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Early Explosive Fat Embolism Syndrome: A Case Report

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Abstract **Case Report**

The Fat Embolism Syndrome (FES) is a set of clinical, biological and radiological signs resulting in the obstruction of microcirculation by micro-droplets of insoluble fats. The clinical signs of the FES are not very specific, the diagnosis is difficult and the risk of misunderstanding this syndrome is very real. The FES appears after a trauma, often few days later. At present, there have been few cases of fat embolism presenting within 3 h after trauma. We present here a rare case of early explosive FES.

Keywords: Fat embolism syndrome, Femoral fracture, Acute respiratory distress syndrome.

INTRODUCTION

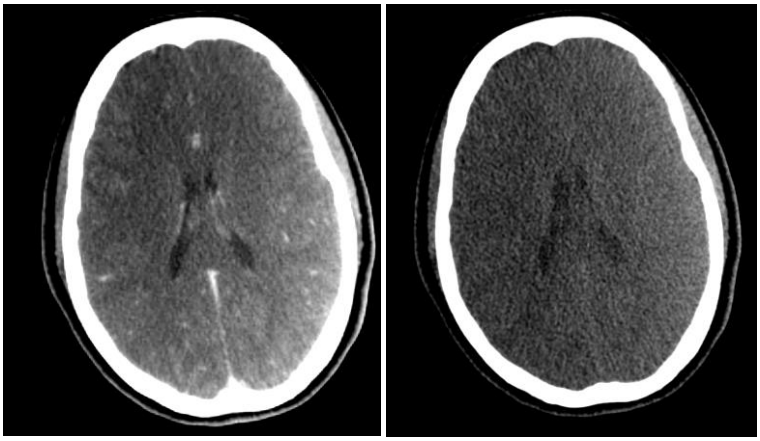
Fat Embolism Syndrome (FES) is a poorly defined clinical phenomenon which has been attributed to fat emboli entering the circulation. This fat can embolise and may or may not produce clinical manifestations [1]. It is common, and its clinical presentation may be either subtle or dramatic and life threatening. It classically presents with respiratory, neurological and dermatological features. It typically occurs after long-bone fractures and total hip arthroplasty, less frequently it is caused by burns and soft tissue injuries [2]. Despite its original description in the 17th century, FES remains one of the least understood complications of trauma. Early stabilization of long bone fractures is thought to reduce its incidence, however the most effective means of achieving this has yet to be determined [3].

CASE REPORT

A fit and healthy 19-year-old woman went to the emergency department of our hospital because of a sports accident and was diagnosed with a fracture of the middle and lower femur of the right lower limb. The patient had no previous medical history. She had no history of surgery, no bad habits such as smoking or drinking, and no history of food or drug allergies.

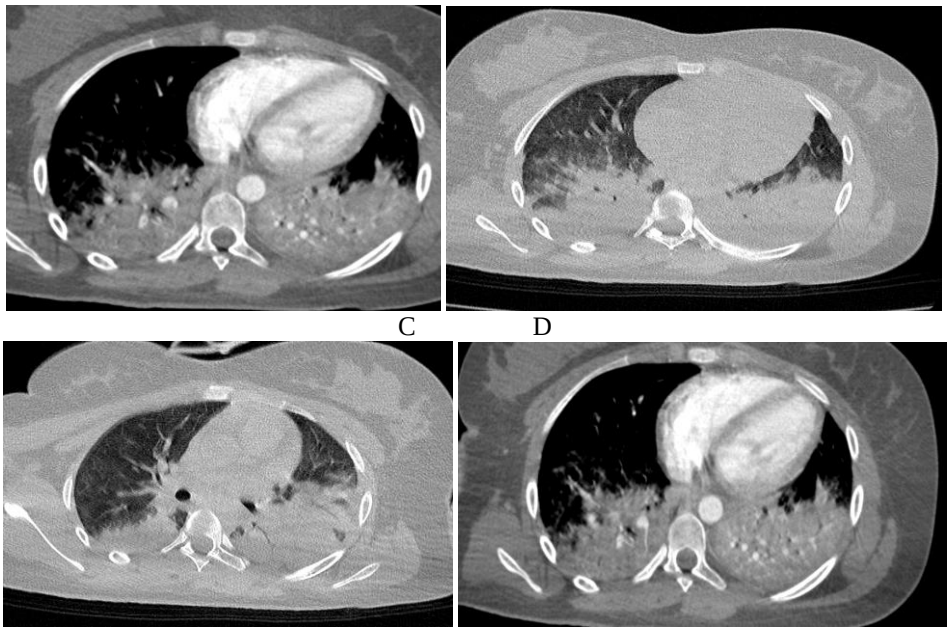
Unfortunately, within one hour of the patient being hospitalized, that is approximately four and a half hours after the injury, the patient's consciousness gradually changed from awake to confused and poor. Then, the patient experienced an increase in heart rate to 115-138 beats/min, an increase in respiratory rate to 30-38 beats/min, and a rapidly progressing decrease in blood oxygen saturation from 95% to70%, and the patient's consciousness quickly deteriorated and was found to be in a deep coma with a Glasgow coma scale (GCS) score of 5. Endotracheal intubation and ventilator assistance were performed, placement of a central venous catheter, was necessary to administer vasoactive drugs and fluids infusion.

