

## **Fat Embolism Syndrome Due to Fracture Right Femur: A Case Report**

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### **ABSTRACT**

Fat embolism syndrome (FES) is usually associated with fractures of long bones and pelvis. Symptoms usually occurs hours to days after injury. We present a case with fat embolism syndrome due to fracture femur. Prompt supportive treatment of patient's respiratory system and additional pharmaceutical treatment provides the positive outcome. There is no specific therapy for fat embolism syndrome. Prevention, early diagnosis, and adequate symptomatic treatment are very important. The incidence of fat embolism syndrome is reduced markedly in last decades due to early stabilization and surgical correction of fractures of long bones.

**Key words:** Fat embolism syndrome, Trauma, Femur fracture.

### **INTRODUCTION**

Fat embolism is fat particles that enters the circulatory system causing vascular occlusion. Fat emboli (macroglobules) can cause a more serious condition called Fat embolism syndrome, in which there is multisystem dysfunction<sup>1</sup>. The majority (95%) of cases of fat embolism syndrome occur after major trauma. The incidence of FES with single long bone fracture is 1-3% and with bilateral femoral fracture it has been reported in up to 33% of patients. An overall mortality of 5-15% has been described<sup>2</sup>.

Clinical symptoms and signs of fat embolism syndrome(FES) are evident hours to days after injury and are characterized by respiratory distress, altered mental status skin petechiae<sup>3</sup>. Pulmonary dysfunction in the form of dyspnea, tachypnea and hypoxemia are the primary manifestations occurring in 75% of cases<sup>4</sup>. 10% of cases may develop respiratory failure and 5-8% of patients may progress to severe acute respiratory distress syndrome(ARDS)<sup>5</sup>. Half

of FES patients develop severe hypoxemia and respiratory insufficiency requiring mechanical ventilation<sup>6</sup>.

Neurological features in the form of agitation, delirium, seizures, or coma are seen in 86% of patients with FES<sup>7</sup>. Dermatological dysfunction is developed as non-palpable petechial rash in conjunctiva, axilla chest and neck in up to 60% of cases.<sup>8</sup> Some other minor symptoms that may also be present are, anemia, low platelets, tachycardia, pyrexia, myocardial depression, and renal changes (e.g., oliguria, or hematuria)<sup>4</sup>.

Clinical findings are important in diagnosis of FES, while biochemical changes may also be of value. The most common classification scheme for diagnosis is that of Gurd and Wilson, providing major and minor diagnosis criteria (Table 1), according to which the diagnosis of FES requires the presence of at least one major and four minor criteria<sup>4</sup>.

**Table 1 Gurd and Wilson, criteria <sup>4</sup>**

Major features	Minor features
<ul style="list-style-type: none"> <li>• Axillary or subconjunctival petechiae</li> <li>• Hypoxemia PaO<sub>2</sub>&lt; 60mmHg</li> <li>• Fio<sub>2</sub>=0.4</li> <li>• Pulmonary edema</li> <li>• Sudden drop in Hb level &gt; 20%</li> <li>• Central nervous system depression</li> <li>• disproportionate to hypoxemia</li> </ul>	<ul style="list-style-type: none"> <li>• Tachycardia &gt;110/min</li> <li>• Pyrexia &gt;38.5</li> <li>• Retinal fat or petechiae</li> <li>• Urinary fat globules or oliguria</li> <li>• Sudden thrombocytopenia&gt;50%</li> <li>• High ESR &gt; 71mm/hour</li> </ul>

The reliability of these criteria has been questioned and other schemes have been proposed based on the involvement of respiratory system alone (Table 1). So now recently semi quantitative measures to

diagnose FES has been established, proposed by Lindeque et al, in which a score of more than five is required for positive diagnosis of FES (Table 2).

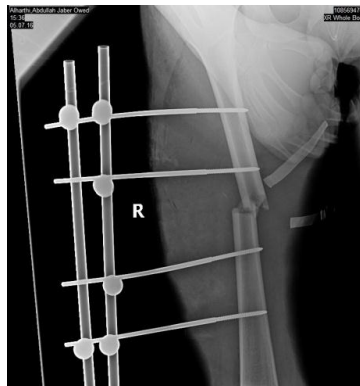
**Table 2 Lindeque's criteria**

<ul style="list-style-type: none"> <li>• A sustained PaO<sub>2</sub> &gt;8 kPa (60 mmHg)</li> <li>• A sustained PaCo<sub>2</sub> &gt; 7.3 kPa (55 mmHg) or pH &lt;7.3</li> <li>• A sustained respiratory rate &gt;35/minutes even after adequate sedation</li> <li>• Increased work of breathing judged by dyspnea, use of accessory muscles, tachycardia and anxiety</li> </ul>
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**Case report**

