Case Report

A case of diffuse alveolar hemorrhage secondary to fat embolism after long bone fracture

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ABSTRACT

Fat embolism syndrome (FES) is a known complication of long bone fractures, frequently presenting with hypoxemia. Diffuse alveolar hemorrhage (DAH) is a rare complication of the condition. Imaging characteristics are frequently non-specific to FES, as are findings on bronchoalveolar lavage. No specific treatment exists, though steroids and albumin can be considered. We present the case of an 18 year old male who presented to the emergency room after a motorcycle collision. His CXR was initially clear, but he developed pulmonary infiltrates, a new oxygen requirement, and hemoptysis. Bronchoscopy confirmed DAH by serial lavage, and BAL was notable for abundant lipid-laden macrophages by Oil-red-O stain. He improved with methylprednisolone and albumin. This case highlights the need for a high index of suspicion both for FES, and for DAH as a complication that may develop days after trauma. We review treatment paradigm for this serious condition.

1. Introduction

Fat embolism syndrome (FES) is a known complication of long bone fractures. While hypoxemia is a common presenting symptom of FES, diffuse alveolar hemorrhage (DAH) is a rare complication of the condition with similar imaging characteristics on chest radiograph. This case describes a young, healthy male who developed hypoxic respiratory failure and hemoptysis from diffuse alveolar hemorrhage secondary to fat embolism after a femoral fracture.

2. Case presentation

A previously healthy 18 year old male presented to the Emergency Room after a motorcycle collision. Initial vital signs were notable for normal temperature, heart rate 98, blood pressure 124/69, oxygen saturation 100% on room air. Imaging showed a right femoral fracture (Fig. 1). Initial CXR was unremarkable (Fig. 2). One day after admission, he developed tachycardia and new hypoxia with a 6L O2 requirement. CXR that day showed new infiltrates (Fig. 3). CT angiography of the chest was negative for pulmonary embolism but notable for extensive ground glass opacities throughout the lung parenchyma with patchy consolidations and areas of crazy paving (Fig. 4). Two days after admission, he underwent right femoral intramedullary nailing. His oxygenation improved; however, later he developed scant bright red hemoptysis. Vital signs were notable for normal temperature, pulse 108, blood pressure 101/56, oxygen saturation 94% on 2L nasal cannula. On physical exam, he was found to be in no acute distress. Pulmonary exam was notable for rhonchi in the bases. No rashes or synovitis were identified. His right thigh was dressed but exam distal to the dressing was unremarkable. Laboratory tests revealed normal chemistries, a normal white blood cell count, hemoglobin of 11.2, and platelets 119. Subsequent CXR showed worsening of his infiltrates. Bronchoscopy with bronchoalveolar lavage was performed, with progressively bloody aliquots (Fig. 5). BAL cultures were negative for bacterial and fungal pathogens. Connective tissue serologies were negative. Cytology from the BAL was notable for abundant lipid-laden macrophages by Oil-red-O stain (see Fig. 6). He was started on...
methylprednisolone and albumin with significant improvement in his radiographic findings (Fig. 7).

3. Discussion

FES occurs as a complication of trauma and orthopedic injuries, but it can also be seen in atraumatic conditions such as bone marrow transplantation, liver injury, and administration of exogenous fat [1]. The clinical incidence is 1–3% with single long bone fractures [2,3]. Intramedullary nailing of long bones is among the most common surgical procedures that lead to FES [3]. The classic

![Fig. 1. Right femur fracture.](image1)

![Fig. 2. Initial admission chest radiograph showing normal lung parenchyma.](image2)
The triad of symptoms includes hypoxemia, petechial rash, and neurologic dysfunction. Average latency between insult and onset of symptoms was 4 hours–15 days in a review of 100 cases [4]. Up to 75% of patients with FES present with respiratory failure to varying degrees [2].

FES is primarily a diagnosis of exclusion. Multiple criteria exist to aid in diagnosis (Table 1) [4–6]. Typical imaging findings on CT chest include bilateral, patchy, well-demarcated ground glass opacities in a non-dependent distribution. Less common findings include crazy paving, lobular consolidation, and nodular septal thickening. These findings are similar to what is seen in diffuse alveolar...
hemothage [7]. The radiographic differential for FES-DAH includes lung contusion, pulmonary edema, aspiration, and DAH from other causes. Fat droplets within cells on bronchoalveolar lavage can be helpful, but is a non-specific finding [7].

DAH is a rare complication of FES. There are approximately 11 reported cases in the literature, which are largely associated with traumatic orthopedic injuries [2]. The exact mechanism by which FES causes DAH is unclear; one theory is that lipoprotein lipase acts on capillary fat, resulting in free fatty acids that induce chemokine-derived cell infiltration leading to damage of the alveolar-capillary membrane [8]. Treatment is largely supportive. Albumin administration has been shown to be helpful, presumably because of its ability to bind free fatty acids [1,2]. Steroid use has also been reported [5,9].

