Fat Embolism Syndrome

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ABSTRACT

Nearly all patients following fractures of bones develop sub-clinical form of fat embolism but the classical form of fat embolism syndrome (FES) presents with triad of respiratory, neurologic and dermal manifestations. Non-traumatic conditions can also have fat embolism, but the incidence is very low. The diagnosis is mainly clinical supported by laboratory and radiological finding. Treatment is mainly supportive with early stabilization of fractured bones. In most cases, prognosis is good if the condition is detected and treated early. High index of suspicion in polytrauma patient is the key to early diagnosis of this condition. This report describes two cases of FES, the second case being fulminant fat embolism with added mortality.

Key words: Fat embolism. Fat embolism syndrome. Polytrauma. Adult respiratory distress syndrome.

INTRODUCTION

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 orthopaedic procedures. Fat embolism syndrome (FES) is a serious clinical manifestation of fat embolism characterized by the triad of respiratory distress, deteriorating level of consciousness and petechiae.1

Zenker (1861) first described the presence of fat droplets on autopsy in lung parenchyma of a railroad worker who sustained fatal thoraco-abdominal trauma.2 The true incidence of fat embolism is difficult to assess as sub-clinical forms go unrecognized. The incidence is less than 1.1%.3 It is frequent in young men and high velocity accidents and rare in children as the bone marrow in children contains more haematopoietic tissue and less fat.3 FES can also be associated with other non-trauma settings such as pancreatitis, diabetes mellitus, liposuction, sickle cell disease, burns, blood transfusion, infections, collagen disease, neoplasm, renal transplantation and orthopaedic procedures as medullary reaming.3

This report describes two cases of fat embolism syndrome (FES) in polytrauma patients. The aim is to highlight this serious condition which has a high morbidity and mortality if unrecognized. Various theories of pathogenesis along with review of recent management and existing literature on this subject is discussed.

CASE REPORT

Case 1: A 21 years old male patient was involved in a road traffic accident (RTA) and sustained bilateral closed fractures of theshaft of femur with open wound of right ankle. Patient was admitted to Nizwa Regional Referral Hospital nearly 3 hours after accident. He was resuscitated in the accident and emergency. The right foot wound was dressed with application of compression bandage. Routine blood work up along with coagulation profile, biochemistry, grouping with cross matching and ECG was performed. All needful radiographs were done prior to shifting the patient to the ward. Patient was admitted for emergency wound debridement for the open bleeding wound of the right foot. Both fractured femora were immobilized in sliding skeletal traction with upper tibial pin.

On the evening following wound debridement of right foot patient developed altered sensorium without any haemodynamic instability. Oxygen saturation (SPO2) of 96% at room air was recorded without any focal neurologic deficit. An urgent CT scan of brain was done to rule out any head trauma which was reported as normal. ABG (arterial blood gases) were within normal limits. Within next few hours patient developed tachypnea (breath rate of 30/minute) and tachycardia (pulse rate 150 – 160 / minute) along with deteriorating sensorium. Oxygen saturation on pulse oximetry (SPO2) had dropped to 85 – 90% at room air and improved to 96% with 60% oxygen flow by ventimask. ECG showed sinus tachycardia with incomplete right bundle branch block. Repeated ABG showed pH 7.4, partial pressure of oxygen in arterial blood (PaO2) as 80 mmHg, partial pressure of carbon dioxide in arterial blood (PaCO2) as 35 mmHg and HCO3 level of 23 mmol/l. Full blood count (FBC) showed thrombocytopenia with other blood parameters within normal limits. Follow-up chest radiograph showed bilateral hilar haziness with basal infiltrates. No petechial or sub-conjunctival
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haemorrhages were found. Physician's consultation was sought as tachycardia persisted along with tachypnea and hypoxemia. A diagnosis of fat embolism syndrome on the basis of clinical findings, ABG report and chest radiograph findings was established. The patient was shifted to the intensive care unit for closer observation where he was sedated, and kept on supportive management with 60% oxygen by ventimask to maintain his SPO$_2$ around 96 – 98%. Over the next 6 days, patient’s level of consciousness improved, his respiratory rate and pulse rate stabilized and oxygen saturation at room air returned to 98%. Chest radiographs showed improvement and thrombocytopenia got corrected. Prophylaxis against deep vein thrombosis and fluid intake and output was maintained during his stay in hospital.

On the 10th and 12th day of admission, patient underwent surgical stabilization of left and right femur respectively. Postoperatively, patient was kept under close observation initially in intensive care unit and then in high dependency ward till his discharge from the hospital on the 18th day of admission with un-eventful recovery.

**Case 2:** A 25 years old male was transferred from a peripheral hospital 6 hours after being involved in RTA. He had been intubated in peripheral hospital due to low GCS. On arrival, patient was attended by the trauma team and connected to the ventilator. Oxygen saturation on pulse oximetery (SPO$_2$) was recorded as 94% with blood pressure of 110/70 mmHg, pulse rate of 130 beats/minute with bilateral sluggish reacting pupils. He was found to have sustained closed fracture of the shaft of right humerus, bilateral fractures of the shaft of femur, with open wound on right knee and fractured patella. Initial chest, abdominal, spine and pelvic examination was normal. Full blood work-up including FBC, biochemistry, coagulation profile, group cross match and radiographs of head, cervical spine, chest, extremities and pelvis were obtained.

Patient was resuscitated, fractured limbs were splinted after preliminary wound debridement of open wound of right knee. CT scan of head was normal. Initial chest radiograph was unremarkable. Investigations showed low haemoglobin 8 gm/dl with normal platelet count, and biochemical parameters. Patient was planned for definitive fracture fixation after correction of his haemoglobin. Blood transfusion was started in emergency.

