## Medical Image of the Week: Acute Amiodarone Pulmonary Toxicity



Figure 1. Chest X-ray showing diffuse interstitial markings, right upper lobe consolidation, small pleural effusions, thoracotomy wires and external leads.



Figure 2. Axial image of the thoracic CT scan showing increased interstitial markings, ground glass opacities and bilateral pleural effusions.

A 71 year old man with a medical history significant for chronic obstructive pulmonary disease, coronary artery disease with post-operative status coronary artery bypass grafting, heart failure with reduced ejection fraction (25%) and atrial fibrillation/flutter underwent an elective ablation of the tachyarrhythmia at another facility and was prescribed amiodarone post procedure. He started complaining of cough and dyspnea one day post procedure and was empirically treated with 2 weeks of broad spectrum antibiotics. He subsequently was transferred to our facility due to worsening symptoms. He also complained of nausea, anorexia with resultant weight loss since starting amiodarone, which was stopped 5 days prior to transfer. Infectious work up was negative.

On arrival to our facility, he was diagnosed with small sub-segmental pulmonary emboli, pulmonary edema and possible acute amiodarone toxicity. His was profoundly hypoxic requiring high flow nasal cannula or 100% non-rebreather mask at all times. His symptoms persisted despite antibiotics, diuresis, anticoagulation and heart rate control. Steroid therapy was then initiated for acute amiodarone toxicity. Although he reported some improvement in symptoms 2-3 days after initiation of steroids, his oxygen requirement did not improve. Unfortunately he suffered a cardiac arrest on day 10 of admission and did not survive.

Amiodarone is a class B anti-arrhythmic used to treat multiple supraventricular and ventricular tachyarrhythmias. Its adverse effects are usually dose and duration dependent. Amiodarone pulmonary toxicity (APT) has been shown to correlate with total cumulative dose; however acute reactions to amiodarone toxicity have previously been reported. Men are at increased risk for APT, and this risk increases with age and those with pre-existing lung conditions. Diagnosis of APT is predominantly a diagnosis of exclusion; however laboratory tests may show leukocytosis with neutrophil predominance (as in our patient) and imaging may provide a clue for diagnosis. Chest x-ray reveals patchy or diffuse infiltrates, which may have predominance in the upper lobes, particularly the right upper lobe (as in our patient). A thoracic CT scan may show bilateral alveolar or interstitial infiltrates with higher attenuation, secondary to the iodine component of the drug. The current mainstay of treatment is discontinuation of the drug permanently along with steroid therapy typically, 40-60 mg of prednisone a day for an extended period of time.

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## Reference

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