

Letters to the Editor

Occult CO poisoning presenting as epileptic fit

Sir,

We read with interest MV Balzan's paper on acute neurological admissions and exposure to carbon monoxide (CO).¹ In his study he screened 307 acute neurological admissions at St Luke's Hospital from December 1994 to April 1995. However, out of 39 cases presenting with epilepsy none was secondary to occult CO poisoning. In a similar study, Heckerling *et al* observed two cases of subacute CO poisoning out of 43 cases with epilepsy.² Balzan highlighted problems in diagnosing occult CO poisoning in patients with concurrent metabolic conditions. We would like to present such a case.

In October 1996 a 66-year-old man was admitted to Accident and Emergency after having fainted, lost consciousness, and sustained involuntary movements to the right side of his body accompanied by urinary incontinence. The episode, witnessed by his brother, happened on leaving the garage after going through his weekly routine of starting the car engine indoors for half an hour. The patient suffered from chronic renal failure and was on haemodialysis. He had no history of epilepsy or head injury and had stopped smoking more than 20 years previously.

On examination he was haemodynamically stable with no neurological deficit except for generalised hyperreflexia. Carboxyhaemoglobin (COHb) on admission was 14.8% (three hours after inhalation), peak COHb of 25% (by extrapolation), haemoglobin 10.3 g/dl, serum creatinine 830 µmol/l, random blood glucose 5.0 mmol/l, PaO₂ 80, pH 7.3, BE -5.7, electrocardiogram within normal limits. A diagnosis of complex partial fit secondary to acute, possibly chronic, CO poisoning was made. Treatment included prompt administration of 100% oxygen using a tight-fitting mask followed by hyperbaric oxygen therapy X 2.8 atm for two hours.

This case illustrates the fact that patients presenting to the emergency room with epilepsy may have occult CO poisoning. Furthermore, the presence of chronic metabolic conditions, such as chronic renal failure, can prevent physicians from seeking an alternative cause of fits, such as CO poisoning.

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- 1 Balzan MV, Aguis G, Galea Debono A. Carbon monoxide poisoning: easy to treat but difficult to recognise. *Postgrad Med J* 1996; 72, 470-3.
- 2 Heckerling PS, Leikin JB, Terizian CG, *et al*. Occult carbon monoxide poisoning in patients with acute neurological illness. *Clin Toxicol* 1990; 28: 29-44.

Cannabis and alcohol in stroke

Sir,

Lawson and Rees reported two transient ischaemic attacks in a 22-year-old man while smoking cannabis.¹ He consumed other drugs and subsequently developed a stroke. They point out that there are reported cases of stroke associated with heavy cannabis abuse. This phenomenon is currently under-recognised even in areas of high cannabis abuse.² However, the authors also mention that all the described cannabis-related cerebrovascular events have occurred "whilst the drug is actually being smoked". In fact, in Zachariah's second case the stroke occurred up to half an hour after smoking a marijuana cigarette.³ Cardiovascular changes (heart rate and temperature) have been shown to persist for at least this length of time.⁴ We have recently had reason to wonder whether cannabis remains a risk factor for stroke after the early effects on the cardiovascular system.

Three months before admission a 29-year-old man, who usually smoked cannabis almost daily, increased his consumption. He continued smoking 10 tobacco cigarettes per day. He denied ever consuming any other illicit drugs. Six weeks before admission he developed transient episodes of numbness in the left arm and leg, some of these occurring with witnessed facial asymmetry and lasting up to five minutes. These episodes increased in frequency to a maximum of six in one day. Although one or two occurred while smoking cannabis, the patient did not feel the events were related. He was an episodic alcohol binger and three days prior to admission, he smoked more cannabis and tobacco cigarettes. Over that weekend he consumed 24 cans of beer. On the following day he developed a dense right-sided weakness and was admitted to hospital. He was normotensive with no family history of stroke.

Computed tomography (CT) scan showed high attenuation in the right middle cerebral artery with an infarct in that territory. SPECT scan confirmed decreased perfusion in the area. Magnetic resonance angiography appearances were normal except for the absence of the right middle cerebral artery. The remaining stroke investigations (erythrocyte sedimentation rate, electrocardiogram, chest X-ray, full blood count, glucose, thrombotic tests, antinuclear antibody, VDRL, transthoracic and transoesophageal echocardiography) were all negative or normal.

Our patient's stroke was probably a result of heavy alcohol consumption prior to admission, a phenomenon well documented elsewhere.⁵ Cannabis is often viewed as a 'safe drug', but increasingly reports suggest a role in cerebrovascular disease,^{1,3} especially after heavy consumption. Its contribution to our patient's pathology is speculative but there remains a need for increased clinical awareness of this putative risk factor.

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- 4 Beaconsfield P, Ginsburg J, Rainsbury R. Marijuana smoking: cardiovascular effects in man and possible mechanisms. *N Engl J Med* 1972; 287: 209-12.
- 5 Wilkins MR, Kendall MJ. Stroke affecting young men after alcoholic binges. *BMJ* 1985; 291: 1342.

Ophthalmic complications of HIV/AIDS

Sir,

I read with great interest the recent article on ophthalmic complications of HIV/AIDS.¹ The section on treatment of CMV-retinitis, however, appears incomplete as authors described only two drugs, foscarnet and ganciclovir. A new third drug, cidofovir, was approved by the US Food and Drug Administration in 1996 and is being widely used. The drug is administered in a dose of 5 mg/kg intravenously once a week for two weeks followed by a similar dose two-weekly as maintenance therapy. The major side-effect of cidofovir is renal failure which can be avoided in the majority of cases by careful selection of patients, intravenous hydration and administration of oral probenecid. The drug has a distinct advantage over ganciclovir and foscarnet, which must be administered five to seven times a week.²

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