



FATAL FULMINANT FAT EMBOLISM SYNDROME: A CASE REPORT

Ghannam Abdelaziz*, Krimech Mehdi Omar, Mekaoui Jalal, Boufettal Moncef¹, Bassir Reda Allah¹, Kharmaz Mohamed¹, Lamrani Molay Omar¹, Berrada Mohamed Salah¹

Department of Orthopaedic Traumatology, CHU Ibn Sina, Faculty of Medicine and Pharmacy of Rabat, Mohamed V University, Rabat, Morocco.

*Corresponding Author: Ghannam Abdelaziz

Department of Orthopaedic Traumatology, CHU Ibn Sina, Faculty of Medicine and Pharmacy of Rabat, Mohamed V University, Rabat, Morocco.

Article Received on 25/11/2021

Article Revised on 16/12/2021

Article Accepted on 06/01/2022

ABSTRACT

Fat embolism syndrome (FES) is a disorder of several organs with potentially serious consequences; he is generally considered to have multiple trauma that follows, including several long bone fractures. The main clinical features of FES include hypoxia, pulmonary dysfunction, changes in mental status, petechiae, tachycardia, fever, thrombocytopenia, and anemia. We report a dramatic and fatal case of a 23-year-old woman who continued to develop FES six hours after nail fixation intramedullary early fractures of the femur and tibia, despite early diagnosis and aggressive supportive treatment.

KEYWORDS: Fat embolism syndrome, femoral stem fracture, intramedullary nailing, fulminant, petechiae.

INTRODUCTION

First described clinically by von Bergmann in 1873^[1], fat embolism syndrome (FES) is still somewhat enigmatic. The complex pathogenesis of FES appears to involve both mechanical (rupture of wounds, soft tissue) and biochemical factors (activation of plasma lipase, phospholipase A2) leading to destabilization of fat circulation and influx of fat. fat in the lungs.^[2,3] Progressive respiratory failure, petechiae and altered mental state, progressive development within 72 hrs 12 hrs after the accident, are the main clinical manifestations of FES.^[3] All of these manifestations can be masked by associated injuries, particularly in multiple injured patients^[4], thus making diagnosis and treatment a real challenge.

CASE REPORT

A 23-year-old woman, weighing 66 kg, was admitted to the emergency room 30 minutes after being involved in a car accident (pedestrian hit in car). She has no remarkable past medical history. The initial clinical examination found a patient well oriented, capable of conversation with a Glasgow score (GCS) of 15 and hemodynamically stable (initial BP 120/60 mmHg, heart rate: 120 beats / min). Closed fractures of the left femoral shaft and right tibial shaft were found (figures). With the exception of a superficial injury to the forehead, no signs of head injury were detected. Evaluation of the abdomen, thorax, and pelvis (including chest and pelvis x-ray) revealed no signs of injury.

Fractures were temporarily immobilized by traction for a fracture of the femur and left tibia splints, and the patient was transferred to the orthopedic surgery department of Ibn Sina hospital where, 6 hours after the trauma, she underwent nail surgery intramedullary of both femur and tibia under general anesthesia. Both femur and tibia were sequentially reamed using flexible 10.5mm reamers, then 9mm diameter antegrade nails with proximal and distal locking screws were placed. (Figure 1.2) During the surgery, the patient was stable, the blood loss was estimated to be approximately 250 ml, and the total running time was approximately three hours.

Immediate postoperative period was uneventful, then 6 hours postoperatively the polypnea patient presented with 94% SpO₂ in room air, low fever (38.3°), tachycardia at 105 beats / min and neurologic impairment with a GCS 10. At the start auscultation, cerebral CT and X-ray were all normal.

The patient was transferred to the intensive care unit (ICU) for further care and investigations where she was intubated due to respiratory deterioration. arterial blood gas analysis revealed a pH of 7.3, a PaO₂ of 100 mm Hg (FIO₂ 1.0) and a PaCO₂ of 45 mm Hg, HCO₃: 23. cardiac examinations and transthoracic lung ultrasound were performed and ruled out pneumothorax and showed neither evidence of an intracardiac shunt, nor any direct sign of pulmonary embolism. 7 hours after admission to intensive care, the patient presented with patent conjunctival and thoracic petechiae which later resolved

spontaneously. All other lab results show almost intact thrombocytopenia, but 105,000 / mm.

