International Journal of Research in Orthopaedics
Khalil KS et al. Int J Res Orthop. 2025 Jul;11(4):914-917
http://www.ijoro.org

DOI: https://dx.doi.org/10.18203/issn.2455-4510.IntJResOrthop20251818

Case Report

Fat embolism syndrome before surgical treatment of open tibial and fibular shaft fracture: a case report

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Received: 14 May 2025
Accepted: 17 June 2025

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ABSTRACT

A 26-year-old male with no significant medical history sustained open fractures of the left tibia and fibula following a workplace accident. Initially alert and stable, he developed sudden neurological deterioration (GCS 9/15) 10 hours post-injury, without respiratory compromise or cutaneous signs. CT was unremarkable; diffusion-weighted MRI revealed widespread acute cerebral infarcts. No intracardiac shunt was found on echocardiography. Supportive care led to gradual neurological improvement, and successful surgical fixation followed. Isolated cerebral fat embolism can precede orthopedic intervention and present without classic signs. Early neuroimaging and high clinical vigilance are crucial for prompt diagnosis and management.

Keywords: Fat embolism syndrome, Cerebral fat embolism, Tibial fracture, Neurological deterioration, Diffusion-weighted MRI, Orthopedic trauma

INTRODUCTION

Fat embolism syndrome (FES) is a rare but fatal condition in which fat droplets enter the bloodstream, potentially leading to vascular occlusion and organ dysfunction. These fat globules typically travel through the venous system to the lungs, resulting in pulmonary fat embolism (PFE) if confined to the pulmonary circulation. In some cases, fat droplets may bypass the pulmonary circulation and enter the systemic circulation, where they can lodge in organs such as the brain, kidneys, skin, eyes, or heart, leading to a multi-organ condition known as FES, which can be life-threatening.[1]

FES is most often associated with traumatic injuries or orthopedic surgeries, with its occurrence in non-traumatic settings being rare. It is associated with a significant mortality rate of 5-30%.[2,3] The syndrome is commonly recognized by the classic triad of respiratory compromise, neurological impairment, and petechial skin rashes. Neurological symptoms, which typically develop within 12 to 72 hours of the inciting event, can include cerebral infarction, spinal cord ischemia, hemorrhagic stroke, seizures, and coma. Additional manifestations may involve confusion, autonomic dysfunction, and retinal ischemia. These neurological signs are often diffuse and of variable severity, posing a diagnostic challenge, especially in the absence of accompanying pulmonary and dermatological signs.[4]

In this report, we present an unusual case of isolated cerebral fat embolism that occurred preoperatively in a patient with an open tibial and fibular fracture, in the absence of acute respiratory compromise or an identifiable right-to-left cardiac shunt. The diagnosis was considered due to the sudden onset of unexplained neurological deterioration, including a low Glasgow coma scale (GCS) score, confusion, and unresponsiveness, despite stable vital signs and a minor fracture. This case is reported to highlight the atypical presentation of FES, emphasize the importance of early recognition in non-classical scenarios, and raise awareness among clinicians to include cerebral

fat embolism in the differential diagnosis when neurological symptoms develop unexpectedly in trauma patients, even before surgery.

CASE REPORT

A 26-year-old Nepali male, weighing 70 kg, sustained open fractures of the tibia and fibula (Gustilo type 2) after his leg became trapped between metal pipes during a fall from height at his workplace (Figure 1). Upon arrival at the emergency department, he was awake, alert, and oriented. He was hemodynamically stable, with an oxygen saturation of 98% on room air and a respiratory rate of 17 breaths per minute. The patient had no significant past medical history. He reported regular alcohol consumption but denied smoking.

On physical examination, all compartments of the left leg were soft. There was a 3 cm bleeding wound on the anterior aspect of the left leg, along with abrasions and a puncture wound, as well as a 10 cm laceration on the dorsum of the left foot. Peripheral pulses were palpable, capillary refill was brisk, and the patient was able to move all toes.

In the emergency department, wounds were irrigated with Dermacyn and Betadine. The anterior leg laceration was closed with three sutures, while the dorsal foot laceration was closed using eight surgical staples. Sterile dressings were applied, and a plaster slab was placed above the knee. The orthopaedic team evaluated the patient and admitted him for observation, limb elevation, and planned surgical fixation.

Approximately 10 hours post-injury, the patient became drowsy and was noted to have a Glasgow coma scale (GCS) score of 9/15. A rapid response code was activated. On assessment, his vital signs included a blood pressure of 105/73 mmHg, heart rate of 75 bpm, oxygen saturation of 94% on room air, and random blood glucose of 6.7 mmol/l. He responded to deep stimuli with facial grimacing and occasional localization to pain, producing incomprehensible sounds. No petechial rash, tremors, diaphoresis, or muscle rigidity were observed.

