

Fat embolism: A report of three cases

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Abstract. Fat embolism syndrome (FES) is a rare complication which usually follows long bone fracture. Clinical manifestations present immediately or 24 to 72 hours after injury. Symptoms are dyspnea, tachypnea, tachycardia, fever, mental status changes and petechial rashes. We reported three cases of FES. The first case had admitted to the pulmonology department with confusion and respiratory distress within 24 hours after tibia fracture. He had petechial rashes on the axillary area and subconjunctival hemorrhages. He was diagnosed as FES and treated. The second case who had right femur fracture had stupor after 48 hours. He had axillary petechial rashes and respiratory distress. He was diagnosed as FES and treated. The third case had admitted to the pulmonology department with the complaints of axillary petechial rashes, subconjunctival hemorrhages after a pelvic fracture. Both first and second patients who had respiratory failure intubated. The third patient died despite treatment. The signs of this syndrome must be carefully examined and considered in the diagnosis of the patients which attend the emergency service with confusion and petechial rashes after long-bone fractures.

Key words: Fat embolism, diagnosis, therapy

1. Introduction

Fat embolism syndrome (FES) is a rare complication that usually seen in long-bone fractures. The incidence and mortality rates are unknown because of comorbid injury and other problems (1). Clinical findings can be seen immediately after the fracture causing embolism or may also occur between 24 and 72 hours after the accident (2). It is a multisystemic disease which pulmonary, neurological, hematological, and dermatological system involvements are observed. Dyspnea, tachypnea, tachycardia, fever, axillary petechial rash, and mental changes are the common symptoms of the disease and early diagnosis and treatment is important (2).

We presented 3 cases of fat embolism as a rare complication of long-bone fractures and we believe that it will contribute for early diagnosis and treatment.

2. Case report

2.1. Case

The 29 years old male patient with tibia fracture had been admitted to the orthopedic clinic and was referred to our clinic because of respiratory distress and confusion. The patient was also referred to neurology clinic because he developed mental confusion in the last 24 hours and this is followed by two generally tonic-clonic seizures each lasted 5 minutes. Treatment was started with intravenous mannitol, steroids, and antiepileptic agents.

In physical examination there was no respond to verbal stimuli. He had tachypnea, and tachycardia. Arterial blood pressure was 100/80 mmHg, and fever was 37.2 °C. In the patient's follow-up, the fever reached to 38°C, subconjunctival hemorrhage, petechial rashes occur in axillary and neck region was observed (Figures 1a, b). Fundus examination was normal. Arterial blood gases (ABG) analysis was pH: 7.48, PO₂: 49.7 mmHg, pCO₂: 30.4 mmHg, SaO₂: 85.7%, HCO₃: 22.9 mmol/L, respectively. hemoglobin (Hb) level was 12.6 g/dL (N: 13.5-18) and platelets (PLT) 344 K/uL at admission and Hb level was 9.8 g/dL and PLT level was 144

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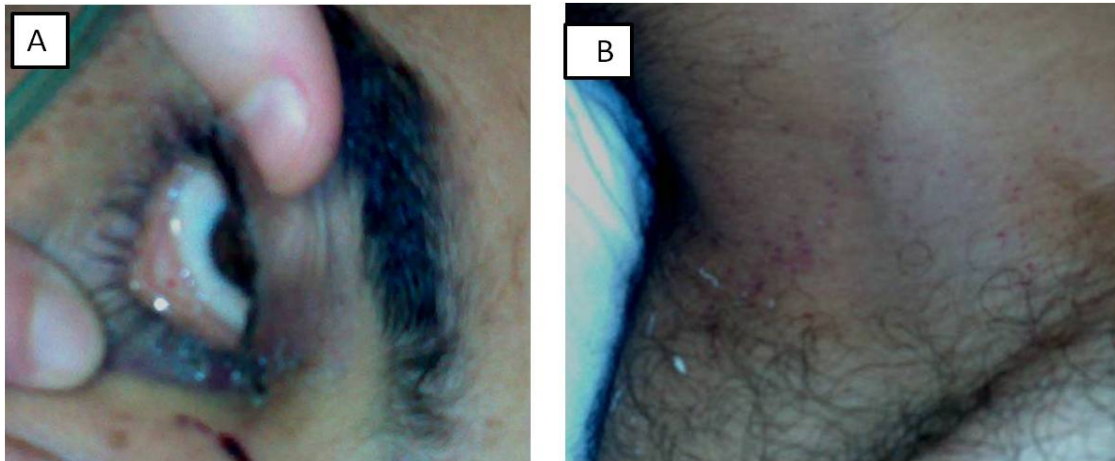


