CLINICAL RESEARCH STUDY

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Clinical Characteristics and Outcomes of Patients with Myocardial Infarction, Myocardial Injury, and Nonelevated Troponins

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ABSTRACT

BACKGROUND: Cardiac troponins have emerged as the preferred biomarkers for detecting myocardial necrosis and diagnosing myocardial infarction. However, current cardiac troponin assays do not discriminate between ischemic and nonischemic causes of myocardial cell death. Thus, when an increased troponin value is encountered in the absence of obvious myocardial ischemia, a careful search for other clinical conditions is crucial.

METHODS: In 2010 to 2011, we prospectively studied hospitalized patients who had cardiac troponin I measured on clinical indication. An acute myocardial infarction was diagnosed in cases of a cardiac troponin I increase or decrease pattern with at least 1 value >30 ng/L (99th percentile) together with myocardial ischemia. Myocardial injury was defined as cardiac troponin I values >30 ng/L, but without signs or symptoms indicating overt cardiac ischemia. Patients with peak values ≤30 ng/L were classified as nonelevated cardiac troponin I. Follow-up was at least 3 years with all-cause mortality as the sole clinical end point.

RESULTS: A total of 3762 patients were included. Of these, 488 (13%) had acute myocardial infarction, 1089 (29%) had myocardial injury, and 2185 (58%) had nonelevated cardiac troponin I values. Patients with myocardial injury frequently presented with dyspnea, were older, and had more comorbidity than patients in the 2 other groups. During a median follow-up of 3.2 years, 1342 patients died. Mortality differed significantly between groups: 39% in those with myocardial infarction, 59% in those with myocardial injury, and 23% in those with nonelevated cardiac troponin I (log-rank test; $P < .0001$). No significant difference in mortality between patients with type 2 myocardial infarction and patients with myocardial injury was observed (63% and 59%, respectively).

CONCLUSIONS: Patients with myocardial injury are older and have more comorbidity than those with acute myocardial infarction. Both groups exhibit a poorer prognosis than patients with nonelevated cardiac troponin I values. Of note, a very high long-term mortality is observed in patients with type 2 myocardial infarction and patients with myocardial injury.

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Cardiac troponins I and T have emerged as the preferred biomarkers for detecting myocardial necrosis and for diagnosing myocardial infarction. In 2007, the universal definition stated that a typical increase or decrease of troponin with at least 1 value exceeding the 99th percentile along with clinical findings or symptoms of ischemia is required to confirm the myocardial infarction diagnosis.[1] In addition, the consensus document featured 5 subtypes of myocardial infarction, which were retained in the 2012 revision.[2] Over the last decade, troponin assays have undergone major improvements and can now detect even minor troponin concentrations. However, current cardiac troponin assays do not discriminate between ischemic and nonischemic causes of myocardial cell death.[3] Thus, when an increased troponin value is encountered in the absence of obvious myocardial ischemia, a careful search for other causes is crucial.

CLINICAL SIGNIFICANCE

- An acute myocardial infarction can be ruled out in more than two thirds of hospitalized patients with significant troponin elevations.
- Patients with myocardial injury most frequently present with dyspnea, are older, and have more comorbidity than patients with acute myocardial infarction.
- After at least 3 years of follow-up, patients with acute myocardial infarction or myocardial injury exhibit a significantly higher mortality than patients with nonelevated troponin values.

The aims of this prospective all-comers study were to compare clinical features and mortality of hospitalized patients with acute myocardial infarction, elevated troponin I without myocardial infarction (myocardial injury), or symptoms suggestive of acute myocardial infarction but with normal cardiac troponin I values (nonelevated troponin). Finally, we aimed to clarify the potential clinical differences between patients with type 2 myocardial infarction and patients with myocardial injury.

MATERIALS AND METHODS

Study Design and Population

The study comprises hospitalized patients at a 1000-bed university hospital (Odense University Hospital) from January 6, 2010, to January 5, 2011.[4,5] The hospital serves as the local hospital for a catchment area of 300,000 residents and is the largest in the Southern Region of Denmark. Patients who had cardiac troponin I measured on clinical indication during admission in any of the hospital's clinical departments were traced through retrieval 3 times daily at the department of clinical biochemistry. In cases of multiple admissions, only the first was considered.

Within 24 hours after the initial cardiac troponin I sampling, patients had a supplementary history taken by a dedicated study personnel paying special attention to symptoms, clinical characteristics, and comorbidity. Data registered were specified in a worksheet (**Supplementary Table 1**, available online). The study personnel did not interfere with patient management. If coronary angiography was performed, the results were collected from the Western Denmark Heart Registry.[6] Cardiovascular prescription medication reimbursed by the patients within 3 months before hospital admission and 3 months after discharge was identified through the Odense-Pharmaco-Epidemiological Database.[7] Management of patients with myocardial infarction was according to guidelines from the European Society of Cardiology.[8,9]

Inclusion Criteria

Patients were included if a physician at any of the hospital's 27 clinical departments requested a cardiac troponin I measurement.

Exclusion Criteria

Patients were excluded if they were aged less than 18 years or were residents living outside the local catchment area.

Definitions of Diagnoses

The diagnosis of acute myocardial infarction was established according to the universal definition criteria, that is, a typical increase or decrease of cardiac troponin I with at least 1 value above the 99th percentile (>30 ng/L) along with evidence of myocardial ischemia.[1] Serial cardiac troponin I measurements were performed as described earlier.[5] Patients with cardiac troponin I values >30 ng/L but without signs and symptoms of overt myocardial ischemia were defined as having myocardial injury. Patients with cardiac troponin I ≤30 ng/L were categorized as having nonelevated cardiac troponin I values.

Analysis of Cardiac Troponin I

Samples of cardiac troponin I were analyzed by Architect c16000 (Abbott Diagnostics, Abbott Park, Ill), a contemporary cardiac troponin I assay, which has a detection limit of 10 ng/L, an upper reference limit of 99th percentile of 28 ng/L, and a coefficient of variation <10% at 32 ng/L.[10] Accordingly, a cardiac troponin I value >30 ng/L was considered elevated.

Adjudication of the Diagnoses

On the basis of the prospectively obtained source information, 3 experienced local cardiologists adjudicated the diagnoses in patients with cardiac troponin I >30 ng/L. In case of diagnostic ambiguity, the Task Force Co-Chairman (KT) of the universal myocardial infarctions definition was consulted to reach consensus. The diagnoses in patients with cardiac troponin I values ≤30 ng/L were classified

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