Case Report

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Radiology Section

'From Broken Bones to Broken Brain' – Unveiling Cerebral Fat Embolism in a Healthy 21-Year-Old Female

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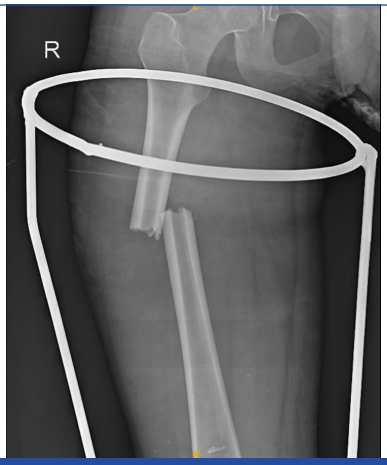
ABSTRACT

Cerebral Fat Embolism (CFE) is an uncommon but deadly complication that can occur following long bone fractures, particularly those of the femur. It is distinguished by a variety of neurological symptoms and may resemble other acute neurological disorders. Early detection along with therapeutic care are critical to better results. We present the example of a 21-year-old girl who suffered a catastrophic injury to her right thigh in a car accident. A first radiographic examination revealed a closed fracture of the right femoral shaft. Within 18 hours following the injury, the patient experienced altered mental state and respiratory discomfort. On diffusion-weighted imaging, brain Magnetic Resonance Imaging (MRI) revealed several hyperintense lesions, which were suggested to be CFE. Other possible explanations were ruled out by laboratory and radiological examinations. Supportive care, such as oxygen therapy and frequent monitoring, was implemented. The patient's neurological condition gradually improved over the next few days. CFE should be evaluated in individuals with long bone fractures who report with unexplained neurological decline. Prompt diagnosis by imaging and supportive treatment are critical for successful results. This example demonstrates the significance of clinical vigilance in trauma patients, especially those with femoral fractures.

Keywords: Diffusion-weighted MRI, Fat embolism syndrome, Femoral shaft fracture, Neurological complications, Supportive management, Trauma-related brain injury

CASE REPORT

A 21-year-old female patient arrived at our Emergency Department having a history of a road traffic collision that resulted in injury to her right thigh as well as other minor soft-tissue injuries, but no head trauma around 18 hours prior. She had a right-sided femur shaft fracture that was inadequately managed in a rural hospital two hours after the accident. Upon arrival at our facility 16 hours later, the patient had a confused mental state and haemodynamically unstable vitals, necessitating urgent resuscitation along with further supportive care. Her right thigh was re-examined and stabilised adequately in congruence with the orthopaedics team following a bedside X-ray confirming fracture [Table/Fig-1]. About two hours into therapy, the patient's Glasgow Coma Scale (GCS) (score of 8) declined, and she had two bouts of generalised tonic-clonic seizures, which led to her being intubated and placed on mechanical breathing.

