Fat Embolism Syndrome in a Surgical Patient

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Fat embolism syndrome, a condition characterized by hypoxia, bilateral pulmonary infiltrates, and mental status change, is commonly thought of in association with long-bone trauma. Fat embolization can frequently take place, however, within the setting of elective and semiacute orthopedic procedures.[1] In particular, there is a high incidence of fat embolization during placement of hip prostheses. Although studies suggest that embolization events infrequently result in a clinically apparent fat embolism syndrome,[1,2] clinicians should be vigilant in considering fat embolism syndrome as a causative agent of postoperative respiratory distress.

Case Report

An 80-year-old woman with a history of hip fracture and prosthesis placement of the left hip came to the emergency department after a fall. A displaced femoral neck fracture of the right hip was diagnosed based on clinical examination and radiologic findings. The patient was admitted to the hospital by the orthopedics service.

The patient was scheduled for operative placement of a bipolar prosthesis of her right hip on the day following admission. Her preoperative course was uneventful. An electrocardiogram (ECG) showed Q waves in leads III, aVF, and V_3, which were interpreted as an old inferior infarction. She was afebrile, her blood pressure was 160/82 mm Hg, and her oxygen saturations were 93% on room air. In the operating room, she was sedated with midazolam and fentanyl and received spinal anesthesia.

Placement of a cemented hip stem (Johnson & Johnson Ultima) and femoral head component (Johnson & Johnson), bipolar shell, and bipolar liner was uneventful. The procedure lasted 96 minutes, during which the patient maintained oxygen saturations of 98% to 100% on 10 L of oxygen. Her blood pressure ranged between 90 and 135 mm Hg systolic, and her pulse was less than 100 beats per minute. Approximately 10 minutes postoperatively, the patient abruptly developed a sinus tachycardia with a pulse of 135 beats per minute. Her blood pressure was 122/88 mm Hg and her oxygen saturations were 85% to 86% on room air at a respiratory rate of 36/min. At this point, consultation by the family practice service was requested by the orthopedic surgeon.

An ECG, complete blood count, and cardiac profile were all obtained. The ECG showed sinus tachycardia without acute ST or T wave changes. Her hematocrit was stable, cardiac enzyme levels were negative, and a thyroid-stimulating hormone level was within normal limits. The patient's tachycardia and tachypnea continued, with her pulse ranging from 135 to 140 beats per minute and respirations between 24 and 36/min. Two hours postoperatively, she developed a fever of 101.7° F and systolic hypertension of 165 mm Hg.

A diagnosis of pulmonary embolus was considered, and arterial blood gas readings and a chest radiograph were obtained. Arterial blood gas on 4 L of inspired oxygen showed a pH of 7.41, carbon dioxide 36 mm Hg, and oxygen 81 mm Hg, with 97% saturation calculated. A chest radiograph showed bilateral perihilar fullness but a lack of infiltrate. Based on the patient's persistent oxygen requirement and her continued tachycardia and tachypnea, a D-dimer assay and ventilation-perfusion scan were obtained. Four hours postoperatively the patient had oxygen saturations of 78% on room air and 91% on 4 L of inspired oxygen.

The D-dimer assay results were between 1,500 and 2,000 μg/L, a positive result. The ventilation-perfusion scan was read as intermediate probability with matched segmental and subsegmental defects bilaterally, predominately at the lung bases and worse on the left. The patient was given heparin. Approximately 1 hour before her initial bolus of

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heparin was given, however, the patient's oxygen saturations suddenly began to improve, from 89% to 95% on 4 L of oxygen. Her pulse returned to 100 beats per minute, and her systolic blood pressure stabilized in the range of 125 to 135 mm Hg.

