



A CASE OF FAT EMBOLISM SYNDROME DUE TO BOTH BONE FRACTURE

Karthikeyan B Jayanthi R

Stanley Medical College

Abstract:

Fat embolism syndrome is a fatal complication of long bone fractures. Classically described as the triad of hypoxia, petechiae, and neurological impairment, it is characterized by bone marrow fat entering the systemic circulation and the individual's inflammatory response to it. Although fat embolization occurs in the majority of patients with long bone fractures or during orthopedic procedures, clinical signs and symptoms occur in only 1-10% of these patients. It is usually seen in the context of poly trauma or a femoral fracture. There are few reports of fat embolism syndrome occurring after isolated long bone fractures other than those of the femur. This case report discusses a case of Fat embolism following fracture of both bones.

Case Report:

A 69 year old male, known case of systemic hypertension on regular treatment for last 4 years with no other comorbidities had a road traffic accident around 4 pm. At the time of admission patient was conscious, oriented, hemodynamically stable and systemic examinations were normal. The patient was found to have comminuted fracture of both bone left leg, apart from this no other injuries were found. The fracture was immobilised with plaster of Paris.



Fig 1 : Comminuted fracture both bone leg – left side.

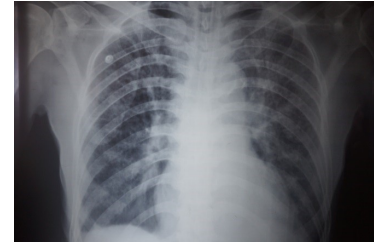


Fig 2 : Chest X ray showing diffuse alveolar infiltrates

Patient was started on treatment with broad spectrum antibiotics, analgesics and other supportive care. His lab Investigations complete blood count, renal function, ECG and chest X ray were normal.

Around 1 am in midnight, patient became breathless, tachypneic and hypoxic. His saturation started to decrease to less than 90 % in spite of oxygen support. His respiratory examination showed bilateral crepitations. His initial ABG showed metabolic acidosis with respiratory alkalosis. Repeat chest x ray was taken; it showed diffuse alveolar infiltrates.

At the same time patient mental status deteriorated from confusion to drowsiness. His CNS examination showed no focal neurological deficit and plantar was flexor bilaterally. CT brain was taken, there were no evidence of infarct or haemorrhage. Serum electrolytes were normal.

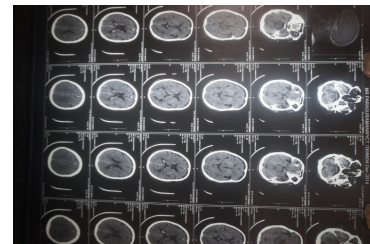


Fig 3 : CT brain showing normal study

In view of decreasing saturation and deteriorating mental status, patient was intubated and mechanically ventilated. The patient was suspected to have fat embolism syndrome. The clinical and investigations confirmed that patient is having fat embolism.

He fulfilled Gurd's criteria; 3 major criteria – hypoxemia, CNS depression out of proportion to hypoxia and pulmonary oedema and 3 minor criteria tachycardia, pyrexia and fat globules in urine. The Schonfeld's score was 10 (more than five is required for diagnosis, a semi quantitative index for fat embolism syndrome).

The patient was evaluated further, his transthoracic echocardiography and lower limb venous Doppler study was normal. His subsequent biochemical investigations showed hypocalcaemia, hypomagnesaemia and thrombocytopenia.

MRI brain was taken it showed multiple acute lacunar infarcts involving bilateral cerebral hemisphere, deep white matter, basal ganglia, thalamus, brainstem and cerebellar hemisphere. MR angiography was normal. The distribution of lacunar infarcts was characteristic for fat embolism syndrome due to multiple shower emboli reaching the brain.

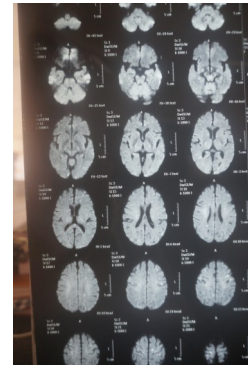


Fig 4: MRI showing multiple acute lacunar infarcts

There were no proven treatments for fat embolism. The patient was treated with adequate oxygenation, ventilatory and supportive care. Steroids and heparin were not given because of controversy and no proven benefits.

The patient improved gradually with supportive care and weaned from ventilator after 7 days. His mental status gradually improved and at the end of 3 weeks patient was able to move all four limbs and no focal neurological deficit was found.

Conclusion

When treating patients with long bone fracture, in case of sudden respiratory impairment and mental status changes the possibility of fat embolism syndrome should be considered. Early immobilization of fractures reduces the incidence of FES and the risk is further reduced by operative correction rather than conservative management. Medical management is mainly conservative once the patient develops syndrome.

References:

1. Gurd AR and Wilson RI. The fat embolism syndrome. J Bone Joint Surg Br 1974; 56:408-16.
2. Kim YH, Oh SW, Kim JS. Prevalence of fat embolism following bilateral simultaneous and unilateral total hip arthroplasty performed with or without cement: a prospective, randomized clinical study. J Bone Joint Surg Am 2002; 84A:1372-9.
3. Schonfeld SA, Ploysongsang Y, DiLisio R, et al. Fat embolism prophylaxis with corticosteroids. Ann Intern Med 1983; 99:438- 43.
4. MG Abbott. Fat embolism syndrome: An in-depth review. Asian Journal of Critical Care 2005; 1:19-24.
5. S Jain, M Mittal, A Kansal et al. Fat embolism syndrome. JAPI • VOL. 56 • APRIL 2008 pg 245- 49