

The Art of Diagnosis of Fat Embolism Syndrome, Treatment and Prognosis – Case Report

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Abstract

Introduction: Fat embolism syndrome (FES) is a rare complication of long-bone fractures caused by the entry of bone marrow fat into the circulation. It mainly affects the lungs and central nervous system and may be difficult to distinguish from other post-traumatic complications.

Case presentation: A 24-year-old male sustained a femoral fracture in a road traffic accident and underwent intramedullary fixation 12 hours after admission. Twelve hours after surgery, he developed tachypnea, tachycardia, hypotension, agitation, confusion, and disorientation. He was transferred to the Intensive Care Unit, intubated, and mechanically ventilated, with hemodynamic support and carefully monitored fluid therapy. Chest X-ray and CT showed bilateral pulmonary changes. Pulmonary contusion, pulmonary thromboembolism, and ARDS were considered in the differential diagnosis. Based on the trauma history, timing of deterioration, clinical presentation, and imaging findings, FES was diagnosed. After two weeks of supportive treatment, the patient improved, was weaned from mechanical ventilation, and transferred to the ward in stable condition. Differential diagnosis was considered pulmonary contusion, pulmonary thromboembolism and ARDS. Key points were mechanism of trauma, patients' history data, clinical and radiological examinations, so the diagnosis of fat embolism syndrome was established.

Conclusion: Early recognition of FES and prompt supportive treatment are essential for a favorable outcome. Respiratory and hemodynamic support, careful fluid management, and timely fracture stabilization remain the main therapeutic measures. Because FES may resemble other post-traumatic conditions, awareness of its characteristic clinical course is important for timely diagnosis and treatment.

Keywords: Fat embolism syndrome; Femoral fracture; Intramedullary fixation; Pulmonary contusion; Pulmonary thromboembolism; ARDS

Introduction

Fat Embolism Syndrome (FES) is a clinical condition caused by the entry of fat droplets, usually originating from bone marrow after long-bone or pelvic fractures, into the circulation. It combines microvascular obstruction with a systemic inflammatory response, predominantly affecting the lungs, brain,

and skin [1,2]. Fat embolism (FE), which is often asymptomatic, should be distinguished from FES, in which clinically develops significant organ dysfunction [1,3,4]. FES is most commonly associated with femoral and other long-bone fractures, multiple or pelvic fractures, and orthopedic procedures, particularly intramedullary fixation [1,3,5]. Its pathogenesis involves both mechanical embolization of marrow fat and a subsequent

biochemical inflammatory response, resulting in endothelial injury, increased capillary permeability, pulmonary edema, and hypoxemia [2-5]. Clinical manifestations usually develop after a latent period of 24–72 hours and classically include respiratory and neurological deterioration accompanied by petechial rash [1,3-5]. Additional findings may include fever, tachycardia, thrombocytopenia, anemia, coagulation abnormalities, and, in severe cases, multiorgan dysfunction [4,5]. Diagnosis is primarily clinical, as no single laboratory or imaging test confirms FES [3,4]. The most commonly used diagnostic systems are the Gurd and Wilson criteria, Schonfeld score, and Lindeque criteria, although none represents a definitive gold standard [3-5]. Laboratory abnormalities may include hypoxemia, an increased alveolar–arterial oxygen gradient, thrombocytopenia, anemia, leukocytosis, elevated CRP, and coagulation abnormalities [4,5]. Fat droplets in blood or urine are not considered reliable diagnostic evidence [4,5].

