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Case Report

When Bone Breaks the Brain: A Case Report of Cerebral Fat Embolism

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Abstract

Objective: Cerebral fat embolism (CFE) is a rare but potentially life-threatening complication of long-bone fractures. It poses a significant diagnostic challenge due to its heterogeneous clinical presentation and frequently normal findings on early investigations. This report describes an atypical case of delayed-onset cerebral fat embolism characterized by neurological deterioration despite unremarkable initial pulmonary and neurodiagnostic findings and the absence of an intracardiac shunt, which is commonly implicated in the passage of fat emboli into the arterial circulation. A focused review of the literature is also provided to emphasize key diagnostic considerations and clinical implications.

Case Presentation: A 20-year-old male presented following a road traffic accident with a right subtrochanteric femur fracture. The patient was neurologically intact and hemodynamically stable at admission. Forty-eight hours later, he developed acute respiratory distress followed by progressive neurological deterioration, culminating in coma and the need for mechanical ventilation. Initial investigations, including computed tomography of the brain (CT), computed tomography pulmonary angiography (CTPA), electroencephalography (EEG), routine laboratory tests, and transthoracic echocardiography (TTE), were unremarkable, with no evidence of a right-to-left intracardiac shunt. Persistent unexplained neurological impairment prompted magnetic resonance imaging of the brain (MRI) on day five, which demonstrated multiple bilateral punctate diffusion-restricted lesions in the subcortical and deep white matter, forming the characteristic starfield pattern consistent with cerebral fat embolism. The patient was managed with supportive critical care, systemic corticosteroids, and rehabilitation, resulting in gradual neurological recovery and a favorable functional outcome.

Conclusion: This case highlights the diagnostic challenge posed by CFE. It emphasizes the importance of maintaining a high index of suspicion in trauma patients who develop unexplained neurological symptoms, particularly when initial imaging is inconclusive. Prompt MRI evaluation and timely life-supportive management are essential for better outcomes.

Keywords: Fat embolism, Femoral fracture, Pulmonary embolism, Traffic accident

Introduction

Cerebral fat embolism (CFE) is an uncommon but potentially life-threatening complication that can arise after trauma to long bones or the pelvis. Its reported incidence ranges from 0.9% to 11%, with an average mortality rate of approximately 10% (1-3). The widely accepted mechanism suggests that following a fracture, fat droplets are released into the bloodstream and form emboli. Two main theories have been proposed to explain the early and delayed symptoms seen in fat embolism syndrome (FES). The mechanical theory holds that the trauma-induced rise in intramedullary pressure forces marrow fat into the venous circulation. This can lead to respiratory and neurological symptoms, as well as the development of non-palpable petechiae, typically on the upper body, including the axillae, trunk, and conjunctiva, within 2 to 3 days after injury (4,5). This classic triad defines FES. Subclinical cases often present as impaired gas exchange and petechial rash, while severe cases may progress to acute respiratory distress, right heart failure, or even sudden brain death. The chemical theory attributes the delayed effects of FES to the body's inflammatory response following trauma. According to this hypothesis, inflammatory mediators convert fat into free fatty acids and glycerol, which can damage vascular endothelium (6,7). This mechanism helps explain the delayed onset of neurological symptoms

such as confusion, hemiplegia, aphasia, apraxia, and coma (8). Both theories contribute to our understanding of the complex clinical presentation of CFE.

Case Presentation

A 20-year-old male presented to the emergency department following a road traffic accident involving a motorcycle and a motor vehicle. He had no history of alcohol consumption, substance abuse, seizure disorder, or any known chronic medical illness. On admission, he was alert and oriented, with a Glasgow Coma Scale (GCS) score of 15/15. His vital signs were stable: blood pressure (BP) 110/70 mmHg, heart rate (HR) 92 bpm, respiratory rate (RR) 20 breaths/minute, and oxygen saturation (SpO_2) 99% on room air. Physical examination revealed swelling and tenderness over the right hip and leg, along with mild tenderness and purpuric rashes in the left axillary region. No other systemic abnormalities were noted. Radiographic imaging confirmed a right subtrochanteric femur fracture (Figure 1). On day 2 of admission, the patient developed sudden tachypnea and tachycardia, accompanied by a drop in oxygen saturation. An arterial blood gas (ABG) performed immediately revealed values within normal limits. At that time, he remained alert, with a BP of 110/78 mmHg, HR of 115 bpm, RR of 24 breaths/minute, and SpO_2 of 91% on room air. Noninvasive ventilation with BiPAP was initiated, resulting in a transient improvement in oxygenation. However, his clinical condition deteriorated progressively over the next few hours, with increasing drowsiness and a further decline in SpO_2 to 85%, necessitating endotracheal intubation and mechanical ventilation. Post-intubation ABG analysis showed a pH of 7.53, pCO_2 of 30.2 mmHg, paO_2 of 80 mmHg, bicarbonate (HCO_3^-) of 24 mmol/L, and lactate level of 1.4 mmol/L.

