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## Prognostic factors in acute pulmonary embolism. Part II: The role of troponins

**The presence of right ventricular dysfunction markedly increases mortality in normotensive patients with pulmonary embolism. Submassive pulmonary embolism is defined by stable hemodynamics and presence of right ventricular dysfunction. A further important risk factor are troponins.**

Giannitsis et al<sup>1</sup> found as early as in 2000 that elevation of troponin T (cTnT) discriminates patients with acute pulmonary embolism with high or low mortality. The mortality in patients with cTnT values  $\geq 0.1$  ng/ml was 44%, vs 3% mortality in patients with cTnT  $< 0.1$  ng/mL.

The prognostic value of troponins in acute pulmonary embolism has recently been evaluated in a meta-analysis including data from 20 studies.<sup>2</sup> Elevated troponin levels were significantly associated with short-term mortality (odds ratio (OR), 5.24; 95% CI 3.28 to 8.38), with death resulting from pulmonary embolism (OR, 9.44; 95% CI 4.14 to 21.49), and with adverse outcome events (OR, 7.03; 95% CI, 2.42 to 20.43). However the data in this meta-analysis also include patients with massive pulmonary embolism. It is known that the mortality in acute massive pulmonary embolism is high, approximately 30%, and in patients with cardiogenic shock it may even reach 50%. In these patients we do not need any risk stratification; because that is based on the clinical evaluation.

More important are studies performed only in normotensive patients. Four such studies were included in the meta-analysis.<sup>3-6</sup> Elevated troponin levels were associated with a high mortality even in this subgroup of hemodynamically stable patients (OR, 5.90; 95% CI, 2.68 to 12.95).<sup>2</sup>

The elevation of troponin T was, in a study of normotensive patients with acute pulmonary embolism, the only indicator of adverse hospitalization outcome.<sup>3</sup> Hospital mortality was 25% in patients with elevated troponin T (values  $> 0.01$  ng/mL),<sup>3</sup> but all normotensive patients without elevated troponin T survived.

Janata et al<sup>7</sup> confirmed that the severity of pulmonary embolism correlates with the elevation of troponin I. Only values of troponin T  $\geq 0.09$  ng/mL were a marker for hospital mortality. Their negative predictive value was 0.99.

Elevation of troponins is most probably caused by right ventricular microinfarctions due to increased right ventricular pressures, leading to an elevation of oxygen consumption in the right ventricle, hypoxemia, systemic hypotension, presence of asymptomatic coronary artery disease, and a decrease in cardiac output.

The elevation of troponins T or I correlates positively, both with the ECG signs of right ventricular overload and with the right ventricular dysfunction observed at echocardiographic examination. Elevated cardiac troponins are a marker of right ventricular involvement and severity of pulmonary embolism, even in patients without coronary artery disease.

**For the stratification of normotensive patients with acute pulmonary embolism, the maximum ability shows a combined use of echocardiography and troponins. In acute pulmonary embolism with both of these adverse findings, survival is markedly decreased at 30 days<sup>8</sup> after the diagnosis; thus, thrombolytic treatment may be indicated.** At present, an individual approach to the indication is necessary, but a prospective, multicenter, double-blind trial comparing tenecteplase + heparin vs heparin + placebo in 1000 patients with submassive pulmonary embolism defined by echocardiographic right ventricular dysfunction and elevated troponins is already under way.<sup>2</sup>

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