Fig. 1(a, b). Subconjunctival hemorrhage, and petechial rashes are seen in the neck (case 1).

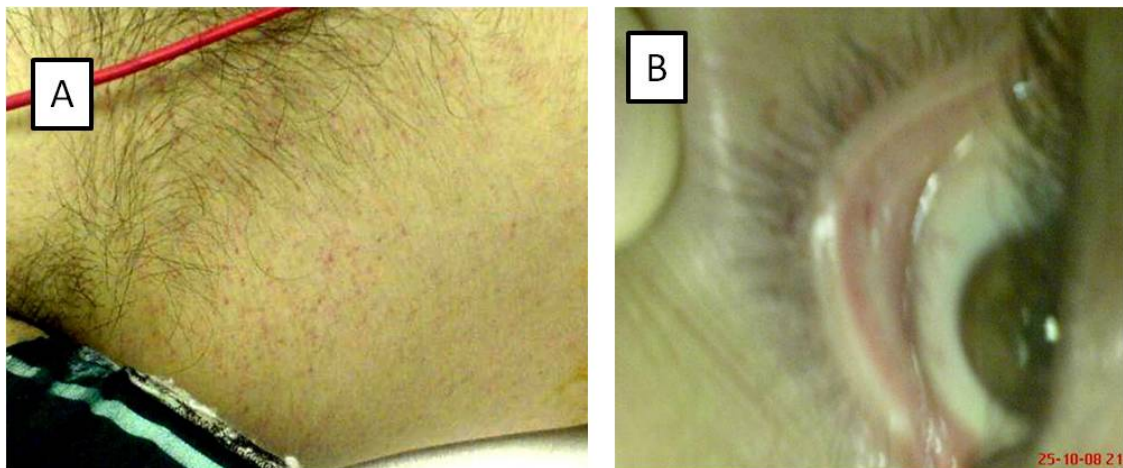


Fig. 2 (a, b). Axillary petechial rash, and subconjunctival hemorrhage are seen (case 2).

K/uL at diagnosis. Other biochemical parameters and urinary findings were normal. D-dimer was 2607 ng/mL (N: 000-500), and fibrinogen was 587 mg/dL (N: 150-400). Chest radiography was normal. In Thoracic CT, suspicious filling defects suggesting of pulmonary embolism were reported in the middle segmental and subsegmental branches of the left pulmonary artery. Bilateral lower extremity venous doppler ultrasound was normal. The current clinical findings suggested FES and the patient underwent supporting treatment, intravenous steroids, and subcutaneous low-molecular-weight heparin. He regained consciousness in the fifth day. The patient clinically recovered and was discharged after being operated.

2. 2. Case

23-year-old male patient was admitted to orthopedics service with right femoral shaft fracture and was consulted to our clinic because

of confusion and respiratory distress, 48 hours after the accident. Considering that confusion may be due to trauma, brain CT was taken within 12 hours interval and no pathology was detected. He did not respond to verbal stimuli in physical examination. Arterial blood pressure was 100/55 mmHg, and pulse rate was 106/min. Respiratory system examination was normal. There was petechial rash on right axillary (Figure 2a). The patient had subconjunctival hemorrhage (Figure 2b) Ocular fundus evaluated by ophthalmology service, and clinical examination was normal. Fever rose up to 39.5 °C during follow-up. ABG analysis results were as follows: pH: 7:51, pO₂: 61.4 mmHg, pCO₂: 26 mmHg, SO₂: 90.4% and HCO₃: 20.7 mmol/L, respectively. Hematology results were as follows: Leucocyte: 15.7 K/uL, neutrophils 84.6%, lymphocytes 8.7%, Hb: 13.5 g/dL, Htc: 38.2%, and PLT: 299 K/uL. He had higher levels of WBC but it returned to normal after one day. Urine output, urea and creatinine

