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# Fat Embolism Syndrome in a Pediatric Trauma Patient: A Rare but Life-Threatening Complication of Long Bone Fracture

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## Abstract

Fat embolism syndrome (FES) is a set of systemic manifestations generally occurring following trauma to a long bone. In children there is little data in the literature because it is a less common pathology than in adults. It is a syndrome which poses a diagnostic problem and whose management is only symptomatic, and where prevention plays the most important role. It is a serious complication which can jeopardize the functional or vital prognosis. Our work is an observation of a 15-year-old girl who presented with FES following a femur trauma whose stabilization was delayed. The aim of this work is to show the benefit of knowing how to make this diagnosis in children when faced with suggestive signs, especially in context of trauma.

## Keywords

Fat embolism, child, trauma

## Main Article

### Introduction

Fat Embolism Syndrome is a serious complication often associated with long bone fractures. It encompasses a polymorphic and nonspecific set of clinical, biological, and radiological signs related to the dissemination of fat particles in the body [1]. Its diagnosis remains controversial due to the polymorphism of clinical signs and the frequent association with concomitant trauma. The Gurd criteria are still the most widely used for the positive diagnosis of this syndrome [2]. The aim of this work is to demonstrate the importance of diagnosing this condition in children when faced with suggestive signs, especially in context of trauma.

### Case history:

This is a 15-year-old patient with no previous medical history, admitted to the emergency department two hours after a fall from the fourth floor. The initial examination found a

conscious patient, hemodynamically stable (BP: 120/70) and respiratory stable (SpO2: 99% in ambient air), with a soft abdomen and deformity of the lower right limb. A whole-body CT scan revealed a complex fracture of the left mandibular arch, minimal bilateral pneumothorax, and pneumomediastinum (Figure 1: initial chest CT showing minimal bilateral pneumothorax and pneumomediastinum).The X-ray of the deformed limb showed a displaced fracture of the right femoral diaphysis (Figure 2: frontal X-ray of the thigh showing a diaphyseal fracture of the right femur). The patient was hospitalized in the surgical department, and surgical indication was established for the femoral and facial fractures; the surgical procedure was postponed to day 4 post-trauma due to logistical issues. On the morning of the procedure, the patient presented fever (39°), tachycardia (FC 122 bpm) and respiratory distress (respiratory rate at 26 c/min, SpO2: 70% in ambient air, PaO2 58 mmhg), with transient confusional episodes, and she was oliguric, but no signs of petechiae in conjunctiva, in oral mucosa or in axillary and cervical regions. The brain CT and metabolic assessment were normal; however, the thoracic angiogram did not show pulmonary embolism, but the parenchymal window of the thoracic CT revealed interstitial opacities suggestive of fat embolism (Figure 3: thoracic CT showing bilateral interstitial pulmonary opacity). The blood count showed anemia at 6 g/dl but without thrombocytopenia, and a normal creatinine level. The patient was admitted to the operating room on the same day for osteosynthesis of the facial and femoral fractures. Subsequently, the patient was transferred to intensive care intubated. The course was marked by the onset of an infectious pneumonia, treated with antibiotics, with significant improvement followed by extubation on day 6 post-op. With improvement in respiratory and neurological status, the patient was transferred back to the surgical department.

## Discussion

This syndrome is not very rare among patients with long bone fractures. The femur is the most common site (accounting for 60% to 92% of Fat Embolism Syndrome cases), as is the case in our study, followed by the tibia [2]. It is particularly frequent in young males [1]. However, it is generally considered rare in children, and little is known about its incidence [3]. This complication is favored by the presence of visceral injuries, the extent of bone fracture displacement, delays in surgical intervention, and the persistence of hypovolemia (leading to the passage of fat emboli into circulation due to increased pressure gradient between the medullary cavity and the venous sinuses) [7,8]. Classically, there is an initial

