International Journal of Research in Medical Sciences
Devaraj DC et al. Int J Res Med Sci. 2024 Aug;12(8):3075-3078
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20242248

Review Article

Key role of orthopaedician and radiologist in successful management of fat embolism syndrome

Deepak Channarayapatna Devaraj[1]*, Anitha Boregowda[2]

[1]Department of Orthopaedics, Dayanand Sagar Medical College, Bangalore, Karnataka, India
[2]Department of Radiology, SAKRA World Hospital, Bangalore, Karnataka, India

Received: 04 July 2024
Revised: 23 July 2024
Accepted: 24 July 2024

***Correspondence:**
Dr. Deepak Channarayapatna Devaraj,
E-mail: aimspublications@gmail.com

ABSTRACT

Fat embolism syndrome (FES) is an emergency fatal condition, if not diagnosed early and timely managed. Careful observation of patients with multiple bone fractures aids in early diagnosis. Rehydration is an important factor in preventing FES. Most often the syndrome is self-limiting. High risk cases are treated with steroids and with respirator assistance.

Keywords: Fat embolism syndrome, Imaging, Magnetic resonance imaging

INTRODUCTION

The actual incidence of fat embolism is unknown, as it is typically asymptomatic and mild cases are self-limiting. Fat embolism syndrome (FES) was first described by Von Bergman in 1873 in patients with femur fractures.[1]

However, two decades earlier, Zenker had noted fat emboli in a patient with a crush injury.

CLINICAL FINDINGS

Patients with FES may present with dyspnoea, tachypnoea, hypoxemia (PaO_2 <60 mm Hg), rales, pleural friction rub, acute respiratory distress syndrome (ARDS), and confusion.

DIAGNOSTIC CRITERIA

Gurd and Wilson established a diagnostic approach for FES based on major and minor criteria. Diagnosis requires one major criterion, four minor criteria, and the presence of fat macroglobulinemia.

Major criteria

Major criteria included: symptoms and radiologic evidence of respiratory insufficiency, cerebral sequelae not related to head injury or other conditions, and petechial rash.

Minor criteria

Major criteria included: tachycardia (heart rate >110 beats/min), pyrexia (temperature >38.5°C), retinal changes of fat or petechiae, renal dysfunction, jaundice, acute drop in haemoglobin level, sudden thrombocytopenia, elevated erythrocyte sedimentation rate, and fat macroglobulinemia.

SYMPTOMS AND ONSET

The symptoms of FES typically begin 12 hours to 3 days after the underlying clinical condition is diagnosed. The three most characteristic features are respiratory distress, neurological symptoms, and skin petechiae.

Respiratory distress

It is present in 75% of cases, ranging from mild distress requiring supplemental oxygen to severe distress requiring mechanical ventilation.

Neurological symptoms

Patients may experience lethargy, restlessness, and a decrease in Glasgow coma scale (GCS) score due to cerebral oedema rather than ischemia. These signs are not lateralized. In severe cases, patients may become unresponsive.

Petechiae

It is seen in 50% of patients, usually on the chest, axilla, shoulders, and mouth. Petechial rash is temporary and can disappear within one day.[2]

Subacute FES (non-fulminant FES)

In subacute FES, the three characteristic features (respiratory distress, neurological signs, and petechial rash) are present. Petechiae, seen in 50-60% of cases, result from the occlusion of dermal capillaries by fat emboli.[3] Neurological signs, such as confusion, stupor, and coma, are usually temporary and not lateralized. Respiratory distress tends to improve by the third day. Retinal changes similar to Purtscher's retinopathy, including cotton wool exudates and small haemorrhages along the retinal vessels and macula, occur in 50% of patients with FES.[4]

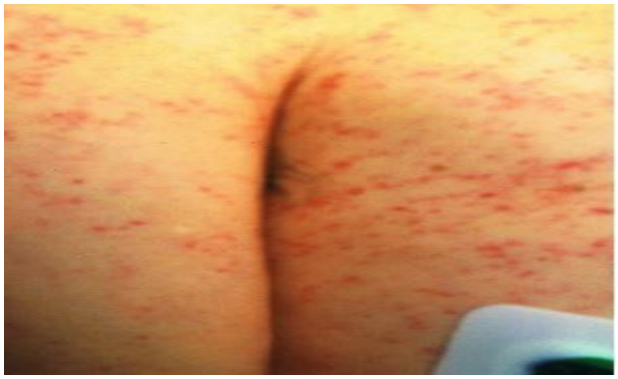

Figure 1: A pathognomonic petechial rash appeared within the first 24 hours, affecting the chest and upper abdomen.

