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Cerebral fat embolism: A case report

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Abstract
FES is a multi-systemic disorder, and its pathogenesis remains unclear to date, though well accepted-mechanical and biochemical mechanism maybe explained. Imaging plays a major role in confirming the diagnosis. Because of its uncommon presentation-here we are reporting a case of a young male with multiple long bone fractures. He was admitted to our Intensive Care Unit, was diagnosed with cerebral fat embolism syndrome (FES) based on clinical suspicion and imaging features. CT brain was normal. Due to strong clinical suspicion MRI was done. Fat embolism was confirmed by typical imaging features on magnetic resonance imaging (MRI) The patient was ventilated and managed appropriately. Surgery was done for the fractures and discharged.

Keywords: Cerebral fat embolism, magnetic resonance imaging, CT brain

Introduction
Fat embolism syndrome (FES) is a serious complication of fracture of the long bones. Though the exact incidence of the syndrome is difficult to estimate as many subclinical forms remain unrecognised, the reported incidence varies from 0.5% to 0.9% [1]. in orthopedic trauma with higher rates in multiple long bone fractures. The mortality rates range from 5% to 15%; however with early diagnosis and appropriate management, a great majority of patients recover. Patients may present with pulmonary, neurological, or dermatological symptoms 24–72 h after initial injury. Cerebral involvement is seen in up to 80% of cases of FES and generally worsens its prognosis. Often respiratory symptoms precede neurological ones, but there are case reports of isolated cerebral involvement. Neurological findings may vary from drowsiness to acute confusional state and seizures, decorticate posturing and focal deficits including hemiplegia, aphasia, apraxia, and visual field defects are also seen [2]. We are here reporting a case of a young male who presented with symptoms of severe CNS manifestations after few hours of trauma with multiple long-bone fractures and diagnosed as FES by neuroimaging studies.

Case Report
A 17yrs old construction worker was brought to our hospital's emergency room with multiple fractures in the lower limbs (closed right segmental fracture femur, Grade 2 right distal tibia and fibula fracture, Grade 1 displaced proximal phalanx fracture of 4h and 5th toe, closed displaced left distal radius fracture after a high-speed collision with a car on the highway, no head injury, loss of conciousness, convulsions and vomiting.

No distal neuro vascular deficits were found No history of hypertension, diabetes mellitus, tuberculosis, bronchial asthma, thyroid disease or epilepsy. On examination patient is conscious, coherent and afebrile BP:100/60mm Hg, pulse: 110b/min, respiratory rate: 60breaths per minute,SpO2 85% at room air. Bilateral air entry present, cardiovascular system, S1 and S2 normal, per addomen: soft non tender.No spine tenderness, chest compression test negative, pelvic compression and distraction test negative, bitrochanteric compression test positive on right side. No distal neurovascular deficits Initial treatment was given with IV fluids, antibiotics, analgesics, POP slab, skin traction, O2 support. CT brain done was normal. On same day evening patient complained of breathlessness and saturation started decreasing, was started on non-invasive ventilation, next day morning patient saturation started further decreasing and GCS (E2V1M3 6/15)started decreasing patient was put on invasive ventilation, urine fat globules was negative, and fundus was normal. In view of low GCS MRI brain was advised. MRI brain f/s/o fat embolism, prognosis was explained to attenders and symptomatic treatment, Hydrocortisone, mannitol was started. Surgery was done, symptomatic treatment given and patient discharged after becoming stable. CT scan brain showed Normal study MRI brain plain showed Multiple discrete T1 hypo-intense and T2/FLAIR hyper-intensities in bilateral cerebral and cerebellar hemispheres and bilateral capsuloganglionic regions with restricted diffusion s/o fat embolism, Takahashi grading –Grade 2 with diffuse cerebral oedema. HRCT chest showed multiple ground glass opacities in both the lungs