levels were normal. There were no fat globules in urine and serum lipase level was normal. Thorax CT revealed pleuritis on both hemithorax and alveolar consolidation areas (areas of ground glass density) consistent with pleural contusion in bilateral lower lobes (Figures 3a, 3b). The patient

was diagnosed as FES due to existing clinical findings. Low molecular weight heparin and steroid treatment was started. The patient regained consciousness and oxygen saturation improved. The patient was discharged from the hospital after being operated.

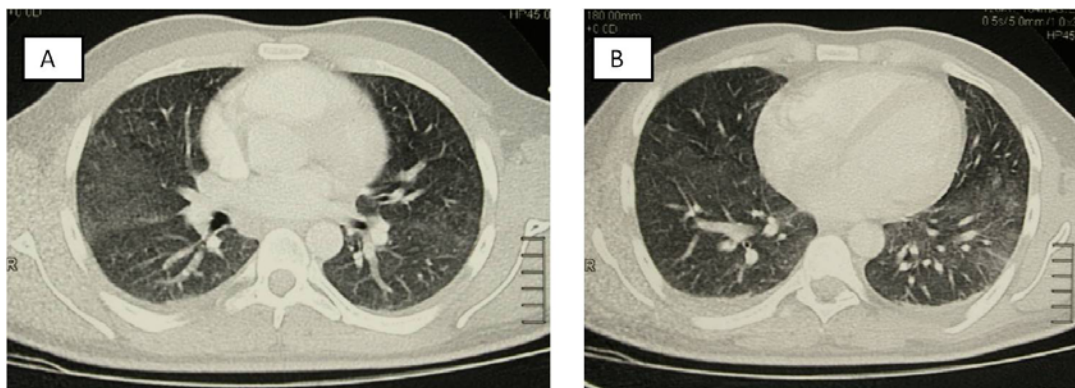


Fig. 3 (a, b). Ground-glass density areas, and bilateral pleural effusion are seen on Thorax CT (case 2).

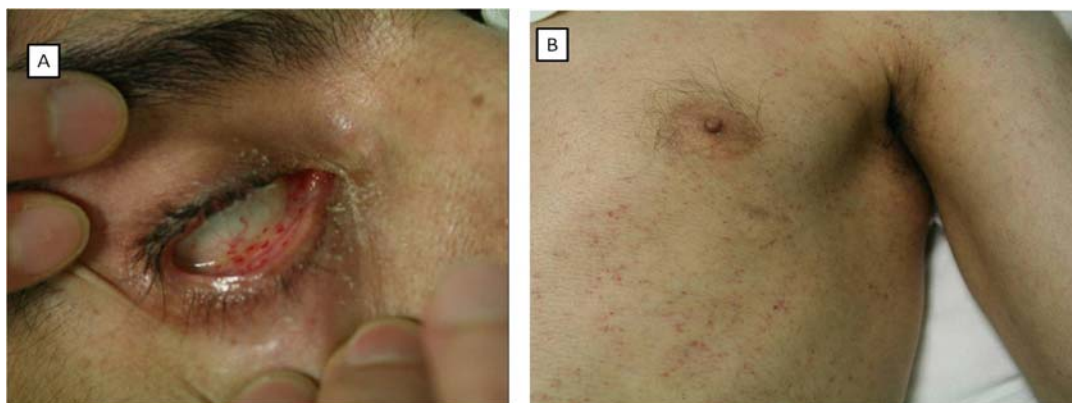


Fig. 4 (a, b). Case 3, subconjunctival hemorrhage, and axillary petechial rashes are seen (case 3).

