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Fat embolism and the accompanying fat embolism syndrome (FES) are conditions that develop when droplets of fat act as emboli. The fat droplets become impacted in the microvasculature, especially of the lungs and brain. The multisystem disorder can also affect the heart, kidneys, eyes, and skin. Fat embolism presents at two different levels:

The microscopic form (subclinical) occurs in more than 90 percent of patients with long-bone fractures and in patients undergoing operative procedures performed on long bones without the use of a tourniquet. Microscopic fat embolism is detected by examination of the serum, urine, or sputum for evidence of fat.

Fat Embolism Syndrome, the most serious form, occurs in 2 percent to 23 percent of patients suffering blunt trauma and related fractures. The varying percentage relates to the severity of the injury. The Mangled Extremity Severity Score was developed to evaluate the potential viability of a limb following trauma and may be a valuable tool in predicting FES (Table 1).³

FES is a serious (potentially life-threatening) condition that usually develops after trauma, most frequently following fracture of a long bone (Figure 1). However, the syndrome has also been associated with blunt trauma, intramedullary procedures, prolonged corticosteroid therapy, osteomyelitis, childbirth, liposuction, fatty degeneration of the liver, pancreatitis, systemic lupus erythematosus (SLE), diabetes, sickle cell anemia, severe burns, coronary artery bypass surgery, massive infection, and conditions causing bone infarction.^{1,2,6}

FAT EMBOLISM a complication



BOLISM of long bone fracture

Recent studies have also shown that FES is not simply a mechanical obstruction by the fat droplets of the small vessels, but that it also causes endothelial injury. The lipoprotein, lipase, causes fatty acids to be released from the impacted fat droplets allowing increased permeability of the microvasculature; fluid leakage into the interstitial spaces (edema) ensues.

History

Experimentally, fat embolism was first observed in 1669 by Richard Lower of Oxford through his work with intravenous injections of various fatty substances, including milk. Lower's work was substantiated in 1842 by François Magendie, a

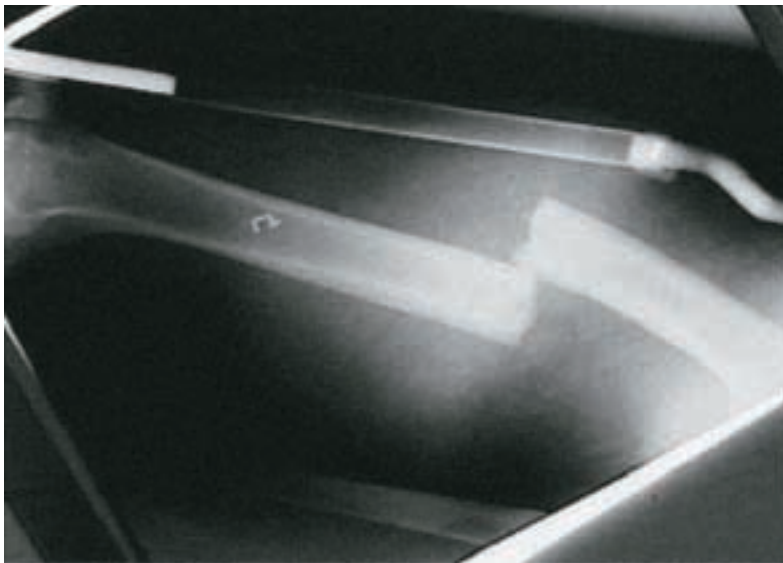


FIGURE 1
Femoral shaft
fracture

French physiologist, while investigating therapeutic intravenous therapy using olive oil. During Magendie's animal studies, the symptoms following the injection of fat were observed, and the changes preceding death were noted. He discovered that fat globules were trapped in the small vessels of the lungs (Figure 2).

Post-traumatic fat embolism was first described by FA von Zenker in 1862. His patient, a railway worker, received a severe thoracoabdominal crush injury that resulted in multiple rib fractures, and rupture of the liver and stomach. He attributed the embolism to aspiration of fatty gastric contents through the exposed hepatic veins.

Fat embolism was first related to bone fracture by Rudolph Wagner in 1862 when he reported lung emboli at necropsy (autopsy) in 48 patients who had suffered bone injury. His further experiments on dogs with bone injury verified the correlation.

The first diagnosis of fat embolism on a living patient was made by Ernst von Bergmann in 1873 on a patient with a fractured femur who subsequently died. It was also von Bergmann who, 10 years earlier through experiments on cats, discovered that the fat was usually trapped in the capillaries of the lungs (pulmonary embolism). In some cases, however, the fat could enter the general circulation (systemic embolism) and affect the liver and other organs, including the kidneys. He also noted that fat could escape into the urine for excretion.

