Fat Embolism Syndrome : A Diagnostic Dilemma

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Introduction

Fat embolism syndrome (FES) is a constellation of clinical manifestations following fracture of long bones. In retrospective review, incidence of FES was less than 1% [1]. Fat embolism syndrome is a clinical diagnosis. This condition is often misdiagnosed and fatal if the treatment is delayed. We present two cases of fat embolism syndrome to highlight problems of missed diagnosis.

Case 1

A 19 year old male sustained bilateral closed tibial and fibular fracture. Plaster of Paris (POP) slab was applied over both lower limbs and surgery planned. While awaiting fixation of fractures patient developed high grade fever from second day. Surgery was deferred to investigate fever. No specific cause for fever could be found and empirical treatment for malaria administered. Four days after the injury, patient suddenly developed tachypnea (breath rate 55/minute), tachycardia (pulse 145/minute), and altered sensorium. Oxygen saturation on pulse oximetry (SpO_2) was 60% with high flow oxygen by facemask. He was intubated and mechanically ventilated with 100 % oxygen using anaesthesia circuit and later placed on ventilator in control mode with positive end expiratory pressure (PEEP). As oxygen saturation did not improve, PEEP was gradually increased to 20 cm of H_2O . The high level PEEP resulted in pneumothorax for which an intercostal chest drain was inserted and patient was shifted to a tertiary care hospital.

On arrival to the intensive care unit quick clinical evaluation revealed marked pallor, pulse 140/minute, blood pressure of 130/70 mm Hg, SpO_2 90%, breath rate and neurological status could not be ascertained as patient was under effect of muscle relaxants. Patient was placed on Seimens Servo i ventilator in synchronized intermittent mandatory ventilation (SIMV) mode with PEEP. Initial ventilator settings were tidal volume 350 ml, mandatory breath rate 16/minute, fraction of inspired oxygen (FiO_2) 0.7 and PEEP 10 cm H_2O . With these settings peak inspiratory pressure of 23 cm H_2O , plateau pressure of 20 cm

H_2O , mean airway pressure of 12 cm H_2O and dynamic compliance of 30 ml/cm H_2O was recorded. There were no petechial or subconjunctival haemorrhages. Chest auscultation revealed bilateral extensive crepitations and bronchial breath sounds. Fundoscopy revealed multiple haemorrhages along the vessel in entire field of both eyes. Doppler study of lower limb showed no evidence of venous thrombosis. Investigations revealed haemoglobin (Hb) 6.8 gm %, chest radiograph showed bilateral non homogenous opacities with predominant basal distribution (Fig.1). Arterial blood gas (ABG) showed pH of 7.25, partial pressure of oxygen in arterial blood (PaO_2) 55 mm Hg, partial pressure of carbon dioxide in arterial blood (PaCO_2) 35 mmHg, bicarbonate (HCO_3) 18 mmol/L and $\text{PaO}_2/\text{FiO}_2$ ratio of 78. Other investigations were within normal limits. Right internal jugular vein was cannulated and central venous pressure (CVP) of 12 cm of saline was recorded. Frank blood was noted on endotracheal suction.

Fig. 1 : Chest radiograph (antero-posterior view) shows bilateral non homogenous opacities in a predominantly basal distribution

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On the basis of clinical background, chest radiograph and arterial blood gas analysis report, the case was diagnosed as fat embolism syndrome with acute respiratory distress syndrome (ARDS). Patient was kept sedated, muscle relaxants discontinued and mechanical ventilation continued. Tachycardia and tachypnea settled down after eight hours of ventilation and patient started responding to simple verbal commands. The case was discussed with orthopaedic surgeon for fixation of fractures on fifth day. A consensus decision was taken to wait for some time. Next day while on ventilator, patient suddenly desaturated (SpO_2 70%), became restless and developed tachypnea (breath rate 60/minute) and tachycardia (heart rate 150/minute). There were no specific changes on electrocardiogram and transthoracic echocardiography did not show any evidence of right ventricular strain. Patient improved within one hour with readjustment of ventilator settings. This episode was thought to be fresh episode of fat embolism.

In view of recurring fat embolism, it was decided to fix fractures early. On sixth day bilateral open reduction and dynamic compression plating of both fractures was done under general anaesthesia. Intraoperative course was uneventful. Postoperatively, ventilation, adequate analgesia, and antibiotics were continued. On seventh day patient improved and he was placed on oxygen mask. After eight hours of extubation patient again developed tachycardia, tachypnea, diaphoresis, and mild desaturation (SpO_2 90%). This time noninvasive ventilation was used to tide over the crisis. After six hours of mask ventilation SpO_2 improved to 97%. Thereafter he made an uneventful recovery.

