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A CASE OF CEREBRAL FAT EMBOLISM

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ABSTRACT

Fat embolism syndrome is a serious clinical event occurring following long bone fractures. Although fat embolism syndrome is a triad of hypoxemia, neurologic abnormalities and petechial rashes, isolated cerebral involvement can occur which is rare. Hence, cerebral fat embolism should be suspected in patients presenting with neurologic symptoms even without respiratory and dermatological manifestations. Here, we present a case of cerebral fat embolism which did not have pulmonary or dermatological manifestations.

KEYWORDS: Cerebral fat embolism, fat embolism syndrome, MRI.

INTRODUCTION

Fat embolism syndrome is a rare but serious clinical event occurring following long bone fractures. The classic triad consists of neurologic, respiratory and dermatological manifestations, thought it need not occur in all. Fat embolism syndrome occurs in 3-10% of long bone fractures. Though the diagnosis is clinical, an early MRI brain which is the most sensitive technique for evaluation of cerebral fat embolism will aid in the diagnosis.

CASE REPORT

24-year-old male presented to our hospital with fracture shaft of left femur (Figure 1). The patient was conscious and oriented with no history of vomiting, seizures or ENT bleed. The left lower limb was stabilized with splint and surgery was planned. After 24 hours, the patient developed altered mental status and abnormal posturing of both upper limbs. Blood investigations showed hemoglobin of 14.7g/dl, platelet count of 286000/mm^3 and D-dimer of 2006 mg/ml. Non-contrast CT brain was normal. CT pulmonary angiography ruled out any embolic phenomenon. Echocardiography did not reveal any significant finding. MRI of brain showed multiple tiny non-confluent T2 and T2 FLAIR hyperintensities (Figure 2) involving bilateral cerebral hemispheres which showed diffusion restriction (Figure 3a and 3b), consistent with cerebral fat embolism. The patient was given supportive treatment with IV fluids and oxygen. On the 6th day, his sensorium improved. On the 9th day, open reduction and internal fixation of fracture was done. He was discharged from hospital after full recovery.

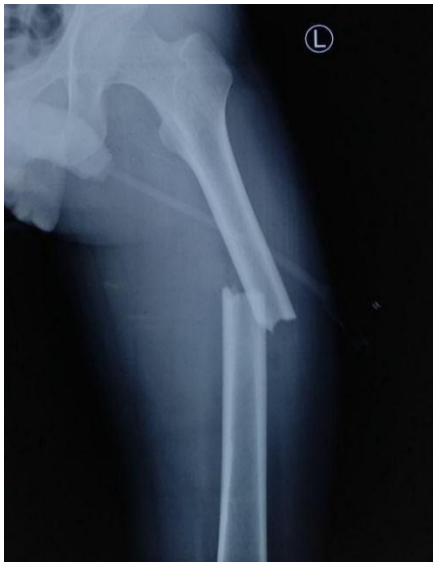

Figure 1: Displaced fracture involving shaft of femur.

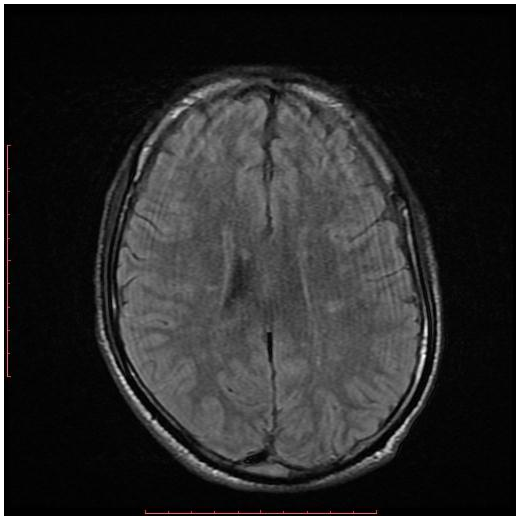

Figure 2: AXIAL T2 FLAIR MRI showing hyperintense signals involving bilateral corona radiata.