As soon as her vital signs were relatively stable, the patient was transferred to the imaging department where urgent examination of the head, chest and abdomen by spiral CT angiography. The scan of the head suggested no abnormality (Figure 1); however there were multiple, diffuse, ground glass opacities and consolidation with interlobular thickening in both lungs in keeping with acute respiratory distress syndrome (Figure 2).



A B

Figure 1: Computed tomography examination. A and B: The results of brain CT angiography showing no obvious abnormality



C D

E F

Figure 2: C, D, E and F: The results of chest CT angiography showing diffuse, ground glass opacities and consolidation in the lung

The diagnosis of Fat embolism syndrome was made and the surgical team was informed. Prompt and adequate immobilisation of fractures is very important in preventing the release of further adipose cells into the circulation. Taking into account the risk of fat embolism induced by intramedullary fixation, open reduction and internal fixation was the chosen surgical method.

The surgery was uneventful, blood transfusion was performed, and fluids were provided to maintain water and electrolyte balance, and the patient was transferred to the intensive care department at midnight for further treatment. The therapy of patient in the intensive care unit was as follows: Central monitoring; lung protective ventilation to maintain adequate gas exchange while minimising the potential for ventilator-associated lung injury (VALI); close observation of mental state and pupillary changes; anticoagulation, supplementation with, plasma, and concentrated red blood cells; maintenance of water and electricity balance; and other supportive treatment. Rapid respiratory recovery was achieved and ventilator support was gradually reduced. Table 1 shows evolution of laboratory values blood gas analysis. And on Day 6, she was successfully weaned off ventilator support to oxygen via partial rebreather mask. Patient's consciousness gradually improved and was weaned of supplemental oxygen on Day 10. Transfer to the orthopedic ward was decided on Day 11 and the patient was discharged on day 14 without any symptoms.

Her GCS score was 15, her brain and neurological system showed no sequelae, and her limb activity was mostly normal six months after the

incident. When the patient was contacted a year after the injury, there were no complaints of discomfort.

Table 1: Evolution of Blood gas analysis laboratory values during the course of illness

	Day1 03/03	Day2 04/03	Day3 05/03	Day5 07/03	Day7 08/03	Day11 12/03
Haemoglobin (g/dl)	13.4	12	9.6	9.1	9.3	11.2
White cells (109 /L)	20.30	15.01	7.10	5	7.96	10.40
Platelets (109 /L)	189	142	132	181	255	518
CRP (mg/L)	200	327.53	296.44	238.8	105.2	19.8
pH	7.21	7.29	7.36	7.40	7.46	7.38
PaO2/FiO2	90	167	223	332	227	314
PaCO2(mmHg)	51	53.8	47.3	49.6	32.4	39

DISCUSSION

FES is the term used to describe a clinical syndrome of systemic alterations brought on by a microcirculation embolism, which is formed when free fat particles with a diameter of 10 to 40 micrometers enter the bloodstream through a ruptured vein following severe trauma [4]. The most typical circumstance is when free fat particles are released into the bloodstream after a bone marrow cavity fracture and impede the microcirculation of the brain, lung, and other organs. Most typically, young men in their third decade of life who suffer multiple leg fractures are affected by FES, according to the most recent literature studies. FES may occur more frequently following a ruptured fracture [5]. Our patient, though, was a young woman who had a fractured femur.

