International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Post-Traumatic Fat Embolism in Intensive Care

Nijimbere Roselyne[1], Lamghari Saraa[2], Idi Djibadje Haboubacar[3], Chabbar Sara[4], Faouji Fatima Zahra[5], Mounir Anass[6], Cherkab Rachid[7], Barrou Lahoucine[8], El Kettani El Hamidi Chafik[9]

[1]Resident, Anesthesiology and Intensive Care department, Ibn Rochd University Hospital, Casablanca
[2]Hospital specialist practitioner, Anesthesiology and Intensive Care department, Ibn Rochd University Hospital, Casablanca
[3]Resident, Anesthesiology and Intensive Care department, Ibn Rochd University Hospital, Casablanca
[4,5]Assistant Professor, Anesthesiology and Intensive Care department Intensive, Ibn Rochd University Hospital, Casablanca
[6,7,8,9]Professor, Anesthesiology and Intensive Care department, Ibn Rochd University Hospital, Casablanca

Abstract
Fat embolism syndrome (FES) is a group of clinical, biological and radiological symptoms resulting from obstruction of the microcirculatory network by microdroplets of insoluble fat. It is a complication most often observed in the aftermath of a polytrauma involving several long bone fractures, but it can also occur outside the traumatic context. The aim of this study was to examine the epidemiological, pathogenic, clinical, paraclinical, therapeutic and evolutionary aspects of fat embolism syndrome. This was a retrospective study including all cases of post-traumatic fat embolism collected in the surgical intensive care unit of the Chu Ibn Rochd in Casablanca over a period of 5 years. 24 cases of post-traumatic fat embolism were collected. Male sex, age less than 40 years and the presence of a femur fracture were the dominant characteristics of the epidemiological profile of the trauma patient presenting with this syndrome occurring mainly within 72 hours of the trauma. The clinico-biological presentation was dominated by respiratory distress, consciousness disorders, anaemia, thrombocytopenia and hypocholesterolaemia. The management of FES was symptomatic, combining early immobilisation of fracture sites, optimal analgesia and maintenance of an effective blood volume. 41.66% of patients required mechanical ventilation and 75% underwent osteosynthesis of their fracture sites. 25% developed ARDS and the mortality rate was 26.08%.

Keywords: Fat embolism - polytrauma - complications - prognosis.

INTRODUCTION

Fat embolism is an often serious complication of long bone fractures. In its complete form, it presents clinically as the GURD triad, with respiratory, neurological and mucocutaneous involvement. In addition to a chest X-ray, further investigations include a chest CT scan, fundus examination, bronchoalveolar lavage, transesophageal ultrasound and possibly cerebral magnetic resonance imaging. In addition to prevention, the treatment of an overt fat embolism is purely symptomatic, based essentially on respiratory

resuscitation. The prognosis is fairly favourable and is mainly related to respiratory and neurological damage.

The aim of this study was to examine the epidemiological, pathogenic, clinical, paraclinical, therapeutic and evolutionary aspects of fat embolism syndrome.

MATERIAL AND METHODS

This was a retrospective study carried out in the surgical intensive care unit of the IBN ROCHD University Hospital in Casablanca between January 2015 and January 2020. All patients who were admitted during the above-mentioned period to the surgical intensive care unit for isolated fracture, polyfracture, polytrauma or directly for suspected post-traumatic FES presenting with FES were included. Diagnosis of the syndrome was based on the modified GURD criteria (one major criteria and four minor criteria or two major criteria and two minor criteria following polytrauma or trauma to the limbs and a significant SCHONFELD index (IEG ≥5) (Tables 1 and 2).The data collected included: demographic data, clinical presentation, biological and radiological data, prognostic and therapeutic data and finally data on the evolution.

