Letter

Role of cardiac troponin as a prognosticator in critically ill patients

Andrew J Turley[1] and Jacqui A Gedney[2]

[1]Cardiology Specialist Registrar, Cardiology & General Adult Intensive Care Unit, The James Cook University Hospital, Marton Road, Middlesbrough, UK
[2]Consultant Anaesthetist and Intensivist, Cardiology & General Adult Intensive Care Unit, The James Cook University Hospital, Marton Road, Middlesbrough, UK

Corresponding author: Andrew J Turley, Andrew.turley@nth.nhs.uk

Published online: 11 November 2005
This article is online at http://ccforum.com/content/9/6/E30

Critical Care 2005, **9:**E30 (DOI 10.1186/cc3920)

See related research by King *et al.* in issue 9.4 [http://ccforum.com/content/9/4/R390]

In a recent issue of *Critical Care*, Dr King and colleagues [1] described the role of cardiac troponin as a prognosticator in critically ill medical patients. They concluded that elevated troponin levels measured on admission are associated with an increased mortality rate.

Our experience [2] supports that reported by King and coworkers. Our retrospective case note study, conducted in 180 consecutive admissions to our general (noncardiothoracic) intensive care unit over a 5-month period, identified 62 patients with a raised troponin T (TnT) level. The all-cause mortality rate was 51.6% in those patients with a raised TnT, as compared with 20.3% in patients with no TnT elevation ($P < 0.001$). The median duration of admission was 5.5 days for patients with a raised TnT and 3 days for patients with a normal TnT ($P < 0.003$). In over 70% of cases the raised TnT occurred within the first 72 hours of admission.

Other groups have also reported that elevated biochemical markers of cardiac myocyte damage are common in critically ill patients and are associated with increased mortality [3]. TnT and troponin I are sensitive and specific for myocardial injury, even at the microscopic level. Although the cardiac troponins are cardiospecific, in many critically ill patients an elevated troponin level will not reflect myocardial ischaemia secondary to obstructive coronary artery disease, as in the setting of acute coronary syndrome (ACS) [3]. In one study [4] 55% of critically ill patients with raised troponins fulfilled criteria for myocardial infarction, and this group had the worst outcome. It is likely, but not proven, that in those patients without myocardial infarction cardiac troponin level represents another surrogate marker of disease severity.

In spite of the different trigger for troponin release in this latter group (i.e. commonly sepsis in critically ill patients versus atheromatous coronary plaque in the ACS population), there may be similarities between the pathophysiology of ACS and that of myocardial dysfunction in the critically ill. Research in ACS patients has revealed complex associations between cardiac biomarkers and outcome, reflecting the different pathophysiological axes that are involved in the ACS setting. The simultaneous measurement of three biomarkers provides independent prognostic information [5]. Troponin is a marker of myocardial necrosis, high sensitivity C-reactive protein is a marker of inflammation, and B-type natriuretic peptide is part of the neurohormonal axis and reflects ventricular loading. The number of elevated biomarkers allows patients to be risk stratified for short-term (30 days) and long-term (>6 months) adverse events, including death, myocardial infarction and congestive cardiac failure, with a fivefold range of risk identified. Also, with each additional elevated biomarker, the risk for death is almost doubled.

Further research is required to unravel the complex pathophysiology that leads to troponin release in the critically ill. However, we believe that it is likely that troponin release forms one part of a much larger puzzle.

Competing interests

The author(s) declare that they have no competing interests.

References

1. King DA, Codish S, Novack V, Barski L, Almog Y: **The role of cardiac troponin I as a prognosticator in critically ill medical patients: a prospective observational cohort study.** *Crit Care* 2005, **9:**R390-R395.
2. Turley AJ, Gedney J: **Elevated troponin T levels in patients with and without renal dysfunction in a general intensive care unit.** *Crit Care* 2004, **8:**S41.
3. Guest TM, Ramanathan AV, Tuteur PG, Schechtman KB, Ladenson JH, Jaffe AS: **Myocardial injury in critically ill patients. A frequently unrecognized complication.** *JAMA* 1995, **273:**1945-1944.
4. Lim W, Qushmaq I, Cook DJ, Crowther MA, Heels-Ansdell D, Devereaux PJ; for the Troponin T Trials Group: **Elevated troponin and myocardial infarction in the intensive care unit: a prospective study.** *Crit Care* 2005, **9:**R636-R644.
5. Sabatine MS, Morrow DA, de Lemos JA, Gibson CM, Murphy SA, Rifai N, McCabe C, Antman EM, Cannon CP, Braunwald E: **Multimarker approach to risk stratification in non-ST elevation acute coronary syndromes: simultaneous assessment of troponin I, C-reactive protein, and B-type natriuretic peptide.** *Circulation* 2002, **105:**1760-1763.