# Fat Embolism Syndrome

**Dr. S. Parthasarathy**
**MD., DA., DNB, MD (Acu),**
**Dip. Diab. DCA, Dip. Software statistics**
**PhD (physio)**
Mahatma gandhi medical college and research institute, puducherry, India

# 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

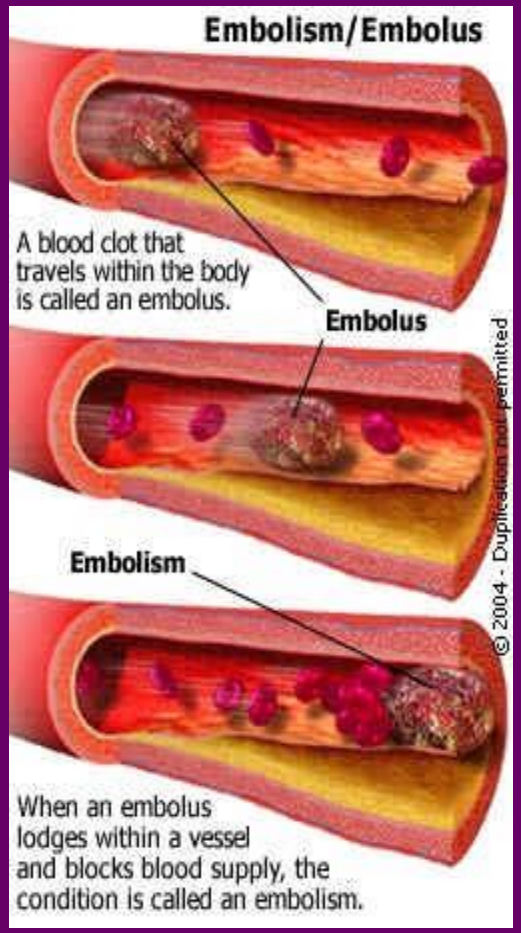
- Three problems :
- **dyspnoea, petechiae and mental confusion**

# 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 – pneumonic

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, hypovolumia
- 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 %

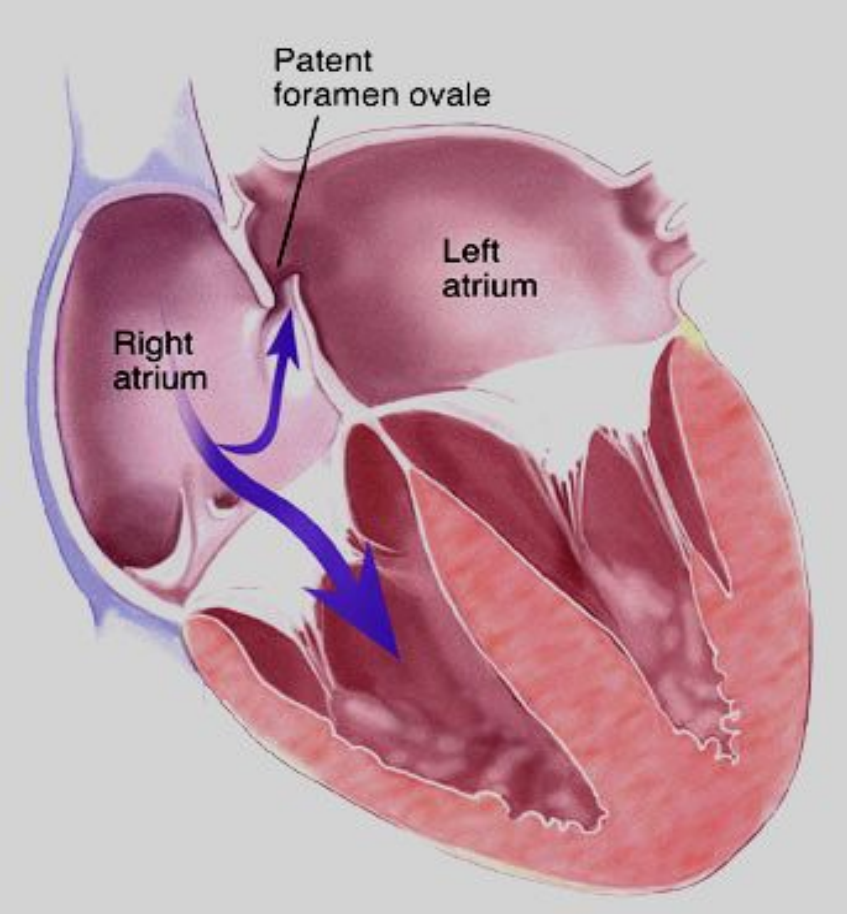
# Pathophysiology ??