2. 3. Case

The patient was 38-year-old male. The patient was brought to hospital because of a traffic accident and was diagnosed with pelvic fracture, subtrochanteric fracture in right femur and shaft fracture on left femur. The patient was referred to our clinic due to the development of drowsiness and respiratory failure within 48 hours. He was tachypneic and blackout of consciousness. Arterial blood pressure was 110/60 mmHg, heart rate was 120/min, and fever was 37.5 °C. Respiratory system examination was normal. Petechial rash and subconjunctival hemorrhage were detected on patient's axillary region of anterior chest wall (Figures 4a, 4b). Fundus examination was normal.

The results of ABG analysis without oxygen were as follows: pH: 7.45, PO₂: 48.2 mmHg,

pCO₂: 31.2 mmHg, SaO₂: 85.7%, HCO₃: 21.7 mmol/L. Other assays were detected as follows: AST 90 U/L (N: 5-37), ALT 59 U/L (N: 10-49), CK 3722 U/L (N: 21-215), CK-MB: 28.5 U/L (N: 0-15), LDH 400 U/L (N: 100-190), Hb: 8.2 g/dL (N: 12-16), Htc: 23.7% (N: 36-45), D-dimer: 4154 ng/mL (0-500), and fibrinogen: 920 mg/dL (N: 150-400). Kidney function tests, erythrocyte sedimentation rate, platelet count and other laboratory parameters were normal. Brain CT was taken with 12 hours interval and no pathology was detected. The patient was diagnosed with FES with the current clinical findings. The patient was transferred to intensive care unit because of general poor health and intubated because did not respond to continuous oxygen therapy and non-invasive mechanical ventilation. Low molecular weight heparin and steroid treatment was started. Chest X-ray on admission

was normal (Figure 5a). CT scan of thorax taken because of the formation of bilateral basal reticular pattern on the tenth day of the follow-up (Figure 5b) and revealed minimal pleural effusion in both lungs and consolidations compatible with

atelectasis in lower lobes. There was fat density in the atelectatic field on the right. The current symptom was reported to be due to fat embolism (Figures 6a, 6b). Despite the supporting treatment patient died on the 13th day of admission.

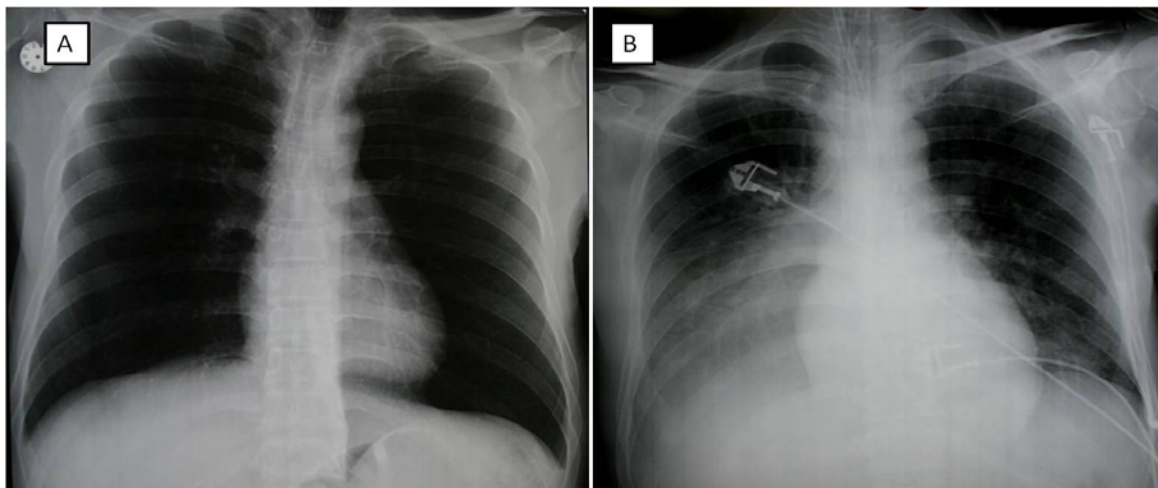


Fig. 5 (a,b). Chest X-ray on admission(Fig. 5a) and on the tenth day (Fig. 5b).

