CASE REPORT

Massive pulmonary embolism presenting as seizures

Shahrukh Hashmani,[1] Fateh Ali Tipoo Sultan,[2] Murtaza Kazmi,[3] Ammarah Yasmeen[4]

Abstract

Pulmonary embolism can present in various ways, though seizure activity has been rarely reported. We report a 38 years old male who had met a road traffic accident, underwent surgery, presented a month later with seizures, chest pain, shortness of breath and low blood pressure. Massive pulmonary embolism was diagnosed on CTPA, with classical features on ECG and echocardiogram and the patient was successfully managed with thrombolysis. Massive pulmonary embolism (MPE) should be kept in mind in patients who present with seizures, chest pain and haemodynamic instability.

Keywords: Case report, Massive pulmonary embolism, Seizure, Emergent thrombolysis, Chest pain.

Introduction

Massive pulmonary embolism (MPE) is a medical emergency and can lead to death, if not diagnosed and managed in time.[1] Patients with MPE usually present with dyspnoea, chest pain, syncope and hypotension. New-onset generalized seizures are rarely reported as a presentation of MPE and the diagnosis may be delayed in such cases. Emergency thrombolysis is the most effective therapy for treatment of MPE.

Pulmonary embolism in countries like Pakistan remains largely an unrecognized and under diagnosed clinical problem due to non-availability of objective tests and lack of awareness among physicians. There are very few reports available from Pakistan on pulmonary embolism.[2]

We report a 38 years age male, who presented with chest pain, worsening shortness of breath and new onset generalized seizures along with hypotension. The patient was successfully managed with thrombolysis, once CTPA showed massive pulmonary embolism.

Case Report

A thirty eight years old male was referred from orthopaedic clinic on 5th March 2016 with symptoms of chest pain, shortness of breath and seizure like activity. He had a history of road traffic accident about one and half months back and underwent surgery for left femur fracture. His activity was limited since the time of surgery and was not on any anticoagulation. He was visiting orthopaedic clinic where he became symptomatic after doing some leg exercises. He started feeling chest pain, which was sharp, severe and associated with shortness of breath, followed by an episode of seizure like activity. After initial resuscitation, he was referred to the emergency room of tertiary care hospital. In the ambulance, he again had an episode of witnessed seizure, which lasted for few seconds. On arrival to the emergency room, his chest pain had already settled, but he was short of breath. On physical examination, he was alert and oriented with temperature of 37°C; pulse rate of130 beats per minute, respiratory rate 24 breaths per minutes, and blood pressure 80/40 mmHg, maintaining oxygen saturation at 5 liters of oxygen through facial mask. He had jugular vein engorgement and mild oedema in both legs. His chest was clear with no added sounds on precordial examination. There was no sign of focal neurological deficit. He was resuscitated with intravenous fluids.

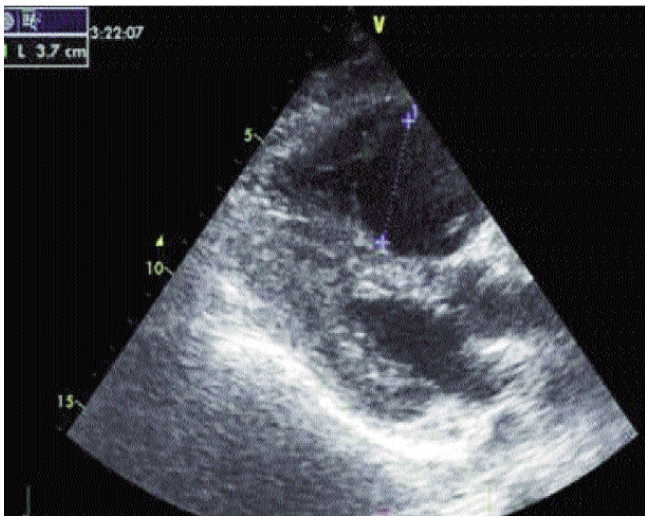

Figure-1: 2D transthroacic echocardiogram in long axis parasternal view, size of RV measured pre-thrombolysis is 37mm.

[1]Cardiology Resident Year II, Department of Medicine, AKUH, [2]Assosiate Professor, Cardiology Section, AKUH, [3]Aga Khan University Hospital, 4Pulmonology Resident Year I, Aga Khan University Hospital, Karachi.
Correspondence: Shahrukh Hashmani. Email: drshahrukhhashmani@gmail.com

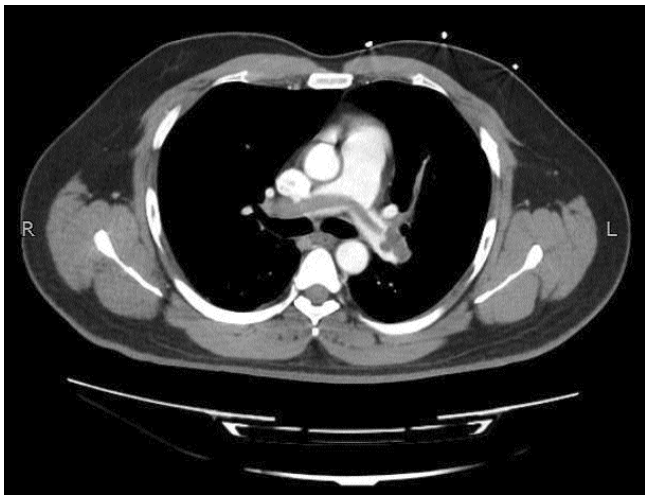

Figure-2: CT chest axial view showing filling defect in bilateral pulmonary arteries "saddle embolism".

