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Cereberal Fat Embolism Syndrome - Case Report

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Abstract

Fat embolism syndrome (FES) is a life-threatening complication in patients with orthopaedic trauma, especially long bone fractures. The diagnosis of fat embolism is made by clinical features alone with no specific laboratory findings. FES has no specific treatment and requires supportive care, although it can be prevented by early fixation of bone fractures. Here we present our patient with cerebral FES secondary to femur and tibia fracture. Our case highlight the importance of a high index of clinical suspicion of FES in patients with long-bone fractures with neurological and pulmonary deterioration . The general recommendation is early surgical fixation to prevent additional emboli in patients with FES. cerebral FES has low mortality when managed with adequate supportive medical care.

Conclusion

Cerebral FES has low mortality when managed with adequate supportive medical care.

Keywords: Fat Embolism, Long bone fractures, Altered sensorium, Seizures, Hypoxia

Introduction

Fat embolism syndrome (FES) is a rare multisystem, clinical syndrome occurring in 0.9%–2.2% of long-bone fractures.[1] Fat globules released from the bone marrow enter the circulation, cause multiple occlusions at various sites. Patients typically present with signs and symptoms within 24–72 hours of the inciting event.[2] The major criteria for FES, as described by Gurd, are neurological impairment, respiratory insufficiency and petechial rash. The other minor diagnostic criteria include: fever, tachycardia, retinal abnormalities, thrombocytopenia and anaemia.[3] However, diagnosis of FES requires a high degree of clinical suspicion since its presentation can vary from subclinical with mild respiratory changes and haematological aberrations to a fulminant state characterised by sudden onset of severe respiratory and neurological impairment.[4] The respiratory involvement typically manifests as shortness of breath, tachypnoea, and hypoxaemia. The petechial rash associated with FES is non-palpable and commonly found in the axillae.[2] The neurological impairment from FES can present as headache, irritability, altered sensorium, stupor, seizures or even coma. The onset of neurological symptoms is often simultaneous with respiratory impairment.[2] Focal findings are less common and often difficult to identify in patients with trauma.[5] Imaging studies are the key in the diagnosis

of FES, especially in patients with neurological involvement. The initial head CT is almost always completely normal. MRI scan of the brain is most helpful in identifying FES. The FES-specific findings are punctate lesions within the white matter on T2-weighted images which are seen as early as 4 hours after onset of FES.[5] The pathognomic 'starfield pattern' can often be seen on MRI diffusion-weighted sequences.

Case Report

Here we present a young patient with cerebral FES secondary to long fracture of right lower limb involving shafts of femur and tibia. This patients exhibited profound neurological impairment, yet the patient didn't have a pulmonary or intracardiac shunt

A 31-year-old man with no significant medical history presented to our emergency department as a transfer from an outside hospital after involvement in a motorcycle versus vehicle collision. The patient was pillion rider. On examination in the ER, his vital signs were stable, and Glasgow Coma Scale (GCS) was 15. Pupils were equal, and reactive. On physical examination, the patient was found to have a right open midshaft femur and mid shaft tibia fracture which had been splinted. Imaging confirmed midshaft fractures of femur and tibia. There was no distal neuro vascular deficit. Initial CT brain had no acute intracranial abnormalities. He underwent wound toileting of the fracture sites in the ER OT and shifted to surgical ICU. He was planned for early fixation of the both the fractures. On day 2 of hospital stay, the patient was observed to have acute onset seizure-like activity. The patient had episodes of tonic stiffening of the arms and legs lasting several minutes each with an associated postictal state. Pupils at this time were equal and reactive. He was emergently intubated to secure airway as he didn't regain consciousness. A loading dose of levetiracetam was administered. Meanwhile a central line triple lumen 7Fr was secured in Rt IJV under ultrasound guidance and rt radial artery was cannulated for invasive blood pressure monitoring. His surgery was deferred for further neurological evaluation. In view of the clinical background of long bone fractures and drop in saturation a clinical diagnosis of fat embolism was considered. Immediate CT scan was negative for any acute pathology. Subsequent MRI of the brain showed findings consistent with fat emboli with multiple tiny acute infarcts in bilateral cerebral hemisphere predominantly in the subcortical white matter of bilateral inner watershed zones, bilateral basal ganglia, splenium of corpus callosum and bilateral cerebellum. In addition multiple petechial hemorrhages with SWI blooming noted in the white matter of bilateral cerebral hemispheres, corpus callosum, internal capsule and cerebellum. Diffuse cereberal edema with effacement of sulci. An electroencephalogram done was normal. His other investigations like chest xray was normal. A transthoracic echocardio

gram (TTE) with bubble study showed no evidence of intracardiac shunting. CT angiogram did not exhibit pulmonary shunting Oh.n Day 2 he underwent surgical fixations of both the fractures under general anaesthesia. His condition continued to be stable during intraoperative and postoperative period. In the subsequent days his GCS was E1VTM2, pupils equal and reactive. On Day 7 he underwent Tracheostomy for early weaning from mechanical ventilation. During the course of his stay in hospital his GCS gradually improved and was weaned from the ventilator. There was no residual neurological deficit. By Day 20, he was successfully decannulated. He was discharged in stable condition.

