

# A Case report of hemodynamic instability, cardiac arrest, and acute severe dyspnea subsequent to inhalation of crystal methamphetamine

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## ABSTRACT

Misuse of stimulants similar to amphetamine is a universal problem. These stimulants cause many complications in their abusers. However, myocardial infarction is rarely reported as a complication of amphetamine abuse. Herein, we report a man aged 42 years presented at the Emergency Department with the chief complaint of acute dyspnea following ice inhalation without history of dyspnea. Within the first hour and a half of admission, the patient was treated by nasal oxygen and bronchodilator aminophylline. However, he did not respond to the initial treatment and lost his consciousness; showed ventricular fibrillation, cardiac arrest, and hemodynamic instability. So, cardiopulmonary resuscitation was immediately initiated for him. The patient was intubated, mechanically ventilated. Also, the synchronized electrical shock was delivered 5 times (200-360 J) along with amiodarone (300 mg intravenously [IV] stat, then 1 mg/min IV infusion for 6 hours and next 0.5 mg/min for 18 hours) to treat the ventricular fibrillation. The arrhythmia was subsequently controlled, and his normal sinus rhythm was resumed. Two hours later, condition of the patient improved, and he was extubated. After two days, when the patient got stable, the echocardiography was performed, which was completely normal.

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## Introduction

Amphetamines belong to the phenylethylamine family compounds with a methyl substitution in the  $\alpha$ -carbon position. Abundant exchanges are possible in the structure of phenylethylamine which results in various amphetamine compounds. One of the frequently abused derivatives of amphetamines is methamphetamine, also known as "speed." Methamphetamines (Meth) can be used in oral or injection forms. They can also be synthesized into a crystalline smokeable form known as "ice." People say that the crystalline form produces a euphoric effect similar to that of the street drug "crack," a smokeable form of cocaine. High-dose abusers use between 2.5 and 15 g of smokeable methamphetamine daily, which is 150 to 1000 times greater than the recommended therapeutic dose and 3 times greater than that used by injection users (1). Crystal Meth damages many body organs such as the brain, kidneys, and liver, and it can cause extensive heart damage. Crystal Meth can cause complications such as arrhythmia, tachycardia, hypertension, brain vasculitis, and or endocarditis.

Study has shown that abusers of methamphetamine have a considerably higher risk of catching heart attacks and strokes due to this damage. Scientists evaluated data of more than 3 million patients referred to Texas hospital with an age range of 18 to 44 years. They found that there is a relationship between heart

attacks and amphetamine use. Excessive consumption of crystal Meth can even cause cardiac arrest or coma (2). Amphetamines and related stimulants are illegal "street" drugs that pose a persistent global problem. It is also very difficult to treat their abuse complications. Although the incidence of severe dyspnea and cardiac arrest following methamphetamine overuse is unknown, the authors describe a patient with acute severe dyspnea with a history of crystal amphetamine misuse via smoking. The patient was refractory to the initial treatment and developed cardiac complications, including ventricular fibrillation, cardiac arrest, hemodynamic instability, and loss of consciousness.

## Case report

A man aged 42 years was introduced to the Emergency Department with the chief complaint of acute dyspnea following ice inhalation without history of dyspnea. His vital symptoms at the admission time were as follows: blood pressure = 140/85 mm Hg, pulse rate = 110 beats/minute, respiratory rate = 34 breaths/minute and temperature = 37.3 °C. His blood sugar was in normal range (130 mg/dl). Within the first hour and a half after admission, the patient was treated by nasal oxygen (3-5 L/min) and bronchodilator aminophylline (70 mg stat, then 70 mg slow infusion in half saline, per

hour until 12 hours). However, the patient did not respond to the initial treatment for severe dyspnea. His electrocardiogram showed a flattening of the T wave (Figure 1). Then, the patient lost his consciousness and developed ventricular fibrillation, cardiac arrest, and hemodynamic instability. Cardiopulmonary resuscitation (CPR) was immediately initiated for him. The patient was intubated, mechanically ventilated. Besides, the synchronized electrical shock was delivered 5 times (200-360 J) along with amiodarone (300 mg intravenously [IV] stat, then 1 mg/min IV infusion for 6 hours and next 0.5 mg/min for 18 hours) to treat the patient's ventricular fibrillation. His arrhythmia was subsequently controlled, and normal sinus rhythm resumed. The patient's initial laboratory tests showed low potassium level of 2.6 mEq/L (normal range: 3.5-5.5 mEq/L). So, he was immediately infused by an intravenous potassium supplement (10 mEq in 100 mL half Saline for 30 minutes stat and then 40 mEq in 1 liter half Saline per 12 hours, IV). Two hours after intubation and CPR, the patient's condition enhanced, and he was extubated. The laboratory results from the following day showed low blood magnesium (0.8 mg/L) (normal range: 1.2-2.5 mg/L), and thus, the patient received an intravenous magnesium supplement (2 g/100 mL half Saline for 30 minutes and then 2 g slow infusion per 12 hours). The patient's serum potassium and magnesium level returned to normal levels after the treatment. After two days, when the patient got stable, the echocardiography was performed that the results were completely normal (without any structural and functional abnormality, while the ejection fraction was 60%).

