RESEARCH PAPER

Spectrum Of Embolic Syndromes: A Two-Case Report Of Cerebral Fat Embolism And Chronic Pulmonary Thromboembolism Mimicking Fat Embolism-A Case Report

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ABSTRACT

Background: Embolic syndromes encompass a wide spectrum of clinical conditions, including fat embolism syndrome (FES) and pulmonary thromboembolism (PTE), both of which can present with overlapping clinical and radiological features. "Fat embolism typically occurs following long bone trauma and commonly involves the lungs and central nervous system, whereas pulmonary thromboembolism arises from intravascular thrombus migration and may progress to chronic pulmonary hypertension. Despite distinct pathophysiological mechanisms, both entities may demonstrate similar imaging findings such as ground-glass opacities and vascular abnormalities, leading to diagnostic challenges. Accurate differentiation is essential due to significant differences in management and prognosis.

Case Presentation: We report two cases illustrating the spectrum and diagnostic complexity of embolic syndromes. The first case involves a 19-year-old male who developed acute neurological deterioration following long bone fracture. Magnetic resonance imaging revealed multiple punctate diffusion-restricted lesions in supratentorial and infratentorial regions, consistent with cerebral fat embolism. The second case involves a 55-year-old patient presenting with breathlessness, following an RTA with past history of progressive exertional dyspnea. Initial thoracic imaging showed bilateral diffuse ground-glass opacities with dilated pulmonary arteries, raising suspicion of pulmonary fat embolism. However, computed tomography pulmonary angiography demonstrated chronic intraluminal thrombi in bilateral pulmonary arteries with features of pulmonary hypertension, confirming chronic pulmonary thromboembolism. The presence of similar parenchymal findings in both conditions posed a significant diagnostic challenge. This report highlights the importance of integrating clinical context with advanced imaging in differentiating embolic syndromes. While cerebral fat embolism presents with characteristic MRI findings following trauma, chronic pulmonary thromboembolism may mimic fat embolism on imaging". Recognition of these differences is crucial to avoid misdiagnosis and to guide appropriate management strategies.

Keywords: Fat embolism syndrome, Cerebral fat embolism, Pulmonary thromboembolism, Chronic thromboembolic pulmonary hypertension, Ground-glass opacities, Starfield pattern, CT pulmonary angiography, Embolic syndromes, Diagnostic challenge, Neuroimaging

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INTRODUCTION

Embolic syndromes represent a diverse group of clinical entities characterized by occlusion of vascular beds by intravascular material, leading to organ dysfunction. “Among these, fat embolism syndrome (FES) and pulmonary thromboembolism (PTE) are important causes of acute and subacute morbidity, particularly in hospitalized and trauma patients (1,2). Although both conditions share overlapping clinical and radiological features, their underlying pathophysiology, management strategies, and prognostic implications differ significantly, making accurate diagnosis crucial (3).

Fat embolism syndrome is a well-recognized complication following long bone fractures, orthopedic procedures, and trauma. It typically manifests within 24–72 hours of injury and is classically characterized by the triad of respiratory distress, neurological impairment, and petechial rash, although all components are not always present (4). The pathogenesis involves mechanical obstruction of microvasculature by fat globules released from bone marrow, along with biochemical injury mediated by free fatty acids (5). The central nervous system is frequently affected, with magnetic resonance imaging (MRI) demonstrating characteristic features such as multiple punctate diffusion-restricted lesions, often described as a “starfield pattern,” reflecting widespread microembolic infarcts (6,7).

Pulmonary thromboembolism, on the other hand, results from the migration of thrombi, most commonly originating from deep veins of the lower limbs, into the pulmonary arterial circulation (8). It ranges from acute life-threatening obstruction to chronic thromboembolic disease, which may lead to pulmonary hypertension and right heart strain. Chronic pulmonary thromboembolism (CPTE) is characterized by organized thrombi, vascular remodeling, and persistent obstruction, often associated with imaging findings such as filling defects, arterial dilatation, and secondary parenchymal changes (9). Ground-glass opacities and interlobular septal thickening may be seen, sometimes mimicking other conditions including pulmonary edema and fat embolism (10).