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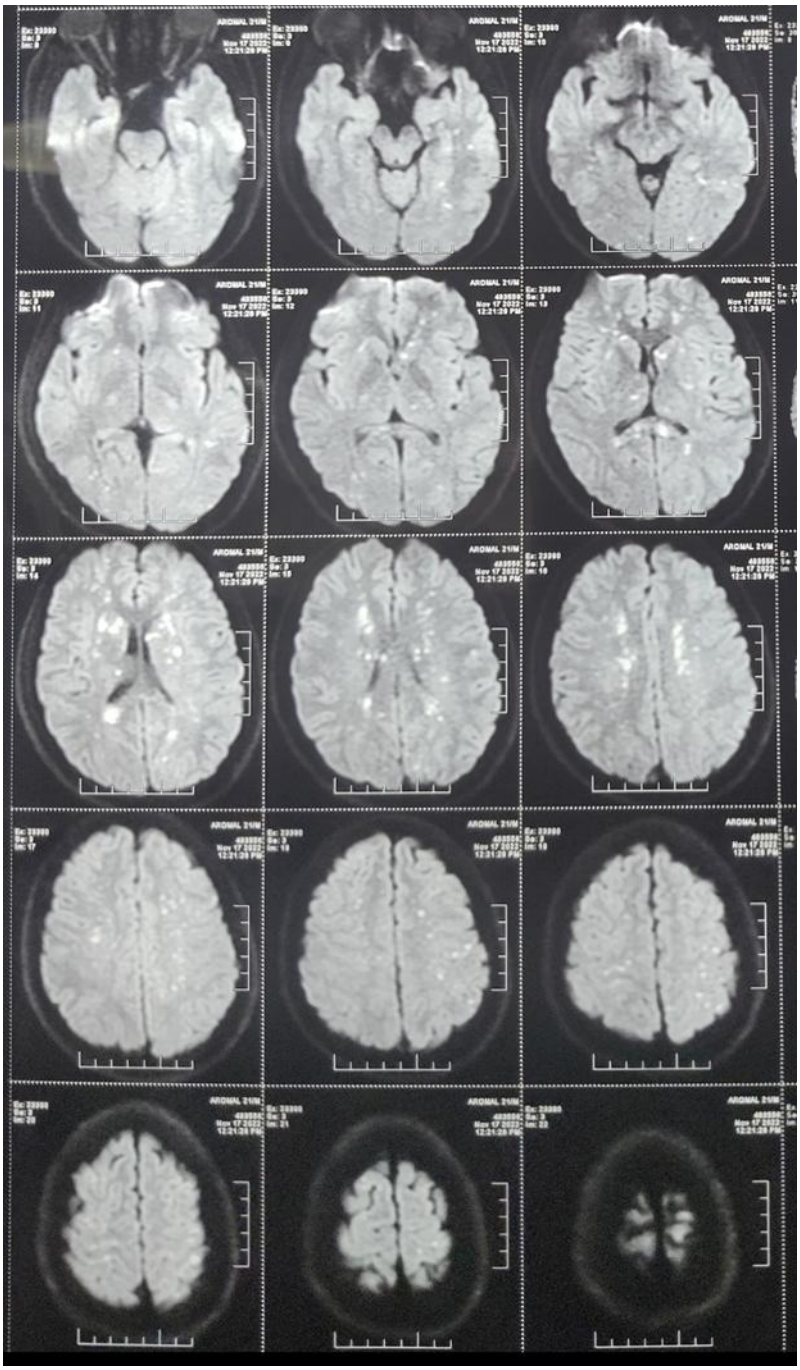

Table 1 Guard's criteria [8] (2 major criteria or 1 major criterion plus 2 minor criteria).

Major criteria	1. Petechial rash
	2. Respiratory symptom with radiographic change
	3. Central nervous system sign unrelated to trauma or other conditions
	Minor criteria 1. Tachycardia (HR 120 bpm)

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	2. Pyrexia (temperature > 39°C)
	3. Retinal change (fat or petechiae)
	4. Acute thrombocytopenia
	5. Acute decrease in hemoglobin
	6. High erythrocyte sedimentation rate (ESR)
	7. Fat globules in sputum

Table 2

Schonfeld's criteria [9] (total score > 5 required for diagnosis).

