Pulmonary Thromboembolism and Right Heart Thromboemboli- An Experience with Tenecteplase

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Abstract

The presence of right heart thromboemboli complicating pulmonary thromboemboli carries with it an increased mortality rate compared to pulmonary thromboemboli alone, but little is known about the optimal management of this difficult clinical situation. We report a case of bilateral pulmonary thromboembolism with right heart thrombi treated successfully with thrombolysis with tenecteplase.

Introduction

Deep venous thrombosis, right heart thrombi and pulmonary embolism represent the spectrum of one disease. Thrombi commonly form in deep veins in the calf and then propagate into the proximal veins, including and above the popliteal veins, from which they are more likely to embolise. About 79% of patients who present with pulmonary embolism have evidence of deep venous thrombosis in their legs. Conversely, pulmonary embolism occurs in up to 50% of patients with proximal deep venous thrombosis. Right heart thromboemboli represent mobilized deep venous thromboses that are lodged temporarily in the right atrium or ventricle, and are often referred to as “emboli in transit”.1

In patients with a mobile right heart thrombus, the incidence of pulmonary embolism is 97% and reported mortality is over 44%.1 At the present time, there is no consensus on how to treat patients with pulmonary embolism associated with right heart clots. The metaanalysis published in 1989 by Kinney and Wright showed no advantage for any treatment option used, namely, Heparin, Thrombolysis or surgery.2 However, the more recent metaanalysis published by Rose et al points to a better outcome with Thrombolysis.3 Here, we describe a case of deep venous thrombosis with pulmonary embolism with right heart thrombi treated successfully with thrombolysis with Tenecteplase.

Case Report

A 47 years postmenopausal, non-smoker, female was admitted to the hospital with the complaints of breathlessness at rest since 12 days. She had road traffic accident 14 days back and was diagnosed to have wedge compression fracture of L2 vertebra which was treated conservatively along with bed rest. After 2 days, she developed sudden onset of breathlessness. Arterial blood gas analysis revealed partial pressure of oxygen 57.2, partial pressure of carbon dioxide was 27.4, and oxygen saturation of 91.8% while breathing 100% oxygen. ECG showed sinus rhythm, T wave inversions in lead V1 to V3. Chest X-ray revealed no abnormality. Her D-dimer levels were 9397.94ng/ml (N-upto 500ng/ml). Baseline blood investigations revealed Haemoglobin- 11.7 gm%, Hematocrit- 34.5%, TLC-9,200 cells/

Discussion

Our patient presented with bilateral pulmonary embolism with right heart thrombi along with right lower limb deep venous thrombosis. In our patient, underlying hypercoagulability with
protein S and protein C deficiency precipitated the development of deep vein thrombosis and pulmonary embolism. Right heart thrombi in our patient represent the migration of thrombus from right lower limb deep veins. The consensus made at the European co-operative study on the clinical significance of right heart thrombi was that two major types of right heart thrombi exist according to morphology, etiology, pathophysiology and prognosis. Type A thromboemboli appear as long, thin and mobile (described as worm like or snake-like in appearance) by trans thoracic echocardiography. This group had a high incidence of deep venous thrombosis and lower incidence of intracardiac abnormalities. The mortality rate in this group was 42%. Type B thrombi are less mobile and associated with intracardiac abnormalities. Our patient had a long linear thrombosis in right atrium, tricuspid annulus, right ventricle and RVOT favouring Type A thromboemboli. Emergent management was required considering mobile thrombus in the right heart and high mortality associated with this condition. In a meta-analysis including all major reported cases in the literature up to 2002, Rose et al reported a 27% mortality rate. However, there is no consensus on how to treat patients with PE and associated RH clots. The metaanalysis published in 1989 by Kinney and Wright showed no advantage for any treatment option used, namely, heparin, thrombolysis or surgery.

However, Thrombolysis may be advocated first. It accelerates thrombus lysis and pulmonary reperfusion, reduces pulmonary hypertension and because of right ventricle–left ventricle interdependence, improved right ventricular function helps to increase both right and left ventricle output and to reverse cardiogenic shock. Moreover, thrombolysis may dissolve the clot in 3 locations at the same time: the intracardiac thrombus, the pulmonary embolus, and the venous thrombosis.

Three lytic drugs have been approved by the Food and Drug Administration in cases of severe pulmonary embolism: urokinase, streptokinase, and rtPA. Our patient received tenecteplase 40 mg IV bolus as a thrombolytic agent followed by enoxaparin (1mg/kg sc bd). Within hours of thrombolysis, patient had dramatic symptomatic improvement and 24 hours after, 2D-echo and pulmonary angiography revealed complete resolution of thromboemboli in the right heart and pulmonary arteries.

There are various reports suggesting that tenecteplase is also useful in pulmonary embolism. A randomized, double blind placebo-controlled study done to assess the effect of tenecteplase on RV dysfunction showed that single bolus tenecteplase was associated with improved reduction of right to left ventricular end diastolic diameter ratio. However, controlled randomised trials showing the beneficial effects of tenecteplase in pulmonary embolism and particularly those with right heart thrombi are lacking. We report a rare case of complete resolution of bilateral pulmonary embolism with right heart thromboemboli after thrombolysis with Tenecteplase.

References