Traumatic Fat Embolism: Recent Clinical Experience in Combined Arterial injury and Long Bone Fracture

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Fat embolization is a frequent accompaniment of long bone fracture; however, this phenomenon only occasionally leads to the clinically recognized traumatic fat embolism syndrome. Three recent cases are presented of fat embolization following long bone fractures and associated with penetrating arterial injury. Consequences illustrated ranged from asymptomatic fat globules in the urine, through serious pulmonary insufficiency, to central nervous system damage with death. Treatment should be directed at effective mechanical ventilatory support to avoid hypoxemia.

TRAUMATIC fat embolism is a striking symptom complex which may complicate long bone fractures. It is usually clinically recognized as pulmonary insufficiency. The manifestations encompass a wide spectrum varying from asymptomatic lipuria, through pulmonary insufficiency, to fatal central nervous system damage. Autopsy records show that patients with multiple fractures frequently have some degree of fat embolization.\(^1\),\(^2\) Unfortunately fat embolism has the grave potential of transforming a successfully executed vascular operative procedure or a routine case of orthopaedic fracture management into a clinical tragedy. Our recent experience in managing three patients who had combined arterial injury and long bone fracture with secondary fat embolism illustrates the variety of forms in which this complication may occur.

Report of Cases

Case 1. A 48-year-old black man sustained a gunshot blast to the right distal thigh (Figure 1). On arrival in the emergency room he was pulseless with no obtainable blood pressure. Resuscitation with rapid crystalloid infusion raised the systolic blood pressure to 90 mm of

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Figure 1
Radiogram of right distal femur in Case I demonstrating the extensive comminuted fracture which resulted from a close range shotgun blast.

Hg and he was then taken directly to the operating room. A compound, comminuted fracture of the distal femur with an associated tense hematoma was stabilized by means of Roger-Anderson external skeletal apparatus. A lacerated femoral artery and vein were primarily repaired. His postoperative course was unremarkable, except for fever lasting eleven days. Throughout the postoperative course, his lungs were radiologically clear. However, his urine consistently contained fat globules. The lowest arterial oxygen tension recorded was 54 mm of Hg. There was no evidence of confusion or neurological impairment.

COMMENT: Although there was early clear-cut evidence of fat embolization by the appearance of lipuria the effect on the patient's clinical course was negligible.

Case 2. A 25-year-old black man was robbed and thrown in the path of an automobile, suffering severe crush and avulsion injuries of the left leg and ankle. In our emergency department he was noted to have evidence of cerebral concussion with unstable vital signs. Examination revealed a compound fracture of the proximal left tibia and fibula, fracture dislocation of the left ankle, and probable associated arterial injury. At surgical operation, the fracture site was explored and debrided. Transection and thrombosis of the posterior tibial artery was identified and, after thorough balloon catheter thrombectomy, a primary end-to-end repair was successfully carried out and a long leg cast applied. An immediate postoperative unexplained drop in blood pressure required peritoneal lavage, confirming the presence of intraperitoneal hemorrhage. Laparotomy revealed a lacerated spleen necessitating splenectomy. Three days later the postoperative course was complicated by serious pulmonary insufficiency due to fat embolism. The changes in the lungs demonstrated the classic radiological picture of pulmonary fat embolization consisting of diffuse patchy infiltrates in all segments of the lung (Figure 2). After clearing of the initial symptoms of cerebral concussion no other central nervous system abnormalities such as seizures or change in level of consciousness occurred. A relative thrombocytopenia developed concomitantly with the pulmonary radiographic picture of fat embolism. The urine tested intermittently positive for fat. Treatment consisted of tracheostomy and controlled ventilation with high oxygen concentration. Even with this aggressive pulmonary support the arterial oxygen tension could only be maintained in the neighborhood of 50 mm of Hg. Although his prolonged

Legend for chest films at right:
Top: Initial Emergency Room film following insertion of right subclavian catheter demonstrating clear lung fields.
Center: Appearance of bilateral patchy infiltrates from pulmonary fat embolization four days post injury.
Bottom: Progressive consolidation and increased areas of infiltration seven days post injury.
hospital course lasted three months, the patient gradually improved and was discharged in satisfactory condition.

COMMENT: Fat embolization, confirmed by positive fat stains of the urine, was undoubtedly responsible for the severe pulmonary insufficiency that occurred three days after the severe multiple system injury. However, there were no cerebral symptoms or petechial hemorrhages. Controlled pulmonary ventilation enabled this patient's subsequent recovery.

