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Case Report

The Benefit of Early Thrombolytic Therapy in a Normotensive Patient with Submassive Pulmonary Embolism

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Abstract

A 65-year old man, presented with syncope and dyspnea. On examination he was tachypneic, hypoxemic, normotensive, with elevated D-dimer and cardiac troponin. ECG showed sinus tachycardia S1Q3T3 syndrome and echocardiography revealed right ventricular dysfunction. Urgent computed tomograph angiograms showed bilateral pulmonary embolism. After treatment with intravenous recombinant tissue plasminogen activator (rtPA) the patient's status improved and echocardiogram showed decreasing of the right ventricular systolic pressure.

The most widely accepted indication for thrombolytic therapy is proven pulmonary embolism with cardiogenic shock; therapy is also frequently considered when a patient presents with systemic hypotension without shock. The use of thrombolysis in submassive embolism-that is pulmonary embolism causing Right Ventricular (RV) dilatation and hypokinesis with normal blood pressure-is debated [1].

The purpose of the study was to demonstrate a case of submassive pulmonary embolism in a normotensive patient who had an excellent clinical electrocardiographic and echocardiographic response to fibrinolysis.

Keywords: Submassive Pulmonary Embolism, Thrombolysis

Case report

A 55-year-old man, with a history of recent hospitalization for orthopedic surgery, presented with exertional syncope and progressively worsening dyspnea. On examination he was tachypneic (33 breaths per minute), hypoxemic (oxygen saturation 84 on room air), with heart rate of 120 beats per minute and blood pressure of 125/70 mmHg. The neck vein was distended, there was no heart murmur and the lungs were clear on auscultation. Laboratory evaluation was remarkable for D-dimer 3025 ng/ml (normal < 234 ng/ml) and a cardiac troponin level of 1,2 ng/ml (normal < 0,06 ng/ml). The Electrocardiogram (ECG) revealed sinus tachycardia with a rate of 120 per minute, a deep S wave in lead I, a Q wave and inverted T wave in lead III (S1Q3T3 syndrome) and a subtle ST elevation in leads aVR and V1 [Figure 1]. Transthoracic echocardiography revealed right ventricular dilatation and hypokinesis with moderate tricuspid regurgitation and an estimated right ventricular systolic pressure of 62 mmHg. Doppler studies of the legs showed bilateral proximal deep venous thrombosis, making the diagnosis of pulmonary embolism likely. Urgent contrast-enhanced computed tomograph angiograms [Figure 2] showed bilateral pulmonary embolism. After screening for contraindication to fibrinolysis was performed, the decision was made to proceed with thrombolysis into low risk of bleeding and increased risk of death. 100 mg of tissue Plasminogen Activator (rtPA) was administered over a 2-hour period, and after completion of the fibrinolysis, unfractioned heparin was started without a bolus as a "bridge" to anticoagulation with warfarin. After treatment with intravenous rtPA, the patient's respiratory status dramatically improved over a period of several hours, the S wave in lead I and the ST elevation in leads aVR and V1 on the ECG disappeared and the heart rate slowed down to 61 beats per minute [Figure 3]. Repeated echocardiography showed that the right ventricular systolic pressure decreased to 31 mmHg. On follow-up, 5 weeks later, the patient's condition was good and the echocardiogram documented normal right ventricular size and function.

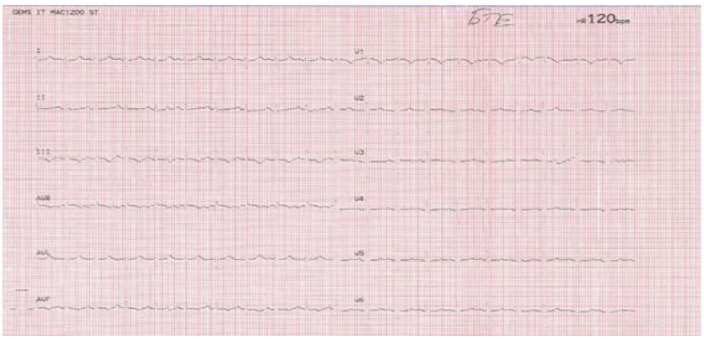

Figure 1: The ECG on admission shows sinus tachycardia with a rate of 120, a deep S wave in lead I, a Q wave and inverted T wave in lead III (S1Q3T3 syndrome) and a subtle ST elevation in leads aVR and V1.