Radiological imaging plays a pivotal role in differentiating these entities. MRI of the brain is highly sensitive in detecting cerebral involvement in fat embolism, while computed tomography pulmonary angiography (CTPA) remains the gold standard for diagnosing pulmonary thromboembolism (11). However, significant diagnostic challenges arise

when imaging features overlap, particularly in cases where pulmonary manifestations of thromboembolism resemble those seen in fat embolism, such as diffuse ground-glass opacities. Such overlap may lead to misinterpretation, delayed diagnosis, or inappropriate management (12).

The present report highlights the spectrum and diagnostic complexity of embolic syndromes through two distinct cases. The first case illustrates classical cerebral fat embolism following long bone fracture, with characteristic MRI findings and neurological deterioration (13). The second case demonstrates chronic pulmonary thromboembolism presenting with bilateral ground-glass opacities and vascular changes, mimicking pulmonary fat embolism on initial imaging (14). Together, these cases underscore the importance of correlating clinical history, imaging findings, and temporal progression to arrive at an accurate diagnosis (15).

This two-case report aims to emphasize the radiological spectrum of embolic diseases, highlight potential diagnostic pitfalls, and reinforce the need for a systematic approach in differentiating fat embolism from its mimics, particularly chronic pulmonary thromboembolism, to ensure timely and appropriate management".

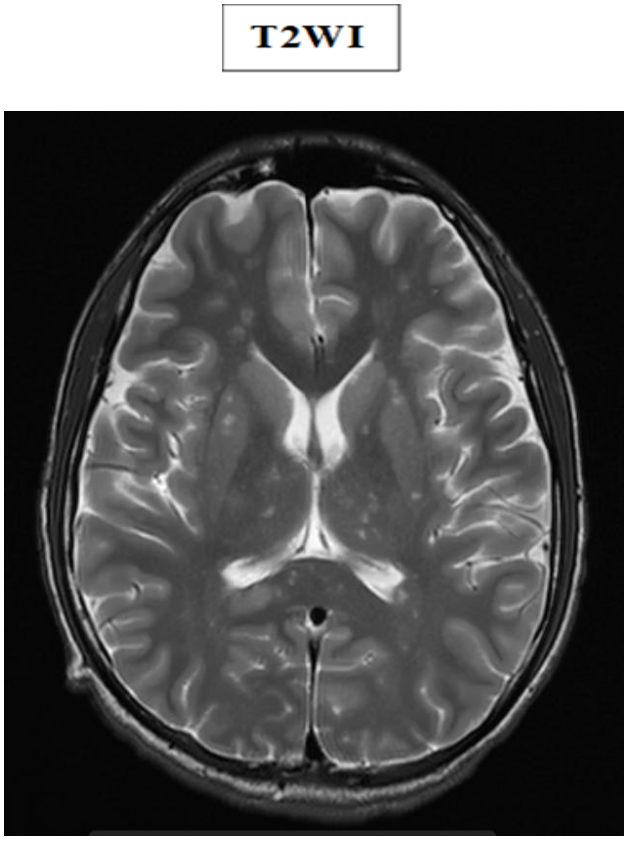
CASE PRESENTATION

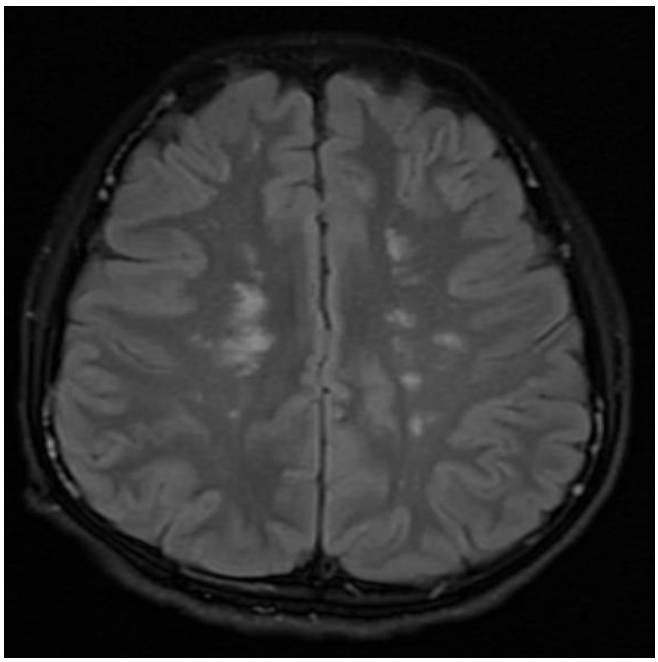
Case 1: Cerebral Fat Embolism Following Long Bone Trauma

