*Case Report*

# Fat embolism syndrome: Management beyond controversies

**M.B.Ummu Guldhum[1]*, Kavu devi[2], Adithya[3]**

[1]Consultant Anesthesiologist, Department of Anesthesia, Kauvery hospital, Tirunelveli, Tamil Nadu
[2]Consultant Neuroanesthesia and Neurocritical Care, Department of Anesthesia, Kauvery hospital, Tirunelveli, Tamil Nadu
[3]Consultant Pulmonary Medicine, Department of Anesthesia, Kauvery hospital, Tirunelveli, Tamil Nadu
*Correspondence

**Abstract**

**Background:** Fat Embolism Syndrome (FES) is a rare, life-threatening complication characterized by an inflammatory response to systemic circulating fat. It occurs in both traumatic and non-traumatic clinical scenarios. Diagnosis relies on a classic clinical triad: respiratory distress, neurological changes, and dermatological manifestations. Early diagnosis and prompt supportive treatment are essential to reduce patient mortality and morbidity.

**Key words:** Fat Embolism Syndrome (FES); Hypoxemia; Dermatological manifestations

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## 1. Introduction

Fat Embolism Syndrome is a rare lethal complication which is an inflammatory response to the presence of fat in the systemic circulation occurring in both traumatic and non-traumatic patients. Diagnosis is by clinical triad of respiratory, neurological and dermatological manifestations. Here we report a patient who had Fat Embolism syndrome diagnosed on the 2nd Day following trauma and we also discuss the role of each intervention. Here we would also like to emphasize that early diagnosis and prompt treatment could reduce the mortality and morbidity of a patient

## 2. Case presentation

A 31 yrs old male patient was admitted on 02.04.2026 at 4 pm with an alleged history of RTA and sustained injury to right lower limb. On further evaluation patient was found to have right shaft of Tibia unicorticate fracture. Patients have nil comorbid conditions. Started treatment with antibiotics ,analgesics and shifted to ward after POP dressing.

On admission patient's vitals:

| | |
|---|---|
| **BP** | 150/90 mm/Hg |
| **PR** | 102bpm |
| **Spo2** | 99% |
| **RR** | 12/min |

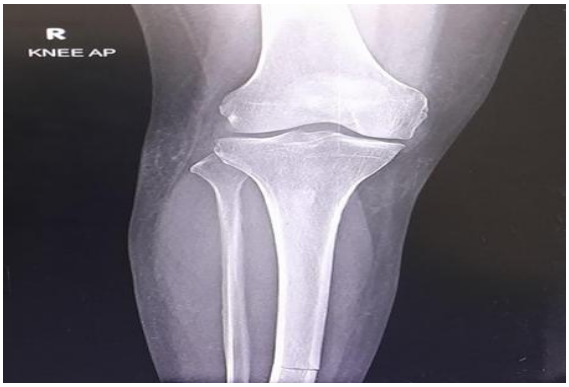



**Fig (1):** X-ray Right Tibia

On 03.04.2026 at 8.00am patient developed sudden desaturation, tachycardia, fever, decreased responsiveness. On auscultation bilateral basal crepts were present. Patients were supported with oxygen and shifted to critical care.

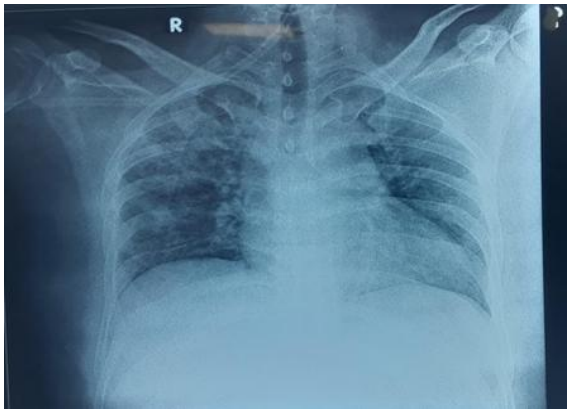
**Vitals**

BP- 130/80mm/Hg

HR-127 bpm

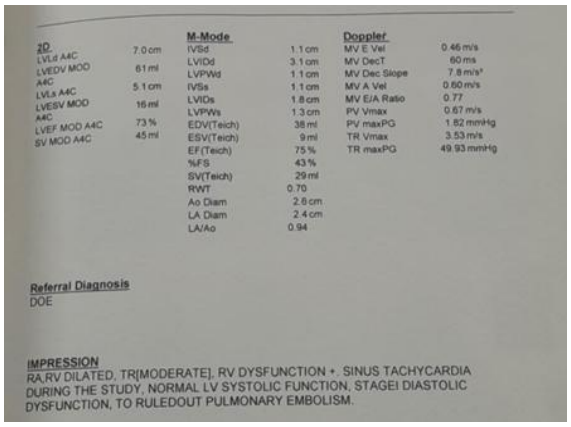
Spo2- 97% with face mask o2 8 lit/min

Patients were supported with NIV with 100% O2 and ABG done showed PH-7.46 Pco2-30 Po2-56 Hco3-21.3 (Hypoxemia with respiratory alkalosis and metabolic acidosis). Provisional diagnosis of possible pneumonia, Fat embolism syndrome, Pulmonary thrombo embolism was suspected. Immediate Chest X-Ray, ECHO and CT-PA done.




