



## *Pulmonary Thromboembolism Masquerading as a First-Onset Seizure, A Rare Manifestation: A Case Report*

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*Citation: Arnab Choudhury, Ashish Kumar, Preksha Joshi, Sumeet Arora, Naresh Dhawan (2026) Pulmonary Thromboembolism Masquerading as a First-Onset Seizure, a Rare Manifestation: A case Report. J.of Adv Int Cri Medicine 2(1), 01-03. WMJ/JAICM-110*

### Abstract

*Pulmonary thromboembolism (PTE) is a life-threatening condition that can present with a wide range of symptoms, including atypical neurological manifestations such as seizures. We present a case of a previously healthy young adult who was admitted with a first-onset generalized tonic-clonic seizure and was subsequently diagnosed with massive pulmonary embolism. Clinical workup revealed hypoxia, tachycardia, and inconclusive brain imaging. Elevated D-dimer prompted computed tomography pulmonary angiography, which revealed extensive bilateral pulmonary arterial embolisms. Treatment with low molecular weight heparin followed by direct oral anticoagulants resulted in significant improvement. Pulmonary embolism rarely presents as a new-onset seizure without cardiopulmonary symptoms, complicating the diagnosis. This case underscores the importance of early suspicion, comprehensive imaging, and multidisciplinary management in patients with atypical seizure presentations accompanied by unexplained hypoxia or tachycardia, as timely intervention can reduce morbidity and mortality.*

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**Submitted:** 22.01.2026

**Accepted:** 27.01.2026

**Published:** 08.02.2026

**Keywords:** Pulmonary Thromboembolism, Seizure, Neurological Manifestation, Hypoxia, Atypical Presentation, Case Report

## Introduction

Pulmonary thromboembolism (PTE) typically originates from deep vein thrombosis and often presents with dyspnea, chest pain, or syncope [1]. However, it can manifest in atypical ways, potentially delaying diagnosis. Seizures may rarely be the first presenting symptom caused by profound hypoxemia, hypotension, or paradoxical embolization. Because this is uncommon, PTE is seldom initially considered in patients with a first-time seizure and no epileptic history [2]. We describe a young adult with no known risk factors who experienced generalized tonic-clonic seizures during exercise [3]. The occurrence of bilateral pulmonary emboli highlights the importance of including PTE in the differential diagnosis of unexplained seizures [4].

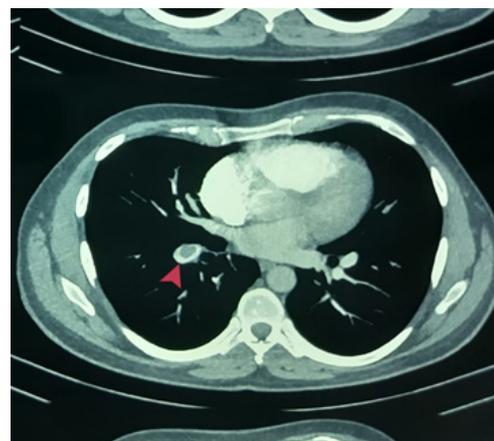
## Case Report

A 38-year-old previously healthy individual presented with 1 episode of generalized tonic-clonic seizure while performing a physical fitness test, followed by 20 minutes of postictal confusion. He had no prior comorbidities or any such episode in the past. On arrival, his Glasgow Coma Scale score was 15, his blood pressure was 146/86 mmHg, his heart rate was 110 beats/min, and his oxygen saturation was 80%, which increased to 90% with 10 L/min oxygen. Physical examination revealed tachycardia, a pronounced pulmonic component of the second heart sound, and clear lungs. There was no preceding history of fever, headache, or focal deficits. Initial laboratory results were unremarkable except for a markedly elevated D-dimer (>10,000 µg/L). Electrocardiography revealed sinus tachycardia with T-wave inversions in lead III (Fig 3) and a brain MRI revealed only nonspecific changes. Chest radiograph done bed side was unremarkable (Fig 4). Bedside ultrasound demonstrated a mildly dilated right ventricle, although no deep vein thrombosis of the limbs was detected. Persistent hypoxia and unexplained tachycardia heightened the suspicion of pulmonary embolism. Confirmatory pulmonary CT angiography revealed bilateral emboli in the right main pulmonary artery, right lower lobe branches, and left proximal artery (Fig 1 & 2). The patient was admitted to the ICU and received oxygen and anticoagulation therapy. Thrombolysis was deferred given his haemodynamic stability and negative cardiac biomarkers, which was consistent with a submassive rather than massive embolism. Low-molecular-