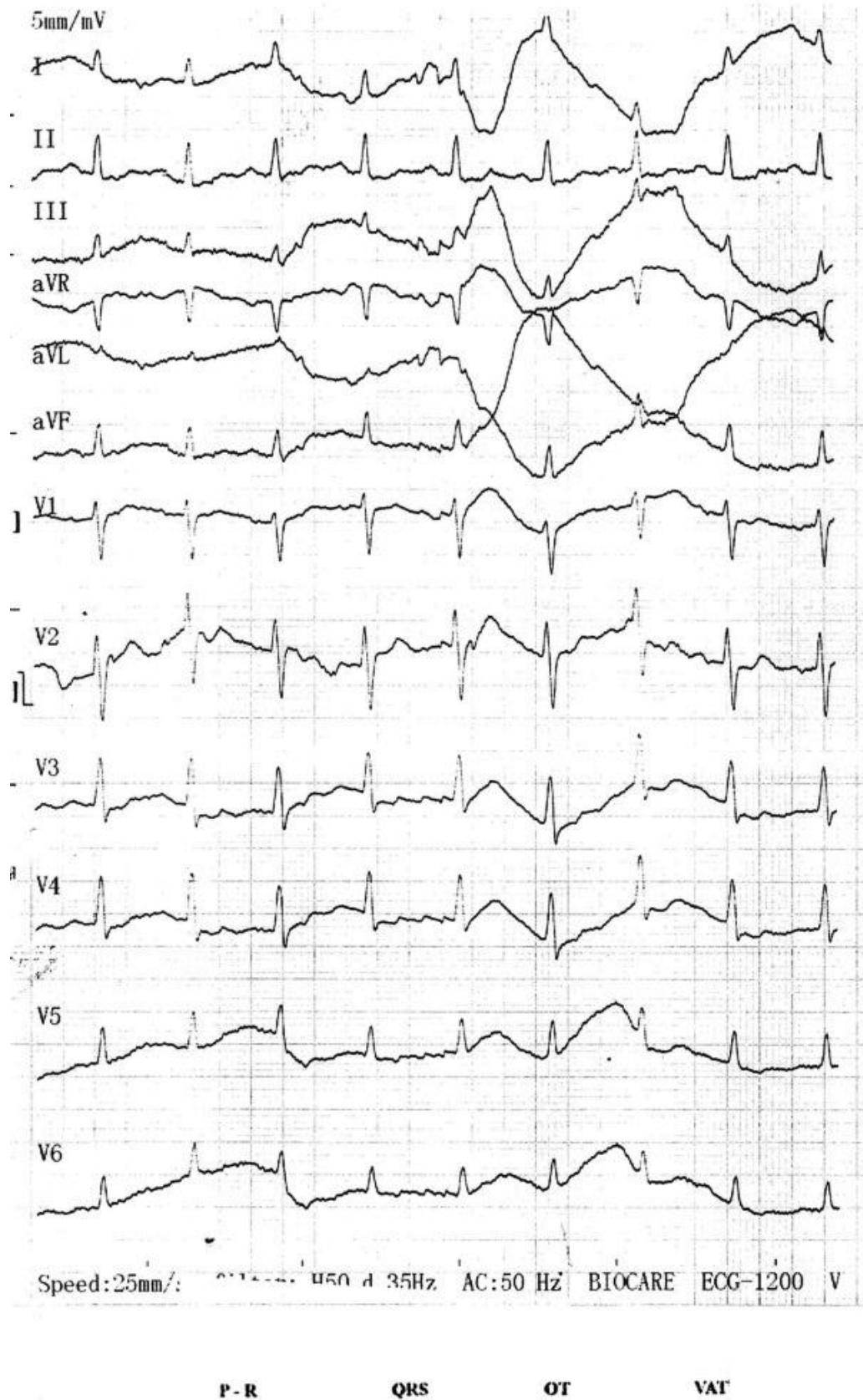
The patient had a 20-year history of oral opium abuse. During a party, he inhaled ice for the first time, resulting in severe dyspnea. Results of his initial arterial blood gas (ABG) were as follows: PaCO<sub>2</sub> = 44 mm Hg, pH = 7.20, HCO<sub>3</sub><sup>-</sup> = 8.6 mEq/L (normal range: pH = 7.35-7.45; HCO<sub>3</sub><sup>-</sup> = 24 mEq/L; PaCO<sub>2</sub> = 40 mm Hg). ABG test results indicated mixed metabolic and respiratory acidosis, which may have caused his hypotension and initial respiratory failure due to respiratory exhaustion. On the second day, his metabolic acidosis was corrected, the patient became conscious (Glasgow coma scale = 15), and oral feeding was initiated. His screening tests for checking toxicity in blood and urine yielded negative results. On day four, the condition of patient was completely normal; he could get out of bed, and all laboratory tests were normal. Echocardiography showed an ejection fraction of 60% without any abnormality. On day 6, the patient was released from the hospital. A week after his discharge, the patient referred to the clinic for follow-up. He had no clinical signs or symptoms. There were no abnormalities in thyroid and parathyroid function. His blood calcium, magnesium, and phosphate levels were all within the normal range.

