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CASE REPORT

Ecstasy induced acute systolic heart failure and Non-Ischemic Cardiomyopathy in a young female: a rare case report and literature review

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ABSTRACT

Ecstasy or 3,4-methylenedioxymethamphetamine (MDMA) is an illicit recreational drug. Effects include euphoria, increased sensory awareness, and central stimulation. Although various arrhythmias, as well as dilated cardiomyopathy, have been previously noted to occur with chronic use, cardiac toxicities are seldom reported in an acute setting. Herein, we present a 28-year-old female patient with no prior medical condition that presented to the Emergency Department with chest pain following intake of MDMA. Electrocardiographic findings, as well as laboratories, were suggestive of possible Acute Non-ST elevation myocardial infarction. Upon admission, cardiac catheterization revealed patent coronary arteries. Stark regional wall motion abnormalities were observed along with reduced ejection fraction. Acute systolic heart failure was treated with standard medical management. Subsequent reassessment of ventricular function with Echocardiography revealed marked improvement. This article describes a case of MDMA induced heart failure, including details of evaluation, management, and monitoring of patient progress. It brings further attention to potential acute harmful effects of MDMA on cardiac function and viability.

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Ecstasy; non-ST elevation MI; heart failure; non-ischemic cardiomyopathy; systolic dysfunction

1. Introduction

MDMA is a synthetic compound with structural and pharmacologic similarities to both amphetamines and mescaline. Molly, E, X, Adam, yellow star, Bugatti, Superman, Ecstasy, etc. are few of its popular names. First developed in 1914 as an appetite suppressant, MDMA found use as a psychotherapeutic agent during the 1970s. It is a commonly abused drug, particularly amongst the young, especially at all-night dance parties (rave) [1]. Among the cardiovascular toxicities documented, MDMA often causes multiple forms of arrhythmia and dilated cardiomyopathy with prolonged use. In this article, we report a unique case of MDMA induced left ventricular dysfunction with details of symptoms, clinical findings of diagnostic testing, and medical management.

2. Case report

A 28-year old female with no known medical comorbidities presented to the Emergency Department with complaints of sudden onset chest pain coupled with a recent fall. Chest pain was described as heaving in nature, intermittent lasting approximately 4 minutes for the last two days before the presentation. It was substernal, 8/10 in intensity, non – radiating, and it resolved spontaneously. The pain was not associated with exertion and lacked any aggravating or relieving factors.

Additionally, the patient also complained of one episode of a fall-related to loss of consciousness lasting around one-minute following a sensation of lightheadedness. Notably, the fall yielded minor trauma to the head and the leg. The patient denied any history of palpitations, nausea, vomiting, headache, shortness of breath, aura, urinary, or fecal incontinence. **The patient had no prior history of heart failure or any symptoms suggestive of heart failure**. The patient endorsed using one tablet of ecstasy, for the first and only time in her life, a day before the incident. The patient also reported marijuana use two days prior. She denied chronic use of any another recreational drug aside from occasional marijuana.

On physical examination, the patient was afebrile, and vitals were unremarkable save for a mildly elevated heart rate of 96 beats per min, respiratory rate of 22 breaths per minute. No orthostatic changes were present. Mild tenderness of the right parietal region was noted overlying the area involved in the fall. Chest, cardiac, and neurological examinations were all unremarkable. Mild epigastric tenderness was detected on abdominal examination. Interestingly, her BMI was 18.0 kg/m2 at the time of presentation but had been as low as at 16 kg/m2 three years prior.

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Laboratories revealed a mild leukocytosis of 13.1x109/L with a concomitant left shift of 73.6%. Both Hemoglobin and Hematocrit were within normal limits. The Chemistry panel did show mild hypokalemia (3.2 mmol/L; 3.5–5.5 normal range), as well as a mildly elevated bilirubin level (2.4 mg/dL) although hepatic enzymes were not elevated. Urine toxicology was positive for marijuana and MDMA.

Finally, the troponin level was elevated at 3.67 ng/mL (Normal: <0.06 ng/mL). However, Electrocardiogram (EKG) on admission (Figure 1) exhibited a normal sinus rhythm with a prolonged QTc interval of 527ms and some non-specific T wave changes in the inferior leads. Chest X-ray was unremarkable. Furthermore, a contrast-enhanced CT of the chest was negative for pulmonary embolism. Head and a cervical spine CT were normal.

Following triage in the ED, the patient was admitted into the Cardiac Care Unit with an initial diagnosis of NSTEMI in the context of syncope. Treatment with Aspirin 325mg, Clopidogrel 75mg, and Low Molecular Weight Heparin was initiated. Supportive measures included Morphine for pain in addition to Nitroglycerin.

