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Case Presentation

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Methamphetamine-Associated Cardiomyopathy (MACM) with Left Ventricular (LV) Thrombus Identified by Computed Tomography (CT) Scan of the Chest

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Abstract

Stimulant abuse is a rapidly growing epidemic both in the United States and around the world. Reports show that nearly 4.7 million Americans have tried methamphetamines at least once. It is known that methamphetamines and related compounds significantly increase cardiovascular morbidity and mortality. Substance-related chronic inflammation and fibrosis can cause irreversible cardiac structural remodeling, leading to conditions like methamphetamine associated dilated cardiomyopathy (MACM) and myocardial infarction with non-obstructed coronary arteries (MINOCA). Left ventricular dilation results in hemodynamic flow impedance which promotes turbulent blood flow and stagnation. Furthermore, the sympathomimetic effects of methamphetamines also disrupt pathways in coagulation homeostasis, providing further incitement of thrombus formation. We report a case of left ventricular thrombus formation in the setting of methamphetamine-associated cardiomyopathy identified by computed tomography scan. Currently, there are no guideline-driven recommendations for methamphetamine user screening with 2D-TTE or for prophylactic anticoagulation for patients with MACM. Although this is the case, we would like to urge clinicians to become familiar with these potential complications and to screen selected patients with symptoms or who are at higher risk for complications. We would also like to emphasize the importance of obtaining a detailed social history and providing patient education on potential complications.

Keywords: Methamphetamine, MACM, Takotsubo, MINOCA, Catecholamine, Cardiomyopathy, Thrombus, Myocardial Infarction with Non-Obstructed Coronary Arteries

Introduction

Stimulant abuse is a rapidly growing epidemic both in the United States and around the world. Reports show that nearly 4.7 million Americans have tried methamphetamines at least once. It is known that methamphetamines and related compounds significantly increase cardiovascular morbidity and mortality [1,2]. Substance-related chronic inflammation and tissue fibrosis can cause irreversible cardiac structural remodeling, leading to conditions like methamphetamine-associated cardiomyopathy (MACM). Left ventricular dilation results in hemodynamic flow impedance, promoting turbulent blood flow and stagnation. Furthermore, the sympathomimetic effects of methamphetamines also disrupt pathways in coagulation homeostasis, providing even further incitement of thrombus formation. Isolated cases of MACM associated intracardiac thrombus formation have been reported in the medical literature [3]. Here we report a case of methamphetamine-associated cardiomyopathy with left ventricular thrombus identified by computed tomography.

Case Presentation

The patient is a 42-year-old male with past medical history of polysubstance abuse (opiates, amphetamines, marijuana, tobacco) who presented with intermittent, sharp, non-exertional chest pain radiating to the left shoulder. The patient was also experiencing shortness of breath and symmetric bilateral lower extremity edema extending to the abdomen. Symptoms began approximately five months prior to presentation and were progressive in nature. Review of systems was positive for decreasing urine output, orthopnea, dyspnea on exertion, and weight gain.

On examination, the patient had jugular vein distention, an audible S3, and diffuse lower extremity and abdominal pitting edema. Breath sounds were diminished and bibasilar crackles were noted. Pertinent laboratory studies include a pro-brain natriuretic

peptide (pro-BNP) of 17,000 mg/ml, lactic acid of 3.7 mmol/L and troponin-I of 0.034 ng/ml. A 12-lead electrocardiogram (ECG) showed sinus tachycardia with a rate of 128 beats/min. Urine drug screen was positive for amphetamines, opiates, and marijuana. Blood and urine cultures showed no growth at 120 hours. Chest radiograph (CXR) showed cardiomegaly with mild pulmonary congestion. A non-contrast computed tomography (CT) scan of the abdomen and pelvis showed intra-abdominal ascites with extensive soft tissue edema. A contrast CT scan of the chest revealed extensive bilateral pleural effusions and atelectasis. A 2.4 cm opacity was seen in the apex of the left ventricle suggesting the presence of a thrombus (Figure 1). 2-dimensional transthoracic echocardiography (2D-TTE) showed severely reduced left ventricular systolic function with diffuse hypokinesis and a calculated left ventricular ejection fraction (LVEF) of 15%. A fixed apical echogenic mass measuring 2.1 × 1.8 cm was identified. Regadenoson (Lexiscan) myocardial stress testing showed no evidence of ischemia or scarring. The patient underwent thoracentesis which yielded 1400 mL of dark yellow transudative fluid. Pleural fluid culture was negative.