Criteria	Point
Petechiea	5
Chest X-ray change (diffuse alveolar change)	4
Hypoxemia (PaO_2 < 9.3 kPa)	3
Fever (temperature > 38°C)	1
Tachycardia (HR > 120 bpm)	1
Tachypnea (>30/min)	1
Confusion	1

Table 3 Lindeque's criteria [10].

1. Sustained PaO_2 < 8 kPa
2. Sustained $PaCO_2$ > 7.3 kPa or pH < 7.3
3. Sustained respiratory rate > 35/min despite sedation
4. Increase work of breathing, dyspnea, accessory muscle use, tachycardia, and anxiety

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Fig 1: Superior saggital sinus and right transverse sinus thrombosis

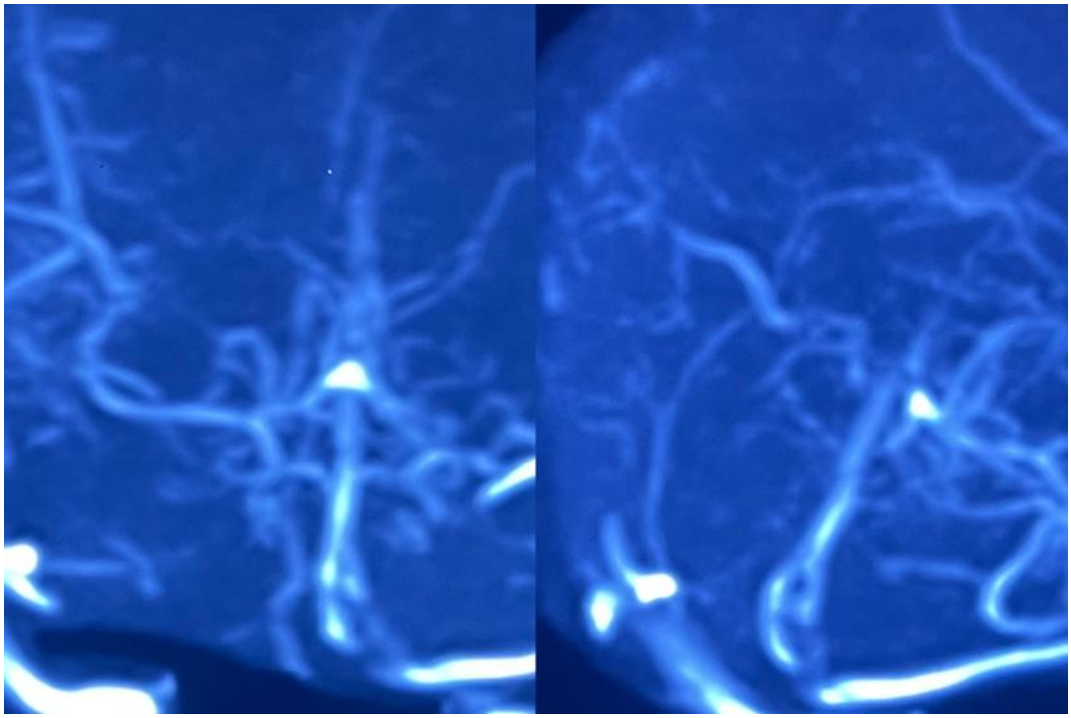
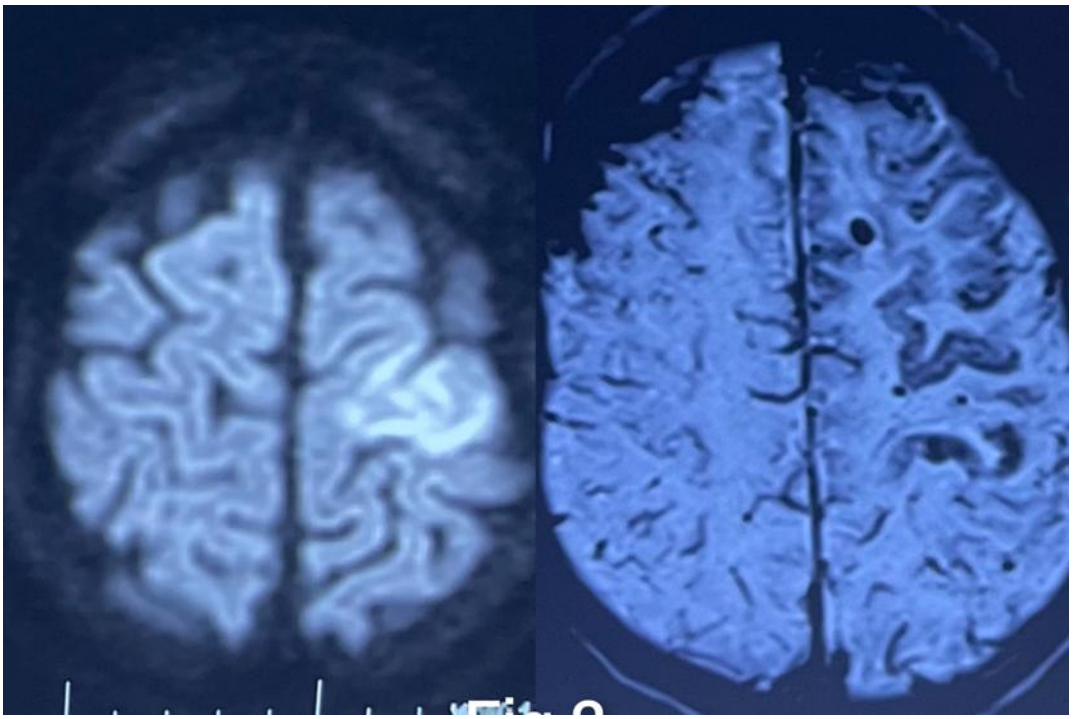


