

Fat Embolism Syndrome

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History

- In 1861, **Zenker** described fat droplets in the lung capillaries of a railroad worker who sustained a fatal thoracoabdominal crush injury.
- In 1873, **Bergmann** was first to establish the clinical diagnosis of fat embolism syndrome.

What is it ??

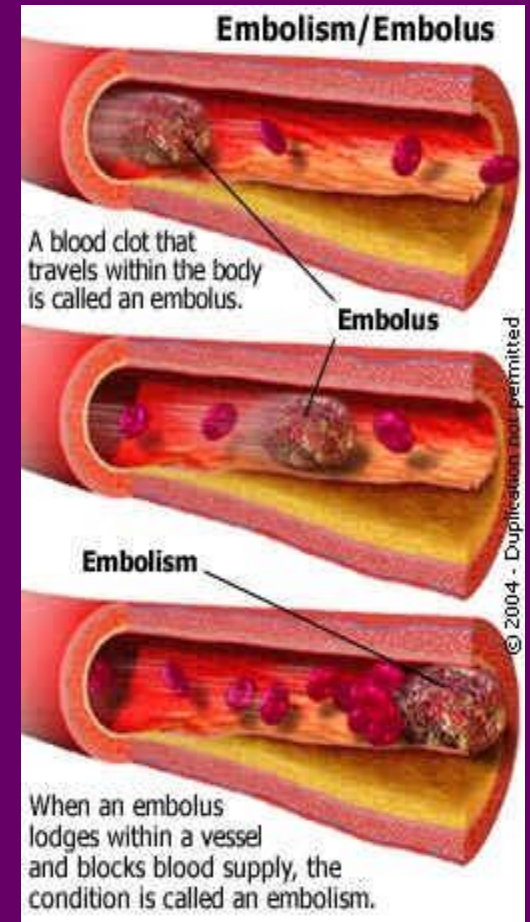
- complex with potentially catastrophic cardiopulmonary and cerebral dysfunction
- Three problems :
- dyspnoea, petechiae and mental confusion

Definitions

Fat Emboli: Fat particles or droplets travel through the circulation

Fat Embolism: fat emboli passes into the bloodstream and lodges within a blood vessel.

Fat Embolism Syndrome (FES): serious manifestation of fat embolism occasionally causes multi system dysfunction, the lungs are always involved and next is brain



Fulminant fat embolism

- sudden intravascular liberation of a large amount of fat causing pulmonary vascular obstruction, severe right heart failure, shock and often death within the first 1-12 h of injury