Table 1: Diagnostic criteria for FES according to GURD and WILSON

Major criteria	Minor criteria
Petechial rash	Tachycardia
Respiratory symptoms with bilateral positive radiographic changes	Pyrexia
Cerebral signs unrelated to head injury	Retinal fat or petechial Urinary fat globules or oligoanuria Sudden drop in hemoglobin level

Table 2: SCHONFELD index of fat embolism

Criteria	Score
Petechiae	5
Chest X-ray changes(diffuse alveolar infiltrates)	4
Hypoxemia (PaO2 <9,3kPa)	3
Fever(>38 degree C)	1
Tachycardia(>120bpm)	1
Tachypnea(>30bpm)	1
Confusion	1

RESULTS

After a retrospective analysis of 355 trauma and polytrauma patients over a period of 5 years, the incidence of FES was 6.7% (24 cases over 5 years).

Demographic data showed a predominance of young males, with 87.5% of patients being male. The mean age was 42.6 years, ranging from 21 to 85 years. 19 patients were hospitalized for polytrauma and 5 for isolated fractures.

Clinically, respiratory distress was observed in 95.83% of patients with hypoxaemia ≤ 90%. Acute respiratory distress syndrome was noted in 25% of patients. Hemodynamically, 83.33% of patients had tachycardia with a heart rate ≥ 100 bpm. Neurological signs were present including consciousness

disorders in 70.83% of cases. 3 patients, or 12.5%, had presented with generalized tonic-clonic convulsive seizures. Petechiae were observed in 16.66% of patients.

In the para-clinical examinations performed on our patients, chest X-rays revealed diffuse bilateral opacities in 37.5% of patients and reactive pleural effusion in 25%. Cerebral computed tomography (CT) was normal in 56.25% of cases. Magnetic resonance imaging (MRI) of the brain was performed in only one patient, and showed multiple punctiform white matter hyper signals associated with ischaemic lesions. Management was based essentially on symptomatic treatment, including correction of any shock by vascular filling and introduction of positive inotropic drugs, oxygen therapy using oxygen goggles or masks in 58.33% of cases, and artificial ventilation in 41.66% of patients.

Complications were both specific and non-specific, and were dominated by anaemia (20 patients), thrombocytopenia (16 patients) and nosocomial pneumonia (7 patients).

The outcome was favourable in 17 patients (73.91%), with an average time to favourable outcome of 8 days and an overall mortality rate of 26.08%, also with an average time to death of 8 days. The causes of death in our patients were respiratory, neurological and septic.

DISCUSSION

Fat embolism syndrome is a group of clinical, biological and radiological symptoms resulting from obstruction of the microcirculatory system by microdroplets of insoluble fat (1).

1. Epidemiological aspects

The incidence of FES is rarer. Various studies report an incidence of between 0.25% and 33% (2). A retrospective study by BULGER (3) reported an incidence of <1%, although a prospective study by Fabien (4) reported a high incidence of 11-29%. A level trauma center in India (5), following a retrospective analysis of the cases of 1,692 patients, reported an incidence of 0.7% of fat embolism syndrome. The incidence of this syndrome also depends on the number and type of fractures, ranging from 0.5% to 2% in the case of an isolated fracture of a long bone, to 5 to 30% in the case of multiple fractures including the pelvic frame(6). In our study, the incidence of FES was 6.7%, which is still high compared with the results of the retrospective studies cited above.

Fat embolism occurs preferentially in young men under the age of 30, with a clear male predominance, for both violent and minor trauma (7). The incidence of FES is four times higher in men (8). In our study, the most common age group was between 20 and 39, and 87.5% of our patients were male. Our results are therefore consistent with those reported in the literature.

2. Diagnosis of FES

The diagnostic criteria proposed by GURD in 1970 (9) and redefined by GURD and WILSON in 1974 (10) are widely used today and are divided into major and minor criteria. FES is considered likely when three major criteria are associated with at least four secondary criteria with lipid macroglobulinemia (11). However, these criteria have been adapted by other authors who have stated that one of the following combinations of major and minor criteria is necessary for diagnosis: two majors (12, 13, 14,15), one major and three minors (16 ,17), two majors and two minors (12); or one major and two minors (18). Another diagnostic system was proposed by SCHONFELD. He proposed a fat embolism index which gives points for the different diagnostic criteria (19). The diagnosis is made with a score of 5 or more. In our series, we used as diagnostic criteria those proposed by GURD and WILSON (20), SCHONFELD (19), as well as those proposed by other authors (21).

