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Diagnostic value of bronchoalveolar lavage in fat embolism syndrome: Report of a case

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Introduction

Fat embolism syndrome is a rare disorder that is commonly associated with traumatic fractures of the long bones and/or pelvis. Gurd's criteria have been widely used in diagnosis, and several other criteria have been advocated [1–3]. All of these criteria are based on nonspecific clinical and biochemical features. Recently, fat staining of cells recovered by bronchoalveolar lavage has been introduced as a rapid and specific diagnostic method [4].

We present a case of fat embolism syndrome in which bronchoalveolar lavage and fiberoptic bronchoscopy were performed early in the patient's clinical course.

Case report

This 21-year-old man who had suffered from bilateral compound fractures of the tibia and fibula in a motorcycle accident was referred to our hospital with hypoxemia and disturbed consciousness. He had been admitted to our affiliated hospital, where surgical debridement and needle traction were performed to correct the bone derangements. About 25 h after the accident, he fell into semicoma. A cranial computed tomographic (CT) scan revealed no abnormality. He subsequently became cyanotic. Arterial blood gas analysis revealed marked hypoxemia (Pao_2 42 mmHg) while he was breathing room air. Administration of oxygen by face mask was ineffective. He was transferred to our intensive care unit 30 h after the accident.

Physical examination on admission showed normal blood pressure (110/60 mmHg), tachycardia (122 bpm) and mild pyrexia (37.6°C). The patient's head and chest wall showed no signs of injury, but he was confused and dyspneic. A few subconjunctival petechiae were present. Rough bronchial breath sounds were heard in both lungs, and a chest roentgenogram revealed bilateral vascular congestion and consolidated radiopacities that suggested pulmonary edema. Laboratory tests showed leukocytosis (19 830/mm^3), hypoproteinemia (5.0 mg/dl), elevated levels of triglyceride (569 mg/dl) and free fatty acids (1.94 mEq/l).

The patient was immediately intubated and ventilated with the positive end-expiratory pressure mode at 5–10 cmH_2O.

Fiberoptic bronchoscopy and bronchoalveolar lavage were performed on the following day. Bronchoscopy revealed numerous petechiae on the lower part of the trachea and on both bronchial trees (Fig. 1). A regional mucosal hemorrhage was observed in the left lower bronchus. Sudan III staining of sediment cells from bronchoalveolar lavage revealed round intracellular fat globules in as many as 30% of the alveolar macrophages (Fig. 2). Pulmonary capillary blood drawn from a Swan-Ganz catheter revealed fat droplets in the plasma. From these results, we diagnosed his illness as fat embolim syndrome.

Methylprednisolone 1500 mg was administered intravenously daily for 3 days to ameliorate the progression of brain edema and lung inflammation that was causing permeability edema. The protease inhibitor, nafamostat mesilate, and albumin were also administered to prevent coagulopathy and hypovolemia, which were likely to promote the development of the fat embolism syndrome.

The patient's clinical response was excellent, and he was removed from the ventilator on the 8th hospital

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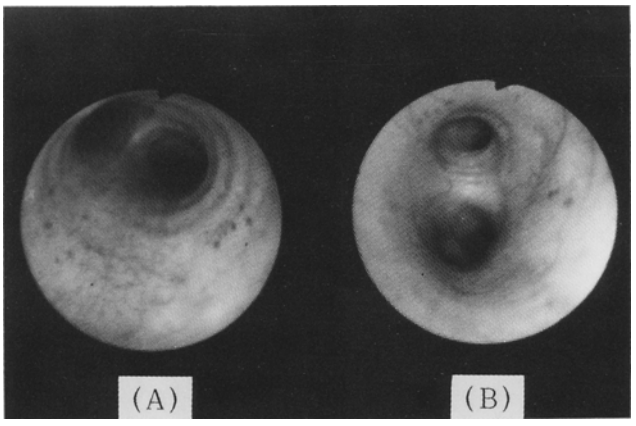