**Fig(2):** Chest X-Ray showed -Bilateral airspace opacities.

ECHO - RA,RV dilated with right ventricle dysfunction ,moderate TR. Normal LV function.

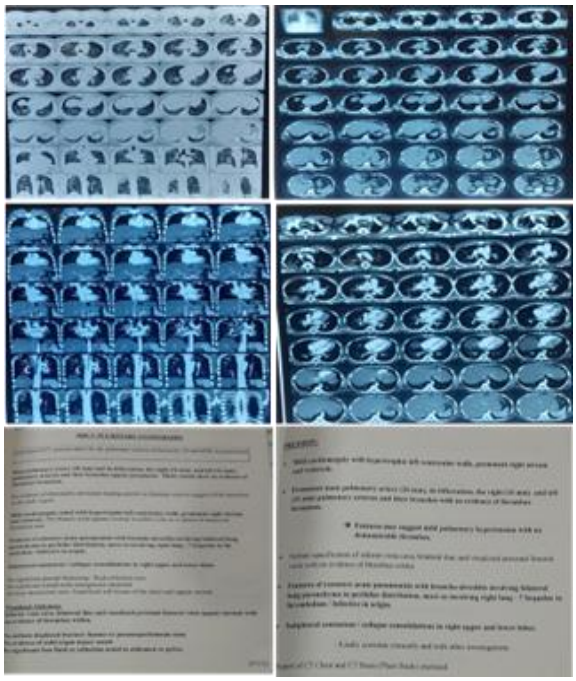


| 2D | | M-Mode | | Doppler | |
|---|---|---|---|---|---|
| LVLd A4C | 7.0 cm | IVSd | 1.1 cm | MV E Vel | 0.46 m/s |
| LVEDV MOD A4C | 61 ml | LVIDd | 3.1 cm | MV DecT | 60 ms |
| LVLs A4C | 5.1 cm | LVPWd | 1.1 cm | MV Dec Slope | 7.8 m/s² |
| LVESV MOD A4C | 16 ml | IVSs | 1.1 cm | MV A Vel | 0.60 m/s |
| LVEF MOD A4C | 73 % | LVIDs | 1.8 cm | MV E/A Ratio | 0.77 |
| SV MOD A4C | 45 ml | LVPWs | 1.3 cm | PV Vmax | 0.67 m/s |
| | | EDV(Teich) | 38 ml | PV maxPG | 1.82 mmHg |
| | | ESV(Teich) | 9 ml | TR Vmax | 3.53 m/s |
| | | EF(Teich) | 75 % | TR maxPG | 49.93 mmHg |
| | | %FS | 43 % | | |
| | | SV(Teich) | 29 ml | | |
| | | RWT | 0.70 | | |
| | | Ao Diam | 2.8 cm | | |
| | | LA Diam | 2.4 cm | | |
| | | LA/Ao | 0.94 | | |

**Referral Diagnosis**
DOE

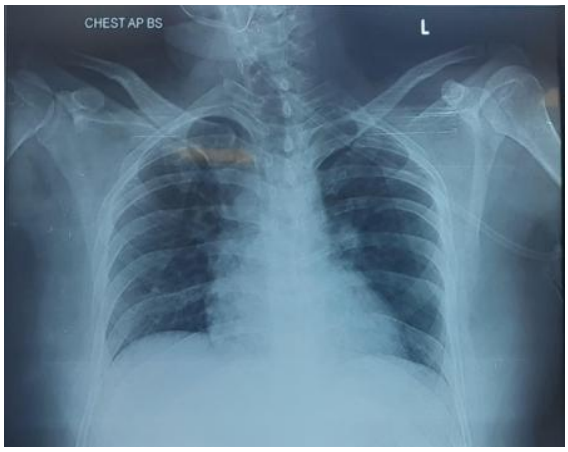
**IMPRESSION**
RA,RV DILATED, TR[MODERATE], RV DYSFUNCTION + SINUS TACHYCARDIA DURING THE STUDY, NORMAL LV SYSTOLIC FUNCTION, STAGEI DIASTOLIC DYSFUNCTION, TO RULEDOUT PULMONARY EMBOLISM.

CT-PA showed - Pulmonary hypertension with no definite thrombus. Mild cardiomegaly with hypertrophic left ventricle walls and prominent right atrium, right ventricle. B/L perihilar bronchoalveolitis suggests Fat embolism or infective origin.



Pulmonologist opinion was obtained and the patient was evaluated for sepsis, broad spectrum antibiotics initiated,Inj.Hydrocort 50 mg q6th hourly given and Inj. LMWH 60 mg S/C OD also started.

