

Title: Case Report: Delirium tremens and acute coronary syndrome

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Delirium tremens is a condition characterized by hallucinations, tachycardia, hypertension, hyperthermia, agitation, and diaphoresis in the setting of acute reduction or abstinence from alcohol. This abrupt discontinuation causes a sympathetic overdrive, increasing oxygen consumption in organs throughout the body. Moreover, chronic alcohol use causes perivascular fibrosis and vascular cell edema in the small vessels of the heart, impairing adequate vasodilation when needed.

A 63-year-old male with a past medical history of non-obstructive coronary artery disease, dilated cardiomyopathy, chronic obstructive pulmonary disease, alcohol and cocaine use disorder, hypertension and hyperlipidemia presented to the emergency department for chest pain and shortness of breath. His last alcohol drink was the night before admission. Physical examination revealed tachycardia and hypertension. Cardiac enzymes were elevated, and an electrocardiogram showed new T wave inversions in lateral leads compared to one done one month earlier. Blood alcohol level was high, and urine toxicology was positive for cocaine. He was admitted as a type 2 myocardial infarction and given medical management in the Intensive Care Unit. He was started on a lorazepam protocol for withdrawal. 48 hours after admission, troponins had trended down, electrocardiogram changes returned to baseline, but the patient developed visual hallucinations, tachycardia, hypertension, and restlessness. Clinical Institute Withdrawal Assessment for Alcohol scale, revised score was 20. Patient had not required benzodiazepines until this point. The patient continued to be restless and combative so a dexmedetomidine drip was started with adequate response. However, 8 hours later, ST-segment elevation was noted on the telemetry strip. Cardiac enzymes and B-Natriuretic Peptide trended up and an electrocardiogram showed ST-segment elevation in V3-V5. The patient denied chest pain but continued to be in active delirium. He was started on a heparin and nitroglycerin drip and transferred to a cardiac catheterization capable institution where he recovered.

This case illustrates the risks involving the care of patients with delirium tremens and an established coronary artery disease. Although the patient was being treated with the standard protocols for alcohol withdrawal, he developed delirium tremens, nonetheless. This hyperadrenergic state leads to coronary vasospasm and increased myocardial oxygen consumption in an already damaged myocardium. In addition to that, active delirium and medications used for withdrawal mask the symptoms of an acute coronary syndrome, making the diagnosis more challenging.