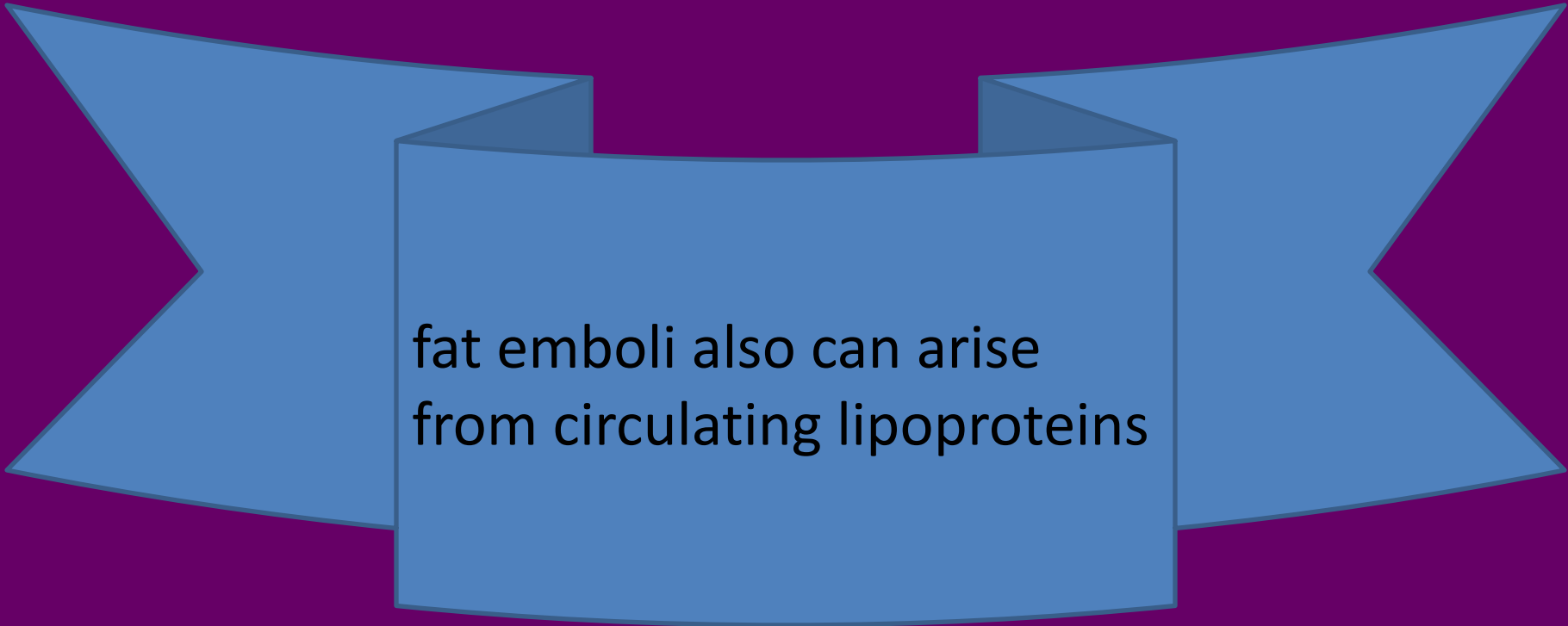
Etiology

Trauma related (95 %)

- Long bone fractures
- Pelvic fractures
- Fractures of other marrow-containing bones
- Orthopaedic procedures
- Soft tissue injuries (e.g. chest compression with or without rib fractures)
- Burns
- Liposuction
- Bone marrow harvesting and transplant

Non-trauma related

- Pancreatitis
- Diabetes mellitus
- Osteomyelitis and panniculitis
- Bone tumour lysis
- Steroid therapy
- Sickle cell haemoglobinopathies
- Alcoholic (fatty) liver disease
- Lipid infusion
- LAST OPD – mnemonic

A blue ribbon graphic with a central rectangular box containing text. The ribbon has a 3D effect with shadows and is set against a dark purple background.

fat emboli also can arise
from circulating lipoproteins

What is frequent ??

- lower extremity and pelvic trauma,
- intramedullary nailing of long-bone fractures,
- hip arthroplasty, and knee arthroplasty

Incidence ??

- incidence of FES was 1 %
- But multiple fractures, adults, high velocity injuries, cementing, hypovolemia
- It can be upto 33 %

Lethal dose

- The acute lethal dose of fat ranges from 20-50 ml.
- The volume of marrow fat from a femur is approximately 70-100 ml.
- Mortality – 10 – 20 %

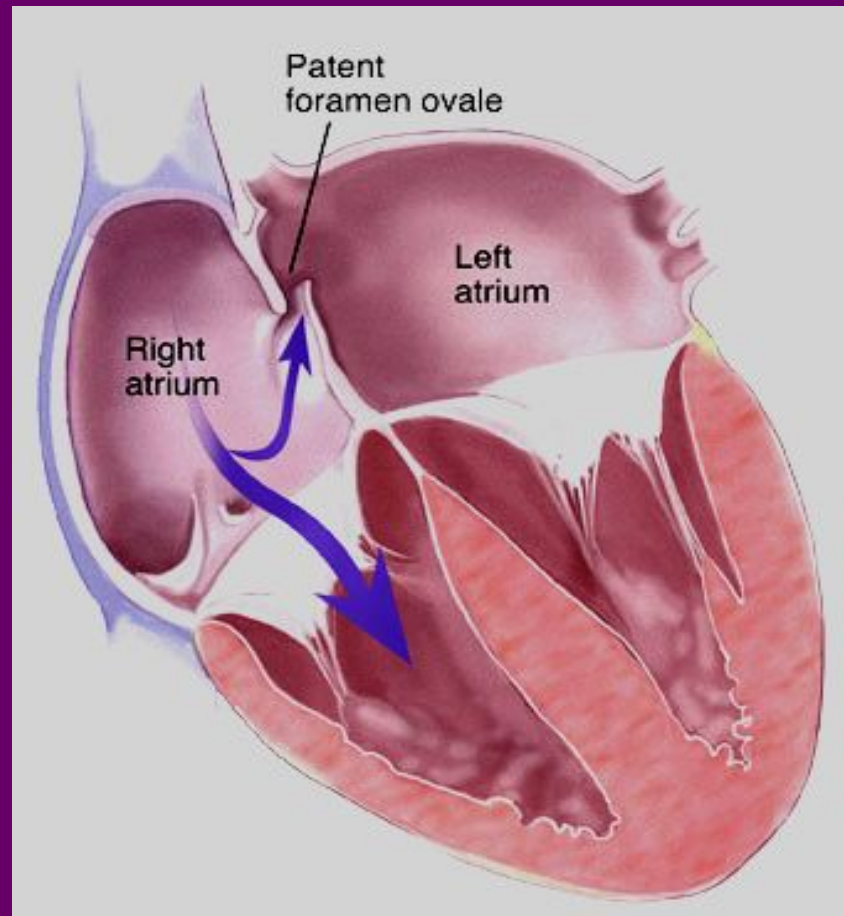
Pathophysiology ??