The clinical condition of the patient deteriorated rapidly; at the neurological level the GCS went to 8 and at the respiratory level of the Pao₂ / Fio₂ went to 80 on the third day. Later showed X-ray lung of the chest infiltrated with cerebral interstitial syndrome CT revealed cerebral edema and CT angiography of the chest showed bilateral condensation images. Although the clinical pulmonary infection (PCIE) scores were 5, the patient was put on empiric antibiotic therapy (Tienam, Amiklin) with sessions of chest ventilation.

On the fifth day, hemodynamics and respiratory failure developed associated with acute renal failure (creatinine clearance at 33) for which the patient was hemodialysis. Thrombocytopenia at 50,000 / mm, anemia at 6.9g / dl, prothrombin time at 50% and signs of lower limb hypoperfusion also developed.

Despite maximum supportive care, including the transfusion of 4 units of red blood cells and 3 units of fresh frozen, ventilatory and inotropic plasma, the patient died on the sixth day after his injury, in a sever setting of distress syndrome. acute respiratory (ARDS) associated with disseminated intravascular coagulation (DIC).

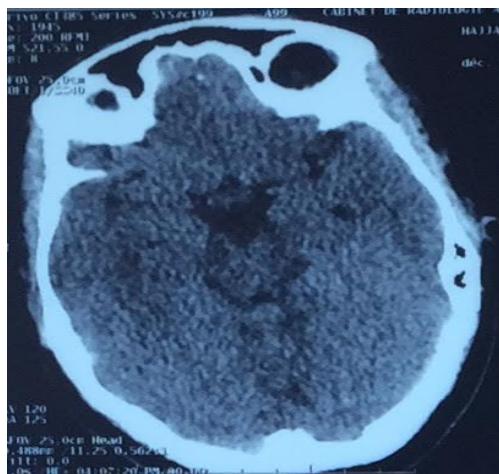


Figure 2: Diffuse brain CT showing hypodense changes in cerebral white matter related to.



Figure 1: Chest x-ray showing diffuse bilateral pulmonary infiltrates 10 hours after surgery.

DISCUSSION

Fat embolism syndrome (FES) is a serious complication most commonly observed following polytrauma, including multiple long bone fractures.^[4,5] In patients with long bone fracture alone, the frequency of FES has been reported to be around 3%, while it reaches almost 30% in the case of long and multiple bones/or pelvic fractures.^[5] The complex pathogenesis of FES appears to involve both mechanical (fracture, soft tissue injury) and biochemical factors (activation of plasma lipase, phospholipase A2) leading to destabilization of fat circulation and influx. fat to the lungs causing ventilation-perfusion mismatch and acute posterior acute respiratory distress syndrome (ARDS).^[2,3]

The diagnosis of FES is primarily clinical^[6], its most common presentation is hypoxia (96 percent), which often occurs before pulmonary symptoms develop.^[7,8] Cyanosis, tachypnea, dyspnea, and hypoxemia are the major clinical manifestations of pulmonary dysfunction that occurs in 75 percent of FES patients.^[6] Hypoxemia has been previously associated with subclinical FES and it is common after long bone fracture.^[6] Although the authors reported that the incidence of critical hypoxemia is similar between a trauma patient with and without ESF^[8,9], they recommended that subclinical hypoxia should be closely monitored with continued monitoring of pulse oximetry for early detection. Initial pulmonary dysfunction can progress to respiratory failure in 10 percent of patients.^[6] Other symptoms include exhibiting changes in cognitive status (59 percent), petechiae, fever, tachycardia, thrombocytopenia and anemia.^[6,7] Our patient was diagnosed with FES after fulfilling two major and five minor criteria of Gurd and Wilson.^[6] These clinical signs included diffuse tachypnea with pulmonary infiltrates, petechiae, tachycardia, hyperthermia, sudden anemia, and thrombocytopenia.

