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LETTER TO THE EDITOR

Case report: Cocaine-induced takotsubo cardiomyopathy☆

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Calcium channel blockers

Cocaine is the second most common illicit drug used, and it is the most common cause of drug-related death.[1–2] Cocaine is a sympathomimetic agent, and it acts by blocking the presynaptic reuptake of norepinephrine and dopamine, leading to an accumulation of these neurotransmitters at the postsynaptic terminal.[3] This results in overstimulation of alpha- and beta-adrenergic receptors. The peak effects of cocaine occur within 1-90 min after exposure and can last anywhere from 15 min to 2-3 h depending on the route of administration.[2] Exposure to cocaine can result in hypertensive crisis, vascular complications including cerebral vascular accidents of either thrombotic or hemorrhagic origin, aortic dissection or rupture, and vasculitis.[2] It most commonly affects the cardiovascular system, increasing the risk for myocardial ischemia, coronary artery spasm, myocardial infarction, atherosclerosis, myocarditis, cardiomyopathy, and arrhythmias.[2] The association of cocaine use with Takotsubo cardiomyopathy is a rare occurrence. In this report, we present the case of a 41-year-old male who developed Takotsubo cardiomyopathy with cocaine use Figure 1.

A 41-year-old male with a history of tobacco and cocaine abuse presented with shortness of breath accompanied by chest pain. Chest pain was located on the left side, rated 10/10 in severity, with radiation to the left arm. The patient admitted to using cocaine the previous night. On presentation, he was hemodynamically stable but hypertensive. His electrocardiogram (EKG) showed ST segment elevation in leads V3, V4, and II. Troponin T levels peaked at 0.34 ng/mL, creatine kinase was 295 μ/L, and creatine kinase-myocardial band (CK-MB) was 17.2 ng/mL. He underwent cardiac catheterization, which did not demonstrate significant coronary artery disease but showed severe left ventricular dysfunction with an ejection fraction of 30-35%; left ventricle basal hypokinesis and apical akinesis was also noted. He was started on enalapril. He was not started on any beta-blockers and did not require any benzodiazepines. Over the course of his hospital admission, his chest pain improved and his condition stabilized. An echocardiogram was repeated 4 months following discharge, demonstrating improved left ventricular ejection fraction of 55-60% and no left ventricular wall motion abnormalities Figure 2.

Takotsubo cardiomyopathy is a rare complication of cocaine abuse. To our knowledge, no previous cases of cocaine use and the development of Takotsubo cardiomyopathy have been reported in literature. The criteria to diagnose Takotsubo cardiomyopathy include transient hypokinesis, akinesis, or dyskinesis of the left ventricle wall with or without apical involvement in the absence of obstructive coronary artery disease; in addition, Takotsubo cardiomyopathy is often related to a stressful trigger.[4–5] It is believed that Takotsubo cardiomyopathy results from sympathetic excitation of the central nervous system, triggering the release of

