
Case Report

Two Ecstasy-Induced Myocardial Infarctions During A Three Month Period in A Young Man

Saeed Sadeghian MD*, Soodabeh Darvish MD*, Shirin Shahbazi*,
Mehran Mahmoodian MD*

Ecstasy normally contains 3,4 methylenedioxyamphetamine (MDMA) that increases the levels of serotonin, dopamine, and epinephrine in the central nervous system with consequent adverse effects on the cardiovascular system. Herein, we presented a case of ecstasy abuse which resulted in two episodes myocardial infarction during a three month period; the second episode led to death due to thrombus formation.

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Introduction

Amphetamine is a synthetic stimulant which has got immense popularity among teenagers and young adults in recent years. Some derivatives of amphetamine that are commonly abused include methamphetamine, also known as “speed”, used in oral or intravenous forms or synthesized into a crystalline smokeable form, the so-called “ice.”¹

3,4 methylenedioxyamphetamine, “ecstasy” (MDMA) and its legal counterpart 3,4 methylenedioxyethylamphetamine, “eve” (MDEA) are analogs of 3,4 methylenedioxyamphetamine (MDA), a drug that was originally used for suppression of appetite.¹

Ecstasy tablets normally contain MDMA that has the similarity to both amphetamines and hallucinogens. MDMA increases the levels of serotonin, dopamine, and epinephrine in the central nervous system, which cause excitation of the sympathetic nervous system with consequent adverse effects on the cardiovascular system.²

Use of MDMA has been associated with sudden death and cardiovascular collapse.³ However; there are few cases of myocardial infarction (MI) after using MDMA.³⁻⁸

Herein, we reported a case with two episodes of MI, the second of which led to death, during a 3-month period of using crystalline (“ice”).

Case Report

A 24-year-old man was admitted to Tehran Heart Center on March 7, 2005 for angiography. He complained of atypical chest pain and nausea. He had also a history of an inferior wall acute MI one month before, which was not treated by thrombolytic therapy because of late referral.

He had used crystalline and denied habitual use of any other kinds of amphetamines. Other habits included smoking (10 packs/year). No heart disease was noted in his family members. He did not have hypertension or diabetes. The electrocardiogram (ECG) showed Q-wave and inverted T in II, III, and AVF (Figure 1). Cardiac catheterization showed normal coronary arteries (Figure 2) with inferoapical akinesia and ejection fraction (EF) of 50%.

The patient received diltiazem 30 mg three times a day and nitroglycerin (Nitrocontin) 6.4 mg two times a day. The patient was free of symptoms

Authors' affiliation: *Research Department, Tehran Heart Center, Tehran University of Medical Sciences, Tehran, Iran.

Corresponding author and reprints: Saeed Sadeghian MD, Research Department, Tehran Heart Center, North Karegar Ave., Tehran1411713138, Iran.

Tel: +98-218-802-9257; Fax: +98-218-802-9256

E-mail: sadeghian15@yahoo.com.

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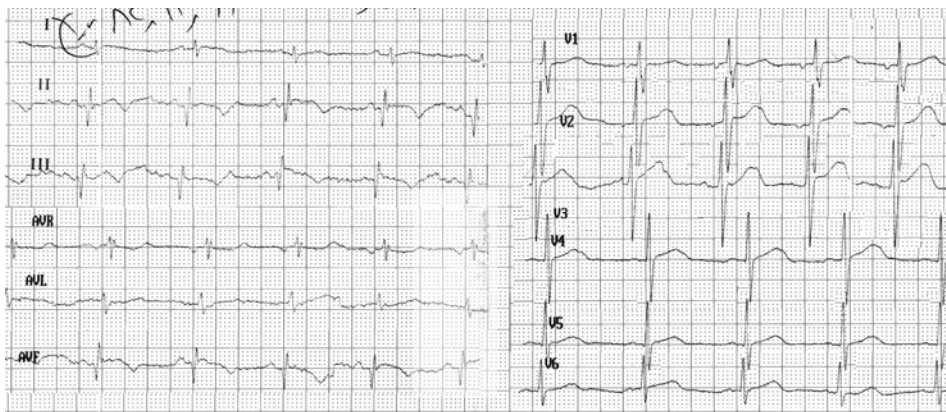


Figure 1. ECG at first admission.

by the second day after the admission and was discharged on March 8, 2005.

