

Letter

Cardiac troponin level is not an independent predictor of mortality in septic patients requiring medical intensive care unit admissionFrançois G Brivet¹, Frédéric M Jacobs¹, Patrice Colin², Dominique Prat¹ and Bogdan Grigoriu¹¹Medical Intensive Care Unit – Hôpital Antoine Bécère, Assistance Publique-Hôpitaux de Paris, France²Cardiologic Department – Hôpital Antoine Bécère, Assistance Publique-Hôpitaux de Paris, and Paris XI University, Paris, FranceCorresponding author: François G Brivet, francois.brivet@abc.aphp.fr

Published: 2 February 2006

This article is online at <http://ccforum.com/content/10/1/404>

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Critical Care 2006, **10**:404 (doi:10.1186/cc3990)See related letter by Turley and Gedney in issue 9.6 [<http://ccforum.com/content/9/6/E30>] and related research by King *et al.* in issue 9.4 [<http://ccforum.com/content/9/4/R390>] and Lim *et al.* in issue 9.6 [<http://ccforum.com/content/9/6/R636>]

Turley and Gedney claim that the cardiac troponin Ic level is a predictor of adverse outcome [1] and that their results support those of King and colleagues [2]. Unfortunately, King and colleagues were unable to demonstrate a link between the troponin Ic level and mortality when using a multivariate model including the Acute Physiology Age and Chronic Health Evaluation II score. Interpretation of the results of Turley and Gedney therefore requires caution since their results are based only on univariate analysis [1].

We prospectively evaluated the accuracy of cardiac troponin Ic levels to predict inhospital mortality in 118 adults with documented sepsis and no history of cardiac arrest or acute coronary syndrome, and we then compared this accuracy with the performance of the new Simplified Acute Physiology II score. Seventy-five patients (63.6%) were in shock (systolic blood pressure <90 mmHg), while 58 patients (49%) had a cardiac troponin Ic level greater than 0.3 ng/ml (detection limit, 0.15 ng/ml) and 43 patients (36.4%) a level greater than 1 ng/ml. Nonsurvivors were more severely ill (Simplified Acute Physiology II score [mean \pm standard deviation], 74.7 ± 25.2 versus 51.5 ± 18.4 ; $P < 0.001$), tended to have higher cardiac troponin Ic levels (5.5 ± 10.3 ng/ml versus 3.6 ± 8.9 ng/ml) and tended to be older. Patients with a cardiac troponin Ic level above 0.3 ng/ml had a twofold risk of dying (odds ratio, 2.56; 95% confidence interval, 1.89–5.542), but the predictive abilities for mortality of cardiac troponin Ic were poor according to the area under the curve of the receiver–operating characteristic curve (0.612; 95% confidence interval, 0.504–0.719) – in contrast to the Simplified Acute Physiology II score (0.775; 95% confidence interval, 0.686–0.864). A logistic regression model identified only the Simplified Acute Physiology II score as an independent predictor of death ($\beta = 0.048$; odds ratio, 1.049; 95% confidence interval, 1.028–1.075; per point increment, $P < 0.001$).

We confirm that nearly one-half of patients with severe sepsis have an elevated cardiac troponin Ic level within the first

72 hours of the intensive care unit stay [3–7]. Whereas there is convincing evidence that the cardiac troponin Ic level reflects and correlates with myocardial damage in septic patients whatever the mechanism [3–6] and that every elevated troponin level should not be diagnosed or treated as acute coronary syndrome [7], the impact of an elevated troponin level on the outcome remains a matter of debate, even in studies performing multivariate analysis [2,3,5–7]. Although two series found that mortality was dependent on the troponin level [3,5], like other studies [2,6,7] we failed to reach a similar conclusion. In our opinion, the troponin level can be useful to assess the risk of myocardial dysfunction in patients with sepsis but is not an independent marker of mortality.

Competing interests

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

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