Chest radiography may demonstrate bilateral diffuse infiltrates (“snowstorm” appearance”), while CT can reveal ground-glass opacities, septal thickening, and centrilobular nodules [6,7]. In cerebral FES, brain MRI with DWI (Diffusion-Weighted Imaging) may demonstrate the characteristic “starfield pattern” [8,9]. Treatment is predominantly supportive and focuses on maintaining adequate oxygenation and ventilation, hemodynamic stabilization, correction of metabolic disturbances, and continuous monitoring [1,3-5]. Early fracture stabilization is an important preventive measure, while the role of corticosteroids remains controversial [1,3-5]. The differential diagnosis of FES is challenging because its clinical and radiological manifestations overlap with other causes of acute respiratory deterioration after trauma. Pulmonary contusion should be considered, particularly after major chest trauma, as it may present with hypoxemia and bilateral pulmonary opacities on imaging [10-12]. Pulmonary thromboembolism is another important differential diagnosis, especially in immobilized or severely injured patients. Unlike FES, it is caused by thrombotic obstruction of the pulmonary vasculature and is primarily evaluated with CTPA – computed tomography pulmonary angiography [13-15]. Acute respiratory distress syndrome (ARDS) may also resemble FES because both can cause severe hypoxemia and bilateral pulmonary infiltrates; however, ARDS represents a syndrome of acute inflammatory lung injury with specific diagnostic criteria and may develop secondary to trauma or other systemic insults [16-18]. The timing of symptoms, mechanism of injury, associated neurological or petechial manifestations, and imaging findings are therefore essential for distinguishing FES from these conditions [1-7].

Case Report

A 24-year-old patient was admitted to the Clinic for Traumatology, Orthopedics, Anesthesia, Resuscitation and

Intensive Care and Emergency Medicine with a fracture of the femur, sustained after a traffic accident as a driver (Figure 1). The patient was conscious, contactable, oriented in time and space. The conducted clinical, laboratory and radiological examinations showed a reduction of erythrocytes in the blood count and severe pain when attempting to move the injured limb, which was relieved with continuous analgesia. Radiographic examinations were normal, except for the finding of a fracture of the left femur.



Figure 1: Femoral fracture.

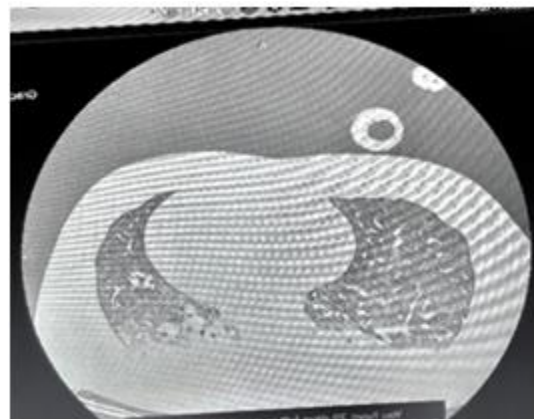


Figure 2: CT of the chest – bilateral ground-glass opacities, septal thickening and centrilobular nodules.



Figure 3: Bedside chest radiography - “snowstorm” appearance.

The patient was operated 12 hours after admission, by implanting an intramedullary nail in the femur. Twelve hours after the end of the intervention, the patient worsened with the appearance of tachypnea, dyspnea, hypoxemia, tachycardia, drop of the blood pressure, mental disorientation, confusion, restlessness and drowsiness. He was immediately transferred to the Intensive Care Unit, where he was intubated and placed on mechanical ventilation. He was hemodynamically stabilized with vasopressors and careful fluid administration to maintain good perfusion of all organs and maintain diuresis. After stabilization of the patient, a CT scan was performed, which showed findings only in the lungs in the form of bilateral consolidations and bilateral diffuse nodular consolidations described as bilateral ground-glass opacities, septal thickening and centrilobular nodules (Figure 2). Bedside radiography was also performed, which findings were described as “snowstorm” appearance” (Figure 3).

Continuous monitoring was performed on the patient. After two weeks of treatment, the patient was stabilized without the need for vasopressor support and was released from mechanical ventilation. The radiographic findings were significantly improved and the patient was transferred to a ward in good general condition. When establishing the diagnosis, fat embolism syndrome, lung contusion, pulmonary thromboembolism and ARDS were considered. Pulmonary contusion was considered as part of the differential diagnosis based on the traumatic mechanism and radiological findings [10-12]. Acute pulmonary embolism was also considered in the differential diagnosis [13-15], while ARDS was considered because of the severe hypoxemia and bilateral pulmonary abnormalities [16-18]. Based on the data on the mechanism of trauma, the clinical examination of the patient, and the time and intensity of the onset of symptoms, a diagnosis of FES was established. The diagnosis was supported by the results of radiography and computed tomography of the lungs.

