

Creatinine Kinase Isoenzyme MB: A Simple Prognostic Marker for Pulmonary Embolism

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Dear Editor,

Acute pulmonary embolism (PE) is the third most frequent cardiovascular disease characterised by high morbidity and mortality. In PE, the pulmonary arterial bed is occluded by thrombi, and different mediators such as thromboxane and serotonin released are associated with vasoconstriction. The obstruction and vasoconstriction of arterial bed lead to an increase in pulmonary vascular resistance, right ventricular (RV) pressure overload, and RV dilatation. RV dilatation increases RV wall tension and myocyte stretching. All of these mechanisms result in RV ischemia and myocardial injury. Therefore, RV dysfunction is an important predictor of mortality in PE [1, 2]. RV dysfunction may be determined by many laboratory markers such as brain natriuretic peptide, N-terminal -pro BNP, cardiac troponin I or T, and heart-type fatty acid-binding protein [3, 4].

Creatinine kinase isoenzyme MB (CK-MB) is a useful biomarker for detecting myocardial injury [5]. To our knowledge, the relationship between admission CK-MB level and in-hospital and long-term clinical outcomes in PE patients who were treated with thrombolytic therapy was investigated in only one study. We reported firstly the impact of admission CK-MB levels and in-hospital and long-term unfavourable clinical outcomes in PE patients

who were treated with thrombolytic therapy [6]. A total of 148 consecutive confirmed acute PE patients who were treated with thrombolytic tissue-plasminogen activator agents were enrolled in this study. Pulmonary multi-slice CT angiography was used to diagnose PE. The study population was divided into two tertiles, based on their admission CK-MB levels. The high CK-MB group ($n = 35$) was defined as having a CK-MB level in the third tertile (>31.5 U/l), and the low group ($n = 113$) was defined as having a level in the lower two tertiles (≤ 31.5 U/l). Patients in the high CK-MB group had a higher incidence of hypotension, shock, use of inotropic drugs, in-hospital haemodialysis treatment, and mechanical ventilation. In-hospital mortality was significantly higher in the high CK-MB group (37.1 vs. 1.7 %; $p < 0.001$). Admission systolic blood pressure and tricuspid annular plane systolic excursion were lower in the high CK-MB group. The CK-MB level of 31.5 IU/l was identified as an effective cut-off point for in-hospital mortality rate (area under curve = 0.893; 95 % CI 0.805–0.98; $p < 0.001$) and it had a sensitivity of 86.7 % and specificity of 83.5 %. The mean follow-up period was 30 months. During long-term follow-up, mortality, recurrent PE, major and minor bleeding, use of fresh frozen plasma, and history of international normalised ratio >5 were similar between the two groups. In summary, CK-MB is an inexpensive, widely available, and strongly prognostic marker for patients with PE. However, at long-term follow-up, CK-MB did not predict either adverse clinical outcomes or mortality.

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Compliance with Ethical Standards

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