IMAGING FINDINGS

Chest

Findings in radiographs can be very subtle and difficult to interpret. Computed tomography (CT) scans are relatively better for establishing the diagnosis, typically revealing three common patterns: ground-glass changes with geographic distribution, ground-glass changes with interlobular septal thickening, and nodular opacities. Fatty filling defects in the pulmonary arteries are rarely described.[5,6]

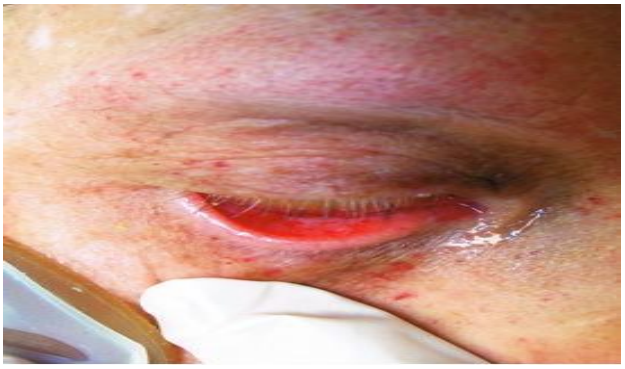

Figure 2: A pathognomonic petechial rash appeared within the first 24 hours, involving subconjunctival haemorrhages.

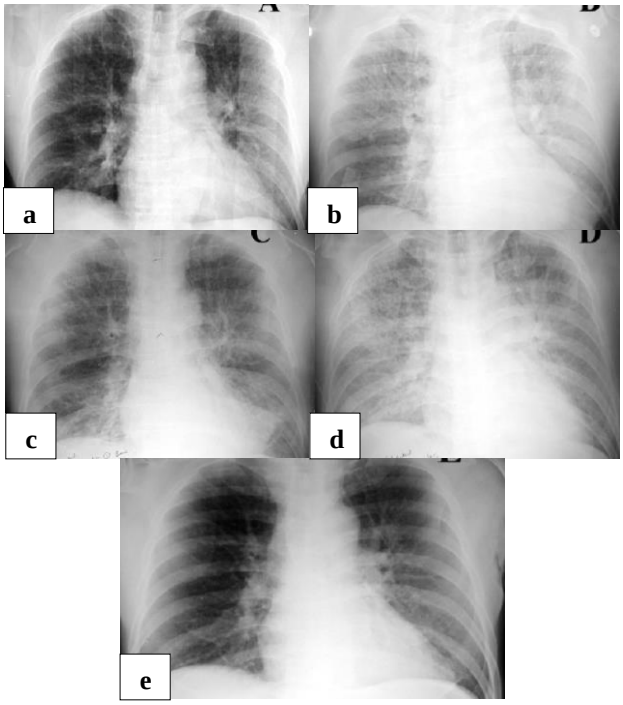

Figure 3 (a-e): Serial supine chest radiographs of a 59-year-old man with a closed fracture of the right femur after falling from a roof.

Fat embolism syndrome developed 24 hours after admission. Figure 3a-e represent the serial films on day 0, day 1, day 3, day 4, and day 15, respectively. This sequence shows the progression of pulmonary opacity, which is a non-specific finding. On day 0 (Figure 3a), the only abnormality detected is perihilar opacity, which progresses to diffuse involvement of both lungs, worst on day 4 (Figure 3d).

After supportive treatment, the lung opacities resolved, and the lung parenchyma appeared normal by day 15 (Figure 3e).[4]

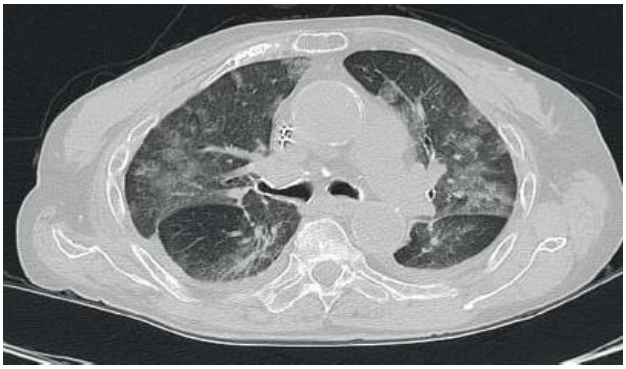

Figure 4: CT scan of the chest revealing peripherally located ground-glass opacities and bilateral patchy consolidations.[7]