General surgery Department, King Faisal Medical Complex, Taif, Saudi Arabia had received a 23 year - old male was injured in a car crash. He was admitted with open fracture right femur (Fig.1). The right leg

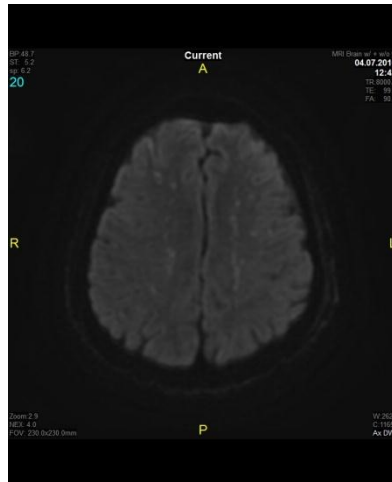
was immobilized with skin traction. The patient was fully conscious and CT brain at time of admission was normal. Respiratory and hemodynamic status was stable.



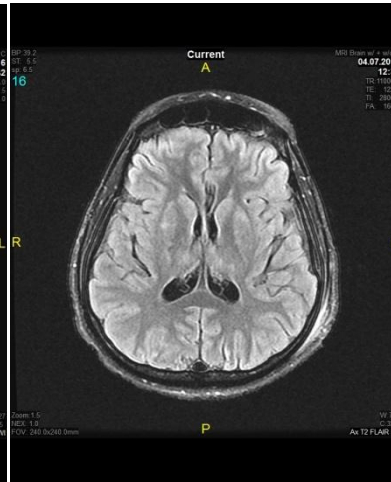
**Fig.1**

Next morning, 18 hours after the accident the conscious level of patient, suddenly deteriorated. He became restless, confused, and unresponsive to verbal stimuli. He developed tachypnea and tachycardia, but there was no focal neurological deficit. The patient was put on mechanical ventilation. CT brain repeated and was normal. CT chest showed bilateral lung contusion/consolidation. CT abdomen was normal. The same day later MRI brain done and showed Multiple minute foci of restricted diffusion, low in ADC were seen in deep periventricular white matter, some are of cortical distribution, basal ganglia

Glasgow coma scale was 9 of 15 (eye opening 4, motor response 2, verbal response 3). Blood gas analysis revealed hypoxemia PaO<sub>2</sub> 65 mmHg and PaCO<sub>2</sub> 33mmHg. He was shifted to intensive care unit, where endotracheal tube was passed and few are in cerebellar hemisphere suggestive of fat embolic acute cerebral infarctions. Fig.2 shows several lesions of high signal intensity, indicating areas of restricted diffusion due to cytotoxic edema. Fig 3. shows multiple punctiform hyperintense lesions in white matter of both cerebral hemispheres



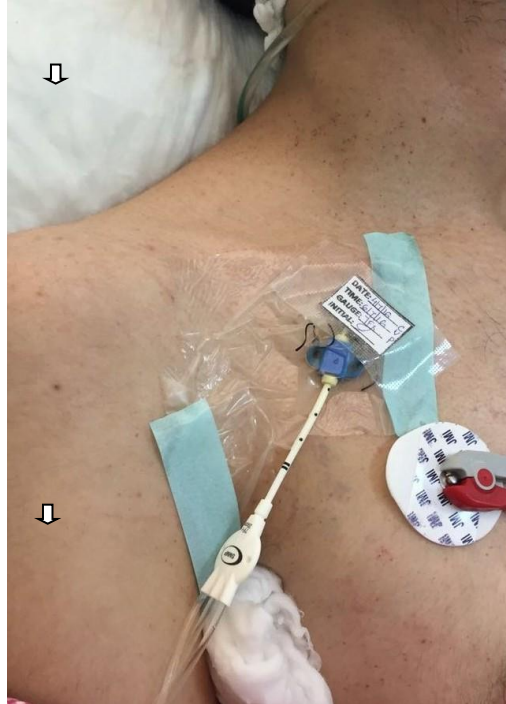
**Fig.2 DW-MRI**



**Fig.3 Axial T2-weighted MRI**

The diagnosis of fat embolism syndrome proposed. On second day after admission in ICU a petechial rash was noted on anterior chest wall and axillary region. Fig.4 on day 3 fracture right femur stabilized with external fixator and later on day 8 his fracture right femur fixed with open reduction and internal fixation. Patient's Glasgow coma scale improved over time but unable to breath

spontaneously and maintain normal Spo<sub>2</sub>, due ARDS. After 7th day tracheostomy done for patient. Later in due course he was weaned off from mechanical ventilation and put on T-piece and later on day 12 his tracheostomy tube removed and patient was shifted to ward on normal diet and physiotherapy started for patient. Repeat MRI after day 15 showed



**Fig.4 Petechial rash on neck chest and axilla**

## **DISCUSSION**

Fat embolism is most commonly associated with skeletal injury and most likely to occur in patients with multiple long bone and pelvis fractures. Some other traumatic causes include: rib fractures, massive soft tissue injury, severe burns, bone marrow biopsy, and liposuction. More rarely, fat embolism is also associated with some non-traumatic disorders, such as pancreatitis, diabetes, diabetes mellitus, and high-dose steroid therapy<sup>7,8,11</sup>.