Fig. 1A,B. Findings on fiberoptic bronchoscopy examination of **A** the lower part of the trachea and **B** the left main bronchus. The airway lumen is dotted with numerous petechiae, which resemble those on the conjunctivae

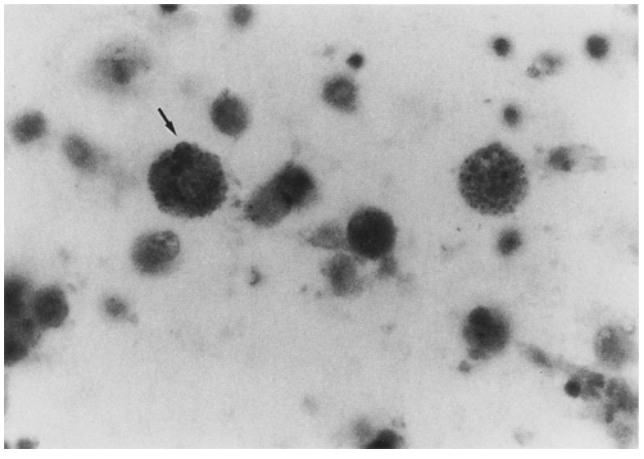

Fig. 2. Light photomicrograph of cells recovered by bronchoalveolar lavage. An alveolar macrophage contains a brown intracellular inclusion (*arrow*) stained with Sudan III (original magnification ×400)

day. He became alert 2 days later. Fiberoptic bronchoscopy performed before extubation showed a decrease in the number of petechiae. The subconjunctival petechiae had also improved. Chest roentogenogram and CT scan on the 16th hospital day were virtually normal. Subsequently, the tibia and fibula were surgically repaired and the patient was discharged after receiving rehabilitation therapy.

Discussion

Bronchoalveolar lavage is routinely used in cytopathological and immunobiochemical investigations in patients with lung disease, and its safety and usefulness are well established. Chastre et al. first applied this technique in studying the fat embolism syndrome and concluded that bronchoalveolar lavage may be a rapid and specific method for confirming the diagnosis of fat embolism syndrome at an early clinical stage [4].

Fat droplets in alveolar macrophages have also been observed in cases of lipid pneumonia that are associated with aspiration pneumonia [5] or with bronchial narrowing [6]. One must therefore exclude these conditions when making a diagnosis of fat embolism syndrome. We were able to exclude them in our patient by bronchoscopy and clinical history.

Fat particles were also demonstrated in pulmonary capillary blood in our patient. This finding suggests a pathogenesis of fat embolism syndrome: intravascular fat breaks through the capillary endothelial barrier and invades the lung interstitium and alveolar space, resulting in lung edema and hemorrhage.

In patients with fat embolism syndrome, petechiae had been reported at points where arteries are supplied by the aortic arch [7]. Because the lower part of the trachea and the larger bronchi are mainly supplied by the bronchial artery, hemorrhagic reactions from fat embolism syndrome are likely to occur in the airway mucosa as frequently as in the conjunctivae and the upper trunk. Respiratory infection, forceful coughing, and bronchial suction each can induce mucosal hemorrhage such as that found in the present case. When these conditions have been excluded and other positive features of the syndrome are simultaneously present, conspicuous and widespread petechiae in the airway mucosa may be strongly suggestive of the occurrence of fat embolism syndrome. Further clinical observation and pathological study are required to confirm this hypothesis.

In summary, we report a patient with fat embolism syndrome in whom the presence of fat globules in alveolar macrophages were demonstrated by bronchoalveolar lavage early in the clinical course. Petechial hemorrhages in the airway mucosa simultaneously observed with other major features also seemed to support the occurrence of fat embolism. Our findings suggest that these measures may be specific and confirmative for diagnosing fat embolism syndrome. We believe that bronchoalveolar lavage and fiberoptic bronchoscopy are useful in making an early diagnosis, and may help to reduce the morbidity and mortality associated with this syndrome.

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