Pulmonary fat embolism

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The author details the history, incidence, symptoms and treatment of pulmonary fat embolism syndrome; a case report of a patient exhibiting the syndrome's classic signs is also presented.

Recovery of an injured patient depends on several factors, among them, the severity of the injury itself, the primary treatment, and the management of post-trauma complications. Fifty years ago, the severely injured patient died of shock or sepsis; thirty years ago, he died of shock or renal failure. Today, if death is not immediate due to hopeless damage, effective resuscitation from the initial shock state and proper treatment of the injury may allow the patient to survive only to later develop a complex post-traumatic syndrome. This syndrome often encompasses endocrinologic, metabolic, nutritional, blood rheologic and clotting disturbances, as well as renal and respiratory insufficiency. The pulmonary damage results from fluid overload, bacterial toxins, shock, oxygen toxicity, blood cell aggregates and tissue and fat emboli. It is this last complication which shall concern us here.

When body homeostasis is altered, blood rheology disturbances are induced and changes in serum fat emulsification occur. More important, however, is the mobilization of depot fat which leads to the commonly found post-traumatic condition of lipemia.

The boundaries between the mobilization of micro- and macro-globules of fat is the important factor here. Fat embolism from large aggregates of fat is the pathologic event. It is found in a large percentage and variety of trauma conditions, especially after damage to fat-containing soft tissues and bone has occurred. These fat macro-globuli adhere to blood cells and fibrin and have the potential to produce embolism in the arteriolar tree. The clinical entity, fat embolism syndrome, is characterized by symptoms exhibited by the affected organ systems. The fat embolus particularly affects the vulnerable pulmonary respiratory function and also the brain, which is so sensitive to low oxygen tension.

History

Fat embolism syndrome has a reported history of more than 100 years. The first human histologic description of the syndrome was made in 1862, in the lung of a patient dying after a severe crush injury. The first clinical diagnosis came in 1873, in a patient treated for a fractured femur.

The first case of pulmonary fat embolism diagnosed in the United States was described by Fenger in the Chicago Medical Journal of 1879. The case report involved a patient admitted to Cook County Hospital who died after...
suffering a fractured femur. In the 1950s, Peltier initiated his series of fat embolism studies which expanded the comprehension of pathogenesis and described the toxic effect of free fatty acids. The result was a description of the respiratory disease theory, now widely accepted as the basis for current therapeutic approaches.

Incidence

The exact incidence of clinical fat embolism syndrome in post-traumatic cases has never been accurately determined in a series large enough to be deemed significant.

The general agreement, however, is that fat embolism syndrome does appear in a high percentage of cases following trauma to fat depots, and is certainly more frequent than it was once believed to be. In fact, one researcher found the syndrome present in 90% of deaths following trauma with fractures. In battlefield casualties, there was a 70% incidence in World War I, a 65% incidence in World War II, and a 39% incidence in the Korean War. In one significant study (10,000 violent deaths), the incidence was 100%. In a study of 557 traffic deaths, 80% had multiple compound fractures and pulmonary fat embolism could be detected in all cases.

Fat embolism syndrome can be seen at any age; there is even a report of a neonatal death attributed to fat embolism. The sparse literature available on this disorder in children gives a false impression of rarity, when, in actuality, several reports have shown postmortem evidence of severe fat embolism in 90% of children dying after injury.

Fat embolism has been associated with many surgical and medical conditions which exist in children as well as in adults.

On the other end of the age scale, clinical evidence shows a higher incidence of the syndrome in elderly patients than in the young. The more severe clinical cases also tend to occur with greater frequency in the elderly. Males exposed to military, industrial and accidental trauma are at the greatest risk of developing fat embolism.

Occurrence

Fat embolism is more likely to occur after multiple fat depot injury—to either soft or bony tissue. One study showed the mortality rate to be 3.4% for tibial fractures with fat embolism, 9% for femoral fractures, and 20% for both femur and tibia. Cases are reported of death from fat embolism after fractures of vertebrae, sternum, riba, clavicle, and ischium. Soft tissue damage, either operative or traumatic, has also been reported as a cause of the syndrome.

The incidence of fat embolism syndrome after open fractures (this in a series from the Vietnam War) was found to be 2%, compared to 30% in closed fractures. However, this incidence was much higher in high velocity missile injuries of the femur than in low velocity damage to soft tissue of the thigh.

An interesting point here is that there is a very low incidence (2%) of fat embolism after intramedullary reaming and nailing of bones. In comparing a series of fractures treated by closed or open reduction, the number of deaths attributed to fat embolism was slightly higher in closed reduction, suggesting the danger of fat embolism in rough manipulative reduction of fractures.