Initial laboratory workup, including toxicology and serologic tests, along with a head CT scan, were unremarkable. An MRI with diffusion-weighted imaging revealed innumerable tiny acute lacunar infarcts scattered across both cerebral hemispheres. (Figure 2). Further investigations were conducted to rule out embolic sources, including echocardiography, ECG, Holter monitoring, carotid doppler ultrasound, and a thrombophilia screen.

The patient was started on dual antiplatelet therapy with aspirin and clopidogrel. The following day, the patient showed improvement in consciousness but continued to be disoriented and unable to follow commands. Clopidogrel was discontinued, and aspirin was continued as monotherapy. Surgery was delayed due to the patient's altered mental status, and the anesthesia team assessed him as unfit for the procedure at that time. Given that he was hemodynamically stable and the wound was clean, the surgery was not considered urgent. On the sixth day of admission, the patient underwent surgical debridement and external fixation of the left leg. This included the placement of 2.6 mm Schanz screws proximally and distally, connected via a carbon rod and secured with a Schanz clamp (Figure 3). His postoperative course was uneventful from a surgical standpoint, though his confusion persisted.

Two weeks after admission, the external fixator was removed, and definitive intramedullary nailing of the tibia was performed. (Figure 4). Patient was discharged 21 days from the date of admission, he was mobilizing on a wheelchair, his GCS has improved, he was alert and oriented, he was prescribed oral antibiotics, aspirin and rosuvastatin.

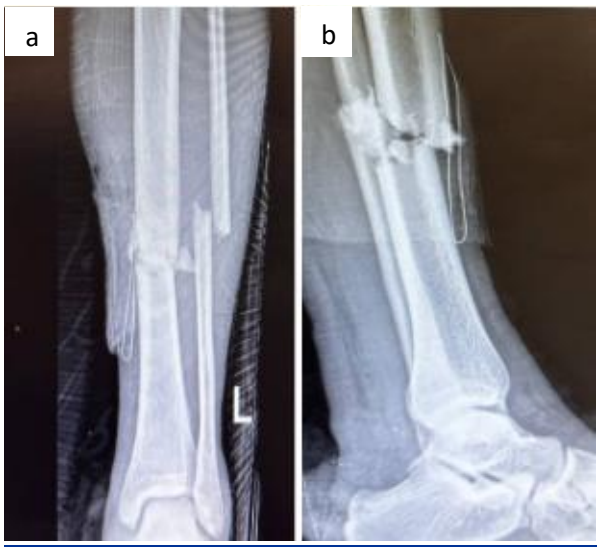

Figure 1 (a, b): Anteroposterior and lateral radiographs showing displaced open fracture of tibia and fibula.

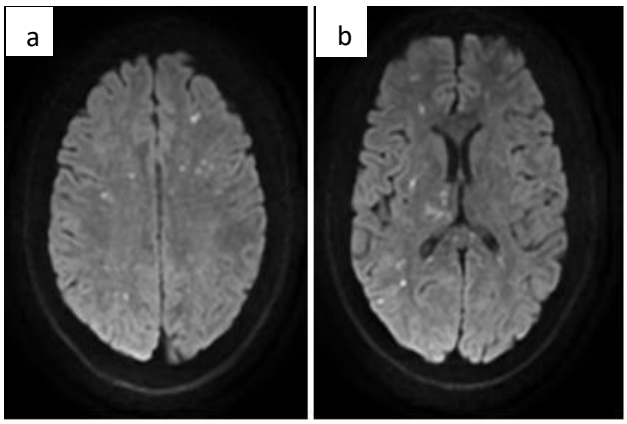

Figure 2 (a, b): Diffusion weighted magnetic resonance imaging with starfield pattern.

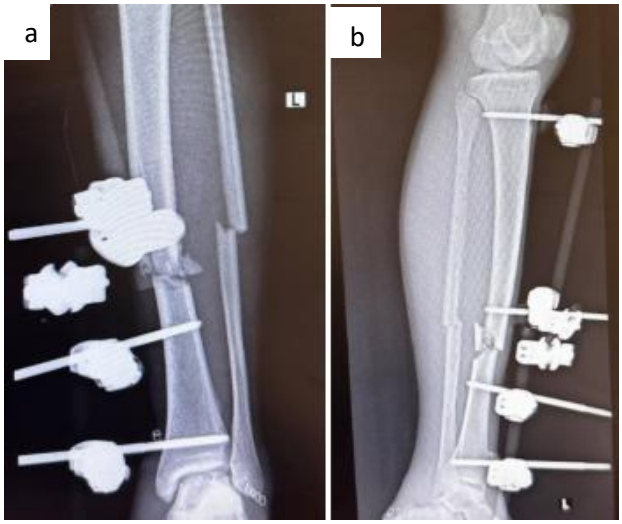

Figure 3 (a, b): Anteroposterior and lateral radiographs showing the external fixation of tibia.