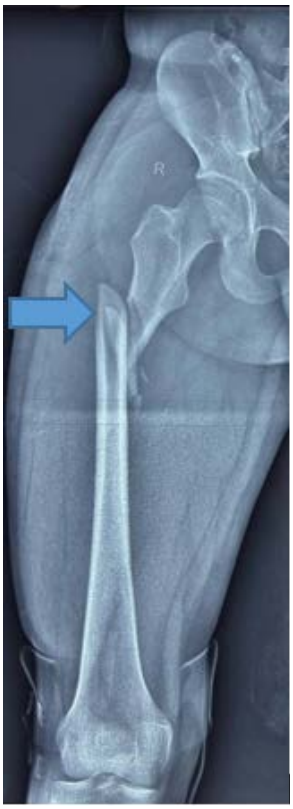

Figure 1. Right femur fracture, shown by the blue arrow

A clinical diagnosis of pulmonary fat embolism was initially considered. However, chest X-ray, D-dimer levels, and CT pulmonary angiography were unremarkable, effectively ruling out pulmonary embolism. Despite ventilatory support, the patient's GCS deteriorated to E1VTM1. A non-contrast CT scan of the brain was performed to exclude intracranial hemorrhage, but the findings were normal. Routine laboratory investigations, including complete blood count, liver and renal function tests, serum electrolytes, and ABG, revealed values within normal limits.

Due to the persistently low GCS, an electroencephalogram (EEG) was performed to evaluate for non-convulsive status epilepticus or metabolic encephalopathy; however, the EEG findings were unremarkable. The patient's neurological status remained unchanged over the subsequent days. On day 5, given the continued poor GCS (E1VTM1) and a normal EEG, a brain MRI was obtained. It revealed hyperintense lesions on FLAIR and diffusion-weighted imaging (DWI) sequences in the bilateral centrum semiovale and occipital subcortical white matter, with associated multiple scattered punctate foci of restricted diffusion, forming the classic "starfield pattern" characteristic of CFE (Figure 2). At that time, neurological examination indicated a comatose state with no response to external stimuli and absence of purposeful movements. The pupils were mid-dilated and sluggishly reactive, and plantar reflexes were bilaterally flexor. A detailed transthoracic echocardiographic evaluation by an experienced cardiologist was performed to assess for a potential intracardiac shunt as a source of embolism, but no abnormalities were detected.

Due to prolonged ventilator dependence, a tracheostomy was performed on day nine. Gradual neurological improvement began around day fourteen; the patient started following verbal commands, and limb power improved to 3/5. MRI of the spine ruled out any spinal pathology. He was extubated successfully with stable vital signs and normal oxygen saturation on room air. His GCS improved to $E4V_TM5$. Subsequently, open reduction and internal fixation (ORIF) of the femoral fracture was carried out using long proximal femoral nailing, with an uneventful postoperative course. After 25 days in intensive care, the patient was discharged in a stable condition with full GCS and motor power of 4/5 in all limbs. He received intravenous (IV) ceftriaxone (1 g twice daily), IV hydrocortisone (100 mg three times daily), subcutaneous enoxaparin (0.6 mg once daily), analgesics including paracetamol (1 g IV every 6 hours) and tramadol (50 mg IV as needed), and intravenous fluids and enteral feeding. Limb immobilization was maintained using skeletal traction.