In relation to total hip arthroplasties, the incidence of fat embolism seems to depend on the type of arthroplasty used, that is, how much reaming and compression of the marrow cavities occurs. Some reports suggest that the cement used in such procedures may be toxic enough in itself to aggravate the mild form of fat embolism.

In a controlled series of 854 patients with hip fractures, the incidence of fat embolism was between 4-7% in those treated without operation and about 1% in patients treated by
pinning, nailing or nail plating. It is not yet determined whether the fixation of fractured bones contributes to the prevention of fat embolism.

Fat embolism is usually seen in cases of multiple trauma or massive surgical injury, but may also complicate minor injuries. It has been described after manipulative release of joint contractures, in club foot operations in children and even in fractures of small bones. Conditions other than damage to bone and soft tissue have been described as associated with emboli to the lungs. The highest incidence—37%—occurred in burn cases. The possible sources of fat embolism here are believed to be heat-ruptured fat cells in the skin and subcutaneous tissue and the fatty liver so commonly found in burn patients.

Other types of operative trauma which have produced fat embolism to the lungs are damage to the liver during laparotomy (where fatty cells are compressed into the hepatic veins), cholecystectomy, thoracotomy, cardiac massage, open heart procedures with extracorporeal circulation, traumatic fetal extractions and other childbirth injuries producing damage to the maternal pelvic fat.

Fat embolism also occurs in fatal poisonings caused by the heavy metals, chloroform, ethyl and methyl alcohols and aviation fuel inhalation. It also occurs in clostridial infections, due to the alpha toxins which are released, and in diabetes, due to hyperlipidemia.

Source of fat embolism

Fat embolism is said to occur when fat macroglobules over 20 microns in diameter enter the tissues through the pulmonary and systemic circulation. The normal rheologic characteristics of the blood are seriously altered in the postinjury period, especially in vessels smaller than 100 microns in diameter. The flow rate and pattern are altered, leading to the following sequence of sludging and occlusion: (1) Aggregation of platelets and fat droplets, with the formation of the nidus for emboli, occurring soon after injury; (2) Aggregation of red cells, also occurring soon after injury; and (3) Decrease of flow rate in the postcapillary venules and sinusoids.

This sequence results in the severe alteration of the perfusion of tissues and organs with the resultant tissue hypoxia.

Fat globules are thought to arise from either extravascular or intravascular sources. Since the extravascular source is more widely accepted, we will examine it only.

This extravascular source results from the disruption of fat-containing tissue with an influx of globules into the circulation. The mechanism for this is: liberation of fat globules, tearing of the venules, and a force which absorbs or pushes the globules into the circulation.

In support of this theory are several clinical and experimental studies which indicate: (1) The existence of sufficient fat in long bones to account for the phenomenon of fat embolism; (2) The existence of three elements at the site of the fracture which are essential for embolism—the rupture of fat cells, the influx of the fat from the fracture hematoma into the general circulation, and an increase in local tissue pressure due to the injury; (3) The finding of bone marrow cells in the lungs, together with fat droplets and even small bony particles, appearing soon after fracture; (4) The finding of fat droplets in the venous blood of the damaged limb, in contrast to none in the normal limb; and (5) Prevention of fat embolism from the fracture site by the application of a proximal tourniquet or by vein ligation.

Triggering factors

Although there need not be any other existing conditions present outside of the primary injury, there are three general conditions which are
known to trigger fat embolism. The first of these is hemorrhagic shock. The theory here is that hypovolemia and hypotension are the factors admitting fat droplets into the circulation at the fracture site. However, there is documentation that adequate and rapid blood transfusion for shock results in a decreased fat influx and a reduced incidence of fat embolism.

Disseminated intravascular coagulation is also a condition known to trigger fat embolism. All that can be said here is that the alteration of blood coagulability seems to predispose an injured patient to fat embolism.

Sepsis, the third condition, aggravates or precipitates fat embolism either as a result of the toxins present or as a factor secondary to the consequent shock.

**Symptoms of fat embolism**

Due to the filter action of the pulmonary capillary bed, the incidence of systemic embolism is much lower than that of pulmonary embolism. Only small amounts of fat globules less than 20 microns in size—and usually even less than 15 microns—enter the systemic circulation and are dispersed to nearly every organ of the body. The amount of fat dispersed to each organ is determined by the cardiac output to that organ. The brain is most influenced here; however, the eyes, the kidneys, the myocardium and the skin may also be affected.

The main pathology of the fat embolism syndrome is in the pulmonary system. The symptoms here are usually labeled as either “early” or “second stages.”