Case 3. A 24-year-old black female sustained a high powered rifle wound to the right femur. She was transported to a nearby medical facility where she was found to be in shock with a systolic blood pressure of 70 mm of Hg. She received intravenous fluids and was transferred to Henry Ford Hospital. Upon arrival in our emergency department her systolic blood pressure was 120 mm of Hg with a regular pulse rate of 120. The pertinent findings were limited to the right lower extremity where there was obvious vascular compromise with absent distal pulses and an extensive compound, comminuted supracondylar fracture of the femur with a large, tense hematoma. Blood transfusions and antibiotic therapy were initiated. The injured extremity was splinted and the patient transferred to the operating room where exploration revealed that the proximal popliteal artery was transected and thrombosed. There was also an associated laceration of the popliteal vein. A thorough catheter thrombectomy and primary end-to-end repair of the artery was carried out. The vein laceration was repaired by lateral suture. This was accomplished after the fracture had been securely immobilized with the Roger-Anderson external skeletal apparatus. The patient was placed in the recovery room approximately seven and one half hours after her injury. She did well for the next four hours, recovered consciousness, and spoke coherently with the attending nurses. Suddenly, approximately twelve hours after the time of her injury (four hours postoperatively) while still in the recovery room, she was noted to be intermittently incoherent in her speech. Arterial blood gases obtained at that time revealed a P02 of 80 mm of Hg while receiving oxygen. During the next two hours the patient's condition progressively deteriorated. She became comatose and decerebrate. A chest x-ray film taken with
portable equipment was perfectly clear. Suddenly she developed severe tachycardia (pulse 164) with pulmonary edema. Endotracheal intubation was performed and her subsequent course was one of progressive pulmonary insufficiency with continued decerebrate rigidity.

Fat globules were identified in the urine approximately 13 hours after the injury and within an hour of the time the patient had her initial symptoms of incoherence. Petechiae were noted 14 hours after injury and within two hours from the onset of symptoms. The petechiae were noted primarily in the upper extremities, in the axillary folds and occasionally in the conjunctiva. There was laboratory documentation of progressive thrombocytopenia with the platelet count dropping to as low as 26,000 per cu/mm. The serum lipase two days after injury was 0.5 unit, three days after injury 1.0 unit, and on the fourth day it was recorded as 2.0 units. Approximately 24 hours after injury, blood hemoglobin had been restored by transfusions to 16 grams/100 ml. However, over the ensuing 72 hours it gradually ebbed to 11 grams/100 ml with no obvious source of bleeding identified. The chest x-ray, obtained after the patient demonstrated clinical pulmonary edema showed the typical picture of multiple infiltrates in both lung fields. No heparin, alcohol or dextran were administered throughout her hospital course. She did receive systemic steroid therapy in an attempt to control the cerebral edema. Stains of the cerebrospinal fluid and sputum were negative for fat.

All supportive attempts failed and the patient expired four days after onset of the ini-
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COMMENT: Although the extent of this patient's injury and the surgical management were not significantly different from the two previously cited cases, there was a fatal outcome. Successful repair of the vascular lesion was accomplished and the patient had apparently recovered from anesthesia without complication. There seems at this time to be no satisfactory method of preventing massive fat embolization with a resultant tragic outcome. Once symptomatic fat embolism occurs, little can be done aside from administering effective pulmonary support.

Discussion

Several clinical findings are ascribed to the fat embolism syndrome. However, classically it consists of mental aberrations, pulmonary insufficiency, petechiae involving the skin and mucous membranes and identifiable fat globules in the urine or blood. This clinical picture usually follows long bone trauma but also has been described in association with burns, sepsis, drug overdose, cardiopulmonary bypass, and cyclic acceleration. Only within the past 10 years has pulmonary insufficiency, associated with the fat embolism syndrome, been recognized and documented by means of arterial blood gas determinations. With more sophisticated diagnostic techniques and an increased clinical attention to the possible existence of fat embolism, this syndrome can now be recognized as a potentially serious complication which may lead to severe morbidity or mortality in patients with otherwise uncomplicated long bone fractures.

Pathophysiology: The pathogenesis of traumatic fat embolism remains obscure and, therefore, heatedly debated. All hypotheses appear to share a common reliance upon the mobilization of neutral fat. It is unclear whether this neutral fat appears in response to 1) direct venous intravasation, 2) lymphatic transport, or 3) in response to catecholamines as a part of the stress reaction. In addition, it should be noted that with the “embolization” of neutral fat there occurs a concomitant mobilization and sequestration of large numbers of platelets and megakaryocytes within the pulmonary circuit. A credible hypothesis is that in response to the stress of an injury associated with mobilization of a large number of neutral fat globules, there is coating of the globules requiring huge numbers of platelets. In addition, there is increased platelet adhesiveness with consequent aggregation and release of harmful platelet vasoactive amines. When one considers that the lung functions as an important pathway in the metabolism of vasoactive amines, prostaglandins, serotonin, bradykinins, and angiotensin then the total pathophysiologic effect of the release of such substances can be more fully appreciated. Compounding this fact is the realization that large pulmonary shunts may occur on a mechanical basis which prolongs the effect of vasoactive products and accentuates their biological mechanisms.