- **The Mechanical theory (Gauss)**
- **Biochemical theory (Lehmann and Moore)**
- **Coagulation theory**

The Mechanical theory (Gauss)

- Trauma to long bones releases fat droplets
- (10-40 μm in diameter)
- fat droplets enter the torn veins near long bone (intramedullary pressure is higher than the venous pressure)
- They enter lungs
- perivascular hemorrhage and edema- picture of ARDS
- but smaller ones (7- 10 mic.) travel to systemic circulation via ? Patent foramen ovale -

Prevalence of PFO = 25 %



Biochemical theory

- Embolized fat is degraded in plasma to free fatty acids.
- FFA can cause lung injury, cardiac contractile dysfunction
- CRP appears to be responsible for lipid agglutination and may also participate in the mechanism of non-traumatic FES.

Coagulation theory

- Tissue thromboplastin is released with marrow elements following long bone fractures.
- Activates intravascular coagulation
- fibrin and fibrin degradation products, leukocytes, platelets and fat globules combine to increase pulmonary vascular permeability
- Catecholamines are involved

- Can it happen in sickle cell disease ??

Sickling

- Bone marrow necrosis as a result of hypoxia may release fat



- Number of theories means
- Poorly understood ??

Clinical Features

- 12-72 hrs after the initial injury
- Rarely two weeks

Features

- Respiratory changes – 95 %
 - Cerebral changes – 60 %
 - petechiae (33% - 60 %).
-
- Not necessary to follow one by one

Respiratory changes

- Dyspnoea, tachypnoea and hypoxaemia are the most frequent early findings.
- Respiratory failure as ARDS

Cerebral

- The more common presentation is with an acute confusional state
- but focal neurological signs including hemiplegia, aphasia, apraxia, visual field disturbances have been described.
- Seizures and decorticate posturing have also been seen.
- Fortunately, almost all neurological deficits are transient and fully reversible.

Petechiae

- Embolization of small dermal capillaries leading to extravasation of erythrocytes. This produces a petechial rash in the conjunctiva, oral mucous membrane and skin folds of the upper body especially the neck and axilla
- No relation to platelets
- Self limiting (36 hours to seven days)

Petechiae



Petechiae

- Petechiae only rarely appear on the legs and they are never seen on the face or the posterior aspect of the body. WHY ??
- May be –
- fat globules float and therefore distribute to branches of the aorta that arise from the top of the arch, and to the side of the body that is uppermost

Gurd – 1 major + 4 minor

- **Major –**

- Axillary or subconjunctival petechiae
- PaO2 < 60 with FiO2 of > 40
- CNS depression disproportionate to hypoxemia
- Pulmonary edema (PODE – Pneumonic)

- **Minor**

- tachycardia, pyrexia, retinal fat emboli, (Purtscher's retinopathy)urine or sputum fat, Increased ESR, Decreased platelet/ hematocrit.

- exclusion of other posttraumatic causes of hypoxemia
- Beware a lung injury

Lindeque's criteria- # femur , #tibia + 1 feature

Sustained $P_{a_{O_2}} < 8$ kPa

Sustained PCO_2 of >7.3 kPa or a pH <7.3

Sustained respiratory rate >35 breaths min^{-1} , despite sedation

Increased work of breathing: dyspnoea, accessory muscle use, tachycardia, and anxiety

Schonfeld's criteria- fat embolism index- 5 or more

Petechiae	5
Chest X-ray changes (diffuse alveolar infiltrates)	4
Hypoxaemia ($\text{Pa}_{\text{o}_2} < 9.3 \text{ kPa}$)	3
Fever ($>38^\circ\text{C}$)	1
Tachycardia ($>120 \text{ beats min}^{-1}$)	1
Tachypnoea ($>30 \text{ bpm}$)	1
Cumulative score >5 required for diagnosis	



The features are acute, but not abrupt

How to confirm ??