Scriba, in 1880, first combined the experimental, clinical, and pathological observations to conclude that fat embolism occurred after every bone injury, especially fractures, via liberation of liquid bone marrow fat into venous circulation. The embolism could vary in importance from subclinical to the cause of death.⁶

Clinical presentation

In 50 to 60 percent of patients, the onset of FES is gradual, becoming apparent within 24 hours; 90 percent of all cases will become apparent within 72 hours.⁷ Patients with sudden onset of symptoms (usually within 12 hours of injury) with great intensity (referred to as a fulminant course) have a high mortality rate. The patient may first appear restless and complain of vague chest pain. The patient may become drowsy and show a decrease in urine secretion (oliguria). Unexplained fever greater than 101°F (38.3°C) and tachycardia may also be present. Clinical diagnosis is based on the presence of all three of the following criteria within 72 hours following injury.

The three main clinical features of FES are:

1. Respiratory failure manifested in one or more of the following ways: dyspnea, tachyp-

nea, cyanosis due to arterial hypoxemia, or radiograph showing diffuse alveolar infiltrates.

2. Petechiae covering the conjunctiva, retina, oral mucosa, or upper half of the body.
3. Cerebral dysfunction demonstrated by delirium, confusion, or coma.

Incidence

Fat embolism is thought to occur in at least 90 percent of patients with a fracture. FES can occur in as many as 23 percent of the patients with fat embolism, with approximately 10 percent or fewer of those cases proving fatal.³ Cerebral, renal, and cardiac complications are less

likely to cause death than respiratory failure. The risk of FES is decreased in young individuals with fractures and with a tourniquet during an operative procedure on a long bone. The risk of FES is increased when the fracture is closed, when the injury is severe and sustained at a high velocity, and in the presence of malignancy (either primary or metastasis, due to enlargement of venous sinuses related to the tumor).³ Fat embolism and FES cannot be prevented, but several steps can be taken to lower the incidence:

- Immediate fracture reduction and stabilization

Table 1 Mangled Extremity Severity Score (MESS) *Adapted from Wheeler's Textbook of Orthopaedics*

MESS value equal to or greater than seven is an accurate prediction that primary amputation is warranted.

Criteria	Severity	Score
1 Skeletal and/or soft tissue injury	• Low energy <i>(stab; simple fracture; pistol gunshot wound)</i>	• 1
	• Medium energy <i>(compound or comminuted fracture; dislocation)</i>	• 2
	• High energy <i>(high speed motor vehicle accident; rifle gunshot wound)</i>	• 3
	• Very high energy <i>(high speed trauma plus gross contamination)</i>	• 4
2 Limb ischemia	• Pulse reduced or absent but perfusion normal	• 1*
	• Pulseless; paresthesia; diminished capillary refill	• 2*
	• Cool; paralyzed; insensate; numb	• 3*
3 Shock	• Systolic blood pressure always greater than 90 mm Hg	• 0
	• Hypotensive transiently	• 1
	• Persistent hypotension	• 2
4 Age (years)	• Less than 30	• 0
	• 30-50	• 1
	• Greater than 50	• 2

* Indicates that the ischemia score is doubled if the time elapsed between injury and intervention is greater than six hours.

- Administration of low-dose corticosteroids
- Implementation of oxygen therapy.

Pathophysiology

Fat embolism is classified into two pathological types: pulmonary embolism, which may occur as a separate entity, and systemic embolism, which is always associated with pulmonary embolism.

The genesis of both pathological types is the same. The fat originates at the site of the trauma, especially the injured marrow of a fractured bone. The fat cells rupture and, due to a difference in pressure between the marrow and the vessel, allow free fat globules to enter torn veins. Within seconds or minutes, the emboli are taken through the pulmonary artery to the lungs, where the fat globules become entrapped within the pulmonary arterioles and/or compressed within the pulmonary capillaries. The buildup of the fatty material may continue for several hours to a few days. New emboli may be introduced intermittently due to lack of mobilization at the fracture site or treatment of the fracture (closed reduction, surgical manipulation, or application of a fixation device: internal, intramedullary or external). The severity of the injury and the presence of multiple fractures dictate the degree of embolism. Histologically, minor to moderate degrees of pulmonary embolism is of little importance due to the enormous capillary bed

and the large functional reserves within the lungs. Severe embolism can be symptomatic and produce death.

