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FIT Clinical Decision Making

DIFFUSE ALVEOLAR HEMORRHAGE SECONDARY TO AMIODARONE TOXICITY: A RARE CAUSE OF CARDIOPULMONARY DECOMPENSATION IN CHRONIC SYSTOLIC HEART FAILURE

Poster Contributions
Poster Hall B1
Saturday, March 14, 2015, 10:00 a.m.-10:45 a.m.

Session Title: FIT Clinical Decision Making: Heart Failure and Cardiomyopathies
Abstract Category: Heart Failure and Cardiomyopathies
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Background: Amiodarone is used for prevention of recurrent ventricular arrhythmias in patients with heart failure. It is generally well tolerated; pulmonary toxicity is usually chronic in nature. Rarely, amiodarone can cause acute diffuse alveolar hemorrhage (DAH) and respiratory failure. Withdrawal of the drug can reverse symptoms.

Case: A 66-year-old male with dilated cardiomyopathy, listed for transplant and on continuous ambulatory milrinone, presents with abrupt-onset shortness of breath. He is intubated for acute hypoxic respiratory failure. His medical history includes chronic kidney disease, ventricular tachycardia, and left ventricular thrombus. Medications, in addition to home intravenous milrinone, include aspirin, amiodarone, torsemide, and warfarin. Exam demonstrates coarse breath sounds, jugular vein pulsation 2 centimeters above the clavicle, and trace bilateral lower extremity edema. Ventilator settings show an FIO2 of 80%.

Decision Making: Chest x-ray demonstrates bilateral patchy opacities. Echocardiogram shows severely reduced systolic function with pulsus alternans. To confirm the suspicion of elevated right- and left-sided filling pressures, the patient undergoes hemodynamic catheterization, which shows right atrial pressure of 9 mm Hg, pulmonary artery systolic pressure of 38 mm Hg, wedge pressure of 16 mm Hg, and cardiac index 2.29 L/min/m2. These values are similar to those obtained on right heart catheterization performed weeks earlier. Given stable hemodynamics, a pulmonary etiology for the patient's acute decompensation is considered. He undergoes bronchoscopy, which yields bloody fluid on three sequential lavages, consistent with DAH. Autoimmune workup is negative. Amiodarone is felt to be the likely culprit and is discontinued; the patient makes an eventual pulmonary recovery.

Conclusion: The present case demonstrates the importance of considering alternative etiologies in patients with chronic systolic heart failure presenting with acute decompensation. Medications may be the cause. Data surrounding acute DAH due to amiodarone are limited to single case reports. A high index of suspicion is warranted when making this rare diagnosis.