This case describes a rare and diagnostically challenging presentation of CFE in a trauma patient who was initially asymptomatic. Two days post-injury, the patient developed

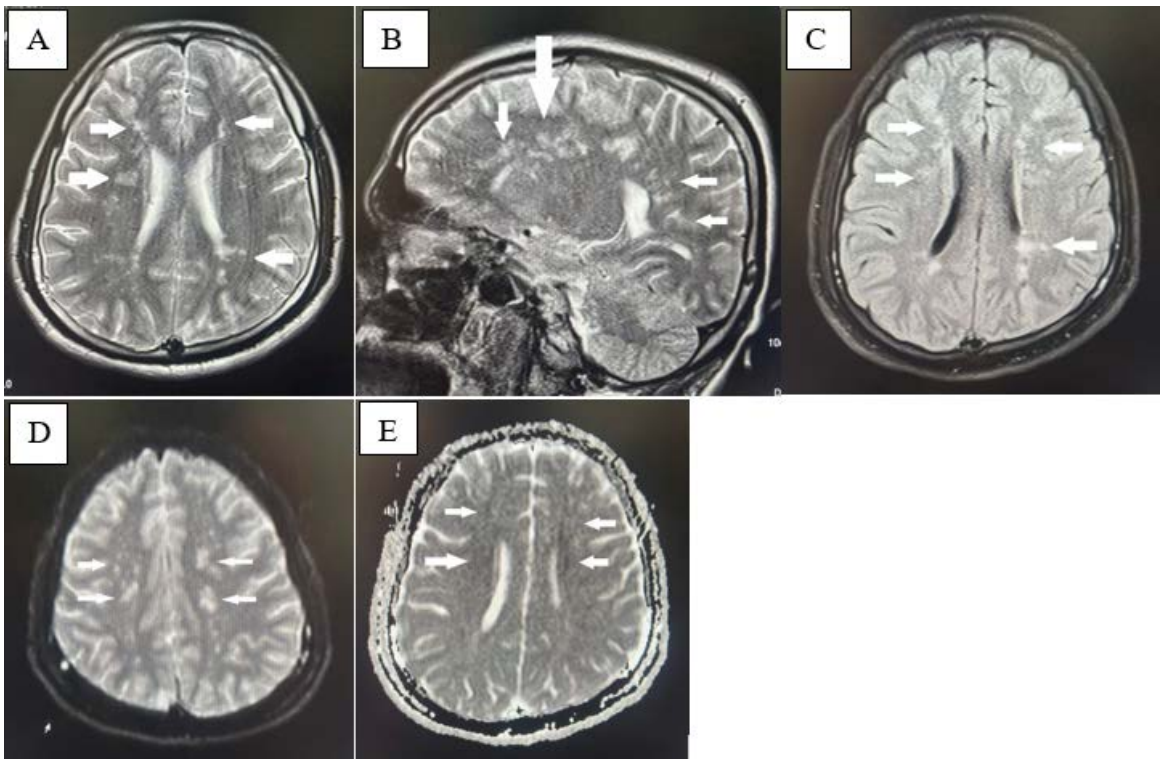

Figure 2. MRI brain images demonstrating multiple bilateral hyperintense foci within the subcortical and deep white matter on axial T2-weighted (a), sagittal T2-weighted (b), axial flair (c), and axial DWI (d) sequences. Corresponding hypointense areas on the axial ADC map (e) suggest areas of restricted diffusion consistent with cerebral fat embolism. These changes are highlighted by white arrows

clinical signs suggestive of pulmonary embolism; however, workup for pulmonary fat embolism, including imaging and laboratory tests, yielded normal results. Despite stable vital signs and normal metabolic, electrolyte, and laboratory parameters, the patient's condition progressively deteriorated, leading to respiratory failure and coma. Initial CT brain and EEG were unremarkable. Repeat neuroimaging with MRI later revealed findings consistent with CFE. This case underscores the importance of maintaining a high index of suspicion for CFE in trauma patients with delayed neurological decline, even when pulmonary assessments, early neuroimaging, EEG, and echocardiography appear normal. Such atypical presentations highlight the role of a timely repeat MRI in patients without prior neurological or systemic illness.

Discussion

Fat embolism is a recognized complication following long bone and pelvic fractures (9). It typically occurs when trauma disrupts marrow-containing bone, allowing fat droplets from the bone marrow to enter the venous system and subsequently the systemic circulation. Similarly, increased intramedullary pressure during orthopedic procedures may also drive marrow fat into the venous bloodstream. Once in circulation, these fat globules can travel to various organs, leading to embolic obstruction, particularly affecting the lungs, brain, and skin (4,10,11).

