



A case of life-threatening intraoperative diffuse alveolar hemorrhage during a femur fracture operation with suspected fat embolism - A case report -

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Diffuse alveolar hemorrhage (DAH) is a rare manifestation of trauma or long bone fracture. A relationship between fat embolism and DAH has been reported. A 73-year-old female developed sudden cardiac arrest during a femur fracture operation. Cardiopulmonary resuscitation (CPR) was repeated for about 130 minutes. During CPR, blood was detected in the endotracheal tube. Diffuse patch ground glass opacity appearance and increased pulmonary artery with bulging of interventricular septum toward the left ventricle were detected on a chest computed tomography scan. After full supportive care including ventilator therapy, the patient's condition became stabilized and she was extubated after 7 days. We report a case of DAH in the course of a suspected fat embolism during femur fracture operation. Although DAH is a rare manifestation of fat embolism, early diagnosis and aggressive treatment likely can decrease morbidity and mortality.

Key Words: Diffuse alveolar hemorrhage, Embolism, Long bone fracture.

Diffuse alveolar hemorrhage (DAH) is a clinicopathological syndrome described by the accumulation of intra-alveolar red blood cells originating from alveolar capillaries [1,2]. DAH is a medical emergency that often results in acute respiratory failure [3,4]. Causes of DAH are diverse. It is known that trauma or a long bone fracture are associated with DAH [3,4]. To the best of our knowledge, this is the first case of DAH during long bone fracture operation reported in the literature. We report a case of life-threatening intraoperative DAH associated with a femur fracture operation.

CASE REPORT

A 73-year-old female (height, 163 cm and weight, 55 kg)

was admitted to our hospital for a right distal femur fracture. She had no previous medical illnesses or surgical history. After 5 days, the patient was transferred to the operating room for open reduction and internal fixation of a right distal femur fracture.

Preoperative laboratory tests revealed no abnormalities. Preoperative electrocardiography, chest radiography, and echocardiography revealed no notable abnormalities. She had skeletal traction on the right femur. Her vital signs before induction of anesthesia were as follows: blood pressure (BP), 170/100 mmHg; pulse rate, 100 beats per minutes (bpm) and oxygen saturation (SpO₂), 97%. General anesthesia was induced with 80 mg of 1% propofol after 40 mg lidocaine and 40 mg rocuronium were administered. The induction was

uneventful.

After induction, BP was 120–140/70–80 mmHg, pulse rate was 80–90 bpm, SpO₂ was 100%, and end-tidal carbon dioxide (ETCO₂) was 30–34 mmHg. Anesthesia was maintained with 1.5 L/min O₂, 2.5 L/min air, and 6 vol% desflurane.

The surgeon removed skeletal traction and skin incision was done at 15 minutes after the induction. ETCO₂ (20 mmHg) and SpO₂ (64%) decreased suddenly at 30 minutes after induction. BP and pulse rate also dropped to 80/42 mmHg and 50 bpm. Estimated blood loss until this time was less than 50 ml. Despite the attempt to normalize vital signs with 10 mg of ephedrine and 0.5 mg of atropine, cardiac arrest occurred. Upon the initiation of cardiac compression, epinephrine (1 mg) was injected. After 5 minutes of cardiac massage, blood pressure was 90–140/50–70 mmHg, pulse rate was 130–150 bpm, and SpO₂ was increased to 98%–100%. However, ETCO₂ remained at 20–22 mmHg. Arterial blood gas analysis showed pH of 6.91, PaCO₂ of 84 mmHg, PaO₂ of 261 mmHg, and bicarbonate of 16.8 mmol/L with oxygen fraction in inspired air (FiO₂) of 1.0 and hemoglobin (Hb) of 8.5 g/dl. The state of repeated cardiac arrest was sustained for 30 minutes. After that, BP was 50–140/40–70 mmHg, pulse rate was 100–150 bpm, SpO₂ was 100% (FiO₂, 1.0), and