Repeat troponin followed at 2 and 8 hours were 3.26 ng/ml and 2.39 ng/ml respectively (Normal: <0.06 ng/mL). On the first day of admission, an Echocardiogram exposed severe left ventricular systolic dysfunction with an Ejection Fraction (EF) of 25–30%. Moreover, hypo-kinesis was observed in the inferior, lateral, anterior, and septal walls. Doppler estimated Right Ventricular Systolic Pressure was also aberrantly elevated at 50–55 mmHg. **There were no signs of a restrictive pattern on Echocardiography**. Lastly, moderate mitral regurgitation, and mild aortic and tricuspid regurgitation was apparent. However, there was **no baseline Echocardiography for comparisoin.**

Urgent cardiac catheterization (Figure 2) was done consistent with echocardiogram results, and severe left ventricular dysfunction was present. Additionally, inferior diaphragmatic and basal anterolateral wall akinesis endured. Left Ventricular End Diastolic Pressure was increased at 38mm Hg. Nonetheless, coronary arteries were normal. **During hospital stay autoimmune disease was excluded**.

Subsequently, the patient was started on heart failure medications, including intravenous Lasix 40mg every 12 hours, Carvedilol 3.125 mg PO q12h and Enalapril 2.5mg PO daily. She was later downgraded to the telemetry unit for further management of Non-Ischemic Cardiomyopathy. She remained there for two more days and received continued medical management. With diuresis, her weight decreased by approximately five pounds. Discharge planning, including consideration for possible placement of AICD/Wearable Cardiac Defibrillator deliberated. Otherwise, the stay in the telemetry unit was uneventful. During the hospital stay, autoimmune disease was excluded.

Nevertheless, an echocardiogram repeated on the fifth day since admission displayed a significant improvement of her EF – up to 45% with a sound resolution of mitral regurgitation. Still, apical and lateral wall hypo-kinesis persisted. The patient was subsequently discharged home on the same doses of Enalapril, and Carvedilol, in addition to, low dose oral furosemide (20mg PO daily). Counseling for abstinence of toxic substances was conducted.

Upon clinic follow-up, the patient reported exertional dyspnea that is relieved with rest. Repeat EKG was notable for a normalized QT duration. **Repeat Echo showed further improvement in EF to 55%, and apical and lateral wall hypo-kinesis also revealed marked improvement**. The patient was recommended to undergo Cardiac MRI for further evaluation to rule out etiologies but opted for secondary consultation.

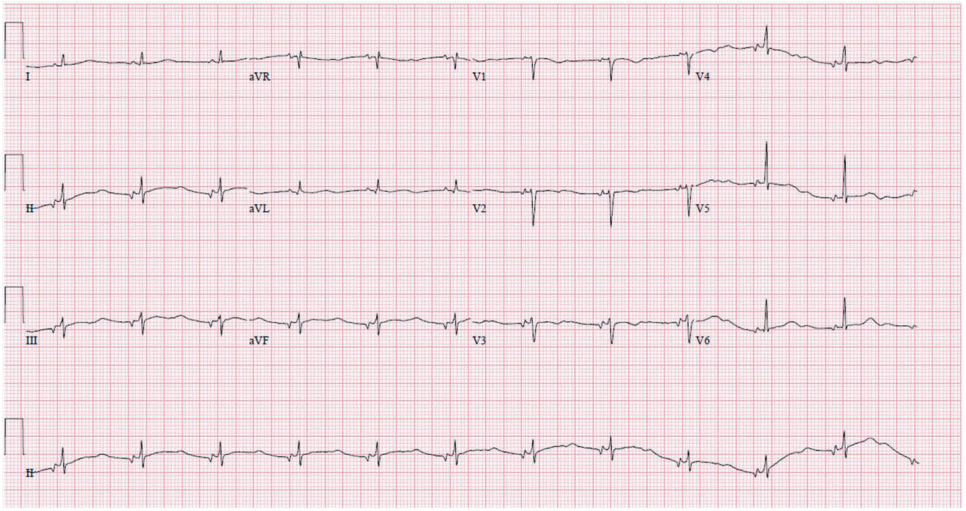

Figure 1. showed normal sinus rhythm with prolonged QTc interval of 527 msec and some non-specific T wave changes over the inferior leads.

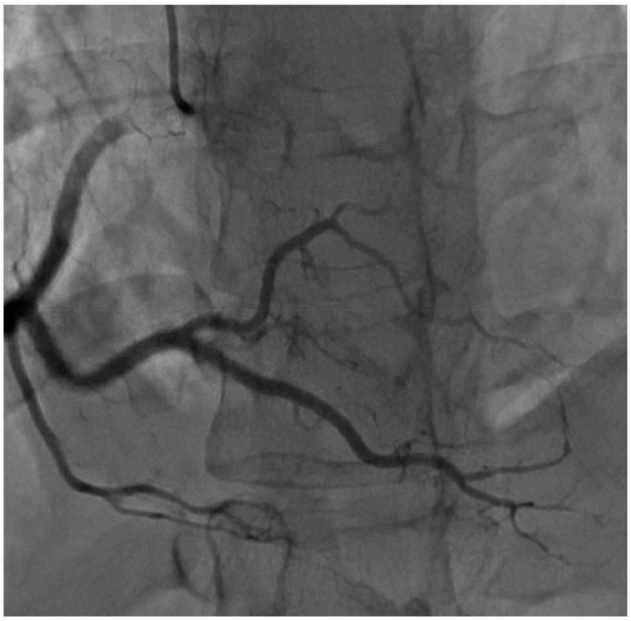

Figure 2. Coronary angiogram showing normal coronary artery.

