (6.5%) had renal failure, 13 (6.5%) had gastrointestinal bleeding, 9 (4.5%) had ventricular or supraventricular tachyarrhythmias, and 3 (1.5%) had pulmonary embolism. Of these 199 patients, 46 (23.1%) died during hospitalization. The mean peak troponin I levels were 6.53 ± 15.1 ng/ml in the 46 patients who died during hospitalization versus 3.27 ± 5.36 ng/ml in the 153 patients who were discharged from the hospital (p not significant). In conclusion, numerous causes other than CAD may cause increased cardiac troponin I levels in hospitalized patients. The in-hospital mortality is high in these patients.

INTRODUCTION

There are numerous causes of increased cardiac troponin levels in hospitalized patients other than coronary artery disease (CAD). [1-10] These causes of increased cardiac troponin levels must be considered when evaluating hospitalized patients with increased cardiac troponin levels.

The present study was performed to determine the prevalence of increased cardiac troponin levels in hospitalized patients who had cardiac troponin I levels measured, the prevalence of different causes of the increased cardiac troponin I levels in patients who did not have CAD, and the in-hospital mortality of these patients.
MATERIALS AND METHODOLOGY

We reviewed the charts of 3,979 hospitalized patients who had cardiac troponin I levels measured at Sound Shore Medical Center, a community hospital affiliated with New York Medical College. We investigated the prevalence of increased cardiac troponin I levels in the 3,979 patients, the prevalence of increased cardiac troponin I levels in 3,398 patients without CAD, the prevalence of the different causes of increased troponin I levels in these patients, and the incidence of in-hospital mortality in these patients. An increased cardiac troponin I level was >0.4 ng/ml [5, 7] and the level considered increased by our laboratory. The diagnosis for the cause of the increased cardiac troponin I levels listed in Table 1 was made by the attending physician, stated in the chart, and reviewed by 2 of the authors. CAD was considered present if the patient had a documented history of myocardial infarction, electrocardiographic evidence of myocardial infarction, coronary angiographic evidence of CAD, a history of angina pectoris, stress test evidence of myocardial ischemia, or sudden cardiac death. The cardiac troponin I levels were measured in plasma with the blood collected in heparin-coated tubes.

Table 1. Causes of increased cardiac troponin I levels in 199 hospitalized patients without coronary artery disease

<table>
<thead>
<tr>
<th>Cause</th>
<th>Number (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sepsis</td>
<td>59 (29.6%)</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>49 (24.6%)</td>
</tr>
<tr>
<td>Stroke</td>
<td>22 (11.0%)</td>
</tr>
<tr>
<td>Hypotension</td>
<td>18 (9.0%)</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td>13 (6.5%)</td>
</tr>
<tr>
<td>Renal failure</td>
<td>13 (6.5%)</td>
</tr>
<tr>
<td>Gastrointestinal bleeding</td>
<td>13 (6.5%)</td>
</tr>
<tr>
<td>Ventricular or supraventricular tachyarrhythmias</td>
<td>9 (4.5%)</td>
</tr>
<tr>
<td>Acute pulmonary embolism</td>
<td>3 (1.5%)</td>
</tr>
</tbody>
</table>

Of the 581 patients with increased cardiac troponin I levels, 362 (65.7%) had coronary artery disease.

RESULTS AND OBSERVATIONS

Of the 3,979 patients, 581 (14.6%) had increased cardiac troponin I levels. Of the 3,398 patients without CAD, 199 (5.9%) had increased cardiac troponin I levels. These 199 patients included 122 men and 77 women, mean age 79 ± 12 years.

Table 1 includes the causes of increased cardiac troponin I levels in 199 patients without CAD. Of the 199 patients with increased cardiac troponin I levels and no CAD, 46 (23.1%) died during their hospitalization. The mean peak cardiac troponin I levels were 6.53 ± 15.1 ng/ml in the 46 patients who died during their hospitalization versus 3.27 ± 5.36 ng/ml who were discharged from the hospital (p not significant).

DISCUSSION

The present study showed that 199 of 581 hospitalized patients (34.3%) with increased cardiac troponin I levels did not have CAD. The causes of these increased cardiac troponin I levels included sepsis, congestive heart failure, stroke, hypotension, respiratory failure, renal failure, gastrointestinal bleeding, ventricular and supraventricular tachyarrhythmias, and acute pulmonary embolism. Mechanisms for causing these increased cardiac troponin I levels in patients without CAD include myocyte necrosis. [1-10] These patients without CAD had a very high in-hospital mortality.

Therefore, increased cardiac troponin levels are not specific for indicating a thrombotic acute coronary syndrome and are present in numerous disease states. Treatment of an increased cardiac troponin level must be directed to treatment of the underlying disorder. Cardiac troponin elevation in the absence of CAD has prognostic value, and its measurement is justified on this basis.

DISCLOSURES

None of the authors have any conflicts of interest.

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