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Case Report

First case of stress cardiomyopathy as a result of methadone withdrawal secondary to drug-drug interaction

Abstract

We describe the first case of stress cardiomyopathy secondary to a drug-drug interaction. A 44-year-old man was admitted for acute agitation, hallucinations, tachycardia, and fever within 2 hours of ingestion of naltrexone prescribed to stop alcohol consumption. He had been receiving methadone (120 mg/d) for several months for a history of heroin use; thus, acute opiate withdrawal syndrome secondary to naltrexone treatment was diagnosed. Because electrocardiography showed diffuse ST-segment elevation, a transthoracic echocardiography was performed. It revealed apical akinesia of the left ventricle with a reduction in systolic function. The echocardiogram showed an ejection fraction of 35%, apical and midventricular wall motion abnormalities of the left ventricle, and a cardiac output of 4 L/min without coronary stenosis. The patient was transferred to the cardiologic intensive care unit with a diagnosis of transient left ventricular apical ballooning syndrome secondary to acute opiate withdrawal syndrome. It is likely that opioid withdrawal, inducing a marked increase in catecholamine plasma concentrations, contributed to the development of stress cardiomyopathy. To our knowledge, this is the first case of stress cardiomyopathy described after abrupt opiate withdrawal secondary to a drug-drug interaction.

Stress cardiomyopathy, or Tako-Tsubo syndrome, is characterized by transient left ventricular dysfunction and electrocardiographic changes that mimic acute myocardial infarction, with no evidence of obstruction in epicardial coronary arteries [1]. We describe the first case of stress cardiomyopathy secondary to a drug-drug interaction.

A 44-year-old man was admitted for acute agitation and hallucinations within 2 hours of ingestion of naltrexone (50 mg/d) prescribed to stop alcohol consumption. He also presented with tachycardia (120/min), fever (38°C), and hypertension (160/90 mm Hg) but did not have chest pain. He had been receiving methadone (120 mg/d) for several months for a history of heroin use; thus, acute opiate withdrawal syndrome secondary to naltrexone treatment was

diagnosed. Intravenous midazolam (10 mg) was used for sedation. Because electrocardiography showed diffuse STsegment elevation on V₁ through V₆, D2, D3, and aVF, a transthoracic echocardiography was performed. It revealed apical akinesia of the left ventricle with a reduction in systolic function. Troponin T was measured at 0.79 and 1.99 ng/mL. The echocardiogram showed an ejection fraction of 35%, apical and midventricular wall motion abnormalities of the left ventricle, and a cardiac output of 4 L/min without coronary stenosis. The patient was transferred to the cardiologic intensive care unit. Electrocardiogram revealed gradual improvement corresponding to improvement of the patient's withdrawal syndrome. The patient was discharged after 11 days with a diagnosis of transient left ventricular apical ballooning syndrome secondary to acute opiate withdrawal syndrome.

The exact mechanism of stress cardiomyopathy has not been identified, but the role of massive release of catecholamines has been demonstrated [2]. It is likely that opioid withdrawal, inducing a marked increase in catecholamine plasma concentrations, contributed to the development of stress cardiomyopathy [3]. Only 2 cases of stress cardiomyopathy have been reported in association with acute opioid withdrawal [4,5]. To our knowledge, this is the first case of stress cardiomyopathy described after abrupt opiate withdrawal secondary to a drug-drug interaction. Given the increased use of morphinic agonists and antagonists for treatment of addiction, which could be prescribed together to the same patients, clinicians should be aware of this potential complication.

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