better). Ophthalmoscopy revealed bilateral cotton wool spots and haemorrhages. A provisional diagnosis of cytomegalovirus retinitis was made and he was immediately transferred to the eye department for an ophthalmic opinion. His best corrected visual acuities were confirmed. There was no afferent pupillary defect. Anterior segment examination revealed no abnormalities. Examination of the retina showed bilateral mild optic disc swelling, venous dilatation, arteriolar attenuation, cotton wool spots, and blot and flame shaped haemorrhages (fig 1). The most striking findings were the large cotton wool spots involving the posterior poles including the maculae. The differential diagnosis of this fundal picture in a patient with this history is wide but is in keeping with malignant hypertension. His blood pressure was found to be 210/140. A provisional diagnosis of malignant hypertension was made and the patient transferred under the care of the physicians for further management.

The patient was started on oral antihypertensives while other investigations relating to the differential diagnosis of the retinal picture and the cause of his hypertension were performed. Blood glucose, blood cultures, urinalysis, plasma viscosity, C reactive protein, liver function tests including plasma proteins, full blood count, auto-antibodies including ANCA, and an HIV test were all normal. An echocardiogram revealed left ventricular hypertrophy but no vegetations and computerised tomography of the brain showed extensive oedema with no focal lesions.

His hypertension gradually came under control and subsequently his vision and retinal appearances improved. Despite extensive investigations no cause for his hypertension has been found.

Discussion

Bilateral visual loss in a man of this age is unusual, as is malignant hypertension. The differential diagnosis of visual loss and retinal cotton wool spots is considerable, and further complicated by the history of intravenous drug abuse. The most common causes of cotton wool spots include diabetes mellitus and hypertension,

but other causes include severe anaemia, vasculitis, blood dyscrasias, and infection including endocarditis.¹ Intravenous drug abusers can develop cotton wool spots due to embolism of infected material (including fungi and bacteria) or talc, as well as HIV, AIDS, and its associated infections. A blood pressure reading of 210/140 narrows the differential diagnosis considerably.

Cotton wool spots are caused by retinal nerve fibre layer infarcts, leading to a cessation of axoplasmic flow and the accumulation of axonal material.² Hypertension causes damage to blood vessels, mainly arterial, leading to obstruction and leakage from these vessels. Changes in the fundus include arteriolar attenuation, arteriovenous nipping and displacement, retinal haemorrhages, hard exudates, cotton wool spots, and optic disc swelling.²

Malignant hypertension can present with heart failure, hypertensive encephalopathy, renal failure, headaches, and grade III-IV hypertensive retinopathy. Initial investigations include assessment of renal function, chest x ray and electrocardiogram. Treatment involves strict bed rest with a gradual reduction of blood pressure, usually with oral antihypertensives such as nifedipine. Initially, chewing a 10 mg nifedipine capsule will reduce the blood pressure over 30 minutes, followed by 10–20 mg of oral nifedipine eight hourly. In the unconscious patient close control of blood pressure can be attained using intravenous sodium nitroprusside.³

Despite the wide differential diagnosis and unusual presentation of this case the diagnosis was made on a simple blood pressure measurement. Subsequent extensive investigations case failed to reveal any cause for the hypertension. This case illustrates the importance of checking the blood pressure in every patient attending for visual loss.

- 1 Brown GC, Brown MM, Hiller T, Fischer D, Benson WE, Magargal LE. Cotton wool spots. *Retina* 1985;5:206–14.
- 2 Wall M, Breen L, Winterkorn J. Optic disk edema with cotton wool spots. *Surv Ophthalmol* 1995;39:502–8.
- 3 Hope RA, Longmore JM, Hodgetts TJ, Ramrakha PS. Accelerated phase (malignant) hypertension. In: *Oxford handbook of clinical medicine*, 3rd ed. Oxford: Oxford University Press, 1993:302.

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Accidental ingestion of Ecstasy by a toddler: unusual cause for convulsion in a febrile child

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Abstract

The case is reported of a toddler who presented with an apparent febrile convulsion. The final diagnosis was that of accidental ingestion of Ecstasy. The child made an uneventful recovery. Ecstasy tox-

icity should be added to the list of differential diagnoses in a child presenting with fever and an unexplained seizure.

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Keywords: Ecstasy; toddler; accidental ingestion

Reports from several countries confirm an increase in the availability and use of Ecstasy (3,4-methylenedioxymethamphetamine or MDMA).^{1,2} Medical papers and current media attention concentrate on its toxic effects in adolescents and young adults. Accidental ingestion of Ecstasy by a young child has been described before under circumstances where the history of ingestion was immediately and frankly made available to medical staff.³ We present a case which shows that—for obvious reasons—parents may not always be so forthcoming with a relevant history of ingestion of illicit substances.

