CASE REPORT

Cerebral Fat Embolism: A diagnostic challenge

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ABSTRACT

Fat embolism syndrome (FES) is a rare but a serious clinical catastrophe occurring after traumatic injury to long bones. Cerebral involvement in the absence of pulmonary or dermatological manifestation on initial presentation may delay the diagnosis of cerebral fat embolism (CFE). We discuss a case series of CFE which posed a challenge in diagnosis. The clinical presentations of these patients did not satisfy the commonly used clinical criteria for aiding the diagnosis of FES. Early MRI brain (DWI and T2 weighted sequences) in patients with neurological symptoms after trauma even in the absence of pulmonary and dermatological findings should be the goal.

Key words: *Cerebral fat embolism, fat embolism syndrome, magnetic resonance imaging*

INTRODUCTION

Fat embolism syndrome is a rare but a serious clinical catastrophe occurring after traumatic injury to long bones. Although subclinical fat embolism develops in almost all the patients with fracture of long bones or in orthopaedic surgeries[1] but only 3–10% of long-bone fracture patients develops clinical manifestations of the syndrome.[2] Classical triad of cerebral, respiratory and cutaneous manifestations may not be present in all the patients of fat embolism syndrome (FES). We discuss a case series of cerebral fat embolism (CFE) wherein the clinical manifestations did not satisfy the Gurd's criterias[3] for aiding the diagnosis.

CASE SERIES

Case 1

A 27 year old male sustained an uncomplicated fracture of the left femoral lateral condyle with cruciate ligament avulsion. The patient was conscious, with no other associated injury and the Injury severity Score (ISS) was 9. The vital parameters were within normal limits. The fractured limb was immobilized and surgery was planned.

On the second day post injury patient developed high grade fever with altered mentation, restlessness and confusion. Acute viral encephalitis, pulmonary embolism, cerebral malaria and CFE were considered as differential diagnosis. Blood investigations showed haemoglobin of 7.9 gm/dl, platelet count of 100,000/mm^3, and biluribin of 2 mg/dl. USG Doppler lower limbs showed no evidence of thrombus and CT pulmonary angiography ruled out any embolic phenomenon. Non contrast CT brain was normal. CSF biochemical analysis was normal and viral encephalitis was ruled out by a negative HSV-PCR. Multiple negative peripheral smears for malarial parasite and no response to anti-malarials made cerebral malaria an unlikely diagnosis. The clinical features did not satisfy Gurd's criteria [Table 1] for diagnosing FES. On third day, the patient had tachypnoea, tachycardia and spO_2 decreased despite supplemental oxygen. The GCS worsened to 7 (E_2 V_2 M_3) for which the airway was secured with endotracheal tube and mechanical ventilation was initiated. Arterial blood gas analysis demonstrated hypoxemia and an increased alveolo-arterial gradient. Chest roentogram showed diffuse scattered infiltrates and ECG showed right ventricular strain pattern. Echocardiography ruled out atrial septal

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defect and patent foramen ovale. Magnetic resonance imaging (MRI) brain showed multiple, nonconfluent hyperintense lesions on T2 weighted images [Figure 1] and relatively low intensity on T1 images in bilateral cerebral hemispheres which confirmed the diagnosis of CFE.

The patient recovered uneventfully 20 days later. MRI was repeated and the changes regressed completely. He was discharged from hospital after full recovery.

Case 2

A 20 year old female sustained fracture midshaft left femur. She was referred twenty four hours later to our hospital. The left lower limb was stabilized in splint. The patient was conscious with no history suggestive of head injury. The initial respiratory and hemodynamic parameters were normal. The patient developed disorientation with aphasia about 48 hrs after admission. Within few hours her Glasgow coma score (GCS) decreased to 7 ($E_2M_4V_1$). The airway was secured with ETT and mechanical ventilation was initiated.On arrival to the intensive care unit marked pallor was observed with a pulse rate of 140/minute, blood pressure of 120/70 mm Hg and spO2 of 99%. Arterial blood gas and other investigations were within normal limits except haemoglobin of 6.7 gm/dl for which 2 units of red blood cells were transfused. Urine and sputum for fat globules was negative; fundoscopy was normal and there were no petechial or subconjuctival haemorrhages. Both chest X-ray and CT head showed no abnormality. There were no specific changes on electrocardiogram and transthoracic echocardiography ruled out septal defect.