Fig 2: Hemorrhagic infarcts in frontal and parietal area



Discussion

The pathogenesis of cerebral FES is currently not clearly understood. There are few theories though controversial [10]. to explain the phenomenon. There are two main theories – Mechanical and Biochemical theory.

There are no universal criteria for diagnosis of FES. Diagnosis is made by clinical suspicion and

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characteristic findings on imaging methods.There are three previously proposed criteria by different authors: Gurd, Schonfeld, and Lindeque. Gurd's criteria is used more widely, and the diagnosis of FES requires at least two major criteria or one major criterion plus two minor criteria. In Lindeque's criteria, FES can be diagnosed using respiratory parameters alone.

Cerebral manifestations of FES are inconstant and not specific. They include headache, irritability, lethargy, stupor, delirium, convulsions, coma or subclinical events. The spectrum of neurological dysfunction seen in FES varies significantly from confusion to encephalopathy with coma and seizures.

Laboratory findings in FES are generally nonspecific. Some patients may develop thrombocytopenia, anemia, or hypofibrinogenemia. Cytological examination of the urine and sputum may show fat globules, but their diagnostic role still remains controversial. Roger et al. used bronchoalveolar lavage (BAL) to identify fat droplets in macrophages by oil red O stain.

Many imaging modalities can aid in the diagnosis of FES, but none is specific. Chest radiographic findings may show diffuse bilateral patchy infiltrates, consistent with acute respiratory distress syndrome. Chest films of some patients were normal The diagnosis based on imaging of cerebral fat embolism is challenging as the brain CT scans are mostly negative. MRI is more sensitive and consistently shows multiple small, scattered, hyperintense non-confluent intracerebral lesions on T2- weighted scans. Signal abnormalities can be noticed in both the gray and white matter. Lesions disappear gradually within weeks to a few months. However, T2-weighted MRI scans are useful in clinching the diagnosis in the (hyper)acute phase. Our case report showed MRI sensitivity in T2WI, DWI and FLAIR images. Transcranial Doppler ultrasound (TCD) can also be used for detecting microembolic signals (MES) in patients with FES. Silbert et al. reviewed 14 studies that used TCD for detecting microembolic signals during orthopedic surgery. Microembolic signals were detected in all 14 studies with prevalence ranging from 20% to 100% of patients. High-intensity transient signal counts were low (<10), but high counts were present in some patients [13)

The treatment of FES is mainly only supportive management. It includes maintenance of adequate oxygenation, hemodynamic stability, hydration, deep venous thrombosis prophylaxis, prevention of gastrointestinal bleeding, and nutrition. No specific drug is recommended or has strong evidence for treatment of FES. Systemic corticosteroids, which may reduce inflammation, perivascular hemorrhage, and edema, may have benefit in patients with worsening of lung functions, but there is no systematic evidence to back up such benefit. Heparin has been proposed as a treatment of FES, due to its stimulatory effect on lipase activity and clearance of lipid from the circulation. Nevertheless, an increase in free fatty acids in the circulation could exacerbate the underlying proinflammatory physiology. Systemic anticoagulation has also been considered as a potential for FES therapy, but in the setting of trauma and pre-existing hematologic abnormalities, anticoagulants may be harmful. Albumin has been recommended as it restores blood levels and combines the fatty acids that may limit lung injury. Early stabilization of long bone fracture is recommended to minimize bone marrow embolization into the venous system Surgical timing and techniques have important roles for preventing FES. Early surgical fixation within 24 hours after trauma has lower risk and severity than delayed fixation.

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Conclusion

FES diagnosis requires high index of suspicion as there are no pathognomonic signs. Treatment of FES is mainly supportive and use of corticosteroids and heparin remains controversial. MRI scan is very

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useful in clinching the diagnosis and must be done at the earliest.

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