About three months later, on June 2, 2005, he was referred to our center from another hospital due to MI, which was not treated by thrombolytic therapy because of late referral. On admission, the patient complained of dyspnea. Examination of the cardiovascular system was normal but examination of the respiratory system revealed a fine bilateral crackle in the lower half of the lungs.

He had a blood pressure of 120/85 mmHg and a pulse rate of 95 beats/min. Laboratory tests showed a creatine kinase of 70 IU/L, troponin of 12.78 $\mu\text{g/L}$, hemoglobin of 14.2 g/dL, hematocrit of 42%, leukocyte count of 16 300/ mm^3 , prothrombin time of 14 s, activated partial thromboplastin time of 21 s, cholesterol of 214 mg/dL, high-density lipoprotein of 44 mg/dL, low-density lipoprotein of 109 mg/dL, lipoprotein A of 62 mg/dL, triglyceride of 306 mg/dL, and fasting blood sugar of 128 mg/dL. The ECG showed Q-wave and ST

elevation from V_{1-6} (Figure 3). Echocardiography demonstrated an EF of 25% with anteroapical dyskinesia.

Other laboratory findings included a positive CRP (35 mg/L) and an elevated ESR (35 mm/hr). Hepatic enzymes were in the upper limit of normal range (alanine aminotransferase 42 IU/L, aspartate aminotransferase 29 IU/L, alkaline phosphatase 309 IU/L, and lactate dehydrogenase 659 IU/L). Serum electrolytes included a sodium of 137 mEq/L, potassium of 4.3 mEq/L, magnesium of 2.3 mg/dL, calcium of 4.2 mg/dL, and a phosphorus of 5.4 mg/dL. Total and direct bilirubin levels were 0.9 mg/dL and 0.3 mg/dL, respectively. Hepatitis B surface antigen, human immunodeficiency virus antigen (HIV Ag), and HIV antibody were all negative.

Antroseptal MI, right bundle branch block, and congestive heart failure were diagnosed. Angiography five days after the admission showed 100% stenosis in the proximal part of the left

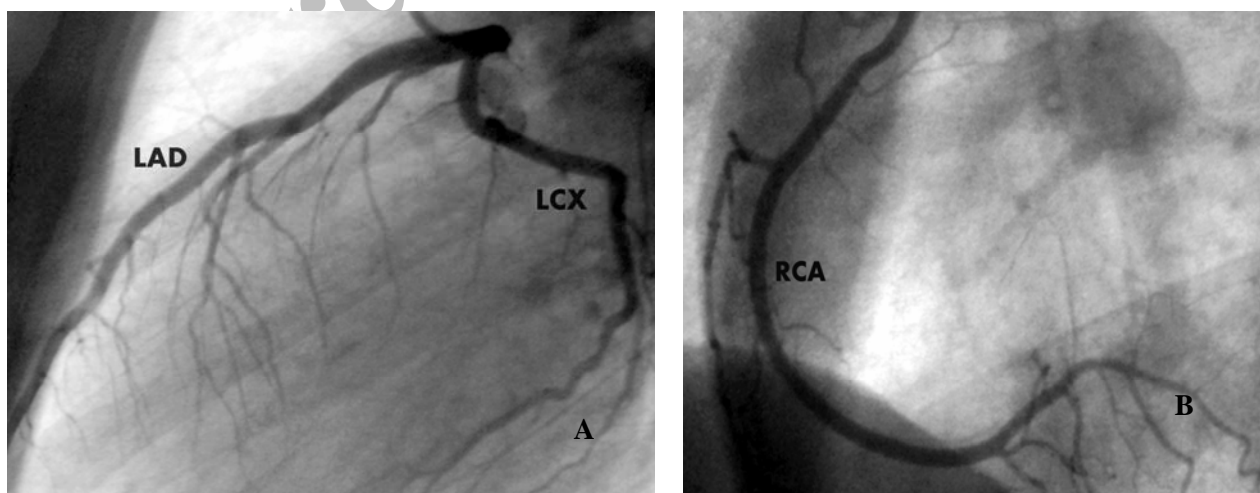


Figure 2. Angiography at first admission (normal coronary artery); A) Angiography of the left coronary system; B) Angiography of the right coronary artery.

