Commentary

# Myocardial infarction on the ICU: can we do better?

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See related research by Lim *et al*., http://ccforum.com/content/12/2/R36

## Abstract

Myocardial infarction remains a major cause of death despite contemporary therapeutic strategies. Diagnosis in the intensive care unit is challenging, but is essential to target therapy accurately. In this issue of *Critical Care* Lim and colleagues present the results of a prospective non-interventional screening study for acute myocardial infarction in patients admitted to the intensive care unit. Myocardial infarction is observed to occur frequently, often without being clinically apparent, with a high associated mortality. Such approaches may facilitate accurate diagnosis of myocardial infarction in this setting, hence opening the way to improved therapy.

Myocardial infarction (MI) in the critically ill presents a diagnostic challenge to the physician and is associated with a particularly adverse outcome for the patient [1]. Such patients have high metabolic demands and are often subject to sustained adverse physiology. Typical signs and symptoms can be difficult to elicit and surrogate physiological markers of impaired coronary perfusion masked or misinterpreted in the context of the index pathology. Cardiac troponin measurements and the 12-lead echocardiogram (ECG) remain sensitive in this setting, but specificity decreases, resulting in diagnostic uncertainty.

Recent consensus guidelines from the European Society of Cardiology, American College of Cardiology Foundation, American Heart Association and World Heart Federation emphasise the role of cardiac biomarkers in defining MI [2]. Diagnosis requires a rise and/or fall in serum levels (preferably troponin) together with evidence of myocardial ischaemia defined: clinically by patient history; electrocardiographically (new ST-T wave changes, new left bundle branch block or evolving pathological Q waves); or by imaging evidence of new regional wall motion abnormality.

Current troponin assays provide a highly sensitive marker of even microscopic levels of myocardial necrosis [3]. This does not define the mechanism of injury, however, and troponin elevation is reported in a variety of non-acute coronary syndrome (non-ACS) pathologies common in the intensive care unit (ICU), including pulmonary embolus, severe sepsis and renal impairment [4,5]. All-cause mortality and duration of ICU admission are increased in critically ill patients with elevated troponin, irrespective of the cause. Lim and colleagues [6] have previously reported on a meta-analysis of 20 studies with 3,278 general ICU patients, where the median incidence of troponin-positivity was 43%. This was associated in an adjusted analysis of 6 of these studies (1,706 patients) with a significant increase in mortality (odds ratio of dying 2.5, 95% confidence interval (CI) 1.9 to 3.4; $p < 0.001$), and in a further unadjusted analysis of 8 of these studies (1,019 patients) with an increase in ICU stay (3 days, 95% CI 1 to 5.1, $p = 0.004$) and a trend towards longer overall hospital admission (2.2 days, 95% CI -0.6 to 4.9; $p = 0.12$). Whether the adverse outcome was due to concomitant ACS, or the severity of the index condition, resulting in troponin elevation, is a critical question in targeting appropriate therapies.

The problems of troponin specificity dictate the requirement for additional diagnostic criteria in defining MI, and nowhere is this more true than on the ICU. Clearly, treatment strategies appropriate for ACS may not improve outcome where elevated troponin is due to an alternative pathology.

Myocardial ischaemia in the setting of mechanical ventilation and weaning is well described [7,8]. Contemporary analyses of ICU patients with current definitions of myocardial infarction are limited. Booker and colleagues [9] prospectively screened 76 consecutive patients admitted to a general ICU. ST-segment changes on continuous telemetry and 12-lead ECGs for the first 24 to 48 hours of admission were recorded with troponin I (TnI) assays 8 to 12 hours after monitoring. There were 37 ECG-defined ischaemic events detected in 8 patients (10.5%), of which 96% were

ACS = acute coronary syndrome; CI = confidence interval; ECG = echocardiogram; ICU = intensive care unit; MI = myocardial infarction.

asymptomatic. Out of the 8 patients, 6 had significant troponin I elevation, and this accounted for 50% of all troponin-positive results. More recently, Lim *et al*. [1] reported on the combined results of ECG, troponin testing and new regional wall motion defects on echocardiography in general ICU patients. Investigations were clinically driven, but of 93 patients included, 24 (25.8%) were diagnosed with coincident MI, and this was associated with a significantly higher ICU (37.5% versus 17.6%; $p = 0.05$) and in-hospital (50% versus 22%; $p = 0.01$) mortality, even after correction for APACHE II score and inotrope/vasopressor requirement.

The current paper by Lim and colleagues [10] continues a series of publications from the McMaster group analysing MI and the diagnostic components in the critically ill patient. A robust screening protocol for MI was devised, defined according to consensus-guidelines by the presence of positive troponin assay and ischaemic ST-T wave changes on ECG. The study includes 103 patients admitted to general ICU and enrolled over a two month period. Serial 12-lead ECGs and cardiac troponin T (cTnT) assays were performed. Tests were performed additionally, and blinded to ICU staff, if not ordered on clinical grounds. Only one patient had an index diagnosis of MI.

Patients were analysed according to: diagnosis of MI (35.9% of patients); presence of positive troponin only (14.6%); and troponin-negative status (49.5%). ICU staff made a clinical diagnosis of MI in 18 patients (17.5%), although 4 did not fulfil diagnostic criteria. Screening identified an additional 23 patients with true infarction. MI was associated with a longer ICU stay (median 5 versus 2 days; $p = 0.001$) and increased hospital mortality (43.2% versus 2%; $p < 0.0001$) compared to troponin-negative patients. MI patients also required a longer period of mechanical ventilation (median 4 days versus 2 versus 1; $p < 0.0001$) with increased ICU mortality (37.8% versus 6.7% versus 2.0%; $p < 0.0001$) compared to the troponin positive and negative groups, respectively.

This paper supports existing literature regarding adverse outcomes for ICU patients with coincident MI, and importantly highlights the additional detection rate afforded by simple screening investigations. Whether this will translate into better patient outcomes through targeted therapy - pharmacological and interventional - will surely be the subject of future studies. Interestingly, in the current paper, mortality rates were similar for MI patients irrespective of whether this was diagnosed prospectively by ICU staff or not. Indeed, there was actually a trend towards better outcome in those not identified, which one would speculate reflects a less unwell subset of patients with smaller infarcts (rather than any iatrogenic effect).

Invasive strategies (coronary angiography and percutaneous intervention) have a clear role in the patient with ACS outside of the ICU. Their role in the ICU setting is less clear - although the potential benefits are very high, so are the risks. Interventional approaches in this population are beset with difficulties arising from the lack of specificity of troponin elevation, and the difficulty in early diagnosis of MI. The work of Lim and colleagues [10] provides further clarification as to which patients could be targeted; further studies are required to ascertain the way in which this should be undertaken.

## Competing interests

The authors declare that they have no competing interests.

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