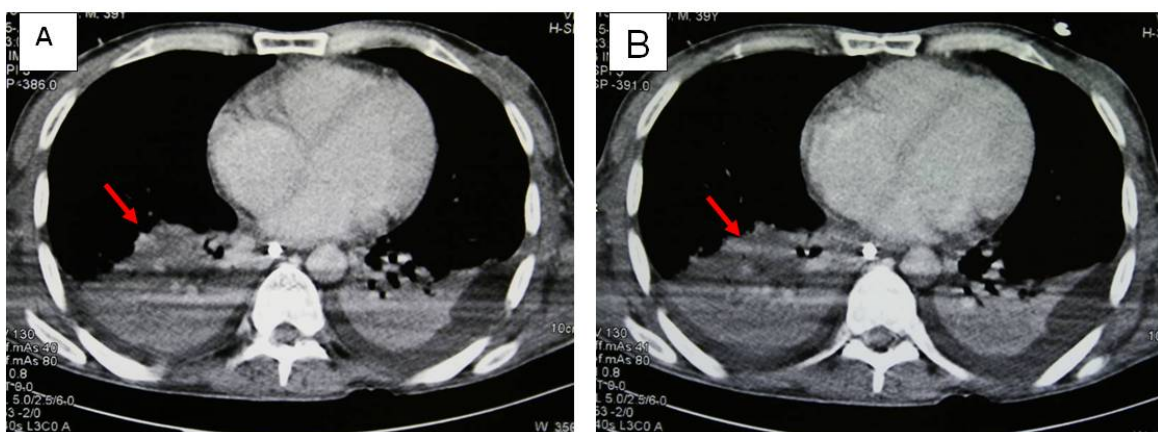


Fig. 6 (a, b). Torax BT, on cross-section of mediastinum, minimal bilateral pleural effusion, areas of atelectasis and fat density areas (arrow) in atelectasis are observed (case 3).

3. Discussion

Fat embolism syndrome was first identified by Von Bergman, in 1873, in a patient with fracture of the femur (3). FES usually occurs as a complication of the lower extremity long bone and pelvic fractures. The other etiological factors include total knee and hip replacement, renal transplantation, sickle cell anemia, osteomyelitis, burns, severe infections, blood transfusions, diabetes mellitus, alcohol-related hepatic failure, high-dose corticosteroid therapy, chronic pancreatitis, parenteral lipid infusion, and liposuction (2-4). Incidence and mortality rates are unknown because of the comorbid injury and

other problems (1). FES has a wide range of incidence between 0% - 35% reported in patients with bone fractures. Incidence depends on bone involvement [isolated or multiple], age and gender. Rarely occurs as a result of medical conditions. Classical triad includes symptoms in pulmonary (dyspnea), skin (petechiae) and central nervous system (mental confusion) (5). History and clinical symptoms are important for the diagnosis. The presence of major and minor clinical symptoms, characterized by Gurd and Wilson (Table 1), should be investigated (1-6). Diagnosis is made by the presence of at least two major, or one major and four minor criteria (1).

All of the major criteria and the minor criteria in part were defined in all three cases we presented.

Two theories have been suggested for pathogenesis of the disease. The mechanical theory explains particularly fat embolisms occurred after long bone fractures; fat droplets released from the bone marrow after fracture cause blockage of the pulmonary and systemic vessels. Biochemical theory explains rather pathogenesis of non-traumatic fat embolisms.

Hormonal changes after the trauma or sepsis induce systemic release of free fatty acids. Fatty acids are toxic on the capillary endothelium and pneumocytes. As a result, vasculitis in lung, brain and skin vessels, hemorrhage, edema and tissue damage occurs (1-3-7). The second effect may be more important because it may cause leakage from cerebral, pulmonary and other vascular veins and diffuse vasculitis (7).