LAD = left anterior descending; LCX = left circumflex; RCA = right coronary artery.

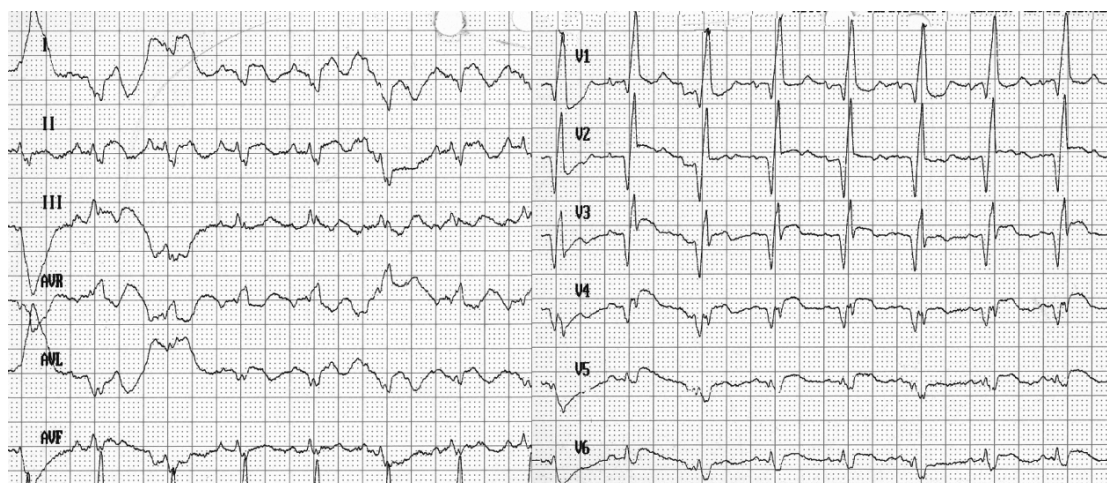


Figure 3. ECG at second admission.

anterior descending artery (LAD) (Figure 4).

Because the patient was in a critical period between recent MI and thrombus resolving, and for fear of thromboembolic event, we did not use any invasive therapy and chose the pharmacologic treatment with heparin instead.

Eight days after the admission on June 10, 2005, he developed cardiogenic shock and died before any chance of revascularization.

Discussion

This case underlines the importance of taking a careful drug history in young patients, as illegal recreational sympathomimetic drugs are widely used in this age range.

Adverse effects of MDMA on cardiovascular system, predominantly relates to activation of the sympathetic nervous system by increased release of catecholamine, inducing various types of

arrhythmias, asystole, and cardiovascular collapse.¹ Arrhythmias are caused by its stimulation of the release of epinephrine, dopamine, and serotonin from the central and autonomic nervous systems.⁹ The role of MDMA on coronary vessels is not well documented.¹⁰ Few cases of MI directly linked to the use of amphetamines have been reported in the medical literature.³⁻⁸ Hong et al³ described a 31-year-old woman admitted for smoking high amounts of crystal amphetamine. She presented with an acute MI due to diffuse arterial vasospasm. On postmortem examination, coronary arteries were found free of atherosclerosis.

