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CE - MEDICAL ILLUSTRATION

Amiodarone-induced pulmonary toxicity

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A 35-year-old woman presented to our clinic with a dry cough and progressive shortness of breath of functional class 3 that had been bothering her for 3 weeks. Her past medical history included severe symptomatic rheumatic mitral valve stenosis with mitral valve area (MVA) of 0.9 cm for which she had undergone percutaneous balloon mitral valvotomy (BMV) 4 years before based on guidelines of the American College of Cardiology (ACC) and the American Heart Association (AHA). Her medication history included digoxin 0.125 mg per day, lasix 20 mg per day, metoprolol 50 mg per day, warfarin (dosage adjusted to reach an INR of 2–3), monthly penicillin injection, and amiodarone 200 mg twice a day, which had been added to her medications due to transient episodes of atrial fibrillation (AF) with rapid ventricular response 2 months prior. The initial dosage was 200 mg q6 h for 1 week and then reduced to 200 mg twice a day.

On physical examination, she appeared mildly tachypneic with a respiratory rate 22 breaths/min, and inspiratory crackles up to the mid-lung fields. She was afebrile. Pulse oximetry was 90% oxygen saturation on room air. The chest X-ray study showed: (Fig. 1), bilateral patchy air space and interstitial infiltrates consistent with mixed interstitial and alveolar pneumonitis in the lower lobes of both the lungs. Echocardiography showed a mildly reduced left ventricular ejection fraction and MVA of 1.2 cm^2, which was similar to the previous study performed 10 months earlier. Given a concern for amiodarone-induced lung toxicity, amiodarone was immediately discontinued. High resolution computed tomography (CT scan) of the lung showed a diffuse ground glass mosaic pattern of the lung, which supported the diagnosis of amiodarone toxicity. Prednisolone 40 mg per day was started, and was tapered in 2 weeks. After 3 months, a control chest X-ray study (Fig. 2) showed significant resolution of the opacities with mild fibrotic lesions in the lower lobe of the right lung.

ACC/AHA recommends BMV in symptomatic patients with moderate to severe MS (i.e., a mitral valve area $< \approx 1$ cm^2/m^2 body surface area (BSA) or <1.5 cm^2 in normal-sized adults), and with favorable valve morphology: no or mild MR, and no evidence for left atrial thrombus as was the case in this patient [1]. Despite widespread use since 1967, amiodarone had not been reported to cause any form of pulmonary disorder until 1980 [2]. Potential risk factors for the development of this

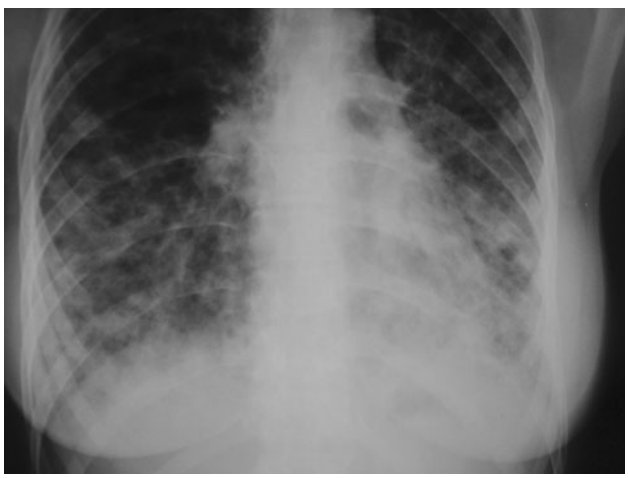

Fig. 1 The initial chest X-ray study showing bilateral patchy air space and interstitial infiltrates consistent with mixed interstitial and alveolar pneumonitis in the lower lobes of both lungs

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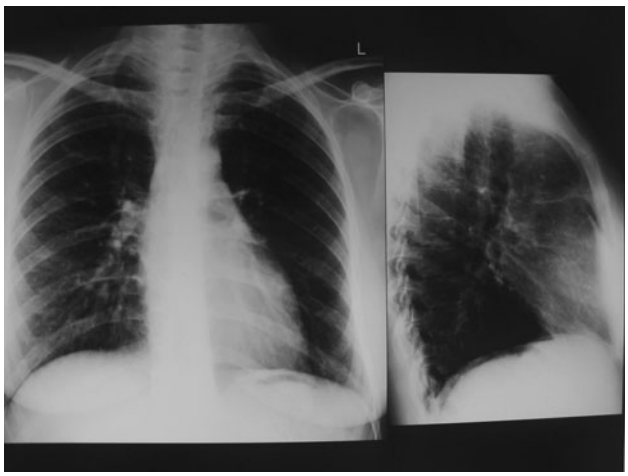

Fig. 2 The second chest X-ray study showing marked improvement with mild fibrotic lesions in the lower lobe of the right lung

complication include a high cumulative dose, a daily dose >400 mg/day, duration of therapy exceeding 2 months, older age, and underlying lung disease [3]. Patients undergoing operation or pulmonary angiography while receiving amiodarone are at increased risk. Several forms of pulmonary disorders may occur in patients treated with amiodarone [4]. The most common form is a chronic interstitial pneumonitis, although bronchiolitis obliterans with organizing pneumonia, ARDS, and a solitary pulmonary mass of fibrosis can also be seen [3, 5]. Once the diagnosis is suspected, the drug should be stopped immediately. If lung involvement is not extensive, discontinuation of the amiodarone may be sufficient. Corticosteroid drugs are considered appropriate in cases with extensive involvement on imaging or hypoxemia, in an attempt to expedite the recovery process and perhaps to minimize the likelihood of lung fibrosis [3]. The prognosis is usually favorable.

Conflict of interest None.

References

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