Successful Thrombolysis of a Large Pulmonary Artery Thrombosis

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Abstract
A 32 yrs old man presented with shortness of breath and syncopal episode with preceding history of DVT 15days above. Patient has tachycardia hypoxia and hypotension, on evaluation ECG showed S1Q3T3 Pattern, bedside Echo showed visible thrombus of 3cm in pulmonary artery, successfully thrombolysed with tenecteplase and streptokinase. This case study is presented to stress importance of urgent bedside echo in all sudden onset dyspnoea and hypoxia to rule out pulmonary Embolism which can be successfully thrombolysed without delay.

Introduction
Pulmonary thromboembolism (PTE) is a major health problem with significant mortality and morbidity. It is a common but life-threatening condition. PTE and deep vein thrombosis (DVT) known collectively as venous thromboembolism (VTE) encompass a single disease entity. PTE implies occlusion of pulmonary arterial circulation by the clot formed elsewhere in deep veins of the leg. Less than 5% of venous thrombosis occurs at other sites. VTE occurs worldwide and is usually but not always associated with specific risk factors.1,2 A crucial point is that DVT and, therefore, PE are often preventable.3,4 Because of the lack of specific symptoms and signs, DVT and PE are frequently clinically unsuspected, leading to substantial diagnostic and therapeutic delays and resulting in considerable morbidity and mortality.1,5

Case Report
A 32-year-old man presented to the emergency department complaining of severe shortness of breath that began abruptly with a bout of cough. He reported that suddenly he was not able to catch his breath, felt light-headed and collapsed on the floor without any loss of consciousness. Associated symptoms included right sided chest pain and diaphoresis. Three weeks prior to this event, the patient reported that he began to notice pain and swelling in his left calf after having a blunt vehicular trauma. He had no significant past medical family, or drug addiction history. His vital signs upon arrival to the emergency room were a heart rate of 112 beats per minute, respiratory rate of 40 per minute with oxygen saturation of 80% and blood pressure of 100/60 mm of Hg. He was pale, diaphoretic, and unable to speak full sentences. His jugular veins were distended up to the angle of the jaw while he was sitting 90° upright. Cardiac examination demonstrated tachycardia, a wide split second heart sound, the presence of a third heart sound at the left lower sternal border and a right ventricular heave. Pulmonary findings consisted of bilateral basal crackles. His extremities were cold and cyanotic with weak peripheral pulses.

The initial electrocardiogram (ECG) showed sinus tachycardia (112 beats per minute), right axis deviation and diffuse ST-segment depression and T-wave inversion with S1Q3T3 pattern. Bedside echocardiogram demonstrated severe right atrial and right ventricular (RV) dilation with signs of RV pressure overload, hypokinesis of the RV free wall and the ventricular septum (Figure 1). There was a pedunculated 3 cm clot attached to main pulmonary artery. Pulmonary artery pressure was estimated to be 38 mmHg by TR jet. Complete blood count, coagulation profile was normal with peripheral blood smear showing macrocytosis. D-dimer test was positive. Lower extremity venous Doppler studies revealed dilatation with an extensive thrombus all along its length upto popliteal vein. Chest X-ray (CXR) revealed oligemic right lung field with enlarged right descending pulmonary artery.

With a diagnosis of pulmonary embolism, the patient underwent thrombolytic therapy with IV tenecteplase 30 mg bolus dose. Despite having thrombolysed with tenecteplase patient had severe distress, tachypnea, and persistence of clot after one hour of thrombolysis on echocardiography, so we decided to start continuous infusion of streptokinase 100,000 U/hr over 24 hours followed by unfractionated heparin 1000 IU/Hr with oral warfarin 5 mg OD. Patient was still tachypneic and was in severe distress with oxygen saturation decreasing rapidly and required endotracheal intubation with mechanical ventilatory support for 2 days. During this time there was progressive improvement in the patient’s condition. The following day, his symptoms improved dramatically, his respiratory rate decreased, his symptoms improved dramatically, his heart rate decreased to 90 beats per minute, his saturation increasing rapidly and he was in severe distress with oxygen saturation decreasing rapidly and required endotracheal intubation with mechanical ventilatory support for 2 days. During this time there was progressive improvement in the patient’s condition. The following day, his symptoms improved dramatically, his respiratory rate decreased, his saturation increased to 98% and his heart rate decreased to 72 beats per minute

Fig. 1: Thrombus in MPA (arrow)
Fig. 2: 2D echo at this 5 days showing complete dissolution of thrombus after thrombolysis

oxygen saturation rose from 85% to 98%, and his blood pressure normalised. Follow up echocardiography after 5 days (Figure 2) showed complete dissolution of clot, improved right ventricular function and decreased right ventricular size and pulmonary arteries. Patient was discharged with warfarin anticoagulation and advised regarding IVC filter placement.

Discussion

Pulmonary emboli are potentially life threatening occurrences associated with significant morbidity and mortality both in the early and late stages. The first step in making a diagnosis is a clinical evaluation that takes into accounts the risks, symptoms, and signs. Identification of breakdown products of clots in the blood (D-dimer) is a useful biomarker that can further assess the likelihood of a thrombus.

Echocardiography is a rapid bedside diagnostic tool which may be useful if the use of thrombolytic therapy or embolectomy is being urgently considered. Bed side echocardiography outlines the right ventricular size and function, size of pulmonary arteries and shows a clot if present.

Patients treated with thrombolytic therapy show rapid improvement of right ventricular function and pulmonary perfusion which may lead to a lower rate of early recurrent PE and a decrease the late sequelae of chronic pulmonary hypertension. Right heart thrombi, particularly when mobile, i.e. in transit from the systemic veins, are associated with a significantly increased risk of early mortality in patients with acute PE. Thrombolysis and embolectomy are probably both effective whereas anticoagulation alone appears less effective.

The main indications for thrombolytic therapy include ongoing hypoxia, respiratory distress, pulmonary hypertension, and right heart failure because thrombolytic therapy often achieves an impressive and almost an immediate clinical benefit in these clinical settings. Tenecteplase has a number of advantages compared with streptokinase, urokinase and tissues plasminogen activator, which are approved treatments for pulmonary embolism. These advantages include a simpler mode of administration via bolus injection, faster onset of action, longer half-life, increased fibrin specificity and higher resistance to inhibition by plasminogen activator inhibitor 1 (PAI-1). Thrombolytic therapy should be considered in all patients with massive pulmonary embolism and hypotension associated with deep vein thrombosis in the popliteal area or higher upto 14 days of pulmonary embolism.

References