

Research Article

Assessment of Right ventricular by Pulsed Doppler peak velocity at the Tricuspid annulus in acute pulmonary embolism

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Abstract

Background: Assessment of the right ventricular (RV) function by echocardiography in patients with pulmonary thromboembolism (PTE) is complex and problematic. Tricuspid annular systolic plane excursion has been used for the assessment of this chamber, although with some limitations. We evaluate the Tissue Doppler S velocity (Pulsed Doppler peak velocity at the annulus) of right Ventricle for this reason.

Objective: To evaluate RV function in PTE using tissue-Doppler echocardiography, in addition to brain natriuretic peptide (NT PRO BNP).

Methods: Patients with PTE were studied using tissue-Doppler myocardial velocities (S) and NT PRO BNP up to 12 hours after diagnosis. RV dysfunction was diagnosed by S velocity less than 10 cm/s. According to their pro NT PRO BNP levels, the patients were divided into Group I, NT PRO BNP < 1000 pg/mL and Group II, NT PRO BNP > 1000 pg/mL.

Results: Of 68 patients (40 men, age = 45 ± 12 years); RV dysfunction was observed in 43%, more frequently in group II (22 vs. 7 patients, $p < 0.001$). Patients in group II were older (45 ± 11 vs. 38 ± 15 years), and had lower S velocity (8.5 ± 2.4 vs. 11.4 ± 2.6 cm/s), and higher pulmonary pressure (44 ± 5 vs. 36 ± 6 mmHg), $p < 0.001$. S velocity has moderate correlation with NT PRO BNP ($r = -0.39$).

Conclusion: RV dysfunction by S velocity is twin with NT PRO BNP elevation. Tissue-Doppler imaging confirms the presence of RV dysfunction.

Keywords: RV, Echocardiography etc.

Introduction

Pulmonary thromboembolism (PTE) is an important cause of morbidity and mortality, with poor prognosis if associated with hemodynamic instability, a situation in which mortality can reach approximately 20 % (1). The adequate assessment of the right ventricular (RV) function is crucial in PTE. However, its analysis using noninvasive methods such as two-dimensional echocardiography is limited because of the anatomical complexity of this chamber. In this sense, other echocardiographic techniques such as tissue Doppler imaging have been added to complement the diagnosis (2) as well as the identification of RV impairment in patients with PTE (4). The assessment of the RV function can also be complemented by the determination of Brain natriuretic peptide (NT PRO BNP) level, which are elevated in the presence of elevated intracardiac pressures (5). Comparing the relationship between RV systolic dysfunction on tissue Doppler echocardiography and the functional reflex of

RV overload, as estimated from NT PRO BNP, would be of potential clinical interest.

The objective of this study was to evaluate the RV systolic function using two-dimensional tissue Doppler echocardiography in patients with acute PTE and to analyze its correlation with NT PRO BNP.

Methods

From March 2014 to August 2016, all patients admitted to the Emergency Unit with clinically suspected PTE were analyzed in this study. PTE was confirmed by thoracic computed tomography showing complete or partial filling defect in pulmonary branches or by ventilation/perfusion pulmonary scintigraphy showing a high probability of PTE. Patients with left ventricular (LV) dysfunction, as characterized by an ejection fraction < 55% on echocardiography, were excluded from the study to minimize the influence of this condition on NT PRO BNP levels. Other exclusion criteria were the presence of chronic obstructive pulmonary disease, arrhythmias (atrial fibrillation or frequent extrasystoles) and an

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inadequate echocardiographic window. All patients received anticoagulation therapy (unfractionated or low-molecular-weight heparin). Patients with hemodynamic instability (systemic blood pressure < 90 mmHg, signs of poor peripheral perfusion) were given thromboembolytic treatment (streptokinase, according to guideline protocol).

The patients underwent echocardiography with measurements of the LV and ejection fraction according to the recommendations of the American Society of Echocardiography 10 up to 24 hours after diagnosis of acute PTE. The LV and RV diameters were measured in the 4-chamber apical view to obtain the RV/LV ratio. The parasternal, apical and subcostal views were used for the subjective assessment of the RV systolic function. Abnormal septal motion (straightening) and/or RV dilatation ($RV/LV > 1$) were added to the subjective analysis of function analysis. The pulmonary artery systolic pressure was derived from the tricuspid regurgitation added to right atrial pressure as estimated from the inferior vena cava diameter and collapsibility. E and A wave velocities were measured from the transmitral flow for the analysis of the LV diastolic function.

Tissue Doppler tracings were obtained from the lateral and septal annulus and from the lateral tricuspid annulus using the 4-chamber apical view to obtain measurements of the myocardial velocities (S and e' waves), with the sample volume as parallel to the wall as possible, so as to minimize the influence of the angle on Doppler velocities. LV filling pressure was estimated from the ratio between the early transmitral filling wave (E) and the mean of septal and lateral e' waves. Diastolic dysfunction was considered in the presence of increased LV filling pressures ($E/e' > 13$).

Plasma NT PRO BNP levels were determined up to 24 hours after PTE had been confirmed. The patients were divided into two groups according to their NT PRO BNP levels: group I, with NT PRO BNP < 1000 pg/mL, and group II with NT PRO BNP > 1000 pg/mL.

Continuous variables are expressed as mean \pm SD, and categorical variables, as percentages or frequencies observed. The groups were tested using the Student t test for continuous variables and the chi square test or Fischer test for categorical variables. Correlation between NT PRO BNP and the variables related to RV were tested using the Pearson's correlation. The analysis was carried out using the SPSS statistical software program (version 17.0; SPSS), and the level of significance was set at 0.05.

