

Street Drug, an Inotrope in Heart Failure?

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Abstract

Methamphetamine is one of the most commonly abused illegal stimulants and causes the release of dopamine, norepinephrine and serotonin. These neurotransmitters trigger vasospasm, causing persistent tachycardia, hypertension, and direct myocardial toxicity. Moderate usage of methamphetamine can increase cardiac output and myocardial contractility. However, larger doses can cause depression of the myocardium. We recently encountered a young man with chronic methamphetamine use who presented with decompensated heart failure after acute cessation of methamphetamine use and his condition improved with low dose inotropic support.

Keywords: Heart failure; Cardiomyopathy; Methamphetamine use; Dilated cardiomyopathy

Introduction

Amphetamines are highly addictive and potent stimulants that are widely abused in United States. Methamphetamine, one of the most commonly used recreational drugs, has a similar structure to ephedrine and other amphetamines. A National Survey on Drug Use and Health in 2013 reported an estimation of 24.6 million Americans were illicit drug users, which represented 9.4% of the populations above age 12. The percentage of methamphetamine users in 2013 was similar to 2012 and 2011 (0.2%), but higher than in 2010 (0.1%) [1]. In a study by Yeo et al, methamphetamine use was seen in 40% of young patients with cardiomyopathy and methamphetamine-associated cardiomyopathy was more severe than other non-ischemic cardiomyopathy [2]. We describe an interesting case of a young man with chronic methamphetamine use, who presented with acute shortness of breath and decompensation from methamphetamine withdrawal.

Case Report

A 27-year-old man, with a history of daily intravenous methamphetamine use, presented with increasing shortness of breath and chest pain for two weeks. The chest pain was atypical in that it was right-sided, sharp, intermittent and aggravated by leaning forward. He had three pillow orthopnea and cough with a small amount of white sputum. He denied any history of cardiovascular diseases. He lost his mother 13 years ago due to unknown reasons and had been using methamphetamine since. Two weeks prior to admission, the patient had hurt his left leg by hitting it against a television stand. He denied any fever or chills.

His examination revealed bi-basilar crackles and bilateral lower limb pitting edema up to the level of the shin. There was a 2 cm x 2 cm area of abrasion with a greenish base, over his left anterior shin with no surrounding erythema or discharge.

His electrocardiogram showed sinus rhythm with non-specific ST-T wave changes. His white blood cell count was 12.2 x 1000 cells /uL, brain-natriuretic peptide was 1483 pg/mL, troponin-I was 0.07 ng/mL and glomerular filtration rate > 60 cc/min at presentation.

The patient decompensated within 24 hours of admission with increasing dyspnea, tachycardia at 130 beats per minute and a fall in oxygen saturation to 70%. He was transferred to the ICU and intubated for respiratory distress. Transthoracic echocardiogram showed an ejection fraction of 7% with severe global hypokinesis and myocardial thinning, findings consistent with severe dilated cardiomyopathy. CT scan did not show any evidence of pulmonary embolism. However, it showed cardiomegaly with pulmonary edema, moderate-sized right pleural effusion, ascites, subcutaneous and mediastinal edema, consistent with congestive heart failure and volume overload. Doppler studies were negative for deep vein thrombosis.

To optimize his fluid status, right heart catheterization was performed which showed an elevated right atrial pressure of 23mmHg, a wedge pressure of 25mmHg and systemic vascular resistance (SVR) of 2800dynes/cm⁵. He was started on intravenous furosemide for aggressive diuresis and as well as sodium nitroprusside for afterload reduction to reduce the SVR. The patient's blood pressure subsequently dropped to 55/40mmHg and he became anuric. He was started on low dose dobutamine at 3 mcg/kg/min and sodium nitroprusside was discontinued. His blood pressure improved to 88/60mmHg. His urine output also improved to >80cc/hour. He had an extensive work up of his elevated WBC count. A repeat CT scan showed possible lower lobe pneumonia and he

was treated for hospital-acquired pneumonia with vancomycin for 14 days. Transesophageal echocardiogram did not show any valvular vegetation. He also developed a rash consistent with genitalia herpes and was treated with valacyclovir for 10 days. Over the course of 2 weeks, dobutamine was slowly titrated down and he was extubated. Carvedilol, oral furosemide, captopril and spironolactone were started. His repeat echocardiogram showed minimal improved EF to 13%. He was stable at the time of discharged, with a plan to follow-up in heart failure clinic.

Discussion

Methamphetamine (MA) is a potent stimulus and is one of the most widespread illegal stimulants abused in the United States [3]. It releases the neurotransmitters dopamine, norepinephrine and serotonin and activates the cardiovascular and central nervous systems, and is highly addictive [4]. It triggers vasospasm, causing persistent tachycardia, hypertension, and/ or direct myocardial toxicity [5,6]. Moderate doses of MA can increase cardiac output and myocardial contractility, but larger doses can cause depression of the myocardium. Chronic use of MA causes cardiomyopathy via cellular infiltration, myocardial hypertrophy, myocardium rupture and fibrosis. Its use also depresses the immune system leading to the suppression of mitogen-stimulated lymphocyte, a reduction in circulating lymphocytes and alternation of T-lymphocyte cytokine secretion as well as B cell pro-inflammatory cytokine secretion [7]. We believe that our patient had severe dilated cardiomyopathy from familial and/or chronic methamphetamine use. He may have been chronically dependent on the inotropic effects of methamphetamine. This is supported by the echocardiographic findings of long-standing, severe dilated cardiomyopathy. The patient was immunosuppressed from chronic MA use. A source of infection and a reduction in cardiac output from MA cessation has led to his cardiovascular collapse. His vitals improved and were stabilized with low dose dobutamine. Dobutamine is not very helpful in sepsis but has a significant role in the treatment of cardiogenic shock. Inotropic support was slowly weaned off throughout the hospital course, with concomitant treatment of his infection.

Conclusion

Chronic use of methamphetamine causes physiological dependence in patients. It is important to be aware of the effects of acute discontinuation of such medications and street drugs.

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