FAT EMBOLISM

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Introduction

- **Fat Embolism** is the presence of fat particles within the microcirculation, while **FES** is the systemic manifestation of fat emboli within the microcirculation. Common systemic manifestations include respiratory distress, altered mental status, and a rash.

- Embolized fat within capillary beds cause direct tissue damage as well as induce a systemic inflammatory response resulting in pulmonary, cutaneous, neurological, and retinal symptoms. This is most commonly seen following Orthopedic Trauma.

- Rare cases of FES have been reported to occur following bone marrow transplantation, osteomyelitis, pancreatitis, alcoholic fatty liver, massive soft tissue injury , severe burns and even liposuction.

- almost all patients with fractures will have fat globules detected in the blood or develop transient hypoxia, the incidence of FES is much lower

- FES is commonly associated with traumatic fracture of femur, pelvis, and tibia, and, postoperatively, after intramedullary nailing and pelvic and knee arthroplasty.

- In his initial study defining the clinical criteria for FES, Gurd reported the incidence of FES as 19% in a group of trauma patients.As **early operative fixation**of long-bone fractures has become standard care, modern studies report an incidence of FES between 0.9% and 11%

Risk Factors

- young age
- closed fractures
- multiple fractures
- conservative therapy for long-bone fractures.
- Factors which increase the risk of FES after intramedullary nailing are over-zealous nailing of the medullary cavity, reaming of the medullary cavity, increased velocity of reaming and increase in the gap between nail and cortical bone.

Pathophysiology

- Fat particles enter the circulation and cause damage to capillary beds.
- The two leading theories for the formation of fat embolism are **the Mechanical Theory(Gassling et al)** and **Biochemical Theory**
- The mechanical theory proposes that obstruction of the systemic vasculature by fat embolism occurs from the direct release of bone marrow into the venous system following trauma. An elevated intramedullary pressure following trauma leads to the release of fat through open venous sinusoids. The embolized fat obstructs capillary beds.
- Microvascular lodging of the droplets produces local ischemia and inflammation, with concomitant release of inflammatory mediators and vasoactive amines and platelet aggregation.

- The biochemical theory provides an alternate explanation for fat embolization. This theory proposes that the inflammatory response to trauma causes the release of free fatty acids from the bone marrow into the venous system. The elevated free fatty acids as well as the inflammatory mediators cause damage to capillary beds. Elevated free fatty acid levels have been associated with hypoxemia. Free fatty acids have also been shown to induce inflammation within the lungs

- Regardless of the mechanism initiating fat embolism, the end result is an intense inflammatory response. Capillary beds develop increased permeability and inflammatory mediators damage surrounding tissues. In the lungs, this induces lung injury that is indistinguishable from ARDS.

Clinical features

- Embolized fat droplets can travel to microvasculature throughout the body.
- Fat has been reported to embolize to the lungs, brain, skin, retina, kidneys, liver, and even the heart.
- Presenting signs are nonspecific and include tachypnoea , tachycardia, and fever. Patient may have a petechial rash. Specific symptoms are dependent on the organ systems involved.
- The pulmonary circulation is most commonly affected in FES, with up to 75% of patients experiencing respiratory depression. The degree of respiratory dysfunction can range from mild hypoxia requiring supplemental oxygen to ARDS
- Patients may decompensate rapidly to respiratory failure, especially during manipulation of fractures while moving the patient or setting the fracture in the operating room.

- FES can cause nonspecific neurological symptoms. Symptoms are believed to arise from cerebral edema rather than ischemia, so symptoms are nonlateralizing . Patients may become lethargic or restless.

- Dermal involvement results in a petechial rash, reported in approximately 50% of patients. This rash tends to be transient, lasting less than 24 h. the torso is most commonly affected.

- Microvascular injury of the retina results in hemorrhagic lesion of the retina, seen in 50% of patients. These lesions are self-limited, disappearing within weeks. Residual visual deficits are uncommon.

Diagnosis

- Since FES is a heterogeneous disease with no pathognomonic features, its diagnosis can be challenging.

- Certain criteria have been put forward.

- **Gurd and wilson's criteria for FES**

- Respiratory distress
- Cerebral symptoms in non-head injury patients
- Petechial Rash

Minor criteria

- Tachycardia (>110 bpm)
- Fever (>38.5 C)
- Jaundice
- Renal changes
- Retinal changes
- Drop in hemoglobin
- New onset thrombocytopenia
- Elevated ESR
- Fat macroglobulinemia

2 major criteria or one major criteria and four minor criteria suggest a diagnosis of FES.