On the third day, MRI of brain revealed white matter hyperintensities on T2 weighted images in bilateral centrum and semiovale, thalami, brainstem, cerebellum and corpus callosum with areas of restricted diffusion and multiple punctate haemorrhagic foci in bilateral periventricular white matter, internal capsule, corpus callosum, brainstem, and cerebellum. [Figures 2a-b and 3a-b] MRI was suggestive of CFE/ haemorrhagic acute demyelinating encephalomyelitis/ demyelinating disease. After neurology consultation patient was started on intravenous methyl prednisolone 1 gm/day administered for five days followed by oral prednisolone (40 mg/day) which was tapered in 21 days. Ventilation and other supportive treatment were continued.

After twelfth day of intubation, fixation of fracture was taken up under general anaesthesia followed by extubation two days thereafter. Follow-up MRI showed nearly complete resolution of the abnormal hyperintensity signal [Figure 4].

Case 3

A 28 year old male presented 18 hrs after sustaining multiple long bone fractures. He sustained fractures to his right femur, both bones of right lower limb and lateral malleolus. Patient arrived in emergency room with respiratory distress for which he was intubated and electively ventilated.

Patient had no history of loss of consciousness, vomiting or ENT bleed. His vital parameters revealed heart rate of 120 /min, blood pressure of 106/90 mm Hg , respiratory rate of 30/min and Glasgow coma scale(GCS) of 9 ($E_3M_5V_1$). Computed tomographic (CT) study of the head and CT angiography revealed normal study. Fat globules were not seen in either the urine or broncho alveolar lavage. Fundus examination revealed blurring of right disc margin and left papilloedema. MRI brain done on third day of ICU admission revealed multiple punctuate scattered nonconfluent (T2 flair hyperintense and T1 hypointense) lesions in bilateral cerebral hemispheres (predominantly in subcortical white matter), basal ganglia, thalamus, pons and cerebellum which are suggestive of CFE.

Table 1: Gurd's criteria: Diagnosis of FES need at least two major criteria or one major and four minor criteria to be present in order to diagnose FES

Major criteria	Petechial rash
	Respiratory insufficiency
	Cerebral involvement
Minor criteria	Tachycardia
	Fever
	Retinal changes
	Jaundice
	Renal signs
	Thrombocytopenia
	Anaemia
	High ESR
	Fat macroglobinemia

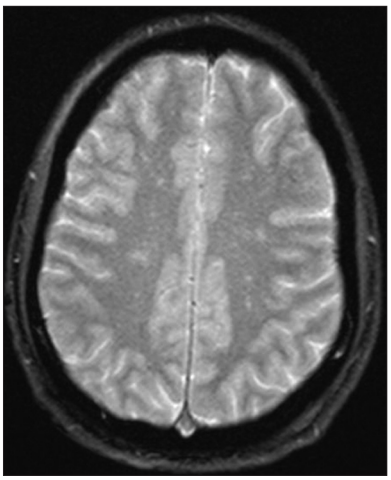

Figure 1: Axial T2-weighted MRI through cenrum semiovale showing multiple punctiform hyperintense lesions in white matter of both cerebral hemispheres

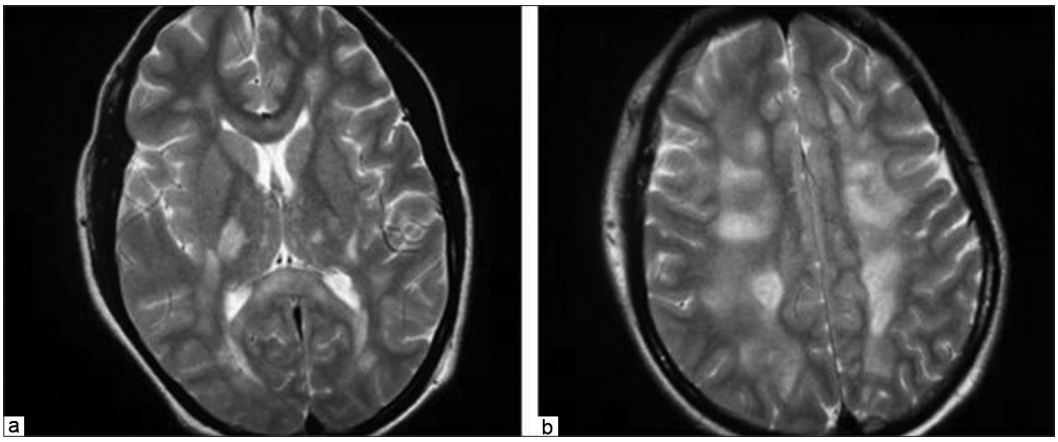

Figure 2: Axial T2 weighted images showing hyperintensities in corpus collosum, bilateral thalmi (a) and centrum semiovale (b)

