

Fat embolism syndrome

What is fat embolism syndrome?

Fat embolism syndrome (FES) is a life-threatening complication in patients with orthopaedic trauma, especially long bone fractures.^[1]

Fat embolism syndrome occurs when embolic fat macroglobules pass into the small vessels of the lung and other sites, producing endothelial damage and resulting respiratory failure (acute respiratory distress syndrome (ARDS-like) picture), cerebral dysfunction and a petechial rash.^[2] ^[3] It can be difficult to diagnose.

The initial symptoms are probably caused by mechanical occlusion of multiple blood vessels with fat globules that are too large to pass through the capillaries. The vascular occlusion in fat embolism is often temporary or incomplete, as fat globules do not obstruct capillary blood flow completely because of their fluidity and deformability. The late presentation is thought to be a result of hydrolysis of the fat to more irritating free fatty acids which then migrate to other organs via the systemic circulation. In the lungs, for example, there is direct toxicity to the lung tissue or capillary endothelium.^[4] It has also been suggested that paradoxical embolism occurs from shunting.^[5]

Fat embolism syndrome aetiology^[6] ^[7] ^[8]

FES most often follows a closed fracture of a long bone but there are many other causes:

- Fractures – closed fractures produce more emboli than open fractures. Long bones, pelvis and ribs cause more emboli than those involving the sternum or clavicle. Multiple fractures produce more emboli.

- Orthopaedic procedures – most commonly, intramedullary nailing of the long bones, hip or knee replacements.
- Massive soft tissue injury.
- Severe burns.
- Bone marrow biopsy.
- Non-traumatic settings occasionally lead to fat embolism. These include conditions associated with:
 - Liposuction.
 - Fatty liver.
 - Prolonged corticosteroid therapy.
 - Acute pancreatitis.
 - Osteomyelitis.
 - Conditions causing bone infarcts, especially sickle cell disease.

How common is fat embolism syndrome? (Epidemiology)

The given incidence of FES ranges from less than 1% to 30% in different studies. It varies considerably according to the cause^[7] and the diagnostic criteria used by studies.^{[4] [8]}

Fat globules have been detected in the blood of 67% of orthopaedic trauma patients. This number increased to 95% when the blood was sampled in close proximity to the fracture site. However, the presence of fat globules in the blood does not automatically lead to FES.^[6]

Men are more likely than women to develop the condition and it is rare in children aged 0-9 years. The age range most commonly affected is 10-40 years.^[4]

Fat embolism syndrome symptoms^{[4] [7] [8]}

There is usually a latent period of 24-72 hours between injury and onset. The onset is then sudden, with:

- **Breathlessness** ± vague pains in the chest. Depending on severity this can progress to respiratory failure with tachypnoea, increasing breathlessness and hypoxia.
- **Fever** – often in excess of 38.3°C with a disproportionately high pulse rate.
- **Petechial rash** – commonly over the upper anterior part of the trunk, arm and neck, buccal mucosa and conjunctivae. The rash may be transient, disappearing after 24 hours.
- **Central nervous system** – varying from a mild headache to significant cerebral dysfunction (restlessness, disorientation, confusion, seizures, stupor or coma).
- **Renal** – oliguria, haematuria, anuria.
- **Drowsiness** with oliguria is almost pathognomonic.

One study reported that 98% of the patients presented with mental status changes, whereas only 22% had focal signs and/or seizures. [9]

There is a fulminant form which presents as acute cor pulmonale, respiratory failure and/or embolic phenomena leading to death within a few hours of injury.

Diagnostic criteria (combined from various sources)

Important information

Diagnostic criteria were first devised by Gurd and have been modified several times since.^[4]

Major criteria

Respiratory symptoms with radiographic changes.

Cerebral involvement unrelated to head injury.

Petechial rash.

Minor criteria

Tachycardia.

Pyrexia (usually >38.5C).

Retinal changes - cotton wool exudates and small haemorrhages, occasionally fat globules seen in retinal vessels.

Jaundice.

Renal abnormalities (oliguria, anuria or lipiduria).

Acute thrombocytopenia.