He was admitted to the intensive care unit and placed on ventilator in synchronized intermittent mandatory ventilation (SIMV) mode with PEEP adjusted to maintain oxygen saturation above 90%, FlO$_2$ 60% and PEEP 7 cms H$_2$O. He was started on broad spectrum antibiotics along with prophylactic low molecular weight heparin. Both lower limbs were placed in traction.

His fluid administration was monitored to maintain CVP and urine output. On the second day of admission, patient was found to be febrile with petechial haemorrhages on the anterior aspect of upper chest wall, axilla and neck. Fundoscopy examination was normal.

On the third day, chest examination revealed bilateral basal crepitations and radiographs of chest showed bilateral haziness with infiltrates suggestive of adult respiratory distress syndrome (ARDS). Patient was diagnosed a case of head injury with multiple fractures, fat embolism syndrome and ARDS. The anaesthetist tried to reduce FlO$_2$ over the following days but failed and patient continued to be febrile with high-grade fever, reaching 40°C, along with persistent tachycardia. Septic screening was unremarkable. On the 7th day of admission, patient had cardio-respiratory arrest from which he could not be resuscitated. The probable cause of death was adult respiratory distress syndrome following FES secondary to multiple injuries.

**DISCUSSION**

The pathophysiology of FES 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 enter the torn veins due to high intramedullary pressure and reach pulmonary vasculature where large fat globules > 8 microns result 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 arteriovenous shunts or patent foramen ovale. The delay of 12 – 48 hours between trauma and development of FES in non-traumatic settings cannot be explained by mechanical theory.

The biochemical theory postulates that the embolised fat is degraded to free fatty acids which leads to endothelial damage, extravasation of fluid, perivascular haemorrhages and oedema. The coagulation hypothesis considers that in FE a disseminated coagulation state is present and that combined fat, platelets, fibrin, leucocytes and red blood cells would be responsible for onset of FES. The last two theories explain appearance of petechiae, acute respiratory distress syndrome (ARDS) and account for non-traumatic FES.

Diagnosis of FES is primarily clinical and investigations are only supportive to clinical diagnosis and monitoring the future course of disease. The diagnostic work-up of suspected FES should include serial arterial blood gases (ABG) as hypoxaemia is one of the early features. Serial chest radiographs repeated over the period of time as the disease progresses may show infiltrates in lungs suggestive of ARDS. Other laboratory markers such as un-explained thrombocytopenia, hypocalcaemia,
hypofibrogenemia, elevated serum lipase and lipiduria may be seen 3 – 5 days after onset of embolization. Centres where facilities are available Transesophageal echocardiography can be used to evaluate the embolization. CT Chest and MRI brain may be valuable tools of investigation for picking-up early emboli to lungs and brain where other investigations are inconclusive.

Gurd and Wilson proposed one major and four minor criteria for diagnosis of FES. Lindeque et al. proposed diagnosis on involvement of respiratory system and Schonfeld et al. proposed seven-feature criteria.5

Gurd and Wilson criteria for fat embolism syndrome are divided into a major and minor criteria. Major criteria include petechiae over the anterior chest wall, axilla and sub-conjunctiva, hypoxaemia PaO2 < 60 mmHg and FIO2 = 0.4, central nervous system depression disproportionate to hypoxaemia and pulmonary oedema. Minor criteria included tachycardia (< 110 beats/minute), pyrexia < 38.5°C, emboli present in retina on fundoscopy, fat in urine or sputum, unexplained drop in platelet count or haematocrit and increasing ESR.

Lindeque’s criteria include sustained PaO2 (< 8 kPa) sustained PCO2 of > 7.3 kPa or a PH < 7.3, sustained respiratory rate > 35 bpm, despite sedation and labored breathing suggested by dyspnea, accessory muscle use, tachycardia and anxiety. Schonfeld’s criteria included petechiae (5), chest X-rays changes (diffuse alveolar infiltrates, (4)) hypoxaemia (PaO2 < 9.3 kPa, 3) fever (> 38°C), tachycardia (> 120 bpm, 1) and tachypnea (> 30 respiratory pm, 1). Cumulative score of > 5 is required for diagnosis. Any patient with one of the above criteria is strongly in favour of FES. Lindeque’s criteria has shortcoming that it is based only on lung function.

Various orthopaedic procedures such as intramedullary nailing for fractures of shaft femur, tibia, hip and knee replacement surgery where reaming of the medullary canal increases intramedullary pressure and may propagate marrow fat into venous circulation increasing risk of FES.8 Unreamed intramedullary nailing and cementless hip arthroplasty procedures decrease risk of FES.7,8

The mainstay treatment of FES is supportive. 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 like adequate hydration, prophylaxis against deep vein thrombosis and stress ulcers should be part of the regimen.

Mechanical ventilation and PEEP should be considered only if FIO2 of > 60% and CPAP > 10 cm are required to achieve a PaO2 > 60 mmHg. One must remember that neither mechanical ventilation nor PEEP is always beneficial as they may promote acute lung injury, so its use is only to accomplish adequate gas exchange.1,3 Corticosteroids use in FES has been a topic of debate. Corticosteroids inhibit activation of complement system, protect capillary endothelium, stabilize the granulocyte membrane and retard platelet aggregation. A recent meta-analysis found that corticosteroids reduce the incidence of FES and hypoxemia in adult patients who have suffered isolated diaphyseal long bone fractures.9

In the first case there was no delay in the treatment and patient did not have any associated head injury, while in the second case definitive treatment got delayed due to time lost in transferring the patient and associated head injury which might have contributed to the mortality. The FES continues to remain a diagnostic dilemma. Therefore, prompt recognition and aggressive multidisciplinary management remains the key in reducing the morbidity and mortality arising from this condition.

REFERENCES


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