

## Fat Embolism Syndrome from Subtle to Full Blown Presentation

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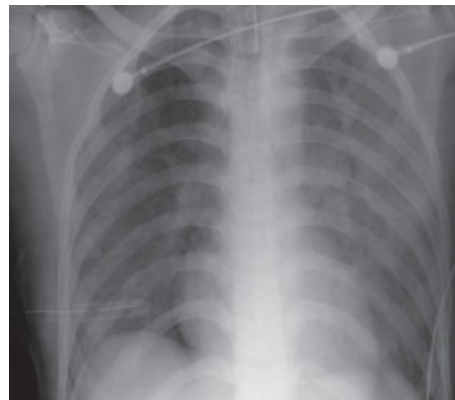
Fat embolism syndrome was first described in 1873 by Von Bergman<sup>1</sup> in patients with fracture of the femur. Fat embolism develops in nearly all patients with long bone fractures or during orthopedic surgical procedures but is usually asymptomatic. However, in a small number of cases, patients develop signs and symptoms due to multisystem dysfunction, mainly involving the lungs, brain and skin. For these cases the term fat embolism or FES is applied.<sup>2</sup> Exact incidence of fat embolism syndrome is not known, but Fabian et al. reported an incidence of up to 30% in their study.<sup>3</sup> The treatment of fat embolism syndrome is primarily supportive. Here we report two cases of fat embolism syndrome encompassing the two ends of the spectrum of presentation of this syndrome.

### CASE REPORT

A 26 year male allegedly met with a road traffic accident. There was a brief period of loss of consciousness with no bleeding from ear, nose or throat. The initial GCS was 15/15 with stable vital parameters. There was contusion on the forehead with swelling on the right thigh. X Ray showed fracture shaft of femur (Rt) and the NCCT of the head was normal. The individual was stabilized and the fracture was immobilized using Thomas splint at the peripheral hospital and later airlifted to our tertiary care hospital for further management. After proper workup he was taken up for open reduction and internal fixation.

On the day of the surgery the patient had mild pallor even though his Hb was within normal limits. A 16 G venous catheter was secured and after preloading with 1000ml of Ringer lactate, 3ml of 0.5% bupivacaine (heavy) was given through a 27 G spinal needle at L2 - L3 level. The analgesia was adequate and fixed at T8 level. During surgery the blood loss of approximately 500ml, was replaced by crystalloids, colloids and two units of blood. The procedure lasted 100 minutes, during which the patient maintained oxygen saturation of 98% to 100% on 6 L of oxygen. His blood pressure ranged between 90 to 130 mm Hg systolic and 54 to 78 mm Hg diastolic, and his pulse was between 88 to 100 beats per minute. In immediate postoperative period, the patient developed a sinus tachycardia with a

pulse of 130 to 140 beats per minute. His blood pressure was 120 - 130/78 - 82 mm Hg and his oxygen saturations were 86% to 88% on room air at a respiratory rate of 32 to 38 breaths per minute. He was put on supplemental oxygen with a face mask at 5l/min with which the SpO<sub>2</sub> of 97 - 98% was achieved. The patient was shifted to the ICU for observation. An ECG, complete blood count, and cardiac biochemical profile were obtained. The ECG showed sinus tachycardia without acute ST or T wave changes. His hematocrit and cardiac enzyme assays were within normal limits but his pulse ranged from 135 to 140 beats per minute with a respiratory rate of 24 to 36 breaths per min. Two hours postoperatively, he developed a fever of 102.4°F. An arterial blood gas revealed moderate hypoxaemia. A chest radiograph showed bilateral perihilar fullness but a lack of infiltrate. The echocardiography revealed a normal study. By the evening on the day of the surgery the patient had become drowsy and had developed petechiae all over his upper trunk including axillae. There was a drop in the hemoglobin level with thrombocytopenia for which he was transfused packed RBC, FFP and platelets.



The chest x ray showed bilateral homogenous opacities. He was diagnosed as fat embolism syndrome by Gurd's criteria.<sup>4</sup> The urine and sputum were sent for fat globules which were negative and fundoscopy was within normal limits. He was managed conservatively with oxygen by face mask. At the end of the third post operative day, the patient was well oriented, the petechiae had started to

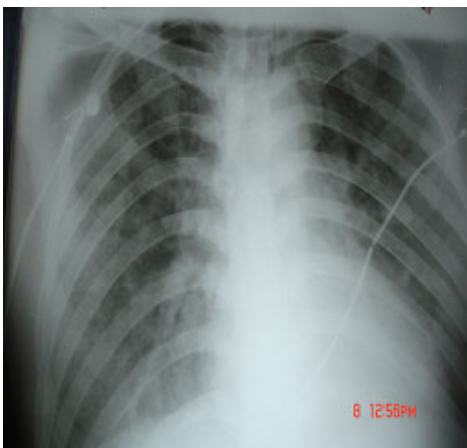
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disappear and the opacities on the chest x ray had started to clear up. He made a remarkable recovery with supportive measures and was subsequently shifted from the ICU.

## CASE REPORT 2

A 48 year old lady was brought to the hospital with alleged history of road traffic accident. There was associated loss of consciousness but no bleeding from the ear, nose or throat. Primary and secondary survey revealed fracture lower one third of left femur along with fracture of shaft of left tibia. There was associated fracture of the pelvis (Left). The ultrasound of the abdomen and NCCT head did not reveal any abnormality. She was adequately resuscitated and planned for open reduction and internal fixation of the femur and tibia on the following day.

