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Regular Article

Endothelin is not elevated in acute pulmonary embolism

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ABSTRACT

Introduction: In acute pulmonary embolism (APE) the increase of pulmonary vascular resistance depends on the thromboli load and potentially on the pulmonary bed contraction caused by neurohormonal reaction. Plasma levels of endothelin were reported to be elevated in pulmonary arterial hypertension. However, there are only a few studies assessing endothelin in patients with APE.
Materials & Methods: Therefore in our study we evaluated endothelin concentration in 55 patients (29M, 26F, age 57 ± 19 yrs) with confirmed APE for potential value in risk stratification. Patients were compared with 24 healthy volunteers at similar age. On admission blood samples were collected for plasma endothelin concentration. The quantitative assessment of right ventricular (RV) function was performed by echocardiography.
Results: Endothelin concentrations were similar in APE patients and in control group (1.41(0.22-9.68)pg/mL vs. 1.62(0.27-8.92)pg/mL; p = NS). There was no differences in endothelin levels between APE patients with and without RV dysfunction (1.46(0.38-4.54)pg/mL vs. 1.41(0.22-9.68)pg/mL; p = NS). Endothelin concentration did not differ between patients with serious adverse events and APE group with event-free clinical course (3.19(0.38-4.27)pg/mL vs. 1.38(0.22-9.68)pg/mL; p = NS). There was no significant correlation between endothelin levels and blood saturation, time from the first symptoms, heart rate, blood pressure, tricuspid valve regurgitation pressure gradient and other echocardiographic parameters.
Conclusions: We concluded that plasma endothelin concentrations assessed on admission are not elevated in patients with APE and it does not play as important role in acute phase of increase of pressure in pulmonary arteries as in chronic pulmonary hypertension.

Introduction

The main reason of fatal outcome in acute pulmonary embolism (APE) is sudden increase in pulmonary arteries resistance resulting in acute right ventricular failure [1]. The increase of pulmonary vascular resistance depends not only on the degree of thromboemboli burden, but also on the vasoconstriction caused by neurohormonal reaction [1,2]. Plasma levels of endothelin (ET), a potent constrictor of pulmonary vascular bed were reported to be elevated in patients with pulmonary arterial hypertension. Moreover, treatment with bosentan, an endothelin receptor antagonist was found not only to decrease pulmonary resistance and to improve functional capacity,[3] but it also decreased ET and brain natriuretic levels [4]. However, there are only a few studies assessing potential role of endothelin in APE [5–7]. Therefore, in our study we evaluated plasma endothelin concentration in patients with APE, its potential role in hemodynamic alterations and a prognostic value for 30-days risk stratification.

Material and Methods

Patients and management of pulmonary embolism

The study population comprised consecutive patients admitted to our department with APE, confirmed by contrast-enhanced spiral computed tomography (16MSCT, GE Bright Speed "G"). Acute PE was diagnosed when symptoms of PE before the diagnosis lasted no longer than 14 days. On admission clinical data and blood samples were collected. Echocardiography for right ventricular function was performed within 24 hours after the diagnosis.

Predefined endpoints of the study included all-cause mortality, and serious adverse events (SAE). SAE comprised at least one of the following in-hospital adverse clinical events: death, cardiopulmonary resuscitation, thrombolysis, or the need for intravenous catecholamines infusion. All events were recorded up to 30 days after the diagnosis of APE. High, intermediate and low risk APE groups were

Abbreviations: APE, acute pulmonary embolism; ET, endothelin; RV, right ventricle; SAE, serious adverse events.

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