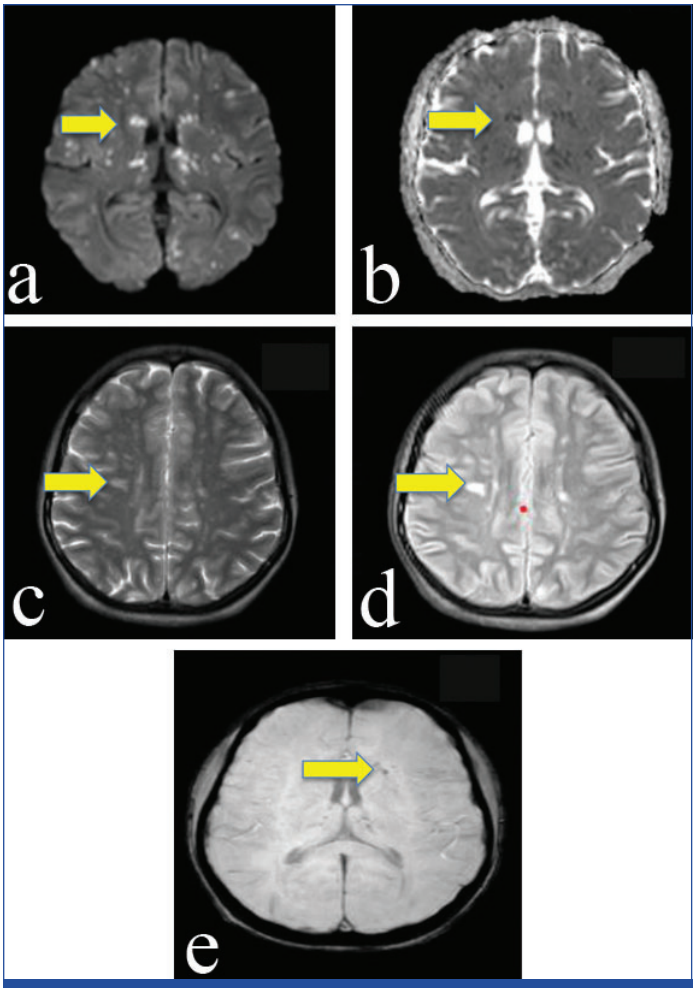


[Table/Fig-1]: X-ray showing right-sided femur shaft fracture with Thomas split in situ.

The CT scan of the brain showed no intracranial abnormality. Routine laboratory investigations, including Complete Blood Count (CBC), Liver Function Tests (LFTs), Kidney Function Tests (KFTs), and coagulation profile, were within normal limits. An echocardiography revealed normal ventricular function in the absence of thrombus. The bilateral lower limb vascular colour Doppler ultrasonography revealed no thrombus signs. MRI brain revealed multiple hyperintensities in T2/FLAIR sequences in bilateral cerebral hemispheres, corpus callosum, bilateral cerebellar hemisphere and pons and hyperintensity noted in Diffusion Weighted Imaging (DWI) sequence. It typically showed the "starfield" pattern in diffusion weighted sequences [Table/Fig-2]. The patient having CFE syndrome was identified after reviewing her usual clinical manifestations and imaging data. The patient was managed with i.v. methylprednisolone (1g/day for 5 days), subcutaneous enoxaparin (40 mg/day for 7 days), IV ceftriaxone (1g twice daily for 7 days), and 20% mannitol (100 mL i.v. thrice daily for 3 days) and admitted under Neurology department. She was extubated after 14 days, showing improvement in motor as well as cognitive abilities with daily physiotherapy. Her femur fracture was reduced and fixed following haemodynamic as well clinical improvement, and she was discharged after 35 days of hospitalisation with minimal residual cognitive deficit and no motor deformities.

DISCUSSION

The CFE is an uncommon, incomplete form of Fat Embolism Syndrome (FES) characterised by neurologic involvement without classic respiratory or dermatologic manifestations. It most often occurs 12-72 hours after trauma, typically following closed, long-bone or pelvic fractures [1]. CFE, because of femur or tibia fracture, is an uncommon occurrence, with a reported frequency of 0.9-2.2% [2] Typically, the presentation includes respiratory dysfunction, petechiae, and neurological symptoms. Neurologic symptoms can appear in a variety of ways, including disorientation, coma, and death. Symptoms can be temporary and may resemble a Transient Ischaemic Attack (TIA) [3]. CFE is classified as a deadly occurrence, with a documented death rate of 20%. While fat emboli may penetrate the microvasculature of the lungs, brain, skin, & kidneys, clinical FES appears in only a tiny percentage (0.1-11%) of individuals with long-bone fractures, most usually affecting the femur [4].



[Table/Fig-2]: MRI of the brain demonstrating multiple foci of abnormal signal involving the bilateral cerebral hemispheres, corpus callosum, bilateral cerebellar hemispheres, and pons. a) Diffusion-weighted imaging (DWI) shows hyperintense foci (arrow), indicative of restricted diffusion consistent with acute ischaemia; b) Corresponding Apparent Diffusion Coefficient (ADC) maps reveal low signal (arrow), confirming true diffusion restriction; c) T2-weighted images demonstrate hyperintense lesions (arrow), reflecting edema and tissue injury; d) FLAIR sequences highlight similar hyperintense foci with CSF suppression (arrow), enhancing lesion conspicuity; e) Susceptibility-Weighted Imaging (SWI) reveals blooming artifacts, suggestive of microhaemorrhagic transformation. These findings are consistent with acute embolic infarction.

