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Fat Embolism Syndrome

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Abstract
Fat embolism syndrome (FES) was first described in 1862, but its frequency today is still unclear. A diagnosis of FES is often missed because of a subclinical illness or coexisting confusing injuries or disease. Fat embolism syndrome develops most commonly after orthopedic injuries, but it has also been reported after other forms of trauma such as severe burns, liver injury, closed-chest cardiac massage, bone marrow transplantation, and liposuction. Although FES usually presents as a multisystem disorder, the most seriously affected organs are the lung, brain, cardiovascular system, and skin. Fat embolism syndrome is a self-limiting disease and treatment should be mainly supportive. Many drugs have been used to treat FES, but the results are inconclusive.

Key words Fat embolism syndrome · Fat embolism · Blunt trauma · Fracture complication

Definition and Incidence

Fat embolism syndrome (FES) has been described as occurring after traumatic, surgical, and atraumatic conditions since the nineteenth century.[1–6] It most commonly occurs after lower extremity trauma and intramedullary surgery; however, it can result in a multisystem manifestation of embolization when fat droplets act as emboli, becoming impacted in different organ microvasculature and microvascular beds, causing damage to the small vessels. The incidence of FES is less than 1% in retrospective reviews,[7,8] and 11%–29% in prospective studies.[9–12]

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Historical Perspective

In 1861, Zenker[1] reported the autopsy finding of fat droplets in the lungs of a railway worker who had suffered a severe thoracoabdominal crush injury. Then in 1873, Bergmann[13] diagnosed fat embolism clinically in a patient with a fractured femur. In 1970, Gurd[14] characterized the clinical findings of this phenomenon, which was subsequently named "fat embolism syndrome."

Overview

Fat embolism syndrome occurs most commonly after orthopedic injuries, but it has also been seen after other forms of trauma such as severe burns, liver injury, closed-chest cardiac massage, bone marrow transplantation, liposuction, parenteral lipid infusion, decompression sickness, extracorporeal circulation, acute hemorrhagic pancreatitis, prolonged corticosteroid therapy, sickle cell disease, and carbon tetrachloride poisoning.[5,7,8] The most common surgical procedures predisposing to FES are intramedullary nailing of the long bones, hip arthroplasty, and knee arthroplasty.[15] Although FES may affect multiorgan systems including the lungs, brain, skin, eyes, and heart, pulmonary involvement is the most serious and life threatening.[16]

Because there are often concomitant injuries and preexisting problems, the true incidence and mortality rate of FES are unclear. Bulger et al.[7] reported an incidence of 0.9% among patients with long bone fractures in a 10-year review, whereas Chan et al.[17] reported an incidence of 8.7% in a prospective series of 80 patients with tibial and femoral fractures. Mortality rates range from 10% to 20% in recent trials.[3,5,12]

Pathophysiology

Among the many theories on the pathophysiology of FES, two have gained acceptance.[2,6,7,16]

The Mechanical Theory

This theory suggests that FES is caused by physical obstruction of the pulmonary and systemic vasculature by fat particles from the medullary channel of long bones. The increased intramedullary pressure that occurs following trauma causes bone marrow to be released into damaged venous sinusoids, from where fatty particles travel to reach the lung and obstruct the pulmonary capillaries.

The Biochemical Theory

This theory suggests that different hormonal changes secondary to trauma or sepsis induce the systemic release of free fatty acids. The increased activity of lipoprotein lipase releases circulating free fatty acids, such as chylomicrons, which are toxic to pneumocytes and the capillary endothelium of the lung, causing interstitial hemorrhage, edema, and chemical pneumonitis. This theory may help to explain nontraumatic FES.[1,17,18]

Clinical Presentation

Although FES usually manifests as a multisystem disorder, the most severely affected organs are the lung and brain. There may also be cardiovascular and skin involvement. The diagnosis of FES is based on the patient's history, supported by clinical signs of pulmonary, cerebral, and cutaneous dysfunction. Evidence of arterial hypoxemia in the absence of other disorders confirms the diagnosis.[3,5,7–9,11,15–17,19–21]

Respiratory System

Up to 75% of patients with FES present with some degree of respiratory failure, ranging from nearly asymptomatic hypoxemia to pulmonary distress requiring ventilatory support. An asymptomatic latent period of about 12–48 h precedes the clinical manifestations. Patients become tachypneic, dyspneic, and hypoxic as a result of ventilation–perfusion abnormalities 12–72 h after trauma. The fulminant form of FES presents as acute cor pulmonale with respiratory failure, resulting in death within a few hours of injury.[2,12,16,21]

Central Nervous System

The second most common involvement occurs in the central nervous system, usually with pulmonary disturbances. Central nervous system signs are usually nonspecific and range from a simple headache to rigidity, disorientation, convulsion, confusion, stupor, and coma. These findings are frequently unresponsive to oxygen therapy.[2,3,12]

Cardiovascular System

An invariable cardiovascular sign of FES is tachycardia, but this does not often help with the diagnosis of FES since there are many causes of tachycardia in the trauma patient. Nonspecific ECG changes can be detected if FES leads to myocardial necrosis.[12,16,19,21]

Skin

A petechial rash appears on the upper anterior area of the body, axillae, neck, upper arm, and shoulder. It may also appear in the oral mucous membranes and conjunctivae. The rash results from occlusion of the dermal capillaries by fat causing increased capillary fragility. It tends to be transient and disappears after 24 h. According to Gossling and Pellegrin,[18] the petechial rash is present in 50%–60% of patients.