ETCO₂ was 20–24 mmHg. Clinical signs (suddenly decreased in ETCO₂, SpO₂, BP and increased difference in arterial CO₂ and ETCO₂) were well correlated with an embolism. We suspected that fat embolism or thromboembolism was the cause of cardiac arrest. Because her vital signs were continually and seriously unstable, we administered 2,000 units of heparin (85 minutes after induction) if a thromboembolism caused the arrest. Under consultation with surgeon, skin incision was sutured and the operation was stopped. At that time, a frothy bloody secretion was seen in the endotracheal tube. The patient was sent to the intensive care unit (ICU) 105 minutes after the induction. Upon arrival at the ICU, cardiac arrest reoccurred and cardiopulmonary resuscitation (CPR) was performed. After 10 minutes, BP was 80–130/40–70 mmHg, pulse rate was 120–150 bpm, and SpO₂ was 100% (FiO₂, 1.0). The patient was then moved to the computed tomography (CT) room for chest CT where cardiac arrest reoccurred and CPR was restarted during chest CT scan. More blood was seen in the endotracheal tube than that seen in the operating room. The amount of blood was increased during suctioning. CPR was continued for about 90 minutes after the CT scan. Her pupils were dilated to 7 mm with a sluggish light reflex.

Diffuse patch ground glass opacity and an increased pul-

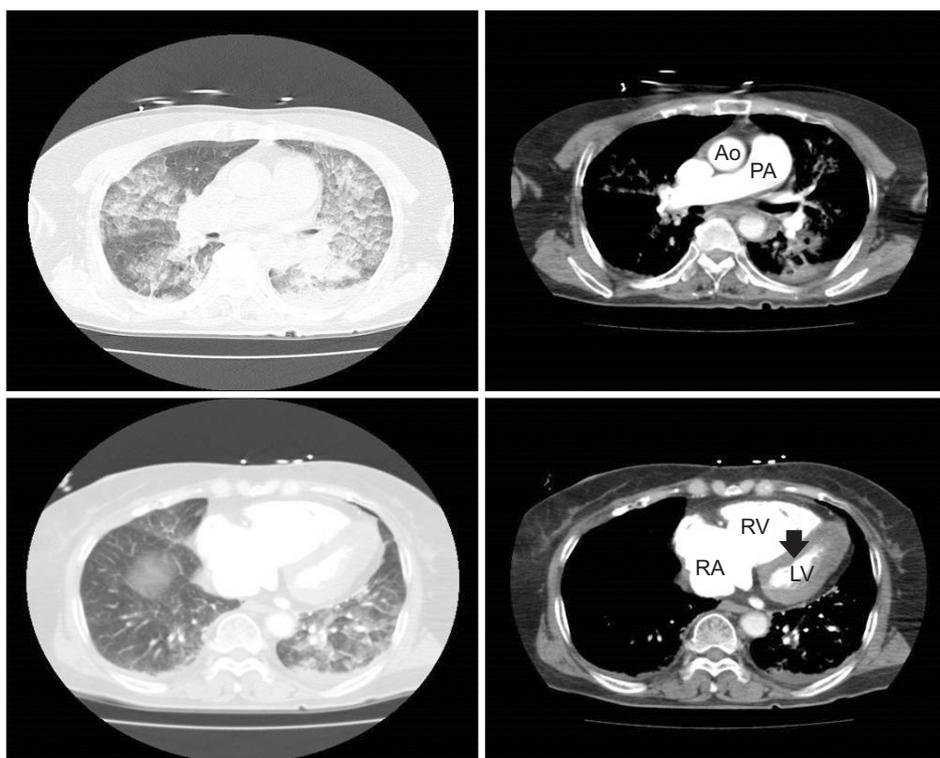


Fig. 1. Chest computed tomography scan shows diffuse patch ground glass opacity appearance and increased pulmonary artery size with bulging of the interventricular septum toward the left ventricle. Ao: aorta, PA: pulmonary artery, RA: right atrium, RV: right ventricle, LV: left ventricle, arrow: bulging of the interventricular septum toward the left ventricle.