The diagnosis of FES is primarily clinical, and the chronology of onset of symptoms must be suggestive. The duration of the interval varies from a few hours (12-36 hours) to a few days (3-4 days) (20), with a median of around the 24th hour(1) . In our study, the interval was <24 hours in 29.16% of patients, which is a poor prognostic factor, and >24 hours in 70.84% of our patients.

Respiratory involvement usually accounts for the severity of the disease. It occurs in almost 98% of cases (20) and results in the onset of acute respiratory failure with hypoxaemia, which becomes very significant when the partial oxygen pressure is less than 60 mmHg in the absence of any anaemia (22), tachypnoea and dyspnoea, which generate respiratory alkalosis with moderate hypocapnia, most often leading to acute respiratory distress syndrome in adults.

According to the literature, almost 50% of patients with a respiratory syndrome during fat embolism will require respiratory assistance (20). In our series, respiratory distress was present in 96% of cases, with ARDS developing in 25%. Artificial ventilation was necessary in 42% of our patients, which is in line with the literature. The radiograph may be normal at first, but overall, diffuse alveolar and interstitial opacities, evenly distributed, bilaterally and symmetrically in both lung fields, involving the perihilar and basilar regions and sparing the apices (23), are images that fall within the non-specific framework of ARDS but are suggestive of FES, in a post-traumatic or post-operative context.

In our patients, 9 presented with a diffuse and bilateral alveolar interstitial syndrome, compatible with the diagnosis of ARDS. However, 6 patients had a reactive pleural effusion and the chest X-ray remained normal in 10 patients.

The neurological manifestations of fat embolism are frequent (60 to 80%) (24, 25, 26, 20), polymorphous, non-specific and variable from one patient to another and sometimes in the same patient. They usually involve vigilance disorders, ranging from temporospatial disorientation, agitation, confusion or delirium to a more or less deep coma. Generalised tonic-clonic seizures have sometimes been reported. It is important to remember that before attributing any neurological manifestations to a possible fat embolism, it is always advisable to rule out a complication linked to a possible associated craniogastric trauma. The electroencephalographic tracing is most often altered, showing diffuse slowing, slow waves and hyporeactivity. CT scans frequently show diffuse cerebral oedema and foci of hypodensity in the white matter that may correspond to foci of infarction (27). Recent observations suggest that these lesions are reversible. The contribution of nuclear magnetic resonance imaging (MRI) seems promising. Recent publications indicate that it would allow differentiation between infarcts and haemorrhagic lesions (28). In our series, neurological syndrome was present in 71% of patients, which exceeds the data in the literature.

The association of petechial purpura with FES was described as early as 1911(22).These haemorrhagic foci, about 2mm in diameter, are pathognomonic of FES, and occur electively on the antero-upper part of the thorax, on the neck and armpits, on the oral mucosa and on the conjunctiva (bulbar and torsal), the latter location being particularly suggestive (1). In our patients, mucocutaneous petechiae were present in 17% of cases, cutaneous petechiae in 8% of cases, and were located on the upper part of the trunk.

Other organs may also be affected during fat embolism, either as a result of hypoxaemia or coagulation disorders, or directly by lipid embolisation. Ocular, cardiovascular and renal damage are the best documented.

Biologically, haemolytic anaemia is very common (1) and is found in two-thirds of patients with FES (20). Like all haemolytic anaemias, it is resistant to transfusion (1), but resistance to transfusion indicates its haemolytic nature and guides the diagnosis. Thrombocytopenia occurs in 48% of haemolytic anaemias.

This reflects the involvement of platelets in thrombotic processes. In our series, 67% of patients had thrombocytopenia and 83% had anaemia.

3. Therapeutic management of FES

The treatment of fat embolism is above all preventive. The first requirement is early immobilisation of any fracture site; this immobilisation must be as complete as possible; it begins at the site of the accident and must be followed by early, solid and definitive surgical fixation; this approach would dramatically reduce the frequency of fat embolism (29).

The second aspect of prophylaxis is based on rapid treatment to prevent hypovolaemic shock, stress and pain, and to correct any hypoxaemia by optimal oxygen therapy.