On the day of the surgery two 16G IV cannulas were secured and in view of her associated fracture pelvis she was given general anaesthesia. Intra operatively blood loss was about 700ml which was replaced with Ringer lactate, blood and blood products. Her heart rate ranged between 120 to 146 beats per minute with a blood pressure of 70/40 mm of Hg to 128/76 mm of Hg and her SpO<sub>2</sub> was between 78 to 95%. In view of her unstable hemodynamics and an intraoperative ABG suggesting moderate hypoxaemia, it was decided to electively ventilate the patient postoperatively. She was put on assist control mode of ventilation with a tidal volume of 400ml, RR of 16bpm, fiO<sub>2</sub> of 0.6, PEEP of 8 with an I:E ratio of 1:2. On the first post operative day her hematocrit dropped and she developed thrombocytopenia with an INR of 1.97.



X-ray chest showed diffuse bilateral infiltrates with right sided pleural effusion; ECG revealed right ventricular strain pattern with tachycardia. ABG showed pH of 7.40 with pCO<sub>2</sub> of 42 mm of Hg and pO<sub>2</sub> of 71 mm of Hg. The fundoscopy was normal. Accordingly as per Gurd's classification<sup>4</sup> she was diagnosed as a case of Fat embolism

syndrome. She was subjected to invasive hemodynamic monitoring maintaining a CVP of 10 - 12 cm of water with a mean arterial pressure of > 65 mm of Hg. She showed dramatic improvement with ventilatory support and could be weaned off and extubated on the second post operative day.

She however continued to have episodes of mild hypoxemia (SpO<sub>2</sub> between 80 to 90%) which could be managed with non invasive ventilation in BIPAP mode. We used the ST mode with an initial IPAP of 14 cm H<sub>2</sub>O and an EPAP of 7 cm H<sub>2</sub>O. She had an uneventful recovery thereafter.

## DISCUSSION

Even in the current era of technology, careful clinical examination with a high index of suspicion remains a "gold standard" for diagnosis of fat embolism syndrome in the perioperative period.<sup>5</sup> The assessment of a patient with acute onset of shortness of breath after an orthopedic operative procedure should include consideration of pulmonary thromboembolism and fat embolism as possible causes. Fat embolism occurs in closed fractures of large bones and pelvis. Fat embolism typically manifests 24 to 72 h after initial insult. Affected patients presents with the classic triad of hypoxemia, neurological abnormality and petechial rash. Among the clinical triad pulmonary manifestation are the earliest to appear. These include tachypnoea, dyspnoea and cyanosis which progress to respiratory failure in 10% of the cases. Cerebral changes are seen in 86% of cases and these ranges from headache and confusion, to stupor, rigidity, convulsions and coma. Petechial rash is reddish brown, non palpable, appears on the upper chest, neck and conjunctiva.<sup>6,7</sup>

According to Gurd's et al,<sup>4</sup> diagnosis of FES need at least two major criteria or one major and four minor criteria to be present in order to diagnose FES.

Our first patient had pulmonary manifestations in the form of tachypnoea, pulmonary infiltrates and mild hypoxemia which could be managed with supplemental oxygen with face mask. He had also shown CNS involvement in the form of drowsiness and subsequently exhibited petechial rash. However, inspite of protean manifestations of fat embolism, this patient did not have a stormy course.

Our second patient had a more turbulent course and in view of moderate hypoxemia in the perioperative period had to be electively ventilated after the orthopaedic procedure. There was radiological and echocardiographic evidence of fat embolism in this patient in the immediate postoperative period. She manifested episodes of mild to moderate hypoxemia even after she was weaned from

invasive ventilation. Since the patients level of consciousness was satisfactory, we decided against invasive ventilation for these episodes of hypoxemia and instead gave her trials of non invasive ventilation in the BIPAP mode. We could achieve satisfactory blood gases with non invasive ventilation and could tide over the periods of hypoxemia.

Besides mechanical ventilation and maintaining adequate blood gases, the other aspects of intensive care that have become the standard of care need to be enforced at the earliest in patients with fat embolism syndrome. Since management of Fat embolism syndrome is mainly supportive, maintaining stable haemodynamics, use of blood products as clinically indicated, hydration, prophylaxis for deep venous thrombosis and stress-related gastrointestinal bleeding and starting nutritional support are all essential elements of care which have to be pursued aggressively to salvage these patients.

Non invasive ventilation (NIV) is being increasingly used in hypoxemic respiratory failure.<sup>8,9</sup> We employed this mode of ventilation in our second patient to prevent repeated invasive ventilation. However it is mandatory for the patient to be closely monitored while on NIV.

The use of corticosteroids in patients with fat embolism syndrome has long been debated in the literature. The theoretical basis for using corticosteroids is sound; they are thought to stabilize granulocyte membranes, reduce catecholamine levels, retard platelet aggregation, inhibit the activation of complement system, and protect the capillary endothelium. Corticosteroids have been shown to reduce the incidence of fat embolism syndrome when given prophylactically<sup>10</sup> in the emergency department, although data showing a therapeutic role<sup>11</sup> for them once clinically apparent fat embolism syndrome has developed have remained elusive.

To conclude, the aim of reporting these cases was to highlight the two ends of the spectrum as far as manifestations and management of this syndrome is concerned. The syndrome can range from mild to very fulminant course requiring very aggressive intensive care. The perioperative physician has a vital role to play in the

management of this condition. Since the management of these cases is mainly supportive, the general principles of intensive care have to be energetically employed and ventilatory support considered at the earliest. All possible laboratory and imaging aids must be used to rule out other treatable conditions. Non invasive ventilation does have a role in the management of this syndrome.

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