Clinical Presentation: A 19-year-old male with no known pre-existing medical illness was brought with a history of high-impact trauma resulting in fracture of the left tibia and fibula. At the time of initial evaluation, the patient was conscious and hemodynamically stable; however, he subsequently developed sudden onset altered sensorium within a short interval. The neurological status deteriorated progressively, with a decline in Glasgow Coma Scale necessitating endotracheal intubation and initiation of mechanical ventilation. The clinical course was further complicated by metabolic acidosis, requiring intensive care monitoring and supportive management. There was no initial evidence of focal neurological deficit, but the rapid decline in consciousness raised suspicion of a diffuse cerebral process.

Neurological Evaluation and Imaging Findings: Detailed neurological assessment revealed global depression of sensorium without lateralizing signs. Magnetic resonance imaging of the brain demonstrated multiple, discrete punctate lesions distributed diffusely across both cerebral hemispheres and infratentorial structures. These lesions appeared

hypointense on T1-weighted images and hyperintense on T2-weighted and FLAIR sequences, with corresponding areas of diffusion restriction on diffusion-weighted imaging, indicative of acute to subacute ischemic insults. The involvement of subcortical white matter, deep grey nuclei, cerebellum, and splenium of the corpus callosum produced a characteristic “starfield pattern,” highly suggestive of cerebral fat embolism. Electroencephalographic evaluation revealed diffuse slowing of background activity, consistent with generalized cerebral dysfunction. Follow-up computed tomography of the brain did not demonstrate any new intracranial pathology, supporting the non-hemorrhagic and embolic nature of the lesions.





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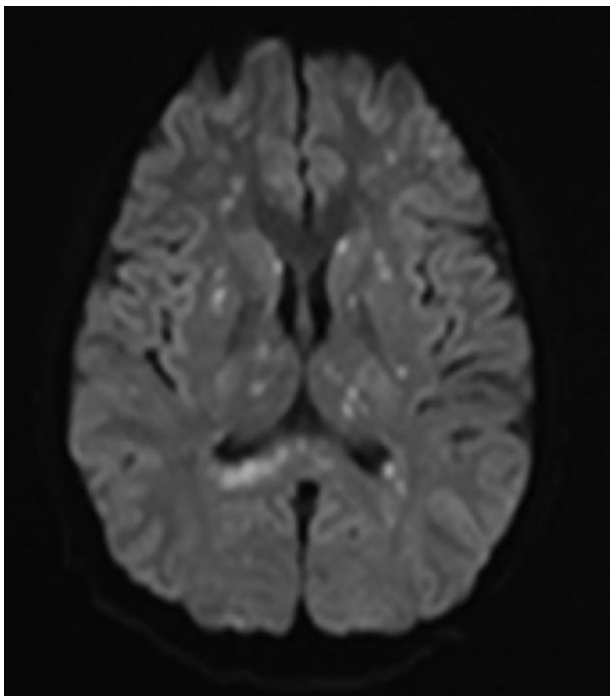