monary artery diameter with bulging of the interventricular septum toward the left ventricle were detected on chest CT. However, no evidence of pulmonary artery thromboembolism was detected on chest CT (Fig. 1). Chest radiography showed diffuse hazy infiltration in both lungs (Fig. 2A). Her Hb level decreased from 13.6 g/dl preoperatively to 8.5 g/dl in the operating room. It continued to decline to 7.1 g/dl at ICU. Bloody secretions were still detected. She did not respond to verbal and painful stimuli at all. Because there was no evidence of pulmonary thromboembolism on chest CT, steroid therapy was started considering autoimmune disease as a possible cause of DAH. Blood sampling for rheumatoid arthritis (RA) factor, fluorescent antinuclear antibody (FANA), anti-glomerular basement membrane antibody (anti-GBM), anti-neutrophil cytoplasmic antibody (ANCA), and lupus anticoagulant was performed. Full supportive care (ventilator therapy, inotropics, and transfusion) was started. The next afternoon, she opened her eyes and hold her fist to verbal command. Bloody endotracheal secretions had also decreased. Continuous renal replacement therapy was started 1 day after the surgery. Echocardiography performed 1 day after the surgery showed a D-shaped left ventricle, dilated right ventricle, and moderate right ventricular dysfunction.

Her condition improved gradually with full supportive care. The bloody secretion in the endotracheal tube disappeared completely on postoperative day (POD) 5 and infiltration in both lungs decreased on plain chest X-ray (Fig. 2B). The patient was extubated on POD 7. RA factor, FANA, anti-GBM,

ANCA, and lupus anticoagulant were all negative. Blood culture results revealed no isolated pathogenic organisms. Transthoracic echocardiography on POD 20 did not show any myocardial dysfunction.

She was rescheduled to undergo open reduction and internal fixation of the right distal femur fracture on POD 21. Spinal anesthesia was induced with 10 mg of 0.5% heavy bupivacaine and the surgery was finished without any incident. The patient was transferred to the general ward on POD 29 and discharged on POD 58.

DISCUSSION

DAH is characterized clinically by hemoptysis, falling hematocrit, hypoxemic respiratory failure, and diffuse pulmonary infiltrates [1,3]. DAH is a medical emergency because significant morbidity and mortality are associated with delayed treatment [3,4]

DAH results in the accumulation of intra-alveolar red blood cells originating from alveolar capillaries. All causes of DAH are associated with injury to alveolar microcirculation [2]. Causes of DAH are diverse [2,5]. Specific etiologies of DAH include Wegener granulomatosis, systemic lupus erythematosus, Goodpasture syndrome, infection, bone marrow transplant, idiopathic pulmonary hemosiderosis, drugs/toxins, mitral stenosis, infection, embolism, and anticoagulant [1,2,5,6].