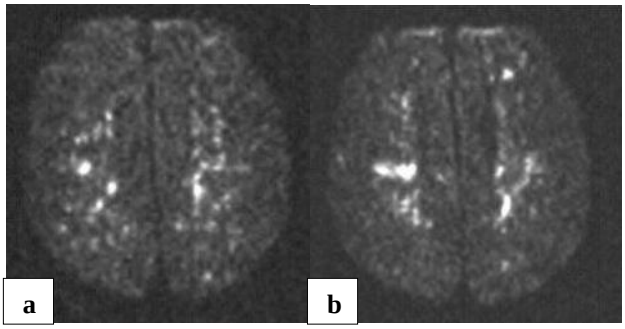

Figure 5: MRI of brain: showing foci of ischemia (a) post-op day 2 showing multiple hypertension areas consistent with multiple emboli, and (b) post-op day 14 shows evolving cortical infarctions.[3]

DISCUSSION

FES has an incidence of 1-3% following long bone fractures and 33% in patients with bilateral long bone fractures. Fat embolism develops in nearly all patients with bone fractures or during orthopedic procedures. Non-traumatic causes are rare and include pancreatitis, liposuction, sickle cell anemia, and orthopedic prosthetic procedures.[8]

Symptoms of FES usually develop 1-2 days after the event. Although fat emboli can reach any organ in the body, the embolic shower's results are most often evident in the lungs, brain, and skin. FES results from innumerable small fat emboli leading to multisystem dysfunction, classically characterized by the triad of: respiratory distress, cerebral abnormalities, and petechial rash.

In the past, FES was encountered not only in injured patients but also in those with acute osteomyelitis or severe frostbite, often resulting in fatal outcomes. Correction of shock is crucial for preventing FES, and adequate pre-operative rehydration is emphasized, especially if injuries were sustained during heavy exercise.[8]

A distinction must be made between the clinical entity of fat embolism syndrome and fat embolism demonstrated pathologically, which may be found post-mortem following fracture without prior evidence of the syndrome. For established cases, treatment aims to ensure adequate arterial oxygen pressure. Treatment consists of general supportive measures, including splinting, maintenance of fluid and electrolyte balance, and oxygen administration.

Once fat emboli enter the circulation, they can lodge in various sites of the body, most commonly the lungs (up to 75% of cases), but also the brain, skin, eyes, kidneys, liver, and heart, causing capillary damage and subsequent organ damage. Two theories describe the formation of a fat embolus.[9-12]

Mechanical theory

Following trauma, fat is released directly from the bone marrow into the circulation due to elevated pressure in the medullary cavity. Fat globules can lodge in the pulmonary circulation and potentially pass into systemic circulation, causing damage.[13-16]

Biochemical theory

Trauma-induced inflammation causes bone marrow to release fatty acids into the venous circulation, damaging capillary beds in the lungs and other organs, leading to conditions like interstitial lung disease and ARDS. This theory can also explain non-traumatic causes of fat embolism.[17-21]

Diagnosing FES requires at least two positive major criteria plus one minor criterion or four positive minor criteria. FES is a clinical diagnosis, with no specific laboratory tests available. Laboratory tests can only support the clinical diagnosis. Chest X-rays may show diffuse interstitial infiltrates, while CT scans reveal diffuse vascular congestion and pulmonary edema. Bronchoalveolar lavage, sputum, and urine tests for fat globules are not specific enough to diagnose FES.[22-25]

CONCLUSION

Orthopaedic surgeons should maintain a high clinical suspicion for FES, while radiologists should confirm the diagnosis with imaging. In patients treated conservatively with immobilization of long bone fractures, the incidence of FES is 22%. Early operative fixation of long bone fractures, particularly with the use of internal fixation devices, can reduce the incidence of FES. Urgent fixation of long bone fractures results in a 7% incidence of acute respiratory distress syndrome (ARDS), compared to 39% in those undergoing fixation after 24 hours. However, movement of the fracture ends during operative fixation can transiently increase fat emboli in the blood circulation. Conservatively treated long bone fractures with immobilization result in persistently elevated cytokine levels, which return to normal following operative fixation. Although reamed nailing increases pressure in the medullary cavity, it does not increase the rates of FES.