This patient was diagnosed with Brain Fat Embolus Syndrome (CFES) after postoperative cognitive impairment (deterioration of GCS). The incidence of CFEs is 0.9 to 2.2 percent [ten]. Encephalopathy is the trademark for the diagnosis of cerebral embolism syndrome in the setting of pulmonary symptoms.^[11] Brain signs include headache, irritability, stupor, seizures, and coma. Less common findings include apraxia, hemiplegia, scotoma, anisocoria, and conjugate eye gap.^[11,12]

Although nonspecific, chest x-rays can show pulmonary images (diffusely increased snowstorm appearance) and right heart dilation.^[13,14] A high resolution chest CT scan will show bilateral or centrilobular opacities^[15]. A transthoracic ultrasound may show evidence of an intracardiac shunt, which may predispose patients to developing CFES.^[16,17] Our patient developed CFES although no evidence of intracardiac shunt was found, this could theoretically be explained by an increased pulmonary arteriovenous anastomosis that occurs during

periods of exercise and hypoxia^[14,18] Potentially creating a means for released systemic fat emboli.

Bronchoalveolar lavage may aid in the diagnosis of FES by showing neutral lipid concentration.^[19] Cerebral CT is usually normal, the first to two days after injury it may show hypodense white matter lesions which usually resolve with residual effusion and subdural cerebral atrophy.^[20] MRI imaging of the brain is the most sensitive imaging technique for diagnosing cerebral fat embolism, and shows multiple nodular hypersignals or pitted foci on T2 sequences as early as four hours after the onset of fat embolism cerebral.^[21,22]

Prevention and treatment of fat embolism syndrome relies on early fracture management, supportive care, and treatment of shock.^[6,22] Albumin has been shown to be effective for volume resuscitation by retaining fatty acids in blood volume and binding to decrease lung damage^[23] The benefits of using methylprednisolone in the prevention and treatment of FES is controversial.^[6,24] Although, a meta-analysis of seven randomized, double-blind studies and 389 patients with isolated fractures of the tibia and femur showed that corticosteroids reduced the risk of FES by 78 percent and hypoxia by 61 percent^[6,24], prophylactic corticosteroids were not administered to our patient, due to limited clinical evidence in associated fractures of the femur and tibia and unknown long-term effects.^[25-27]

Regarding fracture fixation, it was shown that there was a higher incidence of FES in patients who received delayed final intramedullary fixation^[28]. Particularly after ten hours in patients with isolated femoral fractures.^[29] Based on the theory of mechanical pathogenesis, many techniques and devices have been developed in an attempt to reduce intramedullary pressures such as the slow insertion of hollow nails, distal ventilation, narrower reamers, and the reamer irrigator system vacuum cleaner (RIA).^[22,30,31] Muller in his study concluded that most of the pressure build-up was related to the diameter of the flexible conductor, with large pressure drops ranging from a 9mm to a 7mm diameter conductor. In another comparative study of the system, Volgas compared the standard sequential reaming technique with the irrigator-aspirator (RIA) reamer^[31], And concluded that the RIA system reduces intramedullary pressure, but its expense and large size limits its widespread use in orthopedic trauma surgery. Overall, modern commonly used reamer systems have reduced the risk of systemic fat extravasation and subsequent spinal development of FES.

CONCLUSION

Fat embolism syndrome is a relatively rare clinical entity that is most commonly seen in high risk orthopedic injuries, it reflects a multisystem pathology with possible early onset after trauma and rapid development of fulminant clinical consequences. In this case, there was a dramatic development of FES with cerebral

manifestations after definitive closed tibia and stabilization of femoral fracture. The patient, unfortunately, continued with cognitive impairment and respiratory failure and subsequent death, despite early diagnosis and aggressive supportive treatment.