- **Schonfeld's scoring system**

Sign/Symptom
• Petechial Rash (five points) • Diffuse infiltrates on chest x-ray chest (four points) • Hypoxemia (three points) • Fever (one point) • Tachycardia (one point) • Tachypnea (one point) • Confusion (one point)
Score > 5 diagnosis FES

- **Lindeque's criteria for FES**

Criteria
• pO2< 8 kpa
• pCO2> 7.3 kpa
• Respiratory rate >35/min, in spite of sedation
• Dyspnea, tachycardia, anxiety

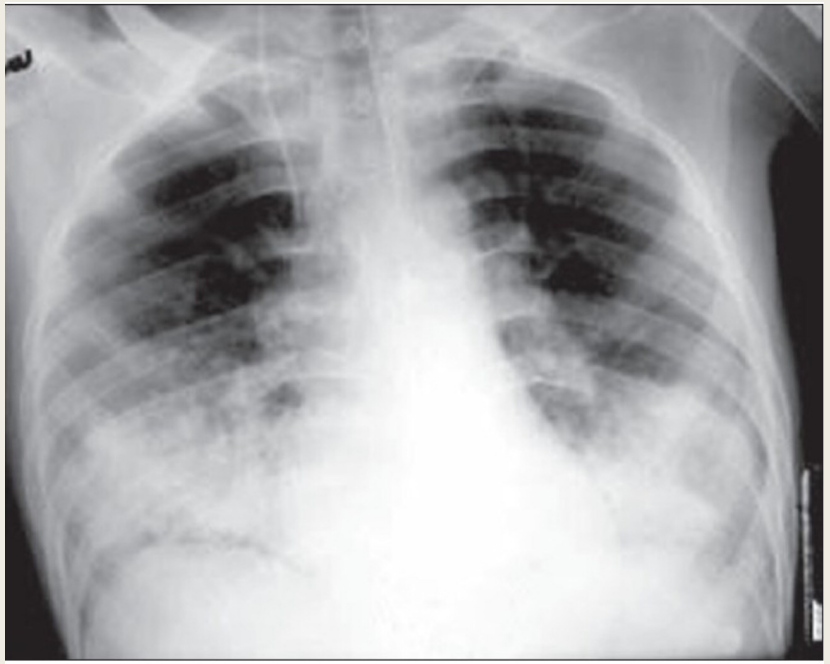
Investigations!!

- None of these criteria have been validated or have been universally accepted.
- In the acute setting **respiratory distress** is the most clinically significant feature of FES.
- Patients will demonstrate **HYPOXEMIA** while on room air on arterial blood gas.
- Laboratory and imaging tests can help in the diagnosis, but are NON-SPECIFIC
- Thrombocytopenia, anemia, hypofibrinogenemia, and increased erythrocyte sedimentation rate (ESR) are seen in FES, but are nonspecific findings. A decrease in hematocrit occurs within 24–48 h and is attributed to intra-alveolar hemorrhage.

- Chest x-ray will often show diffuse interstitial infiltrates while chest CT scan will show diffuse areas of vascular congestion and pulmonary edema.
- Bronchoalveolar lavage (BAL) has been heavily investigated as a diagnostic tool for FES. Lipid inclusions within macrophages can be quantified BAL. BAL though is invasive and time intensive. It has not become widely used in diagnosing FES.

