J Korean Radiol Soc 1997;36:999-1001

Pulmonary Manifestations of Fat Embolism on Thin-section CT : A Case Report[1]

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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 supervention 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 radiogaph and electrocardiogram, were normal. Laboratory study at admission revealed a hemoglobin level of 13.0g/dL, a white blood cell count of 9,400/mm^2 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 dypnea 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-

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dings 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 air-space 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

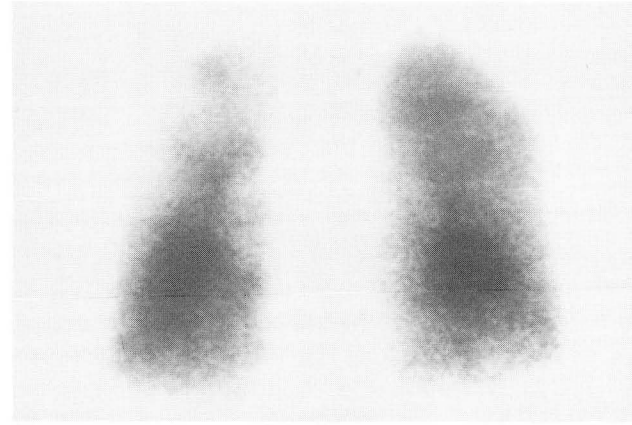
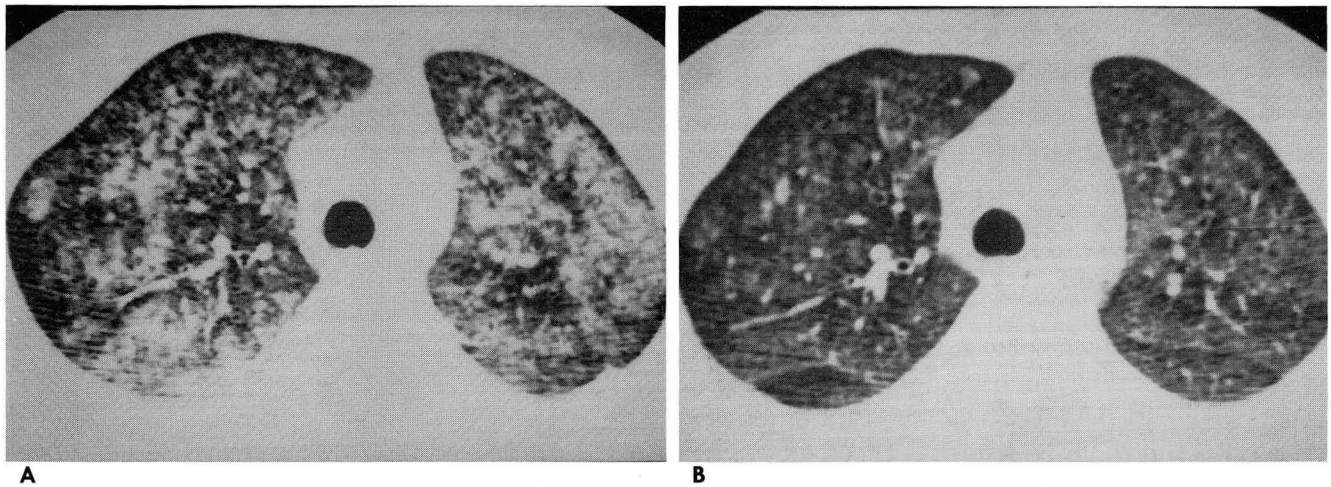
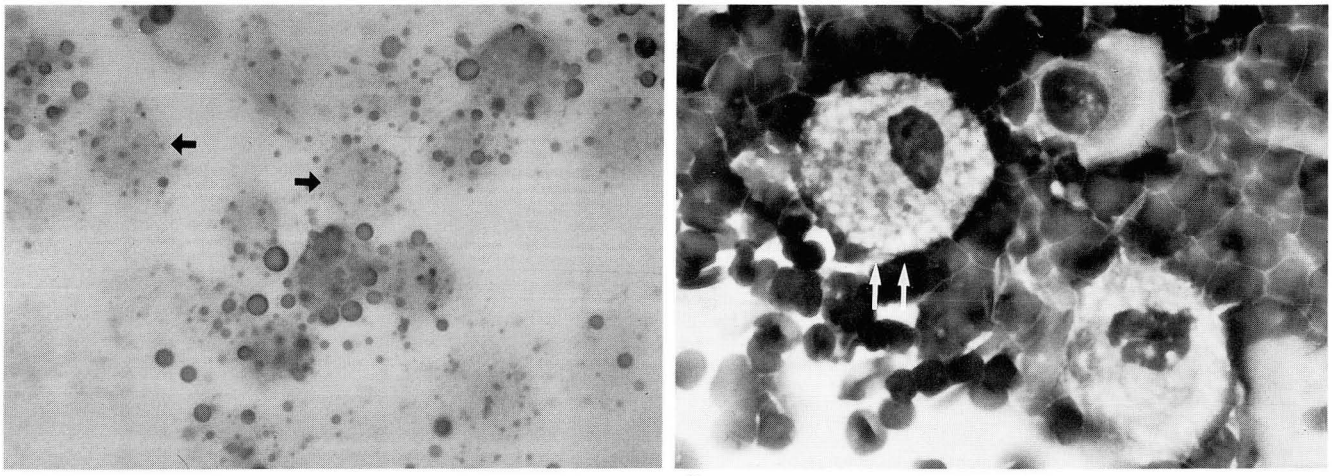


Fig. 2. Perfusion scan of the lung shows multiple small perfusion defects in the peripheral portion of both lungs.



A **B**

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.



A B

Fig. 3. Microscopic examination of cells obtained by bronchoalveolar lavage.
A. Brown-red fat globules are either free or in alveolar macrophages (black arrow) (oil-red O stain, magnification × 200).
B. Brownish-staining cytoplasmic inclusions are seen in alveolar macrophage (white arrows) (H-E stain, magnification × 1000).

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 condition 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 brownish 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.

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대한방사선의학회지 1997;36:999-1001

지방색전증후군의 폐병변의 CT소견 : 1예 보고[1]

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김 경 석 · 김 영 통 · 김 일 영

25세 남자 환자에서 임상 양상과 기관지폐포 세척으로 얻어진 세포의 광확대 소견을 근거로 진단된 폐 지방색전증의 CT소견의 1예를 보고한다. CT소견은 양측 폐에 광범위한 폐포성 경결과 다수의 불분명한 결절이 보였고, 추적 CT소견은 양측 폐에 젖빛유리음영과 희미한 소결절들이 보였다. 관류 주사에서는 양측 폐의 변연부에 작은 관류 결손이 보였다.

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