We report thin-section CT findings of pulmonary fat embolism confirmed by clinical features and microscopic examination of cells obtained by bronchoalveolar lavage. Initial thin-section CT showed extensive air space consolidation and multiple ill-defined nodular densities in both lungs. Follow-up CT revealed ground-glass appearance and faint nodules in both lungs. A perfusion scan showed multiple small perfusion defects in the peripheral portion of both lungs.

**Index Words:** Embolism, fat
Lung, CT
Lung, radionuclide studies

Fat embolism syndrome is characterized by pulmonary edema, and cerebral and cutaneous manifestations that most often occur after long bone fracture or soft-tissue injury. The pathogenesis of the fat embolism syndrome in the lung remains unclear and controversial. Embolization and deposition of fat and fatty acids from bone marrow to pulmonary capillaries appear to be the main event in the supervision of the illness (1-3).

We report thin-section CT findings of pulmonary fat embolism confirmed by clinical features and microscopic examination of cells obtained by bronchoalveolar lavage.

**Case Report**

A 25-year-old man with a fracture of the left proximal femur and of the right distal tibiofibular was transferred to our facility on the second day of trauma.

Initial vital sign were stable and physical examination revealed moderate edema, tenderness and crepitation in the left thigh and right leg with no external wound, but other findings, including initial radiograph and electrocardiogram, were normal. Laboratory study at admission revealed a hemoglobin level of 13.0g/dL, a white blood cell count of 9,400/mm² with normal differential count and serum calcium of 8.4mg/dl. The results of clotting studies, urinalysis, and blood chemistry were all within normal limits.

The patient’s general condition began to deteriorate on the third day of hospitalization, and on the fourth day, he had dyspnea, chest pain and high fever (38.8°C). He was not agitated or confused, however, and petechial rash was not seen. Arterial blood gas analysis on room air revealed arterial oxygen pressure of 32.9 mmHg, arterial carbon dioxide tension of 35.9 mmHg, pH of 7.438, bicarbonate level of 23.8 mEq/L, and oxygen saturation of 65.8 percent. A chest radiograph revealed extensive air-space consolidation in both lungs. With diuretic and oxygen therapy, his condition stabilized.

On the sixth day of hospitalization, he had mild dyspnea and chest pain. Arterial blood gas levels were much improved. Thin-section CT showed extensive air-space consolidation and multiple ill-defined nodular densities in both lungs (Fig. 1A). A perfusion scan revealed scattered perfusion defects in both lungs (Fig. 2).

On the seventh day of hospitalization, his chest radiograph became normal; thin-section CT revealed ground-glass attenuation and small faint nodules in both lungs (Fig. 1B).

On the basis of clinical features, including dyspnea, fever, chest pain and hypoxia, and microscopic fin-
Findings of cells obtained by bronchoalveolar lavage (BAL) (Fig. 3), fat embolism was diagnosed.

Discussion

Fat embolism syndrome was first described at autopsy in 1862 by Zenker, and first clinically diagnosed 12 year later by Von Bergman (1 - 3). Although fat embolization following major trauma associated with long bone fracture is common, the incidence of the clinical syndrome of fat embolism is low (1). The syndrome occurs in many other conditions including burns, chronic pancreatitis, blood transfusion, and cardiopulmonary bypass (3).

Clinical fat embolism syndrome presents with tachycardia, tachypnea, elevated temperature, hypoxemia, hypercapnia, thrombocytopenia, and occasionally mild neurological symptoms (1).

The pathophysiology of fat embolism syndrome falls into two main categories. The mechanical theory is that syndrome results from physical obstruction by embolized fat of the pulmonary and systemic vasculature; the biochemical theory is that circulating free fatty acids are directly toxic to pneumocytes and capillary endothelium in the lung, causing interstitial hemorrhage, edema and chemical pneumonitis (1 - 2).

In mild cases, radiographs may remain entirely normal. In severe cases, they are initially normal; changes appear after an interval of 72 hours or more and consists of bilateral diffuse lung densities. These may appear as veil-like cloudiness, consistent with pathologic findings of interstitial edema, or may resemble the typical pattern of alveolar pulmonary edema. In some patients, the densities are localized, with indistinct margins, and appear patchy; they are due to focal hemorrhages, localized atelectasis, or infarction. Pleural effusion is not a feature of fat embolism. In most cases the pulmonary densities clear after two days to two weeks with an average of about a week. (2)

A CT finding of pulmonary fat embolism was not established. Our case showed extensive bilateral airspace consolidation and multiple ill-defined nodules, findings compatible with pulmonary edema and acute pulmonary hemorrhage, which were previously reported pathologic findings of fat embolism (1 - 2). Follow-up CT one week later showed ground-glass attenuation and small faint nodules in both lungs. These findings are compatible with subacute pulmonary edema.

**Fig. 2.** Perfusion scan of the lung shows multiple small perfusion defects in the peripheral portion of both lungs.

**Fig. 1.** Thin-section CT findings of 25-year-old man with fat embolism.
**A.** Initial thin-section CT scan obtained at the level of lower trachea shows air-space consolidation and multiple ill-defined nodules in the extensive areas of both lungs.
**B.** Follow-up CT scan obtained 1 week later (A) at the same level shows ground-glass appearance and faint small nodules in both lungs.
hemorrhage with improvement of pulmonary edema (4-5).

Perfusion lung scanning can be used to effectively detect fat embolism following skeletal trauma; scan findings are numerous perfusion defects scattered throughout both lung fields. This mottled appearance has been reported to occur in 70% of cases of lower extremity fracture with no ventilation abnormality (6).

Microscopic examination of cells obtained by BAL or tracheobronchial aspirate allowed rapid identification of patients with fat embolism syndrome. Staining of BAL cells for lipids is not a specific test for pulmonary fat embolism, but many conditions involving pulmonary fat embolism are associated with fat droplets in alveolar macrophages. Sudan or oil-red O staining may detect red or brown-red fat globules that are either free or in macrophages, and brown staining cytoplasmic inclusions in alveolar macrophages obtained by BAL (7).

We report thin-section CT finding of pulmonary fat embolism; these were compatible with pulmonary edema and hemorrhage.

References
## 1997년도 제53차 학술대회 사전등록 신청서

### 연락처

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### 학술대회 등록

제53차 학술대회에 사전등록을 하시겠습니까?

예 □ 아니오 □

예 □에 체크하신분은 아래 해당금액을 온라인 구좌로 입금하십시오.

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