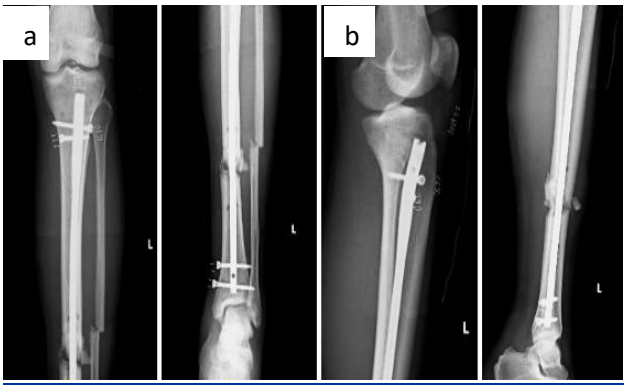

Figure 4 (a, b): Anteroposterior and lateral radiographs post intramedullary nailing.

DISCUSSION

FES is most commonly associated with orthopedic trauma, particularly following long bone fractures, the insertion of intramedullary devices, or procedures such as femoral stem cementing during total hip arthroplasty. It has also been reported, though less frequently, after spinal surgery.[5] The classic clinical trial of FES includes respiratory distress, neurological symptoms (such as altered mental status, seizures, or focal deficits), and petechial rash, typically appearing within 12 to 72 hours following the inciting event.[6]

The underlying pathophysiology involves the release of fat globules from the bone marrow into the venous circulation. These fat emboli are especially prone to occur during intramedullary fixation procedures, either during canal reaming or nail insertion. Once in circulation, the emboli commonly lodge in the pulmonary vasculature, resulting in hypoxia and respiratory symptoms. Some fat droplets may enter the systemic arterial circulation-either through pulmonary capillary bypass or right-to-left cardiac shunts such as a patent foramen ovale (PFO)-and travel to the brain or skin, causing neurologic and dermatologic manifestations.[6,7] Our case is particularly noteworthy for the development of isolated cerebral fat embolism (CFE) in the absence of both respiratory compromise and petechial rash. This is a rare and atypical presentation. In addition, what makes this case particularly unusual is that FES developed before any surgical intervention, a rare presentation since FES typically follows operative procedures such as intramedullary nailing or reaming. Because neurological symptoms may be subtle in such cases, the diagnosis can be easily missed unless cerebral fat embolism is considered early on. While the presence of a PFO has historically been suggested as a mechanism for cerebral fat embolization, more recent studies have found no consistent association between PFO and increased FES incidence.[7-9] In our patient, echocardiography revealed no structural cardiac abnormalities, further supporting the theory that pulmonary capillary bypass alone may permit systemic embolization.

The diagnostic process can be challenging due to the lack of a definitive diagnostic test. Imaging, particularly chest radiographs, is often nonspecific. However, MRI of the brain with diffusion-weighted imaging (DWI) can provide crucial diagnostic support. Our patient's MRI demonstrated a classic “star-field” pattern, consisting of multiple, small, punctate hyperintense lesions across both hemispheres-a hallmark finding in CFE.[6,7] Initial differentials included ischemic stroke, metabolic encephalopathy, and toxic ingestion. However, the clinical context of long bone trauma, combined with unremarkable toxicology and CT findings, shifted the diagnostic consideration toward cerebral fat embolism. This case emphasizes the importance of maintaining a high index of suspicion for FES in trauma patients, even when the presentation deviates from the classical triad.

Management remains primarily supportive, focusing on adequate oxygenation, hemodynamic stability, and neurological monitoring. Our patient was initially started on dual antiplatelet therapy out of concern for embolic stroke, but after confirming the diagnosis, treatment was de-escalated to single antiplatelet therapy with aspirin. Although systemic anticoagulation has been explored in some cases, no conclusive evidence supports its routine use in FES management.[6] Despite the initial severity, the patient showed gradual improvement in neurological status, supporting the notion that early identification and supportive care can lead to favorable outcomes even in cerebral fat embolism.

CONCLUSION

FES does not always follow a predictable pattern. In rare instances, as demonstrated in this scenario, patients may develop isolated neurological symptoms before undergoing any surgical procedure, and without any signs of respiratory distress or skin manifestations. Such presentations can easily be missed or mistaken for other

conditions, delaying appropriate care. Recognizing these atypical features is essential, especially in trauma patients with long bone fractures who experience sudden changes in mental status.

In this case, early brain MRI played a vital role in guiding the diagnosis. Importantly, systemic fat embolism may still occur even in the absence of a cardiac shunt, suggesting that fat particles can bypass the lungs through other pathways. This emphasizes the need for attentive monitoring and a broad differential diagnosis in trauma care, even when vital signs are stable and initial imaging unremarkable.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Khalil KS, Ibrahim SA, Yakout NA. Fat embolism syndrome before surgical treatment of open tibial and fibular shaft fracture: a case report. Int J Res Orthop 2025;11:914-7.