Once diagnosed, the patient must be transferred immediately to an intensive care unit equipped with a cardio respiratory monitoring system(30).Currently, there is no specific treatment for FES, and the treatment of an overt fat embolism is purely symptomatic and based on respiratory resuscitation(1).

Oxygen therapy is essential in any state of respiratory distress (31), and artificial ventilation is indicated in the event of profound hypoxaemia that does not respond to nasal oxygen therapy and/or in the event of neurological disorders. Mechanical ventilation with positive end expiratory pressure (PEEP) should be considered (32). However, in addition to mechanical ventilation, patients with severe pulmonary dysfunction may benefit from prone positioning or extracorporeal membrane oxygenation (29).

Sedation and analgesia in a mechanically ventilated patient must be carefully considered and appropriately chosen to optimise patient comfort while preserving the completeness of the neurological examination, and the use of a sedation agitation scale (SAS) can ensure the consistency of neurological examinations affected by sedation or analgesia (33).

The correction of haemodynamic disorders must be rapid, effective and controlled by appropriate cardiopulmonary monitoring (34). Some data suggest albumin: by binding to oleic acid, it reduces its toxic and oedema-generating potential in the capillary endothelium (35, 36). In fact, oleic acid has been implicated as a key element in fat embolism, particularly in the development of ALI (Acute Lung Injury) and ARDS (37, 38).

Heparin is known to be a "clarifying" factor because, by stimulating serum lipase, it reduces the concentration of lipids in plasma. As lipase increases free fatty acids, which play a determining role in the pathogenesis of the syndrome, heparin could therefore have an amplifying effect (39). On the other hand, pre-treatment with heparin has been shown to reduce the degree of pulmonary compromise (40) and intravascular coagulation (41), but its clinical use has not shown any sustained benefit in reducing lung damage. This is why the results of various studies contradict each other regarding the use of heparin in polytrauma patients (20).

All our patients received early immobilisation on admission, oxygen therapy and volume expansion. 92% of our patients received antiplatelet therapy based on low molecular weight heparin (LMWH), 87.5% received analgesia, 18 patients received surgical fixation of their fracture sites.10 patients received artificial intubation-ventilation and midazolam sedation on admission, with an average duration of intubation of 10 days.

Vascular filling was based on saline and albumin was only administered in one patient.

4. Evolution

In favourable cases, clinical signs subside after an average of 5 to 12 days of symptomatic treatment. The course of the disease may be adversely affected by respiratory complications, which may cloud the prognosis (42), mainly nosocomial pulmonary superinfection, but also acute lung oedema leading to refractory hypoxia with a potentially fatal outcome (40%) (42-43).There are also non-specific complications such as urinary tract infections, skin infections and bedsores, which may occur during the course of the disease. Intra-alveolar haemorrhage is a rare but extremely serious complication of FE.
In our patients, 73.91% had a favourable outcome, with an average time to favourable outcome of 8 days. 25% presented with acute pulmonary oedema leading to ARDS, and intra-alveolar haemorrhage secondary to FES was suspected in one of our patients with an unfavourable outcome.
Death occurs after an average of 5 to 11 days when it is linked to fat embolism and is often due to respiratory and/or neurological failure; it may also be due to associated lesions such as polytrauma, nosocomial pneumopathy or septic shock. In the series by BOUFFARD (44) 14% of patients died, while THEOLOGIS (45) reported 9% deaths. In our study, overall mortality was 26.08% (6 patients). 4 of these patients died as a result of respiratory failure, with an average time to death of 8 days.
Overall, the final prognosis of fat embolism depends fundamentally on pulmonary and neurological damage. When recovery is achieved, it is usually complete.

CONCLUSION

The diagnosis of FES is essentially clinical but often difficult because of its polymorphous presentation and the absence of specific paraclinical examinations. Management is essentially prophylactic, involving early fixation of fracture sites. Treatment of an overt fat embolism remains symptomatic and sometimes requires hydroelectrolytic, respiratory and neurological resuscitation measures.

