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Letter to the Editor

Right ventricle injury during acute pulmonary embolism leads to its remodeling

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Abstract

Right ventricular (RV) overload and hypoxia in acute pulmonary embolism (APE) may lead to RV myocardium injury reflected by elevated cardiac troponin levels. We studied 26 patients aged 57.2±17.8 years with first episode of APE. On admission troponin T (TnT) was measured. Transthoracic echocardiography was performed after 6 months of anticoagulation. Myocardial injury (TnT ≥0.03 ng/ml) was observed in 8 (30.8%) patients at the diagnosis. At follow up RV diastolic area tended to be larger in group with myocardial injury (25.0 (20.8–38.6) vs 18.4 (17.7–23.3) cm^2, $p=0.06$). Tricuspid annulus systolic velocity at tissue Doppler was lower in group with myocardial injury (0.12 (0.11–0.13) vs 0.15 (0.13–0.21) m/s, $p=0.04$), while no such a relationship was found for mitral annulus systolic velocity. TnT concentration correlated with RV diastolic area ($r=0.61$) and tricuspid annulus systolic velocity ($r=-0.58$) although not significantly ($p=0.08$ and $p=0.09$. respectively). Our data suggest that RV injury in acute phase of PE may lead to its remodeling.

Keywords: Acute pulmonary embolism; Right ventricle; Myocardial injury

In acute pulmonary embolism (APE) myocardial injury [1] or even RV infarctions [2] were observed. Since left ventricular infarction may lead to its remodeling, it seems justified to hypothesise that RV injury in APE may influence RV morphology and function at follow up. Complex RV structure limits echocardiographic evaluation of its function, such as ejection fraction. Moreover, RV contraction pattern varies from that of LV. RV ejects blood mainly by shortening of its longitudinal axis, while LV mainly by the reduction of transverse diameter [3]. It was demonstrated that myocardial velocities measured with tissue Doppler at the level of tricuspid annulus precisely reflect RV systolic function [4,5].

We studied 26 patients aged 57.2±17.8 years, 6 months the after first episode of APE confirmed at spiral CT. Patients after acute coronary syndromes, with LV ejection fraction <50% and valvular abnormalities with obstructive pulmonary diseases were not included. At the diagnosis and 6 months later transthoracic echocardiography to assess RV overload was performed as described previously [6]. In the 6th month also tissue Doppler tracings of mitral and tricuspid annuli were recorded (mean of 3 values measurements at end-expiration).

On admission TnT concentration was assessed (ECLIA, Roche; detection limit ≥0.01 ng/ml). Serum TnT ≥0.03 ng/ml was considered to indicate myocardial injury (group TnT+) [7].

Patients were treated according to the current guidelines [8]. After 6 months of anticoagulation spiral CT was repeated to detect pulmonary artery thrombi.

Clinically submassive APE was diagnosed in 12 (46.2%) patients, nonmassive APE in 14 (53.8%) patients. Three (11.5%) patients, with submassive APE received thrombolysis because of progressive deterioration.

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