CASE REPORT

Postoperative hypoxemia due to fat embolism

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ABSTRACT

Although the reported incidence of fat embolism syndrome (FES) is low (approximately 1%), it is likely that microscopic fat emboli are showered during manipulation of long bone fractures. Even though there continues to be debate regarding the etiology and proposed mechanism responsible for FES, significant systemic manifestations may occur. Treatment is generally symptomatic based on the clinical presentations. We report a 10-year-old girl who developed hypoxemia following treatment of a displaced Salter-Harris type II fracture of the distal tibia. The subsequent evaluation and hospital course pointed to fat embolism as the most likely etiology for the hypoxemia. We discuss the etiology for FES, review the proposed pathophysiological mechanisms responsible for its clinical manifestations, present currently accepted diagnostic criteria, and discuss its treatment.

Key words: *Hypoxemia, fat embolism, postoperative hypoxemia*

INTRODUCTION

Originally described in 1873 as fat embolism syndrome (FES), the embolic phenomenon resulting from long bone fractures continues to be a diagnostic challenge during the perioperative time frame. Although the incidence of the clinical manifestations (respiratory failure, petechial rash, pyrexia) is less than 1%,[1] the embolization of fat seems to be almost unavoidable during long bone manipulation. Despite that it may infrequently cause clinical signs and symptoms, the potential diagnosis of fat embolism must be considered in the perioperative etiology of hypoxemia. We present a patient who developed progressive hypoxemia which was thought to be the result of a fat embolism following reduction of a tibial fracture. The case report is followed by a discussion of the etiology, presentation, diagnosis, and treatment of fat embolism.

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CASE REPORT

Institutional Review Board approval for retrospective case reports is not required at Nationwide Children's Hospital. The patient was a 63 kg, 10-year-old girl who presented with a displaced Salter-Harris type II fracture of the distal tibia. The patient had suffered a twisting injury which resulted in greater than a 6-mm displacement. The patient had mild, well-controlled asthma with no history of hospitalizations. She had no other comorbid diseases and was on no medications. The patient was transported to the operating room where routine American Society of Anesthesiologists' monitors were placed. This was followed by intravenous induction, uncomplicated endotracheal intubation, and maintenance anesthesia with fentanyl and sevoflurane. Initially, a closed reduction was attempted; however, a residual fracture dislocation of greater than 3 mm was present and the decision was made to proceed with internal fixation. The patient's vital signs remained stable throughout the case with an oxygen saturation of 99–100% on an F_iO_2 of 0.4–0.5. A popliteal fossa block was placed for postoperative analgesia. At the completion of the case, her trachea was extubated and she was transported to the Postanesthesia Care Unit (PACU). During the first hour in the PACU, the patient's oxygen saturation decreased to 85–90% while breathing in room air and therefore she was provided supplemental oxygen at 2 liters/minute via nasal cannula. Deep breathing did not change her oxygen

saturation and she was changed to a simple facemask with an oxygen flow rate of 5 liters/minute which maintained her oxygen saturations of 91–93%. However, her oxygen saturation decreased to 80% with even brief periods off of supplemental oxygen. The patient's respiratory rate varied from 12–16 breathes/minute and there were no signs of respiratory distress such as nasal flaring or use of accessory muscles of respiration. Physical examination revealed fine rales over the left lower lung field. No change in the physical examination or oxygen saturation was noted after the administration of a high-flow nebulization treatment with albuterol. A chest radiograph obtained in the PACU [Figure 1] demonstrated a wedge-like opacity in the left lower lobe likely from an embolic phenomenon. The patient was admitted to the hospital for further monitoring. Her respiratory status improved overnight and she was weaned from supplemental oxygen. Her vital signs remained stable throughout her admission and a petechial rash was noted on the lateral aspect of her neck later that day. A follow-up chest radiograph was taken on postoperative day 1 [Figure 2], which showed improvement of the left-sided density. The patient was discharged later that day in stable condition.