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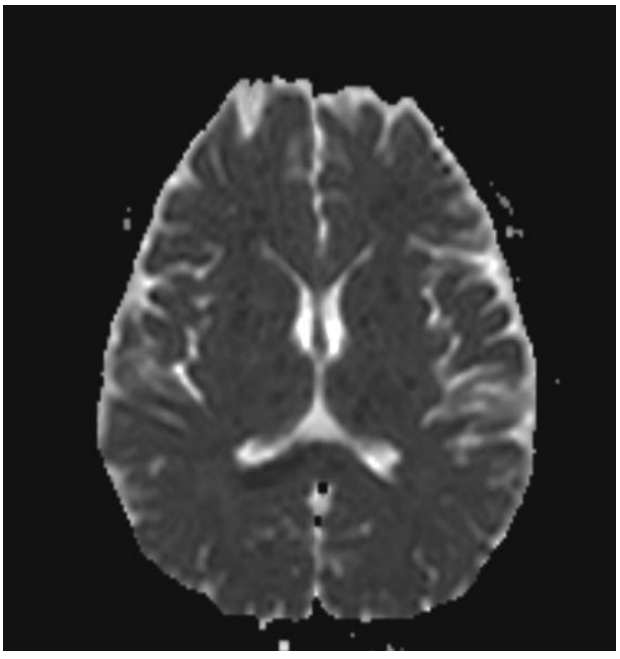


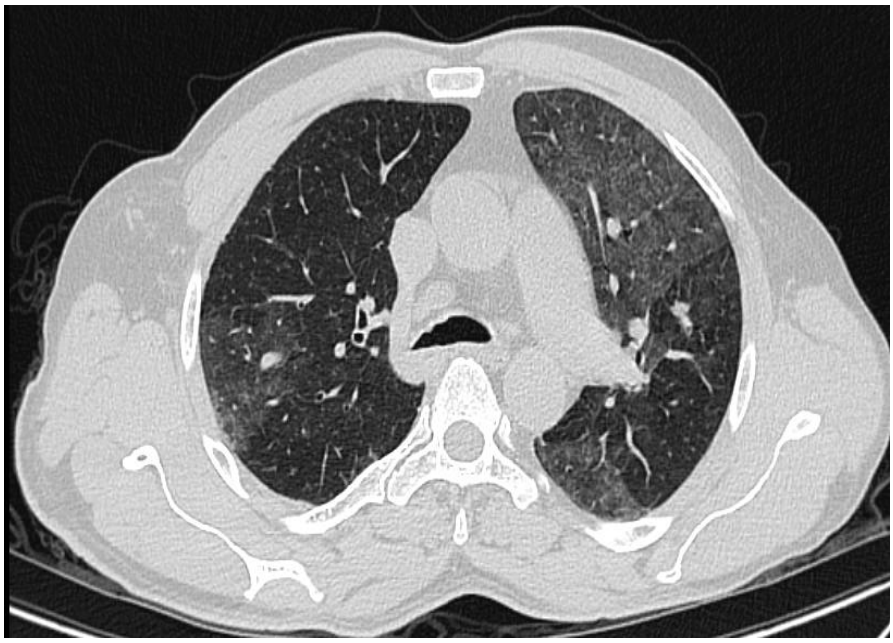
ADC

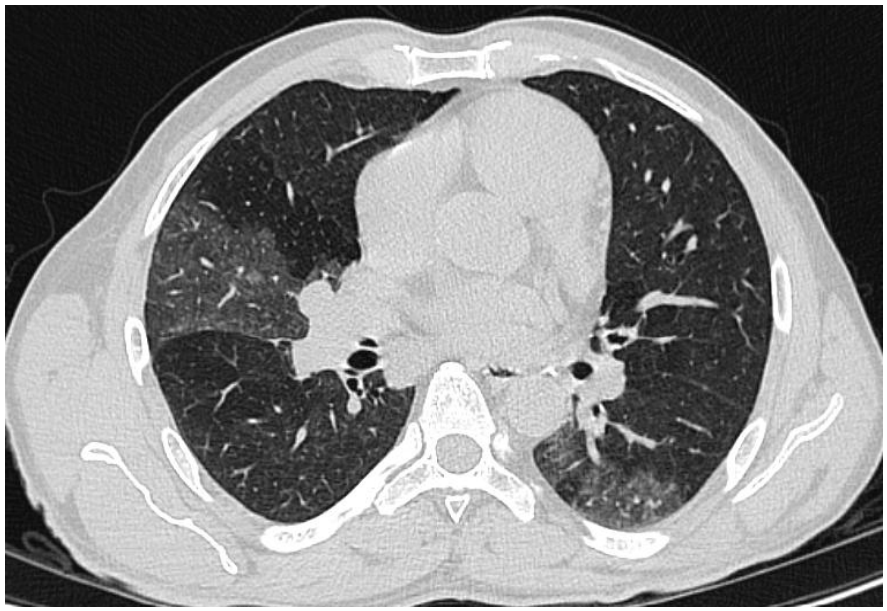
Systemic Evaluation and Hospital Course: During the course of hospitalization, the patient remained ventilator-dependent and required prolonged respiratory support, ultimately necessitating tracheostomy. Laboratory investigations showed leukocytosis with predominant neutrophilia, raising concern for secondary infection and sepsis. Cardiovascular assessment revealed preserved left ventricular systolic function; however, features of moderate pulmonary arterial hypertension were noted, likely secondary to systemic inflammatory and embolic processes. Orthopedic stabilization of the long bone fracture was performed using definitive surgical fixation. Based on the temporal relationship with trauma, rapid neurological deterioration, and characteristic MRI findings, a diagnosis of cerebral fat embolism syndrome with associated hypoxic ischemic encephalopathy was established. The patient was managed with comprehensive supportive care including anticonvulsant therapy, broad-spectrum antibiotics, anticoagulation, corticosteroids, and meticulous critical care monitoring, leading to gradual stabilization of clinical status.

