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“Bath salts” induced severe reversible cardiomyopathy

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Data Interpretation D
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Patient: Male, 27
Final Diagnosis: Bath salt induced cardiomyopathy
Symptoms: Agitation • fever • pedal edema
Medication: Intravenous nor-epinephrine for less than 6 hours
Clinical Procedure: —
Specialty: Internal medicine • cardiology

Objective: Unusual clinical course





Background: “Bath salts” is the street name for a group of recently identified and increasingly abused stimulant synthetic cathinones that are associated with multiple systemic effects. We present a case of a patient who developed reversible dilated cardiomyopathy secondary to their use.

Case Report: A 27 year old male with no past medical history was brought to emergency department with agitation. He had been inhaling and intravenously injecting “bath salts”, containing a mephedrone/ Methylendioxypropylvalerone (MDPV) combination. On presentation, he was tachycardic, hypotensive and febrile. His initial labs showed an elevated white count, creatinine and creatinine phosphokinase levels. His erythrocyte sedimentation rate; C-reactive protein; urinalysis; urine drug screen; Human Immunodeficiency Virus, hepatitis, coxsackie, and influenza serology were normal. EKG showed sinus tachycardia. An echocardiogram was done which showed dilated cardiomyopathy with an ejection fraction (EF) of 15–20% and global hypokinesia. A left heart catheterization was done and was negative for coronary artery disease. At a 20 week follow up, he had stopped abusing bath salts and was asymptomatic. A repeat echocardiogram showed an EF of 52%.

Conclusions: Bath salts (MDPV, mephedrone) are synthetic cathinones with amphetamine/cocaine like properties with potential cardiotoxic effects. Cardiovascular manifestations reported include tachycardia, hypertension, myocardial infarction, arrhythmias and cardiac arrest. “Bath salts” can also cause severe reversible dilated cardiomyopathy. Prior to diagnosis, other causes of cardiomyopathy including ischemic, infectious, familial, immunological, metabolic and cytotoxic may need to be ruled out; as was done in our patient.

Key words: dilated cardiomyopathy • 3,4-methylenedioxypropylvalerone • mephedrone

Full-text PDF: <http://www.amjcaserep.com/download/index/idArt/889381>

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Background

“Bath salt” is the street name for a group of synthetic cathinones that are emerging drugs of abuse. Two major pharmacological constituents of these drugs are MDPV (Methylenedioxypropylvalerone) and mephedrone [1]. They are becoming increasingly common and are a rising cause of emergency room visits, fatalities and affect multiple organ systems. The use of “Bath salts” has not been previously associated with the development of cardiomyopathy.

Case Report

A 27 year old male with no past medical history was brought to the emergency department with complaints of severe agitation. In spite of being very agitated and violent, he was oriented to time, place and person and was able to provide a good history. He was not complaining of chest pain, dyspnea, orthopnea, recent febrile illnesses or syncope. His review of systems was negative. He had been inhaling and intravenously injecting a combination of MDPV/mephedrone a few hours before presentation. The patient had brought the substance abused with him, though the amount of substance used was not known. He was not on any medications, had an unremarkable family history and denied use of other drugs, alcohol or tobacco. On examination, he was tachycardic (117 bpm), mildly hypotensive (90/60 mmHg) and febrile (101.2F). He had trace pedal edema and track marks on the left antecubital area. Cardiovascular exam was normal with no jugular venous distention, murmurs or added sounds. Lungs were clear. Laboratory findings included WBC – 14.6×10^3 /mcl; Hemoglobin – 11 g/dl; Platelets – 118×10^3 /mcl; Creatinine – 2.33 mg/dl; CPK – 723 U/l, myoglobin – 220 mg/dl and lactate – 2.8 mmol/l. Electrolytes were normal with an anion gap of 10. ABG showed mild acidosis with pH of 7.30. Troponins; erythrocyte sedimentation rate; C-reactive protein; urinalysis; urine drug screen, blood alcohol and cortisol levels were normal/negative. EKG showed sinus tachycardia. Chest Xray did not reveal any acute processes. His blood pressure continued to remain low on day 2 and a potential cardiogenic shock was suspected and an echocardiogram was done. It showed dilated cardiomyopathy with an ejection fraction (EF) of 15–20% and global hypokinesia (Figure 1). His blood pressure continued to drop requiring vasopressor support with low dose intravenous norepinephrine for duration of less than 6 hours. Mean Arterial Pressure (MAP) was consistently kept over 60 mm Hg at all times. Human Immunodeficiency Virus, Hepatitis, Coxsackie, and Influenza serology were normal. He improved clinically with conservative management and was discharged in a stable, asymptomatic condition after extensive counselling against bath salt use on day 5. He had an outpatient ischemia workup immediately after discharge that included a left heart catheterization that was negative for coronary artery disease. At a 20 week follow up, he was doing well, was

asymptomatic and had stopped abusing bath salts. An echocardiogram was repeated at this time and showed an EF of 52% with significant improvement in hypokinesia.