Qasim et al⁶ reported a 23-year-old man with inferolateral MI who had used ecstasy and crack cocaine. On angiography, he demonstrated normal coronary arteries and left ventricular function. Similar to this study, Ragland et al⁴ reported a normal angiography in a 45-year-old woman with lateral wall MI. Dowling et al¹¹ reported a 25-year-

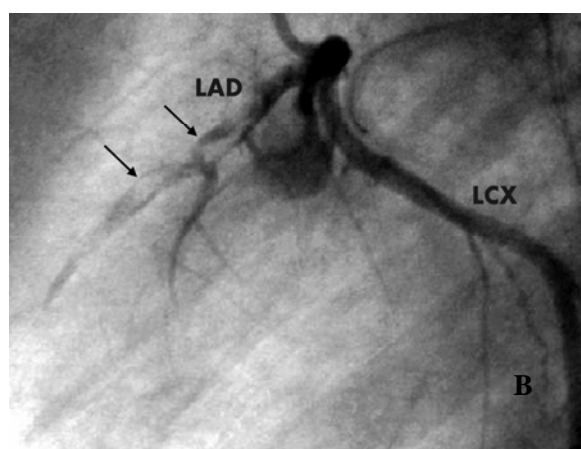
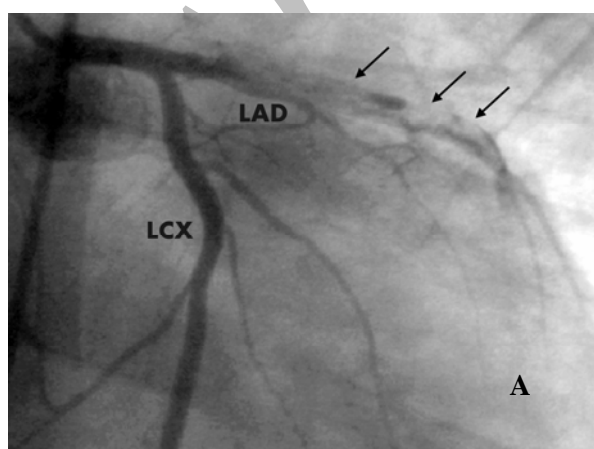


Figure 4. Angiography at second admission (thrombus formation in the proximal part of the left anterior descending [LAD] coronary artery); A) Angiography of the left coronary system left anterior oblique (LAO); B) Angiography of the left coronary artery (lateral).

old man with MI and insignificant atherosclerotic plaques. Dowling et al also described an 18-year-old woman who presented with ventricular fibrillation after ingestion of ecstasy tablet. Similarly, Packe et al⁵ reported a 25-year-old man with MI and subsequent ventricular fibrillation with angiographically normal coronary arteries.

Unlike these studies, in one case reported by Lai et al⁸ and another presented by Bashour¹² the existence of thrombus in coronary arteries was documented.

Chronic amphetamine abuse has also been reported to be associated with necrotizing vasculitis. Angiographically, this is demonstrated by “beading” and narrowing of small- and medium-sized arteries. This can involve multiple organ systems and may contribute to MI in amphetamine abusers.¹³ There is also postmortem examination and animal evidence of patchy myocardial necrosis,⁶ which may result from direct myocardial toxicity of MDMA metabolites.

In most reports describing amphetamine-associated MI, the mechanism implicated is that of catecholamine excess. Similar to cocaine, amphetamines can potentially cause coronary vasospasm and platelet aggregation due to catecholamine release.¹

Compared with the previous case reports, our case presented with normal coronary arteries at first admission. It can be due to coronary artery vasospasm or resolved thrombosis. On the other hand, in his second admission, angiography showed total occlusion of the proximal part of the LAD — similar to reports of Bashour¹² and Lai et al.⁸ These findings indicate that induction of thrombosis is probably the likely mechanism of amphetamine-related acute MI.

Coronary vasospasm can cause a disturbance in the endothelial function of the coronary arteries and plays a role in initiating coronary occlusion, which in turn perpetuates the thrombotic event that leads to MI.¹⁴ Therefore, medical treatment for amphetamine-induced MI, like for cocaine-induced MI, deviates from the standard treatment in certain aspects. There are good reasons to consider early coronary angiography and establish whether there is atherosclerotic coronary artery disease, thrombus, or vasospasm. This would allow better targeting of treatment with antiplatelet agents,

percutaneous intervention, or drugs to treat coronary artery spasm.

This case demonstrated that, in young patients without apparent risk factors who present with acute coronary syndrome, high suspicion of drug abuse should be considered.

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