A high level of suspicion should be taken when patients present with hypoxia, confusion or rash following long bone fractures and/or postoperatively. Differential diagnoses include pulmonary embolism, acute respiratory distress syndrome, pulmonary oedema and atypical infections. The severity of the condition can vary, most cases are self- limiting, but mortality has been reported as high as 5–15% [6]. The mainstay of treatment is supportive; ensuring good arterial oxygenation and maintaining good intravascular volume, as shock can exacerbate lung injury [7, 8].

Early diagnosis and treatment are beneficial in reducing mortality [9]. Gurd's, Lindeque's, and Schonfeld's criteria have each been previously proposed for the diagnosis of FES and exclusion of other potential diagnoses [10, 11].

Since all of the diagnostic standards for FES were developed using small sample sizes and with little validation, they may all be criticized. In our case, the patient was diagnosed with FES according to Gurd's criteria (Table 2).

Table 2: Gurd's diagnostic criteria; The presence of one major and four minor criteria were proposed as sufficient for a diagnosis of fat embolism syndrome

Major criteria	+Axillary or subconjunctival petechiae +Hypoxaemia with bilateral radiographic changes +Cerebral signs unrelated to head injury or any other condition
Minor criteria	+Tachycardia +Pyrexia +Emboli present in the retina on fundoscopy +Fat present in urine +A sudden decrease in haematocrit or platelet concentrations +Increasing erythrocyte sedimentation rate +Fat globules present in the sputum

The pathophysiological mechanism of FES has not been fully elucidated. Proposed pathophysiology of most cases of FES combines mechanical and biochemical processes. The mechanical process includes that fat cells activate platelets and accelerate fibrin generation, lodging in the pulmonary capillary, ultimately leading to interstitial haemorrhage and edema, alveolar collapse and reactive hypoxemic vasoconstriction. Massive fat emboli may also cause macrovascular obstruction and shock. The biochemical process suggests that a pro-inflammatory cytokine cascade is caused by toxic free fatty acids, eventually leading to endorgan dysfunction. In the lung, toxic injury to pneumocytes and pulmonary endothelial cells leads to vasogenic and cytotoxic edema as well as haemorrhage. Ultimately, acute lung injury or acute respiratory distress syndrome develops [12, 13].

The patient in our report developed ARDS and required intubation and lung-protective ventilator support very early. Most FES cases occur between 12 and 72 h postinjury [14], and only a few cases have occurred within 12 h after injury [15]. Our case is extremely rare, and the patient showed rapid progression within 4 hours after the injury.

There are currently no specific treatments for FES, hence it is mostly managed by providing respiratory and hemodynamic support. Inflammation induced by free fatty acids (FFAs) plays a crucial role in acute respiratory distress syndrome (ARDS). Given the pathogenesis, the early administration of corticosteroids may be effective. A great range has been reported for the effective dosage of methylprednisolone (total 9–90 mg/kg) [16, 17]. In our case, we used short-term, low-dose methylprednisolone (total 12 mg/kg), and no adverse effects were observed. Finally, we used LMWH to prevent venous thromboembolism effectively in the case with no hematologic abnormalities.

CONCLUSION

Explosive fat embolism is uncommon and potentially deadly. Due to its subclinical presentation or confusing injuries in patients who have been harmed more severely, FES diagnosis is sometimes missed. With supportive care, the death rate is less than 10%, and pulmonary, neurological, and dermatological problems typically completely disappear. A good prognosis is influenced by a high suspicion of FES and early diagnosis. The severity of this disease will be lessened by improved knowledge of the function of the still-research-based "targeted" pharmaceutical therapies and improvements in the perioperative care of polytraumatized patients. Similar to this, a deeper understanding of how FES affects lung damage immunomodulation will aid guide treatment.

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