Additionally, a few related studies report that the factors that increase the risk of FES development are: young age, closed fractures, multiple fractures, and conservative therapy of long-bone fractures<sup>10,12</sup>. Overall mortality for FES is estimated at 5%-15% and up to 36% in patients who require mechanical ventilation<sup>9,14</sup>.

For the development of FES, a mechanical theory and a biochemical theory have been proposed. According to the mechanical theory, FES occurs when large fat globules enter the venous circulation resulting in the

obstruction of the pulmonary vascular system. However, this theory cannot substantiate the delay in the development of symptoms<sup>15</sup>.

The biochemical theory suggests that hormonal changes after extensive trauma induce hydrolysis of triglycerides and release of free fatty acids, causing toxic endothelium damage in pulmonary capillary beds, as well as ARDS in animal models<sup>16</sup>. In this theory, the time required to produce these toxic intermediaries explains the delay in development of symptoms. Despite the large number of studies supporting the involvement of these mechanisms in the development of FES, evidence is considered circumstantial<sup>10,12,14</sup>.

Among the reasons for difficulty in diagnosis of FES is the complication of widely different clinical conditions that may vary in severity. Diagnosis is established on the basis of patients' clinical condition and symptoms, using the process of exclusion for other possible causes. The most useful examination in diagnosing FES includes imaging studies such as chest radiography,

CT scans, pulmonary ventilation/perfusion scans, and cerebral magnetic resonance imaging, as well as cardiac investigations so as to exclude cardiac causes. In this case the, the diagnosis of FES was prompted on the basis neurological symptoms, hypoxemia, tachycardia, petechial rash, and MRI brain findings of FES, with no evidence of brain injury, sepsis, cardiogenic pulmonary edema or any cause of ARDS. The patient had three major and one minor criteria of Gurd and Wilson, classification to establish the diagnosis of FES<sup>16</sup>.

The treatment of fat embolism is only supportive and includes maintenance of adequate oxygenation, stable hemodynamic, normal blood levels, hydration, prevention of deep venous thrombosis and gastrointestinal bleeding and nutrition<sup>17</sup>.

The purpose of medication is to reduce morbidity and prevent complications. High-dose corticosteroids have been effective in preventing the development of FES in several studies, their use as prophylaxis remains controversial<sup>18</sup>. Albumin has also been recommended because it not only restores blood levels but also combines the fatty acids that may limit lung and brain injury<sup>19</sup>.

The timing and type of surgery for fractures constitute modifiable factor for the development of FES. Studies have revealed that after traumatic injury, early surgical fixation in patients with isolated femoral fractures could prevent the development of FES. In our case, surgical stabilization was delayed more than 12 hours, till symptoms and signs of FES developed.

On the other hand, a number of studies have pointed out that surgical orthopedic treatment, especially intramedullary nailing is associated with higher probability of fat embolism and pulmonary and neurological complications due to release of fat emboli from the bone marrow of medullary canal<sup>20</sup>.

In summary, there is no specific therapy for FES; prevention, early diagnosis, and adequate symptomatic treatment are very important. Most of studies in the last 20

years have shown that the incidence of FES is reduced by early stabilization of the fracture and the risk is even further decreased with surgical correction rather than conservative management.<sup>21</sup> In the present study delay in surgical stabilization of right fracture femur was assumed to be most significant responsible for the development of FES.

## CONCLUSION

The diagnosis of FES may be complex because there are no pathognomonic signs (except for petechiae). Early suspicion combined with chest radiography and MRI brain is the key answer to diagnosis. Fracture of long bones should be approached for damage management with principle of orthopedics in order to eliminate potential complications. As the literature to date is limited, further research is needed to investigate the controversial relationship between femoral fractures and FES.

## Consent

The study was approved by the Ethics Board of King Faisal University, written informed consent was obtained from the patient and relatives for subjecting to the research and publication of this case report and all accompanying images.

## Author, contributions

All authors contributed equally to the preparation of the manuscript

## Disclosure

The author reports no conflicts of interest in this work

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