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

Major Criteria	Minor Criteria
1. Hypoxemia with PaO ₂ < 60 mmHg, FIO ₂ ≤0.4	1. Pyrexia (temperature > 38.5°C)
2. Petechiae in a vest distribution	2. Tachycardia (heart rate > 110 beats per minute)
3. Pulmonary edema	3. Emboli visible in retina
4. Central nervous system depression disproportionate to hypoxemia	4. Fat in sputum
	5. Fat urine
	6. Unexplained drop in hematocrit or platelet count
	7. Increasing erythrocyte sedimentation rate (>71 mm/h)

There are no specific laboratory and imaging methods for fat embolism syndrome (6). Hypoxia and hypocapnia observed with the measurement of arterial blood gases. Laboratory findings include decrease in platelet and hematocrit levels, increased sedimentation rate, increase in the level of lipase, presence of fat globules in urine, sputum, and bronchoalveolar lavage (3-8). Some authors suggest bronchoalveolar lavage for rapid and specific diagnosis of FES, but being an invasive method restricts the availability of it (9). Radiological findings are nonspecific in FES. Radiographic examinations of the patients may be normal. Although most patients had normal radiographs initially after the trauma, symptoms may occur within approximately 72 hours. Resolution is expected usually in the second week of the hospitalization (10). Frequently observed CT findings include focal or diffuse areas of consolidation, and/or ground-glass opacities, nodules smaller than 10 mm and rarely filling defects of fat density which were determined with a Hounsfield unit in pulmonary arteries. Filling defects consistent with subsegmental pulmonary embolism were observed in the chest CT of the first patient. There were bilateral ground-glass density and bilateral minimal pleural effusion in the second patient.

In the third patient there were patchy infiltrates bilaterally in the lower zones on chest radiograph

and areas with fat density in the atelectasis areas in the lower lobes were observed on the thorax CT. Although, often one of the first signs of FES is respiratory failure, cerebral symptoms may be prominent. In the acute phase cranial diffusion MR has high sensitivity to detect cerebral fat embolisms and may be preferred for diagnosis of suspected patients (9).

Clinical approach to patients with FES includes, general patient assessment involving the traumatic situation, coordination of patient care, active nutritional support, symptomatic treatment, and adequate physical intervention (11). Accepted treatment dosage and duration of treatment with steroids is not known for FES (12). However, heroically known beneficial effects of steroids includes stabilizing pulmonary capillary membrane, suppression of inflammatory response, reduction of interstitial edema, preventing activation of the complement system and has such as preventing platelet activation (12). The use of albumin in patients with fat embolism syndrome, causes a decrease in free fatty acid concentrations (11,12). Different treatment methods with medications such as heparin, ethanol, dextran, nonsteroidal anti-inflammatory and heparin-glucose infusion had been tried, but no contribution to a decrease in morbidity and mortality had been reported. Therefore, none of these are considered in routine practice (11,12). On the other hand, Wang et al.

(13) reported that they achieved 92.3% success with administering hydrocortisone, dextran 40 glucose and Dan Shen Root injection as a treatment along supporting impaired respiratory functions and restoring hypoxemia in patients with fat embolism syndrome at 12 May 2008 Wenchuan earthquake.

A meta-analysis of studies showing the effects of prophylactic steroid therapy in patients with lower extremity fractures, revealed a reduction in the incidence of hypoxemia and FES. Suggested prophylactic treatment is to administer a total of 5 doses of 1 mg/kg intravenous methylprednisolone in every 8 hours after admission to hospital (14). Our first and second cases were supported by oxygen inhalation therapy; in the third case ventilator support was needed. All three of our cases were not given prophylactic steroid treatment. We preferred to start steroid and low molecular weight heparin therapy with strict monitoring of bleeding, after the differential diagnosis.

As a result, in patients with confusion and axillary petechial rash after bone fractures, fat embolism should be considered in the diagnosis and signs should be sought. Early diagnosis and prompt supporting treatment is important in terms of clinical course.

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