

Review Article

Fat Embolism Syndrome: A Review

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Abstract: The subject of fat embolism is of recurring interest to those managing trauma. Even though fat embolism as a complication of bony trauma has been recognized for over 100 years, still it remains diagnostic challenge for treating clinicians. There has been rapid progress in knowledge and understanding of this relatively rare syndrome over few years. Presently, as a result of improvements in technology, new information has been derived to reduce significantly the morbidity and mortality of fat embolism. Although rare, clinicians should consider fat embolism as a cause of postoperative respiratory distress. This article covers the topic of fat embolism in general, highlights the importance of clinical expertise, and whatever technological aids are available to diagnose and appropriately treat this relatively rare, but highly significant form of the syndrome.

Keywords: Fat embolism, Trauma, Postoperative respiratory distress.

INTRODUCTION

FA Zenker in 1861 first described fat embolism in a rail road worker with a thoraco-lumbar crush injury [1]. Ernst von Bergmann in 1873, was first to make clinical diagnosis of fat embolism [2]. He arrived at this diagnosis because of his knowledge of the pathology of fat embolism. In 1911, the term 'cardiac syndrome' was used by Grandahl to describe the early acute onset of tachycardia and hypotension seen with fat embolism. He attributed this to blockage of the pulmonary arteries by fat [3].

Definition

Fat Embolism Syndrome (FES) may be defined as a complex alteration of haemostasis which occurs as an infrequent complication of fractures of pelvis and or long bones and manifests clinically as acute respiratory insufficiency [4]. Any case in which fat globules are demonstrated within the lung parenchyma or peripheral microcirculation can be described as 'fat embolism [3]. Fat embolism syndrome follows long bone fractures. Its classic presentation consists of an asymptomatic interval followed by pulmonary and neurologic manifestations combined with petechial haemorrhages.

INCIDENCE

Many aspects of the fat embolism syndrome remain poorly understood. It is therefore difficult to determine

the incidence of this complication. It ranges from less than 2% to 22% in different studies [5].

CAUSES

Fat embolism is most common after skeletal injury, and is most likely to occur in patients with multiple long bone and pelvic fractures. It is more common in patients with fractures involving the middle and proximal parts of the femoral shaft. It is more frequent in closed rather than open fractures.

Causes of fat embolism

Traumatic :

- Long bones fractures
- Blunt trauma
- Soft tissue injury
- Bone marrow harvest
- Joint reconstruction [6]
- Liposuction

Non traumatic :

- Acute pancreatitis
- Burns
- Cardiopulmonary bypass
- Decompression sickness
- Sickle cell crisis [7]
- Parenteral lipid infusion [8]
- Diabetes mellitus

Risk factors for FES

- Young individuals with fractures.
- Multiple fractures.
- Closed fractures.
- Movement of unstable bone fragments.

Greater amount of fat will be released into the marrow vessels following multiple fractures than do single fractures, thereby increasing risk for FES.

Movement of unstable bone fragments and reaming of the medullary cavity cause distortion of and increased pressure within the medullary cavity, permitting entry of marrow fat into torn venous channels that remain open even in shock because they are attached to the surrounding bone.

PATHOPHYSIOLOGY

Many aspects of the fat embolism syndrome remain poorly understood. Several theories have been proposed for its pathogenesis. Two theories which have gained importance are:

- **Mechanical theory:** described by Gossling *H et al.* [9] states that large fat droplets are released into the venous system. FES results from physical obstruction of the pulmonary and systemic vasculature with embolized fat. Increased intramedullary pressure after injury forces marrow into injured venous sinusoids, from which the fat travels to the lung and occludes pulmonary capillaries.
- **Biochemical theory:** described by Baker *et al.* [10]. Local hydrolysis of triglyceride emboli by pneumocyte lipase together with excessive mobilization of free fatty acids from peripheral adipose tissue by the catecholamines results in toxic pulmonary concentration of these acids. The biochemical theory helps to explain non-traumatic forms of FES.

CLINICAL FEATURES

- Classic presentation - asymptomatic interval for about 12-72 hours followed by triad of pulmonary changes, cerebral changes and petechial rash.
- FES follows biphasic clinical course.
- 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 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.

- Pulmonary Changes [11]:
 - Earliest manifestations and seen in 75% of cases.
 - Dyspnoea, tachypnoea, tachycardia and cyanosis.
 - Respiratory failure - 10% cases.
- Cerebral Changes [12]:
 - Nonspecific and seen in 86% of patients.
 - Ranges from acute confusion, headache, stupor, coma, rigidity or convulsions. (Cerebral edema contributes to the neurological deterioration)
- Dermatological Changes [13]:
 - A petechial haemorrhage seen within 24-36 hours and usually distributed to the upper anterior portion of the body—chest, neck, shoulder, axilla, upper arm and conjunctivae.
 - It results from occlusion of dermal capillaries by fat and increased capillary fragility.
 - The distribution is theorized to the related fat particles floating in the aortic arch are like oil in water and are embolized to non-dependent skin areas via subclavian or carotid arteries [14].
 - Rashes disappear within a week.