Clinical signs (suddenly decreased ETCO_2 , SpO_2 , BP and

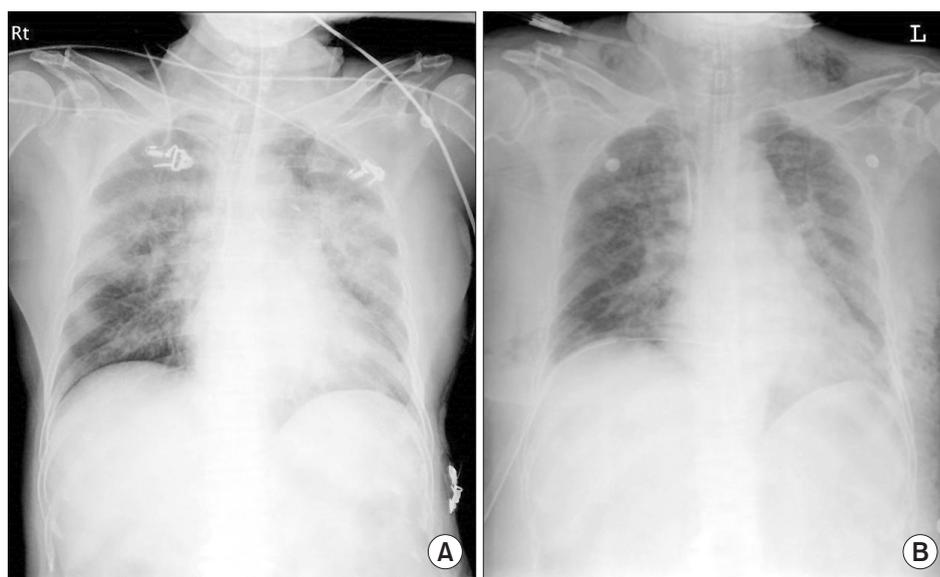


Fig. 2. (A) Chest radiography shows diffuse hazy infiltration in both lungs. (B) Chest radiography shows infiltration in both lungs had decreased on postoperative day 5.

increased difference in arterial CO₂ and ETCO₂) were well correlated with an embolism in this case. However, pulmonary thromboembolism was excluded from the cause of DAH in this case because there was no evidence of pulmonary thromboembolism on chest CT.

It has been reported that fat embolism and DAH can occur in association with an orthopedic injury and repair [4,7]. A fat embolism rarely presents as a macroembolism with a visible obstruction of the pulmonary artery on radiologic imaging [8]. Imaging findings of a fat embolism are similar to acute respiratory distress syndrome and consist of widespread homogeneous and heterogeneous areas of increased opacity [8]. We inferred that a fat embolus might have abruptly obstructed the pulmonary artery, leading to the development of DAH. Two mechanisms of lung injury by a fat embolism are currently accepted. One is mechanical obstruction of pulmonary vessels by fat. The other is intravasation of fat triggers an inflammatory response which causes further lung damage [9]. Pulmonary, cerebral, and cutaneous symptoms usually occur together and the prognosis is good if fat embolism syndrome is diagnosed early and treated adequately [9]. However, adult respiratory distress syndrome with a fat embolism due to a fracture of the femur and/or tibia is life-threatening. These patients showed alveolar edema and hemorrhage with fat droplet deposition and fibrin thrombi [10].

Flexible bronchoscopy should be done to confirm the clinical diagnosis of DAH. The diagnosis is established when sequential bronchoalveolar lavage (BAL) aliquots from the same location are more hemorrhagic progressively [1,2,5]. Allan et al. [4] reported that patients with fat emboli-associated DAH show increasing hemorrhaging in serial BAL specimens and hemosiderin-laden macrophages and lipid-laden macrophages in microscopy. However, we could not analyze BAL because of her serious condition.

Several cases of heparin associated with DAH have been reported [11–13]. They used heparin more than 4 days and administered a high dose of heparin (19,500–39,000 units). However, heparin was probably not the cause of DAH in our case for two reasons. First, we only administered a single small dose of heparin (2,000 units). Second, her Hb level decreased from 13.6 (preoperative) to 8.5 g/dl (operating room) before heparin was administered. Estimated blood loss until this time was less than 50 ml. Since the steep drop of hemoglobin level prior to heparin injection was unexplainable by

the injection itself, the heparin injection was not the cause of the DAH.

We considered an autoimmune-based cause and started steroid therapy. However, RA, FANA, anti-GBM, ANCA, and lupus anticoagulant were all negative. No growth was detected in the blood culture. She had no mitral stenosis either. We could not find a definite cause for the DAH such as immune disease, collagenous disease, renal disease, infection or heart disease.

In summary, here we report a case of DAH during a femur fracture operation with a suspected fat embolism. Although DAH is a rare manifestation of fat embolism, early diagnosis and aggressive treatment decrease morbidity and mortality. Therefore, clinicians should be aware of DAH associated with a fat embolism.

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