- **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 hemorhage 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

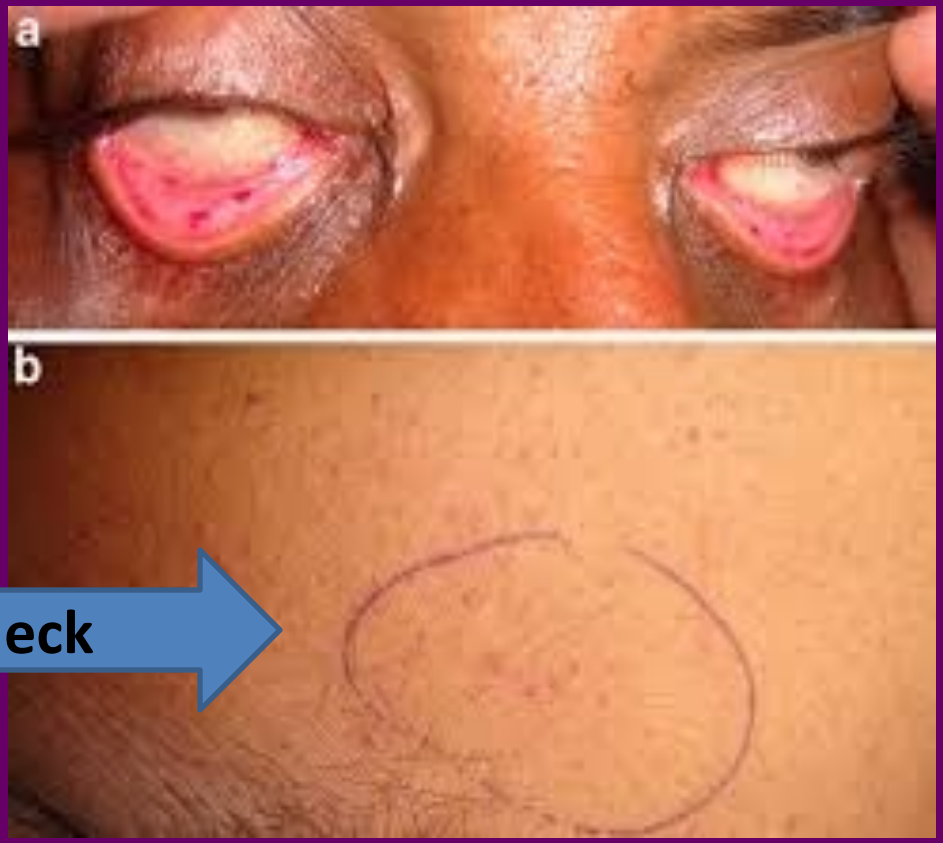
# Cerebral

- 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 subconjuctival **p**etechiae
- Pa**O**2 < 60 with FiO2 of > 40
- CNS **d**epression disproportionate to hypoxemia
- Pulmonary **e**dema ( 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 $Pa_{o2}$ <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