REFERENCES

1. **O Mimoz, P Incagnoli, A Édouard, K Samii. Le syndrome d'embolie graisseuse. Conférences d'actualisation 1997, p. 587-98. 1997 Elsevier, Paris, et SFA**
2. **JohnsonMJ, LucasGL**. Fatembolism. Syndrome. Orthopedics.1996; 19:41-8.
3. **Bulger EM, Smith DG, Maier RV, Jurkovich GJ**. Fat embolism.
4. **Fabian TC, Hoots AV, Stanford DS, Patterson CR**, **Mangiante EC**. Fat embolısm syndrome, prospective evaluation of 92 fracture patients. Cri Care Med. 1990; 18:42-6.
5. **Cupta B, D'souza N, Sawhney C, Farooque K, Kumar A, Agrawal P, et al**. Analysing fat embolism syndrome in trauma Patients at AlIMS apex trauma centre, New Delhi India. J Emerg Trauma Shock. 2011; 4:337-41.
6. **Mimoz O.** Le syndrome d'embolie graiseuse. In: Samii K, éd. Traité d'anesthésie réanimation chirurgicale. 2e édition. Paris: Flammarion, 1995: 165 1-6.
7. **Pollak R, Myers RAM**. Early diagnosis of the fat embolism syndrome. J Trauma 1978; 18:121-3.
8. **Stein PD, Yaekoub AY,** Matta F, et al. Fat embolism syndrome. Am J Med Sci (2008): 336(6):472-7.
9. **Gurd AR**. Fat embolism: an aid to diagnosis. J Bone Joint. (1970); 52(4):732-737
10. **Bulger. EM, Smith.DG, Maier.RV, Jurkovich**,GJ FAT syndrome Arch surg. 1997; 132 (4):435-439
11. **Gurd AR,Wilson RI**. The fat embolism syndrome. BritJ Bone Joint Surg 197; 56:408-16.
12. **JeanStéphane David,Christian Guillaume,PierreYves_Gueugni aud**.embolie graisseuse. Le praticien en anesthésie réanimation 2006. Elsevier Masson SAS.

13. **Aoki N, Soma K, Shindo M, et al**. Evaluation of potential fat emboli during placement of intramedullary nails after orthopedic fractures. Chest (1998); 113(1):178-181.
14. **Chastre J, Fagon JY, Soler P, et al.** Bronchoalveolar lavage for rapid diagnosis of the fat embolism syndrome in trauma patients.Ann Intern Med (1990); 113(8):583-588.
15. **Roger N, Xaubet A, AgustiC, et al**. Role of bronchoalveolar lavage in the diagnosis of fat embolism syndrome. Eur Respir J (1995); 8(8):1275-1280.
16. **Bulger EM, Smith DG, Maier RV, Jurkovich GJ**. Fat embolism syndrome. A 10 year review. Arch Surg (1997); 132: 435-439.
17. **Wong MW, Tsui HF, Yung SH, et al**. Continuous pulse oximeter monitoring for inapparent hypoxemia after long bone fractures. Trauma (2004); 56(2):356-362
18. **Pinney SJ, Keating JF, Meek RN.** Fat embolism syndrome isolated femoral fractures: does timing of nailing influence incidence? Injury (1998): 29(2): 131 133.
19. **Capan LM, Miller SM, Patel KP**. Embolism IL. Fat embolism. Anesth Clin NorthAm (1993): 11(1): 25-54.
20. **C. Forstera, M. Jöhrb, J,-0. Gebbersa** .Embolie graisseuse et syndrome d'embolie graisseuse. Forum Med Suisse No 28 10 juillet 2002.
21. **Talbot M, Schemitsch EH**. Fat embolism syndrome: history, definition, epidemiology, Injury, Int. J. Care Injured (2006) 37S, S3-S7.
22. **Park HM, Ducret RP, Brindley DC**. Pulmonary imaging in fat embolism syndrome. Clin Nucl Med (1986); 11(7):521-2.
23. **Larcan A, Lambert H, Laprevotre-Heuilly MC**. Les embolies graisseuses. Rey Prat, 35: 2087-2096. 1985.
24. **Saulnier F, Durocher A, Durois D, Mathieu D, Fourrier F, Chopin C, Watrel F**. L'embolie graisseuse. Apropos de 44 observations. LARC Medical, 10 :679-686, 1983
25. **Tenaillon A, Longchal J, Jacqueson A, Boutier J, Planchon M**. Problèmes d'anesthésie et de réanimation en cas d'embolie graisseuse (pp 97-1 12). In: Problèmes d'anesthésie et de réanimation en situation d'exception.Arnette, Paris, 1981.
26. **Peltier LE** Fat embolism. III. The toxic properties of neutral fat and Iree fatty acids. Surgery (1956):40(4):665-70.
27. **Akhtar S**. Fat Embolism. Anesthesiology Clin (2009); 27: 533-550.
28. **Riska EB, Millynen P**.Fat embolism with multiple injury. J Trauma 1982; 22:891-4.
29. **JacobGeorge, Reeba George, R.Dixit,R.C Gupta and N Gupta**,. .fat embolism syndrome. Lung India. 2013 Jan-Mar; 30(1):47-53.
30. **JP Estèbe**.Des emboles de graisse au syndrome d'embolie graisseuse. Ann FrAnesth R&cmm 1997;16: 138-510 Elsevier, Paris
31. **Webb DP, McKamie WA. Pietsch JB.** Resuscitation of fat embolism syndrome with extracorporeal membrane oxygenation. JExtra Corpor Technol (2004):36(4):368-370.
32. **Habashi NM, Andrews PL, Scalea TM**. Therapeutic aspects of tat embolismsyndrome, Injury, Int. J. Care Injured (2006) 37S, S68-S75.
33. **JP Estèbe**.Des emboles de graisse au syndrome d'embolie graisseuse. Ann FrAnesth R&cmm 1997;16: 138-510 Elsevier, Paris
34. **Agnantis N, Gyras M, Tserkezoglou A, et al**. Therapeutic effect of bovinealbumin in the experimental fat embolism syndrome. Respiration (1988); 53(1):5057.

