CASE REPORT

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Amphetamine-type Stimulant-induced Cardiomyopathy with Reversible Left Ventricular Dysfunction: A Case Report

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Abstract

The global prevalence of methamphetamine and other amphetamine-type stimulants (ATS) continues to rise, contributing substantially to cardiovascular morbidity and mortality. ATS-associated cardiomyopathy (ATSAC) is an increasingly recognized but underdiagnosed cause of heart failure in young adults. This case report aims to describe the clinical presentation, management, and outcome of ATSAC and to highlight its potential reversibility with stimulant cessation and guideline-directed medical therapy (GDMT). A 24-year-old male with a long-standing history of polysubstance ATS abuse presented with progressive dyspnea and symptoms of acute decompensated heart failure. Comprehensive clinical evaluation, laboratory testing, echocardiography, and cardiac magnetic resonance imaging excluded alternative etiologies of cardiomyopathy. On admission, the patient demonstrated severe left ventricular systolic dysfunction with a left ventricular ejection fraction of 14%. GDMT for heart failure was initiated, alongside sustained cessation of stimulant use and multidisciplinary follow-up. Serial echocardiographic assessments over 12 months documented marked clinical and functional improvement. This case illustrates that ATSAC should be considered in young patients presenting with otherwise unexplained cardiomyopathy. Importantly, it demonstrates that significant—and potentially complete—recovery of cardiac function is achievable with early recognition, abstinence from stimulant use, and appropriate medical management. Further research is warranted to identify predictors of reversibility, clarify underlying mechanisms of myocardial injury, and develop standardized diagnostic and therapeutic strategies for ATSAC.

Keywords: Amphetamine, methamphetamine, cardiomyopathies, ventricular dysfunction, heart failure

INTRODUCTION

Amphetamines were first synthesized in the late 1920s, and by the late 1940s, they had achieved considerable medical and commercial success as antidepressants and weight-loss medications. In the late 1980s, crystal methamphetamine (METH) "ice" emerged, further contributing to widespread use. More recently, 3,4-methylenedioxymethamphetamine (MDMA), commonly known as "ecstasy", has gained popularity due to its ease of availability and relatively low cost. Although regulatory restrictions were introduced once the high addictive potential of these substances became evident, diversion to the illicit market has fueled a continuing global epidemic.

METH exerts profound effects on multiple organ systems, with the most clinically significant manifestations involving the central nervous and cardiovascular systems. In the brain, METH stimulates euphoria and heightened alertness through increased dopamine release in the nucleus accumbens, reinforcing addictive use behaviors.[1] However, chronic or high-dose exposure leads to reduced dopamine synthesis and receptor downregulation, resulting in deficits in memory,

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attention, and decision-making. Long-term use is strongly associated with psychiatric complications, including psychosis, depression, paranoia, delusions, violent behavior, and an alarmingly high lifetime suicide attempt rate, reported in up to 30% of users.[2]

Cardiovascular complications are equally concerning. The heightened catecholaminergic state induced by METH, combined with direct vasoconstriction, oxidative stress, and endothelial dysfunction, contributes to hypertension, vasospasm, and ischemia. METH use has been linked to hemorrhagic stroke, intracardiac thrombi in approximately one-third of patients with methamphetamine-associated cardiomyopathy (MACM), and a high incidence of atrial and ventricular arrhythmias.[3]

MDMA, a semi-synthetic entactogenic phenylethylamine, also produces a spectrum of adverse effects. Acute symptoms may include appetite loss, trismus and bruxism, nausea, muscle aches, fatigue, excessive perspiration, and ataxia. More severe complications have been documented, including psychosis, hyperthermia, seizures, cardiac arrhythmias, rhabdomyolysis, disseminated intravascular coagulation, hyponatremia, hepatotoxicity, aplastic anemia, pneumomediastinum, cerebral hemorrhage, and multiorgan failure. Fatal outcomes most commonly result from malignant hyperthermia, heat stroke, or acute hepatic failure.[4]

Given the escalating use of METH and MDMA worldwide, alongside their well-documented neuropsychiatric and cardiovascular toxicities, there is an urgent need for continued research to better understand their mechanisms, complications, and public health implications.

We present this case to highlight the potential reversibility of amphetamine-type stimulant-associated cardiomyopathy (ATSAC) in the absence of myocardial fibrosis, as demonstrated by cardiac magnetic resonance (CMR) imaging. The patient achieved complete recovery of left ventricular function following stimulant cessation and guideline-directed medical therapy (GDMT), underscoring the importance of early recognition and abstinence in improving outcomes.