Experimental studies have shown an increase in the lung content of lipase, an enzyme which acts upon neutral fat releasing glycerol and fatty acids. The respiratory distress syndrome can be reproduced by the injection of small amounts of oleic acid. Fatty acid release results in direct injury of the alveolar capillary membrane. Consequent to this pulmonary injury there is marked decrease in the number of expanded alveolae and also a lack of pulmonary sur-
factant which results in further alveolar collapse and atelectasis. The effect of the release of fatty acids by the action of lipase upon neutral fat within the pulmonary circuit is recognized as an important factor in producing pulmonary insufficiency. Hypoperfusion states, such as those seen in traumatic or hemorrhagic shock, are also associated with a decrease in pulmonary surfactant. This synergistic effect would help to explain the frequent association of the fat embolism syndrome and clinical shock.

**Diagnosis:** The antemortum diagnosis of traumatic fat embolism is established by relying on the characteristic clinical signs and symptoms supported by laboratory evidence of fat embolization. Following an episode of major long bone trauma confusion or coma develops, associated with pulmonary insufficiency and petechiae of the skin and mucous membranes. Fat globules may even be observed in the eye grounds. This symptom complex dictates a presumptive diagnosis of traumatic fat embolism.

The most reliable yet simply performed laboratory test is the examination for fat globules in the urine.\(^{11}\) The decreased P02 reflects the alveolar disruption and edema at the level of the alveolar capillary membrane. Diffuse, fine, patchy infiltrates are typically seen in the chest x-ray. Serum lipase levels gradually rise during the hospital course, a late indicator that fat embolization has occurred. The slow drop in hemoglobin reflects the amount of pulmonary hemorrhage and can be used as an index of the patient's progress. The platelet count is usually depressed, at times severely. Fat may be demonstrated in the sputum or blood but these have been found to be less helpful forms of laboratory documentation.\(^{12,13}\)

**Treatment:** Once the diagnosis of fat embolism is established it is often too late to institute effective treatment other than intensive ventilatory support for the pulmonary insufficiency. Treatment therefore is basically preventive. The initial recognition of long bone fractures by paramedics or ambulance personnel is important so that early extremity immobilization and rapid transportation to appropriate medical facilities can be accomplished with the least amount of delay and additional trauma. The old boy scout adage of “splint them where they lie” remains appropriate. It is the first step in the prevention of fat embolism. Upon arrival at the emergency facility there should be a careful estimation of the patient's fluid losses with adequate early replacement. All efforts should be made to avoid shock in any form. Once the patient has been adequately assessed and resuscitated, attention should next be directed at early stabilization of the fracture site along with repair of any associated vascular injuries. These simple but often neglected principles of early management are the first lines of defense against the complication of fat embolism.

Adjunctive pharmacological treatment is the next consideration in the prevention of this syndrome. Corticosteroids may be employed in dosages similar to those found beneficial in gram negative sepsis and shock.\(^{5,13}\) The use of ethanol has also been suggested, not because it has an anti-emulsifying effect, but rather because it is a lipase inhibitor.\(^6\) Either a 5% solution of alcohol or 50 cc of 50% glucose has been advocated by some authors on a prophylactic basis in all patients who are considered to be at increased risk of developing fat embolism.\(^5\) In a similar fashion, but less frequently, low dose heparin therapy or dextran may be employed because of their antiplatelet effect.
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For established traumatic fat embolism, preventive measures and effective treatment of the pulmonary insufficiency by intensive ventilatory support are all that can be done. Basically, an attempt is made to maintain an arterial partial pressure of oxygen of between 70-80 mm Hg by whatever means is required. This may include the introduction of an endotracheal tube with the administration of continuous positive pressure ventilation. Tracheostomy is often required. Our approach has been aggressive in regards to the early enlistment of positive endexpiratory pressure. We attempt to maintain the oxygen concentration in the inspired air at less than 50-70%. We have also been pleased with the use of intermittent mandatory ventilation for weaning patients from the ventilator. During the severe state of pulmonary insufficiency it is paramount that adequate ventilatory support and oxygen exchange be maintained to allow as total recovery of pulmonary function as possible. In order to accomplish this a membrane oxygenator with extracorporeal perfusion may even be required.

Summary and Conclusions

At Henry Ford Hospital, during the twelve months from July 1, 1973 through June 30, 1974, three documented cases of traumatic fat embolism were associated with major arterial injury and long bone fracture of the lower limb. These three cases are presented to illustrate the wide range of clinical manifestations that can be associated with fat embolization. Our conclusions are in basic agreement with other reports: 1) fat embolism is a more frequent complication of major skeletal trauma than is generally recognized, 2) fat embolism may be accompanied by significant morbidity and mortality, 3) attention must be directed to preventive treatment in those patients who are highly susceptible to its occurrence, and 4) the associated pulmonary insufficiency requires aggressive management.

References


