Letter to the Editors

'Ally McBeal heart?' – Drug induced cardiomyopathy in a young woman

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Dilated cardiomyopathy is relatively uncommon in women of child bearing age. When it does occur, it is most commonly as a sequela of pregnancy, viral myocarditis or excessive alcohol intake. However, individuals in this age group are also amongst the most likely to consume other recreational drugs. We present the case of a young woman whose long-term amphetamine habit resulted in severe impairment of left ventricular function.

Mrs X was a 30-year-old mother of two children who was referred from her general practitioner with a four month history of ankle swelling, together with increased abdominal distension and breathlessness over the previous 3 days. Past medical history included an episode of pneumonia 7 years earlier, mild asthma (never requiring hospitalization), and regular panic attacks.

Symptomatic enquiry revealed that she had been experiencing a sensation of panic most nights in the last 2 months when she found she had been waking from sleep breathless. These symptoms were eased by sitting forward and she was adamant that they were quite distinct from the symptoms she would typically experience during a traditional panic attack.

She had a 20-pack year smoking history but professed only very occasional alcohol consumption. She did, however, admit to having taken amphetamines on a daily basis for the last 4 years. Her motivation for this habit was a desire to stay thin – and indeed she had initially begun to abuse the drug at a time when she was trying to give up smoking and subsequently gained weight. She purchased the drug illegally, consuming approximately one tablespoon per day. She estimated that she would utilize 15 g of amphetamine per month at a price of roughly 10 pounds sterling per gram.

On examination she was thin with a BMI of 19 kg m⁻². Respiratory rate was increased and saturations on air were low at 92%. A sinus tachycardia of 120 bpm

was present. Blood pressure was 125/90. The apex beat was displaced and the jugular venous pressure was elevated 8 cm. Auscultation revealed a gallop rhythm and a left basal effusion. There was hepatomegaly, mild ascites and peripheral pitting oedema.

ECG demonstrated biphasic P waves in keeping with left atrial enlargement, a left ventricular hypertrophy and strain pattern, and Q waves in the anteroseptal leads. CXR showed cardiomegaly, interstitial pulmonary oedema and a small left pleural effusion. Full blood count with MCV of 84, urea and electrolytes normal. Liver function tests were marginally abnormal with bilirubin 22 mmol l⁻¹ [4–20], alkaline phosphatase 83 (30–100 IU l⁻¹), ALT 70 (5–31 IU l⁻¹) and albumin 28 g l⁻¹ [35–50].

A diagnosis of amphetamine-related cardiomyopathy was made and she was commenced on a diuretic, warfarin and an ACE inhibitor. Subsequent echocardiography confirmed a significantly dilated left ventricle (systolic and diastolic dimensions 6.1/6.9 cm, respectively; normal upper limits 3.5/5.7 cm). There was global impairment of contraction with an estimated ejection fraction of 26%.

Left atrium was 4.1 cm (1.9–4.0 cm).

During a 7 day stay she made a dramatic improvement on therapy with both clinical and radiographic resolution of her pulmonary oedema. At that stage her left ventricular dimensions had improved to 5.7/6.8 cm and left atrium to 2.9 cm with an ejection fraction of 33%. She expressed a firm wish to stay off amphetamines for good and coped well with minor withdrawal symptoms, requiring only occasional small doses of diazepam.

Discussion

Amphetamine abuse is a more widespread phenomenon in the United Kingdom than is perhaps appreciated. There are more confiscations of this substance by the police than for any other drug except cannabis. Synthetic amphetamines produced in illegal laboratories find their way to the street, whilst 'legal' amphetamine

abuse may occur with the aid of prescription medications and assorted over-the-counter diet pills.

These compounds are related by their phenylethylamine structure, which bears strong resemblance to that of naturally occurring biogenic amines (dopamine, serotonin, catecholamines, etc.) and helps to explain their plethora of peripheral and central nervous system effects.

There have been sporadic reports in the literature of cardiomyopathy associated with amphetamine use over the last 30 years [1, 2]. On at least one occasion the degree of left ventricular failure was severe enough to lead to death despite therapy [1]. Several authors report acute reversible left ventricular function following intravenous administration which is a common route of abuse along with smoking or ingesting the drug [3, 4]. Fortunately our patient was not taking amphetamines during either of her pregnancies, as prenatal exposure has also been associated with cardiomyopathy in the neonate [5].

Amphetamine users generally fall into two groups – firstly, those who indulge for the chemically induced euphoria it provides. Use of the drug in this context has become inextricably linked with the club music scene. The second group of users take amphetamines as a way of combating weight gain. These individuals tend to be female, in the reproductive years and not infrequently in professional occupations. With ever thinner actresses debuting on our screens (the lead actress in the popular legal drama *Ally McBeal* is reputedly a UK size 6!), it is perhaps not surprising that some turn to drugs to maintain an otherwise unsustainable body image. Although our patient was taking a large amount of illegal amphetamine, acute cardiomyopathy has also been described occurring after taking prescription amphetamines at the appropriate dose [6].

The mechanism of cardiomyopathy is uncertain. It is possible that adrenergically driven recurrent hypertensive crises ultimately result in failure of the ventricle in exactly the same way as essential hypertension. Our patient had classical left ventricular hypertrophy and strain pattern on the ECG yet echocardiographic measurements showed the septum and posterior wall thickness to be within normal limits but estimated left ventricular mass was definitely elevated at 355 g (uncorrected).

Also unexplained in our case is the presence of anteroseptal Q waves on the ECG.

Clearly, in another setting, these would generally indicate prior myocardial infarction but this appearance is not uncommon in dilated cardiomyopathy. There was little evidence to support a prior myocardial infarction

in our patient: young age, no family history; no antecedent chest pain; no regional wall motion impairment in the left anterior descending artery territory (i.e. dysfunction was global rather than regional). Ideally, we would have liked to proceed to magnetic resonance imaging with late gadolinium enhancement to confirm the absence of scar. This technique is now widely accepted as the gold standard for infarct-detection [7]. Unfortunately, the patient was needle-phobic and refused all but the minimum of interventions.

Conclusion

Amphetamine-induced cardiomyopathy is likely to be increasingly recognized in younger age groups and should be considered once the usual differential for cardiomyopathy has been exhausted. The few case reports in the literature suggest that standard therapy is appropriate and that in most instances abstinence from the drug will lead to an improvement in cardiovascular function both clinically and on serial imaging.

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