During the next 24 hours, she was weaned from oxygen, her low-grade temperature resolved, and her blood pressure remained stable. Several new petechiae were noted on the patient's anterior chest wall. A skin biopsy of the petechial lesions revealed intravascular fat, and a diagnosis of fat embolism syndrome was made. A review of her symptoms from the previous night indicated the patient met five of Gurd and Wilson's criteria for fat embolism syndrome, including petechiae, hypoxemia, pyrexia, tachycardia, and relative thrombocytopenia.[3] The heparin was discontinued.

The patient did suffer some bleeding from her wound site and required a transfusion of 2 U of packed red blood cells. She had no hematoma and began to make excellent progress with physical therapy. She was released from the hospital 8 days postoperatively.

At her 6-month follow-up, the patient was doing well. She had suffered no complications of the prosthesis itself and was progressing well in physical therapy, with good return of function to her affected hip. She had no long-term sequelae of her embolization event.

Discussion

The workup of a patient with acute onset of shortness of breath after an orthopedic operative procedure should include consideration of pulmonary thromboembolism and fat embolism as possible causes. Fat embolus is a common occurrence in many orthopedic procedures. Although it has been described extensively in the setting of long-bone fractures and multiple trauma, fat embolism syndrome has not been widely reported as a complication of total hip arthroplasty.[1,3–5]

In the patient described above, the diagnosis of fat embolism syndrome was entertained after the possibility of pulmonary thromboembolism was ruled out. The fat embolism syndrome was first described clinically by Von Bergmann,[6] who cared for a man with a broken femur and symptoms of the syndrome in 1873. The prevalence of fat embolism syndrome among all fracture patients is reported to be between 0.25% and 1.25%.[7] Among patients with multiple bone fractures, the prevalence can reach 5% to 10%.[8,9]

The pathophysiology of fat embolism syndrome has not yet been definitively characterized. A mechanical theory holds that the embolization event results from a transient rise in pressure in a fat-containing cavity in association with torn blood vessels, allowing escape of marrow or adipose fat cells into the circulation.[10] Two alternative biochemical theories posit explanations for fat embolism syndrome, both of which could account for the observation of the syndrome in nontraumatic settings. In one, fat droplets already in the circulation are broken down at distal sites to free fatty acids, which then exert a local toxic effect on the tissues. This theory explains the appearance of petechiae and the histologic changes in pneumocytes in association with fat-embolism–induced acute respiratory distress syndrome (ARDS).[11] The obstructive explanation for fat embolism syndrome proposes that free fatty acids are mobilized by circulating catecholamines. Fat droplets in the circulation eventually coalesce and embolize, causing destructive effects.[12]

Fat embolism syndrome can occur in immediate conjunction with a precipitating factor or it can be delayed for up to 3 days, although 85% of cases are apparent within 48 hours.[13] The diagnostic workup of a patient suspected of having fat embolism syndrome should include serial arterial blood gas measurements, as hypoxemia is one of the cardinal features. Serial chest radiographs can be used to observe the progression of ARDS infiltrates in the lungs, although it should be noted that chest radiographic changes are often not apparent in the initial stages of the syndrome. An ECG might show a new right bundle-branch block or nonspecific T-wave changes. A late laboratory marker of fat embolism syndrome is serum lipase, which becomes elevated 3 to 5 days after embolization and peaks at 5 to 8 days.

Gurd and Wilson[3] proposed the most widely accepted guidelines for the diagnosis of fat embolism syndrome, which require at least one sign from the major and at least four signs from the minor criteria (Table 1). An alternative set of standards was later proposed by Lindeque et al,[14] who believed that the criteria of Gurd and Wilson were too restrictive. The criteria of Lindeque et al are seldom used among clinicians, in part because of

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Table 1. Criteria for Fat Embolism Syndrome by Gurd and Wilson.