asymptomatic period lasting 48 to 72 hours until signs appear [3]. In our patient, the free interval was 3 days. Typically, respiratory symptoms arise first, presenting as tachypnea, dyspnea, and hypoxemia, which can be severe in half of the cases and may require mechanical ventilation, as was the case with our patient. The thoracic CT scan typically shows an alveolo-interstitial syndrome [2, 4]. The incidence of neurological manifestations varies between 59% and 76%; these symptoms are nonspecific and can range from simple agitation to a comatose state. Brain CT is often normal aside from any associated traumatic lesions [2], as was the case with our patient. However, MRI is relevant in diagnosing Fat Embolism Syndrome, highlighting diffuse abnormalities, appearing as low signal on T1-weighted sequences and high signal on T2-weighted sequences [2]. Petechial purpura, considered specific in this syndrome but inconstant, involves the conjunctiva, oral mucosa, and axillary and cervical regions [2]. Fundoscopic examination may reveal dysoric nodules (60%), pinpoint hemorrhages or flame-shaped lesions (30%), and macular edema (30%), with these abnormalities persisting for 10 to 15 days [9]. Biologically, thrombocytopenia is very common and may sometimes be part of a disseminated intravascular coagulation picture. Hemolytic anemia is also frequent, but no specific biological test exists. The search for intracellular lipid particles in tracheal samples or bronchoalveolar lavage is neither sensitive nor specific [2]. Gurd proposed diagnostic criteria in 1970 that are still in use today; they include major and minor criteria. The diagnosis of Fat Embolism Syndrome is made when a major criterion is associated with four minor criteria, along with the presence of macroglobulinemia (Table 1). Most authors have abandoned this last criterion since fat globules can be found in both traumatized individuals and healthy subjects [2,5]. Fat Embolism Syndrome remains primarily a diagnosis of exclusion [2]. The treatment of Fat Embolism Syndrome is primarily prophylactic and relies on the early stabilization of fracture sites. Once it occurs, treatment is purely symptomatic [1]. Several specific treatments have been proposed (heparin, dextran, corticosteroids), but none have proven effective to date [1]. Fat Embolism Syndrome is considered severe because it is responsible for 5% to 20% of cases [6]. The final prognosis depends on pulmonary and neurological involvement, but fortunately, advancements in resuscitation have reduced mortality from severe forms [2]. Recovery occurs within about fifteen days. However, sequelae, particularly neurological and psychiatric (such as seizures, deficits, etc.), may persist [2]. In our case, the patient was declared cured on the 9th day, which aligns relatively well with the literature.

## Conclusion

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Certainly, fat embolism syndrome is rare in children compared to adults, but it remains a very serious complication and can be fatal. This is why it is essential to make this diagnosis even in pediatrics when faced with the onset of respiratory distress and/or unexplained neurological signs, especially in a traumatic context. It is a diagnosis of exclusion based on a collection of evidence, and its management is primarily preventive, focusing on the rapid stabilization of long bone fractures.

## Figures:

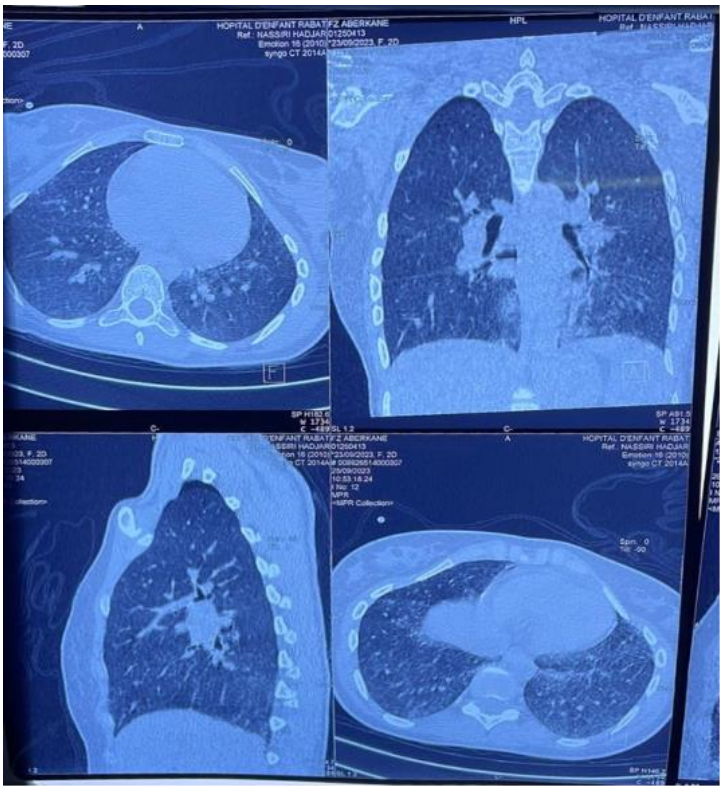

*Figure 1: initial chest CT showing minimal bilateral pneumothorax and pneumomediastinum*