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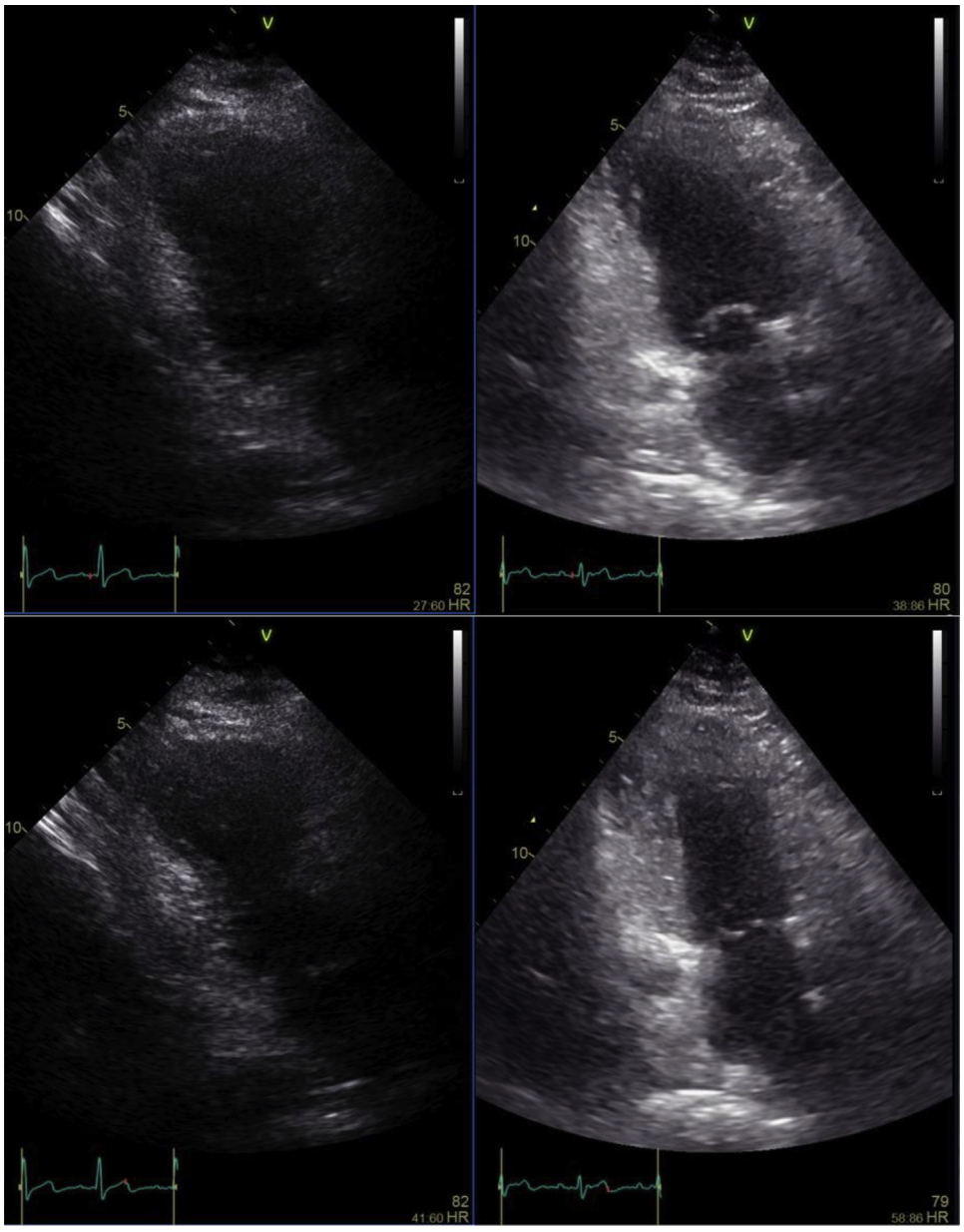

Figure 1 Two-chamber echocardiogram view demonstrating diastolic phase on admission on the top left and 4 months later on the top right. Two-chamber echocardiogram view demonstrating systolic phase on admission on the bottom left and 4 months later on the bottom right. Left ventricle basal hypokinesis and apical akinesis noted in the bottom left image.

catecholamines, causing hyperdynamic basal contraction and apical systolic dysfunction.[4–5] As such, it is not surprising that cocaine use can be associated with the development of Takotsubo cardiomyopathy when it results in excess release of catecholamines and excitation of adrenergic receptors. As with other forms of cardiomyopathy, treatment of Takotsubo should be directed at improving cardiac function and reducing the risk of congestive heart failure. This is often achieved with angiotensin-converting enzyme inhibitors and beta-blockers.[2] Benzodiazepines can also be used to suppress excess adrenergic stimulation, while nitrates and calcium channel blockers can be used to relieve symptoms of coronary ischemia.[2] In many cases, cessation of cocaine use has been associated with improvement in cardiac function. Counseling on drug addiction, detoxification, and rehabilitation programs are therefore integral to reducing the risk of relapse and worsening cardiovascular complications. Takotsubo cardiomyopathy is a rare complication of cocaine abuse and must be distinguished from other causes as monotherapy with beta-blockers could have fatal consequences for the patient.

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