Case Report

Toxic Dilated Cardiomyopathy: Recognizing a Potentially Reversible Disease

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Introduction

The use of illicit drugs has increased in recent years[1]. Related to this increase, there is a growing need to recognise and properly treat the adverse effects associated with the consumption of these drugs.

These substances can induce several cardiovascular (CV) complications, being this acute or chronic[1]. Some of them are the ischemic and arrhythmic events, the development of dilated cardiomyopathy (DCM) and the left ventricular systolic dysfunction (LVSD).

There are several types of drugs with different pharmacological and pathophysiological properties, and synergism is described between them[1]. Among these, cocaine and heroin stand out.

The key to a successful intervention towards one of these adverse effects is the high index of suspicion and early intervention.

Case Report

We describe the case of a 46-year-old Portuguese woman, a heroin and cocaine addicted (intra-venous consumption, as well smoking) since the age of 23, a chronic bearer of hepatitis B and C virus, with a previous history of pulmonary tuberculosis (treated) and moderate drinking habits.

The patient was admitted to the Emergency Department (ED) with exertional dyspnea of four months' evolution and progressive worsening in the last month (dyspnea on mild exertion). She denied chest pain, fever, chills or other associated symptoms. She denied recent travelling outside Portugal. She was tachycardic (115 beats per minute) and hypertensive (168/90 mmHg). In cardiac auscultation, she showed a holosystolic murmur, grade II/VI, at the level of the mitral focus, with axillary irradiation. She also showed signs of pulmonary congestion, hepatomegaly and mild peripheral edemas.

The chest x-ray showed an increased cardiothoracic index, signs of vascular cephalization, with hypotransparency at the level of the lower thirds of both lung fields, compatible with pulmonary congestion. The electrocardiogram revealed sinus tachycardia and left bundle branch block (LBBB). Analytically, she had normochromic and normocytic anemia (hemoglobin of 11.9 g/dl), normal renal function and ionogram; overall elevation of liver enzymes (AST 287 U/L, ALT 207 U/L, Gamma-GT of 109 U/L, total bilirubin of 2.04 mg/dl, direct bilirubin of 0.4 mg/dL) as well as of BNP's levels (3600 pg/mL). She presented no significant elevation of myocardial necrosis or inflammatory biomarkers. Computed tomography angiography excluded signs of pulmonary thromboembolism and showed the presence of bilateral pleural effusion and alveolar consolidation.

For additional assessment, the patient underwent an echocardiography, which showed dilatation of the left cardiac chambers (**left** ventricular [LV] end diastolic volume of 125 ml/m^2; maximal atrial volume of 39 ml/m^2), with global and severe LVSD (ejection fraction [EF] of 11%), without asymmetries in segmental contractility, and two small apical thrombi were identified (Figure 1). Mild right ventricular dysfunction was also evident.

Given these findings, we conducted the diagnosis of decompensated heart failure (HF) in a patient with dilated cardiomyopathy and ventricular dysfunction, with probable toxic etiology.

She started pharmacological treatment of HF (loop diuretics, β blockers [BB] and angiotensin-converting enzyme [ACE] inhibitor), as well as anticoagulant, with favourable clinical evolution. She was discharged by the 5thday of hospitalization, and she was oriented to the outpatient Consultation in the hospital and to the Support Centre for Addicts. She remained in functional class I of NYHA since the first month after discharge. After six months of total abstinence from drugs, under optimized treatment for HF, she was completely asymptomatic, with BNP values below 10 pg/mL. The echocardiographic reassessment showed normal cardiac chambers size and recovery of the biventricular systolic function (LV end diastolic volume of 57 ml/m^2; maximal atrial volume of 21 ml/m^2; LV EF 62%), with no evidence of intracavitary thrombi. The oral hypocoagulant was suspended, keeping up the therapy with BB (carvedilol) and the ACE inhibitor (lisinopril).

Discussion

All the types of recreational drugs may induce important CV complications and they are responsible for high morbidity and mortality.

Following the cannabinoids, the psychostimulant drugs are the most widely consumed illicit substances. Acute intoxication by these drugs is a frequent cause of resort to

Keywords

Cardiomyopathy, Dilated; Cocaine; Heroin.