Symptoms of CFE generally manifest within 24 to 72 hours post-injury and tend to develop progressively (11). The classic clinical presentation includes a triad of respiratory distress, altered mental status, and petechial rash. However, this triad is not always present, with the rash absent in 50–80% of cases. Several diagnostic frameworks exist, including Gurd›s criteria, which combine clinical and laboratory indicators, and Lindeque's criteria, which focus solely on respiratory findings (12,13). However, neither includes neuroimaging, which has shown high specificity for CFE. CFE arises when fat emboli access the arterial system. This can happen either through a right-to-left cardiac shunt, such as a patent foramen ovale (PFO), or by direct passage of small fat droplets through the pulmonary capillaries. Although PFO has been documented in some cases, its absence in others suggests that both mechanisms are plausible (10-4). In our case, no intracardiac shunt was detected, making trans-pulmonary passage the most likely explanation for the CFE.

Neurological symptoms in CFE vary widely, from confusion to deep coma, and may occasionally include seizures or focal deficits. These manifestations can result from either cerebral embolization or hypoxia secondary to pulmonary compromise (4). Interestingly, many cases demonstrate good neurological recovery over time. Mortality rates range between 5% and 15%, and respiratory failure is the most common cause of death. Diagnosis of CFE is primarily clinical, though brain imaging, especially MRI, plays a key role. CT scans often appear normal, whereas MRI may reveal a "starfield" pattern, considered characteristic of CFE (14,15). Treatment remains largely supportive, focusing on respiratory stabilization and neurological protection. The use of corticosteroids has been explored, though evidence of benefit remains inconclusive and their routine use remains debated (2,16).

Radiological findings in cerebral fat embolism vary with the type of embolism. In microembolism, early CT

brain scans are typically normal, as seen in our case. In macroembolism, imaging may reveal a hypodense vessel sign, and as the condition progresses, features consistent with ischemic stroke may develop (9). However, cerebral fat microembolism has distinct radiographic features on MRI. MRI is more sensitive in detecting cerebral fat microembolism and reveals characteristic patterns. Lesions are typically bilaterally symmetric and involve the subcortical and deep white matter, including areas such as the subcortical U-fibers, corpus callosum, and internal capsule. On diffusion-weighted imaging (DWI), early findings (1-4 days) often show multiple scattered punctate foci of restricted diffusion, the classic "starfield pattern" similar to the one observed in our case. Later stages (5–14 days) may demonstrate more confluent regions of cytotoxic edema in white matter. Susceptibility-weighted imaging (SWI) may reveal numerous microhemorrhages, described as the "walnut kernel pattern (15,17,18). T2/FLAIR sequences can show small hyperintense areas consistent with vasogenic edema, while T1-weighted images may exhibit corresponding hypointense signals. CFE is predominantly a clinical diagnosis, though neuroimaging can offer valuable supportive evidence. While CT scans of the brain are frequently unremarkable, MRI, particularly DWI, often demonstrates the classic "starfield" pattern with numerous punctate areas of restricted diffusion indicative of widespread microvascular embolization, as observed in our case (8). Similar to our case, several reports in the literature have diagnosed CFE based on clinical suspicion corroborated by the characteristic "starfield" pattern on brain MRI (19).

In summary, although fat embolism syndrome and its cerebral form are relatively rare in clinical practice, their asymptomatic occurrence is likely underdiagnosed, particularly in patients with long bone or pelvic fractures. Clinicians should maintain a high index of suspicion in trauma patients who develop respiratory compromise, neurological symptoms, or petechial rash. FES lacks specific diagnostic tests or definitive criteria and is primarily a diagnosis of exclusion. Management focuses on supportive care, particularly treating complications. Early surgical stabilization of long-bone fractures has been shown to reduce the incidence of FES.

Conclusion

CFE should be suspected in trauma patients presenting with unexplained neurological decline, particularly when initial evaluations, including brain CT, appear normal. A high index of clinical suspicion, coupled with supportive MRI findings, is essential for early diagnosis.

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None.

Authors' Contribution

Conceptualization: Asif Dabeer Jafri, Rehana Ansari, Tanmoy Ghatak, Ratender Kumar Singh.
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Formal analysis: Asif Dabeer Jafri, Ratender Kumar Singh.
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Competing Interests

None.

Ethical Approval

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