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PHOTO QUIZ

An enlarged heart with hyperdense consolidation

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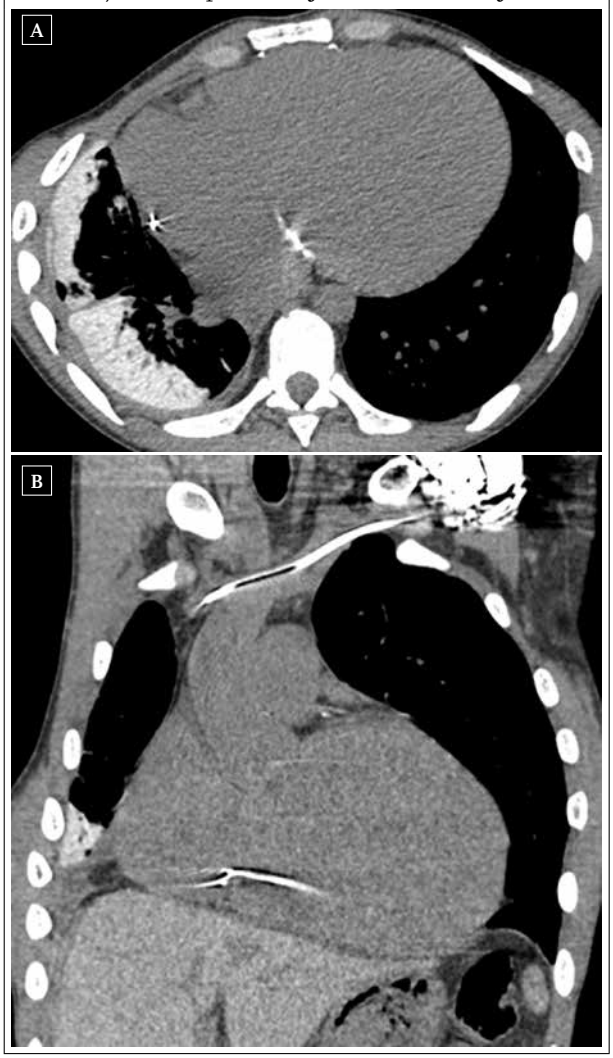
CASE REPORT

A 47-year-old man presented with progressive dyspnoea and dry cough. He had a history of dilated cardiomyopathy and ventricular tachyarrhythmia. The patient had been treated with placement of an implantable cardioverter-defibrillator and amiodarone (200 mg/day for two years). On chest auscultation, fine crackles were audible at the right lung base. Blood count and routine serum biochemistry test results were normal except for a slightly elevated alanine transaminase level (60 U/l). Chest X-ray and computed tomography (CT) demonstrated an enlarged heart, right pleural effusion, and hyperdense consolidation in the right lung (*figure 1*). The liver parenchyma was also dense.

WHAT IS YOUR DIAGNOSIS?

See page 321 for the answer to this photo quiz.

Figure 1. *Axial (A) and coronal (B) computed tomographic images demonstrating increased heart volume, right pleural effusion, and peripheral hyperdense consolidation in the right lower lobe of the lung. Note also the hyperdensity of the liver (the liver is denser than the heart) and the presence of a cardioverter-defibrillator*



ANSWER TO PHOTO QUIZ (PAGE 317)

AN ENLARGED HEART WITH HYPERDENSE CONSOLIDATION

DIAGNOSIS

These tomographic changes were consistent with amiodarone-induced pulmonary toxicity (APT). Amiodarone was stopped, and an alternative antiarrhythmic agent was used. The patient's symptoms improved, and follow-up CT demonstrated regression of the consolidation.

Amiodarone is one of the most commonly used antiarrhythmic medications worldwide, frequently employed in the treatment of supraventricular and ventricular arrhythmias. This drug is an iodine-containing compound that tends to accumulate in several organs, including the liver and lung parenchyma. It has been associated with significant adverse effects, the most serious of which is APT.

The spectrum of APT ranges from mild chronic or subacute to rapidly progressing acute lung disease, including acute respiratory distress syndrome with high mortality. Symptoms can include progressive dyspnoea, dry cough, fever, malaise, and pleuritic chest pain. The most common CT findings include septal thickening, interstitial fibrosis, and consolidations. The drug's high iodine content enables the detection of amiodarone deposits in the lung by CT as high-attenuation focal or multiple parenchymal opacities. The association of dense lung air-space consolidations with high density of the liver and/or spleen is characteristic of amiodarone impregnation.

The diagnosis of APT is based on exclusion because the signs and symptoms are non-specific, and no laboratory test allows the diagnosis. APT should be suspected in any patient taking amiodarone who has new or worsening symptoms and/or new infiltrates on a chest X-ray. Early recognition of APT is important because discontinuation of amiodarone could prevent its progression. The prognosis is usually good in cases of chronic or subacute disease. Open lung biopsy should be avoided because of the tendency for APT to worsen after thoracic surgery, and because affected patients usually have impaired cardiac and pulmonary functions.