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ORIGINAL ARTICLE

Suspected ACS Patients Presenting With Myocardial Damage or a Type 2 Myocardial Infarction Have a Similar Late Mortality to Patients With a Type 1 Myocardial Infarction: A Report from the Australian and New Zealand 2012 SNAPSHOT ACS Study

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Background Cardiac troponin (T and I) are considered the standard markers for detection of myocardial damage and the diagnosis of acute coronary syndrome (ACS) among patients who present to an emergency department with chest pain. However, these markers can be released in other situations and may be associated with short- and long-term clinical outcomes. In this study, we examine late mortality rates among patients presenting with a suspected ACS due to an unstable coronary plaque and those patients having a non-ACS.

Methods 4,388 patients were hospitalised with suspected ACS, between the 14th and 27th of May 2012 in the Australia and New Zealand SNAPSHOT ACS study. Those patients were categorised in five diagnostic groups: 1)

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ST elevation MI (n=419); 2) non-ST elevation MI (n=1012); 3) unstable angina (n=925); 4) non-ACS diagnoses (n=837); and 5) chest pain considered unlikely ischaemic (not otherwise specified, n=1195).

Result The respective mortality rates at 18 months in these groups were 16.2%, 16.3%, 6.8%, 12.8%, and 4.8%; Pearson χ^2=110 p<0.001. Among non-ACS diagnoses patients (group 4) those with the highest mortality rates (cardiac (14.4%), respiratory (18.2%), sepsis (15.4%) and neoplastic (67%) diagnoses) had the highest rates of elevated troponin levels (48%, 31%, 38% and 67% respectively). By contrast, those with the lowest mortality rates (musculoskeletal (2.9%), gastrointestinal disorders (3.9%) and non-specific chest pain (7.4%)) had the lowest rate of elevated troponin levels (9%, 18% and 15.8% respectively). However, after adjusting for baseline clinical and demographic characteristics, the mortality rate at 18 months for patients with elevated troponin was similar for ACS or non-ACS diagnoses (Hazard Ratio, 95% C.I.0.98–1.07, P=0.333).

Conclusions Among patients in the 2012 SNAPSHOT ACS study, non-ACS diagnoses characterised by high rates of elevated troponin levels had high mortality rates similar to those diagnosed with ACS. Therapies known to be effective in ACS patients, including early invasive management, should be examined in these non-ACS patients with troponin elevations within adequately powered randomised trials.

Keywords Cardiac troponin • Type 2 MI • Mortality rate • ACS

Introduction

As cardiac troponins (cTn) T and I are the most sensitive and specific biochemical markers for myocyte necrosis[1–4], the third universal definition of myocardial infarction (MI) recommends them as preferred cardiac biomarkers[5]. Assessing cTn levels in patients with chest pain potentially due to an acute coronary syndrome (ACS) is pivotal in diagnosis and management. However, troponin rises due to myocardial injury in these patients may be related to supply/demand ischaemia (type 2 MI) or injury not related to myocardial ischaemia [5]. Causative conditions of troponin elevation include cardiac conditions, such as heart failure, cardiomyopathy, pericarditis and tachyarrhythmia[4], as well as in various non-cardiac disorders such as sepsis, pulmonary embolism, tumours, stroke and renal failure [6–9].

The mortality rate increases with increasing levels of cTn, irrespective of the aetiology of the elevation [10–12]. Indeed, for a given level of cTn elevation, those patients with a non-cardiac cause may have higher mortality rates than those with an ACS [13,14]. Differentiating between type 1 and type 2 MI or other causes of myocardial injury based on troponin elevations can be challenging, even if there are sufficient samples obtained to demonstrate a characteristic kinetic profile of cTn levels, which often does not occur outside a clinical trial [15–18].

In this study, we evaluated the late mortality among patients with suspected ACS or without ACS hospital diagnoses.

Methods

Study Design and Population

The 2012 Australia and New Zealand SNAPSHOT ACS study was a prospective audit of care provided to patients admitted for a suspected or confirmed ACS within a study window of May 14th to May 27th, 2012. As previously described, 478 sites across Australia and New Zealand participated, from which 286 provided data [19]. Data entry was undertaken at sites with training and support from state co-ordinators. Late follow-up at 18 months has recently been reported [20]. Ethical approval in New Zealand allowed for a consent waiver, which also applied to in-hospital deaths in Australia. An opt-out consent process applied for all other Australian patients. Written study protocols were provided to all participating sites; participation was dependent on local resources [19].

Patient Eligibility and Classification

Consecutive first admissions within the audit period were enrolled. Patients were tracked for the duration of the acute care episode including all transfers between hospitals. Clinical characteristics, including clinical variables enabling the calculation of the global registry of acute coronary events (GRACE) risk score, as well as the logistical details of patient presentation and transfers between hospitals were recorded on a case record form (CRF). Data on the CRF included care provided at all hospitals involved in the initial acute care episode, including inter-hospital events [19].

Patients were classified by investigators at the sites (without central adjudication) with respect to primary discharge diagnosis in the following groups: Group 1, ST elevation MI (STEMI)/left bundle branch block (LBBB) required ST elevation or LBBB on an electrocardiogram (ECG) at any time during the admission, with elevated cardiac biomarkers except where patients died prior to biomarkers being drawn; Group 2, Non-ST-elevation MI (NSTEMI) required evidence of biomarker elevation with or without ECG changes consistent with ischaemia; and Group 3, unstable angina (UA), was clinically based on symptom history suggestive of myocardial ischaemia, with or without ECG changes, and without biomarker elevations. These three groups of patients were classified as having an ACS diagnosis.

There were two other broad diagnostic groups: Group 4, patients who had another diagnoses, of a non-ACS condition (see Figure 1); and, Group 5, chest pain unlikely ischaemia

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