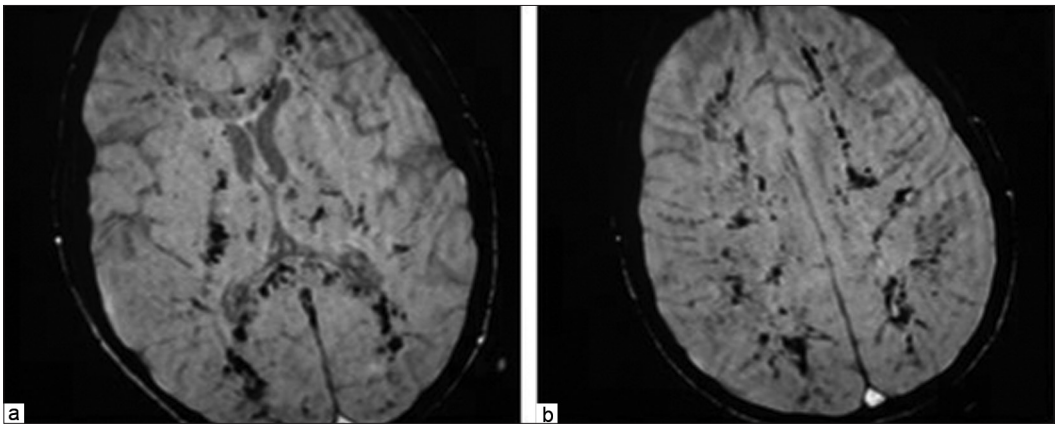

Figure 3: Axial gradient echo MRI showing multiple punctate haemorrhagic foci in corpus callosum, bilateral thalmi (a) central semiovale and grey-white matter junctions (b)

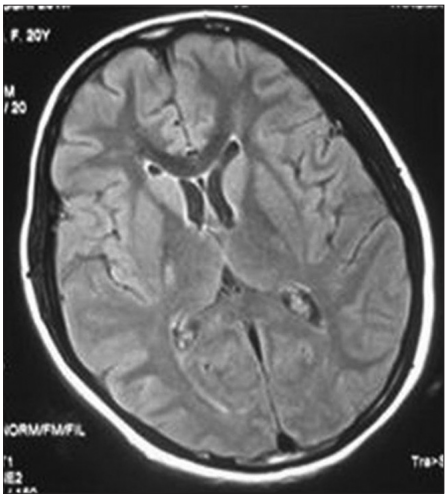

Figure 4: Follow up FLAIR MR image showing resolution of T2 hyper intensities in Splenium of corpus callosum and bilateral basal ganglia

Tracheostomy was done on third day of ICU admission in view of expected long duration of mechanical ventilation. On fourth day of ICU stay, patient had focal seizures and GCS decreased to 8. Phenytoin therapy was initiated for control of siezures. Supportive treatment was continued and early fracture stabilization was achieved once patient improved. Patient was weaned from ventilator and tracheostomy was decannulated 28 days post trauma. There was complete regression of lesions on MRI and the patients recovered completely.

Case 4

A 27 year male with history of motor vehicle accident was referred to our centre with polytrauma and head injury. He had multiple fractures of right humerus, femur and pubic rami. He had history of loss of consciousness, nasal bleed and his GCS on admission was 12 ($E_3V_4M_5$). CT head showed left parietal contusion and phenytoin therapy was initiated. Patient was shifted to ICU after adequate resuscitation and stabilization of fractures in cast. During the five days course of stay in ICU, GCS of patient improved to 15. He was shifted to ward in hemodynamic and neurological stable condition. In the ward 8 days after injury, the patient had respiratory distress with vital parameters of pulse 118/min, blood pressure of 108/60 and spO2 of 62%. Patient was intubated and shifted to ICU for further management. Chest x-ray showed bilateral lung haziness and ECG showed tachycardia and T wave inversions in inferior leads. Patient was evaluated on high

clinical suspicion of pulmonary thromboembolism/FES. Duplex scan lower leg veins did not reveal any thrombus. Pulmonary angiography being the gold standard was used to rule out pulmonary embolism. Axial T2-weighted MRI showing multiple punctiform hyperintense lesions in white matter of both cerebral hemispheres confirmed the diagnosis of CFE.