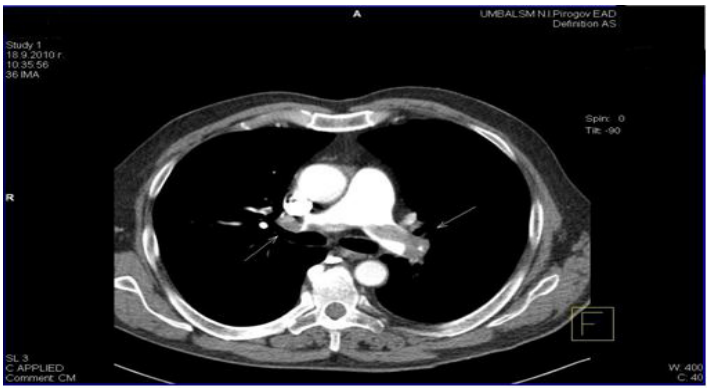

Figure 2: Multidetector spiral computer tomogram showing bilateral defects (arrows) in the pulmonary artery.

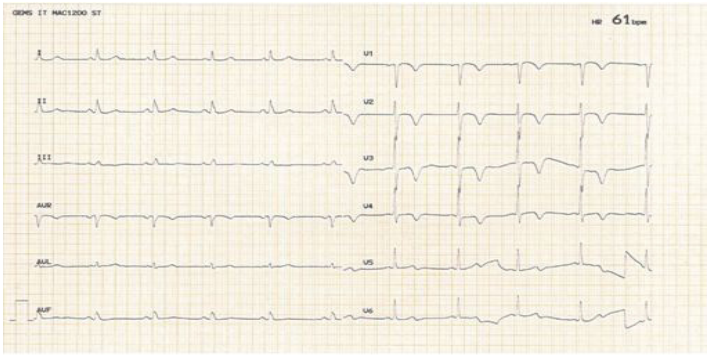

Figure 3: The ECG performed after completion of fibrinolysis revealed disappearance of the S wave in lead I and the ST elevation in leads aVR, V1 and slowing down of the heart rate to 61.

Discussion

Puulmonary embolism is a common disease associated with high mortality rate. Death due to pulmonary embolism occurs more commonly in undiagnosed patients before hospital admission or during the initial in-hospital stay. Thus, mortality could be reduced by prompt diagnosis early risk stratification and more intensive treatment. Mortality is particulary high in patients with pulmonary embolism presenting with arterial hypotension or cardiogenic shok. In patients with pulmonary embolism and normal blood pressure, a number of clinical features and objective findings have been associated with a high risk of adverse in-hospital outcome. Submassive Pulmonary Embolism (PE) defines patients who appear hemodynamically stable on admission, but have evidence of Right Ventricular (RV) dysfunction. This group of patients can be identified by the presence of RV dysfunction detected on physical examination, cardiac biomarkers, ECG, echocardiography and chest CT.

The decision to administer a fibrinolytic agent in addition to heparin anticoagulation requires individualized assessment of the balance of benefits versus risk. Potential benefits include more rapid resolution of symptoms, stabilization of respiratory and cardiovascular function without need for mechanical ventilation or vasopressor support, reduction of RV damage, improved exercise tolerance, prevention of PE recurrence and increased probability of survival. Potential harm includes disabling of fatal haemorrhage, including intracerebral haemorrhage and increased risk of minor haemorrhage, resulting in prolongation of hospitalisation and need for blood product replacement. Fibrinolysis is most successful when administered within several days of acute PE. Although the efficacy of fibrinolysis is inversely proportional to the duration of symptoms, effective thrombolysis can be observed up to two weeks after an acute event.

Thrombolysis is indicated in the case of patients with PE who have arterial hypotension or are in shock. In contrast, the benefits of thrombolysis in a normotensive patient's with acute PE are less well established. Despite growing interest in thrombolytic agents to treat submassive pulmonary embolism, their role in this scenario remains controversial. Needed is a way to identify patients with this condition who are at risk of clinical deterioration and who would benefit from thrombolytic therapy [2]. Results from randomised trials suggested that selected patients with evidence of RV dysfunction and a low risk of bleeding may benefit from early fibrinolysis [3,4]. The decision to select thrombolysis for submassive PE or to maintain anticoagulation alone must be individualized because of paucity of trials to help guide management. On the other hand, some authors concluded, that there is no scientific support for thrombolytic therapy in this case [5] In our patient the dilemma was whether the normotensive patient with RV dysfunction, as detected on echocardiogram and CT scan, and with evidence of myocardial injury, as indicated by a positive troponin test, may benefit from early thrombolytic treatment.

After completion of fibrinolysis, the patient's respiratory status and gas-exchange derangements improved over a period of

several hours. The ECG returned to normal and the echocardiogram revealed decreasing of the RV systolic pressure.

Conclusion

The best that can be said for the idea of treating submassive pulmonary embolism with thrombolytic therapy, is that it is unsupported by the available literature, which is very limited. Nevertheless, a selected group of normotensive patients with submassive pulmonary embolism and evidence of right ventricular dysfunction may benefit from early thrombolysis. We believe that this paper illustrates a case that clinicians may encounter in their practice and it can help them to make better decision.

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