Early symptoms are as follows: (1) The patient is pale; (2) The patient has a mild tachycardia of 100-120; (3) There is a drop in hemoglobin—considered a “common” finding after fractures; (4) There is a drop in platelets—frequently considered “normal” with a hematoma; (5) There is an increase in temperature interpreted initially as “resorption of the hematoma;” (6) A chest x-ray shows initial haziness and localized or multiple infiltrates; (7) Arterial blood gases show hypoxemia with a drop in PaCO$_2$ tending toward alkalosis.

These symptoms usually occur within two to three days after the injury. Second stage symptoms then occur. These include: (1) Deepening of the hypoxemia, even with an increase in the inspired oxygen content; (2) Acidosis of combined origin: Respiratory, due to alveolar-capillary block, and metabolic, due to tissue hypoxic metabolites such as lactic acid; (3) Compared with an early 5-15% A-V shunt in the lungs, a 25-30% shunt is now found, with a severe hypoxemia of PaO$_2$ below 50 mmHg; (4) EEG and EKG dysrhythmias occur now and may be severe. Cardiac extrasystoles and fibrillation may be fatal at any instant; (5) Sequelae of hypotension and decreased perfusion appear, including renal and hepatic abnormalities with azotemia and hyperbilirubinemia; (6) Radiologically, progressive patchy infiltrates appear to form lobar or pneumonic consolidation; most commonly found is “snowstorm” patchy infiltrates. These findings will persist for several days after clinical improvement is noted; (7) Blood clotting disturbances may occur, such as drop in platelet count, increase in prothrombin time, decrease in fibrinogen level, decrease in serum calcium, and a clotting factor deficiency. All of these deficiencies are signs of consumption coagulopathy and may lead to disseminated intravascular coagulation.

The overall result of this combination of factors will be a pathologic chain reaction. Commonly, a tracheobronchopulmonary infection appears with subsequent septicemia, adding to the already existent pulmonary damage. The anoxia of the hypothalamus and central nuclei can enhance the pulmonary edema by a neurogenic pathway. Death is a result of severe hypoxemia and metabolic disturbances.
The early stages are reversible, as is occasionally the full-blown lung involvement, provided early, adequate therapy is initiated.

Making the diagnosis

There are no real pathognomonic signs or tests that can be used in diagnosing fat embolism. Diagnosis is, however, suggested in a patient at risk by signs which are significant if there appears to be no other explanation. Some of these signs include: (1) A temperature above 38°C if there is no other sign of infection; (2) Pulse rate of 100-120 accompanied perhaps by an elevation of diastolic pressure; (3) A drop in the hemoglobin without obvious bleeding—due to trapping of red cells in pulmonary and peripheral capillary systems; (4) Petechiae over the neck, shoulders, upper chest and conjunctiae. This is present in only 25% of patients and results from either embolus-produced skin ischemia or from increased capillary fragility and thrombocytopenia; (5) Fat in the urine. This is common, but not necessarily indicative of fat embolism; (6) Staining of tracheal aspirate for fat. This is a significant sign; (7) Elevation of serum lipase following trauma for seven to eight days (elevation for three to five days after trauma is normal).

Treatment of fat embolism

Part of the treatment for a patient with this condition should be prophylaxis for fat embolus, including the immediate and satisfactory immobilization of the injured limb, the replacement of blood volume, and the prevention of hypotension. If the patient does develop signs of fat embolism, the following measures should be instituted: (1) Maintenance of adequate acid-base balance and electrolytes; (2) Frequent EKG and chest x-rays; (3) Administration of corticosteroids to combat chemical and hemorrhagic pneumonitis; (4) Avoidance or treatment of hypoxia by keeping inspired oxygen high enough to keep \(\text{PaO}_2\) above 80 mmHg, but not continuing an \(\text{FiO}_2\) greater than 40% for any longer than 24 hours. To do this, mechanical ventilation may need to be instituted with PEEP, because PEEP reexpands the collapsed alveoli and decreases the shunt; (5) Frequent arterial blood gases are also needed. An arterial line is a must in severe cases; (6) Pharmacologic agents such as aminophylline should be administered for their pulmonaryologic effect; (7) Oxygen consumption should be reduced by lowering body temperature to normal or even subnormal using antipyretics, surface cooling or a lytic cocktail; (8) Two to three units of rheomacrodex over four to six hours should be administered. This desludges the pulmonary and systemic capillary flow; (9) Heparin should also be administered cautiously over the acute phase; (10) Fresh blood should be given; and (11) Antibiotics for pulmonary infections should be administered.

Case report

A 19-year-old male was admitted to our emergency room at 0100 hours, after being involved in a questionable vehicle accident. He was admitted having suffered multiple trauma, including fracture of the transverse process of L 5, fracture dislocation of the pubic symphysis, pelvic hematoma which displaced the bladder to the right, midshaft fracture of the left femur, and a fracture of the fifth metacarpal of the right hand.

