

Syncope as a Rare Clinical Presentation of Pulmonary Thromboembolism: A Case Report

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Abstract: A 50 - year - old male adult with no co - morbidities experienced sudden onset loss of consciousness for approximately 2 - 3 minutes at home. The patient had no history of chest pain, diaphoresis, palpitations, abnormal body movements, or incontinence. On examination, he presented with hypotension, tachycardia, and normal saturation at room air. Diagnostic tests revealed T - wave inversions on ECG, dilated right pulmonary artery on chest X - ray, and thrombus presence in the pulmonary artery on CT - Pulmonary angiography. The patient was diagnosed with acute pulmonary thromboembolism and successfully treated with thrombolysis. Follow - up examinations showed no syncope episodes, stable vitals, and normal 2DEcho results.

Keywords: Syncope, Pulmonary Thromboembolism, Case Report, Thrombolysis, Hemodynamic Compromise

1. Case Presentation

50 year old male adult with no co - morbidities was in usual state of health, when he had sudden onset loss of consciousness for approximately 2 - 3 minutes while at home. He denied history of chest pain, diaphoresis, palpitations, any abnormal tonic clonic body movements or incontinence.

He was admitted from emergency department and on examination he had hypotension, BP - 86/50, tachycardia, PR 110/min, and normal saturation at room air, spo2 98%. Systemic examination was within normal limits.

Routine Investigations like complete blood count, renal function tests, liver function test, serum electrolytes were normal. His ECG showed T - Wave inversions in lead II, III, avf and sinus tachycardia. Chest X - Ray showed dilated right pulmonary artery (PALLA SIGN). Fig I



An electrocardiogram was done which showed RA/RV dilatation, severe PAH, moderate TR, Concentric LVH. Possibility of pulmonary thromboembolism was kept. As a part of work up, compression USG legs was done which showed filling defect in left popliteal vein – Left limb DVT. However patient was asymptomatic with no pain/swelling of leg. Further, D - Dimer was raised – 2.5 u/ml. CT - Pulmonary angiography was done which showed hypodense contents in both branches of pulmonary artery - s/o thrombus.

Diagnosis of acute pulmonary thromboembolism was made. In view of hypotension, he was given a bolus of 500 ml normal saline. BP not improving so thrombolysis was done with TENECTEPLASE 35gm bolus. After 1 hour his BP improved to 112/90 mmHg and he was started on LMWH. Meanwhile, his coagulation profile was sent which was normal. There was no evidence of Protein C, S antithrombin 3 deficiency. He was discharged in hemodynamically stable state on rivoroxaban. He was followed up after 1 month, 3 months and 6 months. There was no syncope episode, vitals are stable and repeat 2DEcho was normal.

2. Discussion

Acute PE is a general term for a group of diseases or clinical syndromes caused by various emboli obstructing the pulmonary artery system, including pulmonary thromboembolism (PTE), fat embolism syndrome, amniotic fluid embolism, and air embolism.

The classic triad of pleuritic chest pain, dyspnea, and hemoptysis, and clinically apparent DVT is present in only 11% of confirmed cases of pulmonary embolism in patients without underlying cardiopulmonary disease (2) However, the clinical picture of pulmonary embolism is variable and most patients suffering from acute pulmonary embolism present with one of three different clinical syndromes.

Syncope has been classified as cardiovascular (reflex and cardiac syncope), noncardiovascular and unexplained. It occurs in approximately 10% of patients with acute pulmonary embolism and is commonly ascribed to a massive, hemodynamically unstable acute pulmonary embolism. However, submassive emboli can also present as syncope and it may be the only symptomatic presentation of the disease as in our case (3)

The occurrence of syncope during PTE may result from different possibilities: the first is acute right ventricular failure, caused by massive embolism consequent to a reduction in the cross-sectional pulmonary vascular area and pulmonary arterial hypertension. This failure could trigger a significant decrease in left ventricular filling, with concomitant tachycardia, arterial hypotension, and low cerebral flow, which may be the most probable mechanisms of syncope in the presence of acute PTE. (4)

The second possibility is the reflex syncope due to a vasovagal mechanism triggered by pulmonary thromboembolism. The third possibility is complete atrioventricular block in the presence of preexisting complete left bundle - branch block. The development of

acute right bundle - branch block due to pulmonary embolism supposedly accounts for complete A. V. blockage and syncope in these individuals.

For most cases of acute PE without haemodynamic compromise, LMWH or fondaparinux, given subcutaneously at weight - adjusted doses is the treatment of choice. Patients not suffering from shock or hypotension require further risk stratification after the diagnosis of PE has been confirmed.

Patients with PE presenting with shock or hypotension (as in our case) are at high risk of in - hospital death, particularly during the first few hours after admission. Primary reperfusion treatment, particularly systemic thrombolysis, is the treatment of choice and in those in whom thrombolysis has failed or is contraindicated to improve the haemodynamic status, surgical embolectomy is recommended if surgical expertise and resources are available. (5)

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