Results

A total of 68 patients had the diagnosis of PTE confirmed by tomography (n = 60) or pulmonary scintigraphy (n = 8). Predisposing factors for PTE included neoplasia (10%), surgery (45%) and long-distance travel (15%). Predisposing factors for PTE were not identified in 30% of patients. Only 4 patients

were hemodynamically unstable and, therefore, underwent thrombolysis; the other patients were treated medically. Most of the patients were males (59%), with a mean age of 45 ± 12 years. Tricuspid regurgitation was observed in 60 patients, and the pulmonary artery systolic pressure from the tricuspid regurgitation was 38 ± 15 mmHg.

Of 68 patients RV dysfunction was observed in 43%, more frequently in group II (22 vs. 7 patients, $p < 0.001$). Patients in group II were older (45 ± 11 vs. 38 ± 15 years), and had lower S velocity (8.5 ± 2.4 vs. 11.4 ± 2.6 cm/s), and higher pulmonary pressure (44 ± 5 vs. 36 ± 6 mmHg), $p < 0.001$. S velocity has moderate correlation with NT PRO BNP ($r = -0.39$). Proper pulsed tissue Doppler tracings could be obtained for S wave measurement in all patients. The tissue Doppler analysis showed a mean S wave velocity of 11.6 ± 4.5 cm/s. Of the 68 patients, 29 had RV systolic dysfunction on two-dimensional echocardiography. The ability of tissue Doppler S wave to predict RV systolic dysfunction was analyzed by the ROC curve, which showed a cut-off value of 10.3 cm/s, with 87% specificity, 63% sensitivity, and area under the curve of 0.82. The correlation between NT PRO BNP levels and tissue Doppler myocardial velocities was tested, showing a significant, but modest, inverse correlation ($p = 0.01$) ($r = -0.39$).

Twenty six patients showed NT PRO BNP < 1000 pg/mL, and were included in group I. Group II (n = 42) consisted of older patients, with a higher prevalence of tricuspid regurgitation (92% vs. 64%, $p < 0.01$) and higher pulmonary artery pressures (44 ± 5 vs. 36 ± 6 mmHg, $p < 0.001$) when compared to group I patients. Group II also showed a higher prevalence of RV systolic dysfunction (58% vs. 12%, $p < 0.001$). The estimated LV filling pressures (E/e' ratio) was similar in both groups.

Discussion

Pulmonary thromboembolism is an important cause of morbidity and mortality, and its prognosis is associated with the hemodynamic instability resulting from RV systolic dysfunction (1). In the literature, the prevalence of RV dysfunction on echocardiography varies from 30% to 50% (1, 2). Increased NT PRO BNP levels (> 1000 pg/mL) were associated with a higher prevalence of RV systolic dysfunction. Tissue Doppler imaging is a simple, fast and noninvasive method for the assessment of ventricular performance. However, cut-off values for the identification of RV systolic dysfunction are not fully consistent (3-6). Hsiao *et al* (2) studied patients with PTE using tissue Doppler imaging and showed that these patients had lower S wave values (10.6 cm/s) when compared to a group of normal individuals (13.1 cm/s). Meluzin *et al*, in turn, studied patients with heart failure and right ventricular dysfunction, and obtained a slightly lower cut-off point (10.5 cm/s) for concomitant RV systolic dysfunction, also in comparison to normal individuals.

Since our population consisted exclusively of patients with the disease (PTE), we observed different RV performances, ranging from completely normal ventricles to global chamber failure; in this situation, there is a higher probability of estimate overlapping, and consequently a lower sensitivity of the method. In some cases, we also observed that the assessment of the basal RV region by tissue Doppler imaging did not reflect the change seen in the other walls, which occasionally could be hypokinetic and/or with local dilatation.

In relation to NT PRO BNP, increased levels of this biomarker have been found primarily in the presence of RV systolic dysfunction (predominantly marked RV dysfunction), thus suggesting that a significant impairment of this chamber is crucial for the elevation of the levels of this peptide. Because of the pulmonary vasoconstriction resulting from PTE, there is an increase in the RV workload in an attempt to keep the pulmonary pressure high; depending on the degree of pressure elevation, RV dilatation and failure may occur. Since NT PRO BNP is secreted in response to increased ventricular or atrial stress, it reflects volume or pressure overload, which results in its elevation in patients with RV systolic dysfunction. However, the correlation between RV dysfunction and NT PRO BNP is not linear, with countless factors influencing this relationship, including the biomarker secretion and clearance. Left ventricular systolic dysfunction, for instance, is strongly associated with increased intracardiac pressures (and consequently increased NT PRO BNP levels); because of this association, patients with decreased LV ejection fraction were excluded. Since the diastolic dysfunction is also correlated with increased NT PRO BNP levels (7-10), its influence was analyzed from the estimated intraventricular pressure by tissue and conventional Doppler imaging (obtained from the E/e' ratio); in our case series, despite patients with higher NT PRO BNP levels were older, and presumably with a higher prevalence of diastolic dysfunction, both groups had similar LV filling pressure measurements. Diastolic dysfunction shows only a modest correlation with NT PRO BNP levels (11, 12), particularly in patients without associated systolic dysfunction. Thus, we believe that the influence of diastolic dysfunction on NT PRO BNP levels may be minimized in this group.

Limitations

Other techniques, such as the measurement of the RV fractional area change (FAC) and ejection fraction by Simpson's method could add more consistency to the results

Conclusion

Increased NT PRO BNP levels are a marker of RV dysfunction on echocardiography in patients with acute PTE.

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