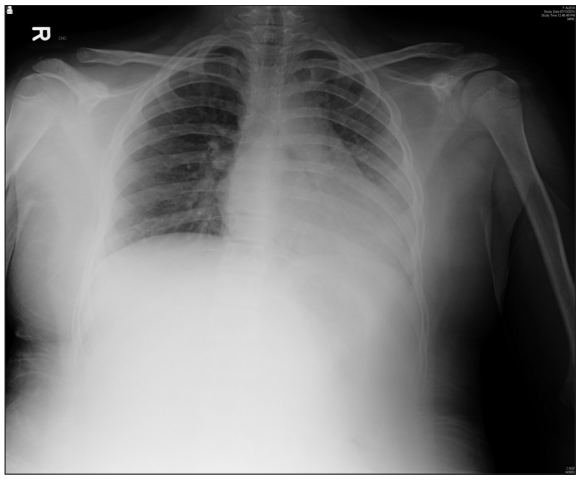

Figure 1: Chest radiograph taken in the post-anesthesia care unit showing left lobar consolidation

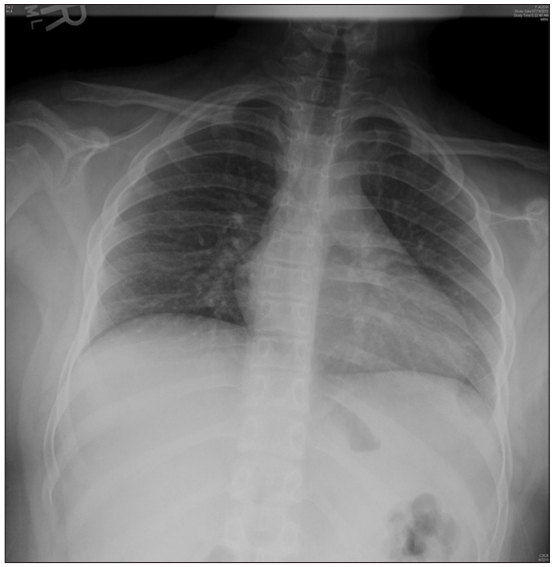

Figure 2: Chest radiograph taken on postoperative day 1 showing improved aeration in left lung base

DISCUSSION

Fat embolism syndrome results from the presence of fat in the circulation, most frequently the result of long bone fractures, combined with the clinical sequelae from the subsequent inflammatory process. The clinical signs and symptoms of FES can vary significantly from the asymptomatic patient to severe morbidity and even death. Most commonly there is a gradual onset of neurological symptoms, hypoxemia, and petechial rash; however Gurd and Wilson suggested the use of a more specific classification system for FES [Table 1].[2]

Respiratory signs and symptoms predominate. Dyspnea, tachypnea, and hypoxemia may occur anywhere from 12–24 hours after the initial insult and may mimic an acute lung injury type presentation.[3] The impact on ventilatory status may vary significantly with as many as 50% of patients diagnosed with FES requiring mechanical ventilation.[1,3] Central nervous system (CNS) involvement is usually reversible and may range from confusion and drowsiness to seizures and coma.[4] Although CNS involvement generally coexists with pulmonary manifestations, there have been reports of CNS involvement in the absence of pulmonary symptoms. The petechial rash may be the last of the triad to develop; however, it is generally considered pathomnemonic for FES. It may occur on the skin folds of the neck and axillae, mucus membranes, or conjunctivae.

The presence of signs related to the arterial side of the

Table 1: Gurdand Wilson criteria for fat embolism syndrome

Major criteria
Petechial rash
Respiratory insufficiency
Cerebral involvement
Minor criteria
Tachycardia
Fever
Retinal changes
Jaundice
Renal signs
Thrombocytopenia
Anemia
High erythrocyte sedimentation rate
Fat macroglobinemia

circulation such as petechiae and CNS findings is postulated to occur when fat globules traverse a patent foramen ovale or other right-to-left shunt and occlude the arterial side of the capillary bed.[5] In the skin, occlusion of the capillary arch by fat globules blocks blood flow to the dermal capillaries resulting in wall rupture and extravasation of blood. Alternatively, proponents of the free fatty acid and lipase mechanism (see below) suggest that systemic activation of various enzymes results in the coalescence of fat globules on the arterial side without.[6]