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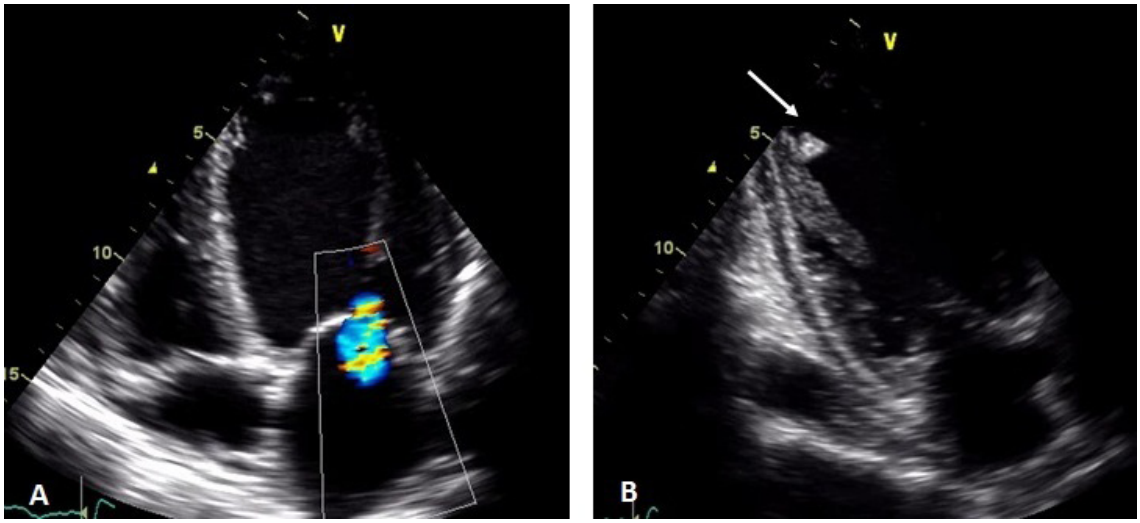

Figure 1 - *Transthoracic echocardiogram at admission showing the presence of dilated cardiomyopathy, with mitral regurgitation due to poor coaptation of the valve leaflets (A) and thrombus adhering to the apex (arrow) (B).*

the ED, particularly for chest pain, as well as an important cause of drug related death[2].

Psychostimulants and opioids have been related with LVSD. However, few clinical reports have illustrated the role of drug abstinence in the recovery of heart failure and myocardial dysfunction, which can be achieved in a short period of time[3,4]. The major cardiovascular manifestations, secondary to cocaine and heroin drug abuse, will be reviewed here.

The CV toxicity associated with psychostimulants is well described and it is an phenomenon independent from the consumption standard, dosage or administration route.

CV effects of cocaine abuse derive essentially from the activation of the sympathetic nervous system, contributing to the occurrence of arrhythmias and ischemic events[3,5].

The ischemic events are undoubtedly the more frequent CV complications in consumers of stimulants such as cocaine[2]. There is even a temporal relation between the consumption and the event. It was found that two thirds of acute myocardial infarctions (AMI) related to the effect of cocaine occur within the first three hours after its consumption[6].

Ischemia may result from the increased maximum oxygen consumption by the myocardium, as well as from phenomena of coronary vasospasm, probably in relation to the activation of α-adrenergic receptors in the coronary vessels[5]. In addition, cocaine promotes thrombogenesis, by atherosclerotic plaque formation and platelet activation and aggregation[3,7].

More rarely, the chronic use of cocaine is associated with the development of DCM and LVSD, the latter being potentially reversible with a consumption discontinuation[7]. The mechanisms which cause the systolic dysfunction include direct toxic effects of cocaine, the presence of sustained ischemia, the persistent hyperadrenergic state and inflammatory mechanisms including the alteration of cytokine production and induction of myocyte apoptosis[5].

Opioids are another type of recreational drugs. Heroin is the most widely consumed illicit opiate. They act by increasing parasympathetic activity and decreasing sympathetic activity, which can cause bradycardia and hypotension[1].

Like psychostimulants, this kind of drugs has also been associated with the occurrence of several types of arrhythmias, ischemic events and potentially reversible LVSD[1,3]. It is believed that the most likely mechanism of myocardial ischemia is also vasospasm.

Moreover, acute heroin intoxication can cause non-cardiogenic pulmonary edema due to the disruption of alveolar-capillary membrane integrity.

The indicated treatment for the vast majority of patients admitted under recreational drug use is the conventional treatment, considering its complications and supportive measures. However, the approach to chest pain in patients consuming cocaine is still somewhat controversial with relation to the use of BB, as it can exacerbate coronary vasospasm in a more serious phase of acute coronary syndrome (ACS), by inhibiting the vasodilatory effect inherent to the stimulation of α2adrenergic receptors[8]. Although there are not enough studies about the best therapeutic strategy in this clinical context, current recommendations suggest that nitrates and calcium channel blockers are the preferred drugs for the initial control of arterial hypertension, coronary vasoconstriction and tachycardia (verapamil). In case of insufficient response to this therapeutic strategy, it is reasonable to administer a β blocker with additional α-blocking effect (for example, labetalol)[9].

Conclusion

The consumption of recreational drugs can induce an extensive range of cardiovascular manifestations, causing many serious medical conditions which include arrhythmias, ischemic events and LVSD.

The case described significantly illustrates the importance of recognizing the toxic etiology, particularly for recreational drug abuse, associated with dilated cardiomyopathy, which when treated properly and with proof, is potentially reversible and may have a favourable prognosis.

Author contributions

Conception and design of the research: Rangel I; Acquisition of data: Rangel I, Amorim M, Gonçalves A; Analysis and interpretation of the data: Rangel I, Gonçalves A; Writing of the manuscript: Rangel I, Amorim M; Critical revision of the manuscript for intellectual content: Gonçalves A, Sousa C, Bettencourt P, Maciel MJ.

Potential Conflict of Interest

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Study Association

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