Due to the liquid nature of the fat globules and capillary pressure, it is possible for the fat to continue to move forward in the blood stream through the lungs, enter the aortic circulation, and produce a systemic effect. All tissues and organs are involved with systemic embolism, with the brain (Figure 3) and kidneys the most heavily affected. As with pulmonary fat embolism, systemic embolism also varies considerably in its severity, depending on the degree of pulmonary embolism and the nature of the injury.

Diagnosis

A criterion for diagnosis of FES was established by Fraser Newman Gurd in 1970 (Table 2).⁷ Diagnosis of FES requires that the patient exhibit at least one sign from the major criteria category and at least three minor signs or two major and two minor signs.

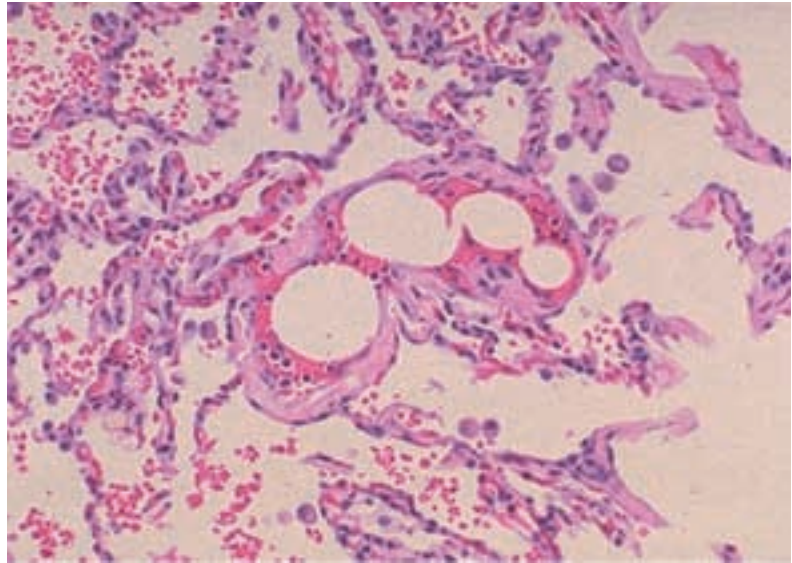
The clinician should be suspicious of the development of FES following any fracture, especially closed long bone, rib, and pelvic fractures. Open fractures, and fractures of the clavicle and sternum show lower incidence of FES.¹ The diagnosis is based on the clinical presentation of the syndrome, making diagnosis in an anesthetized patient difficult. No single specific

Table 2 Gurd's diagnostic criteria for fat embolism syndrome *Adapted from Wheeler's Textbook of Orthopaedics*

Gurd's major criteria	Gurd's minor criteria	Miscellaneous
<ul style="list-style-type: none"> • Hypoxemia • CNS depression that is disproportionate to hypoxemia, and pulmonary edema • Axillary or subconjunctival petechiae • Occurs within 4-6 hours of skeletal trauma 	<ul style="list-style-type: none"> • Tachycardia (greater than 110 beats per minute) • Pyrexia (greater than 38.3 °C) • Retinal emboli upon fundoscopic examination • Fat present in urine • Fat present in sputum • Drop in hematocrit not related to blood loss • Drop in platelets (thrombocytopenia less than 150K) • Increased sed rate 	<ul style="list-style-type: none"> • Occurs within 72 hours of skeletal trauma • Short of breath • Altered mental status • Long tract signs and posturing • Urinary incontinence

diagnostic tool positively confirms the presence of FES; however, several exams provide useful information:^{1,3,5,7}

1. Radiography shows evidence. Fat embolism is thought to have a similar appearance to pulmonary edema (snow storm appearance: fleck-like shadows that are evenly distributed) on chest X-ray.
2. Hematology
 - *Platelet count is decreased (thrombocytopenia).*
 - *Coagulation times are increased (related to thrombocytopenia).*
 - *Hematocrit is decreased.*
 - *Hemoglobin is decreased.*
 - *Serum lipase level is increased.*
 - *Arterial blood gas (ABG), such as arterial hypoxemia or respiratory alkalosis, appears.*
3. Urinalysis bears evidence of fat globules.
4. Neurological examination indicates decreased level of consciousness, convulsion, or personality changes.
5. ECG reveals tachycardia, ST depression, T wave flattening, AV block or bundle-branch block, evidence of right heart strain, or ischemic patterns.
6. Pulse oximetry shows decreased O₂ saturation.
7. Cerebrospinal fluid analysis contains fat globules.
8. Visual examination reveals cyanosis or the presence of petechial rash on the upper body, upper extremities, conjunctiva, and oral mucosa.
9. CT scan discloses evidence of cerebral edema.
10. Sputum analysis uncovers fat globules. A specimen may be obtained with the use of bronchioalveolar lavage (BAL).
11. Fundoscopic exam (ophthalmoscopy) reveals the following: retinal hemorrhage; presence of “cotton-wool” exudates, pallor, and edema in the macular region; or scotomata (area of decreased vision) in the central fields.



