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Case Report

Ischemic cardiomyopathy in a 43-year-old male with stroke during admission: the role of chronic amphetamine-dextroamphetamine use

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Abstract: Cardiomyopathy associated with amphetamine-dextroamphetamine (Adderall) use is an emerging and under-recognized clinical concern, particularly in the context of chronic stimulant exposure. While most reported cases involve non-ischemic myocardial dysfunction, the potential for Adderall to accelerate atherosclerosis and contribute to ischemic cardiomyopathy remains unexplored. This case report aims to document the potential severity of Adderall-induced cardiomyopathy with concomitant coronary artery disease (CAD), examine the pathophysiological link between chronic stimulant exposure and accelerated atherosclerosis, and emphasize the need for vigilant cardiovascular monitoring in patients on long-term stimulant therapy. We report the case of a 43-year-old man with no known cardiovascular history who presented with progressive dyspnea and signs of heart failure. He disclosed a five-year history of high-dose Adderall use (45-65 mg daily) and tobacco consumption but had no prior history of hypertension, diabetes, or known CAD. Evaluation revealed a severely reduced left ventricular ejection fraction (10-15%), consistent with dilated cardiomyopathy. Coronary angiography unexpectedly revealed severe three-vessel CAD, necessitating urgent coronary artery bypass grafting (CABG). Postoperative recovery was uneventful, and the patient was initiated on guideline-directed heart failure therapy, with structured follow-up and strict recommendations for stimulant cessation and lifestyle modification. This case illustrates the multifactorial cardiotoxicity of chronic Adderall use, including direct myocardial injury, fibrotic remodeling, vasospasm, and accelerated coronary atherosclerosis. Unlike prior reports of reversible non-ischemic cardiomyopathy, this case required surgical revascularization, underscoring the irreversible nature of the damage in some patients. It uniquely highlights the synergistic contribution of stimulant-induced toxicity and underlying CAD to the development of severe cardiac dysfunction.

Keywords: Adderall-induced cardiomyopathy, coronary artery disease, stimulant cardiotoxicity, heart failure

Introduction

Cardiomyopathy is a progressive myocardial disorder that can lead to heart failure, arrhythmia, and increased morbidity. Traditionally associated with genetic, metabolic, and ischemic causes, stimulant-induced cardiomyopathy has gained recognition as a significant but underreported etiology [1].

Amphetamine-based medications, such as amphetamine-dextroamphetamine (Adderall), are prescribed for attention-deficit/hyperactivity disorder (ADHD) and narcolepsy. Adderall exerts its effects by increasing dopamine and norepinephrine levels and enhancing cognitive function and alertness [2]. However, its sympathomimetic properties may contribute to cardiotoxicity through mechanisms such as catecholamine toxicity, oxidative stress, myocardial fibrosis, and endothelial dysfunction [3]. Stimulants such as amphetamines negatively affect the body through catecholamine signaling, leading to tachycardia, hypertension, vasoconstriction, and vasospasm. Prolonged exposure to catecholamines induces cardiotoxic effects, including alterations in myocardial contractility and fibrosis. Structural changes may manifest

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as dilated cardiomyopathy with or without a reduction in ejection fraction or as hypertrophic cardiomyopathy [4]. Several reports have documented amphetamine-induced cardiomyopathy, presenting as severe heart failure and reduced ejection fraction, occasionally necessitating heart transplantation [5]. Additionally, acute Adderall overdose has been linked to Takotsubo cardiomyopathy, further underscoring its potential to induce significant cardiac complications [6].

With the increase in ADHD medication prescriptions and stimulant misuse, the cardiovascular implications of chronic Adderall use warrant greater clinical awareness. This case report underscores the potentially severe nature of Adderall-induced cardiomyopathy, particularly when compounded by underlying coronary pathology. Through this case, we aim to emphasize the need for vigilant cardiovascular risk assessment, early detection strategies, and long-term monitoring in patients receiving chronic stimulant therapy. Furthermore, we highlight the interplay between stimulant-induced cardiotoxicity and pre-existing coronary artery disease, providing insights into the mechanisms contributing to severe cardiac dysfunction in such cases.