Case 2

A 21 year old male sustained compound fracture left femur, closed fracture right femur, closed fracture right tibial shaft, and fracture right radius. There was no other injury. Initially fractures were stabilised temporarily by bilateral skin traction and POP slab. Prophylactic dose of fractionated heparin was started and definitive surgery was planned. On third post injury day, patient became drowsy and developed vomiting. Within few hours glasgow coma score (GCS) dropped to 6/15 but non contrast computed tomography scan (NCCT) of head was normal. Neurosurgical consultation ruled out traumatic brain injury. The patient was intubated to secure airway and placed on ventilator. Arterial blood gases and other investigations were within normal limits. On fourth day magnetic resonance imaging (MRI) of brain showed multiple punctate foci of hyperintensity in the cerebellar hemispheres, subcortical white matter of both cerebral hemispheres, thalami and splenium of corpus callosum probably as a result of fat embolism (Fig. 2). Fundoscopy was normal and there were no petechial or subconjunctival haemorrhages. Ventilation, decongestive therapy and other supportive treatment continued. On fifth day, patient improved neurologically and was taken up for fixation of fractures under general anaesthesia. All fractures were fixed with interlocking nail. Intraoperative course was uneventful. However there was neurological deterioration postoperatively with GCS 10/15 that was thought of due to fat embolism during surgery.

Fig. 2: MRI of the brain (flair axial images) shows multiple punctate foci of hyperintensity in the cerebellar hemisphere, subcortical white matter of both cerebral hemispheres, thalami and splenium of corpus callosum

Postoperative ventilation and other supportive treatment continued. The patient was extubated on eighth day.

Discussion

In patients with long bone fractures, a high index of suspicion should be maintained to diagnose fat embolism syndrome. Even in the current era of technology, careful clinical examination with a high index of suspicion remains the “gold standard” for diagnosis of fat embolism syndrome [2]. FES commonly presents with triad of pulmonary, cutaneous and cerebral signs. Numerous tests have been proposed to aid in the diagnosis of FES, including serum lipase, triglycerides, staining for fat of bronchoalveolar lavage specimen and evaluation of blood for fat obtained from a pulmonary artery catheter. None have been shown to be specific.

Two theories have been proposed to explain the appearance of fat droplets in blood in FES. Mechanical theory states that fat from the bone marrow may enter torn medullary veins. Fat droplets may then pass through the pulmonary blood vessels into systemic circulation through arterio-venous shunts or an intracardiac shunt to become arterial emboli resulting in cerebral symptoms and petechial haemorrhages [3]. Biochemical theory explains that FES is caused by toxic effects of free fatty acids liberated at the endothelial layer which causes endothelial disruption, perivascular haemorrhages and oedema [4]. This biochemical theory explains FES seen with inflammatory conditions such as pancreatitis, severe burns and sickle cell disease.

Surgical risk factors for FES include intramedullary nailing (especially of the femur and tibia) and joint replacement of the hip or knee with prostheses that displace large volumes of marrow [5]. Any instrumentation of the marrow of long bones may produce fat emboli. Intramedullary nailing is the treatment of choice in stabilizing femoral and tibial fractures. Intravasation of bone marrow content can

occur in reamed femoral nailing [6].

The treatment of FES is supportive, in the form of ventilatory and haemodynamic support. Once patient develops FES following long bone fracture, it is best to delay surgery till the patient improves. Early fixation of fractures should be considered to avoid recurrent embolization. Several small studies suggest a decrease in the incidence and severity of hypoxemia when prophylactic steroids are given at the time of injury [7].

Neurologic symptoms of FES are usually non specific and consist of dementia, disturbance in consciousness, convulsion or coma. Rarely FES presents as a neurologic disorder without accompanying respiratory symptoms. The characteristic appearance of cerebral fat embolism on flair axial magnetic resonance images is diagnostic [8]. Our first patient presented with fever and later developed respiratory distress while second patient presented as coma and due to unusual features definitive treatment was delayed in both the cases. Fat embolism syndrome takes 24-72 hours to develop following long bone fracture. Incidence is higher with multiple bone fractures or movement of fracture segments. Fat embolism syndrome carries a mortality of 10-20 %. Pinney et al [9], did not come across any case of FES in a study of 60 patients, who underwent surgery within ten hours of injury. Therefore early fixation of fractures within 12 hours of injury could prevent the development

of fat embolism syndrome.

Conflicts of Interest

None identified

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Answers to MCQs

1. (c)	2. (c)	3. (b)	4. (d)
5. (a)	6. (c)	7. (d)	8. (a)
9. (b)	10. (c)	11. (d)	12. (d)
13. (d)	14. (b)	15. (d)	