

## Post-traumatic Cerebral Fat Embolism: A Case Report and Review of the Literature

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### Abstract

Fat embolism syndrome (FES) is a serious clinical manifestation of fat embolism. This report describes a case of post traumatic Cerebral fat embolism (CFE) following surgery on long bone fractures. The course of disease, clinical picture, management have been described highlighting that early diagnosis and good supportive and intensive care can help in achieving meaningful recovery in most cases.

**Keywords:** Fat embolism; Cerebral fat embolism syndrome; Seizures; Coma Computed tomography; Magnetic resonance imaging.

### Introduction

Fat embolism is a subject of great interest to orthopedic surgeon. The term 'Fat embolism' indicates the presence of fat globules in the peripheral circulation after major trauma associated with fractures of long bones, pelvis and in setting of elective or emergency orthopedic procedures. Fat embolism syndrome (FES) in was first described by Van Bergman (1873) in a patient with fracture femur.<sup>[1]</sup> The true incidence of which is difficult to assess as subclinical forms go unrecognized. Its incidence varies from 0.5 - 30 % of fractured patients<sup>[2]</sup> with higher rate in multiple injured patients with mean mortality of 10 %.<sup>[3]</sup> Cerebral fat embolism (CFE) is a variant of FES characterized by predominance of neurologic manifestations often without initial pulmonary, dermatologic manifestations.<sup>[4]</sup> Neurologic manifestations may vary from decreased level of consciousness to seizures, coma, paresis, altered or loss of speech and vision.<sup>[5]</sup>

### Case report

An 18 years old previously healthy male patient was brought to accident and emergency department, Nizwa hospital following motorbike accident with Type 3A open fracture right tibia and ipsilateral closed fracture of shaft femur. Patient had no evidence of head, chest or abdominal trauma. Upon admission patient was attended by trauma team. Patient had GCS 15/15, normotensive, with normal pulse, respiratory rate. Oxygen saturation (SpO<sub>2</sub>) was recorded as 98 %. Blood work up including FBC, biochemistry, coagulation profile, group cross match and relevant radiographs of fractured extremity along with chest,

**Received date:** September 12, 2015

**Accepted date:** January 13, 2016

**Published date:** January 18, 2016

**Citation:** Dhar, D. Post-traumatic Cerebral Fat Embolism : A Case Report and Review of the Literature. (2016) J Anesth Surg 3(1): 65-67.

**DOI:** 10.15436/2377-1364.16.528

pelvis and cervical spine were obtained. All along the patient was resuscitated, fractured limbs were splinted after preliminary wound debridement of open wound right leg.

Five hours after admission patient underwent emergency wound debridement right leg along with external fixation of fracture tibia and closed Interlock nailing right femur under general anesthesia. The surgery lasted for 3½ hours. After surgery he was transferred to the high dependency unit of orthopedic ward for close monitoring. Post operatively he was vitally stable with normal response to verbal commands and normal eye opening. Eight hours post operatively he was found to be drowsy but still responding to verbal commands with normal hemodynamic and oxygen saturation of 98 %. An urgent chest radiograph and CT brain was done. Both revealed no abnormality. Patient over next two hours had further decline in level of consciousness with Glasgow coma score of 6 (eye opening 2, motor response 2 and verbal response 2) with normal response of pupils to



light. Eleven hours post operatively patient had first tonic-clonic seizures following which he was intubated and ventilated, transferred to intensive care unit for neuromonitoring and supportive care. His repeat blood work up showed hypocalcaemia with normal arterial blood gas levels and platelet count. Urine examination did not show any fat globules. Hypocalcaemia was corrected with calcium gluconate infusion over a period of next two days. Second postoperative day patients fundus examination suggested features consistent with fat embolism. On third postoperative day Chest radiograph demonstrated diffuse pulmonary infiltrates and patient was noticed to have petechial rash on the anterior chest wall, axilla and supraclavicular region which disappeared over period of two days. Subsequent chest radiographs demonstrated diffuse involvement of both lungs, worst on day 6 which resolved with continuing supportive treatment and the lung parenchyma appeared to be normal by day 10. Repeat CT scan of brain was reported as showing diffuse edema. MRI was contemplated but could not be done as it was not available in our hospital and shifting patient to tertiary centre for MRI was not found to be feasible in view of his general condition. On the basis of clinical findings diagnosis of cerebral fat embolism (CFE) was confirmed. Patient remained in ICU on mechanical ventilation with general supportive care for 15 days. He was extubated on 16 days post operatively but found to have weakness of left arm with grade 3/5 power with sluggish reflexes and apraxia without any cranial nerve deficit. Patient was reviewed by neurologist and started on physiotherapy and by day 25 of admission to the hospital had made full neurologic recovery with normal speech. He was eventually discharged from hospital by 35 days of hospitalization. Patient has been on follow up since with us regularly and is doing fine.

