Commentary

Myocardial infarction complicating critical illness

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Abstract

Cardiac troponins are highly sensitive and specific indicators of myocardial injury. Although the mechanism of this injury is not entirely clear, it carries important prognostic information. Elevated serum levels of cardiac troponins have been described in a wide variety of conditions other than myocardial infarction (MI). The current study is an important first step in trying to determine the exact frequency of MI among critically ill patients with elevated troponin. At present, the rate of MI in these patients is unknown and its implications on outcome and management will have to await future prospective clinical trials.

In this issue of *Critical Care*, Lim and colleagues examine the frequency of myocardial infarction (MI) among a non-selected group of critically ill patients [1].

Cardiac troponins I (cTnI) and T, myocardial regulatory proteins of the thin actin filament, are considered highly sensitive and specific indicators of myocardial injury. Over the past decade, measuring cardiac troponin levels has become the corner stone of detecting myocardial injury to the extent that it is now an inseparable part of the current guidelines for diagnosis and management of acute coronary syndromes (ACS) and MI [2].

It is well established that reasons other than thrombotic MI can cause elevated serum levels of cardiac troponins [3,4]. Such elevation has been described in severe sepsis, pulmonary thromboembolism, and a wide variety of additional conditions. The mechanism responsible for this myocardial injury is unclear. Aggravation of pre-existing ACS in the context of extreme stress associated with critical illness and uncoupling of oxidative phosphorylation during sepsis have been described [5]. Systemic inflammatory response syndrome-induced cytokine-mediated (lipopolysaccarhides, tumor necrosis factor-α, interleukin-1β, and interleukin-6) direct myocardial injury and increase in intra-cellular calcium in cardiac cells have also been implicated [6,7]. Regardless of the mechanism, it is widely accepted that in addition to indicating myocardial injury, troponin elevation provides prognostic information. Debate still exists though whether such elevation is an independent outcome predictor or should be viewed as a surrogate of organ failure in the broader context of multi-organ dysfunction. Whereas some have suggested that cTnI levels correlate with myocardial damage and poor outcome [8,9], the study by Lim and colleagues [1], as well as studies by our group and others [10,11], could not confirm this association. Recently, the natriuretic peptides have emerged as promising prognostic markers in patients with congestive heart failure, chronic ischemic heart disease and ACS as well as in patients with severe sepsis [12-14]. These data are consistent with the notion that a biological marker could be of supplementary value in assessing prognosis, and myocardial dysfunction.

To what extent does troponin elevation in critically ill patients reflect a thrombotic ACS is obviously a different question. It can be easily postulated that patients with critical coronary lesions are more likely to develop a thrombotic MI while critically ill. The imbalance between pro- and anti-coagulant mechanisms as well as endothelial dysfunction so characteristic of sepsis may play an important role in limiting coronary flow. In addition, tachycardia hypoxemia and diminished oxygen delivery may tip the balance of regional myocardial oxygen consumption over the critical edge. Bhatti and colleagues [15] showed that, among patients admitted to an intensive care unit (ICU) due to gastrointestinal bleeding, those with risk factors for ischemic heart disease developed ACS more frequently than those without and that this subgroup tended to have longer ICU stay. Another observational cohort study suggested that the occurrence of clinically recognized cardiac dysfunction is common (21.2%) among critically ill medical patients and is an independent determinant of hospital mortality. The finding of acute cardiac

ACS = acute coronary syndrome; cTnI = cardiac troponin I; ECG = electrocardiogram; ICU = intensive care unit; MI = myocardial infarction.

injury, assessed using serial blood measurements of cTnI, was also common (15.8%), but did not independently contribute to hospital mortality [11].

It is clear that elevated troponin levels alone are unable to differentiate between thrombotic and non-thrombotic etiologies. In their article, Lim and colleagues [1] take an important first step in trying to answer a vital question; what is the true frequency of MI among acutely ill patients with elevated troponin? The *a priory* defined criteria used to diagnose MI were a combination of elevated troponin and electrocardiogram (ECG) changes as well as echocardiographic evidence of new myocardial wall motion anomalies. They found that of the 93 patients for whom both troponin measurements and ECG recordings were available, 44 (47.3%) had elevated troponin levels and 24 (25.8%) had MI. Although troponin elevation was not an independent outcome predictor, MI was associated with a significantly higher mortality rate and was found to be an independent predictor of hospital mortality [1]; however, only 23 patients had echocardiograms and none had angiography performed. Moreover, no data were provided indicating whether the wall motion abnormalities found were diffuse or segmental and, importantly, whether a correlation was found between ECG territory and echocardiograms. Even though the ECG criteria were defined as either ST elevation or depression, most MIs were eventually categorized as non-ST MIs. Thus, an unequivocal determination of the exact frequency of MI in the ICU setting is not presently possible.

The importance of ACS complicating critical illness is beyond merely an additional prognostic marker. Ultimately, therapeutic modalities such as anti-platelet agents, beta blockers whenever relevant, statins and possibly even revascularization once the patient is stable enough, may all become relevant if we wish to change a patient's course and outcome. At present, however, the exact frequency of MI in the setting of critical illness, the best way to diagnose it and its implications on outcome and management strategies will have to await future prospective clinical trials.

Competing interests

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

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