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THE RADIOLOGICAL MANIFESTATION OF FAT EMBOLISM SYNDROME: CASE STUDY

Radiology

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ABSTRACT

A clinical condition known as fat embolism (FE) or fat embolism syndrome (FES) is defined by the systemic dispersion of fat emboli throughout the circulatory system. Dissipation of fat emboli will cause a systemic inflammatory response syndrome by disturbing microcirculation and damaging the capillary bed. The diagnosis of cerebral fat embolism may take longer if there is cerebral involvement and no pulmonary or dermatological symptoms at the time of presentation. Not every patient with fat embolism syndrome will exhibit the classic triad of brain, the respiratory system, and dermatological symptoms. We diagnosed a case of cerebral fat embolism (CFE) in which the clinical symptoms did not meet to make the early clinical diagnosis. Because there is currently no gold standard for diagnosing fat embolism syndrome (FES), physicians continue to have difficulties in making this diagnosis.

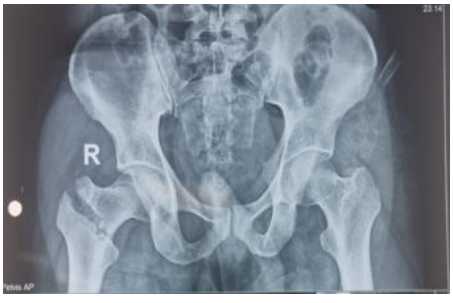
KEYWORDS

INTRODUCTION

Patients who have had orthopaedic surgery or who have broken long bones develop fat emboli. Clinical signs appear in just 3–10% of individuals with long-bone fractures. Not every patient with fat embolism syndrome will exhibit the classic triad of brain, the respiratory system, and dermatological symptoms. We examined a case of cerebral fat embolism in which the clinical symptoms met Gurd's criteria (1).

Case Report

A 28-year-old man alleged history of road traffic accident. He was given first aid in local primary health care centre and patient was oriented to time, place and person. Patient developed swelling in right leg and was unable to stand. X-Ray findings revealed intercondylar fracture of right femur.



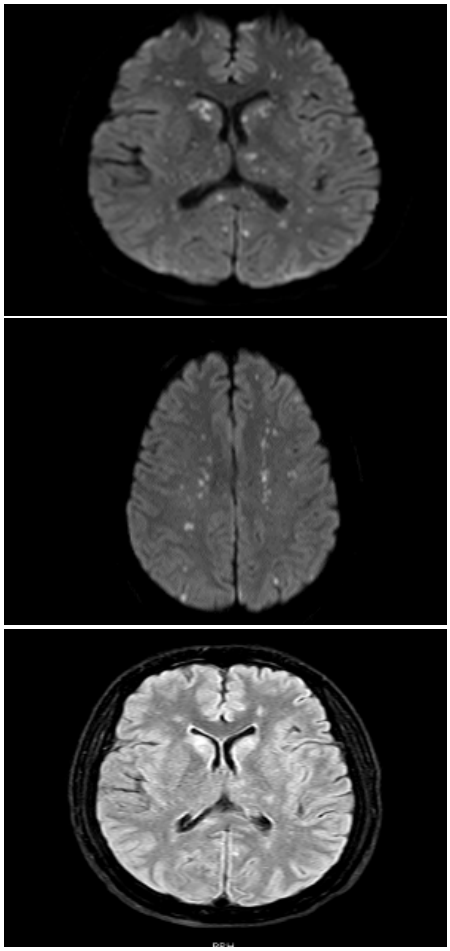
24 hours after injury patient developed altered sensorium which was sudden onset. Patient was referred to higher centre. Patient presented to National Institute of Medical Sciences & Research, Jaipur, Rajasthan with chief complaints of altered sensorium and fracture of right leg. The patient's Injury Severity Score (ISS) was 9 (fracture of femur). The critical values were: HR 105 bpm, BP 90/60 mmHg, and Spo2 78% at room air. An operation was scheduled and the broken limb was immobilised.