- High index of suspicion and some investigations

CXR usually normal early on, later may show 'snowstorm' pattern- diffuse bilateral infiltrates



Lab values

- **Arterial blood gases :**
- **This reveals a low partial** pressure of oxygen and a low partial pressure of CO₂ with respiratory alkalosis.
- **An unexplained anemia (70% of patients) and thrombocytopenia (platelet count <1,50,000 mm⁻³ in up to 50% of patients).**

Hypocalcemia (due to binding of free fatty acids to calcium) and elevated serum lipase have also been

Reported

Hypofibrinogenemia

CVS

- ECG : sinus tachycardia ; Non specific ST T changes, RBBB,
- Lung scan : ? V/Q mismatch.
- **Transesophageal echocardiography : Fat droplets. PFO, Rt sided dilatation if present**

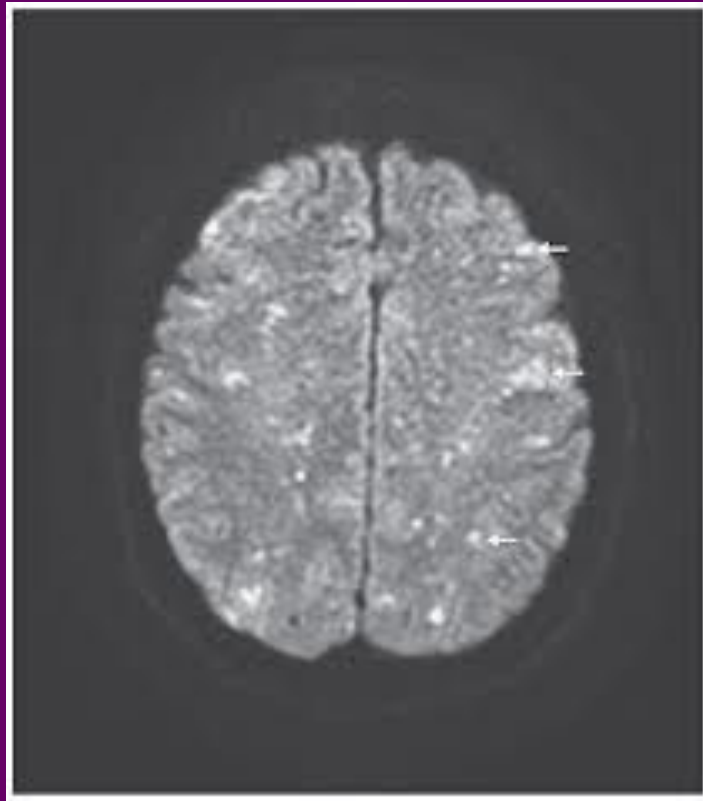
Broncho alveolar lavage

- **BAL : fat droplets.**
- The staining of cells with oil red O after recovery by a standard 150- to 200-mL lavage can identify intracellular fat droplets.
- Can be there in minimal fat embolism – but!!
- **quantitative count of lavage cells containing fat of greater than 30% being significant of fat embolism syndrome**

CT Brain

- White matter petechiae
- Cerebral edema
- Rarely cerebral atrophy due to
- full embolisation

MRI brain – increased signal intensities



Treatment

- Prevention and supportive
- **adequate oxygenation and ventilation,**
- **stable haemodynamics,**
- blood products as clinically indicated, hydration,
- prophylaxis of deep venous thrombosis and stress-related gastrointestinal bleeding,
- Nutrition care

Prevention

- Hole and drill the long bones
- Early immobilization of fractures
- Cementless prostheses or
- bone-vacuum cementing technique
- Less reaming
- **Albumin** also binds fatty acids and may decrease the extent of lung injury
- Methylprednisolone 1.5 to 7.5 mg / kg IV 6 to 12 doses (depending on the risk) ?? Advantage

Prevention

- during cementing
- Hydration
- Oxygenation
- No nitrous

Treatment

- Aspirin
- Heparin
- N acetyl cysteine
- Other speculated therapies such as glucose and insulin, alcohol infusion therapy have theoretical benefit
- Details of mechanical ventilation, Inhaled nitric oxide, inhaled prostacyclins – not covered

Prognosis who survived

- The prognosis for patients who survive fat embolism is good, with recovery from the fat embolism syndrome usually being complete within 2-4 weeks.
- neurological signs may remain for up to 3 months

Summary

- Definitions
- Incidence
- Etiology
- lethal dose
- Theories
- Prevention
- Treatment

Thank you all