Despite its rarity, physicians should consider FES-DAH as a cause of hemoptysis after long bone fracture, especially as it may have implications for anticoagulation protocols in the post-operative period [10]. Fortunately, prognosis is generally good. Our patient met criteria Lindeque’s criteria for FES even prior to his intramedullary nailing; he may have met the other criteria as well if arterial blood gas data had been available. After initiation of steroids and albumin, he had rapid improvement in his chest radiograph and was discharged on room air. He had no pulmonary complaints at his orthopedic follow up appointment 6 weeks later.
Declaration of competing interest

The authors TNA and KB have no conflicts of interest to report.

References

[1] K. Taviloglu, H. Yanar, Fat embolism syndrome, Surg. Today 37 (1) (2007) 5–8.
[2] H. Chieng, B. Saha, L. Foulke, G.P. Wu, A.A. Chopra, 24-Year-Old man with dyspnea and a broken left femur, Chest 161 (4) (2022 Apr) e225–e231.
[3] M.J. Johnson, G.L. Lucas, Fat embolism syndrome, Orthopedics 19 (1) (1996 Jan) 41–48, discussion 48–49.
[4] A.R. Gurd, R.I. Wilson, The fat embolism syndrome, J. Bone Joint Surg. Br. 56b (3) (1974) 408–416.
[5] B.G. Lindeque, H.S. Schoeman, G.F. Domnisse, M.C. Boeyens, A.L. Vlok, Fat embolism and the fat embolism syndrome. A double-blind therapeutic study, J. Bone Joint Surg. Br. 69 (1) (1987 Jan) 128–131.
[6] S.A. Schonfeld, Y. Phongsang, R. Dilisio, J.D. Crissman, E. Miller, D.E. Hammerschmidt, H.S. Jacob, Fat embolism prophylaxis with corticosteroids. A prospective study in high-risk patients, Ann. Intern. Med. 99 (4) (1983 Oct) 438–443.

Table 1
Diagnostic criteria for fat embolism.

| Criteria for diagnosis                                      | Gurd’s criteria                  | Lindeque’s criteria | Schonfeld’s criteria |
|-------------------------------------------------------------|----------------------------------|--------------------|----------------------|
| Major                                                        | Axillary/subconjunctival petechial rash | Sustained PaO2 < 60 mmHg, FiO2 < 0.4 | Petechiae (5 points) |
| Hypoxemia (PaO2 < 60 mmHg, FiO2 < 0.4)                      | CNS depression                   | Sustained PCO2 < 55 mmHg or pH < 7.3 | Chest X-ray changes (4 points) |
| Hypoxemia (PaO2 < 60 mmHg, FiO2 < 0.4)                      | Pulmonary edema                  | Sustained respiratory rate > 35 breaths/min | Hypoxemia (PaO2 < 70 mmHg) (4 points) |
| CNS depression                                              | Minor                            | Increased work of breathing              | Fever (> 38°C) (1 point) |
| Tachycardia (> 110 beats/min)                               | Retinal emboli                   | Tachycardia (120 beats/min) (1 point)   | Tachypnea (> 30 breaths/min) (1 point) |
| Tachycardia (> 110 beats/min)                               | Urinary fat                      | > 1 criteria in patient with long bone fracture | |
| Tachycardia (> 110 beats/min)                               | Increased ESR                    | > 5 points                            | |
| Tachycardia (> 110 beats/min)                               | Fat globules in sputum           |                                  | |
| Increased hematocrit or platelets                           |                                  |                                  | |
| Increased ESR                                              |                                  |                                  | |

Fig. 7. Final chest radiograph after administration of steroids and albumin showing resolution of alveolar opacities.
[7] K. Newbigin, C.A. Souza, C. Torres, E. Marchiori, A. Gupta, J. Inacio, M. Armstrong, E. Peña, Fat embolism syndrome: state-of-the-art review focused on pulmonary imaging findings, Respir. Med. 113 (2016 Apr) 93–100.
[8] E. Seixas, P.G. Ferreira, Fat embolism syndrome presenting as diffuse alveolar hemorrhage: a rare (known) association, Pulmonology 24 (5) (2018 Sep-Oct) 314–315.
[9] A. Banerjee, R. Aggarwal, K. Dev Soni, A. Tirkha, Prone positioning in a patient with fat embolism syndrome presenting as diffuse alveolar haemorrhage: new perspective, BMJ Case Rep. 13 (3) (2020 Mar 10) e233452.
[10] P.F. Allan, D.K. Anjadi, R.L. Haynes, Diffuse alveolar hemorrhage: a rare manifestation of trauma, Mil. Med. 176 (9) (2011 Sep) 1071–1076.