Case presentation

A 43-year-old male construction worker with no significant past medical history presented to the emergency department with progressively worsening dyspnea over the preceding two weeks. Initially, he experienced shortness of breath during physical exertion, but his symptoms rapidly progressed to dyspnea at rest. The patient also reported orthopnea, palpitations, and intermittent dizziness. Although he denied chest pain, lower extremity swelling, or recent infections, he mentioned a family history of heart failure, noting that his 70-year-old father had been diagnosed with the condition. Concerned about the similarity in symptoms, the patient had recently discontinued smoking and chronic Adderall use before seeking medical attention.

Upon further inquiry, the patient disclosed a five-year history of chronic Adderall abuse, consuming high doses ranging from 45 to 65 mg daily to feel better and focus on work. He also admitted to a history of tobacco use and occasional alcohol intake and denied recent illicit drug consumption. He had no known history of hypertension, diabetes, or cardiovascular disease and had not previously undergone cardiovascular evaluations.

Upon arrival, physical examination revealed mild tachycardia, although his blood pressure remained within normal limits. Laboratory investigations revealed a markedly elevated B-type natriuretic peptide (BNP) level of 3474 pg/mL, indicating significant cardiac strain. Troponin levels were within normal limits, suggesting the absence of an acute myocardial infarction. Electrocardiography (ECG) revealed normal sinus rhythm with left axis deviation and signs of possible left atrial enlargement. Chest radiography revealed cardiomegaly consistent with an enlarged heart, prompting further diagnostic evaluation.

Echocardiography revealed dilated cardiomyopathy (DCM) with a severely reduced left ventricular ejection fraction (LVEF) of 10-15%, consistent with heart failure with reduced ejection fraction (HFrEF). Given the severity of his presentation, clinicians performed coronary angiography to assess potential ischemic contributions. Surprisingly, the angiogram revealed severe three-vessel coronary artery disease (CAD). Extensive coronary involvement suggests a chronic, progressive atherosclerotic process, potentially exacerbated by long-term stimulant use.

The patient required inotropic support to stabilize his hemodynamics. A subsequent cardiac MRI was performed to evaluate myocardial viability and revealed significant areas of viable myocardium despite severe dysfunction. This finding supported the decision to perform coronary artery bypass grafting (CABG) to restore myocardial perfusion. The patient tolerated the surgery well and did not experience any intraoperative complications.

His postoperative course was uneventful, and he was initiated on guideline-directed medical therapy, including beta-blockers, angiotensin-converting enzyme (ACE) inhibitors, and aldosterone antagonists. He was also strongly advised to maintain stimulant abstinence, stop smoking, and adhere to lifestyle modifications to optimize long-term cardiac function. A structured follow-up plan was established with

scheduled echocardiographic monitoring to assess ventricular recovery and ongoing risk management.

This case illustrates the profound cardiovascular impact of chronic Adderall abuse, highlighting the complex interplay between stimulant use, accelerated atherosclerosis, and resultant cardiomyopathy.

Discussion

Cardiomyopathy associated with Adderall use is an emerging concern, particularly in individuals with prolonged stimulant exposure. Several reports have documented the cardiotoxic effects of stimulant medications, ranging from hypertension and arrhythmia to heart failure and irreversible myocardial damage [1, 3, 5]. Our case expands on the existing literature by illustrating the multifactorial interplay between chronic Adderall use, severe three-vessel CAD, and resultant end-stage heart failure requiring surgical intervention.

