



Dual Mechanism of Methamphetamine-Induced Cardiomyopathy on Magnetic Resonance Imaging

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Highlights

Methamphetamine is associated with a number of adverse cardiovascular effects and diseases. We present a case of a 50 year old male presenting with heart failure and angina and long-standing methamphetamine use. With the aid of cardiac magnetic resonance imaging and also coronary angiography, we speculated the dual pathophysiology of methamphetamine-induced cardiomyopathy in this patient.

Keywords: cardiomyopathy; cardiac magnetic resonance imaging

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Case

Mr S is a 50 year old Pakeha male presenting to hospital with 1 month of shortness of breath, orthopnoea and paroxysmal nocturnal dyspnoea, and on the day of admission developed central chest tightness radiating down both arms. He has no past medical history, but was a regular methamphetamine user up until 1 year ago. Examination was significant for sinus tachycardia and oxygen saturations 94% on air, but no heart murmurs or signs of heart failure. Electrocardiogram showed left bundle branch block of unknown duration, chest X-ray showed cardiomegaly, and troponin I was 7700ng/L rising to 11400ng/L 6 hours later. Antithrombotic therapy and heart failure medications were commenced on admission.

Inpatient coronary angiogram was performed the following day, and this revealed coronary dissection of the second obtuse marginal lesion (figure 1), but otherwise normal coronaries. Transthoracic echocardiography was undertaken later the same day, showing severe left and right ventricular systolic impairment but normal left ventricular size (end diastolic/systolic diameter of 5.6/5.2cm), with resultant mild-moderate mitral and tricuspid regurgitation, left atrial dilatation (29cm²), mild pulmonary hypertension and a small pericardial effusion 8mm maximum. Cardiac magnetic resonance imaging (MRI) was requested and occurred one week later. This indicated severe left and right ventricular

impairment with ejection fraction 21% and 30% respectively. On delayed gadolinium enhancement imaging, there is transmural delayed enhancement in the apical lateral and apical inferior segments, and mid-myocardial delayed enhancement in the basal anteroseptum (figure 2a and 2b respectively). The patient clinically improved in hospital and was discharged with bisoprolol 5mg daily, cilazapril 2.5mg daily, spironolactone 12.5mg daily and furosemide 40mg daily, for subsequent up-titration and follow-up in cardiology clinic, and anti-thrombotic therapy ceased.

Discussion

Speculating the mechanism of cardiomyopathy in our case is fascinating, in the setting of significant previous methamphetamine use. A number of cardiac complications have been linked to methamphetamine use including hypertension, arrhythmias, sudden cardiac death, cardiomyopathy, acute coronary syndrome, vasospasm, coronary, aortic or carotid dissections[1]. Theories of underlying mechanisms of cardiomyopathy include direct myocardial toxicity, vasospasm, free radical formation and mitochondrial injury.

The focal transmural scar in the apical inferior and lateral segments suggests an ischaemic origin, and in the absence of coronary atherosclerotic disease on angiogram, this is likely a result of

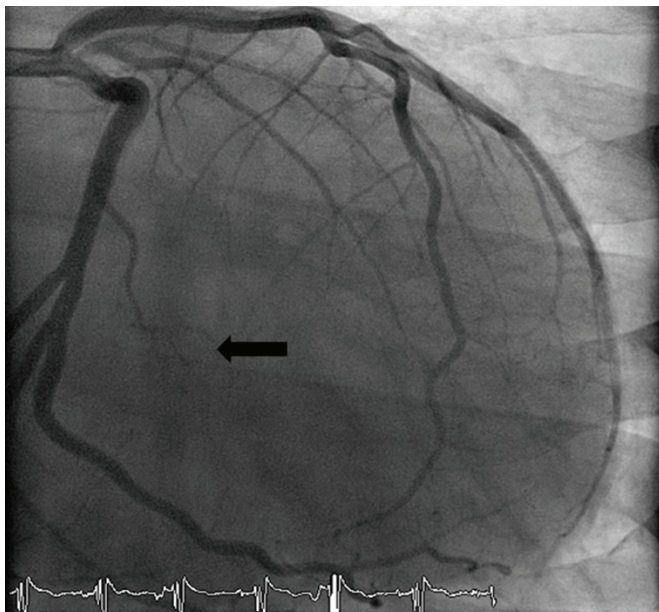


Figure 1. Coronary angiogram in the caudal 30 degrees view. Note the dissection in the obtuse marginal artery as marked by the arrow.

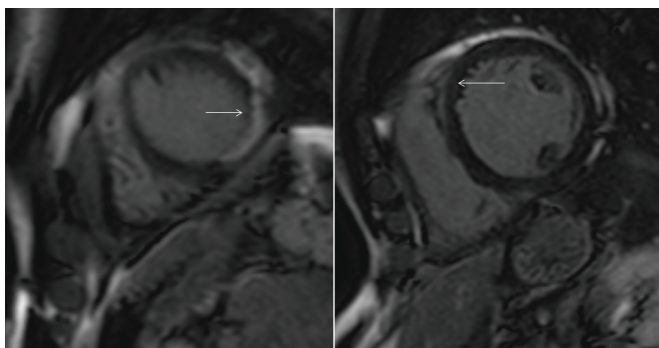


Figure 2. Cardiac magnetic resonance imaging of a) transmural and b) mid-myocardial scar (arrows)

the corresponding dissection of the second obtuse marginal branch, although another possibility would be vasospasm, both of which may be associated with methamphetamine[1]. On the other hand, the mid-myocardial scar on the basal anteroseptum suggests a non-ischaemic origin for example from direct toxicity of methamphetamine[2]. Although non-methamphetamine causes of cardiomyopathy cannot be ruled out, our MRI findings highlights the possibility of dual pathophysiologies of methamphetamine-induced cardiomyopathy, and this is the first such reported case in the literature.

Risk prognostication is also important, and a number of predictors of left ventricular function recovery in methamphetamine cardiomyopathy have been identified, including smaller left ventricular mean end diastolic diameter (49 vs 73mm) and atrial (mean 18 vs 29cm²) size, shorter duration of methamphetamine use, reverse Takotsubo rather than global systolic dysfunction pattern and absence of scar [2,3]. Our patient had adverse features of global systolic dysfunction, left atrial dilation and scar on MRI but also protective features of normal left ventricular size of high troponin levels. Cessation of methamphetamine use and heart failure therapy will both be important pillars of management to assist cardiac function recovery [2-4], however

in some patients severe dilation and dysfunction of left ventricle may persist despite optimal treatment, and this is associated with a poor prognosis [5].

Declarations of interest

The authors declare no conflict of interest.

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The authors state that they abide by the authors' responsibilities and ethical publishing guidelines of the International Cardiovascular Forum Journal [6].

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