CASE REPORT

Patient History

A 24-year-old male student presented to the emergency department with a one-month history of progressive shortness of breath, which had worsened over the preceding week, causing severe dyspnea and orthopnea. Initially reluctant to disclose substance use, he later admitted to a six-year history of periodic consumption of amphetamine-type stimulants (ATS), including METH, MDMA, and amphetamine, sometimes combined with alcohol.

Five years earlier, while under the influence of stimulants, he sustained traumatic injuries in an accident with significant blood loss, requiring intensive care admission and multiple sessions of dialysis for acute kidney injury. Renal function subsequently recovered, but echocardiography at that time revealed a reduced left ventricular ejection fraction (LVEF) of approximately 40%. As he was asymptomatic, no further cardiological investigations were pursued. Following recovery, he abstained from stimulants for a period but relapsed due to deteriorating mental health and depression. He was not on any regular medication and had no significant family history of cardiomyopathy.

Examination on Admission

On presentation, the patient was alert and oriented to time, place, and person but appeared diaphoretic. Vital signs showed tachycardia (heart rate 120 bpm), blood pressure 150/90 mmHg, respiratory rate 27 breaths/min, SpO_2 88%, and temperature 36.6 °C.

Electrocardiography revealed sinus tachycardia with T-wave inversion in leads V4-V6, as well as evidence of ventricular hypertrophy (Figure 1).

Investigations

Laboratory findings revealed elevated NT-proBNP (2800 pg/mL), slightly increased platelet count (367×10^3/μL), and C-reactive protein (8.62 mg/L). High-sensitivity troponin I was marginally elevated at 0.016 ng/mL (normal <0.014 ng/mL) without dynamic change on repeat testing. The estimated glomerular filtration rate was 99 mL/min. Thyroid function tests were normal. Laboratory findings demonstrated markedly elevated NT-proBNP, mild troponin elevation without dynamic change, and increased inflammatory markers, while renal and thyroid function remained within normal limits.

Bedside transthoracic echocardiography showed a dilated left ventricle (left ventricular end-diastolic diameter 6.9 cm) with severely reduced LVEF of 21% and global hypokinesis (Table 1). Other chambers were also dilated: right-ventricular end-diastolic diameter 4.1 cm, LA 4.8 cm, RA 4.4 cm. Pulmonary artery systolic pressure was elevated (42 mmHg), and mild mitral regurgitation was present.

Lung ultrasonography revealed large bilateral pleural effusions, estimated at approximately 1500 mL on the right and 1000 mL on the left, consistent with significant volume overload and contributing to the patient's respiratory distress. Thoracentesis was performed sequentially, resulting in significant symptomatic relief.

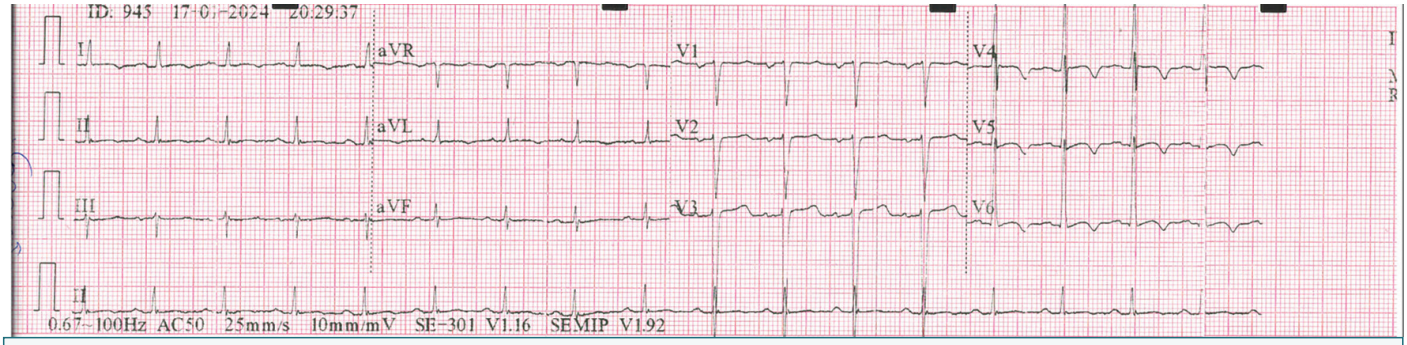

Figure 1. Electrocardiogram (ECG) on hospital day 3 ECG showing sinus tachycardia, T-wave inversions in leads V4-V6, and evidence of left ventricular hypertrophy

