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Cerebral Fat Embolism Following Orthopedic Surgery (CaseReport).

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Abstract

Fat embolism syndrome (FES) is a rare but a serious clinical condition occurring after traumatic injury to long bones. Cerebral involvement in the absence of pulmonary or dermatological manifestation on early presentation may delay the rapid diagnosis of cerebral fat embolism (CFE). We discuss a case of CFE with a challenge in diagnosis with absence of acute chest pain. The clinical presentations of these patients did not satisfy the clinical criteria the diagnosis of FES. Early MRI with neurological symptoms after trauma even in the absence of pulmonaryand dermatological findings should be the goal.

Key Words: *Fat embolism syndrome, trauma.*

I. Introduction

Fat embolism syndrome (FES) is a change in physiology resulting from mechanical causes, trauma, sepsis, or orthopedic surgery. Fat globules generated within the systemic circulation induce pulmonary dysfunction, neurological changes, dermal symptoms, and dysfunction of several other organs. Although very rare, FES is a fataldisease that develops that develops within 12–72 hours [1, 2].

The characteristic triad of respiratory failure, petechial rash, and neurological manifestations is simultaneously present in less than half al all cases. Although CFE (cerebral fat embolism) is a clinical diagnosis, the clinical criteria lack specificity, so the role of magnetic resonance imaging (MRI) is preponderant for the diagnosis, especially diffusion-weighted imaging (DWI) sequences and susceptibility-weighted imaging (SWI) which is knownto be very sensitive to blood products [3,4].

We report an unusual case of cognitive impairment following a motor car accident. The case with multiple bilateral femoral fractures. After internal fixation, the patient condition was deteriorated and MRI brain helping the diagnosis.

II. Case report

A 25-year-old man was admitted to our institute after motor car accident. On arrival, he was alert, with external evidence of head trauma and no signs of neurological deficit.

The vital parameters were within normal limits. The fractured limb was immobilized and surgery was planned. After2 days bilateral internal fixation was done [Fig 1].

A few hours after surgery, the patient suddenly developed confusion followed by a rapid decline in consciousness, with a GCS score of 10 (and fever (38°). There was no sign of respiratory involvement.

Acute viral encephalitis, pulmonary embolism and traumatic brain injury were considered a differential diagnosis. USG Doppler lower limbs showed no evidence of thrombus and CT pulmonary angiography ruled out any embolic phenomenon. Non contrast CT brain was done that elicited bilateral subcortical low attenuation densities.

The patient was requested to MRI of the brain to rule out traumatic brain injury.

The brain MRI on the 2nd day showed multiple small, scattered hyperintense lesions on DWI and T2 Weighted images in the subcortical white matter and cerebellum, with corresponding lower apparent diffusion coefficient (ADC)-values[Fig 2] . Also drop of signal at susceptibility weighted images[Fig 3] .

No apparent abnormalities were detected in MRA.

The clinical examination on the third day revealed petechiae on the upper thorax and neck. Combined with the patient's typical clinical manifestations, laboratory tests, and imaging studies, we diagnosed the patient with cerebralfat embolism syndrome.

The patient was given neurotrophic, anti-infection, and symptomatic support treatment, and was then gradually weaned from the ventilator and steadily recovered consciousness. After 3 months of follow-up, the patients' neurological symptoms and signs had disappeared.

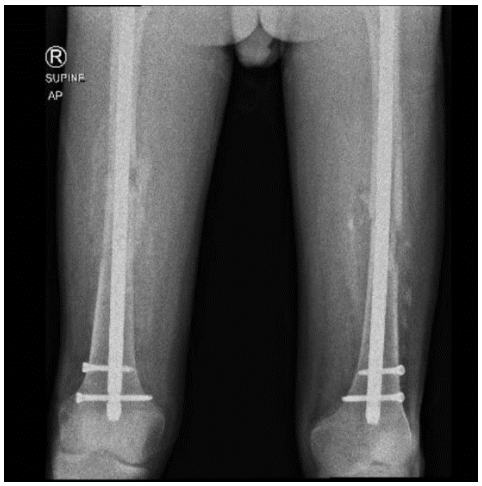

Figure 1: Radiograph A-P view after internal fixation of both femoral bones

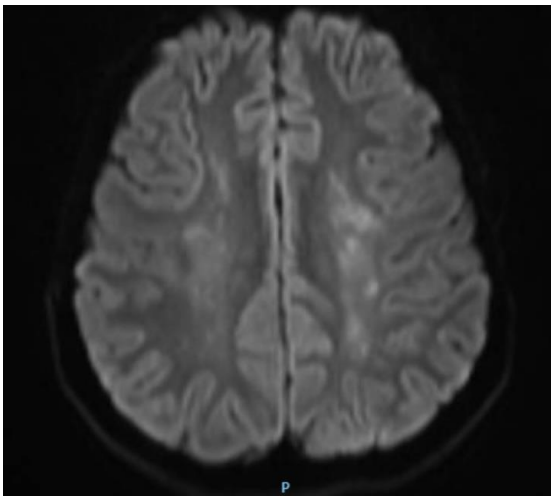

Figure 2: Diffusion weighted images revealed multiple bilateral white matter tiny areas of restricted diffusion.