## Discussion

Cerebral fat embolism is commonly seen in age group of 16-30 years.<sup>[2,3,6]</sup> Our patient also was in the same age group. Neurological manifestations are seen usually in postoperative period following surgeries on long bones and pelvis.<sup>[3,6,7]</sup> as was seen in our patient.

The pathophysiology of CFE is not definitively understood. Various theories have been put forward. The Mechanical theory states that fat droplets released from the bone marrow after fracture of long bones enters the torn veins due to high intramedullary pressure and is transferred to pulmonary vasculature where large fat globules > 8 microns results in mechanical obstruction of lung capillaries. Smaller fat globules of 7 microns or less may cross pulmonary circulation into systemic circulation causing embolization to brain, kidney and skin. Another way the fat globules can cross over to the systemic circulation is through the pre-existing arterio-venous shunts or patent foramen ovale.<sup>[8]</sup>

The biochemical theory postulates that the embolised fat is degraded to free fatty acids which leads to endothelial damage, alteration of capillary permeability with ischemic pathological changes seen in white matter due to poor collateral circulation although fat embolism occurs in grey matter predominantly due to good capillary circulation.<sup>[9]</sup>

Neurologic manifestations of CFE can present in form of confusion, encephalopathy, seizures or coma.<sup>[10,11,12]</sup> Diagnosis is based on clinical features and is collaborated by associated pulmonary and cutaneous manifestations. Subclinical CFE is

difficult to diagnose because in early stages CT scan and MRI of brain is negative.<sup>[9]</sup> In our case the diagnosis of CFE was made that the patient had cerebral manifestations following orthopedic operations in form of decreased level of consciousness followed by seizures and other findings in form of petechial rash, fundus findings of FES, hypocalcaemia and ARDS changes on chest radiographs.

The neuroradiological diagnosis of CFE based on CT and MRI imaging modalities in early stages are non specific. Cerebral CT is used primarily to exclude other causes of neurological dysfunction as it is usually negative despite focal deficit or clinical encephalopathy.<sup>[13]</sup> MRI is more sensitive and typical findings are in form of multiple small, scattered, no confluent hyper intense intracerebral lesions on T2-weighted scans occurring in both gray and white matter.<sup>[3,14]</sup> Their number correlates with the Glasgow coma scale<sup>[15]</sup> with lesions disappearing over period of few weeks.<sup>[14,15]</sup> DW-MRI has been however found to be more sensitive and specific in picking up early high intensity lesions in brain in form of star field pattern of scattered bright spots on a dark background.<sup>[16,17]</sup>

The mainstay treatment of CFE is supportive with ventilator support the duration of which may extend from 3 weeks to 6 months with good recovery.<sup>[3,18]</sup> Pfeffer et al have reported poor outcome in cases where there was associated hypoxia and hypotension. Cerebral edema if present leads to rapid neurological deterioration in these cases.<sup>[3,18]</sup> Further neurological findings of CFE may be masked if there is associated head trauma. Long term ventilator support is needed in these patients to prevent hypoxic brain injury which if avoided leads to good prognosis.

Early diagnosis, prevention by early immobilization of fractures, stable haemodynamics and adequate tissue oxygenation, with idea of maintaining arterial oxygen saturation of more than 90 % should be the goal. Ancillary measures as adequate hydration, prophylaxis against deep vein thrombosis, DIC and stress ulcers should be part of the regimen. Corticosteroids use in FES has been a topic of debate. It is known that corticosteroids inhibit activation of complement system, protect capillary endothelium, stabilize the granulocyte membrane and retard platelet aggregation. Corticosteroids therapy used prophylactically in selected cases has been found to reduce the incidence of FES.<sup>[16,17]</sup>

The prognosis of CFE if recognized and treated early is good with complete recovery but can prove fatal if its diagnosis remains elusive in early stages.

## Conclusion

Any patient with fractures of long bones following orthopedic surgery who develops an expected deteriorating level of consciousness or seizures in absence of head trauma or other pre-existing medical condition should be evaluated for suspected CFE keeping in mind the clinical features of FES, and where facilities are available to go in for early neuroimaging so as to rule out other differential diagnosis and treat CFE as soon as possible to prevent long term disability or fatality.

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