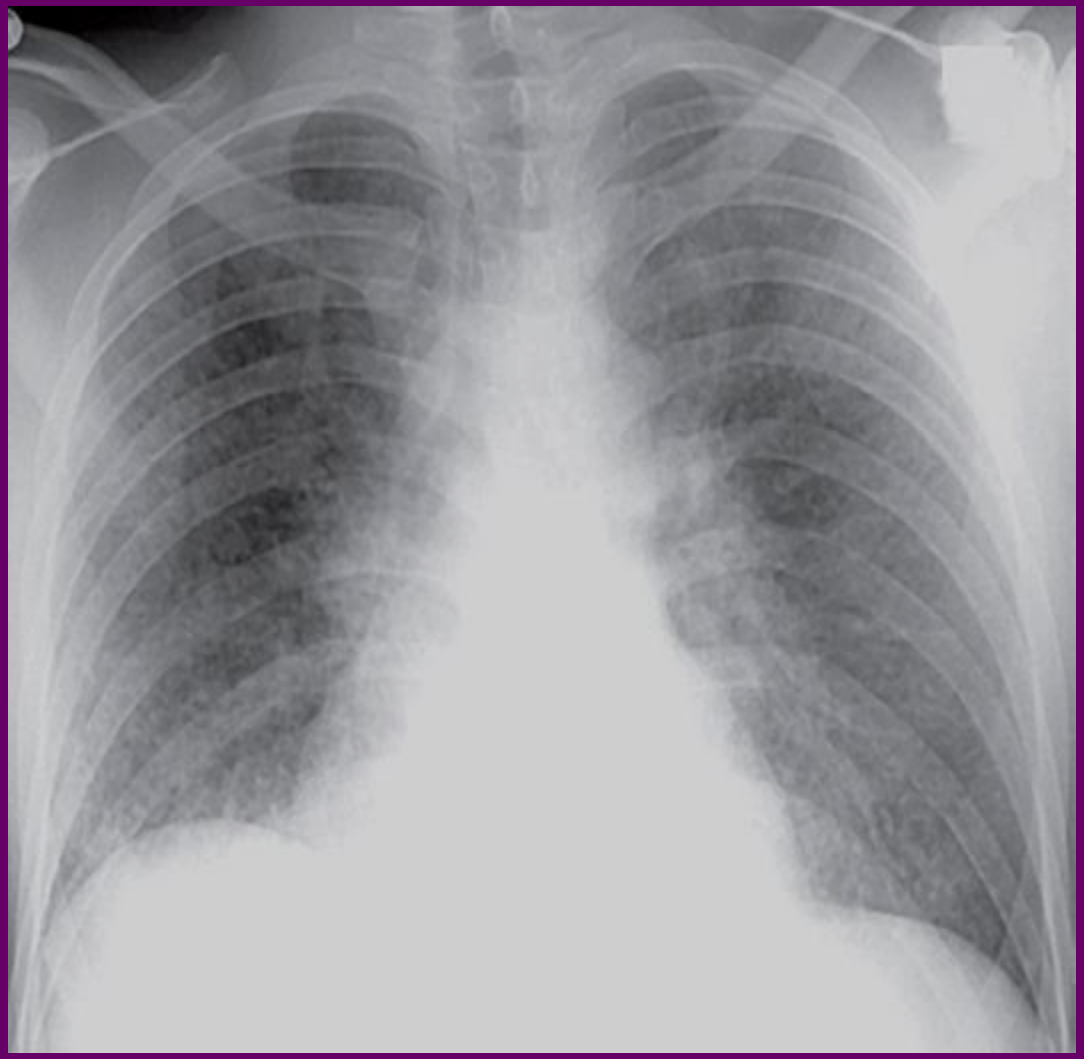
| | |
|---|---|
| Petechiae | 5 |
| Chest X-ray changes (diffuse alveolar infiltrates) | 4 |
| Hypoxaemia ($Pa_{O2} < 9.3$ kPa) | 3 |
| Fever (>38°C) | 1 |
| Tachycardia (>120 beats $min^{-1}$) | 1 |
| Tachypnoea (>30 bpm) | 1 |
| Cumulative score >5 required for diagnosis | |

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

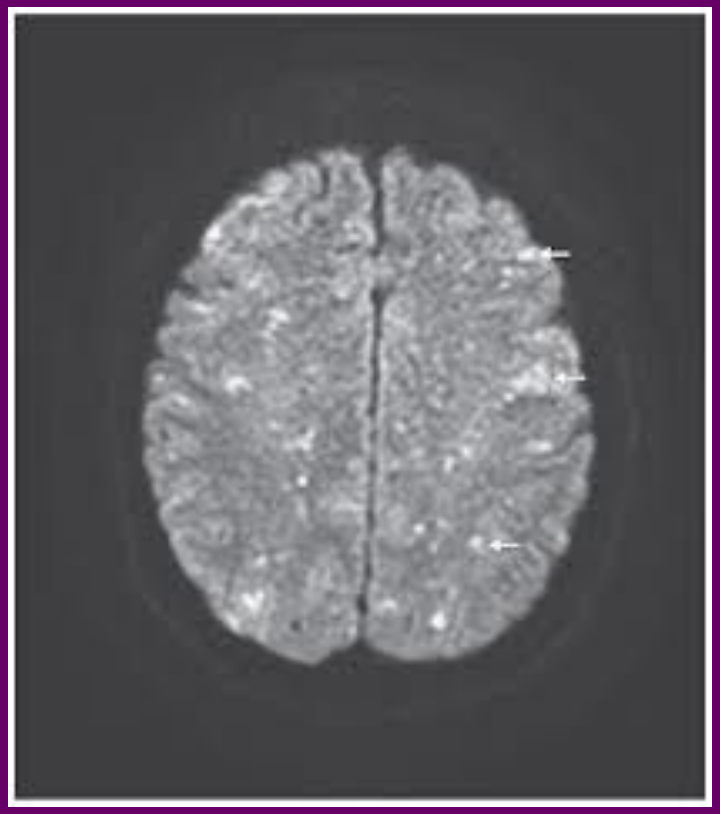
- **BAL : fat droplets.**
- The staining of cells with oil red O after recovery by a standard 150- to 200-mL lavage can identify intracellular fat droplets.
- Can be there in minimal fat embolism – but!!
- quantitative count of lavage cells containing fat of greater than 30% being significant of fat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