

# fat embolism

## general

- fat embolism occurs in approximately 90% of patients with long bone fractures
- only 3-4% of these go on to develop florid fat embolism syndrome with severe hypoxia, decreased level of consciousness & petechiae

## risk factors

- FES is most frequently seen status post lower extremity and pelvic trauma, intramedullary nailing of long-bone fractures, hip arthroplasty, and knee arthroplasty.
- However, FES has also been described in association with diverse diseases, such as sickle cell disease, acute pancreatitis, and diabetes mellitus and with liposuction procedures, burns, decompression sickness, and total parenteral nutrition infusion.

## clinical presentation

- there is a spectrum of presentation ranging from mild hypoxia to full blown FES
- 90% of patients will develop symptoms within 24 hours of injury but some have a latent period up to 72 hours post injury

- diagnosis is normally made using Gurd's criteria with the presence of one major and four minor signs necessary to make the diagnosis:

### MAJOR CRITERIA:

- (i) petechial rash
- (ii) respiratory symptoms with bilateral signs and positive radiographic changes
- (iii) cerebral signs unrelated to head injury of other condition

### MINOR CRITERIA:

- (i) tachycardia
- (ii) pyrexia
- (iii) retinal changes (fat or petechiae)
- (iv) urinary changes (anuria, oliguria, fat globules)
- (v) sudden drop in haemoglobin
- (vi) sudden thrombocytopenia
- (vii) high ESR
- (viii) fat globules in sputum

- other diagnostic criteria include Schonfeld's and Lindeque's criteria

### cardiorespiratory features:

- FES always involves pulmonary compromise. The presentation may range from subclinical shunting to fulminant pulmonary failure.
- In response to the lodging of fat particles in the pulmonary vasculature, the patient may present with right-sided heart failure, cardiovascular collapse, or severe hypoxia.

### neurological features:

- Frequently there is cerebral involvement. Cerebral symptomatology may be due to paradoxical fat embolization to the central nervous system and/or a response to the severe hypoxia associated with this syndrome.

### skin:

- a petechial rash (axillae, anterior chest or conjunctivae) will appear in 25-50% of patients

### eyes:

- retinal findings include exudates, haemorrhage and cotton wool spots

### systemic:

- fever & tachycardia are common

## pathophysiology

- The pathophysiology of FES is complex and probably has both a mechanical component as well as a secondary biochemical process.
- In the initial phase, fat and marrow are displaced from the bones, enter the venous system, and travel through the heart to enter the lungs. There the emboli may cause shunting, severe hypoxemia, and right ventricular dysfunction. Analogous to gas emboli, the fat may travel, paradoxically, to other organs via the systemic circulation either by transpulmonary passage or through an intracardiac shunt, most commonly through a patent foramen ovale.
- The secondary phase may involve inflammatory mediators responsible for the interstitial edema or acute respiratory distress syndrome that may ensue.
- Additionally, bone marrow contains thromboplastin that may activate coagulation cascades.
- These mechanisms may be responsible for the delayed petechial rash seen 24 to 48 hours after the initial event in approximately 50% of patients with FES.

## diagnosis

- BAL can aid the diagnosis with sensitivity and specificity increased by serial lavage; look for fat laden macrophages

- at CT scan may show cerebral oedema and while petechial haemorrhage is common large haemorrhages are rare

### laboratory findings include:

- (i) hypoxaemia often with a large Aa gradient
- (ii) hypocarbia
- (iii) decreased Hb
- (iv) thrombocytopenia
- (v) coagulopathy
- (vi) hypocalcaemia
- (vii) bilateral pulmonary infiltrates on chest X-ray

## management

### prophylaxis:

- early immobilisation of fractures reduces the incidence of FES

### treatment:

- the mainstay of treatment is supportive & there is no definitive treatment available
- principle of therapy is to maintain adequate oxygen delivery through adequate Hb, cardiac output & SaO2
- right sided pressures may be high due to pulmonary hypertension
- inotropes, vasopressors and diuretics may be useful
- there is no role for aspirin, dextran, ethyl alcohol or heparin and the place of corticosteroids is unclear

## prognosis

- the pathophysiology in fat embolism is completely reversible and with appropriate supportive care survival rates should approximate 100%; historical mortality rates are 10% with most deaths attributable to respiratory failure