The pathophysiology of FES comprises two basic mechanisms: a mechanical theory, in which fat from the wounded marrow penetrates the venous circulation as well as travels to the end organs, and a biochemical/inflammatory hypothesis, in which trauma-induced inflammatory responses cause fat to become more emboli. A CFE occurs when fat globules circumvent the pulmonary filter, commonly through a patent foramen ovale, resulting in multifocal brain damage [5]. CFE often manifests as altered awareness, seizures, as well as coma within 24-72 hours following trauma; however, it can occur earlier [6].

Long bone fractures (femur longer than the tibia), total replacement of the hip or knee surgery, liposuction, sickle cell crises, altitude sickness, burns, severe pancreatitis, and propofol injection are all risk factors [7,8]. The clinical manifestation of CFE ranged from 24-72 hours following orthopaedic injury, according to the literature [9].

The CFE causes a variety of neurological signs, the most common of which are acute encephalopathy and generalised symptoms. The patient's mental condition may deteriorate from fully aware to confused to comatose. If the basal ganglia is implicated, symptoms such as ataxia, aphasia, hemiplegia, dystonia, and even seizures might occur. Transient ischaemic episodes can serve as an essential differential diagnosis. Hypoxia can be the initial presentation in 96% of cases, progressing to respiratory failure in 10% [1].

CFE is a challenging clinical diagnosis due to its variable and non-specific symptoms, often accompanied by a normal Computed Tomography (CT) scan of the brain.

Schonfeld's Fat Embolism Index is used to aid in the diagnosis of fat embolism syndrome; a cumulative score of 5 or more points within the first three days of hospitalisation is considered indicative of the condition [10].

In our patient, the calculated score was 4 (confusion, fever >38°C, heart rate >120/min, respiratory rate >30/min). Despite a score below 5, the diagnosis of CFE was confirmed through characteristic clinical features and MRI findings, highlighting the unique presentation of this case.

CT scans have a greater probability of false negatives, which limits their diagnostic utility [9]. MRI is a very sensitive diagnostic tool that produces a hypointense signal on T1 and a hyperintense signal on T2 and diffusion weighted sequences. The "starfield" pattern found on diffusion weighted sequences is pathognomonic for cytotoxic oedema induced by numerous micro emboli [9]. To exclude out other disorders, the patient should have a chest X-ray, an echocardiography, and brain imaging. A chest X-ray may reveal nonspecific abnormalities, but an echocardiogram may reveal results related to right heart strain.

Diffuse axonal injury is another possible alternative diagnosis with a comparable neurological presentation and a history of a traffic collision. Unlike CFE, Diffuse Axonal Injury (DAI) shows normal CT and MRI brain findings and is characterised by an abrupt decrease in neurological status, which can lead to death [4].

A recent study by Vetrugno L et al., in 2021 included 268 cases. It was more common in the male gender (81.6% vs. 18.4%) [11]. The mean age of the participants was 33±18 years. The mean age of males (29±14 years) was significantly lower than that of females (51±26 years) ($p<0.001$). The femur was the most common fracture site (71% of cases). Patent foramen ovale was found in 12% of all cases.

In a similar case report by Nakada D et al., , Methylprednisolone (1 mg/kg/day) was initiated as treatment for Acute Respiratory Distress Syndrome (ARDS) associated with FES, resulting in gradual symptom improvement [12]. The methylprednisolone regimen was continued for 10 days. By day 7, the patient's level of consciousness gradually improved, and communication became possible. The patient was extubated on day 9 and began rehabilitation the following day, with an initial goal of non-weight-bearing ambulation using crutches. By day 16, the patient was able to ambulate with crutches, although weight-bearing remained restricted. However, residual higher-order cognitive dysfunction posed challenges during rehabilitation. On day 17, the patient was transferred to a rehabilitation hospital for further recovery.