Case 2: Chronic Pulmonary Thromboembolism with Parenchymal Changes Mimicking Fat Embolism

Clinical Presentation: A 55-year-old patient presented with complaints of increased breathlessness, following an RTA with past history of progressive exertional dyspnea. The symptoms were suggestive of an underlying cardiopulmonary pathology, with gradual functional limitation. There was no definitive history of acute chest pain or hemoptysis; however, the chronicity of symptoms raised suspicion for a long-standing vascular or parenchymal pulmonary disorder.

Thoracic Imaging and Initial Assessment: Initial evaluation with high-resolution computed tomography of the thorax revealed bilateral, patchy geographic ground-glass opacities involving multiple lobes, including both upper and lower lung zones. These findings were accompanied by cardiomegaly and significant dilatation of the main pulmonary artery and its branches, indicating the presence of pulmonary arterial hypertension. The diffuse parenchymal involvement, in conjunction with vascular enlargement, led to consideration of differential diagnoses including pulmonary fat embolism and pulmonary edema. Further evaluation was warranted to assess the pulmonary vasculature.

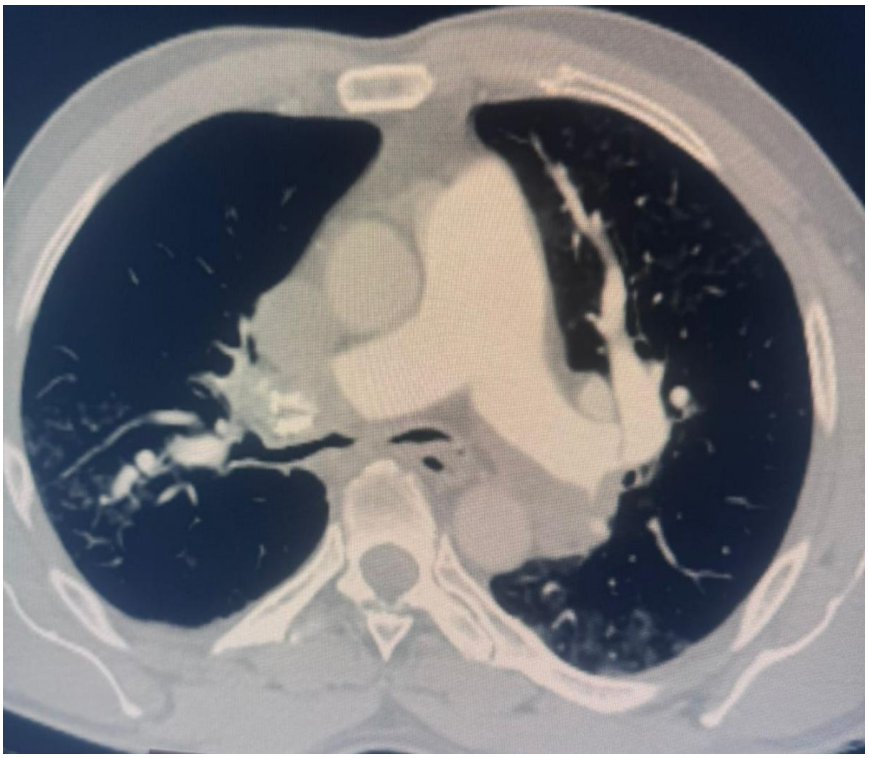


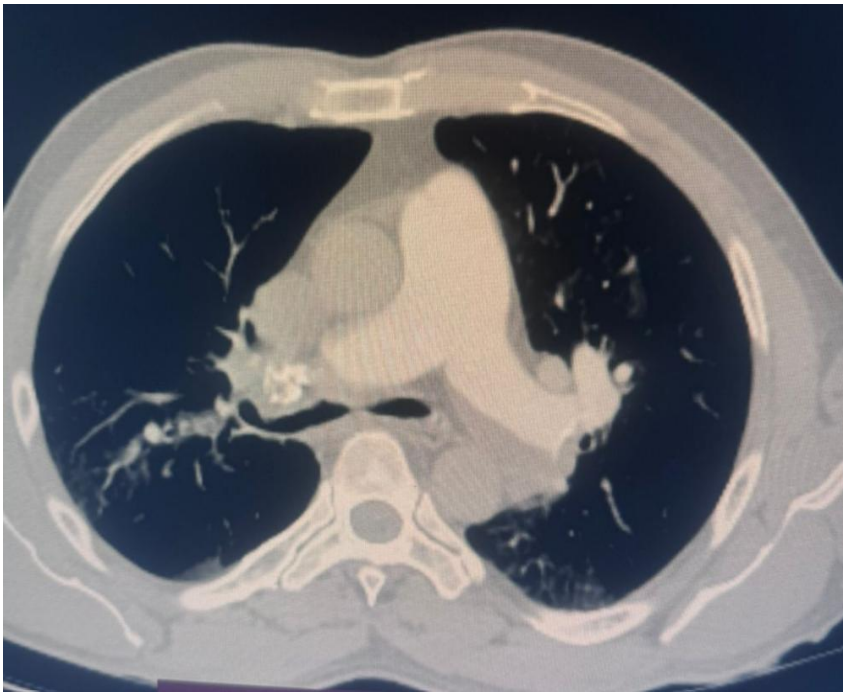


HRCT

CT Pulmonary Angiographic Findings: Subsequent computed tomography pulmonary angiography demonstrated intraluminal filling defects within both the right and left main pulmonary arteries. On one side, the thrombus extended into the lobar branches and resulted in near-complete luminal compromise, while on the contralateral side, a long segment partial filling defect was observed along the vessel wall. Notably, the presence

of calcific foci within the thrombus suggested chronicity and organization. These findings were diagnostic of chronic pulmonary thromboembolism.





CTPA

Associated Vascular and Parenchymal Changes: In addition to the central thromboembolic disease, there was evidence of secondary vascular remodelling, with segmental pulmonary arteries appearing dilated and tortuous. Adjacent lung parenchyma demonstrated areas of ground-glass attenuation and interlobular septal thickening across multiple segments, reflecting altered perfusion and chronic vascular compromise. The coexistence of these parenchymal findings with vascular abnormalities created a radiological picture that closely mimicked pulmonary fat embolism, thereby posing a significant diagnostic challenge. However, the direct visualization of organized thrombus within the pulmonary arterial system on angiographic imaging confirmed the diagnosis of chronic pulmonary thromboembolism with associated pulmonary hypertension.

DISCUSSION

Fat embolism syndrome (FES) and pulmonary thromboembolism (PTE) represent important yet often overlapping entities within the spectrum of embolic disorders, posing significant diagnostic challenges in clinical and radiological practice. “The present two-case report highlights this spectrum by demonstrating classical cerebral fat embolism in one case and chronic pulmonary thromboembolism mimicking fat embolism in the other, emphasizing the importance of clinicoradiological correlation.

