

Case Report

Pulmonary Embolus in Transit treated with Thrombolysis: Risk Stratification with Transthoracic Echocardiography

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ABSTRACT

Right heart thromboemboli and submassive pulmonary emboli are medical emergencies that can carry high mortality, if not treated promptly and aggressively. Thrombolysis is a preferred treatment option in acute submassive pulmonary emboli, whereas, optimal therapy for right heart thromboemboli is yet unclear. We report a case of echocardiographically documented pulmonary embolus in transit. This echocardiographic finding led to the treatment with recombinant tissue

plasminogen activator (rt-PA). There was clinical improvement in symptoms and hemodynamics immediately after thrombolysis and there were no complications. This case demonstrates the utility of two-dimensional transthoracic echocardiography for risk stratification in patients with documented pulmonary emboli, which may affect the subsequent management. The pertinent literature is reviewed and relevant issues in decision-making are discussed.

KEY WORDS: echocardiography, embolism, pulmonary heart disease and prognosis, thrombolysis, thrombus

INTRODUCTION

Case reports in the literature of right heart thromboemboli or so-called pulmonary emboli in transit are more frequent with the increasing use of two-dimensional transthoracic echocardiography in suspected or confirmed cases. We report a case of acute submassive pulmonary embolus with a large mobile right heart thrombus extending from the inferior vena cava to the right ventricle and discuss the appropriate management.

Case presentation

A 52-year-old male presented with acute progressive shortness of breath. He was an obese, hypertensive male on long acting nifedipine and hydrochlorothiazide. He complained of progressive bilateral leg swelling and shortness of breath over a two-month period, which suddenly became worse on the day of admission. He denied any chest discomfort, cough or hemoptysis. He had marked respiratory difficulty while taking a shower, nearly collapsed and then presented to the emergency room.

On examination, he was in moderate respiratory distress with a respiratory rate of 26 breaths per minute. The arterial oxygen saturation was 84% on room air and 96% on 50% oxygen, the pulse was 110 beats per minute and the blood pressure was 115 / 55 mmHg (his usual blood pressure was 140-150 /

80 mmHg). Chest examination showed minimal wheezing and was otherwise unremarkable. Heart examination revealed an audible S4. There was bilateral lower limb swelling consistent with edema, more evident in the right leg.

The initial laboratory data included serum creatinine of 380 $\mu\text{mol/L}$ (normal range < 130 $\mu\text{mol/L}$). Troponin (I) was elevated at 3.72 ng/mL (normal range is 0.0 - 0.15 ng/mL). Arterial blood gas on 50% oxygen showed significant hypoxemia ($\text{PaO}_2 = 69.1$ mmHg) without acidemia ($\text{PH} = 7.48$). The electrocardiogram showed sinus tachycardia, and right ventricular hypertrophy with strain pattern. The chest X-ray showed bibasilar atelectatic changes.

A transthoracic echocardiogram (TTE) was performed (Fig. 1 and 2) and it showed a long serpiginous highly mobile clot (type-A) measuring 1.1 x 9 cm in the right atrium prolapsing into the right ventricle, and originating from the inferior vena cava. Right ventricular dysfunction and dilatation were present, with estimated systolic pulmonary artery pressure of 51 mmHg. Left ventricular ejection fraction was estimated at 40-45% with mild diffuse hypokinesis.

Ventilation perfusion scan, performed after the first TTE, showed extensive bilateral pulmonary emboli with obliteration of more than 50% of the vascular bed. Doppler scan of the lower limbs

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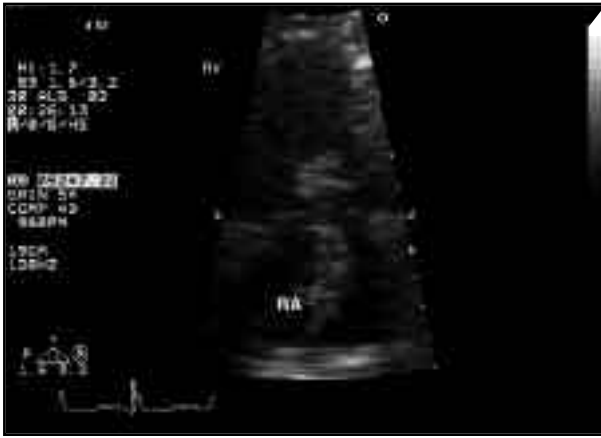


Fig. 1: Transthoracic echocardiogram showing the clot in the right atrium (RA) extending through the tricuspid valve into the right ventricle (RV)

