CASE REPORT

Ecstasy induced acute myocardial infarction

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Abstract

A 23 year old man presented with a clinical history and ECG compatible with acute myocardial infarction, having taken a single tablet of ecstasy (3,4-methylenedioxymetamphetamine) 18 hours previously. He was treated with aspirin and thrombolytic therapy; however, cardiac catheterisation showed angiographically normal coronary arteries and left ventricular function. Sympathomimetic drugs are freely available and widely abused in Britain, but there is little evidence of the mechanisms or management of cardiac complications. In such cases the use of standard treatment for acute myocardial infarction is recommended with agents such as glyceryl trinitrate and phentolamine to reduce coronary artery spasm. Early coronary angiography may help to determine the relative contribution of spasm, thrombus, and underlying atherosclerotic disease.

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Keywords: ecstasy; 3,4-methylenedioxymetamphetamine; acute myocardial infarction

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A 23 year old white man presented to his local hospital with a six hour history of crushing chest pain, nausea, sweating, and breathlessness. He reported taking a single tablet of ecstasy or 3,4-methylenedioxymetamphetamine (MDMA) 18 hours previously. He had also used ecstasy and crack cocaine three months earlier. He was a smoker with a six pack-year history. There was a strong family history of ischaemic heart disease, with no known diabetes or hypertension. He used inhaled salbutamol as required for asthma. He was pale, sweating, and tachycardic with blood pressure 130/80 mm Hg. Heart sounds and respiratory examination were normal. ECG on admission (fig 1) showed widespread ST segment elevation confirming acute myocardial infarction. He was given aspirin, oxygen, and diamorphine, and thrombolytic treatment with recombinant tissue plasminogen activator (rt-PA) and an infusion of glyceryl trinitrate were initiated. Twelve hours later his chest pain recurred with further ST elevation, and thrombolysis with rt-PA was repeated. Echocardiography showed good left ventricular systolic function with mild anteroapical hypokinesia. Creatine kinase was 553 IU/l on admission. Laboratory analysis of a urine specimen showed the presence of MDMA.

He was transferred the following day to the regional cardiac centre, where left heart catheterisation showed angiographically normal coronary arteries and left ventricular function. He was asymptomatic and discharged seven days after admission, at which time his ECG showed significant residual ST elevation in the inferolateral leads without Q waves.

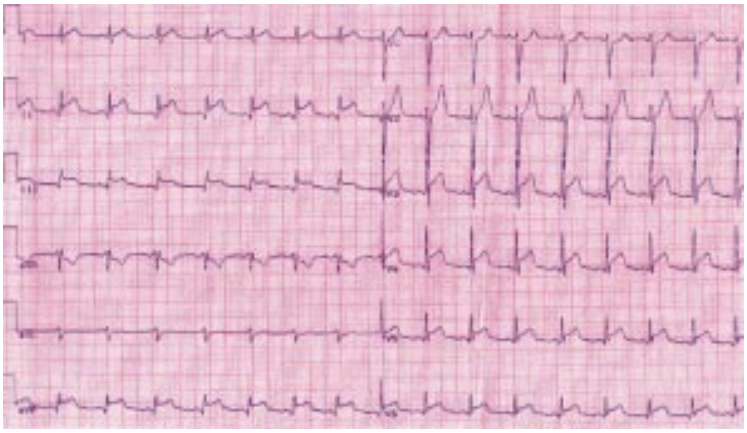

Figure 1 Admission ECG from a 23 year old man with ecstasy induced acute myocardial infarction.

Discussion

This case illustrates the importance of a careful drug history in young patients, as illegal recreational sympathomimetic drugs are widely used and freely available in Britain. MDMA is used regularly by large numbers of young people in nightclubs, and its effects on body temperature and fluid balance have been widely publicised. There has been a previous reported case of a patient presenting with ischaemic sounding chest pain and ECG changes of acute myocardial infarction after using MDMA.[1] As with acute myocardial infarction caused by cocaine or amphetamine abuse it is thought that in the case of MDMA severe vasospasm is the main mechanism,[2] and the contribution of thrombus formation is uncertain.[3] There is also postmortem examination[4] and animal model evidence of patchy myocardial necrosis,[5] which may result from direct myocardial toxicity of MDMA metabolites.

Developed initially as an appetite suppressant and then used as a psychotropic agent, MDMA stimulates release of noradrenaline (norepinephrine), dopamine, and serotonin from the central and autonomic nervous

systems and has a monoamine oxidase inhibitor effect, inhibiting the reuptake of catecholamines in sympathetic synapses.[6] The effects start around 20 minutes after ingestion and last up to 48 hours, and can be potentiated by alcohol. The resulting sympathetic surge can cause tachycardia, hypertension, and arrhythmias, as well as myocardial ischaemia.[2]

There is no evidence base for the management of these patients and so treatment should be pharmacologically based. Coronary artery spasm can be treated effectively with infusions of glyceryl trinitrate or phentolamine, and aspirin can safely be given. The need for fibrinolytic agents is unclear and although streptokinase has been used effectively in myocardial infarction caused by cocaine abuse, its safety and efficacy in this situation are not known. Treatment with β blockers for hypertension or arrhythmias should be avoided because unopposed α-adrenoceptor activation may result in severe hypertension. There are good reasons to consider early coronary angiography and establish whether there is atherosclerotic coronary artery disease, thrombus, or vasospasm. This would allow better targeting of treatment with antiplatelet agents, percutaneous intervention, or drugs to treat coronary artery spasm.

In many cases the exact nature and dose of recreational drugs are difficult to establish when patients present acutely requiring urgent treatment. We feel that the safest approach to the management of patients with chest pain and ECG ST segment elevation after ingestion of sympathomimetic recreational drugs is with standard treatment for acute myocardial infarction, with a relatively low threshold for coronary angiography when there is persistent or recurrent chest pain with dynamic ECG changes.

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