Discussion

“Bath salts” are synthetic cathinones and are structurally derived from amphetamines [1]. Their popularity primarily began in Europe beginning in 2007–08. By the end of the year 2011, it was recognized as an emerging public health threat by the national poison center’s data system [2]. In October 2011, legislation was passed classifying both mephedrone and MDPV as Schedule I drugs. However, in 2012, even after active legislation, approximately 2600 exposures were still documented by the AAPCC (American Association of Poison Control Centers) [3]. There is no mandatory reporting for “bath salt” exposures and this number may simply be the tip of the iceberg.

They are usually ingested or insufflated, though rectal, intravenous and intramuscular abuse has also been reported [4]. The regular urine drug screen is likely to be negative in the setting of “bath salt” use. Urinary analysis using liquid chromatography and mass spectrometry has been shown to be effective in the identification of “bath salts” in the urine [5] as has analysis with flow injection analysis and mass spectrometry [6]. However, these are all new and expensive techniques and have not been thoroughly validated. They can be used when a good history of definitive use is not available.

They are commonly reported to cause central nervous system effects including agitation, seizures, hyperthermia, paranoid psychosis [7] excited delirium [8], posterior reversible encephalopathy syndrome [9], serotonin syndrome [10] and dependence [11]. They are also associated with methemoglobinemia [12] and acute kidney injury [13] which can be direct or secondary to rhabdomyolysis [8]. Several studies have also found associations with death [14,15]

The most common cardiovascular effects however, are hypertension and tachycardia [2]. Animal studies have shown responses similar to methamphetamines with increased stroke volume, increased cardiac output without a direct pro-arrhythmic effect [16].

This patient presented with severe cardiomyopathy and cardiogenic shock. The causes for cardiomyopathy considered initially included ischemic, infectious, immunological, familial, metabolic versus cytotoxic.

The patient was very young, did not smoke and had no family history or other risk factors for cardiovascular disease. However, since occult disease has been reported to cause cardiomyopathy in a significant percentage of patients, ischemic causes