Upon arrival in the emergency room, our patient was conscious, oriented, and cooperative, but obviously in pain. Following initiation of intravenous therapy, Foley catheter and x-rays, he was transferred to the intensive care unit where a Steinmann pin was inserted in the left tibia. The patient was subsequently placed in traction. Oxygen was given through a cannula, and throughout that morning he received several units of whole blood in
addition to plasmanate and lactated Ringers’ solution. He was also started on a regimen of heparin, 5000 units every 6 hours. His initial chest x-ray was read as “clear,” but proved to be a technically inadequate film.

For the next 24 hours or so, the patient remained in stable condition. His hematocrit remained at approximately 40% with the help of the whole blood and other volume expanders. However, a look at his TPR record showed that a significant change was taking place. His pulse, temperature and respiration rate were all slowly increasing, so that by 1000 hours on the second day, he had a pulse of 130, a respiratory rate of about 50, and a temperature of 101.5°F. The patient also complained that he was short of breath and very uncomfortable. During this particular morning, petechial hemorrhages were noted over the upper chest. At this time, the patient’s arterial blood gases showed a pH of 7.46, pO₂ 42, pCO₂ 34 and he was still receiving nasal oxygen. A chest x-ray at this time showed an alveolar pattern with air space consolidation.

A decision was subsequently made to intubate the patient and place him on the MA-1 ventilator, due to his rapidly worsening condition. He was intubated nasally and started on a regime of morphine and Valium® for sedation, and pancuronium for ventilator control. The first arterial blood gas (ABG) following intubation showed a pH of 7.40, pO₂ 62, pCO₂ 34 and he was still receiving nasal oxygen. A chest x-ray at this time showed an alveolar pattern with air space consolidation.

On the fourth day, a chest x-ray showed more consolidation in the lower lobes. We were able to reduce the patient’s PEEP to 12 cm with a FIO₂ of 45% and still maintain adequate oxygenation. His ventilator settings at this time were FIO₂ 45%, PEEP 12 cm, tidal volume 1100 cc, rate 10/min.

During these days, the patient remained febrile (temperature 101-102°F) and was receiving Keflin®. A culture from his tracheal aspirate grew staphylococcus, coagulase positive, which was sensitive to Keflin®.

On the next day, there was no change on the patient’s chest x-ray. During this day, we were able to reduce his PEEP gradually to 3 cm, and keep his FIO₂ at 40%. During the following three days, he tolerated the ventilator well with the help of the sedation and occasional relaxants, plus much verbal reinforcement. The patient remained on a regime of frequent tracheal suctioning. Due to the traction, he could not be turned. However, he did remain in as much of a semi-Fowler’s position as his fractures would allow.

On the sixth day, we were finally able to do away with the PEEP and even instituted intermittent mandatory ventilation (IMV), which can be done with the MA-1. The patient maintained his PaO₂ above 100 mmHg on the IMV with an FIO₂ of 40%. His hourly sedation was discontinued and he was given only small doses of Demerol® as needed for pain.

On the seventh day the patient’s chest x-ray was read as clearing, and he was placed on a T-piece with FIO₂ of 40%. When it became apparent that he could do quite well without ventilator support and still maintain adequate oxygena-
tion, he was extubated and placed on a face mask with 40% oxygen.

The patient continued to do well that day and the next; consequently, the arterial line was discontinued. There was no sequelae from the week's cannulation. The patient received oxygen by mask for the next two days until ABGs were adequate on room air. He had no laryngeal problems resulting from the prolonged intubation since a soft-cuffed tube had been used.

Summary

Fat embolism syndrome seems to be found with increased incidence in violent deaths. Its occurrence is not limited by age; however, cases in the elderly seem to be more severe. Children are usually expected to recover.

Fat embolism syndrome most often occurs in post-traumatic and postoperative patients, although it may also arise as a complication of several medical conditions. Adequate immobilization of fractures is essential in the prevention of this syndrome.

There is no specific treatment for fat embolism syndrome; however, correction of the condition’s homeostatic disturbances, using daily intensive monitoring, has resulted in many cases of recovery.

Preventive measures should include the effective treatment of shock and the stabilization of all fractures, as well as maintenance of a normal blood rheology.

Curative means should include the avoidance of hypoxia by mechanical ventilation and PEEP, anti-inflammatory action of corticosteroids, maintenance of a proper blood flow, utilization of clotting mechanisms and general supportive measures.

REFERENCES


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This article was written while Maj Lenig was stationed with the U.S. Army in Nurnberg, Germany and was originally presented at the U.S. Army—Europe's annual conference for nurse anesthetists and operating room nurses in 1977.