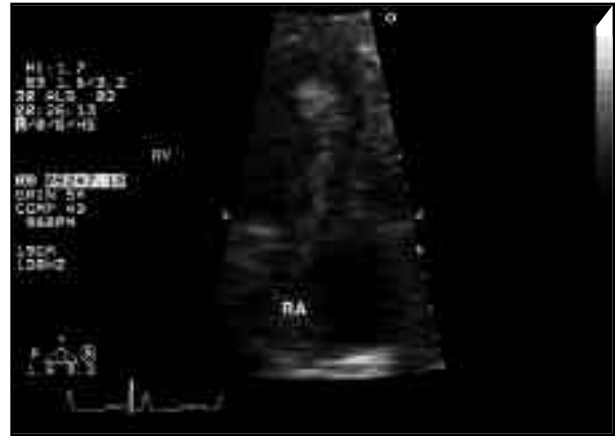


Fig. 2: Transthoracic echocardiogram showing the clot extending into the right ventricle

documented the presence of bilateral above knee-deep venous thromboses without extension into the iliac veins or the inferior vena cava.

Initially the patient was treated with intravenous unfractionated heparin. A repeat transthoracic echocardiogram showed disappearance of the clot but persistence of right ventricular dysfunction. The patient became more dyspneic and hypoxemic. Subsequently, thrombolytic therapy with rt-PA (100 mg intravenous 2-hr infusion) was administered. Immediately after infusion, the blood pressure increased from 110 / 50 to 150 / 70 mmHg, the heart rate decreased from 110 to 80 bpm, and the patient became less tachypneic with improved oxygen saturation. There was also significant improvement in the urine output and the renal function (down to his baseline) over the next 24 hours. Four days later, repeat transthoracic echocardiogram showed significant improvement in the right ventricular function and normalisation of the pulmonary artery pressures.

DISCUSSION

Pulmonary embolism is a life-threatening but treatable condition. This case illustrates two aspects of the problem: right heart thrombo-embolism or so-called pulmonary embolism in transit, and documented pulmonary embolism with acute right ventricular dysfunction or so-called submassive pulmonary embolism.

Right heart thrombo-embolism is a medical emergency and a potentially fatal condition, if left untreated. In a study by Chartier *et al*^[1], the mortality rate for patients with right heart thrombi and pulmonary embolism was 44.7%. In another recent study by Rose *et al*^[2], the mortality rates associated with no therapy, anticoagulation therapy, surgical embolectomy, and thrombolysis were 100.0%, 28.6%, 23.8%, and 11.3%, respectively.

Thrombolytic therapy (rtPA) was shown to be safe and rapidly effective in right heart thrombo-emboli resolution and pulmonary perfusion. It also resulted in an improvement in the hemodynamics and clinical status^[3,4]. The current optimal therapy is not well defined as different therapies may produce a variable degree of clot fragmentation and embolisation with unpredictable results. While we await a well-designed prospective randomised trial, it is reasonable to use systemic thrombolysis in the presence of a mobile (type-A) right heart thrombus as the first line therapy, and to reserve surgical embolectomy for patients with formal contraindications to thrombolysis^[5].

Submassive pulmonary emboli, or pulmonary emboli with right ventricular dysfunction and stable hemodynamics, have higher mortality rates (than those without dysfunction), if not treated appropriately^[6,7]. In acute massive pulmonary embolism and cardiogenic shock, thrombolysis is the standard treatment^[8,9]. On the other hand, in submassive pulmonary embolism the results of thrombolysis were variable in previous studies^[10-12]. Recently, the largest prospective randomised double-blind study by Konstantinides *et al*^[13] showed that alteplase (rtPA) in conjunction with heparin can improve the clinical course of stable patients who have acute submassive pulmonary embolism (manifested as right ventricular pressure overload and dysfunction) and can prevent clinical deterioration requiring the escalation of treatment during their hospital stay.

Acute right ventricular dysfunction can be assessed by clinical examination and electrocardiogram but the echocardiogram is more sensitive. Myocardial markers have been studied in acute pulmonary embolism. Cardiac troponins (I and T) were found to be significant predictors of mortality, morbidity, and recurrence of pulmonary embolism. Elevated troponins correlated with the presence of right ventricular dysfunction on transthoracic echocardiography^[14].

CONCLUSION

Transthoracic echocardiogram has a significant role in the diagnosis of pulmonary embolism in transit and in risk stratification, which may alter the management approach. As illustrated in this case, thrombolysis is a valid treatment for mobile right heart thromboemboli and/or submassive pulmonary emboli and may provide hemodynamic and clinical benefits.

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