Major Criteria	Minor Criteria
Petechiae in a vest distribution	Tachycardia (heart rate > 110 beats per minute)
Hypoxemia with $PaO_2 < 60$ mm Hg, $FI0_2 \leq 0.4$	Pyrexia (temperature > 38.5°C)
Central nervous system depression disproportionate to hypoxemia	Emboli visible in retina
Pulmonary edema	Fat in urine
	Fat in sputum
	Unexplained drop in hematocrit or platelet count
	Increasing erythrocyte sedimentation rate

PaO_2 – arterial oxygen pressure, FIO_2 – forced inspiratory oxygen.

they are unable to distinguish fat embolism syndrome from other causes of respiratory distress.[15]

The histologic diagnosis of fat embolism syndrome relies on observing fat globules in vascular spaces. This finding is most reliably obtained by a biopsy of superficial cutaneous petechial lesions. Fat globules can also be found in sputum and urine, although this evidence is made more elusive by the fact that fat must be actively circulating at the time the sample is collected.

The treatment of fat embolism syndrome is primarily supportive. As with other causes of ARDS, maintaining adequate tissue oxygenation and an arterial oxygen saturation of more than 90% should be the clinician's goal. The patient's lung disease might necessitate the use of positive airway pressure or even mechanical ventilation. Because many patients suffer fat embolism syndrome in conjunction with multiple trauma, general supportive measures, including hemodynamic stabilization, maintenance of normal electrolyte values, and prompt attention to orthopedic and soft-tissue injury should be maintained.

The effects of steroids on patients with fat embolism syndrome have long been debated in the literature. The theoretical basis for using corticosteroids is sound; they are thought to stabilize granulocyte membranes, reduce catecholamine levels, retard platelet aggregation, inhibit the activation of complement system, and protect the capillary endothelium. Corticosteroids have been shown to reduce the incidence of fat embolism syndrome when given prophylactically in the emergency department,[16] although data showing a therapeutic role for them once clinically apparent fat embolism syndrome has developed have remained elusive.

Orthopedic surgeons might be able to reduce their patients' risk of fat embolism syndrome. Early fracture fixation has decreased the incidence of pulmonary complications[17] and fat embolism syndrome[18] related to long-bone trauma. Using a distal drain hole or a proximal and distal vacuum during the cementing stage of total hip arthroplasty has been associated with markedly reduced embolization. Recent studies using ultrasound have detected embolic events in routine total hip replacement operations in 94% and 100% of patients studied.[1,19] No patients in either group, however, showed clinically observable symptoms, underscoring the complexity of the factors that contribute to the genesis of the fat embolism syndrome.

It is thought that the technique used to cement the intramedullary component of the prosthesis causes embolic events during total hip arthroplasty.[5,20,21] In the traditional method, the femoral canal is first reamed out. Next, glue is inserted into the intramedullary canal, then the stem of the prosthesis. This technique generates tremendous pressures in the canal, which might cause the extravasation of marrow or cement into the vasculature. Use of a distal drain hole or vacuum greatly reduces the intermedullary pressures during total hip arthroplasty. Although such new approaches seem to reduce a patient's risk of fat embolism syndrome, surgeons caution that operative techniques which use a distal port might be associated with increased incidence of cement failure and femoral shaft fracture.

Conclusion

Long thought to be a problem unique to trauma patients, the fat embolism syndrome is common in other settings as well (Table 2). In particular, it should be considered in the differential diagnosis of shortness of breath that occurs after any orthopedic surgical procedure. It can be encountered in old patients as well as young, and by family physicians as well as surgeons and intensivists.

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Table 2. Settings for Fat Embolism Syndrome.

Blood transfusion
Burns
Cardiopulmonary bypass
Collagen disease
Decompression from altitude
Diabetes mellitus
Hemoglobinopathy
Infections
Medullary reaming
Multiple trauma
Neoplasm
Osteomyelitis
Renal transplantation
Suction lipectomy

New approaches have been used to decrease the rate of operative embolization, and to provide prophylaxis to patients who suffer multiple trauma, but only supportive therapies have been proven effective once ARDS associated with fat embolism syndrome becomes manifest.

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