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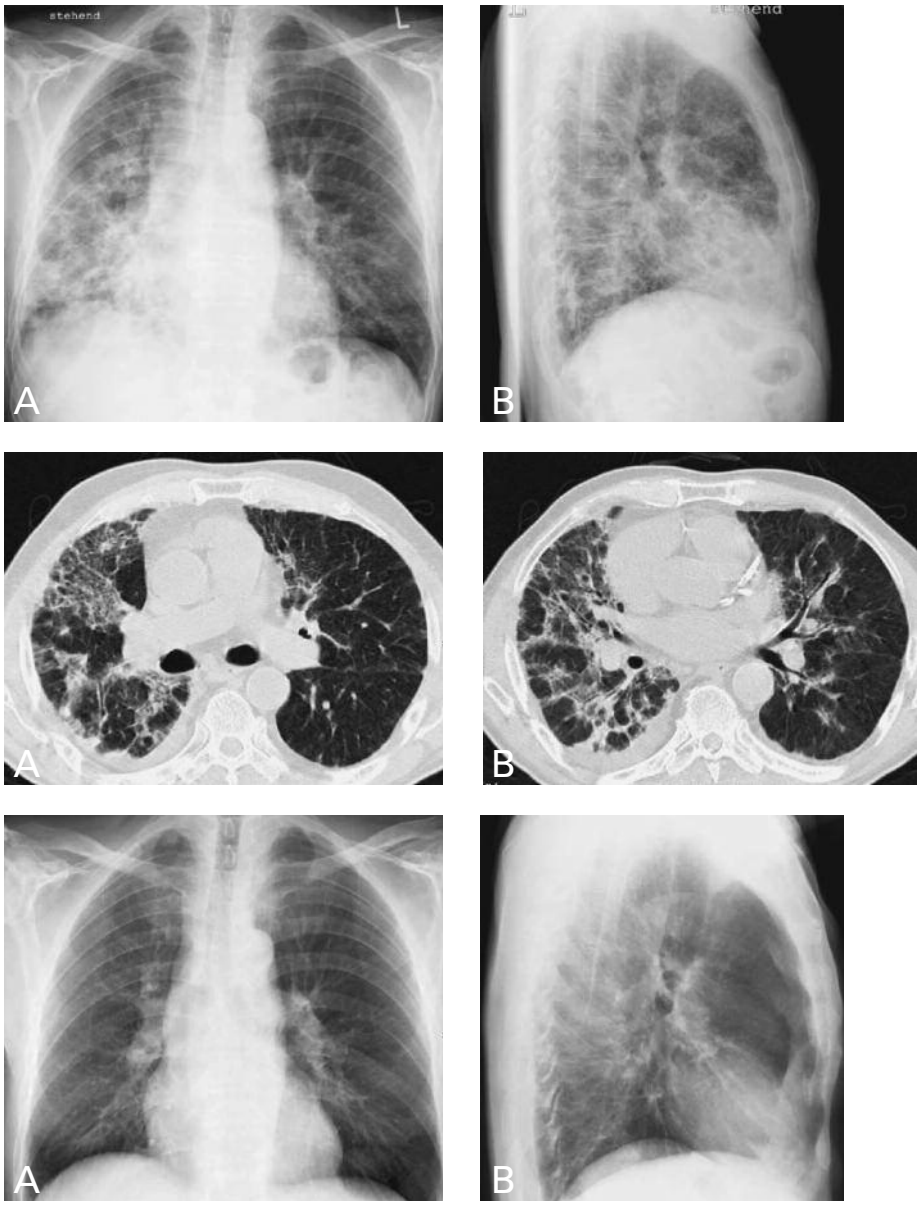
AMIODARONE PNEUMONITIS

T.A. Bley[1,2], C.J. François[1]

Key-word: Lung, effects of drugs on

Background: A 65-year-old male patient presented with progressive shortness of breath over the past 3 months, recently even at rest. He neither complained about fever nor about chest pain. The laboratory findings were normal with only a slight increase in leucocytes.
A significant restrictive ventilation disorder was noted on plethysmography. His past medical history included three vessel coronary artery disease, arterial hypertension with hypertensive cardiomyopathy and atrial flutter. Carcinoma of the urinary bladder and prostate cancer were diagnosed and successfully treated 10 and 12 years earlier, respectively.