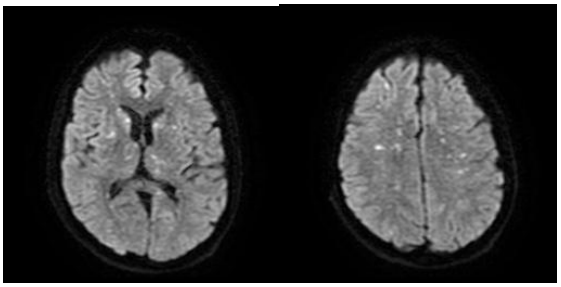

Figure 3a **Figure 3b**

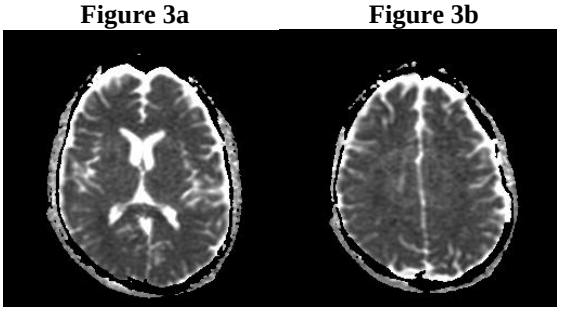

Figure 3c **Figure 3d**

Figure 3a and 3b: DWI showing high signal intensity areas involving bilateral centrum semi ovale, caudate nuclei, lentiform nuclei and thalami. Figure 3c and 3d - all showing corresponding fall in ADC.

DISCUSSION

Fat embolism syndrome is the triad of hypoxemia, neurologic abnormalities and petechial rashes.[1,2] It usually occurs in adults than children since bone marrow of children contain more hematopoietic tissue and less fat.[2] Acute hypoxia is the most common and usually the earliest manifestation of fat embolism syndrome which can progress to respiratory failure. Neurologic manifestations include headache, irritability, delirium, seizures and coma; almost all symptoms are fully reversible. Petechial rashes occur on chest, axilla and subconjunctiva which too resolve quickly. Fat embolism syndrome is a clinical diagnosis and a diagnosis of exclusion.[3,4] There is no gold standard for the diagnosis of fat embolism syndrome. Gurd's criteria has been described for the diagnosis of fat embolism syndrome.[1]

Though subclinical fat embolism occurs in almost all cases of fractures of long bones or orthopedic procedures, symptoms occur in only 3-10% of long bone fractures.[1] Non-traumatic causes of fat embolism include pancreatitis, sickle cell disease, severe infection, coronary artery bypass graft, alcoholic liver disease, renal transplantation, parenteral lipid infusion, orthopedic procedures liposuction and decompression sickness.[3] Two theories have been put forward to explain the origin of fat droplets: mechanical and biochemical theory.[3] According to mechanical theory, trauma to long bone leads to mobilization of fat globules which enter the circulation through damaged venules at the fracture site. Once they reach pulmonary vasculature, they cause local ischemia and inflammation. Fat globules reach arterial circulation though a patent foramen ovale or by micro emboli that pass through the lungs. Biochemical theory states that the release of catecholamines during trauma mobilizes free fat acids from fat stores or the acute phase reactants affect the solubility of fat causing embolization.

Usually there's a latent period of 12-72h after trauma before the manifestation of symptoms.[3] Management includes immobilization of fracture site, supportive treatment like hemodynamic and ventilatory support and prevention of secondary brain injury due to hypoxia and hypotension.[4]

Neurologic abnormality in fat embolism syndrome develops due to occlusion of cerebral arterioles by embolized fat globules and breach in blood brain barrier by chemical effect of free fatty acids which lead to cerebral microinfarcts, edema and microbleeds.[4] They are fully reversible due to their very small size. However, neurologic sequelae can occur in severe cases of cerebral fat embolism.

MRI is the most sensitive technique for the evaluation of cerebral fat embolism. Diffusion weighted images show the presence of "star field" pattern.[1] However, susceptibility weighted images are more sensitive and specific for the detection of cerebral fat embolism.[5]

Diffuse axonal injury is the close differential diagnosis for cerebral fat embolism. However, neurologic symptoms and loss of consciousness occur immediately after injury.

CONCLUSION

Though the diagnosis of fat embolism syndrome is mainly clinical, an early MRI brain will aid in proper diagnosis of cerebral fat embolism. Management of the patient includes mainly supportive treatment. Most patients recover fully and uneventfully.

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