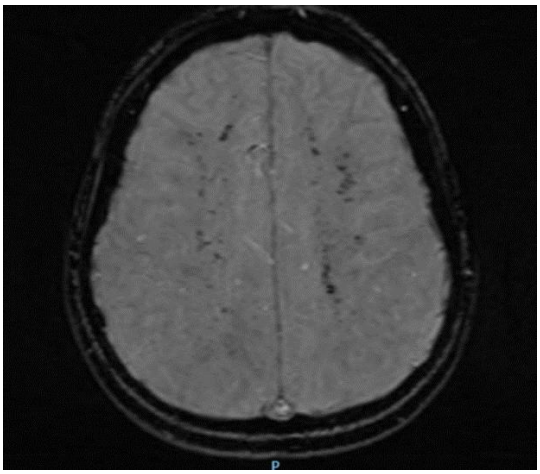

Figure 3: susceptibility-weighted image shows multiple signal drop with associated petechial hemorrhage.

III. Discussion

CFE is a severe complication secondary to long bone fractures, and is rarely observed in clinical practice pathogenesis of FES is unclear but is thought to involve mechanical obstruction and biochemical injury [5,6,7]. The mechanical theory postulates that fat micro emboli enter venous sinusoids, collect in the pulmonary microvasculature, and occasionally migrate into the systemic circulation via the pulmonary capillary bed or right-to-left shunt. The incidence of intracardiac shunt has been described to occur in 20– 34 % of the population. Additionally, micro-fat droplets can theoretically traverse the pulmonary circulation without sequestration, resulting in systemic symptoms. Neuronal ischemia followed by cytotoxic oedema occurs in most patients with cerebral fat embolism. Ischaemic changes typically occur inwatershed areas, seen as a “star field” pattern [8].
Although neurological symptoms are typically accompanied by respiratory failure and skin eruptions,isolated cerebral fat embolism cases have been reported in several instances [9,10]

Neurological features are present in 80–85% of FES [11], often acute encephalopathy with non-localizing symptoms or initial mental status changes that may quickly progress from confusion to coma. Other features are focal neurological signs (hemiplegia, aphasia, apraxia, agnosia, seizure, or dystonia) and neurovegetative disorders (tachycardia, profuse sweating, and hyperthermia) which are observed when the basal ganglia are involved.

Petechial rash is considered pathognomonic of FES and is reportedly present in up to 60 % of patients,usually on the conjunctiva, oral mucous membranes, skin folds of the neck and axillae [11]
The main differential diagnosis in the context of traumatic injury consists of multiple microhemorrhages with diffuse axonal injury (DAI), but neurological symptoms do not occur after a lucid interval. Moreover,in DAI, MRI shows DWI and T2-FLAIR hyperintense abnormalities predominantly in the grey-white matter junction, corpus callosum, basal ganglia, brainstem, and occasionally in the cerebellum [4].

Clinically apparent FES is unusual and requires a high index of suspicion, especially in long bone and pelvic fractures. When patients have long bone and pelvic fractures, multiple bone fractures or deteriorated neurological status, CFE should be considered. Additionally, CFE may occur without an intracardiac shunt.To prevent FES, risk factors should be corrected if possible, and careful anaesthetic management should beundertaken.
In FES, prevention, early detection, and appropriate treatment are important. First, anaesthesiologists and surgeons should recognize patients at risk for developing fat embolism syndrome. Some case reports havesuggested that the use of intracranial pressure monitoring and cerebral tissue oxygenation monitoring clearly defines neuro-protective targets for optimum perfusion, and intracranial pressure control is helpful[12].
Furthermore, the early diagnosis and appropriate management of FES are important, and prior to andfollowing surgery, patients should be monitored comprehensively in the intensive care unit. With appropriate treatment, CFE patients may achieve good results.

IV. CONCLUSION

CFE should be suspected in patients with acute respiratory failure, petechial rash, and deteriorating mentalstatus after trauma or orthopedic surgery. SWI combined with DWI has high sensitivity and specificity for the distribution, range, and characteristics of CFE lesions, and these can be used as the characteristic imaging manifestations for the clinical diagnosis of CFE.

We report the case of a patient with mental deterioration after few hours from orthopedic surgery withinternal fixation of femur, so we recommend MRI brain if the CFE is suspected.

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