CONCLUSION(S)

In the final analysis, CFE, albeit uncommon, should be explored in patients with long bone fractures who experience unexplained neurological deterioration. A high-index of suspicion, early MRI diagnosis, intense supportive therapy with high Positive End Expiratory Pressure (PEEP), and fast surgical repair of the fractures can all improve outcomes and lower the chance of long-term neurological sequelae or mortality.

REFERENCES

[1] Akoh CC, Schick C, Otero J, Karam M. Fat embolism syndrome after femur fracture fixation: A case report. Iowa Orthop J. 2014;34:55-62. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4127739/.
[2] Godoy DA, Di Napoli M, Rabinstein AA. Cerebral fat embolism: Recognition, complications, and prognosis. Neurocrit Care. 2018;29(3):358-65. Doi: 10.1007/s12028-017-0463-y.
[3] Kassimi M, Amriss O, Guerroum H, Habi J, Salimi Z, Mahi M. Cerebral fat embolism syndrome after long bone fracture: A case report. Radiol Case Rep. 2021;17(2):283-85. Doi: 10.1016/j.radcr.2021.10.060. Erratum in: Radiol Case Rep. 2023 Jan 25;18(4):1641-42. Doi: 10.1016/j.radcr.2023.01.013.
[4] Perron A, Canaple S, Ullmer A, Deramond H, Roussel M, Godefroy O. Cerebral fat embolism: An unusual case report and literature review. Interdiscip Neurosurg. 2021;25:101101. Doi: 10.1016/j.inat.2021.101101.

[5] Ippolito M, Benenati V, Gambino V, Lapi M, Nicosia D, Pace A, et al. Cerebral Fat embolism after traumatic long-bone fracture: A case report. Clin Case Rep. 2024;12(12):e9671. Doi: 10.1002/ccr3.9671.
[6] Kellogg RG, Fontes RBV, Lopes DK. Massive cerebral involvement in fat embolism syndrome and intracranial pressure management: Case report. J Neurosurg. 2013;119(5):1263-70. Available from: https://doi.org/10.3171/2013.7.JNS13363.
[7] Mijalski C, Lovett A, Mahajan R, Sundararajan S, Silverman S, Feske S. Cerebral fat embolism: A case of rapid-onset coma. Stroke. 2015;46(12):e251-e253. Doi: 10.1161/STROKEAHA.115.011440.
[8] Mellor A. Fat embolism. Anaesthesia. 2001;56(2):145-54. Doi: 10.1046/j.1365-2044.2001.01724.x.
[9] Wang Y, Si Z, Han J, Cao S. Imaging findings of cerebral fat embolism syndrome: A case report. J Int Med Res. 2020;48. Doi: 10.1177/0300060520950559.
[10] Schonfeld SA, Ploysongsang Y, DiLisio R, Crissman JD, Miller E, Hammerschmidt DE, et al. Fat embolism prophylaxis with corticosteroids. A prospective study in high-risk patients. Ann Intern Med. 1983;99(4):438-43. Doi: 10.7326/0003-4819-99-4-438. PMID: 6354030.
[11] Vetrugno L, Bignami E, Deana C, Bassi F, Vargas M, Orsaria M, et al. Cerebral fat embolism after traumatic bone fractures: A structured literature review and analysis of published case reports. Scand J Trauma Resusc Emerg Med. 2021;29(1):47. Doi: 10.1186/s13049-021-00861-x. PMID: 33712051; PMCID: PMC7953582.
[12] Nakada D, Hino H, Okuma N, Fujimoto Y, Miyashita M, Matsuura T, et al. Deterioration and manifestation of cerebral fat embolism triggered by bone reaming during femur fracture surgery: A case report. Clin Case Rep. 2025;13(8):e70806. Available from:https://doi.org/10.1002/ccr3.70806.

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