Clin Res Cardiol
DOI 10.1007/s00392-010-0205-z

LETTER TO THE EDITORS

Ecstasy-induced myocardial infarction in a teenager: rare complication of a widely used illicit drug

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Received: 11 March 2010 / Accepted: 3 August 2010

Sirs:

Ecstasy [methylene 3,4 dioxymethamphetamine (MDMA)] is a popular so-called recreational illicit drug, especially consumed in adolescents and young adults [9]. Well-known severe adverse effects include psychiatric complications, hyperthermia, rhabdomyolysis, hepatic and renal failure, stroke, seizures or disseminated intravascular coagulation [5, 7]. Cardiovascular problems like arrhythmias and hypertension are also recognized [1, 3], but there are only very few cases of myocardial infarction reported in the literature [1, 8, 11–14]. We report on a 16-year-old adolescent, who developed myocardial infarction after ingestion of the drug.

A 16-year-old male was admitted to our hospital presenting with retrosternal chest pain after having been on a party the night before. After falling asleep he complained of thoracic oppression, retrosternal burning and shortness of breath lasting for several hours. He had smoked and had "a few beers", but later on he reluctantly admitted the intake of "ecstasy". His medical history was free except for uncomplicated remote children's diseases.

Physical examination revealed no cardiac murmur, gallop or friction rub, or arrhythmia. Heart rate was 100/min, blood pressure was 140/90 mmHg. Lungs were clear. There was no jugular distension or edema.

ECG showed sinus rhythm and ST-segment elevation in leads I, II, aVL, aVF and V4 through V6 (see Fig. 1).

The echocardiogram showed normal chamber sizes and biventricular normal global systolic function. There was slight hypokinesis in the apical anterior, septal und mid-posterior segments. Valves were normal. No pericardial effusion was seen. The entire routine lab was normal, except for elevated levels of troponin I, 7.5 ng/ml (normal $\leq$0.3); creatine kinase, 870 U/l; maximum 1,020 U/l the following day (normal $\leq$174 U/l); CK MB, 75 U (normal $\leq$24 U/l) and SGOT, 71(normal = 10–50 U/l). Semi-quantitative urine analysis for amphetamine derivatives was strongly positive [urine amphetamine, >2,000 ng/ml (normal $\leq$300 ng/ml)]. The patient received aspirin and heparin and left heart catheterization was performed.

Left ventricular angiogram revealed normal ejection fraction with discrete regional hypokinesis as seen in the echocardiogram. Coronary angiogram (CA) showed global vasospastic narrowing that immediately resolved after intracoronary (IC) nitroglycerine, accompanied by a prompt relief of chest pain (Fig. 2a, b) The diagnosis of drug-induced vasospastic myocardial infarction was made. The patient was transferred to the coronary care unit. Nitro IV and amlodipine as well as aspirin and clopidogrel per os were instituted.

The clinical course was uneventful. Repeat echocardiographic studies before discharge were normal, as it was the ECG. All medications were stopped. The patient and his parents were comprehensively informed about the potential danger of further ecstasy misuse and ambulatory psychological support was established.

Ecstasy and its main component MDMA are synthetic analogues of amphetamine. They exert similar psychoactive effects that begin as early as 20 min after ingestion and usually last about 6 h. At higher doses it may even last up to 48 h [4]. Ecstasy is distributed in form of colored and coined tablets that besides pure MDMA often consist of several related substances like methylenedioxyamphetamine (MDA), MDE (methylenedioxyethyl-amphetamine/"eve") and others.

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Published online: 07 September 2010

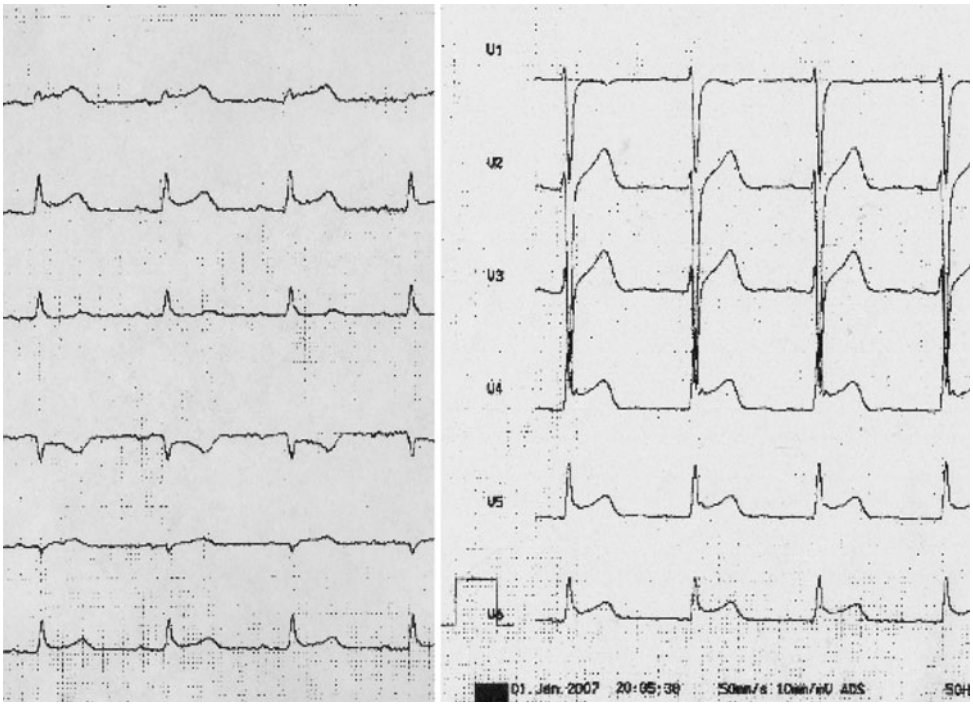

Fig. 1 The ECG on admission shows sinus rhythm at 99 bpm. There are marked ST-segment elevations in leads I, II, aVL, and V4 through V6

Pharmacologic or toxic effects are similar [4, 9]. Although illicit, the drug is widely used mainly by adolescents and young adults as a so-called recreational drug. Customers aim for its generally stimulating and euphorisating effect, however, panic attacks, depression or paranoid psychosis, collapse, seizures and loss of consciousness may occur [8, 9].

