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CASE REPORT

Cardiology

Delayed cardiomyopathy and cardiogenic shock due to intravenous methamphetamine use requiring hemodynamic support with veno-arterial extracorporeal membrane oxygenation

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Abstract

Cardiovascular disease is the leading cause of mortality in chronic methamphetamine users. We present the case of a 29-year-old man, a prior heroin user, who presented following first-time use of intravenous methamphetamine, with delayed development of cardiomyopathy and severe cardiogenic shock, treated with veno-arterial extracorporeal membrane oxygenation (VA ECMO), and subsequent recovery. His initial chief complaint was shortness of breath, a common presentation to the emergency department. However, this case presentation is unique in three aspects: (1) a delayed presentation, (2) methamphetamine was administered intravenously as opposed to the common methods of being snorted or smoked, (3) and the effects were seen after first-time usage as compared to in a chronic user. This unique presentation can bring awareness to an uncommon etiology of shortness of breath due to intravenous methamphetamine usage.

1 | INTRODUCTION

Methamphetamine is a psychostimulant used as a recreational drug. Its use has been estimated at fifteen to sixteen million users worldwide, making it the most abused illicit drug after cannabis.[1] Methamphetamine is often smoked or snorted, and thus, less commonly injected intravenously. Chronic use is known to be a cause of heart failure, cardiomyopathy, arrhythmias, vasospasm, and myocardial infarction because of a surge of adrenergic stimulation. Among chronic users, cardiovascular disease is the leading cause of mortality. Moreover, chronic methamphetamine usage accounts for up to 6% of heart failure etiologies.[2] Although cardiac complications in the setting of chronic use are well documented, acute cardiomyopathy causing cardiogenic shock is a rare complication of initial methamphetamine use. There have only been two reported cases that we could find,[3,4] of veno-arterial extracorporeal membrane oxygenation (VA ECMO) being used in cardiogenic shock related to methamphetamine usage, the second report in a patient with chronic methamphetamine use.

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2 | CASE PRESENTATION

A 29-year-old Caucasian man with a prior history of heroin use and untreated hepatitis C presented with acute progressive shortness of breath after two episodes of intravenous methamphetamine use, ~3 and 4 hours before presentation. History was obtained solely from the patient who adamantly reported he had been drug-free for years and had never used methamphetamines in his lifetime. He had a temperature of 96.6°F, heart rate of 132 and regular, blood pressure 115/96 mm Hg, 27 respirations/min. Because of the work of breathing, he was put on bipap, saturating 100% on FIO_2 of 40%. On physical examination, he was in mild distress, spoke in full sentences, was alert and oriented to person, place, and time, increased work of breathing, tachycardic without any murmurs, no jugular venous distension, and no peripheral edema. Laboratory values were initially significant for a pH 7.072, pCO_2 61.4, pO_2 <21 (prior to bipap), WBC of 25.8 K/mcL with 85% neutrophils, sodium 137 mmol/L, potassium 2.7 mmol/L, chloride 96 mmol/L, CO_2 14 mmol/L, anion gap of 27, creatinine 1.43 mg/dL (baseline normal), ALT 129 units/L, AST 97 units/L, BNP 185 pg/mL, lactic acid 3.8 mmol/L, troponin 0.288 ng/mL, procalcitonin negative, ethanol negative, and D dimer 1440 ng/mL. Urine drug screen was positive for methamphetamine, fentanyl, and benzodiazepines. Electrocardiogram showed sinus tachycardia, normal axis, and no interval or ischemic changes. Chest X-ray showed diffuse bilateral patchy opacities with a perihilar predominance. A quick initial bedside transthoracic echocardiogram was performed by the emergency medicine attending for a global assessment of the cardiac function and was documented as grossly normal left ventricular function of 60%–65%.

He was transferred to the intermediate care unit for acute hypercarbic and hypoxic respiratory failure responsive to bipap (repeat arterial blood gas pH 7.335, pCO_2 39.9, and pO_2 299) and lactic acid anion gap metabolic acidosis, which was attributed to the polysubstance use. He was initially treated with 2 L of intravenous fluids and 1 mg of lorazepam as needed for tachycardia and agitation; however, he continued to develop progressive respiratory failure and shock. He required mechanical intubation for worsening respiratory failure and hemodynamic support with norepinephrine uptitrated to a mean arterial pressure >65 mm Hg. To further elucidate the etiology of shock, a complete bedside transthoracic echocardiogram was performed ~12 hours after initial presentation and revealed severe global hypokinesis with a left ventricle ejection fraction of barely 5% and a grossly normal-sized right ventricle with severely reduced function. Milrinone was initiated. Swan-Ganz catheterization measured a pulmonary artery pressure of 40/25 mm Hg and a pulmonary artery saturation of 39%. Cardiac output was not measured because of shift to cannulation for VA ECMO through his femoral vasculature. Lactic acid peaked at 10 mmol/L and troponin peaked at 9.994 ng/mL. Repeat electrocardiogram did not show any ischemic changes. Norepinephrine and milrinone were continued; he remained on cardiovascular support with VA ECMO and invasive mechanical ventilation with daily echocardiographic assessment of cardiac function (Table 1).