## Discussion

Methamphetamine is a synthetic amine stimulant. It is very addictive and currently is the most common illicit amine medicine utilized in the United States (1). During the past two decades, consumption of it has increased particularly among teenagers (2, 3). The most recent report from the Iran Drug Control Headquarters indicates that only 3.6 percentages of drug users in Iran use Meth (3). However, in last years, the local manufacture of Meth has increased, and its cost has reduced, resulting in increased consumption of the drug. Nonofficial reports indicate that it is recently the second or third most illegal substance which is widely utilized in Iran (4). The manufacture of Meth is relatively easy, allowing its inexpensive production and wide availability. Its long duration of action and low price have made it a very popular medicine for utilize (4). On the other hand, its stimulant impacts and high addictive potential have made it a serious health problem (5-6). Meth influences multiple organs, such as the cardiovascular system (7).

One study reported that 40 percentages of young patients with cardiomyopathy are Meth addicts (11). Another research reported that at least 5 percentages of all patients admitted to the emergency departments with congestive heart failure are Meth addicts (12). Previous clinical series and case reports indicate that Meth misuse can potentially cause functional and structural changes in myocytes, resulting in clinical manifestations of congestive heart failure and cardiomyopathy (13). Hong *et al* reported a woman aged 31 years hospitalized for smoking high amounts of crystal methamphetamine. She was admitted with severe myocardial infarction (MI) because of diffuse arterial vasospasm. She subsequently died of cardiogenic shock (1). Sadeghi *et al* reported a man aged 28 years and two women aged 29 and 31 years with the histories of Meth consumption who were presented to the Emergency Department with acute dyspnea at rest. Each of patients had sinus tachycardia with tachypnea and an abnormal echocardiogram showing acute systolic dysfunction in accordance with heart failure. Additional assessment in the hospital showed that cardiomyopathy had no other etiology other than the use of Meth (8).

Obvious cardiovascular impacts consist increased contraction, vasoconstriction, and heart rate. Both effects significantly increase blood pressure. The most likely mechanisms for Meth cardiotoxicity are relate to the strong peripheral and central sympathomimetic impacts of Methamphetamine (9). Increased in circulating catecholamine levels (caused by this medicine) lead to direct myocardial toxicity, persistent tachycardia, coronary vasospasm, and or hypertension (10-12). Methamphetamine may increase levels of synaptic catecholamine by preventing monoamine



**Figure 1** Electrocardiogram (ECG) on admission of a man aged 42 years with methamphetamine-induced flattening of T waves. ECG: Sinus rhythm, first degree AV block (prolonged PR interval), normal axis, flattened T waves

oxidase, which is the enzyme responsible for the oxidation of serotonin and norepinephrine (12).

Amphetamine can cause hypokalemia by sympathomimetic stimulants. In cases of high clinical doubt for a disorder, a urine and or serum drug screen for amphetamines, diuretics, and other sympathomimetic stimulants should be performed. Evaluation of TSH levels in cases of tachycardia or clinical suspicion of hypokalemic periodic paralysis is required (13).

In conclusion, the main aim of presenting this case was to highlight the high risk of death in these people due to cardiac complications, related arrhythmia, and the possibility of serious electrolyte imbalance. All these situations require critical, important, and prompt treatment.

### Declarations of interest

The authors have no declarations of interest.

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