Acute anaemia.

High ESR.

Fat macroglobulinemia.

Fat globules in sputum.

Typically, at least two symptoms for the major criteria or one symptom for the major criteria and four symptoms for the minor criteria must be present to diagnose FES.^[4] ^[8]

Differential diagnosis^[7]

Dyspnoea, hypoxia and abnormal CXR can occur with thromboembolism and pneumonia.

Investigations^[7]

The diagnosis of fat embolism syndrome is made by clinical features and there are no specific laboratory findings.^[1]

- Cytological examination of urine, blood and sputum may detect fat globules that are either free or in macrophages. This test has low sensitivity and a negative result does not exclude fat embolism.
- The CXR may show evenly distributed, fleck-like pulmonary shadows (snow storm appearance), increased pulmonary markings and dilatation of the right side of the heart.

- Arterial blood gases will show hypoxia, pO_2 usually less than 8 kPa (60 mm Hg) and hypocapnia. Continuous pulse oximeter monitoring may enable hypoxia from fat embolism to be detected in at-risk patients before it is clinically apparent (suggested by recurrent desaturations below 90%).^[10]
- Platelets are reduced.^[10] Decreased haematocrit occurs within 24–48 hours and is attributed to intra-alveolar haemorrhage.^[7] Lipase is elevated but this is not pathognomonic, as it occurs in any bone trauma.^[10] Calcium is reduced.^[11]
- Brain MRI scan may help in the diagnosis of cerebral fat embolism.
- Transoesophageal echocardiography (TEE) may be of value in detecting intra-operative release of marrow contents into the bloodstream during intramedullary reaming and nailing.^[6]
- One study reported hypointense areas consistent with fat globules at the grey-white matter junction.^[9]

Fat embolism syndrome treatment^[7]

Management of FES is supportive and consists primarily of ensuring good arterial oxygenation.^[1] High flow rate of oxygen is given to maintain the arterial oxygen tension in the normal range. Restriction of fluid intake and the use of diuretics can minimise fluid accumulation in the lungs so long as circulation is maintained.

On the other hand, maintenance of intravascular volume is important because shock can exacerbate the lung injury caused by FES.^[10] Albumin has been recommended for volume resuscitation in addition to balanced electrolyte solution, because it not only restores blood volume but also binds fatty acids and may decrease the extent of lung injury.

Mechanical ventilation and positive end-expiratory pressure (PEEP) may be required to maintain arterial oxygenation.

Drugs^{[6] [8]}

Early experiments using dextrose to decrease fatty acid mobilisation, ethanol to reduce lipolysis and heparin anticoagulation have been largely shown to be of unproven benefit. Recent studies have indicated a potential role for the renin-angiotensin system in the treatment of FES but more research is required to confirm this.

Surgical^[6]

Prompt surgical stabilisation of long bone fractures reduces the risk of the syndrome.

Prognosis

- Advances in supportive care have reduced the mortality rate from FES to <10%.^[4] Even severe respiratory failure associated with fat embolism seldom leads to death. .
- Neurological deficit and coma may last for days or weeks. Residual deficits may include personality changes, memory loss and cognitive dysfunction.
- Pulmonary sequelae usually resolve completely within a year, although residual diffusion capacity deficits may exist.^[7]
- One study reported that patients presenting with mild mental status changes, focal deficits, or seizure had a better outcome than those presenting with coma or abnormal posturing.^[9]

Prevention

Early immobilisation of fractures seems to be the most effective way of reducing the incidence of this condition.^[1] Prophylactic corticosteroids with long bone fractures may reduce the incidence of FES^[12] although a large-scale RCT is required prior to widespread recommendation of this approach.^[4] . Early (within 24 hours of trauma) compared to delayed fixation has been associated with a lower risk of FES.^[4]

Historical

Fat embolism was first described as an autopsy finding by Zenker in 1862. In 1873 von Bergmann described it as a clinical syndrome for the first time.^[7]

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Last updated by: Dr Jo Seale 29/11/2022	
Peer reviewed by: Dr Toni Hazell 29/11/2022	Next review date: 28/11/2027

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