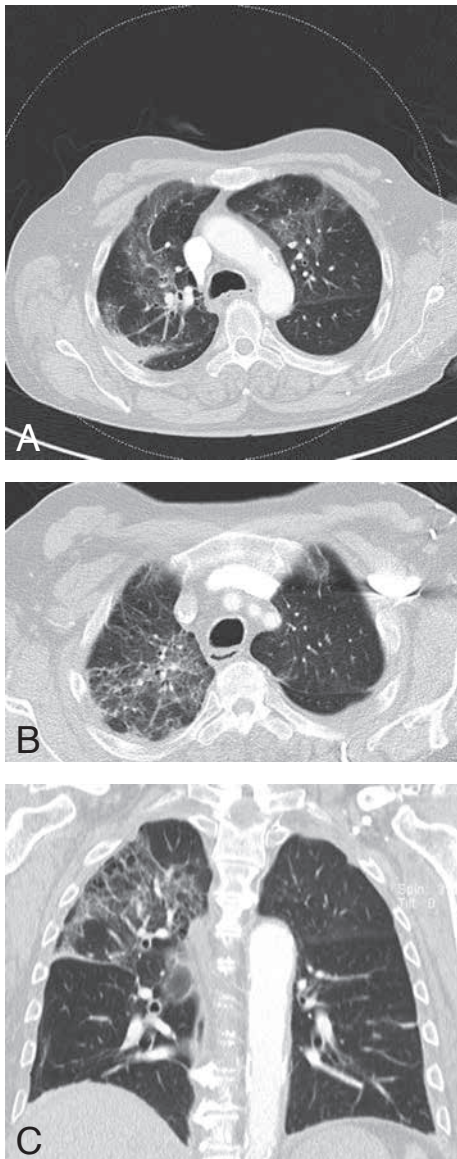


IMAGES IN CLINICAL RADIOLOGY



Pulmonary fat embolism

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A 64-year-old man with a subcapital femoral fracture after falling with a scooter underwent a total hip prosthesis. Two days after surgery he developed sudden dyspnea and tachycardia, improved with the administration of supplemental oxygen at 2 liters per minute. Arterial blood gas analysis revealed a pO₂ of 75 mmHg.

Conventional chest x-ray showed patchy alveolar opacities in the right upper lobe, which were new compared to the x-ray pre-op. Suspecting pulmonary emboli, a chest CT scan (Fig. A-B-C) was performed. No arterial filling defects were visualized. A patchy crazy paving pattern with ground glass opacities and thickened interlobular septa was seen right upper and lower apical lobe and in the left upper lobe. Seen the recent history of hip fracture and surgery, the diagnosis of pulmonary fat embolism was made. The patient was admitted for monitoring and treated with supplemental oxygen and fully recovered within a few days. There was no need for mechanical ventilation.

Comment

Pulmonary fat embolism is an infrequent complication of long bone fracture, occurring in 1%-3% of patients with simple tibial or femoral fractures but in up to 20% of individuals with more severe trauma. Less common causes are hemoglobinopathy, major burns, pancreatitis, overwhelming infection, tumors, blood transfusion, and liposuction.

Clinically, fat embolism manifests 24 to 72 hours after the trauma of surgery, but may rarely occur as early as 12 hours or as late as two weeks after the inciting event. Patients present with a spectrum of severity. This can range from subclinical fat embolism, over tachycardia, tachypnea and fever to fulminant acute fat embolism syndrome involving cor pulmonale, diffuse petechiae and neurological signs such as confusion, stupor and coma.

The mechanisms of fat embolism are not completely understood but presumably are twofold. First there is the production of free fatty acids, which initiates a toxic reaction in the endothelium. This process is complicated additionally by the accumulation of neutrophils and other inflammatory cells, which cause damage to the vasculature. The second mechanism is the mechanical obstruction of the pulmonary vasculature by fat globules and aggregates of red blood cells and platelets.

Chest radiograph findings are important in the diagnosis of this syndrome. However the radiographic appearance is nonspecific and variable. Chemical pneumonitis with secondary damage such as micro hemorrhage and edema account for the classic onset of multiple, diffuse, bilateral

opacities and interlobular septal thickening on CT 1 to 3 days after a traumatic event or intervention. These findings often resemble pulmonary edema without pleural effusion and demonstrate a predilection for the basilar and peripheral lung regions. The radiologic differential diagnosis includes lung contusion, pulmonary edema and aspiration.

Supportive care is the mainstay of therapy for clinically apparent fat embolism and most patients fully recover.

Fat embolism is a condition with a complex presentation and there are no specific tests for its definitive diagnosis. Early recognition of the classic pulmonary manifestations on CT is an important step in the diagnosis of fat embolism.

Reference

1. Han D., Lee K.S., Franquet T., et al.: Thrombotic and Nonthrombotic Pulmonary Arterial Embolism: Spectrum of Imaging Findings. *Radiographics*, 2003, 23: 1521-1539.

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