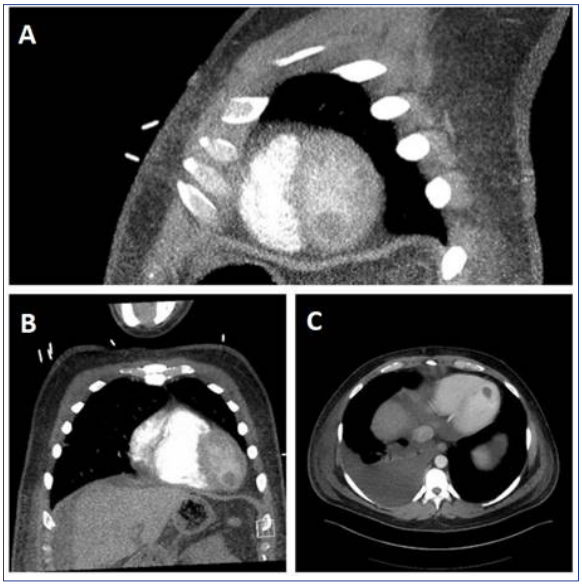

Figure 1: Contrast CT Scan of the Chest: A 2.4 cm opacity can be seen within the apex of the left ventricle (LV) consistent with thrombus formation. The LV thrombus can be seen in the sagittal (A), coronal (B) and axial (C) views

The patient was started on medical therapy for non-ischemic dilated cardiomyopathy with reduced left ventricular ejection fraction with carvedilol (6.25 mg twice daily), lisinopril (2.5 mg daily), furosemide (80 mg twice daily) and spironolactone (25 mg daily). Rivaroxaban (20 mg daily) was initiated for management of the left ventricular thrombus. Over the following days, the patient's condition improved with diuresis and he was discharged home with close outpatient follow up. Unfortunately, over the next 6-months, the patient had many admissions for hypertensive urgency and congestive heart failure exacerbation secondary to medication non-compliance. Repeat 2D-TTE at 6-months revealed an LVEF of 15% and persistence of the left ventricular thrombus, measuring 2.0 x 1.0 cm. About 12-months after initial presentation, the patient was again readmitted for acute hypoxic respiratory failure secondary to flash pulmonary edema and acute systolic congestive heart failure with cardiogenic shock. The patient was intubated, mechanically ventilated and invasive pressure monitoring with inotropic assisted diuresis was started. The patient was started on continuous renal replacement therapy (CRRT) due to insufficient diuresis. After lack of patient status improvement and discussion with family members, the patients code status was changed to comfort care. Later that evening, the patient developed profound hypotension and pulseless electrical activity. The patient passed with family at bedside.

Discussion

Considering that amphetamine containing products are listed by national reports as one of the most widely abused illicit substances, clinicians must be aware of the many complications associated with their use [1,2]. Pertinent to the case presented, MACM is not an uncommon cardiac complication within this patient population.

Methamphetamines play a significant causative role in many cardiac pathologies, including hypertension, tachyarrhythmias, coronary vasospasm, and cardiomyopathies. Abuse of centrally acting stimulants results in excess production of catecholamines, creating a hyperactive sympathetic response. This response leads to uncontrolled hypertension, tachycardia, and stress induced cardiomyopathy, also known as Takotsubo. This unstable catecholamine driven pathway is thought to create myocardial ischemia via direct free radical oxidation, mitochondrial injury, and metabolic derangements [4-7]. This is thought to be the cause of left ventricular systolic dysfunction with apical ballooning and hypokinesis [8-11].

Some cases have been reported of myocardial infarction without evidence of epicardial coronary stenosis, suggesting global coronary microvascular vasospasm and a diagnosis of myocardial infarction with non-obstructive coronary arteries (MINOCA). Some reports suggest that the myocardial structural changes can be reversible with cessation of methamphetamine use [8,9]. Unfortunately, with persistent or prolonged use, these changes become irreversible, leading to fibrosis and myocardial thinning. As mentioned prior, left ventricular dilation results in hemodynamic flow impedance, which promote both turbulent and stagnant blood, inciting thrombus formation.

Methamphetamine-dependent individuals have been shown to have lower dopamine receptor availability in the lower striatal and orbitofrontal brain regions, which have known associations to increased impulsivity [12]. This dopamine receptor depletion also seems to persist even with cessation of substance use, revealing the potential for permanent cognitive changes as well [12]. Given this information, it is imperative for clinicians to engage in early interventions for this select but growing patient population.

Conclusion

Here we have reviewed the potential cardiac complications of methamphetamine use, focusing on catecholamine-driven dilated cardiomyopathy and associated thrombus formation. Currently, there are no guideline-driven recommendations for methamphetamine user screening with 2D-TTE or for prophylactic anticoagulation for patients with MACM. Although this is the case, we would like to urge clinicians to become familiar with the potential cardiac complications seen with methamphetamine use and screen select at increased risk. We would also like to emphasize the importance of obtaining a detailed social history and providing patient education on potential complications.

Disclosures: None

Conflicts of Interest: None

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