1. Department of Radiology, University of Wisconsin–Madison, Madison, Wisconsin, USA and 2. Department of Diagnostic Radiology and Medical Physics, University Hospital Freiburg, Germany

Fig. 1A 1B 2A 2B 3A 3B

Work-up

Chest X-ray (Fig. 1, A: AP view and B: lateral view) shows asymmetrical reticular opacities, mainly in the right lower and middle lobe. Please note volume loss of the right lung as depicted by elevation of the diaphragm and mediastinal shift to the right.

Chest CT (Fig. 2) (lung window settings) demonstrates thickening of the bronchial walls and inter- and intralobular septae as well as ground glass opacities and peripherally located areas of consolidation. Bilateral pleural effusions are present, greater on the right.

Chest radiographs after six months following cessation of amiodarone and after corticosteroid treatment (Fig. 3, A: AP view and B: lateral view) shows complete remission of the initial findings. The reticular pattern with patchy opacities that was seen initially has resolved entirely.

Radiological diagnosis

Given the history of this patient, which also included the use of amiodarone, drug induced lung toxicity, *amiodarone pneumonitis* was suspected. The diagnosis was validated by trans-bronchial biopsy. Amiodarone treatment was stopped and treatment with corticosteroid treatment was initiated. Differential diagnosis of amiodarone pneumonitis include: ventricular dysfunction, infectious, eosinophilic, or organizing pneumonia, pulmonary infarction, exogenous lipoid pneumonia, bronchoalveolar carcinoma and lymphoma.

Discussion

Amiodarone is a class III anti-arrhythmic drug used for treatment of refractory cardiac tachyarrhythmias.

It accumulates in the liver and lung, and may lead to potentially fatal pulmonary toxicity in 5% of the patients.

Amiodarone pneumonitis was first described in 1980. Its prevalence in patients treated with amiodarone reached up to 15%. With higher age and higher dosage of amiodarone, the risk of amiodarone pneumonitis increases. Due to its long tissue half life, both onset of lung toxicity and clearing following cessation may take several months. However, pulmonary toxicity may begin after a few days of amiodarone usage or more than a decade after initiation of treatment. Patients typically present with progressive dyspnea that may have lasted for several months and that is often accompanied by malaise, nonproductive cough, and pleuritic chest pain. Elevated erythrocytes sedimentation rate and leucocytosis are commonly found.

Amiodarone pneumonitis manifests in the pulmonary interstitium or in the alveolar space. Diuresis helps to distinguish amiodarone pneumonia from interstitial pulmonary edema. To confidently establish a specific diagnosis, a lung biopsy of significant size may be required, the recognized diagnostic gold standard.

On chest radiographs and CT scan, focal, patchy or diffuse opacities may be found bilaterally, typically peripheral in location. These opacities may have high attenuation on non-contrast-enhanced CT as amiodarone gets incorporated into type II pneumocytes.

Amiodarone pneumonitis may result in amiodarone induced pulmonary fibrosis, which develops in approximately 5-7% of patients with amiodarone pneumonitis. Amiodarone induced pulmonary fibrosis is an irreversible condition with limited or short term response to corticosteroid treatment and an adverse outcome. Mortality among hospitalized patients with amiodarone pneumonitis is high with approximately 30%. Chest radiographs and pulmonary function tests are recommended in every patient to detect onset of potential amiodarone pneumonitis. Follow-up studies are warranted within the first two years on a regular basis, especially in patients who are at greater risk to develop amiodarone pneumonitis, e.g. patients with poor lung function, COPD, pulmonary emphysema or previous pulmonary surgery.

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