FAT EMBOLISM SYNDROME: A CASE REPORT

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ABSTRACT

A young motorcyclist, who had met a road traffic accident resulting in multiple fractures, later developed fat embolism syndrome. He had to be instituted mechanical ventilation for hypoxemia and deteriorating levels of consciousness and was rendered vigorous management for his critical general condition.

Key Words: *Road Traffic Accident, Multiple Fractures, Fat Embolism Syndrome, Ventilatory Support.*

INTRODUCTION

Fat embolism syndrome (FES) follows long bone fractures. In 1862, Zenker first described this syndrome at autopsy. In 1873, von Bergmann clinically diagnosed fat embolism for the first time in a patient with femur fracture.[1] Because of its variable clinical course, it is difficult to determine the incidence of this complication. Different studies have shown the range from less than 2% to 22%.[2] Fat embolism has been associated with many non traumatic disorders, but is most common after skeletal injury and is most likely to occur in patients with multiple long bone and pelvic fractures. Patients with fractures involving the middle and proximal parts of the femoral shaft are more likely to develop fat embolism.[3] Age also seems to be a factor in the development of fat embolism, young men being at an increased risk. Timely management, these patients have excellent prognosis but the patients with multiple underlying medical problems and less physiologic reserves have worse outcomes.[4]

CASE REPORT

A young man of 19 was admitted at 2015 hours on 2nd January 2005 in surgical intensive care unit (SICU) of POF Hospital Wah, with a 30-minute history of motorcycle accident. He was fully conscious and oriented. He looked pale, in severe agony, pulse 130/minute, blood pressure 130/70 mm Hg and respiratory rate 18/min. His arms, left thigh and leg were tender with restricted movements and deformities. He also had multiple abrasions and bruises all over the body. The examination of all other systems was unremarkable. He had an Oxygen saturation >97% on air and a Glasgow Coma Scale (GCS) of 15/15. A clinical diagnosis of poly trauma with multiple fractures was made. Intravenous lines were established and crystalloids were given, cross-matched blood transfusion started and analgesics administered. All the fracture sites were splinted. Next day, while shifting the patient to another hospital on parents decision, the patient suddenly became dyspnoeic, cyanosed, started becoming irritable and drowsy with impaired consciousness. His parameters were, pulse 170/min, Oxygen saturation70%, B.P.175/85mmHg, ECG was normal. He became febrile (102F) and developed crackles in the chest. He was given oxygen, which improved oxygen saturation initially but he became further cyanosed and tachypnoeic (respiratory rate 38/min and Oxygen saturation 50%) within a few hours. A clinical diagnosis of Fat Embolism Syndrome (FES) was made. With the worsening hypoxemia and deteriorating level of consciousness, it became difficult to maintain a patent airway. He was instituted mechanical ventilation along with PEEP.He was started low molecular weight Heparin 40 mg 12 hourly and injection Decadron 8mg 6 hourly. X-ray chest at this stage was normal and arterial blood gases (ABGs) showed compensated respiratory alkalosis without hypoxemia. Next day, petechial spots appeared in both axillae and conjunctivae, which persisted for a few days. X-ray skull, coagulation profile, routine hematological and biochemical investigations remained normal. Urine for fat globules was negative. During ventilation the record of ABGs is shown in table 1.

Patient remained comatose and febrile

ARTERIAL BLOOD GASES (DURING VENTILATORY SUPPORT)

	1st day (before starting ventilation)	3rd day (On ventilator)	5th day (On ventilator)	7th day (On ventilator)
pH	7.35	7.52	7.46	7.40
$PaCO_2$	28.3	27.6	29.3	26.0
PaO_2	46.0	75.3	69.0	84.2
HCO_3	24.2	22.8	21.0	18.6
BE	+1.1	+1.8	-5.2	-8.2
SaO_2	72.2%	96.6%	94.3%	95.3%
FiO_2	0.4	0.6	0.5	0.4

Table 1

with chest X-ray showing evidence of adult respiratory distress syndrome (ARDS). While on ventilatory support, surgery was performed to stabilize the fractures under general anaesthesia. Patient showed gradual improvement in oxygenation and pulmonary condition and was finally weaned off the ventilator after 9 days. Chest physiotherapy and incentive spirometery was continued. On 12th day, when he was stable but drowsy with maintained airway and oxygenation, he had a CT Scan brain, which showed diffuse brain edema. He was given Mannitol with supportive treatment. Patient showed spontaneous eye opening on 14th day. His MRI brain was normal except for evidence of fluid collection in mastoid air cells. His aphasia recovered gradually, receptive component before the expressive. He had been on parental feeding initially and then on entral nutrition and prophylactic antibiotics. The patient was discharged from the hospital on 45th day of his admission in a stable general condition and is being followed up in OPD.