The true incidence of ecstasy-induced complications is unknown, but only one emergency admittance is estimated to occur among 10,000 intakes [8]. Most medical problems are considered modest and selflimiting, and therefore may be underreported [8]. Nevertheless, severe, even life-threatening complications are well known. They include hyperthermia and rhabdomyolysis as a consequence of extensive dancing activity ("rave parties") [8, 9], and severe liver failure or renal failure [4, 9].

Ecstasy exerts its effects by releasing central nervous noradrenaline, dopamine and serotonin and inhibiting the re-uptake of catecholamines into the sympathetic synapses [1, 4]. There are only few cases of myocardial infarction caused by the drug reported in the literature [1, 4, 6, 11–14]. Hence, no definitive conclusions about the underlying mechanism can be drawn. Myocardial necrosis has been found in an anatomical animal study attributed to a direct toxic effect [10]. Similar to cocaine, excess alpha adrenergic stimulation is thought to lead to vasoconstriction, thereby probably enhancing autochthonic thrombus formation [1, 4, 7]. A few angiographic studies either showed thrombosis, entirely normal or stenosed coronary arteries [6, 11–14]. One case reported presumed microvascular spasm [2].

In our patient global epicardial vasospasm was identified, but no thrombus could be detected. It is speculative, whether thrombotic material was too small to be detected on the angiogram or already had dissolved spontaneously or embolized distally.

Nevertheless, our case provides evidence of ecstasy-induced ubiquitous epicardial vasospasm, which was promptly reversible to Nitro IC. It underlines the presumed and discussed mechanism in ecstasy-induced MI [1, 2, 10–13].

We treated the patient with aspirin and clopidogrel to reduce thrombocyte aggregation and with nitroglycerin and a calcium antagonist to provide coronary vasodilatation.

Betablockers were not used as these drugs are thought to further enhance hypertension and vasoconstriction in the ecstasy-induced prevailing alpha-sympathomimetic situation, as it is known from cocaine toxicity [1, 4, 7, 11].

Thrombolysis is considered to be safe in cocaine-induced MI [4, 7], and in some cases, patients with ecstasy-induced MI were treated safely with thrombolysis [1, 9, 11]. However, ecstasy consumption has been associated with excessive hypertension and intracranial hemorrhage [1, 4]. Therefore thrombolysis could further increase the bleeding risk. In our case vasospasm was identified as the underlying mechanism. In accordance with the proven superiority of percutaneous intervention (PCI) in "classic" MI we preferred performing CA with the option of immediate PCI [1].

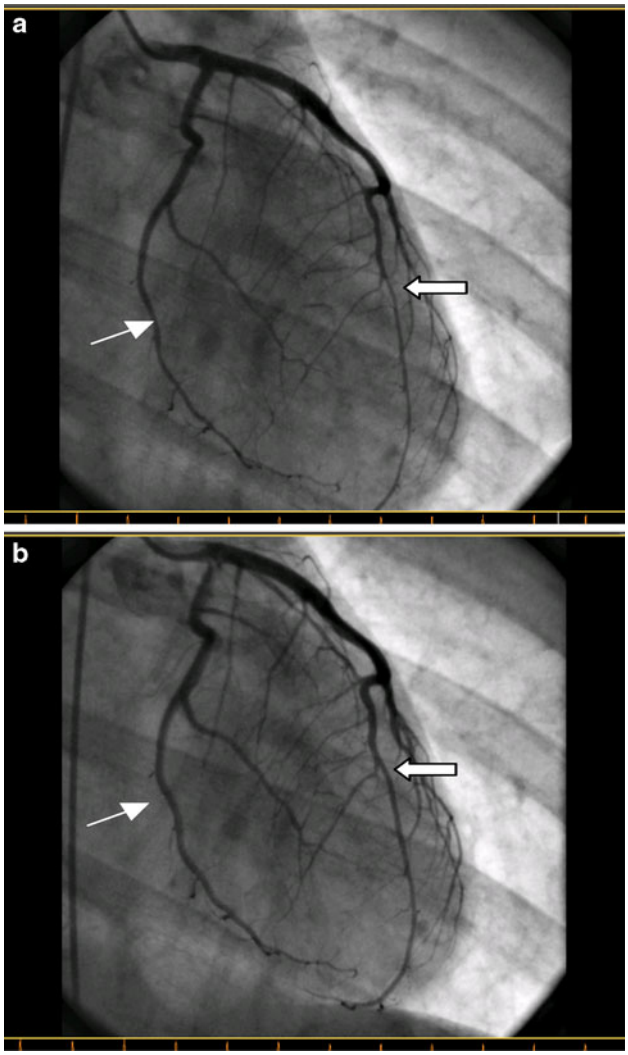

Fig. 2 **a** Shows marked coronary vasoconstriction in the left anterior descending branch (LAD) (*big arrow*) and circumflex artery (RCX) (*small arrow*). **b** Shows normalized coronaries in LAD and RCX after Nitro IC

We have reported on a 16-year-old teenager, who experienced a myocardial infarction after ingestion of ecstasy. Coronary angiography revealed global vasoconstriction and no thrombus, providing insights into the pathophysiology of a very rare complication of ecstasy misuse.

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