

CASE REPORT

Acute myocardial infarction caused by intravenous amphetamine abuse

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Abstract

A man aged 27 years presented with an acute myocardial infarction after injecting himself intravenously with amphetamine. Soon after admission ventricular fibrillation developed. This was successfully cardioverted. Coronary arteriography was normal.

Myocardial infarction is a rarely reported complication of amphetamine abuse. We describe a patient in whom an acute myocardial infarction developed after intravenous injection of amphetamine.

Case report

A 27 year old man injected himself intravenously with two doses (total 1.5 g) of amphetamine at an interval of half an hour. An hour later he felt unwell and after four hours severe central chest pain developed. He had previously taken amphetamine without symptoms and had misused other drugs such as heroin and cannabis. He smoked 15 cigarettes per day and drank alcohol occasionally. His father had a history of ischaemic heart disease.

Two and a half hours after the onset of chest pain he was admitted to hospital. The electrocardiogram showed evidence of an anterolateral myocardial infarction (figure). The chest radiograph was normal. He was treated with intravenous diamorphine 5 mg

and prochlorperazine 12.5 mg. Soon afterwards he had a cardiac arrest and the cardiac monitor showed ventricular fibrillation. He was successfully cardioverted by a single direct current shock of 400 J. He was subsequently given 1.5 megaunits of streptokinase. Over the ensuing 24 hours the electrocardiographic monitor showed frequent ventricular extrasystoles, several self-terminating runs of ventricular tachycardia, and intermittently a nodal rhythm. Thereafter he made an uncomplicated recovery. Blood taken after he was resuscitated showed a rise in serum alanine aminotransferase reaching a maximum of 224 U/l the day after admission, and in lactate dehydrogenase reaching a maximum of 556 U/l two days after admission. Gamma glutamyl transferase rose to 104 U/l two days after admission. Antibodies to hepatitis B antigen and human immunodeficiency virus were not detected in his serum. Before discharge from hospital he was able to complete a limited treadmill exercise test without symptoms developing: inverted T waves, which were evident on the electrocardiogram before exercise, became upright during exercise. He was discharged after one week and advised to take aspirin 150 mg daily. He was strongly urged to avoid drug misuse in the future.

After two months he was readmitted for additional investigations. His electrocardiogram had by now reverted to normal. Fasting blood lipids were normal. He performed a

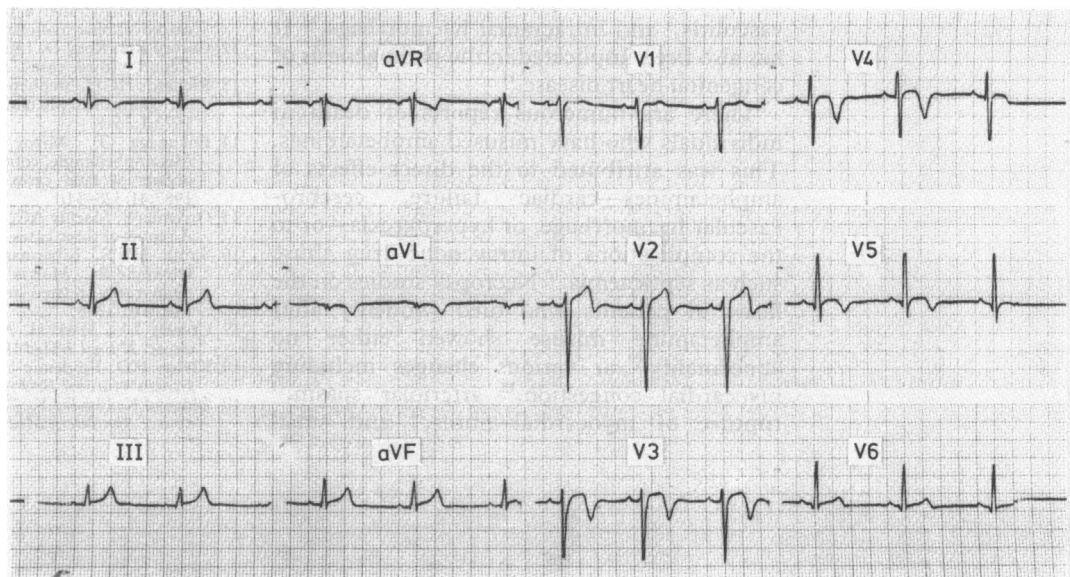


Figure Electrocardiogram on admission to hospital.

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repeat exercise test using the full Bruce protocol and was able to accomplish 12 minutes without symptoms developing; the electrocardiogram remained normal throughout the test. Coronary arteriography was normal but the ventriculogram showed a small area of apical dyskinesia.

Discussion

Our patient sustained an acute myocardial infarction after intravenous injection of amphetamine. There have been only two previous reports showing a clear relation between amphetamine misuse and myocardial infarction. Carson *et al* described a patient aged 33 years in whom chest pain developed one hour after intravenous injection of amphetamine.¹ His electrocardiogram showed an inferolateral myocardial infarction; coronary arteriography was normal. In the patient described by Marsden and Sheldon an anterior myocardial infarction developed after he had ingested the contents of a propylhexedrine aerosol inhaler; adult respiratory distress syndrome also developed.² Four of six patients reported by Anderson and Scott experienced chest pain after receiving amphetamine.³ In one heart block also developed but the electrocardiographic findings in the other patients were not described.

Myocardial infarction after amphetamine abuse may have been caused by coronary artery spasm. Amphetamine stimulates release of noradrenaline from sympathetic nerves; this has a pressor effect on the coronary circulation.⁴ Alternatively, transient occlusion of the coronary vessels may have been caused by platelet thrombi: catecholamines are known to induce platelet aggregation.⁵ These effects may have been aggravated by an increase in myocardial oxygen demand induced by catecholamines.⁴

In addition to myocardial infarction, amphetamine abuse has been implicated in the aetiology of other cardiovascular disorders including acute⁶ and chronic cardiomyopathy,⁷ cor pulmonale,⁸ necrotising vasculitis,⁹ and intracranial haemorrhage.¹⁰ It has also been implicated in the pathogenesis of congenital heart disease.¹¹

There are numerous reports of death in individuals who have misused amphetamines. This was attributed to the direct effects of amphetamines—cardiac failure, cerebrovascular haemorrhage, or hyperpyrexia—or to the complications of intravenous drug abuse such as septicaemia.¹⁰ Necropsy studies on the heart in patients who died suddenly after amphetamine misuse showed either no abnormality¹⁰ or various changes including myocardial congestion,¹² arteriolar spasm,¹³ rupture of myocardial fibres,¹⁴ and small

interstitial¹⁴ and endocardial haemorrhages¹⁵; there are no reports of myocardial infarction. Although amphetamine abuse has the potential to cause myocardial infarction, it is likely that in many cases ventricular fibrillation supervenes before the changes of myocardial infarction become manifest.

Cocaine abuse has been strongly associated with myocardial infarction.^{16,17} Like amphetamine, it enhances noradrenaline release and probably causes myocardial ischaemia by inducing coronary artery vasospasm and by increasing myocardial oxygen demand.^{16,17} Recreational use of the purer, free-base form of cocaine (crack) has reached epidemic proportions in North America and there are fears that use of crack could become widespread in the United Kingdom.¹⁸ Cocaine is often diluted (cut) with other agents such as amphetamine before it is sold, so drug misusers are often unaware of the exact composition of the drugs they are taking.¹⁹ This raises the possibility that the patient we describe may inadvertently have taken a combination of amphetamine and cocaine which may have had an additive effect in provoking myocardial ischaemia.

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