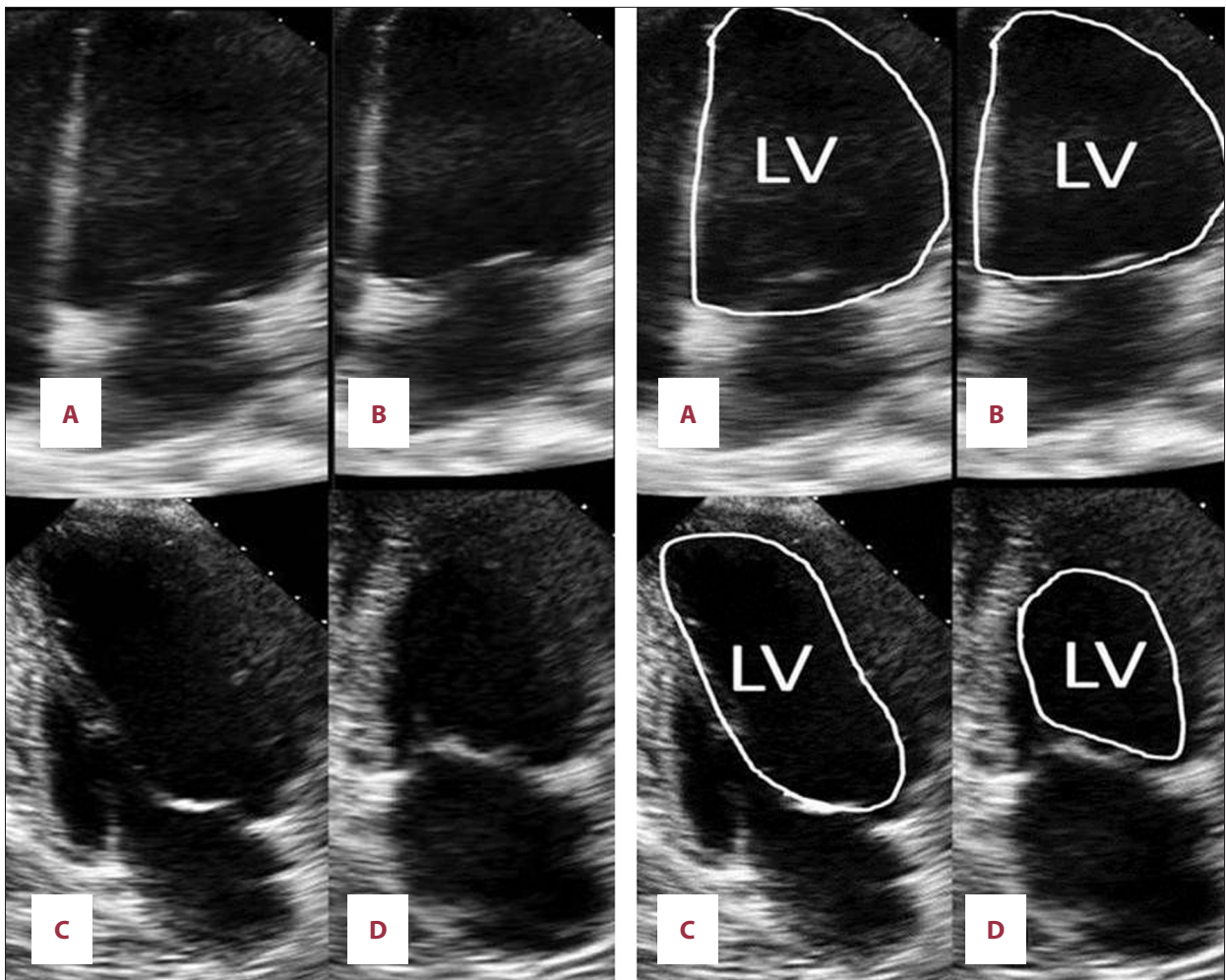


Figure 1. Serial echocardiographic findings: (A, B) diastolic and systolic images at presentation; (C, D) diastolic and systolic images at 20 week follow up. LV – left ventricle.

were definitively ruled out with a cardiac catheterization [17]. The catheterization revealed clean coronaries and a depressed ejection fraction of 20%.

His troponins remained negative and he had no electrocardiographic changes making myocarditis as well as stress induced cardiomyopathy an unlikely scenario. The pattern of hypokinesia was global, which is atypical for stress induced cardiomyopathy. Also, he did not meet the Mayo clinic criteria [18].

Reversal of cardiomyopathy at 20 weeks argues against a connective tissue related, hypertensive or infiltrative cause for his cardiomyopathy. He did not consume alcohol (blood alcohol levels were also negative at admission), did not smoke, did not use cocaine and had never been on chemotherapy. Established causes of toxic cardiomyopathy were therefore also deemed unlikely.

Patient's fever and tachycardia resolved after the first day and the tachycardia was not out of proportion to the fever. He

also did not have evidence of pericardial involvement, muscle aches or any other systemic manifestations that may have suggested an infectious cause for his cardiomyopathy. Human Immunodeficiency Virus, Hepatitis, Coxsackie, and Influenza serology were also negative. It is not possible to serologically confirm the lack of every infectious cause of cardiomyopathy, though there was no clinical necessity to run any further testing.

Serological or urine testing for "Bath Salt" and in this patient for mephedrone or MDPV was not undertaken as it was felt to be unnecessary given the good history. Also, the lack of easy availability, cost and the lack of thorough validation of these tests also played a role in the decision to not go ahead with the testing.

Heart failure and cardiogenic shock, as in the patient described above is an uncommon effect of bath salt abuse. The exact mechanism is unknown, though adrenergic excess may play a role. However, the pattern of presentation was not similar to stress induced cardiomyopathy (also known as takotsubo

cardiomyopathy) as pointed out earlier. Serial clinical assessment and echocardiograms can help monitor cardiac function. Abstinence from bath salts may be a good prognostic sign in this regard.

Conclusions

Even after legislation against their sale and use, "bath salts" continue to be rampantly abused. They affect multiple organ

systems and can present with severe cardiomyopathy and shock. This must be suspected when a patient presents with hypotension and a history of "bath salt" abuse. "Bath salts" are not detected by standard drug screens. Prior to diagnosis, other causes of cardiomyopathy including ischemic, infectious, familial, immunological, metabolic and cytotoxic may need to be ruled out; as was done in our patient. Clinical outcome was good with conservative management, though further studies may need to ascertain this.

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