Discussion

Fat embolism syndrome (FES) is a rare but potentially life-threatening complication most commonly associated with long-bone fractures, particularly fractures of the femur and pelvis, as well as orthopedic surgical procedures. Although fat embolization occurs frequently after long-bone fractures, only a small proportion of patients develop clinically manifest FES with organ dysfunction [1-5]. Current evidence supports a multifactorial pathophysiology involving both mechanical obstruction of the microcirculation and biochemical mechanisms, including endothelial injury and inflammation induced by free fatty acids and other lipid mediators [2,3,5]. In the presented case, the femoral fracture represents the major predisposing factor.

Following long-bone fracture, displacement of fracture fragments and manipulation of the medullary cavity may facilitate the entry of bone marrow fat into the venous circulation. The subsequent inflammatory response and disruption of the alveolar-capillary barrier can result in systemic involvement, predominantly affecting the lungs, brain, and skin [1-3].

Clinical Presentation

The classic clinical triad of FES consists of respiratory dysfunction, neurological abnormalities, and petechial rash [1-5]. However, the complete triad is not present in all patients, and the absence of petechiae should not be used to exclude the diagnosis [3,4]. In the present case, the temporal relationship between femoral fracture and the subsequent development of neurological and respiratory dysfunction strongly supports FES. The patient developed confusion and drowsiness, followed by tachypnea and hypoxemia, with subsequent tachycardia and hypotension. This combination of neurological and severe respiratory dysfunction in the setting of a femoral fracture is highly suggestive of FES [2-5]. Pulmonary involvement is the most common manifestation and ranges from mild hypoxemia and tachypnea to severe acute respiratory failure, including ARDS requiring mechanical ventilation [3,5,6]. Qi et al. reported dyspnea, neurological abnormalities, and acute respiratory failure among the most frequent manifestations of pulmonary FES, findings that are consistent with the present case [6].

Neurological involvement may range from confusion, restlessness, and drowsiness to seizures, profound impairment of consciousness, and coma [3,8,9]. Cerebral involvement is typically diffuse rather than characterized by focal neurological deficits. In this patient, the combination of altered mental status and acute hypoxemia supports a neurological component of FES. Petechial rash is a characteristic but non-mandatory manifestation and may be transient, most commonly involving the upper body, conjunctivae, and axillary regions [1,3,5]. Its absence in the present case does not substantially reduce the likelihood of FES given the characteristic mechanism of injury, clinical course, hypoxemia, and neurological dysfunction.

Differential diagnosis

Because the clinical and radiological findings of FES are nonspecific, several alternative causes of acute hypoxemic respiratory failure should be considered in the traumatized patient, particularly pulmonary contusion, acute pulmonary thromboembolism (PE), and ARDS. Pulmonary contusion is a direct traumatic injury to the lung parenchyma that may cause hypoxemia, tachypnea, and bilateral or multifocal pulmonary opacities [10-12]. Radiological abnormalities may progress during the first hours after trauma [10]. However, no significant primary thoracic trauma was described in the present patient.

Furthermore, pulmonary contusion would not adequately explain the associated neurological dysfunction. Therefore, it is unlikely to account for the overall clinical presentation.

Acute PE is an important differential diagnosis in trauma and postoperative patients because of the increased risk of venous thromboembolism. PE may present with dyspnea, tachycardia, hypoxemia, and, in severe cases, hypotension or shock [13]. Unlike FES, PE results from thrombotic occlusion of the pulmonary vasculature, with CTPA typically demonstrating an intraluminal filling defect [13-15]. In FES, diffuse pulmonary parenchymal abnormalities may predominate without a typical thrombotic filling defect [6,13]. Nevertheless, the absence of a filling defect alone does not establish FES; the diagnosis should be based on the overall clinical context. In this patient, the combination of femoral fracture, neurological dysfunction, hypoxemia, and bilateral pulmonary abnormalities favors FES over isolated PE.

