

CASE REPORT

Massive Pulmonary Embolism Presenting As Seizures

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INTRODUCTION

Pulmonary embolism has varied clinical presentations; there are no specific signs and symptoms associated with this disease, so a high level of suspicion is required to diagnose this potentially fatal disease. Although syncope secondary to pulmonary embolism has been reported in the literature, generalized seizures secondary to PE are infrequent, and only a few cases have been reported in the literature. Therefore, we present a case of massive pulmonary embolism with seizures as the initial presentation.

CASE

A 65-year-old female presented in an unresponsive state to a peripheral hospital. She had slept normally the previous evening and was found unresponsive in the morning. Her family members denied any history of illicit drug use. She had urinary and faecal incontinence and evidence of a tongue bite on examination. Her past medical history was significant for hypertension and primary hypothyroidism. She was on a tablet of amlodipine 5mg once a day and a levothyroxine replacement.

On presentation, her GCS was 8/15; her pulses were feeble, with a rate of 110/min, and her blood pressure was 80/40 mmHg. Her SpO₂ was 34% on room air, and her chest examination revealed crepitations in the right infra-scapular area; CNS examination did not reveal any focal weakness. She was resuscitated and required endotracheal intubation for respiratory support, intravenous fluids, and inotropic infusion for circulatory support. Her investigations revealed a pH of 6.9, HCO₃ of 17.2 mEq/L, PCO₂ of 88 mmHg, and

serum lactate of 11.8 mmol/L. Her serum sodium, potassium and blood sugars were 138 mEq/L, 3.6 mEq/L, and 187 mg/dl, respectively. ECG revealed sinus tachycardia, and the chest X-ray was normal. Her cardiac troponin (TnT) tested negative. CT-scan brain did not indicate any bleeding. An initial diagnosis of generalized seizure with aspiration pneumonitis with shock, likely septic, was made. The patient was referred to the tertiary care centre for further management.

The patient was transferred to the intensive care unit. She was managed with ventilatory support, empirical antibiotics, vasopressors, a prophylactic dose of unfractionated heparin and other supportive care. Her hemogram revealed: haemoglobin of 15.1g/dl, total leukocyte count of 17.5×10³/ul and platelet count of 145×10³/ul. Kidney functions revealed serum urea of 128 mg/dl and serum creatinine of 3.1 mg/dl. Liver functions showed serum bilirubin of 2.57 mg/dl, ALT of 93 U/L, AST of 55 U/L and serum albumin of 3.48 g/dl. Her serum calcium and blood sugars were 9.4 mg/dl and 123 mg/dl, respectively. Her serum sodium and potassium levels were 138mEq/L and 4.1mEq/L, respectively. CSF analysis was unremarkable. Blood and urine cultures were sterile. Her echocardiography revealed normal left ventricular contractility and functions and moderate pulmonary hypertension. Her hypotension and oxygenation improved. She was extubated on the third day of hospitalization and required oxygen supplementation @ 5L/min via nasal

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prongs. Her repeat investigations revealed improvement in leukocyte count and renal functions.

Given unexplained shock at presentation, evidence of pulmonary hypertension, persistent hypoxemia and normal chest X-ray, a suspicion of pulmonary embolism was made. A CT pulmonary angiography was done, which revealed the presence large saddle thrombus at the bifurcation of the pulmonary artery (figure 1). A diagnosis of pulmonary embolism was made, and her anticoagulation was increased to a therapeutic dose. MRI brain was subsequently done, which showed hyper-intensities in basal ganglia & periventricular areas, likely representing hypoxic brain insult. Her EEG was done subsequently, which did not reveal any epileptiform activity. She was discharged in stable condition on oral anticoagulants after seven days and did well on subsequent OPD follow-ups.

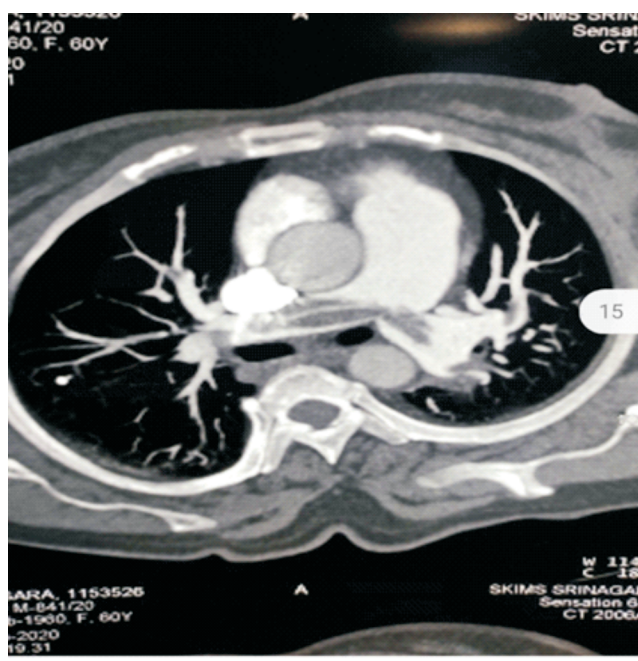


Fig.1: CT-pulmonary angiography showing the presence of clot in pulmonary artery

DISCUSSION

Pulmonary embolism remains a significant cause of morbidity and mortality throughout the world. More than 10 million cases of venous thromboembolism are reported annually¹. It accounts for 5-10% of in-hospital deaths & as a cause of sudden death; it is second only to sudden cardiac death.^{2,3}

Pulmonary embolism is called a “great masquerader”

because of its varied clinical presentations.⁴ Majority of patients present with new onset or worsening of breathlessness, chest pain, cough or unexplained hypotension. Atypical symptoms such as syncope, palpitations, loss of consciousness & abdominal pain may also be present in some patients.⁵

Generalized seizures secondary to underlying pulmonary embolism are rare and may be easily missed during routine evaluation. Literature regarding seizures as a presentation of pulmonary embolism is restricted to case reports only. Sagall et al. described two patients with pulmonary embolism who had presented with loss of consciousness and convulsions, which were attributed to the cerebrovascular event at the presentation time. Similarly, Hamilton et al. described two cases of fatal pulmonary embolisms erroneously diagnosed with the cerebrovascular disease at presentation.⁷ More recently, Fred HL et al., Marine JE et al. and Meyer MA also reported cases of pulmonary embolism presenting with seizures.^{8,9,10}

The exact pathophysiological mechanism for the pathogenesis of seizures in pulmonary embolism is unknown; however, it is suggested that seizures result from hypoxemia and acidosis resulting from respiratory and circulatory failure and transient cerebral hypo-perfusion similar to the mechanism in cardiogenic seizures.

A high degree of suspicion is required for the diagnosis of pulmonary embolism in the setting of a seizure. Diagnosis of pulmonary embolism in the setting of a seizure can be challenging because of many reasons:

1. The patient's post-ictal confusional state may compromise the clinical evaluation.
2. Well-established signs of pulmonary embolism like tachycardia, tachypnea and hypoxemia may be attributed to aspiration pneumonitis, as in our patient.
3. The presence of seizures may lead to neurological workup, which may distract the physician from consideration of pulmonary embolism as the cause of seizures.

However, prompt recognition of this condition is of paramount importance.

CONCLUSION

This case highlights the varied presentation of pulmonary embolism. Therefore, a high degree of suspicion is vital for

diagnosis in atypical case scenarios, as a delay in diagnosis may be detrimental to patient outcomes.

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