Clinical Reports

Cerebral infarction in association with Ecstasy abuse

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Summary: A previously fit 35 year old man presented with a right hemiparesis and dysphasia 36 hours after abuse of Ecstasy (3,4-methylenedioxymethamphetamine). Computerized axial tomography scan demonstrated an extensive acute left cerebral infarction and carotid digital subtraction angiogram, 2 days after admission, revealed left middle cerebral artery occlusion. There were no other known risk factors and all other investigations were negative. The patient made a partial recovery. We propose an association between Ecstasy abuse and cerebral infarction.

Introduction

Although intracranial haemorrhage and infarction are rare complications of amphetamine abuse,^{1–4} cerebral infarction consequent upon Ecstasy abuse is not recorded. We report a case of major cerebral infarction within 36 hours of Ecstasy abuse.

Case report

A previously fit 35 year old male presented with sudden collapse, dense right hemiparesis and severe mixed dysphasia. Blood pressure on admission was 110/60 mmHg. He was apyrexial, not clinically dehydrated and the remainder of the examination was normal.

History from his partner was that the patient had taken Ecstasy (3,4-methylenedioxymethamphetamine) several times, 36 hours before and had been smoking cannabis when he collapsed without warning. There was no suggestion that the acute effects of Ecstasy were more extreme than normal and there was no history of trauma.

Biochemistry, haematology, chest radiography and electrocardiography on admission were normal. Investigations performed within the following 3 days included full clotting profile, erythrocyte sedimentation rate, antithrombin III levels, two-dimensional echocardiogram and fasting lipid profile. All were normal. Serum and cerebrospinal fluid Venereal Disease Research Laboratory and Treponema Pallidum Haemagglutination tests were negative, as was a full autoimmunity screen. A

computerized axial tomography brain scan 3 days after admission showed a large acute left cerebral infarct including frontal, upper temporal and mid and lower parietal lobes. Repeat scan 8 weeks later confirmed an established large middle cerebral artery territory infarct with no other abnormalities. Carotid digital subtraction angiogram 2 days after admission showed complete occlusion of the left middle cerebral artery, with no other abnormalities and, in particular, no evidence of an intimal tear.

Following prolonged rehabilitation motor function and mobility have improved and he has returned to independent living at home. However, communication is extremely limited by severe expressive dysphasia and verbal dyspraxia.

Discussion

Ecstasy or MDMA (3,4-methylenedioxymethamphetamine) is a semi-synthetic hallucinogenic amphetamine structurally similar to endogenous catecholamines. Acute adverse reactions follow from the effects of variable central and peripheral sympathetic stimulation of alpha- and beta-adrenergic receptors. The toxic dose of MDMA is not known and severe adverse reactions are commonly idiosyncratic.⁵

Described effects have been reviewed recently⁵ and range from mild usually acute reactions at low doses, including nausea, sweating, dry mouth, anorexia, insomnia, ataxia, nystagmus and increased body temperature, to moderate reactions including hyperreflexia, tachycardia, initial hypertension leading to hypotension, hyperventilation and visual hallucinations. More severe reactions include delirium, loss of consciousness,

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convulsions, cardiac arrhythmias, hyperpyrexia, rhabdomyolysis and acute renal failure.

Amphetamine-related death from intracerebral haemorrhage is documented and it is postulated that sudden transitory extreme hypertension soon after substance intake⁶ or amphetamine-related vasculitis may be contributory. Immunosuppressives have produced clinical and radiological improvement in the latter.⁷

The possibility of infarctive stroke as a complication of amphetamine abuse has also been suggested. In rhesus monkeys intravenous injection of methamphetamine produces almost immediate 'beading' of cerebral arteries on arteriography, and subsequent vasculitis, focal areas of infarction and ischaemia.² Cerebral vasculitis is described in association with amphetamine overdose⁸ in humans. Infarctive stroke has been sporadically associated with intranasal use of methamphetamine^{3,4} with an interval between reported usage and stroke of between 12 hours and 2 weeks. The apparent delay in presentation, also seen in the present case, may be the result of patient under-reporting of the frequency of drug abuse⁴ or part of the pathogenic mechanism (for example, perhaps delayed cerebral vasculitis).³ Either may apply to the present case as the history was not obtained directly from the patient.

It is unclear whether the proposed effect of Ecstasy in the present case may have been idiosyncratic or dose related, although the lack of an unusual or extreme acute effect of the drug points tentatively to the former. Another possible mechanism might be undocumented physical trauma experienced whilst under the influence of Ecstasy, though the absence of intimal tearing on angiography only 2 days after the onset of stroke makes this unlikely.

We believe the present case in which there were no other recognized risk factors for cerebrovascular disease, is the first to suggest a link between cerebral infarction and Ecstasy abuse. It may be that cannabis use exerted a potentiating effect, although if so we are unable to suggest a possible mechanism. We propose an association between cerebral infarction and Ecstasy abuse.

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