ECG showed sinus tachycardia, with RBBB and S1Q3T3 pattern. Chest X-ray was normal with enlarged right pulmonary artery (Fleishner's sign). Transthoracic echocardiogram (Figure-1) revealed severely dilated right ventricle, measuring 37mm, with severely reduced systolic function. McConnell's sign was present (an echocardiographic pattern of RV dysfunction consisting of akinesia of the mid free wall but normal motion at the apex). Possibility of intracranial bleed was ruled out by CT head. After initial stabilization, CTPA (Figure-2) was done that showed extensive pulmonary embolism extending from main pulmonary trunk to right and left pulmonary arteries. No further seizure activity was observed after stabilization of blood pressure. After confirming the diagnosis and within half an hour of his arrival to the emergency room, 10mg of intravenous alteplase was given as a bolus over 10-15 minutes, followed by 90mg of alteplase as slow intravenous infusion over 2 hours. He was later started on intravenous heparin. Significant improvement was observed after thrombolytic therapy. The patient became haemodynamically stable with normal respiratory rate and maintaining oxygen saturations at room air. Repeat echocardiogram showed normal right ventricular size and systolic function. He was discharged on fourth day of his presentation on rivaroxaban, in a stable condition and was doing well on follow up visit.

Discussion

Pulmonary embolism (PE) is the leading cause of morbidity and mortality following surgical procedures. The incidence of Deep Vein Thrombosis (DVT) varies from 10 to 70% in hospitalized patients.[1] Death from DVT is attributed to massive PE, which causes as many as 300,000 deaths annually in the United States. Hospitalized patients are particularly at high risk and studies have shown that only 21 to 35% of the surgeons take appropriate prophylactic steps to prevent DVT and PE for their patients.[3] Our patient was not on any anticoagulation despite limited activity after an orthopaedic surgery.

About 90% of patients with fatal pulmonary embolism die within one to two hours of the first symptom.[4] The patient presented here was lucky enough that his symptoms began when he was in the clinic and received appropriate treatment in time.

Pulmonary embolism can present in various ways ranging from acute unexplained dyspnoea and chest pain to syncope and acute cor-pulmonale. Acute cor-pulmonale syndrome is the extreme condition caused by the complete obstruction of 60 to 75% of pulmonary circulation and can result in sudden death.[5] Syncope occurs in approximately 10% of patients with acute pulmonary embolism and is commonly ascribed to a massive, haemodynamically unstable acute pulmonary embolism.[6]

New onset generalized seizures as a presentation of pulmonary embolism are rare but not a new observation and has been reported in literature.[7] Seizures in the setting of pulmonary embolism can be the result of two possible mechanisms. First, greater than 50% occlusion of the pulmonary vascular tree causes right ventricular failure and impaired left ventricular filling, leading to a reduction in cardiac output, arterial hypotension, reduced cerebral blood flow, and ultimately seizures. Hypoxaemia secondary to ventilation perfusion mismatch may also play an important role in the development of seizure. Our patient had seizures, probably due to hypotension and hypoxia both. However, the diagnosis of generalized seizures was clinical in our case and no EEG was done to confirm seizure activity. The clinical picture was that of generalized seizures and not of syncope or pseudoseizures.

Suspicion of pulmonary embolism was made on the basis of overall clinical picture, which guided us to investigate on the lines of pulmonary embolism. ECG and transthoracic echocardiogram (TTE) supported our clinical suspicion.

In pulmonary embolism, the most common ECG finding is sinus tachycardia; however 33% of patients can have a normal ECG. ECG findings typical of PE as suggested by Chou[8] include S1Q3T3, rightward QRS axis, complete or

incomplete RBBB and sinus tachycardia. Our patient had typical ECG changes.

Transthoracic echocardiography is of tremendous value in the setting of major pulmonary embolism. Echocardiographic findings in patients with MPE include right ventricular dilatation, pulmonary artery dilatation, paradoxical septal motion, right ventricular hypokinesis and tricuspid regurgitation.[9] Echocardiographic findings were also classical in the patient presented here.

Thrombolytics are often used to dissolve or reduce the size of the thromboembolism and improve haemodynamic status and gas exchange. Emergent thrombolysis is the first line of treatment in patients with high risk of PE who present with persistent systemic hypotension.[10] Anticoagulation with heparin should be initiated in the absence of contraindication, once the diagnosis of PE is considered. Early and adequate anticoagulation has shown to decrease mortality;[11] while surgical embolectomy is reserved for refractory shock, ongoing cardiac arrest, or patients with contraindications to thrombolytics. Thrombolytic — alteplase was used appropriately with a good result in our patient.

In our institution we follow an algorithm for major PE, which includes an urgent bedside echocardiogram and a spiral CT scan. Anticoagulation is initiated as soon as PE is clinically suspected if there is no contraindication to it. Thrombolytics and surgical embolectomy are considered in patients presenting with shock and in those with refractory hypoxia.

Conclusion

Massive pulmonary embolism presenting as seizures is an unusual finding. To avoid misdiagnosis and over investigation, clinical suspicion should be made on the basis of overall clinical picture and not just the presence of seizures. TTE along with CTPA can help in making a rapid diagnosis and prompt treatment, which can be lifesaving as was in our case.

Informed Consent: Written informed consent was taken from the patient.

Disclaimer: None.

Conflict of Interest: None.

Funding Disclosure: None.

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