DISCUSSION

FES is a clinical complication which follows long bone and pelvic fractures. Though in case 1, the medullary cortex of the femur was intact, there have been reports of even minor trauma causing symptoms of CFE.[4]

The fat embolism syndrome (FES) still remains a diagnostic challenge for clinicians as there is no gold standard for the diagnosis of FES. The commonly used clinical criteria like Gurd's criteria[3] [Table 1] may not be satisfied in all the cases as in all our four cases. FES may mimic widely variable clinical conditions and hence it is a diagnosis of exclusion. Signs and symptoms may vary greatly in its presentation and severity. Neurological symptoms are variable and nonspecific, and the clinical diagnosis is difficult solely based on it. Neurologic symptoms consist of dementia, confusional state, disturbed consciousness, convulsion, focal deficits and coma.[5] In the majority of patients, neurologic abnormalities often develop after the development of respiratory distress.[6] But this may not be always true as in case 1 and case 2.

Although the neurologic findings are transient and fully reversible in most cases, the condition is often misdiagnosed and fatal if the treatment is delayed. The early diagnosis not only prevents morbidity and mortality but also reduces cost burdens of additional investigations for correct diagnosis. Presently MR imaging is the most sensitive technique to evaluate cerebral fat embolism. The characteristic appearance of cerebral fat embolism on flair axial magnetic resonance images is diagnostic.[7]

Reviewing literature till date, only a few reports have stressed the usefulness of MRI brain as a valuable and sensitive indicator in the diagnosis of FES. Cerebral CT scan results are usually negative or may show diffuse edema with low density area and haemorrhage in white matter.[5] In all our four cases there were no lesions demonstrated by CT scan. Clinical symptoms and CT are not always diagnostic while MRI is more sensitive imaging modality for diagnosing CFE.[8]

Diffusion-weighted MRI is more sensitive and can hasten the establishment of diagnosis in such cases. There is limited data on MRI findings in patients with FES. MRI usually shows typical multiple, nonconfluent, hyperdense lesions in white matter and deep gray matter along the boundary zones of major vascular territories[9] as in all four cases. Area of distribution is centrum semiovale, subcortical white matter, matter, ganglionic regions and in thalami. Presence of a "starfield pattern" of scattered bright spots on a dark background is characteristic.[10,11] MRI of the brain may reveal high intensity T2 signal and relative low intensity on T1 weighted images.

Table 2: Takahashi grading according to size and distribution of brain lesions on T2 MRI images

Grade	MRI Finding
Grade 0	No abnormality
Grade 1	Several small spotty high-intensity lesions seen in the deep white matter or deep brain structures
Grade 2	Either many small spotty high intensity lesions or macular lesions which represented confluent spotty lesions in the deep white matter or deep brain structures
Grade 3	Large macular high-intensity lesions in the deep white matter

It has been suggested that diffuse hyperintense areas on diffusion weighted images reflect immediate cytotoxic edema secondary to ischemic occlusion of cerebral arterioles, whereas hyperintense lesions on T2- weighted images reveal vasogenic edema developing at a later stage.[12] The high intensity T2 signals appear as early as 4 hrs after the onset of CFE; fuse and enlarge with time and diminish within 2 weeks in cases with good outcome.[13] According to size and distribution of brain lesions on T2 MRI images, Takahashi[6] graded these changes from grade 0 to grade 3 [Table 2] which correlated with GCS at onset of CFE and degree of clinical neurological impairment.

Incomplete FES in the form of CFE may be confounded with other neurological processes and is particularly challenging. Thus early MRI (DWI and T2 weighted sequences) in patients with neurological symptoms post trauma even in the absence of pulmonary and dermatological findings should be the goal. MRI if done in the earlier course of treatment can hasten the establishment of diagnosis and prevent expenses of other unnecessary investigations.

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