Other methods, such as drilling holes in the bony cortex, lavaging bone marrow prior to fixation, and using tourniquets to prevent embolization, have not been shown to reduce the incidence of FES.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Shaikh N, Parchani A, Bhat V, Kattren MA. Fat embolism syndrome: Clinical and imaging considerations: Case report and review of literature. Indian J Crit Care Med. 2008;12(1):32-6.
2. Shaikh N. Emergency management of fat embolism syndrome. J Emerg Trauma Shock. 2009;2(1):29-33.
3. Ten Duis HJ. The Fat Embolism Syndrome. Injury. 1997;28:77-85.
4. Graziani A, Moretti CC, Cappa FM. Fat embolism syndrome: chest CT findings. J Bras Pneumol. 2018;44:3.
5. Gurd AR, Wilson RE. The Fat embolism syndrome. J Bone Joint Surg Br. 1974;56:408-16.
6. Georgopoulos D, Bouros D. Fat embolism syndrome: Clinical examination is still the preferable diagnostic method. Chest. 2003;123:982-3.
7. Fenire M. Fat Embolism Syndrome. Images Med. 2014;2(5):21-3.
8. Mcmermott ID, Culpan P, Clancy M, Dooley JF. The role of rehydration in prevention of FES. Injury. 2002;33:757-9.
9. Akhtar S. Fat embolism. Anesthesiology Clin. 2009;27(3):533-50.
10. Laurence K. Fat embolism syndrome. Available at: patient.info. Accessed on 14 March 2024.
11. Adeyinka A, Pierre L. Fat Embolism. Stat Pearls. 2020.
12. Fukumoto LE, Fukumoto KD. Fat Embolism Syndrome. Nurs Clin North Am. 2018;53(3):335-47.
13. George J, George R, Gupta D, Gupta N. Fat embolism syndrome. Lung India. 2013;30(1):47-53.
14. Michael E, Kwiatt, Mark J, Seamon. Fat embolism syndrome. Int J Crit Illness Injury Sci. 2013;3(1):64-8.
15. Korhan T, Hakan Y. Fat embolism syndrome. Surg Today. 2007;37(1):5-8.
16. Joseph D, Puttaswamy RK, Krovvidi H. Non-respiratory functions of the lung. Continuing Educ Anaesth Crit Care Pain. 2013;13(3):98-102.
17. Saigal R, Mittal M, Kansai A, Singh Y, Jain. Fat embolism syndrome. Journal of the Association of Physicians of India. 2008;56(392):245-9.
18. Shaikh N. Emergency management of fat embolism syndrome. J Emerg Trauma Shock. 2009;2(1):29-33.
19. Silva DF, Carmona CV, Calderan TR, Fraga GP, Nascimento B, Rizoli S. The use of corticosteroid for the prophylaxis of fat embolism syndrome in patients with long bone fracture. Rev Col Bras Cir. 2013;40(5):423-6.
20. Lewis SL, Dirksen SR, Heitkemper MM, Bucher L. Medical-Surgical Nursing: Assessment and Management of Clinical Problems, Single Volume. Elsevier Health Sciences. 2013;1523.
21. Nolan J, Soar J. Anaesthesia for Emergency Care. OUP Oxford. 2012;86.
22. Bederman SS, Bhandari M, McKee MD, Schemitsch EH. Do corticosteroids reduce the risk of fat embolism syndrome in patients with long-bone fractures? A meta-analysis. Can J Surg. 2009;52(5):386-93.
23. Dondelinger RF. Imaging and Intervention in Abdominal Trauma. Springer Science & Business Media. 2004;477.
24. George RB, Light RW, Matthay MA, Matthay RA. Chest Medicine: Essentials of Pulmonary and Critical Care Medicine. Lippincott Williams & Wilkins. 2005;222.
25. Kumar V, Abbas AK, Aster JC. Robbins Basic Pathology. Elsevier Health Sciences. 2007;505.

Cite this article as: Devaraj DC, Boregowda A. Key role of orthopaedician and radiologist in successful management of fat embolism syndrome. Int J Res Med Sci 2024;12:3075-8.