Clinical Medicine 2011, Vol 11, No 2: 199–200

lesson of the month (1)

Ictal asystole due to unsuspected cocaine abuse

This lesson describes a patient who had a cardiac arrest during an episode of status epilepticus provoked by a first fit. This is an exceptional sequence of events and should lead to investigation for an underlying cause. Unsuspected cocaine abuse is common and may provoke prolonged status epilepticus, particularly if there is a low seizure threshold. A toxic screen should be undertaken in all patients presenting with unexplained status epilepticus even if abuse of illicit substances is denied.

Lesson

A 26-year-old man presented to the accident and emergency (A&E) department in status epilepticus complicated on arrival by an asystolic cardiac arrest. He had felt unwell for three or four days prior to admission with fever and headache. On the morning of his admission, his girlfriend heard him 'banging about' in the bathroom, he reported that he felt dizzy and returned to the bedroom to lie down. He then started to shake and became unresponsive, at which point an emergency ambulance was called. Unfortunately, the ambulance was delayed in traffic and despite giving 20 mg of diazepam there was no resolution of his seizure activity. On arrival in A&E, approximately 40 minutes after his collapse, he was centrally cyanosed and acidotic. He had had a pulse throughout the ambulance ride but had an asystolic cardiac arrest in A&E. Cardiac output returned after one cycle of cardiopulmonary resuscitation (CPR) and he was transferred to intensive care (ITU). Treatment for bacterial and viral meningitis was commenced and he was loaded with phenytoin. After 24 hours on ITU sedation was weaned and he was well enough to be transferred to the ward. A magnetic resonance imaging (MRI) scan showed a small cavernoma in the left parietal cortex but an electroencephalogram (EEG) was normal. An electrocardiogram was unremarkable. No further relevant history was elicited and, in particular, he denied use of illicit substances. He made a full recovery and was discharged home on carbamazepine 200 mg twice daily.

He reported being unwell on his return home, feeling unsteady and dizzy and being unable to sleep. He attended A&E on two occasions when routine investigations were normal. He declined admission and it was concluded his symptoms were due to the anti-epileptic medication.

Two weeks later the patient and his partner were contacted by telephone because he had failed to attend a clinic appointment. He reported continuing to feel dizzy and tired despite reduction in the dose of anti-epileptic drugs. Ten days later he collapsed at home in the bathroom. His partner was in the next room and heard him fall. She reported that he fell to the floor and appeared vacant. He jerked several times while an ambulance was called. No resuscitation attempts were undertaken before emergency staff arrived. He had been pulseless for at least 10 minutes before ambulance staff commenced CPR which was unsuccessful.

At post mortem the cardiac examination was entirely normal and no significant abnormality in the brain was noted apart from some dilated vessels in the left posterior hemisphere. Toxicology screen, however, showed the presence of cocaine and cocaine metabolites as well as the presence of lidocaine. The level of benzoylecgonine, a cocaine metabolite was particularly high at 5.6 mg/l, the range of concentration associated with fatality being 0.7–31 mg/l.

Discussion

The abuse of illicit drugs is known to be associated with neurological complications.[1–3] Cocaine is a potent sympathomimetic and central nervous system (CNS) stimulant.[3,4] Acute toxicity is dose related and characterised by profound hypertension and tachycardia with arterial vasoconstriction and supraventricular or ventricular arrhythmias due to direct myocardial stimulation. CNS toxicity eventually causes cerebral vasoconstriction, and haemorrhagic or ischaemic stroke resulting from sympathomimetic activation.[5] Seizures are relatively uncommon following cocaine abuse but epidemiological evidence is limited.[6] While cocaine can reduce seizure threshold in patients with underlying epilepsy, seizures may occur without a previous history of epilepsy and may follow first time use of the drug.[7] They are usually single, generalised tonic-clonic fits occurring within 90 minutes of cocaine use, often associated with a severe acidosis and usually follow intravenous injection or the use of 'crack cocaine'. The European Monitoring Centre on Drugs and Drug Addiction (EMCDDA) has confirmed very high levels of cocaine use in some recreational settings and a dramatic increase in cocaine availability.[8] Cocaine is the second most used illicit drug in Europe, after cannabis, with the UK having the highest prevalence (7.7% in the 15–64 age group and 12.7% in the 15–34 age group). The regular use of cocaine is increasing, particularly in the 16–24-year-old demographic, with just 1.4% using the drug within the last year in 1996 compared to 6.6% in 2008.[9] Cocaine

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use is most common in young men who regularly drink alcohol. One in five young men who drink alcohol on three or more days a week will have used cocaine in the last year. Overall it seems likely that the acute presentation manifest in the present patient will be increasingly recognised.

Multiple seizures or status epilepticus may be observed in patients with a history of seizure disorders or patients who concomitantly abuse other drugs or those in whom the drugs are adulterated by other substances, eg lidocaine, amphetamines or quinine. In this patient there was a high concentration of lidocaine suggesting the cocaine that had been used was contaminated.

Acute cocaine use can present with severe generalised epilepsy which may be associated with asystole either due to a direct neurogenic mechanism or because of concomitant cardiac involvement. In adults with an unexplained first fit, particularly if the EEG is normal, a toxic screen should be undertaken even if the patient denies using illicit drugs. It is important to emphasise that reliance on the patient and their friends, or even the relative's, history is potentially misleading. Drug abusers tend to minimise or deny their use, behaviour and its consequence. The present case was exceptional because of the severity of the initial presentation with a first fit leading to cardiac arrest and acidosis. While this was related to cocaine it is of interest that the ingestion was apparently nasal. There was no evidence of a primary cardiac arrhythmia although the heart may well have been rendered more vulnerable by the use of cocaine. The presence of a cavernoma may have lowered the seizure threshold even though the EEG was normal. Unexplained status epilepticus, particularly if accompanied by severe acidosis or cardiac arrhythmias, may be due to unsuspected cocaine usage even if this is denied by the patient or their partner. In such circumstances a toxicology screen is a mandatory part of the investigation.

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