In addition to the above clinical features, there are number of other features, often related to fat embolism which, if present, may be helpful in diagnosis. These include:

- Retinal Signs [4]: retinal haemorrhage, mucular oedema, fluffy exudates and, rarely, presence of fat droplets in the vessels;
- Hepatic Signs: rarely, jaundice may be the presenting sign;
- Fever: may be secondary to underlying infection or may result from the fat embolism;
- Tachycardia: again may result from an underlying infection; and
- Renal Signs: transient oliguria, lipuria, proteinuria, and haematuria may all result from fat emboli in the renal system.

DIAGNOSIS

Diagnosis is always made on clinical grounds. Various criteria were proposed by different authors.

Table 1: GURD's Criteria [11]

Major criteria (one essential for diagnosis)	Petechial rash
	Respiratory insufficiency
	Cerebral involvement
Minor criteria (four essential for diagnosis)	HR >120 bpm
	Temp > 39.4°c
	Retinal signs- fat or petechiae
	Jaundice
	Renal signs- anuria or oliguria
Laboratory findings (one essential for diagnosis)	Thrombocytopenia
	Anaemia
	High ESR
	Fat macroglobulinemia

Table 2: SCHONFELD's Criteria [15]

Petechiae	5
Chest x-ray changes (diffuse alveolar infiltrates)	4
Hypoxemia (PaO2 < 9.3 kPa)	3
Fever (> 38°c)	1
Tachycardia (>120 bpm)	1
Tachypnea (>30/min)	1
Confusion	1

Total score > 5 required for diagnosis

Table 3: LINDEQUE's Criteria [16]

1	Sustained PO2 < 8 kPa
2	Sustained PCO2 > 7.3 kPa or pH < 7.3
3	Sustained respiratory rate >35/min despite sedation
4	Increased work of breathing- dyspnea, accessory muscle use, tachycardia and anxiety

Lindeque *et al.* [16] suggested that the FES can be diagnosed on the basis of respiratory status alone (Table 3).

Laboratory studies

Mostly nonspecific

- Thrombocytopenia, anemia and hypofibrinogenemia can occur.
- Decreased hematocrit occurs within 24-48 hours and is attributed to intra-alveolar hemorrhage.
- Cytologic examination of urine, blood, CSF and sputum may detect fat globules.
- ECG findings are usually normal but may show right heart strain or ischemia.

Imaging Studies

- **Chest radiography:** Serial radiographs reveal increasing diffuse bilateral pulmonary infiltrates within 24-48 hours of onset of clinical findings (snow storm appearance).
- **Noncontrast head CT:** CT performed because of alterations in mental status. Findings may be normal or may reveal diffuse white-matter petechial hemorrhages consistent with microvascular injury.
- **Nuclear medicine ventilation /perfusion imaging of the lungs:** Performed for suspicion of pulmonary embolus, the findings from this

scan may be normal or may demonstrate subsegmental perfusion defects.

- **MRI:** sensitive in detecting cerebral FES. Multiple, nonconfluent, hyperintense lesions were seen on proton-density- and T2-weighted images [17].
- **Transcranial Doppler sonography:** Cerebral micro embolic signals were detected as long as 4 days after injury [18].
- **Transesophageal echo cardiography (TEE):** TEE may be of use in evaluating intra operative release of marrow contents into the bloodstream during intramedullary reaming and nailing. The density of the echogenic material passing through the right side of the heart correlates with the degree of reduction in arterial oxygen saturation [19].
- **Bronchoalveolar lavage (BAL)** with staining of alveolar macrophages for fat [20].

TREATMENT [4, 19]

Treatment is essentially preventive and supportive. These include cardiovascular and respiratory resuscitation and stabilization. Maintenance of intravascular volume is important because shock can exacerbate the lung injury caused by FES. 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. Adequate analgesia is important to limit the sympathomimetic response to injury.

No specific drug therapy for FES is currently recommended. The use of corticosteroids in massive doses may be considered for initial prophylaxis, or as therapy if lung function deteriorates. It must be emphasized, however, that while steroids may be of benefit, there is no documented and standardized proof of this.

Other specific measures which have been proposed for the treatment of fat embolism include alcohol, heparin, dextran 40, and aprotinin. All of these agents have supporters, but no agreement has been reached as to the benefits of their use, largely because trials have shown conflicting results. Early stabilization of long bone fractures is recommended to minimize bone marrow embolization into the venous system.

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