ARDS represents another important consideration. According to the Berlin definition, ARDS is characterized by acute onset, bilateral pulmonary opacities, and hypoxemia that cannot be fully explained by cardiac failure or fluid overload [16,17]. The 2024 global definition expanded the diagnostic framework by allowing broader use of pulse oximetry and high-flow nasal oxygen and by incorporating lung ultrasound as an imaging modality [18]. In the present case, severe hypoxemia and bilateral pulmonary opacities may fulfill criteria for ARDS depending on the $\text{PaO}_2/\text{FiO}_2$ or $\text{SpO}_2/\text{FiO}_2$ ratio and the respiratory support provided. However, ARDS describes a pattern of acute lung injury, whereas FES may represent the underlying causal syndrome. Therefore, the formulation “FES with severe acute lung injury/ARDS” may be more appropriate than considering FES and ARDS as mutually exclusive diagnoses.

Treatment

There is no specific pharmacological treatment with sufficient evidence to be routinely recommended for established FES. Management is primarily supportive and focuses on maintaining adequate oxygenation, ventilation, hemodynamic stability, and organ perfusion [1-5]. In severe cases, invasive mechanical ventilation may be required, as in the present patient. Lung-protective ventilation strategies should be applied when ARDS is present, while prone positioning and, in selected patients with refractory hypoxemia, extracorporeal membrane oxygenation (ECMO) may be considered [3,18]. Hemodynamic instability should be managed with appropriate assessment of volume status, cautious fluid resuscitation, and vasopressor support when required, while avoiding unnecessary fluid overload.

The role of corticosteroids remains controversial. Although some evidence suggests a possible prophylactic benefit in high-risk patients, evidence supporting their routine use for established FES

remains inconsistent. Miyake et al. emphasized this uncertainty and the need for further clinical studies [3]. Consequently, corticosteroids should not currently be considered standard specific therapy for established FES. Anticoagulation is likewise not a specific treatment for FES. It should be used when venous thromboembolism or PE is proven or strongly suspected, but fat embolization itself should not automatically be managed as a thromboembolic event [3,5,13].

Early fracture stabilization and prevention

Early stabilization of long-bone fractures, when permitted by the patient's overall condition, remains an important preventive strategy [2,3,5]. Early fixation may reduce continued mobilization of the fracture site and further release of marrow fat into the circulation. In the present case, surgical treatment of the femoral fracture was performed 12 hours after admission, representing relatively early stabilization. Nevertheless, FES developed despite timely fixation, emphasizing that early stabilization reduces but does not completely eliminate the risk. Current evidence supports early stabilization, particularly within the first 24 hours when the patient's clinical condition permits [3].

Assessment of the diagnosis

Based on the available clinical and imaging findings, FES represents the most likely diagnosis in the present patient. The diagnosis is supported by the strong temporal and pathophysiological association with the femoral fracture, the development of respiratory and neurological dysfunction, hemodynamic instability, and characteristic bilateral pulmonary CT abnormalities.

The combination of hypoxemia, neurological dysfunction, and bilateral ground-glass, nodular, and consolidative pulmonary changes in the setting of a long-bone fracture is consistent with contemporary descriptions of FES [3,6]. Importantly, CT findings alone are not diagnostic of FES. Rather, imaging should be interpreted as part of an integrated clinical assessment and is particularly valuable for excluding or differentiating other causes of acute respiratory failure, including PE, pulmonary contusion, and other forms of acute lung injury.

Conclusion

Fat Embolism Syndrome is an uncommon but potentially life-threatening complication of long-bone and pelvic fractures. Early recognition of its characteristic clinical manifestations, prompt exclusion of important differential diagnoses, and timely supportive management are essential to minimize complications and improve patient outcomes. Early fracture stabilization remains an important preventive strategy.

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