12. Transesophageal echocardiography may detect the emboli as they enter pulmonary circulation during a surgical procedure.

Treatment

Treatments for fat embolism and FES vary according to the severity of the symptoms. This is a self-limiting condition; therefore, no “cure” for fat embolism or FES exists. The treatments are considered supportive until the patient spontaneously returns to a homeostatic state. Successful treatment depends on oxygenation to peripheral tissues. Several conventional treatment options are described below:^{1,3,5,7}

1. Provide pulmonary support (according to need)
 - *Supplemental oxygen by face mask*
 - *Mechanical ventilation (positive end-expiratory pressure [PEEP] may be helpful)*
2. Optimize cardiac output to maintain perfusion
 - *Maintain blood pressure (fluid administration; use of inotropic agents such as dobutamine, dopamine, epinephrine, isoproterenol, or norepinephrine)*
 - *Maintain hematocrit*
3. Fluid management according to circulatory status (to decrease pulmonary edema)
 - *Fluid restriction*
 - *Diuretic administration*

FIGURE 2

Lung pathology

Stained section of lung tissue showing arterial vacuoles that result from lipid removal during tissue processing

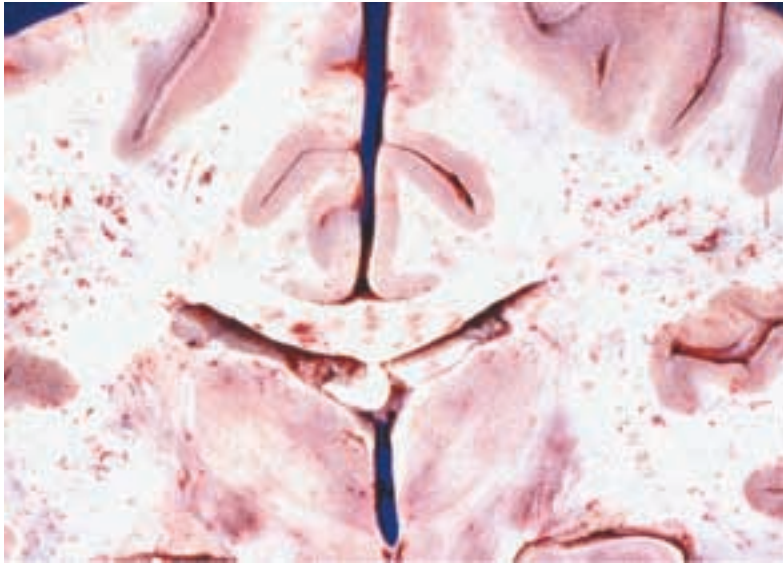


FIGURE 3

Brain pathology

“Purpura”
petechial
hemorrhages in the
brain, prominently
visible in the white
matter

4. Early reduction and stabilization of fracture(s)
5. Administration of corticosteroids

Controversial treatment options include IV ethyl alcohol infusion to inhibit lipase and clofibrate (an antihyperlipidemic) to increase free fatty acid metabolism. Theoretically, use of lipase inhibitors is sound, as they increase the metabolism of intravascular lipids, but the formation of more free fatty acids may cause further damage to the pulmonary capillary endothelium. Administration of aspirin, heparin (also considered a lipase inhibitor), or dextran may be helpful in decreasing platelet adhesiveness; however, the benefits of the anticoagulants in treating FES may be outweighed by additional risk of hemorrhage from recent trauma.

Conclusion

Most individuals with FES recover fully within two to three weeks with appropriate supportive treatment. The overall prognosis is very good, with most patients suffering little to no residual effects of the event. Morbidity and mortality are related to the degree of pulmonary and central nervous system complications.⁷

The patient may suffer from multisystem trauma, making diagnosis and treatment difficult. Other conditions to be considered include pulmonary or cardiac contusion, pulmonary

embolism, shock (septic or hypovolemic), intracranial injury, aspiration pneumonitis, and other types of ARDS (acute respiratory distress syndrome).⁷ FES may be accompanied by intravascular coagulation and osteonecrosis as part of a triad of pathological conditions.¹¹

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