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FIT Clinical Decision Making

COCAINE TOXICITY PRESENTING AS ACUTE REVERSIBLE PULMONARY HYPERTENSION AND RIGHT HEART FAILURE

Poster Contributions
Poster Hall B1
Saturday, March 14, 2015, 3:45 p.m.-4:30 p.m.

Session Title: FIT Clinical Decision Making: Structural Heart Disease and Pulmonary Hypertension
Abstract Category: Pulmonary Hypertension and Pulmonary Thrombo-embolic Disease
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Authors: *Mahesh Anantha Narayanan, Toufik Mahfood Haddad, Aiman Smer, Mohamed Ayan, Aryan Mooss, The Cardiac Center of Creighton University, Omaha, NE, USA*

Background: While chronic pulmonary hypertension is a known manifestation of cocaine intoxication, acute reversible pulmonary hypertension with right ventricular dysfunction has been rarely reported and poses diagnostic challenges.

Case: A 56 year old African-American male with polysubstance abuse and chronic pancreatitis was brought to the emergency room after he was found unresponsive. He was hypotensive (70/40 mmHg) and tachycardiac (110/min) with widely dilated pupils. Immediate fluid resuscitation was done and patient was intubated and sedated. EKG revealed sinus tachycardia with right ventricular strain pattern. Bedside echocardiogram showed preserved left ventricle ejection fraction and massively dilated right ventricle (RV) with RV: LV ratio of 0.9 and positive McConnell's sign (mid free wall akinesis of the right ventricle sparing apex) with severe tricuspid regurgitation. His labs showed creatinine 4.4 mg/dl (baseline 1.0), AST 1205 units/L, ALT 1547 units/L, troponin 3.8 ng/ml and lactic acid 9 mmol/L. Urine drug screen showed cocaine, methadone and benzodiazepines.

Decision Making: Differential diagnoses included pulmonary embolism, septic shock and right ventricular infarction. He was started on heparin and antibiotics along with fluids and pressors. Emergent CT chest with angiography did not show pulmonary embolism (PE), pulmonary edema or pericardial effusion. Troponin peaked at 16.3 ng/ml. Left heart catheterization showed normal epicardial coronaries with dominant right coronary and preserved ejection fraction. Right heart catheterization showed elevated pulmonary artery systolic pressure of 50mm Hg with a mildly elevated wedge pressure. At discharge, his RV dilatation significantly improved with RV systolic pressure of 42mm Hg, which improved further at three months follow up.

Conclusion: After excluding coronary artery thrombosis and PE, the only possible explanation for the acute RV dilatation in our case was reversible acute pulmonary hypertension. It is important for clinicians to recognize rare presentations of transient cocaine induced pulmonary vasospasm since it mimics multiple conditions and has a high mortality.