The pathophysiology of stimulant-induced cardiomyopathy is complex and involves several mechanisms [6-8]. Chronic stimulant use leads to persistent norepinephrine and dopamine release, resulting in increased sympathetic activation, hypertension, and a heightened myocardial oxygen demand [5, 6]. This prolonged catecholaminergic stimulation induces direct myocardial toxicity, similar to stress-induced cardiomyopathy, by increasing calcium overload, oxidative stress, and apoptosis within cardiac myocytes [7, 8]. Amphetamines have also been associated with coronary vasospasm, a transient but severe reduction in myocardial perfusion, leading to ischemic injury even in the absence of atherosclerosis [5-9]. This effect is particularly concerning given the prothrombotic state induced by stimulants, which may exacerbate underlying CAD, as seen in our patient.

Chronic amphetamine exposure has been linked to structural myocardial changes, including interstitial fibrosis and adverse ventricular remodeling [5, 9]. This phenomenon is mediated by sustained adrenergic signaling, which promotes fibroblast activation, mitochondrial dysfunction, and increased oxidative stress, leading to progressive cardiac dilation and systolic dysfunction [5, 7]. Amphetamine toxicity has also been associated with a variant of stress-induced cardiomyopathy that mimics inverted Takotsubo cardiomyopathy [9, 10]. In such cases, the cardiac apex remains functional while the basal segments become akinetic, resembling a catecholamine surge-induced myocardial stunning pattern. While Adderall-induced cardiomyopathy has been predominantly reported as non-ischemic, recent evidence suggests that stimulant use can also accelerate atherosclerotic plaque formation. Chronic stimulant users exhibit increased vascular inflammation, endothelial dysfunction, and arterial stiffness, factors that may hasten the progression of CAD [3, 7]. Our patient's severe three-vessel CAD, despite a lack of significant preexisting cardiovascular risk factors, underscores this relationship.

Adderall has the potential to induce severe three-vessel CAD through multiple interconnected mechanisms. It stimulates excessive catecholamine release, leading to chronic vasoconstriction and hypertension, which subsequently damages the endothelial lining of the coronary arteries. Endothelial damage facilitates atherosclerosis, whereas diminished nitric oxide availability compromises vascular health, permitting low-density lipoprotein (LDL) cholesterol infiltration and inflammatory cell adhesion, thereby accelerating the formation of plaques [11]. Furthermore, amphetamines elevate oxidative stress and pro-inflammatory cytokine levels, exacerbating the progression of atherosclerosis. Clinical evidence indicates that stimulant users develop more severe coronary disease, even in the absence of traditional risk factors, such as obesity. Prolonged exposure results in vascular remodeling, smooth muscle hypertrophy, and reduced arterial elasticity, impairing blood flow and increasing the risk of plaque rupture [12]. Additionally, chronic sympathetic overactivity induced by amphetamines causes direct cardiac toxicity through myocardial fibrosis and microvascular damage, further compromising the coronary perfusion. The combined effects of hemodynamic stress, endothelial injury, inflammation, and structural vascular changes create a high-risk environment for severe multivessel CAD in patients taking Adderall [11-13].

Our case aligns with previously published reports of stimulant-induced cardiomyopathy but

presents unique distinguishing features [8-10, 14]. In 2023, Lewars et al. [9] documented a 38-year-old woman with non-ischemic cardiomyopathy secondary to chronic Adderall use, where discontinuation of the stimulant resulted in partial left ventricular function recovery. In contrast, our patient had a severely reduced ejection fraction (10-15%) and required surgical intervention. In 2024, Al-Juhani et al. [14] described a 32-year-old male with HFrEF (<10%) from amphetamine use, who required an implantable cardioverter-defibrillator (ICD). The patient did not have underlying CAD, reinforcing the distinct interplay of stimulant-induced cardiotoxicity and preexisting coronary pathology in our report. However, the patient did not have concurrent CAD, making our case unique in demonstrating the combined impact of stimulant use and atherosclerosis progression. Alsidawi et al. [10] described a 19-year-old woman who developed inverted Takotsubo cardiomyopathy following an acute Adderall overdose. Our case highlights the chronic effects of long-term Adderall use on myocardial structure and function.

