

"30-minute-delta" of high-sensitivity troponin I improves diagnostic performance in acute myocardial infarction

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Introduction

Acute myocardial infarction (AMI) is a major cause of mortality and morbidity worldwide. Patients with suspected AMI must be evaluated immediately to identify life-threatening emergencies. The universal definition of MI is based on rise or fall of cardiac biomarkers such as cardiac troponin (cTn) with at least one value above the 99th percentile of the upper reference limit associated with symptoms and/or ST-T changes on electrocardiogram (ECG) suggestive of myocardial ischemi

Methods

Study patients:

We prospectively enrolled 71 consecutive patients presenting to the Hirosaki University Hospital between May 2015 and January 2016 with symptoms suggestive of AMI within 12 h from onset

A clinical assessment was performed for all patients including <u>history taking</u>, <u>physical examinations</u>, a <u>12-lead ECG</u>, laboratory test including <u>cardiac</u> <u>enzyme values</u>, <u>echocardiography</u>, and <u>coronary angiography</u>. Blood samples for routine tests were collected from all patients at presentation. The left ventricular ejection fraction (<u>LVEF</u>) was assessed by echocardiography or left ventriculography.

The final diagnosis of AMI was determined based on all available data by two independent cardiologists. A primary diagnosis of AMI was done according to the current guidelines when there was evidence of myocardial necrosis that was consistent with myocardial ischemia together with clinical symptoms and/or ECG changes suggestive of new ischemia (new ST-T changes or new left bundle branch block) or imaging evidence of a new loss of viable myocardium or a culprit coronary lesion classified according to the Ambrose criteria

Results

- Among the enrolled 71 patients, 55 (77%) were adjudicated as AMI (AMI group), and the remaining 16 were non-AMI (non-AMI group).
- The hs-cTnl levels at presentation were significantly higher in the AMI group than in the non-AMI group [306.2 (77.3–1809.9) pg/ mL versus 22.5 (7.2–115.5) pg/mL, p < 0.01] (Fig. 1A). Furthermore, the hs-cTnl levels at 30 min after presentation were also significantly higher in the AMI group than in the non-AMI group [394.5 (121.2–2136.5) pg/mL versus 24.4 (7.4–117.4) pg/mL, p < 0.01] (Fig. 1B). The "30-minute-delta," a difference between 1st and 2nd hs-cTnl values, was significantly higher in the AMI group than in the non-AMI group [54.6 (13.5–288.0) pg/mL versus 1.9 (0.6–6.3) pg/mL, p < 0.01)</p>

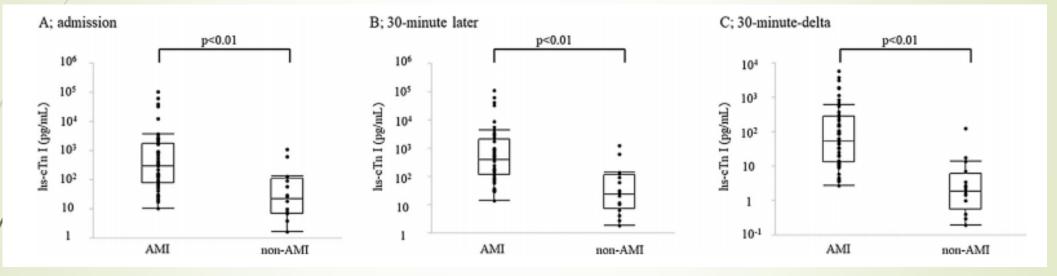


Fig. 1. Comparison of high-sensitivity cardiac troponin I (hs-cTnI) levels between AMI patients and non-AMI patients. (A) hs-cTnI levels at presentation. (B) Hs-cTnI levels 30 min after presentation. (C) "30-minute-delta" of hs-cTnI.

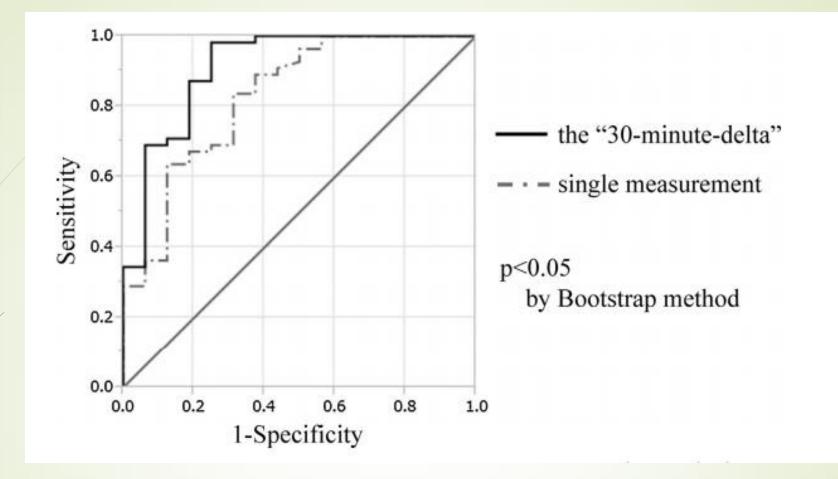


Fig. 2. Comparison of receiver operating characteristic (ROC) curves between "30-minute-delta" and the 1st high-sensitivity cardiac troponin I (hs-cTnI) value at presentation. The black line indicates the ROC curve for the delta measurement of hs-cTnI, whereas the dotted line indicates the ROC curve for a single measurement of hs-cTnI at presentation. The difference between the two areas under the curve was significant according to the bootstrap method (p < 0.05)</p>

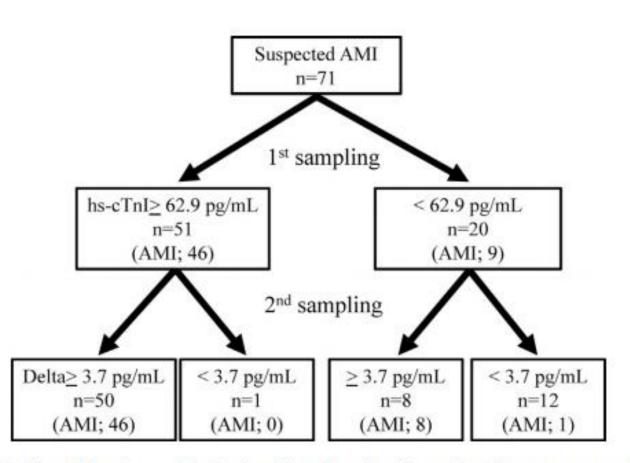


Fig. 3. The "30-minute-delta" algorithm for the diagnosis of acute myocardial infarction (AMI) according to our results. Delta indicates a difference between 1st and 2nd high-sensitivity cardiac troponin I (hs-cTnI) values.

Conclusions

The "30-minute-delta" of hs-cTnl improves the early diagnostic performance of AMI detection compared with the use of 1st hscTnl value at presentation. The "30-minute-delta" approach may lead to the earlier initiation of effective medicaltreatment aimed at better prognosis for AMI patients and contribute to reduction of unnecessary examinations