Ophthalmologist opinion obtained and presence of retinal fat globules were ruled out. Blood investigations showed no significant evidence of sepsis. Antibiotics deescalated-dimer was-4.65 mg/L. But serial blood levels showed a fall in Hemoglobin levels and platelet counts. Patient was on intermittent NIV and facemask O2 support. On 06.04.2026 ECHO repeated which showed- Moderate TR, dilated RA,RV. Chest physiotherapy, deep breathing exercises given. Antibiotics and steroids continued. The patient was gradually improving. The patient was shifted to the ward on 07.04.2026.




**Fig(3):** Repeat chest X-Ray taken on 07.04.2026.

**3. Discussion**

Incidence

Variable data on the incidence of fat embolism and FES have been reported. Clinical diagnosis of small fat embolism or mild cases of FES may be missed and go unnoticed. In 1 study, about 67% of orthopedic trauma patients have fat globules in their blood. If the blood sample was taken from a site close to the area of the fracture, the incidence is closer to 95%. Fat embolism and FES can also occur intraoperatively while repairing a long bone fracture. A transesophageal echocardiogram detected fat embolism in nearly 41% of patients. Fat embolism has a higher incidence than FES. In the landmark study carried out by Gurd, using the established clinical criteria, an incidence of 19% of FES was reported in a group of trauma patients. Since early open reduction and internal fixation have become the standard of care for repairing fractures of long bones, the incidence of fat embolism and FES has gradually decreased. Most recent studies show an incidence of about 1% to 11%.[4]

Mortality rate

Overall, the mortality of FES is estimated to be 5–30%. Despite critical care improvement, a mortality rate as high as 30% was reported in a recent meta-analysis. The difference in incidence rates and mortality rates may be due to a difference in the age of the FES of the studies[5]

Role of corticosteroids

Many clinical trials have been conducted regarding the use of corticosteroids for the prevention of FES. Since the development of FES has been attributed to the release of free fatty acids, endothelial injury, vasculitis, and direct toxicity to pneumocytes, corticosteroids have been proposed to limit the elevation of free fatty acid levels thus blunting the inflammatory response.

Since steroids were beneficial in critically ill patients in the treatment of ARDS its role in the prevention of FES could also be considered.

Despite its use, complications leading to infection and even death have limited its use in such patients. Studies have Conducted not only the benefits but also a second outcome

of possible infection and avascular necrosis in those patients. There were also trials regarding low dose vs high dose steroids. There were no proven benefits of survival with high dose steroids in any kind of similar lung pathologies; rather it could lead to superinfections and complications as mentioned before. Low dose steroids for a longer period have been in use for patients with ARDS ,although Fat Embolism Syndrome is like ARDS but a different entity, trials of outcome with low dose steroids have been done. [1]

Which steroid to use?

When it comes to the type of steroid, again trials still remain in place of using Inj.Methylprednisolone and the debate of low dose vs high dose was Conducted based on this drug. Since the patient was getting better with NIV, we continued with Inj.Hydrocortisone 50mg IV Q6th hrly.

Role of anticoagulants

Anticoagulants are used in trauma patients to prevent thromboembolism in view of prolonged immobilization unless there are no major injuries and significant bleeding risks.

Heparin vs LMWH

Historically Inj.Heparin was used in Fat Embolism Syndrome due to its lipase activity which can dissolve the fat globules and relieve the mechanical obstruction.

One other theory points towards the mechanism of free fatty acids in the Pathogenesis of fat embolism syndrome which damages the pulmonary alveolar capillary membranes that lead to ARDS. We saw that Heparin could dissolve fat globules and produce free fatty acids.[2]

For this reason, there are controversies of using Heparin as it can provoke Fat Embolism Syndrome. On the other hand, LMWH does not have this lipase activity and could be considered as it has no role in either prevention or provoking FES. Since the Pathogenesis of the disease is not comprehensively determined, we preferred to use Inj.LMWH 60 mg S/C OD considering the above-mentioned theories only to prevent thromboembolism due to immobilization.

Is it only a major trauma?

What we see till now is the occurrence of Fat Embolism Syndrome in major trauma, long bone fractures and crush injuries. Also, we know there are non-traumatic causes present as well. Our patient here sustained a traumatic injury to the tibia which is only an unicorticate fracture and not a major one but landed up in this phenomenon.

So, either traumatic or non-traumatic, major or minor ,the basic pathology lies in the breaching of the medullary cavity which displaces the Fat globules into the medullary veins.[3]. Above all ,diagnosing it early and intervening effectively can halt progression and reduce further complication

**4. Conclusion**

Fat embolism syndrome can occur even with minor, single-cortex long bone fractures, leading to a sudden onset of low blood oxygen that can progress to acute respiratory distress syndrome following trauma. The condition can manifest in subclinical, fulminant, or non-fulminant forms, though it typically resolves on its own within three to

seven days. Ultimately, securing a quick diagnosis and providing timely intervention through a collaborative, multidisciplinary medical team remains the cornerstone of effective patient management.

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