

Methamphetamine-induced cardiomyopathy causing severe mitral valve regurgitation

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A 29-year-old woman with a history of methamphetamine abuse presented with acute pulmonary oedema, palpitations, dyspnoea and cough in the context of sinus tachycardia. Past medical history was unremarkable.

On examination, a systolic murmur was elicited and trans-thoracic echocardiography demonstrated severe mitral valve regurgitation. In addition, moderate left ventricular dysfunction with significant dilatation was observed. Further assessment of the mitral valve showed marked restriction of the posterior leaflet, which, although not calcified, appeared almost rigid. In contrast, the anterior leaflet appeared entirely normal (Figure 1). An magnetic resonance imaging (MRI) cardiac scan confirmed a dilated left ventricle with increased trabeculation and reduced function. Furthermore, it revealed moderate rigidity of the mitral subvalvular apparatus beneath the posterior leaflet, which was thicker than normal. The mechanism of the regurgitation was postulated to be as a result of restricted posterior leaflet motion, with the previous methamphetamine abuse thought to be the cause of the left ventricle abnormality.

The patient underwent prompt surgical mitral valve repair. The myocardial thickening involved the top of the posterior papillary muscle leading to posterior leaflet movement restriction. The free body of this leaflet had otherwise a normal appearance. A resection of the fibrotic primary chordae was performed and 4 couples of CV-4 Gore-Tex artificial chordae (W.L. Gore & Associates, Flagstaff – AZ) were inserted into the posterior leaflet. A pericardial ring was inserted into the posterior tract of the mitral valve annulus. The intra-operative trans-oesophageal echocardiography confirmed excellent results of the repair with no evidence of residual regurgitation (Figure 2). Surgery and postoperative recovery were uneventful and the patient was discharged home on postoperative day 6. Histopathological analysis of a fragment of the papillary muscle showed focal fibrosis, which in association with the dilated left ventricle supported the hypothesis of a methamphetamine-induced cardiomyopathy.

Methamphetamine is a synthetic stimulant that can have a strong effect on the cardiovascular system. The most common physical symptoms after acute intoxication are chest pain, arrhythmias, palpitations and hypertension [1]. Although more rarely, acute coronary syndrome, myocardial infarction, aortic dissection and sudden cardiac death [2] have also been described.

The primary mechanism behind its cardiovascular effect is the action on the catecholamine in the peripheral nervous system that modulates heart rate and blood pressure [2]. The increased level of catecholamine

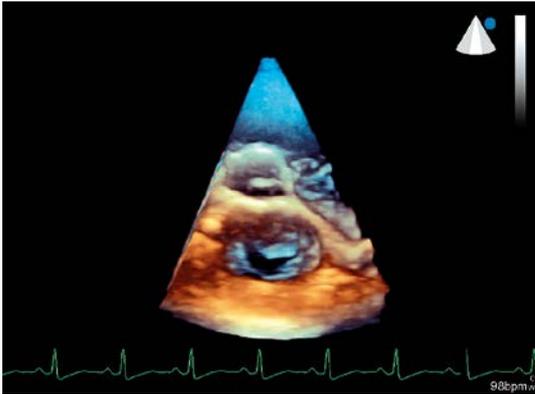


Figure 1. Pre-operative 3-dimensional transthoracic echocardiography showing a restricted posterior mitral valve leaflet and a normal anterior leaflet

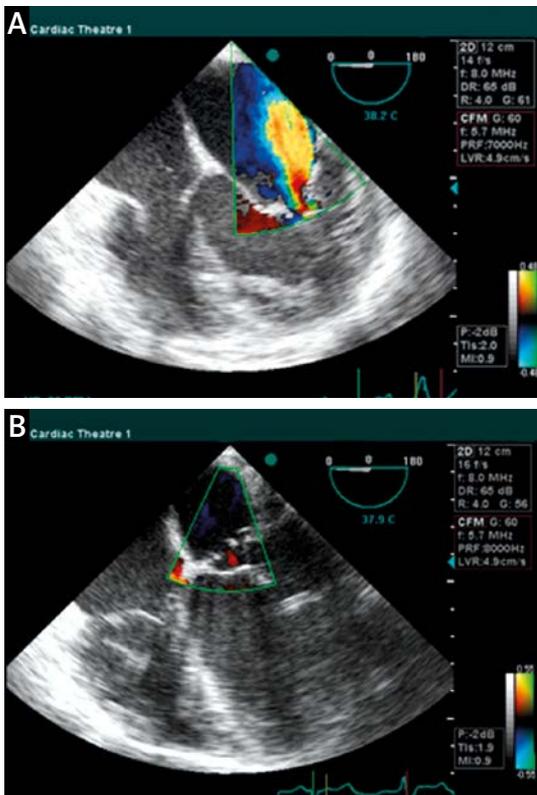


Figure 2. Intra-operative trans-oesophageal echocardiography demonstrating severe mitral valve regurgitation (A) and no residual regurgitation after the surgical mitral valve repair (B)

can be responsible for cardiotoxicity due to coronary vasoconstriction, calcium overload and production of oxygen free radicals [3]. The long-term effect on the heart appears to be related to higher oxygen consumption due to an increased workload as well as the oxygen-wasting effect [4]. Several histological myocardial alterations have been demonstrated in autopsy studies including contraction band necrosis, eosinophilic degeneration, atrophy, hypertrophy, disarray, oedema, cellular

infiltration, myolysis, granulation, vacuolization and fibrosis [4]. Most of these alterations could be reversed upon cessation, except for fibrosis, which can be irreversible in long-term methamphetamine use, with consequent deterioration of the cardiac tissue and function [4]. Hence, although less common than other illicit substances such as cocaine, an amphetamine-induced cardiomyopathy is possible [5]. In our case, the specific localized retraction of the posterior leaflet appeared mainly related to an abnormal left ventricle cavity that led to the deformity of the papillary muscle and mitral valve chordae. The fibrotic process was the cause of the restrictive motion of the posterior leaflet and the abnormal segmental wall motion; these features were severe enough to trigger the acute presentation. Our patient remains well 6 months after surgery with no residual mitral regurgitation on follow-up echocardiography. However, doubts remain regarding the risk of progressive degeneration of the left ventricular wall fibrosis and its consequent long-term prognosis.

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