Fat embolism syndrome is most commonly associated with long bone fractures and typically manifests within 24–72 hours following trauma, as described by Kwiatt and Seamon (2013) and Adeyinka and Pierre (2022) (1,2). The first case in the present study closely aligns with this classical presentation, wherein a young patient developed acute neurological deterioration following a long bone fracture. The pathophysiology involves both mechanical obstruction of microvasculature by fat globules and biochemical injury mediated by free fatty acids, leading to widespread endothelial damage and inflammation, as elaborated by Alrasheed et al. (2026) (7). The characteristic “starfield pattern” observed on MRI in our case has been consistently reported in literature as a hallmark of cerebral fat embolism, reflecting multiple microinfarcts in the brain parenchyma (Kassimi et al., 2021; Wei et al., 2026) (5,13).

Neurological involvement in FES is often associated with diffuse cerebral dysfunction, as also noted in our patient, supported by abnormal

electroencephalographic findings. Jude et al. (2025) further emphasized the role of transcranial Doppler in detecting cerebral microemboli, highlighting the dynamic nature of embolic load and its correlation with clinical status (6). The presence of hypoxic ischemic encephalopathy in our case underscores the severity of systemic involvement, consistent with previously reported severe presentations.

In contrast, pulmonary thromboembolism represents a distinct pathological entity involving thrombotic occlusion of pulmonary arteries. Chronic thromboembolic disease, as seen in the second case, results from unresolved thrombi leading to vascular remodeling, pulmonary hypertension, and secondary parenchymal changes. Sabbula et al. (2024) described chronic thromboembolic pulmonary hypertension as a progressive condition characterized by organized thrombus and persistent vascular obstruction, findings that correlate with the calcified intraluminal thrombus and dilated pulmonary arteries observed in our case (9).

The radiological overlap between FES and PTE is a well-recognized diagnostic pitfall. Ground-glass opacities and interlobular septal thickening, seen in both of our cases, are non-specific findings that may represent edema, hemorrhage, or inflammatory changes. Qi et al. (2024) and Newbigin et al. (2016) reported that pulmonary fat embolism commonly presents with diffuse ground-glass opacities and patchy consolidations, which can closely mimic other pulmonary pathologies (11,12). Similarly, Donuru et al. (2024) highlighted that interlobular septal thickening may be seen in a variety of vascular and interstitial conditions, further complicating diagnosis (10).

The second case in the present study exemplifies this diagnostic challenge, where initial imaging suggested pulmonary fat embolism; however, CT pulmonary angiography confirmed chronic thromboembolism. A similar overlap has been described by Munagama et al. (2024), where fat embolism mimicked pulmonary embolism, reinforcing the need for definitive vascular imaging (14). Peracaula et al. (2024) emphasized that CT pulmonary angiography remains the gold standard for diagnosing pulmonary embolism, allowing direct visualization of intraluminal thrombus and assessment of disease chronicity (8).

Thus, the present study highlights the importance of integrating clinical history, temporal progression, and advanced imaging modalities in differentiating embolic syndromes. While FES is typically acute and trauma-related with characteristic MRI findings, chronic PTE presents with organized thrombus, pulmonary hypertension, and secondary parenchymal

changes that may mimic fat embolism". Recognizing these distinctions is essential to avoid misdiagnosis and ensure appropriate management.

CONCLUSION

This two-case report highlights the diverse spectrum of embolic syndromes and the diagnostic challenges posed by overlapping clinical and radiological features. Cerebral fat embolism, as demonstrated in the first case, typically follows long bone trauma and presents with characteristic MRI findings such as the "starfield pattern," aiding in early recognition. "In contrast, the second case underscores chronic pulmonary thromboembolism presenting with bilateral ground-glass opacities and vascular changes that can closely mimic pulmonary fat embolism. These similarities may lead to diagnostic ambiguity, particularly on initial imaging.

Accurate differentiation requires careful correlation of clinical history, temporal evolution, and advanced imaging modalities, especially CT pulmonary angiography for definitive vascular assessment. Early and precise diagnosis is crucial, as management strategies and prognostic implications differ significantly between these entities". This report emphasizes the importance of a systematic approach to embolic syndromes to avoid misdiagnosis and ensure appropriate and timely patient care.

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