Table 1. Echocardiographic and cardiac MRI findings at presentation

Parameter	Value
LVEDD (cm)	6.9
LVEF (%)	21% (echo); 14% (CMR)
LV function	Global hypokinesis
RVEDD (cm)	4.1
LA diameter (cm)	4.8
RA diameter (cm)	4.4
PASP (mmHg)	42
Mitral regurgitation	Mild
LVEDV (mL, CMR)	313
Stroke volume (mL, CMR)	44
Cardiac output (L/min, CMR)	5.0
LVEDV/BSA (mL/m², CMR)	226
LGE-CMR	No fibrosis/necrosis

MRI: Magnetic resonance imaging, LVEDD: Left ventricular end-diastolic diameter, LVEF: Left ventricular ejection fraction, RVEDD: Right-ventricular end-diastolic diameter, PASP: Pulmonary artery systolic pressure, LGE: Late gadolinium enhancement, CMR: Cardiac magnetic resonance

Initial imaging revealed severe biventricular dilation and global systolic dysfunction with markedly reduced LVEF (21% by echocardiography, 14% by CMR). Importantly, late gadolinium enhancement [(LGE)-CMR] did not demonstrate fibrosis or necrosis, suggesting absence of irreversible myocardial damage. Echocardiographic and CMR imaging findings at presentation and during follow-up are summarized in Table 1.

A post-procedure chest radiograph showed vascular congestion, interstitial edema, Kerley B lines, cardiomegaly, and free costophrenic sinuses (Figure 2).

Post-procedural chest X-ray demonstrating cardiomegaly, pulmonary vascular congestion, interstitial edema, and Kerley B lines. Pleural effusions were resolved after drainage, with free costophrenic sinuses visible.

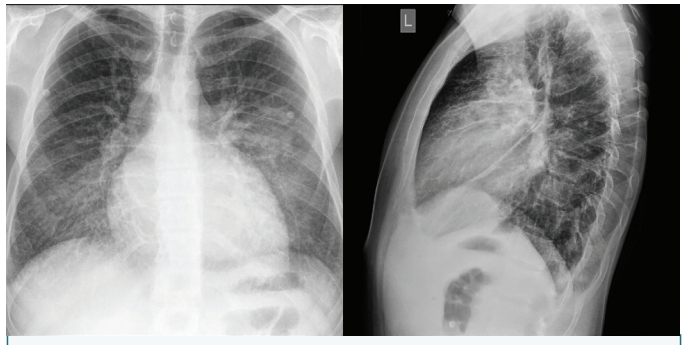

Figure 2. Chest radiograph following thoracentesis

Management and Hospital Course

The patient was initiated on inotropic support and intravenous diuretic therapy. Ivabradine was introduced for initial rate control given the patient's marked tachycardia and low ejection fraction (EF), in whom early initiation of beta-blockers was considered unsafe. Beta-blocker therapy (carvedilol) was subsequently introduced gradually once hemodynamic stability was achieved. Enoxaparin was administered to prevent intracardiac thrombus formation. GDMT for heart failure was then initiated, including sacubitril/valsartan, carvedilol, dapagliflozin, eplerenone, and torasemide. This approach aligns with guideline principles that prioritize stabilization in acute heart failure with reduced ejection fraction (HFrEF) and allow the use of adjunctive agents for rate control in sinus rhythm when beta-blockers cannot be promptly up-titrated.

Despite clinical improvement, repeat thoracentesis was required on day three, draining an additional 1000 mL of transudative fluid from the right pleural space.

Coronary angiography was deferred as per ESC/ACC/AHA guideline recommendations, given the patient's young age, absence of cardiovascular risk factors, typical history of stimulant abuse, and CMR imaging findings that did not suggest ischemic injury. CMR imaging was performed, showing severely

impaired systolic function [EF 14%, left ventricular end-diastolic diameter (LVEDV) 313 mL, stroke volume 44 mL, cardiac output 5.0 L/min, LVEDV/BSA 226 mL/m²]. Importantly, no evidence of intramyocardial fibrosis, necrosis, or active inflammation was detected on early or LGE (Figure 3).

CMR demonstrating severely reduced left ventricular systolic function (LVEF 14%), increased LV end-diastolic volume (LVEDV 313 mL), and no evidence of fibrosis, necrosis, or inflammation on LGE.