35. **Hofman WF, Ehrhart IC**. Albumin attenuation of oleic acid edema in dog lung depleted of blood components. J Appl Physiol (1985); 58(6):1949- 1955.
36. **Jacobs RR, Wheeler EJ, JelenkoC II, et al**. Fat embolism: a microscopic and ultrastructure evaluation of two animal models. JTrauma (1973); 13(11):980-993.
37. **Beilman G.** Pathogenesis of oleic acid-induced lung injury in the rat: distribution of oleic acid during injury and early endothelial cell changes. Lipids (1995):30(9):817-823.
38. **Roth B. Ekelund M, Fan BG, Ekstrom U, Nilsson-Ehle P.** Effects of heparin and low molecular weight heparin on lipid transport during parenteral feeding in the rat. Acta Anaesthesiol Scand (1996):40: 102-11.
39. **Saldeen T**. Intravascular coagulation in the lungs in experimental fat
40. embolism.Acta Chir Scand (1969); 135(8):653-662.
41. **Rokkanen P, Lahdensuu M, Kataja J, et al.** The syndrome of fat embolism: analysis of thirty consecutive cases compared to trauma patients with similar injuries. J Trauma (1970); 10(4):299-306
42. **Guartite.A, AL Harrar, Haida.F, Benyahia.B, Abbassi.O** Hémorragie intra-alveolaire asphyxiante: forme rare du syndrome d'embolie graisseuse Ann fr anesth réanim. 1998; 17(7):743-6.
43. **Wiel.E, M Fleyfel, J Onimus, O Godefroy, X Leclerc and P Adnet** tetraplegie au cours de l'embolie graisseuse Annales au Françaises d'Anesthésie et de Réanimation, Volume 13, Issue 5, Pages 730-733.
44. **Bouffard.Y, C. Guillaume, D. Perrot, B. Delafosse and J. Motin**. Bone marrow FAT in the circulation: clinical entities and pathophysiological mechanisms Injury, Int. J. Care Injured (2006) 37S, S8-S18.
45. **Thicoipe.M, M. André, P. Maurette, P. Lassie and J.P. Claverie** Embolies graisseuses post-traumatiques Annales Françaises d'Anesthésie et de Réanimation, Volume 3, Issue 5, 1984, Pages 335-338.