Eyes

Adams[20] reported observing retinal manifestations of FES in about 50% of patients. These changes consist of cotton-wool exudates and small hemorrhages along the vessels and macula. Most of these findings disappear within a few weeks, but some patients have been left with residual scotomata.[2,3,7,16,20]

Diagnosis

The clinical manifestations of FES were first described by Gurd[14] in 1970, and then refined by Gurd and Wilson[19] in 1974. They stated that at least two major symptoms or signs or one major and four minor symptoms or signs must be present to diagnose the syndrome (Tables 1 and 2).

Hematological Tests

There may be a sudden decrease in the hematocrit level, with thrombocytopenia, hypocalcaemia, hypoalbuminemia, and coagulation abnormalities during FES. The serum lipase and free fatty acids levels may also increase, possibly as a result of the escape of lipoprotein lipase unrelated to pancreatic lipase. High levels of lipoprotein lipase after 5–8 days do not aid in the diagnosis.[7,8,15,16]

Table 1. Gurd's criteria for the diagnosis of fat embolism syndrome[14]

Major criteria	
1	Axillary or subconjuctival petechia. This occurs transiently over 4–6 h in 50%–60% of patients
2	Hypoxemia (PaO_2 <60 mmHg; FiO_2 <0.4)
3	Central nervous system depression disproportionate to hypoxemia, and pulmonary edema
Minor criteria	
1	Tachycardia (>110 beats/min)
2	Pyrexia (>38.5°)
3	Emboli in the retina on fundoscopic examination
4	Fat present in urine
5	Sudden unexplained drop in hematocrit or platelet values
6	Increasing erythrocyte sedimentation rate
7	Fat globules in the sputum
8	Symptoms within 72 h of skeletal trauma
9	Shortness of breath
10	Altered mental status
11	Occasional long tract signs and posturing
12	Urinary incontinence

Table 2. Criteria for the diagnosis of fat embolism syndrome according to Gurd and Wilson[19]

Major criteria	
1	Respiratory insufficiency
2	Cerebral involvement
3	Petechial rash
Minor criteria	
1	Pyrexia (usually <39°C)
2	Tachycardia (>120 beats/min)
3	Retinal changes (fat or petechiae)
4	Jaundice
5	Renal changes (anuria or oliguria)
6	Anemia (a drop of more than 20% of the admission hemoglobin value)
7	Thrombocytopenia (a drop of >50% of the admission thrombocyte value)
8	High erythrocyte sedimentation rate (ESR >71 mm/h)
9	Fat macroglobulinemia

Arterial Blood Gases

Hypoxemia is noted secondary to impaired gas exchange and hypoventilation.

Urine Examination

The presence of fat globules in urine following trauma is neither specific nor pathognomic.

Radiological Findings

Chest X-ray shows multiple flocculent shadows, defined as the "snow storm appearance," caused by diffuse bilateral alveolar infiltration. Computed tomography and magnetic resonance imaging of the brain are used to confirm the extent of the respective organ involvement and exclude alternative pathologies.

ECG

Nonspecific ECG changes may be detected in the presence of myocardial involvement.

Bronchoalveolar Lavage

Many researchers have investigated the role of bronchoalveolar lavage, in the treatment of trauma patients. Oil red-O-positive macrophages may also be related to FES.[2,3,7–9,15–17,20,21]

Presentation of FES

The incidence of FES may be reduced by adequate immobilization of the fracture prior to patient stransport. Although low-dose methylprednisolone provides protection against FES and pulmonary dysfunction after skeletal trauma, the safety of this therapy needs further evaluation. The severity of the clinical syndrome can be reduced by the prophylactic use of oxygen.[10,12]

Treatment

Several trials have shown that FES is usually a self-limiting disease; thus, treatment should be mainly supportive. The results of treatment with drugs, including clofibrate, dextran-40, ethyl alcohol, heparin, aspirin, and steroids, are inconclusive.[17,18,21] It is generally thought that early surgical immobilization and fixation decrease the incidence of pulmonary complications of FES.[2,3,5,15] Maintaining the oxygenation of the peripheral tissues is of utmost importance. Facemask mechanical ventilatory support is indicated when the partial pressure of oxygen cannot be maintained over 60 mmHg.[16] Human albumin should be given for fluid replacement because of its ability to bind to free fatty acids. The use of dextran-40 is helpful for decreasing blood viscosity, reducing platelet adhesion, reversing thrombocytopenia, and reducing cell aggregation. Aspirin and heparin are not routinely used because of the risk of bleeding from other sites of trauma. Finally, corticosteroids limit the rise of free fatty acids, stabilize membranes, and inhibit complement mediated leukocyte aggregation. Methylprednisolone is the preferred drug, given once or twice, often as divided doses ranging from 9 to 90 mg/kg, to prevent or treat FES. The dose and optimal timing of administration have not been established.

In summary, FES occurs in many traumatic and atraumatic conditions. Preventative measures include early immobilization of fractures and methods to reduce intramedullary pressure during surgical interventions. Treatment is largely symptomatic and should be mainly supportive.

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