As the patient's condition stabilized, inotropic support was discontinued. Primary prevention of sudden cardiac death was discussed. Although implantable cardioverter-defibrillator (ICD) therapy was considered, the absence of sustained ventricular arrhythmia, sudden cardiac arrest, or prior ventricular tachycardia, together with the potentially reversible etiology, led to the decision to defer ICD implantation until after reassessment at three months of optimized medical therapy.

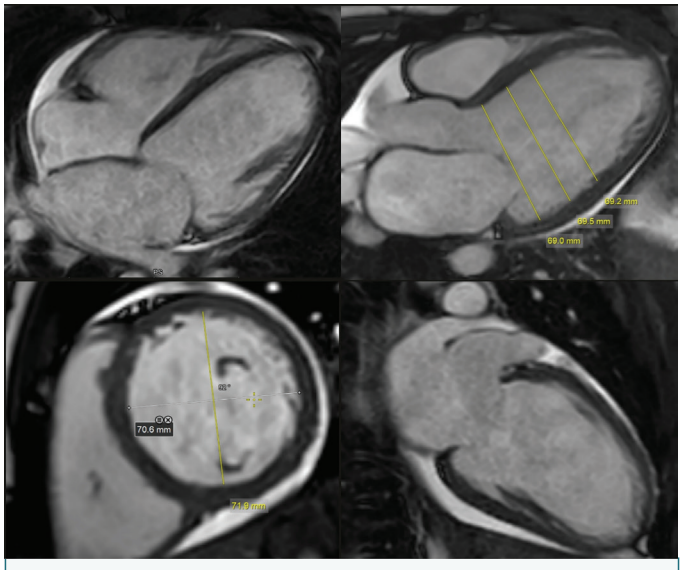

Figure 3. Cardiac magnetic resonance imaging

The patient was hospitalized for seven days. At discharge, he was prescribed sacubitril/valsartan, carvedilol, dapagliflozin, eplerenone, and torasemide. He was counseled regarding strict abstinence from stimulants and offered referral to a rehabilitation center, which he declined, though he committed to cessation of drug use.

Follow-up and Outcome

Serial echocardiographic findings demonstrating progressive recovery of cardiac function are summarized in Table 2. At two months, there was partial recovery of systolic function (LVEF 35%) and reduction in LV dimensions. At 12 months, LVEF normalized to 50%, chamber sizes were within reference ranges, and pulmonary pressures had resolved. He reported significant improvement in both physical and psychological well-being. He continued on GDMT, with the exception of loop diuretics, which were discontinued.

Abstinence from stimulant use during follow-up was assessed based on patient self-report and clinical evaluation, as routine toxicological screening was not performed.

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Research Ethics Committee of Caucasus University (approval no: CU-17-45/2025). Informed consent was obtained from the patient included in the study.

DISCUSSION

The global incidence of ATS use, including MACM or ATSAC, continues to rise. In 2022, approximately 0.6% of the world's population aged 15-64 reported ATS use in the preceding year.[5] Among these substances, METH is the most widely consumed stimulant, and its cardiovascular complications are the best documented. Reported manifestations include malignant hypertension, arrhythmias, aortic dissection, myocardial infarction secondary to vasospasm or coronary artery disease, stroke, pulmonary arterial hypertension, endothelial

Table 2. Echocardiographic recovery during follow-up

Parameter	Admission	2 months	12 months
LVEDD (cm)	6.9	5.8	~5.0
LVEF (%)	21%	35%	50%
RVEDD (cm)	4.1	Improved	Normalized
LA diameter (cm)	4.8	Reduced	Normalized
RA diameter (cm)	4.4	Reduced	Normalized
PASP (mmHg)	42	Improved	<30
Mitral regurgitation	Mild	Trivial	None

Echocardiography demonstrated progressive improvement in ventricular function and remodeling following stimulant cessation and GDMT. By 12 months, LVEF normalized to 50% and all chamber sizes were within reference ranges

LVEDD: Left ventricular end-diastolic diameter, LVEF: Left ventricular ejection fraction, RVEDD: Right-ventricular end-diastolic diameter, PASP: Pulmonary artery systolic pressure, GDMT: Guideline-directed medical therapy

dysfunction, and dilated cardiomyopathy (DCM). Recent large-scale analyses indicate METH users face a 32% higher overall risk of cardiovascular disease, including significantly elevated rates of heart failure and pulmonary hypertension. The mechanistic basis for these injuries spans catecholamine-mediated vasoconstriction, direct myocardial cell toxicity, oxidative stress, and inflammation, as well as structural and electrical remodeling of the heart.