REFERENCES

1. Von Bergmann E. Ein Fall Tö dlicher Fettembolie. Berliner Klinische Wochenschrift, 1873; 10: 385-7.
2. Aoki N, Soma K, M Shindo, Kurosawa T, Ohwada T. Assessment of potential fat emboli during placement of intramedullary nails after orthopedic fractures. Chest., 1998; 113: 178-81.
3. In Mellor, Soni N. fat embolism. Anesthesia, 2001; 56: 145-54.
4. Mudd KL, Hunt A, Matherly RC, et al. Analysis of pulmonary fat embolism of the number of blunt kills. Journal of Trauma, 2000; 48: 711-5.
5. Ten Duis HJ. Fat embolism syndrome. Injury, 1997; 28: 77-85.
6. Gurd AR, RI Wilson. Fat Embolism syndrome. JBJS Br., 1974; 56B(3): 408-416.
7. Bulger EM. Fat Embolism Syndrome: a 10-year review. Arch Surg, 1997; 132: 435-439.
8. Wong MW. Continuous monitoring of the pulse oximeter for inconsistent hypoxemia after long bone fractures. J Trauma, 2004; 56(2): 356-62.
9. Talucci RC. Early intramedullary nailing of femoral body fractures: a cause of fat embolism syndrome. Am J Surg., 1983; 146(1): 107-111.
10. Müller C, Rahn B, et al. Pathogenesis of incidence, diagnosis and treatment of fat embolism. Orthop Rev., 1994; 23(2): 107-17.
11. Jacobson DM. Neurological manifestations of fat embolism. Neurology, 1986; 36: 847-851.
12. Thomas JE, Ayyar DR. Systemic fat embolism: a diagnostic profile in 24 patients. Arche Neurol, 1972; 26: 17-23.
13. Muangman N. Chest radiographic course in fat embolism syndrome. J Med Thai Assoc, 2005; 88(12): 1854-1860.
14. Eriksson EA. Cerebral Fat Embolism Without Intracardiac Shunt: A New Presentation. J Emerg Shock Trauma, 2011; 4(2): 309-312.
15. Malagari K. Chan P, R Fishman, Editors. High resolution CT scans in benign pulmonary fatty embolism. Chest., 2003; 123(4): 1196-1201. cerebral edema: induction in cortical slices by polyunsaturated fatty acids. Science, 1978; 201: 358-360.
16. Gössling HR. Pelegrini VD. Fat Embolism Syndrome: A review of the pathophysiology and physiological basis of treatment. CORR. nineteen eighty one., 165: 68-82.
17. Nastanski F. Paradoxical posttraumatic fat embolism to the brain: a case report. J Trauma, 2005; 58(2): 372-4.
18. Lovering AT. intrapulmonary and shunt pulmonary gas exchange during normoxic and hypoxic exercise

- in healthy humans. *J Appl Physiol*, 2008; 104(5): 1418-1425.
- 19. Karagiorga G. Biochemical parameters of bronchoalveolar lavage fluid in fat embolism. *Intensive care Med.*, 2006; 32: 116-23.
 - 20. Sakamoto T. Computed tomography for the diagnosis and evaluation of cerebral fat embolism. *Neuroradiology*, 1983; 24: 283-285.
 - 21. Takahashi M. Magnetic resonance Brain imaging findings in fat Embolism: correlation with clinical manifestations. *J Trauma*, 1999; 46(2): 324-327.
 - 22. akoh CC1,Schick C1,Otero J1,Karam M1. Fat embolism syndrome after femoral fracture fixation: a case report. *Iowa J. Orthop*, 2014; 34: 55-62.
 - 23. Abbot MG. Fat Embolism Syndrome: A Closer Look. *Asian Journal of Critical Care.*, 2005; 1: 19-24.
 - 24. Lindeque BG, Schoeman HS, et al. Fat embolism syndrome: A double-blind therapeutic study. *J Joint Bone Surg Br.*, 1987; 69: 128-31
 - 25. Shier MR. Fat embolism prophylaxis: a study of four treatment modalities. *J Trauma*, 1977; 17(8): 621-629.
 - 26. J. Kallenbach Low-dose prophylactic corticosteroids for fatty embolism. *J Trauma*, 1987; 27(10): 1173-1176.
 - 27. Alho A. Corticosteroids in patients at high risk for fat embolism syndrome. *Surg Gyn Ob.*, 1978; 147: 358-362.
 - 28. Svennisenngsen S. Prevention of fat embolism syndrome in patients with immediate fracture of the femur or fixation of delayed operation. *Chirurgiae et gynaecologiae Annales*, 1987; 76(3): 163-166.
 - 29. Pinney SJ. Fat embolism syndrome in isolated femoral fractures: the choice of timing to nail the incidence of influence? *Injury*, 1998; 29(2): 131-3.
 - 30. Müller C. Effect of flexible drive diameter and reamer design on increasing pressure in medullary canal during reaming. *Injury*, 1993; 24(3): 40-47.
 - 31. Volgas DA. Fat embolism in femoral fractures: a comparison of the two reaming systems. *Injury*, 2010; 41(2): S90-3.