Case report

A two year old girl was brought urgently to the accident and emergency department by her mother in the early afternoon. She had suddenly become unwell and her mother gave a history of a probable generalised seizure. She was on a course of amoxycillin for an upper respiratory tract infection. The girl had a history of speech delay for which she was attending speech therapy. Her 21 year old mother appeared somewhat withdrawn, although appropriately anxious about her child's condition.

On examination the girl was agitated and crying inconsolably. She had a respiratory rate of 48/min, an axillary temperature of 39°C, a sinus tachycardia of 207 beats/min, and a blood pressure of 120/60 mm Hg. She was noted to have dilated (7 mm), reactive pupils. Examination was otherwise unremarkable. The initial diagnosis was of a febrile convulsion secondary to an upper respiratory tract infection, although it was recognized that the child's agitation and extreme tachycardia were not typical of a febrile convulsion. She was tepid sponged, given oxygen and rectal paracetamol, and admission was arranged.

Following admission the child began grinding her teeth and had an oculogyric crisis. She was prescribed intravenous diazepam. It was noted on the ward that the mother also had dilated pupils. On further questioning the mother reluctantly mentioned possible ingestion of an unknown substance, as she said that she had found some white chalky matter in her child's mouth. Given this history of ingestion, gastric lavage was performed under general anaesthesia. The girl subsequently made an uneventful recovery with no change in any biochemical or haematological indices.

A drugs screen was requested on urine and gastric lavage samples and the results were available the following day. Chromatography of the stomach contents detected the presence of MDMA. Chromatography of the child's urine detected the presence of MDMA and its metabolite MDA (methylenedioxyamphetamine). Confronted with the toxicology results the mother admitted that her child had managed to put one Ecstasy tablet in her mouth when she was left unattended. The tablet had been left in a cup on a bed awaiting consumption. On re-entering the room the mother had immediately realised that the

tablet was missing and was able to clear some of it from her daughter's mouth.

Discussion

Acute Ecstasy toxicity may cause cardiac dysrhythmias, severe hyperthermia, coma, convulsions, rhabdomyolysis, hepatic and renal impairment, and disseminated intravascular coagulation.⁴ Bruxism (grinding of the teeth) and an acute dystonic reaction have also been reported.⁵ Toxicity does not appear to be necessarily related either to the amount ingested or the serum concentration.⁴ In the United States, few cases of death or severe reaction have been reported, despite its widespread use.⁵ Observed differences in toxicity between the United Kingdom and the United States have been attributed to the circumstances in which Ecstasy is consumed.^{4,7} In the USA it is usually taken alone or at parties while in the United Kingdom it is used largely as a "dance drug", ingested at a time of marked physical exertion, generally in a warm environment with variable fluid intake. There is only one previously reported case of Ecstasy toxicity in a young child.³ In neither the previous case nor this one did any haematological or biochemical abnormalities develop. Both children made a full recovery. It is assumed that they were both normally hydrated before ingesting the drug.

Young children may ingest any harmful substance that is left unattended or insecure. As the use of recreational drugs increases, the incidence of their accidental ingestion by children may similarly rise. Children's exposure to Ecstasy may be limited in the United Kingdom, as it is largely consumed in nightclubs and at "raves". Children may conversely be put at more risk by the continuing false perception of Ecstasy as a safe drug outside the club environment. Such beliefs may lead to a more relaxed attitude to supervision and security when Ecstasy is present in the home.

This case illustrates the difficulty in making a diagnosis of drug ingestion in a young child if the parent or guardian conceals relevant history. Ecstasy toxicity should be added to the list of differential diagnoses in a child who presents with fever and an unexplained seizure. To make the diagnosis of a drug induced convulsion in these patients it is essential that the physician has a high index of suspicion.

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- 1 Clegg MT, Tracey JA. Ecstasy abuse in Ireland. *Irish Med J* 1993;86:118-9.
- 2 Cuomo MJ, Dymont PG, Gammino VM. Increasing use of "Ecstasy" (MDMA) and other hallucinogens on a college campus. *J Am Coll Health* 1994;42:271-4.
- 3 Bedford Russell PJ, Schwartz RH, Dawling S. Accidental ingestion of Ecstasy. *Arch Dis Child* 1992;67:1114-5.
- 4 Henry JA, Jeffreys KJ, Dawling S. Toxicity and deaths from 3,4-methylene-dioxymethamphetamine ("ecstasy"). *Lancet* 1992;340:384-7.
- 5 Priori A, Bertolasi L, Berardelli A, Manfredi M. Acute dystonic reaction to Ecstasy [letter]. *Movement Disord* 1995; 10:353.
- 6 Dowling GP. Human deaths and toxic reactions attributed to 3,4-methylene-dioxymethamphetamine and MDEA. In: Peroutka SJ, ed. *Ecstasy: the clinical, pharmacological, and neurotoxicological effects of the drug 3,4-methylenedioxy-methamphetamine*. Norwell, MA: Kluwer, 1990:63-75.
- 7 Henry JA. Ecstasy and the dance of death. *BMJ* 1992;305: 5-6.