DISCUSSION

The causes of FES include blunt trauma and accidents (90% of cases) involving closed fractures of long bones, acute pancreatitis, diabetes mellitus, burns, joint reconstruction, liposuction, cardiopulmonary bypass, decompression sickness,parenteral lipid infusion and sickle cell crisis. [4]

FES usually occurs 24 to 72 hours after fracture or orthopedic surgery. Bone marrow from a fractured bone or injured adipose tissue releases fat globules, which enter the blood stream through torn veins at the site of injury. These fat globules travel to the lungs, where they form an embolus that blocks pulmonary circulation. Lipase breaks down the trapped fat emboli into free fatty acids, which causes a local toxic effect that damages the epithelium, increases capillary permeability, and inactivates lung surfactant. The increased capillary permeability allows protein-rich fluid to leak into the interstitial space and alveoli, increasing the workload on the right side of the heart and causes pulmonary edema. The decreased surfactant causes alveolar collapse, a decrease in functional reserve capacity, and ventilation/perfusion mismatch, leading to hypoxemia. Platelet aggregation on fat, normal injury-related platelet consumption and platelet dilution through intravenous crystalloid administration, all contribute to thrombocytopenia, petechiae, and possibly, disseminated intravascular coagulation.

FES is a clinical diagnosis with nonspecific and insensitive diagnostic tests. [5] Clinical suspicion is based on the appropriate signs and symptoms and underlying risk factors. Gurd's criteria are often used to diagnose FES clinically. [6] At least one major and three minor criteria are required for the diagnosis. The *major criteria* include hypoxemia with a PaO_2 less than 60mm Hg, pulmonary edema, cerebral manifestations, petechiae in a “vest” distribution, mucous membrane and conjunctivae. The *minor criteria* include tachycardia with a heart rate greater than 110 beats/minute, pyrexia with a temperature higher than 103°F, retinal changes, fat in urine or sputum, an unexplained drop in hematocrit or platelet count, an increasing ESR, jaundice and renal changes. Neurological manifestations (agitation, confusion, stupor or coma) represent capillary damage to cerebral circulation and cerebral edema; which may be exacerbated by hypoxemia. [7]

In the early stages of FES, the patient's $PaCO_2$ may be low secondary to hyperventilation but it increases as the respiratory insufficiency continues.Changes seen on the X-ray chest caused by fat embolism are a late sign of FES. Findings may vary from patchy areas of consolidation, fleck like pulmonary shadows (“snow storm” appearance) to complete whiteout if the condition progresses to adult respiratory distress syndrome

(ARDS).Most of the laboratory tests like Serum lipase level, cytologic examination of urine, blood and sputum to detect fat globules are not sensitive and do not exclude FES. CT scan of brain performed because of alterations in mental status, may be normal or may reveal diffuse white-matter petechial hemorrhages consistent with micro vascular injury. It also rules out the intracranial lesions. Similarly, MRI is helpful to rule out other conditions like brain injury and cortical vein thrombosis. A ventilation/perfusion scan can rule out pulmonary embolism from other sources. The differential diagnoses of FES include deep vein thrombosis (DVT) and cerebral insult of varying degree. The most effective prophylactic measure is to reduce long bone fractures as soon as possible. Maintenance of intra vascular volume is important because shock can exacerbate the lung injury caused by FES. Mechanical ventilation and PEEP are required to maintain arterial oxygenation and the adequacy of ventilation monitored with serial ABGs. [8]

After weaning the patient off the ventilator, deep breathing exercises with incentive spirometery improves the lung capacity by recruiting the atelactatic areas of lungs. Corticosteroids have been effective in preventing development of FES in several trials but their role is still controversial.

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