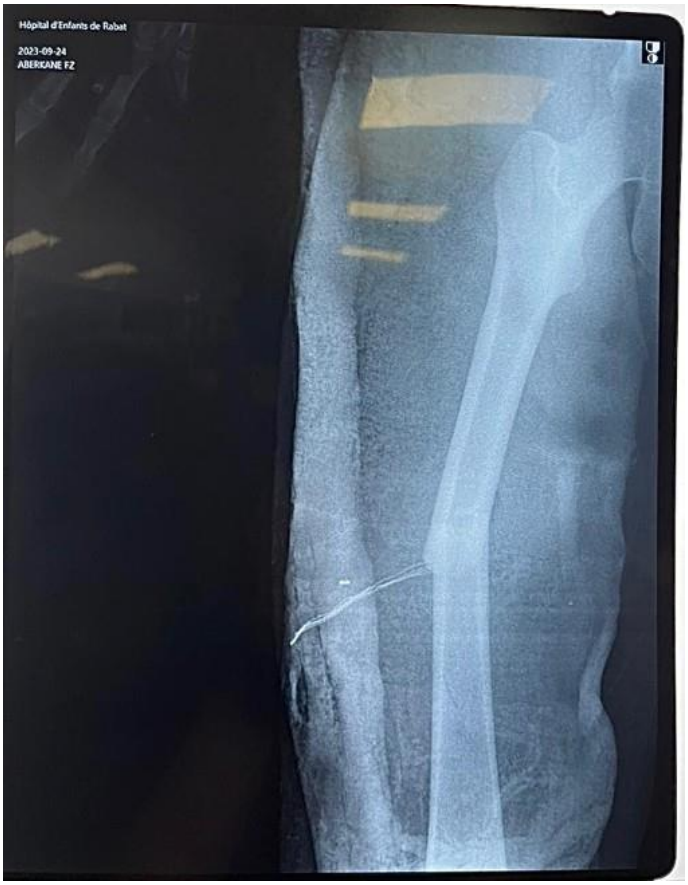


*Figure 2: frontal X-ray of the thigh showing a diaphyseal fracture of the right femur*

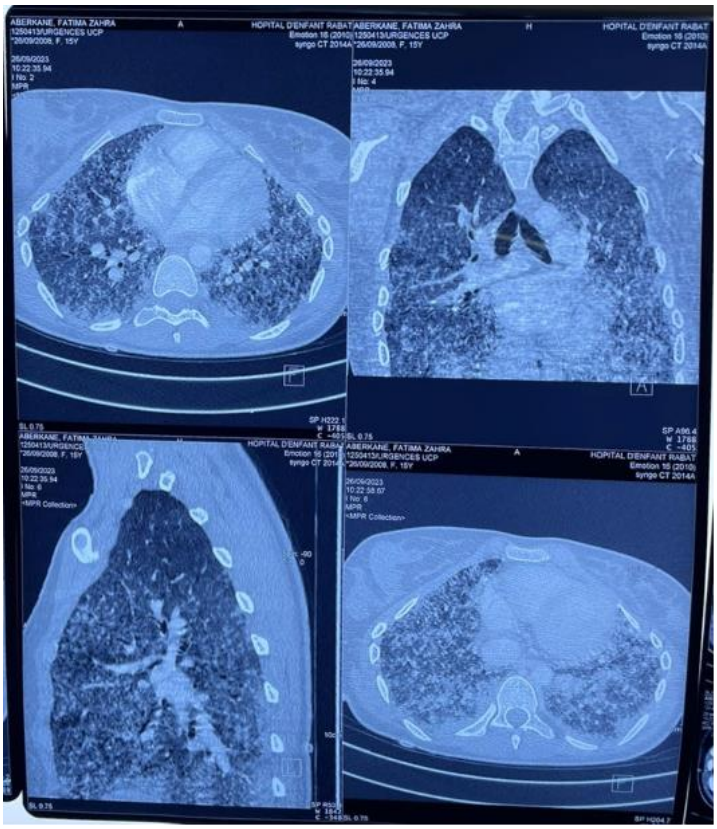


*Figure 3: thoracic CT showing bilateral interstitial pulmonary opacity*

## Tables :

*Table 1 : Gurd's criteria.*

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|---|---|
| Major criteria | Petechial rash |
| | Respiratory insufficiency |
| | Cerebral involvement |
| Minor criteria | Tachycardia |
| | Fever |
| | Retinal changes |
| | Jaundice |
| | Renal signs |
| | Thrombocytopenia |
| | Anaemia |
| | High ESR |
| | Fat macroglobinemia |

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