Case report

We report a 39-year-old Caucasian man who was diagnosed with constrictive pericarditis in the setting of modest recreational methamphetamine use by smoking for over ten years. He did not have a significant medical history or taking prescribed medications. Symptomatic with pleuritic chest pain six months prior when he was diagnosed with acute pericarditis, yet transthoracic echocardiogram was remarkable at the time. Over the next six months, he developed progressive dyspnoea on exertion, abdominal distention and extensive lower limbs pitting oedema. Initially, his diagnosis was delayed due to the deceptive symptoms and history of drug use, and the investigations were directed towards a primary liver pathology due to the deceptive history of drug use and clinical symptoms of right heart failure.

The diagnosis of constrictive pericarditis was firstly suggested by cardiac imaging studies which revealed a marked pericardial thickening, pericardial tethering, minimal pericardial effusion and a pronounced diastolic interventricular septal bounce (Figure-1). Invasive hemodynamic measurements by the left and right heart catheterisation established the diagnosis of constrictive pericarditis by demonstrating a biventricular elevation and equalisation of diastolic pressure (16 mmHg). Pathology studies include Acid Fast Bacilli (AFB), dense hyalinized fibrosis consistent with constrictive pericarditis. Eventually, the patient underwent a successful pericardiectomy and obtained histologic specimen confirmed thickening of the pericardium by dense hyalinized fibrosis consistent with constrictive pericarditis.

Discussion

To our knowledge, this is the first report of constrictive pericarditis in the setting of recreational MA abuse. Due to the paucity of knowledge in this field, the authors reviewed and collected existing data to identify the possible pathophysiological aetiology of this finding. Several studies demonstrated a compelling association between MA use and increased 5HT (a clinically effective precursor of serotonin) release in the striatum [6]. On the other hand, the role of elevated 5HT in pericardial disease and constrictive pericarditis has been explained in the literature [7]. In this case, we postulated that the long term MA abuse could result in elevated serotonin and further development of chronic pericardial disease and constrictive pericarditis. Quantifying serotonin level was not feasible, due to the absence of a consensus method to measure circulating serotonin and the difficulties in measuring this hormone [8]. The other known causes for constrictive pericarditis have already been excluded in this case.

Conclusion

This case highlights an extremely rare but debilitating cardiac side effect of recreational MA use. Although pericardial involvement as a side effect of MA use is extremely rare and unusual, awareness of all the potential cardiac complications is warranted to commence an early and appropriate treatment.

Abstract

Recent public health reports suggesting a global rise in methamphetamine use among youth. Current data show the most common forms of chronic cardiovascular disease associated with methamphetamine use are cardiomyopathy and coronary artery disease. We report a 39-year-old Caucasian man who was diagnosed with constrictive pericarditis in the setting of modest recreational methamphetamine use by smoking for over ten years. Primarily, his diagnosis was delayed, and the investigations were directed towards a primary liver pathology due to the deceptive history of drug use and clinical symptoms of right heart failure.

Methamphetamine use and constrictive pericarditis; a rare case of heart in a cage

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Introduction

Emerging evidence reveal methamphetamine (MA) use has substantially grown worldwide and in Australia over the last few years [1]. The association between long term MA abuse and a wide range of cardiovascular disease has been well described [2]. Current data show the most common forms of chronic cardiovascular disease associated with MA use are cardiomyopathy followed by severe coronary artery disease [3]. The pathophysiological mechanism of MA abuse is due to release of endogenous catecholamines (dopamine and noradrenaline), subsequent stimulation and over activation of both alpha and beta-adrenergic receptors. These result in developing hypertension, tachycardia, vasospasm, vasoconstriction and further risk of acute coronary syndrome [3,4]. Although several cardiopulmonary complications of MA have been formerly reported [5], the chronic pericardial disease is extremely rare, and it is not well known in this context.
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