There are multiple theories proposed for the pathophysiological etiology of FES.[1] The *infloating theory/ mechanical theory* suggests that fat is forced out of the marrow and into the venous due to shear-mechanical pressure gradients during manipulation of the intramedullary cavity. Normal intramedullary pressures range from 40 to 60 mmHg; however, the pressure can exceed 600 mmHg during procedural reaming. The resultant showering emboli may coalesce forming thrombotic masses, pulmonary emboli, and/or right ventricular outflow tract obstruction.[7] The *free fatty acid and lipase theories* suggest that an elevation in the respective serum biochemical markers results in the formation of microscopic fat globules, severe vasculitis, and initiation of the inflammatory cascade. This may be the etiology of systemic manifestations in patients who do not have a right-to-left shunt. Although, the inflammatory pathway may be initiated locally, it quickly becomes systemic and may result in widespread end-organ involvement. The *shock and coagulation theory* proposes that the hypovolemic nature of trauma patients leads to a sluggish circulation and sludging of microaggregates in the lungs. Resultant vascular damage leads to platelet activation and their adherence to bone marrow fat particles. This process further activates the clotting cascade, which potentially leads to circulatory collapse via a thromboembolic phenomenon.

The diagnosis of fat embolism is usually clinical as described above. Findings on the chest radiograph may include diffuse patch infiltrates, alveolar hemorrhage, or subsegmental atelectasis. It is a common misconception that the presence of fat globules, either in sputum, urine, or a wedged PA catheter, is necessary to confirm the diagnosis of FES. However, there is a growing body of literature regarding the use of bronchoalveolar lavage to detect fat droplets in alveolar macrophages as a means to diagnose fat embolism. Treatment is mainly supportive with mortality rates ranging from 5% to 15%.[8] Early immobilization and operative treatment within 24 hours of the injury has been shown to reduce the incidence of fat embolism. Although there is no consensus regarding management, it is imperative to focus on early resuscitation and stabilization to minimize the inflammatory cascade as well as the treatment of hypotension caused by hypovolemia and hypoxemia. Since pulmonary manifestations are the most common, appropriate supplemental oxygenation and ventilatory support may be necessary. The use of corticosteroids remains controversial as studies lack the power to prove efficacy in a phenomenon with such a low incidence. However, corticosteroids have been studied extensively in animal models and have been shown to reduce the severity of FES when given prophylactically.[9] The proposed mechanism of the effect is a blunting of the inflammatory response and stabilization of the capillary membranes. More recently, surgical techniques have been modified to possibly decrease the severity of embolic material including limiting the elevation of intraosseous pressure by venting the intramedullary canal, using unreamed nails, or using sharp reamers as well as hollow nails.

Even though the embolism of fat particles frequently occurs with long bone fractures, clinical signs and symptoms may vary widely making the diagnosis difficult. Although hypoxemia generally develops later in the course of FES, as demonstrated by our patient, FES must be considered in the diagnosis of hypoxemia in the PACU. Our patient presented with two of the three "classic symptoms" as described by Gurd and Wilson including respiratory difficulty and a petechial rash. Although there is no consensus regarding treatment, early recognition and supportive management should be administered to limit the potential morbidity and mortality of FES.

REFERENCES

1. Mellor A, Soni N. Fat Embolism. Anaesthesia 2001;56:145-54.
2. Gurd AR, Wilson RI. The fat embolism syndrome. J Bone Joint Surg Br 1974;56B:408-16.
3. Gittman JE, Buchanan TA, Fisher BJ, Bergeson PS, Palmer PE. Fatal fat embolism after spinal fusion for scoliosis. JAMA 1983;249:779-81.
4. Bulger EM, Smith DG, Maier RV, Jurkovich GJ. Fat Embolism Syndrome. A 10-year review. Arch Surg 1997;132:435-9.
5. Fabian TC, Hoots AV, Stanford DS, Patterson CR, Mangiante EC. Fat embolism syndrome: Prospective evaluation in 92 fracture patients. Crit Care Med 1990;18:42-6.
6. Joffe D, Elrefai A, Thomas M. Fatal marrow emboli in a pediatric patient having posterior spinal instrumentation for scoliosis repair. Pediatr Anesth 2006;16:89-91.
7. Lafont ND, Kalonki MK, Barre J, Guillaume C, Boogaerts JG. *et al*. Clinical features and echocardiography of embolism during cemented hip arthroplasty. Can J Anaesth 1997;44:112-7.
8. Carr JB, Hansen ST. Fulminant fat embolism. Orthopedics 1990;13:258-61.
9. Pell AC, Hughes D, Keating J, Christie J, Busuttil A, Sutherland GR. Brief report: Fulminating fat embolism syndrome caused by paradoxical embolism through a patent foramen ovale. N Engl J Med 1993;329:926-7.

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