Patient was unresponsive, agitated, and confused. Despite receiving more oxygen, the patient had tachypnoea, tachycardia, and a drop in spO2. The endotracheal tube was used to secure the airway, and mechanical ventilation was started when the GCS deteriorated to 10 (E3 V2 M5). The examination of arterial blood gas revealed elevated hypoxemia. The chest radiograph showed no abnormalities. Echocardiography did not revealed any abnormality. Differential diagnoses included cerebral malaria, pulmonary embolism, acute viral encephalitis, and cerebral fat embolism.

On second day of admission, blood examinations indicated moderate anaemia (HB 9.3 gm/dl, platelet count of 2,80,000/mm3, and bilirubin of 1.1 mg/ dl). USG Doppler lower limbs revealed no signs of thrombus, Brain non-contrast CT scan results were normal.

On day third, CT pulmonary angiography ruled out pulmonary embolism. A negative HSV-PCR ruled out viral encephalitis, and the CSF biochemical screening was normal.

MRI was done which showed multiple punctate diffusion restriction foci noted in bilateral involving cerebral hemisphere in cortical/ sub cortical region, white matter, both caudate nucleus, centrum semi-ovale, thalami and few in both cerebellar hemispheres. Suggestive of micro fat-embolism. (k/c/o inter-condylar fracture of femur likely fat embolism). Gurd's criteria for the diagnosis of fat embolism syndrome are met.



On fifth day, D-dimer was 4.61(raised), urine for fat globules were absent, coagulation profile was dearranged. Patient was given 4 units of fresh frozen plasma and managed conservatively. Surgery was cancelled by the orthopaedician.

For the next three days, patient was managed with proper neurological and respiratory care with signs of improvement in the Glasgow coma scale and haemodynamic changes.

DISCUSSION

Fat embolism syndrome is a clinical condition that develops after pelvic and long bone fractures. In our case, there have been moderate injuries resulting in cerebral fat embolism symptoms (2).

Clinical criteria that are often applied, such as Gurd's criteria, might not always be fulfilled. This syndrome is an exclusion diagnosis since it can resemble a wide range of clinical diseases. There is a wide range in the appearance and intensity of signs and symptoms. Dementia, disorientation, altered awareness, convulsions, focal impairments, and coma are examples of neurological symptoms. The neurologic symptoms can be completely reversed. This illness is frequently misdiagnosed and deadly if medical intervention is put off. (3).

Currently, the most sensitive method for assessing cerebral fat embolism is magnetic resonance imaging. Diagnostic information is provided by the distinctive appearance of cerebral fat embolism on DWI sequence and FLAIR axial magnetic resonance imaging (4).

The distinguishing discovery is the existence of a "**starfield pattern**"—scattered brilliant specks on a black background—as shown in a study of Parizel PM, Demey HE et al (5). Our study also displayed a starfield design.

In a study carried out by Jacobson DM, Terrence CF et al has shown that MRI brain imaging is a good and sensitive diagnostic tool for fat embolism syndrome (3). The findings of a cerebral CT scan are negative or might indicate diffuse swelling. In the present case, the CT scan revealed no lesions.

Greater sensitivity is achieved using diffusion-weighted MRI, which displays characteristic numerous, nonconfluent, hyperdense lesions in deep grey matter and white matter along the main vascular regions' border zones.

CONCLUSION

Therefore, even in the absence of pulmonary and dermatological signs, early MRI (DWI and T2 weighted sequences) should be the first choice for patients exhibiting neurological symptoms following trauma. If MRI is performed early in the course of treatment, it can expedite the development of a diagnosis and save costs associated with further needless studies.

The most sensitive imaging method for identifying cerebral fat embolism appears to be MRI- DWI and T2-weighted imaging, which also shows good correlations with the clinical extent of brain injury. If pulmonary fat embolism is appropriately treated, cerebral fat embolism is a potentially curable illness with favourable prognoses.

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