Alternative etiologies were carefully considered. Viral myocarditis was deemed unlikely due to the absence of clinical features suggestive of acute infection, lack of dynamic troponin elevation, and CMR findings demonstrating no evidence of myocardial edema, inflammation, or LGE on CMR. Genetic cardiomyopathy was also considered less likely in the absence of a family history and given the marked reversibility of left ventricular dysfunction following stimulant cessation.

In recent years, the use of other stimulants such as MDMA, 4-fluoroamphetamine, Adderall, and α-PVP has also increased, largely due to low cost and wide availability. MDMA is particularly prevalent among young adults, with the highest reported incidence in the Netherlands, where 9.3% of individuals aged 15-34 reported use in 2023.[6] In Georgia, a 2021 survey revealed that over half of psychoactive substance users had consumed MDMA in the past year, and amphetamine use rose from negligible levels in 2016 to 10% in 2022.[7] These drugs are often used in social and party contexts, but also by students and young adults aiming to enhance academic or occupational performance.

Our case highlights the frequent pattern of polysubstance abuse, as many patients combine different ATS with alcohol. Another concern in the Georgian context is the purity of stimulants sold on the illicit market: more than half (57%) of MDMA products are either adulterated or entirely substituted with other substances.[8] This significantly increases the risk of unexpected toxicities, complicates diagnosis, and underscores the importance of public health interventions such as drug-checking programs, surveillance, and education campaigns. A widespread misconception persists that MDMA is safer than METH; however, accumulating evidence demonstrates its cardiotoxic potential, which is comparable to other amphetamines.

ATS-related cardiotoxicity is multifactorial, involving catecholamine excess, oxidative stress, reactive oxygen species-mediated injury, and inflammation, ultimately leading to myocardial fibrosis and DCM. Clinical outcomes depend strongly on whether patients achieve abstinence. Improvement in cardiac function following METH cessation was independently associated with the extent of myocardial fibrosis, whereas continued use was linked to progressive heart failure and poor prognosis.[9] Similarly, the degree of irreversible fibrosis is considered a major predictor of recovery. A smaller study suggested that patients presenting with a reverse Takotsubo pattern and less ventricular dilatation may achieve earlier recovery of function.[10]

Currently, no randomized trials have evaluated specific pharmacologic therapies for MACM. Until such data are available, GDMT for HFrEF remains the standard of care.[11] Endomyocardial biopsy is the gold standard for assessing myocardial fibrosis, but LGE-CMR has emerged as a valuable, non-invasive tool for detecting fibrosis and inflammation and may help predict left ventricular recovery.[12]

In the present case, our patient had several years of stimulant use with intermittent abstinence. Importantly, CMR did not reveal evidence of myocardial fibrosis. Following sustained ATS abstinence and initiation of GDMT, he demonstrated substantial recovery, with LVEF improving from 21% at baseline to 50% after 12 months. This favorable outcome is consistent with existing evidence that highlights abstinence and limited fibrosis as key determinants of reversibility in MACM.

This case has several limitations. First, objective toxicological confirmation of ATS use (e.g., urine drug screening) was not performed at presentation, and the diagnosis relied on patient-reported history in conjunction with clinical findings. Second, abstinence during follow-up was assessed based on patient self-report and clinical evaluation without routine toxicological monitoring. Third, although alternative etiologies were carefully considered and deemed unlikely based on clinical, laboratory, and imaging findings, definitive exclusion of all potential causes, including viral or genetic cardiomyopathies, cannot be fully established. Despite these limitations, the temporal association between stimulant cessation and marked recovery of cardiac function strongly supports a causal relationship.

CONCLUSION

ATSAC should be suspected in young patients with unexplained cardiomyopathy, as stimulant cessation and guideline-directed therapy can lead to meaningful recovery. This case underscores the importance of early recognition, integrated multidisciplinary care, and ongoing research to clarify the mechanisms of myocardial injury, identify predictors of reversibility, and define optimal management strategies that integrate cardiovascular care with substance use treatment.

Ethics

Informed Consent: This study was conducted in accordance with the Declaration of Helsinki and was approved by the Research Ethics Committee of Caucasus University (approval no: CU-17-45/2025). Informed consent was obtained from the patient included in the study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: T.K., V.S., M.L., N.G., T.V., Concept: T.K., V.S., M.L., N.G., T.V., Design: T.K., V.S., M.L., N.G., T.V., Data Collection or Processing: T.K., V.S., M.L., N.G., T.V., Analysis or Interpretation: T.K., V.S., M.L., N.G., T.V., Literature Search: T.K., V.S., M.L., N.G., T.V., Writing: T.K., V.S., M.L., N.G., T.V.

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