This case is scientifically significant and novel for the following reasons. While stimulant-induced cardiomyopathy has been well-documented, most cases have been non-ischemic. Our case uniquely presents a patient with severe three-vessel CAD, suggesting that chronic Adderall use may contribute not only to myocardial dysfunction but also to accelerated atherosclerosis, an underexplored aspect of stimulant-related cardiovascular disease. Many reported cases of Adderall-related heart failure demonstrate some degree of left ventricular recovery after stimulant cessation. However, in our patient, the left ventricular ejection fraction (LVEF) was critically reduced (10-15%), and recovery was not possible without surgical revascularization. This highlights the irreversibility of myocardial damage in certain cases of chronic stimulant use. Most previous case reports focus on a single pathophysiologic mechanism (e.g., catecholamine toxicity or fibrosis). Our case demonstrates a multifactorial process, including sympathomimetic toxicity, coronary vasospasm and ischemia, fibrotic remodeling leading to dilated cardiomyopathy, and atherosclerosis acceleration leading to CAD progression. This comprehensive pathophysiologic interplay makes our case one of the most thoroughly documented examples of severe stimulant-induced cardiomyopathy with concurrent ischemic pathology. Given the widespread prescription of Adderall for ADHD, our case emphasizes the importance of cardiovascular monitoring in patients on long-term stimulant therapy [1, 3], particularly those with preexisting risk factors for CAD. Routine echocardiographic and electrocardiographic (EKG) screening may help detect early signs of myocardial dysfunction before irreversible damage occurs.

For patients necessitating prolonged administration of stimulants such as Adderall, which may elevate the risk of cardiac damage, including cardiomyopathy and heart failure, the primary approach to prevent or mitigate cardiac injury involves meticulous monitoring and management. The most crucial intervention is the discontinuation of the stimulant upon the emergence of cardiomyopathy or heart failure symptoms, as cessation has been demonstrated to enhance cardiac function in certain patients [13]. Physicians should perform comprehensive cardiovascular assessments prior to and throughout treatment, incorporating EKGs, echocardiograms, and vigilance for signs of cardiac dysfunction, particularly in young adults who may be susceptible despite the absence of other cardiac risk factors. Management of cardiovascular risk factors, such as hypertension, and the prevention of stimulant misuse are also imperative. In instances where cardiomyopathy arises, conventional heart failure treatments (e.g., beta-blockers and ACE inhibitors) may be employed in conjunction with the cessation of stimulants. Regular follow-up and multidisciplinary care are vital for the early identification of cardiac alterations and prompt intervention to avert irreversible damage. Although the overall risk remains relatively low, awareness and proactive cardiac monitoring are essential to minimizing stimulant-related cardiac damage [9, 13, 14].

This case underscores the urgent need for heightened awareness of Adderall associated cardiac complications. Discontinuing Adderall remains the cornerstone of treatment, as seen in multiple case reports. However, stimulant cessation alone was insufficient in our case due to severe CAD. Close hemodynamic monitoring is essential for stimulant-related cardiomyopathy patients. Our case reinforces the

need for routine coronary artery assessment in chronic stimulant users, as they may have silent, yet significant, atherosclerosis progression. Surgical intervention such as CABG should be considered in patients with severe CAD and heart failure. Patients recovering from stimulant-induced cardiomyopathy require lifelong echocardiographic monitoring, as some may develop progressive heart failure despite stimulant cessation.

Conclusion

This case report highlights the severe cardiovascular consequences of chronic Adderall use, particularly in patients with underlying CAD. Unlike previously reported cases, this patient exhibited severe three-vessel CAD in addition to cardiomyopathy, demonstrating an accelerated ischemic process not commonly reported in the literature. Given the increasing prevalence of stimulant prescriptions, clinicians must recognize the potential for irreversible myocardial and CAD progression, advocating for routine cardiovascular screening in long-term Adderall users.

Disclosure of conflict of interest

None.

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