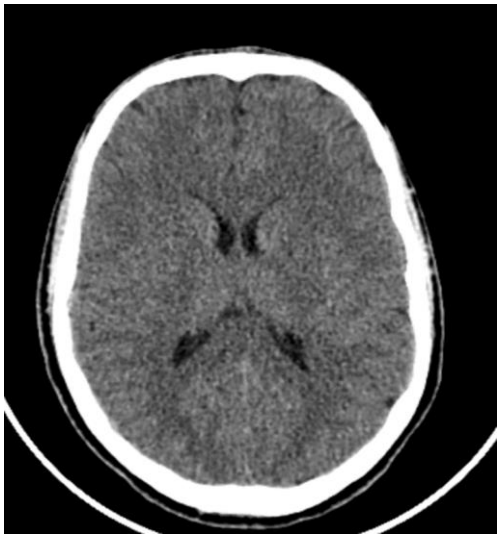

Fig 1: CT Brain Axial

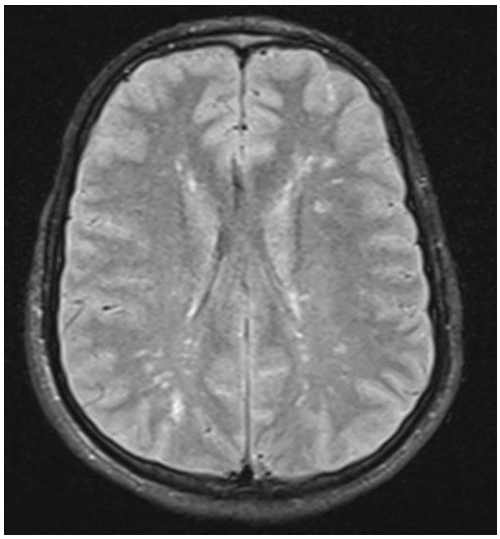

Fig 2: FLAIR

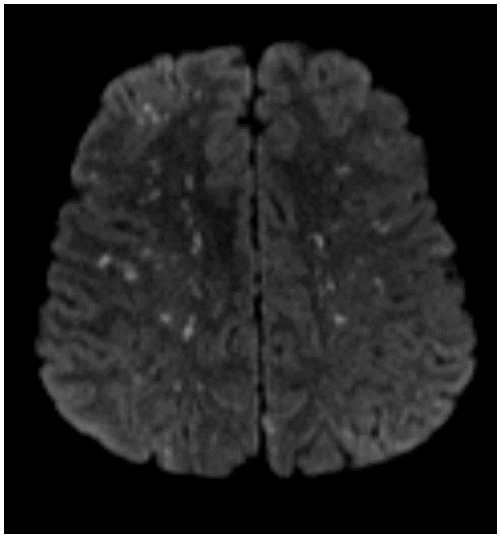

Fig 3: DWI

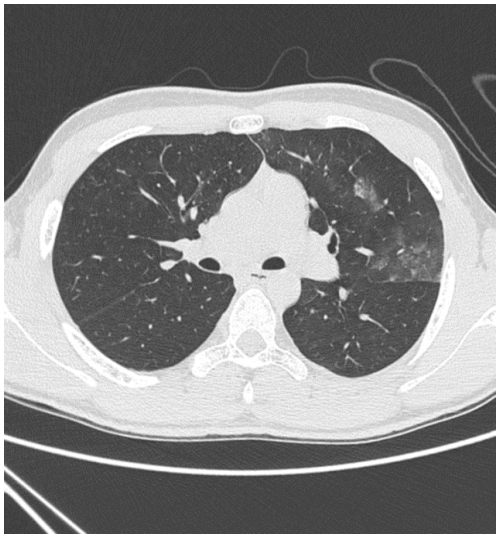

Fig 4: HRCT Chest showing Ground glass opacities in both lungs

Discussion

The fat embolism syndrome was first described as a clinical entity by E Von Bergmann in 1873. It is thought to have been clinically described as a post-mortem finding by Zenker in 1862 [3].

The diagnosis of FES is an exclusion and depends mainly on clinic manifestations, which is characterized by hypoxemia, neurological impairment, and petechial rashes, Guard's criteria [4, 5]. In Lindeque's criteria, FES can be diagnosed using respiratory parameters alone [6].

Cerebral fat embolism typically occurs in patients with bony fractures (usually long bones of the lower limb) or as part of a sickle cell crisis with bone marrow fat necrosis and subsequent embolism [7]. The neurological abnormality in cerebral FES occurs from two mechanisms, the occlusive effect of embolized fat globules in the cerebral arterioles and the cytotoxic effect of released free fatty acids causing increase in capillary permeability. These lead to cerebral microinfarcts with edema and microbleed; these lesions are particularly distributed over the watershed territory of the brain. In most cases, the acute microinfarcts and cytotoxic edema are reversible as the emboli are of very small size. However, in some cases of severe cerebral FES, sequelae may occur from persistent ischemia leading to chronic infarcts and neurological deficits. Though diagnosis of cerebral FES is a clinical one, neuroimaging with MRI is a sensitive and useful tool for confirming it and predicting outcome. The CT brain can be normal in most cases [8]. The lesions are seen as early as 4 h after neurological deterioration. On T2WI, multiple, diffusely scattered foci of hyperintensities are seen in the deep white matter, basal ganglia, brain stem and cerebellum. These represent microinfarcts in a penumbra of micro-hemorrhages and cerebral edema. In addition, the fluid-attenuated inversion recovery sequence is useful in detecting these lesions in their acute stage and revealing their watershed distribution. On diffusion-weighted imaging (DWI), the areas of cytotoxic edema appear as a "starfield," i.e., multiple scattered white spots against dark background. In addition, hyperintensities On DWI indicate low diffusivity, appearing as hypointensities on apparent diffusion coefficient map [9, 10].

- Based on the size on T2WI, the lesions can be graded from 1 to 3 as follows: Grade 1 = small spotty hyperintensities, grade 2 = several small spotty or macular hyperintensities, grade 3 = large macular hyperintensities. Takahashi *et al*. showed that the maximum grade of lesion on MRI during the course of cerebral FES correlated with patient's GCS at the time of presentation [11] However, the initial MRI done soon after onset of cerebral FES may not reveal the true severity of neurological insult as the cerebral lesions take time to develop; hence, serial scans are useful.
- Based on clinical features and initial MRI, the patient was diagnosed with cerebral FES, and supportive care therapy started. The MRI showed grade 2 lesions corresponding with patient's initial low GCS and severe cerebral insult. In most cases of cerebral FES, recovery is seen within 4–6 weeks; however, there are a few reports of prolonged coma and delayed recovery up to 2 months. In our case, the patient response was poor to supportive intensive care therapy initially. A high degree of suspicion for cerebral FES is warranted in patients of orthopedic trauma without head injury who develop altered sensorium. Early recognition of cerebral FES is important for appropriate medical management and to avoid adverse outcomes and improve prognosis. Immobilization of the fracture site, maintenance of intravascular volume, hemodynamic, and ventilatory support are the main elements of treatment. Care should be taken to prevent secondary brain injury due to hypoxia, hypotension, or seizures.

A differential to consider for the starfield pattern on MRI includes many other causes of multiple small foci of infarction or haemorrhage, only fat emboli will result in the very large number of tiny lesions characteristic of a starfield appearance. Other diagnoses like diffuse axonal injury, cerebral vasculitis, minute hemorrhagic cerebral metastasis, septic emboli may considered to consider [12]. Good supportive care during patient recovery can decrease mortality rate to less than 10%. Dermatologic, neurological, and respiratory manifestation generally resolve without consequences [13].

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Conflicts of interest
There are no conflicts of interest.

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