TABLE 1 Echocardiographic assessment of cardiac function while on VA ECMO

Day of ECMO	Ejection fraction	ECMO settings
0—Initiation	5%–10%	Flow at 4.6 L at 4500 rpm
1	5%–10%	Flow at 4.6 L at 4500 rpm
2	5%–10%	Flow at 4.6 L at 4500 rpm
3	15%–20%	Flow at 4.5L at 4500 rpm
4	20%–25%	Flow at 4.5L at 4500 rpm
5	15%–20%	Flow at 3.5L at 4400 rpm
6	15%–20%	Flow at 3.5L at 4000 rpm
7	15%–20%	Flow at 3.5L at 4000 rpm
8	45%–50%	Flow at 3.5L at 4000 rpm
9—Decannulation	55%–60%	Flow at 3.0 L at 3000 rpm

After the initial 3 days, he began to develop signs of cardiac recovery and inotropes were gradually weaned. His ICU course was complicated by North South or Harlequin syndrome, as a result of improved cardiac function and atelectasis. This was rapidly resolved by transfer of the pulse oximeter to the right hand along with bronchoscopy and ventilator titration for plugging and atelectasis. By day 9, his left ventricle ejection fraction recovered to 55%. ECMO was weaned under transesophageal echocardiography (TEE) visualization, and he was decannulated.

A send-out comprehensive toxicology panel from a blood specimen eventually resulted positive for methamphetamines, fentanyl, and benzodiazepines. Blood specimens to address other etiologies for his cardiomyopathy were drawn soon after initiation of VA ECMO, and included unremarkable culture data, human immunodeficiency virus, infectious (syphilis, gonorrhea, chlamydia, enterovirus, herpes simplex virus, Epstein-Barr virus), autoimmune (antinuclear antibody, glomerular basement antibody, pANCA, cANCA), and vasculitis panels. His hepatitis C viral load was 349,000. Coronary angiography was not performed.

As a complication of his refractory cardiogenic shock, he developed acute kidney injury, bland diffuse alveolar hemorrhage, coagulopathy, and acute liver injury, all of which resolved as his cardiac function improved. He was also treated for withdrawal syndrome from his polysubstance abuse, as well as *Staphylococcus aureus* ventilator-associated pneumonia as indicated by bronchoscopy cultures.

Repeat transthoracic echocardiogram, before discharge from his 3-week hospitalization, showed a small left ventricular cavity, normal left ventricular wall thickness, hypokinetic septal wall with normal wall motion of the remaining walls, left ventricle ejection fraction 50%–55%, trace mitral regurgitation, trace tricuspid regurgitation, and a right atrial pressure of 3 mm Hg. He was neurologically intact before discharge to an acute rehabilitation facility.

3 | DISCUSSION

Chronic use of methamphetamines is known to cause a multitude of pathologies with cardiovascular complications as the leading cause of

mortality. These cardiovascular complications include cardiomyopathy, hypertension, aortic dissection, arrhythmias, myocardial infarction, and strokes.[5] This is because of an increase in norepinephrine, serotonin, and mostly dopamine, through multiple mechanisms including redistribution of the above catecholamines into the cytosol from the synaptic vesicles, reverse plasma membrane transporters, blockage of monoamine transporters, decreased cell surface expression of transporters, inhibited activity of monoamine oxidase, and increased activity of tyrosine hydroxylase.[6,7] Chronic use leads to vasoconstriction, and thus, the previously listed cardiac complications. The resultant catecholamine surge is likely the etiology of acute systolic cardiomyopathy seen in this setting, similar to the catecholamine-related cardiomyopathy seen in patients with pheochromocytoma and Takotsubo cardiomyopathy.[8,9]

The half-life of intravenous methamphetamine is 11.4 hours with a mean residence time of 16 hours.[10] The time course between the second intravenous methamphetamine usage and hemodynamic instability in this patient was ~16 hours, and thus, it is possible that the catecholamines were masking his hemodynamics; however, it is difficult to elucidate why there was an acute change from the initial normal left ventricular function. When assessed by security, there was no objective evidence of any substances within his possession, and thus, unlikely that re-exposure resulted in the delayed progression of cardiomyopathy.

Other etiologies of cardiomyopathy were explored to include infection, autoimmune, and vasculitic. Ischemic workup was not pursued because of his improvement without intervention and negative electrocardiogram findings to suggest an ischemic event. Of note, the urine drug screen and serum toxicology screen were both positive for fentanyl, which unfortunately is becoming more commonly mixed into recreational drugs; however, it is difficult to assess the impact of fentanyl on this clinical presentation.

Although systolic cardiomyopathy and chronic congestive heart failure are well-recognized complications of chronic smoked or snorted methamphetamine use, acute severe cardiomyopathy resulting in cardiogenic shock is rare, and even more so in the setting of initial intravenous methamphetamine use. The existing literature describes only two cases of cardiogenic shock due to methamphetamine usage with successful recovery following cardiovascular support with VA ECMO.[3,4] One case described a 22-year-old woman who presented after intravenous methamphetamine use, in cardiogenic shock, which led to a cardiac arrest, successful resuscitation, and subsequently supported with VA ECMO for 82 hours, until recovery. A second case described a 31-year-old man, with longstanding and heavy methamphetamine use, who also required hemodynamic support with VA ECMO for cardiogenic shock with subsequent partial recovery of ejection fraction a month later.

This case report serves to illustrate that severe cardiomyopathy is potentially an uncommon complication of intravenous methamphetamine usage, as there was no evidence of another etiology in this patient. This is the only reported case to demonstrate a delayed presentation of cardiomyopathy with an unremarkable transthoracic echocardiogram at initial presentation to the emergency department. It is important for health care providers to be aware of this potentially fatal complication of substance abuse, because the management of intoxication would focus on hospital admission, follow-up echocardiograms, cardiovascular and hemodynamic support.

CONFLICT OF INTEREST

The authors have no conflicts of interest to declare.

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