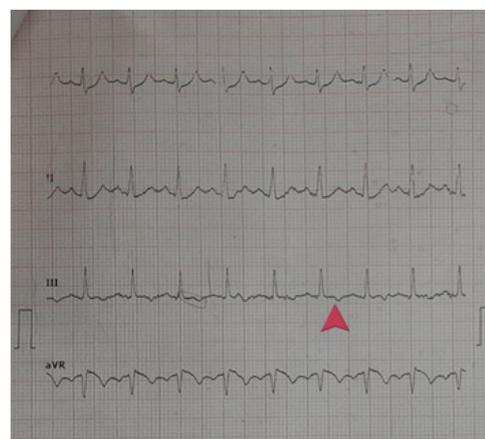
weight heparin (60 mg subcutaneously every 12 hours) prompted rapid improvement, with oxygen saturation exceeding 94% within 24 hours. He was then transitioned to apixaban 5mg twice daily, pending thorough evaluation for potential prothrombotic factors and close monitoring. This case underscores the necessity of considering pulmonary embolism in first-time seizures without obvious neurogenic causes, especially when tachycardia and hypoxia persist.



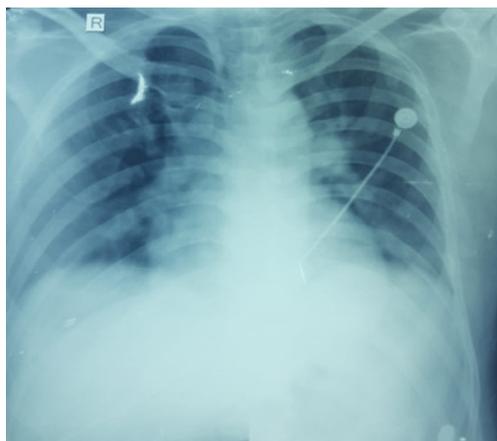
**Figure 1:** Non-Occlusion Partial Filling Defect in Right Proximal Pulmonary Artery



**Figure 2:** Polo Mint Sign- Central Filling Defect Surrounded by Thin Rim of Contrast.



**Figure 3:** T Wave Inversion in Lead III



**Figure 4:** Bedside Chest Xray- AP View

### Discussion

Pulmonary thromboembolism (PTE) is recognized for its cardiopulmonary manifestations but can occasionally present with primarily neurological symptoms [5]. Hypoxic brain injury from significant ventilation–perfusion mismatch and systemic hypotension can result in generalized seizures mimicking primary epileptic events [6]. Additionally, paradoxical embolization through a patent foramen ovale can cause cerebral emboli, leading to ischaemic or seizure-like episodes.<sup>3</sup> Typically, first-onset seizures are attributed to neurogenic causes, and PTE is not routinely considered without classic cardiopulmonary symptoms [7]. However, delays in diagnosing PTE can be fatal. Hence, a high index of suspicion is critical in atypical presentations [8]. When neurological evaluations fail to explain seizures fully especially with persistent hypoxia, D-dimer testing and CT pulmonary angiography can rapidly confirm or exclude PTE. Anticoagulation is the main therapy, and systemic thrombolysis is an option in intermediate-risk patients with right ventricular strain or instability [9]. In this instance, the patient responded well to anticoagulation alone, underscoring the role of risk stratification. Close follow-up is needed to monitor for recurrence and assess the appropriate anticoagulation duration. This case highlights the

importance of early recognition of possible PTE in new-onset seizures and underscores a collaborative, multidisciplinary approach for timely, life-saving management.

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