A peripheral diagnostic evaluation was notable for marked Vitamin D deficiency. For this, a mega-dose treatment regimen was initiated.

3. Discussion

MDMA typically causes increased energy, feelings of euphoria, wakefulness, intimacy, sexual arousal, and disinhibition. This is due to its sympathomimetic amphetamine, which causes the release of endogenous catecholamine (Norepinephrine and Dopamine). Its half-life varies from 12–34 hours, but typically, the effects last 3 to 6 hours and may persist beyond 24 hours [2].

The exact pathophysiology behind the cardiotoxicity remains unknown. It is believed to be due to vasospastic nature, as seen in amphetamine and cocaine. A thrombus, as a cause of MDMA induced MI, has also been reported although rarely.

Data for human and animal studies suggest, MDMA causes lysosomal destabilization by activation of the autophagy-lysosome pathway resulting in myofibril damage and thus LV systolic dysfunction after 24 hours of use [3]. There are reports of various cardiotoxicities with chronic use of MDMA in literature, for instance, dilated cardiomyopathy, cardiac hypertrophy, and cardiomyocyte necrosis [4,5]. A single dose of MDMA has also been demonstrated to induce oxidative stress and myocardial band necrosis, which have mostly been seen with rat models [6,7]. In both humans and rats, MDMA is metabolized by multiple pathways into different products. Two of these products, Dihydroxymethamphetamine (DHMA) and Dihydroxyamphetamine (DHA) are known to generate reactive oxygen species.

Our patient was diagnosed as NSTEMI with severe ventricular dysfunction and had a normal coronary angiogram. Most reports (Table 1), however, have shown that the acute myocardial infarctions due to MDMA are ST-elevation Myocardial Infarction (STEMI). In a case report by Qasim et al.; MDMA associated MI was a STEMI with Antero-apical hypokinesia and the Left Ventricular function was preserved. Similarly, Lai et al. reported a case of STEMI and anteroposterior wall hypo-kinesis following MDMA intake. Moller et al. also described a STEMI following MDMA use showing normal left ventricular systolic function

Table 1. Comparisons of MDMA induced myocardial infarction in different studies.

STUDY	Symptoms Onset.	EKG	Echo Finding	Cath finding	Max troponin/CK
Qasim et al	18 hr	II, III, AVF, V3-6	antero-apical with good LV function	Normal coronaries and normal EF.	CK 553IU
Lai et al	3 hours prior		anteroposterior wall hypokinesis	Thrombus in RCA(8hrs) Thrombus in proximal RCA (day5) Patent RCA no thrombus (day 10)	18.8
Feriyde et al				D1 and AVL <1mm	<0.01–<0.01 679CKMB
Moller et al	<24 hours	I, II, AVL, AVF, V4-6- STE	normal left ventricular systolic function with slight hypokinesis in the apical-anterior, septal and mid-posterior segments	Discrete regional hypo-kinesis, global vasospastic narrowing resolved immediately after intra coronary nitroglycerine.	7.5
Sadeghia et al		Qwave and STE in V1-6	EF20-25% with anteroapical dyskinesia (history of recent MI)	After 5 days 100% stenosis in proximal LAD	CK70, T12.78

with slight hypo-kinesis in the apical-anterior, septal, and mid-posterior segments. Only, Sadeghi et al. reported a case of STEMI and heart failure with reduced ejection fraction (HFrEF) following MDMA use like ours, however, left ventricular dysfunction did not show subsequent improvement. Unlike ours, in the cases mentioned above, the chronicity of MDMA intake has not been described. Likewise, the presence of other confounding factors such as the history of coronary artery disease (CAD), smoking, cocaine abuse which are independent predictors of Coronary Artery blockade and Myocardial Infraction have not been described.

In conclusion, though myocardial infarction with MDMA use is known, the overall incidence appears to be rare. There have been only a handful of reported cases, mostly STEMI with MDMA use. To our knowledge, this is the only case of NSTEMI following MDMA with acute left ventricular dysfunction, its subsequent reversibility, and a normal coronary angiogram.

More extensive observational studies would probably be required to understand better the consequences of MDMA in the heart and its tendency to cause MI.

Disclosure statement

No potential conflict of interest was reported by the authors.

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