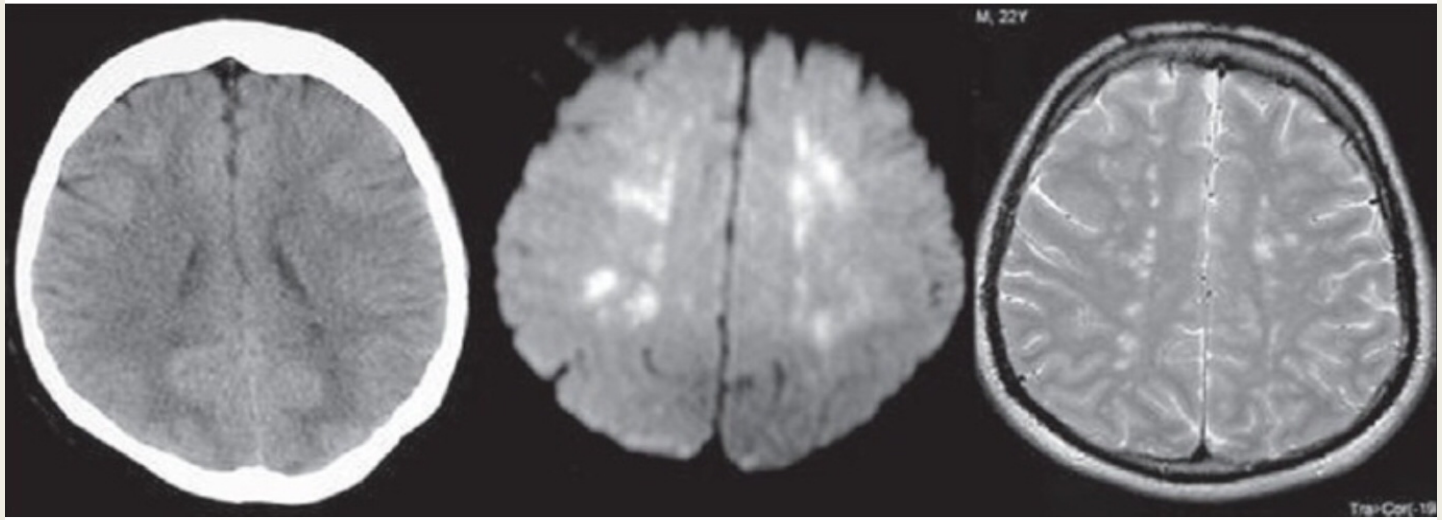
- Microscopic examination of blood, urine, or sputum may show fat globules, but again this finding is nonspecific.
- With the absence of specific tests or criteria the diagnosis of FES is dependent on the clinical acumen of the treating physician.

- Chest radiography: Serial radiographs reveal increasing diffuse bilateral pulmonary infiltrates, fleck-like pulmonary shadows (‘snow storm’ appearance), increased pulmonary markings, and dilatation of the right side of the heart within 24–48 h of onset of clinical findings.



AP radiograph of the chest showing bilateral basal air space–filling lesions (consolidation)

- Magnetic resonance imaging brain is more sensitive than CT scan and diagnosis can be made earlier. It will show typical white matter changes along the boundary zones of major vascular territories



CT image showing minimal hypodense changes in periventricular region, which are more evident in DWI and T2WI as areas of high signals. Constellation of findings along with clinical data is characteristic for FES.

TREATMENT

- Corticosteroidtherapy has been proposed as a potential therapy for FES by limiting freefatty acid levels, stabilizing membranes, and inhibiting complement mediated leukocyte aggregation. Meta-analysis of seven randomized trials using prophylactic corticosteroids in patients with long-bone fractures found that corticosteroids reduce the risk of FES by 77% (95% CI: 40–91%).
- Methylprednisolone is the most commonly used steroid and dosages range from 6 to 90 mg/kg.
- A 2004 randomized trial found no difference in incidence of FES between patients treated with methylprednisolone. While still controversial, some clinicians administer corticosteroids to patients with long-bone fractures as FES prophylaxis.

- Once a patient develops FES the only proven treatment is **SUPPORTIVE CARE** of the involved organ systems.
- Supplemental oxygen
- mechanical ventilation
- intravenous fluid
- Albumin has been recommended for volume resuscitation in addition to balanced electrolyte solution, because it not only restores blood volume but also binds with the fatty acids and may thus decrease the extent of lung injury.
- ionotropic support with dobutamine may be needed.
- Early operative fixation of long-bone fractures is advocated to reduce the incidence of FES.

- Johnson *et al.* further demonstrated that patients undergoing fixation urgently had an incidence of ARDS of 7% compared to an incidence of ARDS of 39% in patients that had fixation delayed by more than 24 h
- Increased intramedullary pressure during fixation increases the amount of fat emboli entering the circulation. Care must be taken during operative fixation to limit intamedullary pressure.While reaming may increase intramedullary pressure, reaming has not been shown to increase the incidence of FES.

Prevention

- Continuous pulse oximetry monitoring in high-risk patients may help in detecting desaturation early, allowing early institution of oxygen (and possibly steroid) therapy; it would thus be possible to decrease the chances of hypoxic insult and the systemic complications of FES.
- The early fixation of long-bone fracture is important to prevent or to decrease the severity of FES.
- External fixation or fixation with plate and screw produces lesser lung injury than nailing the medullary cavity and venting the medullary canal during nailing, reduces the number of emboli.
- Preoperative use of methylprednisolone may prevent the occurrence of FES.
- Smaller-diameter nails and unreamed nailing have been mentioned as being useful in the prevention of FES.

Conclusion

- With supportive care and early fixation FES has a favorable outcome. The most significant morbidity is associated with the development of ARDS. However, most patients can expect a complete recovery of pulmonary, neurologic, and retinal abnormalities.

- The mortality rate from FES is 5–15%. Even severe respiratory failure associated with fat embolism seldom leads to death.

Thank you