Mean platelet volume predicts early death in acute pulmonary embolism

Maciej Kostrubiec, Andrzej Łabyk, Justyna Pedowska-Włoszek, Anna Hrynkiewicz-Szymańska, Szymon Pacho, Krzysztof Jankowski, Barbara Lichodziejewska, Piotr Pruszczyk

Department of Internal Medicine and Cardiology, The Medical University of Warsaw, Warsaw, Poland

Correspondence to
Dr Maciej Kostrubiec, Department of Internal Medicine and Cardiology, The Medical University of Warsaw, ul. Lindleya 4, Warszawa 02-005, Poland;
maciej.kostrubiec@wum.edu.pl

Accepted 27 October 2009
Published Online First 11 November 2009

ABSTRACT

Background Recently, mean platelet volume (MPV) was reported to predict venous thromboembolism. Moreover, MPV correlates with platelet reactivity and indicates poor outcome in acute coronary syndromes.

Objective To examine the hypothesis that in acute pulmonary embolism (APE) MPV is elevated and may predict mortality.

Methods and results The study included consecutive 192 patients with APE, (79M/113F, 64±18 years) and 100 controls matched for age, sex and concomitant diseases. On admission blood samples were collected for MPV and troponin measurements. Although MPV did not differ between patients with APE and controls (10.0±1.2 vs 10.1±0.8 fl), it differed between low- and intermediate- or high-risk APE (9.4±1.2 fl, 10.3±1.1 fl, 10.3±1.8 fl; respectively, $p<0.0001$). Eighteen (9%) patients with APE died during the 30-day observation. MPV was higher in non-survivors than survivors (10.7±1.4 fl vs 9.9±1.2fl, $p<0.01$). The areas under receiver operating characteristic curves of MPV were 0.658 (95% CI 0.587 to 0.725) for predicting 30-day mortality, and 0.712 (95% CI 0.642 to 0.775) for 7-day mortality. MPV >10.9 fl, showed sensitivity, specificity, positive predictive value and negative predictive value for death within 30 days (39%, 81%, 18%, 93%, respectively) and for 7-day mortality (54%, 82%, 18%, 96%). Multivariable analysis showed that MPV was an independent mortality predictor for 7- and 30-day all-cause mortality (HR=2.0 (95% CI 1.3 to 3.0), $p<0.001$)) and 1.7 (95% CI 1.2 to 2.5), $p<0.01$)), respectively). MPVs were higher in patients with myocardial injury than in those without troponin elevation (10.2±1.1 fl vs 9.8±1.2 fl; $p=0.02$). There were correlations between MPV and right ventricular diameter and right ventricular dysfunction ($r=0.28$, $p<0.01$ and $r=0.19$, $p<0.02$, respectively).

Conclusion MPV is an independent predictor of early death in APE. Moreover, MPV in APE is associated with right ventricular dysfunction and myocardial injury.

INTRODUCTION

Platelets have an important role in the pathogenesis of atherothrombosis. It has been shown that platelet size, measured by mean platelet volume (MPV), correlates with their reactivity.[1] MPV is positively associated with indicators of platelet activity, including expression of glycoprotein Ib and glycoprotein IIb/IIIa receptors, and can be regarded as a marker of platelet activation.[2] Increased values of MPV have been recognised as an independent risk factor for myocardial infarction and stroke.[3–5] Moreover, increased MPV was associated with poor clinical outcome in survivors of myocardial infarction[6 7] and with the severity of acute ischaemic cerebrovascular events.[8] Therefore, MPV is still regarded as an easy, useful tool for indirect monitoring of platelet activity in different situations. Recently, increasing levels of MPV were identified as a predictor for venous thromboembolism—in particular, venous thromboembolism of unprovoked origin.[9] Moreover, platelet activation was also observed in patients after acute pulmonary embolism (APE), and correlated with the severity of right ventricular (RV) dysfunction.[10] In our study we tested the hypothesis that in patients with APE, MPV is elevated and may predict mortality. We also examined whether platelet activation assessed by MPV on admission may be a predictor of mortality in patients with APE.

MATERIAL AND METHODS

Patients and management of pulmonary embolism

The study population comprised consecutive patients with APE admitted to our department between 2006 and 2009. APE was confirmed by contrast-enhanced spiral CT. APE was diagnosed when symptoms of PE had been present for no longer than 14 days before the diagnosis. Clinical data were collected and echocardiography was performed on admission. One hundred subjects matched for age, sex and frequency of concomitant diseases, who were randomly chosen from patients admitted to our hospital for elective ophthalmological surgery or advanced diagnostics of primary hypertension, served as controls.

The end points of the study were defined as all-cause death and APE-related mortality, which comprised deaths caused by clinically diagnosed irreversible RV insufficiency or recurrent PE. All events were recorded up to 30 days after the diagnosis of APE. High-, medium- and low-risk APE groups were defined according to European Society of Cardiology Guidelines,[11] based on systemic systolic blood pressure on admission and the presence of right ventricular dysfunction at echocardiography and elevated plasma troponin levels.

All patients received standard anticoagulant therapy with intravenous unfractionated heparin or subcutaneous body weight-adjusted dose of low molecular weight heparin. High-risk APE was an indication for thrombolysis (infusion of recombinant tissue plasminogen activator—either 100 mg over 1 h, or 0.6 mg/kg body mass max; 50 mg over