Intracerebral haemorrhage in previously healthy young adults

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A 31-year-old right-handed man was admitted to hospital with vomiting, severe headaches, photophobia and weakness of the left arm and leg. There was no medical or family history of note. He smoked about 20 cigarettes a day. On examination, he was drowsy but rousable to a Glasgow Coma Scale of 15/15. There was mild neck stiffness but Kernig's sign was negative. His oral body temperature on admission was 35.7°C. The radial pulse was 70 beats/min, regular and of good volume. Blood pressure was 125/60 mmHg. Examination of the nervous system confirmed severe flaccid left hemiparesis (muscle power MRC 3/5 and 0/5 in the lower and upper limbs, respectively). There was also a left upper motor neurone facial weakness. Tendon reflexes were brisk on the left side and the left plantar response was extensor. There were no sensory signs, visual field defects or dyspraxia. Language function appeared intact. Formal psychological assessment confirmed a significant deterioration in cognitive function, especially in performance items. Attention, concentration, and delayed recall were particularly severely affected. The rest of the neurological and general physical examination was unremarkable.

Haemoglobin was 14.1 g/dl and the total white blood count was $12.8 \times 10^9/l$ with 93% neutrophils. Platelet count was normal. Liver function tests and urea and electrolytes were also normal on admission. An electrocardiogram was normal. A computed tomography (CT) brain scan with contrast enhancement confirmed the presence of a large right frontal lobe haemorrhage approximately 6 cm in diameter with some mass effect. Blood was also present in the right Sylvian fissure.

The patient's headaches, photophobia and vomiting resolved in 2–3 days and his neurological signs also improved quickly. Two weeks after his stroke he had fully recovered muscle power and function of the left arm and leg. However, there was mild but progressive deterioration in his liver function. On the fourth day of admission his γ -glutamyl transpeptidase and aspartate transaminase were 377 and 105 IU/l, respectively. One week later his γ -glutamyl transpeptidase was 609 IU/l and aspartate transaminase was 152 IU/l. Other liver enzymes and serum bilirubin remained within the normal limits. A urine test suggested the cause of the intracerebral bleed. Six weeks after the stroke all liver enzymes were within the normal limits.

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Questions

- 1 What urine test suggested the cause of the patient's intracerebral bleed?
- 2 What other investigation would you ask for?
- 3 What are the possible mechanisms of intracerebral haemorrhage in this patient?
- 4 How would you explain the abnormalities of liver function?

Answers

QUESTION 1

Urine amphetamine levels. Amphetamines are excreted in the urine unchanged. The rate of excretion is dependent on the urine pH. Approximately 70% of the ingested dose is excreted in 16 hours if the urine is acidic but less than 5% if the urine is alkaline.

Amphetamines (eg, Speed) and their ring-substituted derivatives, 3,4 methylenedioxyethyl amphetamine (Ecstasy, Adam, etc) and methylenedioxyethyl amphetamine (Eve) are widely used, especially by young adults, for their mood-elevating effect and their tendency to enhance sociability. An anonymous survey of the attitudes of college students towards the recreational use of amphetamines has shown that 39% of the students in a university campus had taken the drug at least once.¹ However, despite this widespread use, mortality and severe morbidity due to these drugs is rare. Most patients die from heat stroke, rhabdomyolysis, disseminated intravascular coagulation, renal and liver failure.² Stroke has been reported only in a small number of cases. Nevertheless, amphetamines seem to be a leading cause of stroke in this patient population. In a review of 72 young patients (aged 15–45 years) who presented with non-traumatic intracerebral haemorrhage, amphetamines were found to be the cause of the stroke in five cases.³

QUESTION 2

Magnetic resonance angiography. This is a powerful non-invasive method for the diagnosis of intracerebral vascular malformations. Cases

of ruptured congenital berry aneurysms following the ingestion of amphetamine derivatives (presumably due to the severe increase in blood pressure) have been reported.⁴

QUESTION 3

The mechanism of the intracerebral haemorrhage cannot be established from the available clinical data. The patient had a normal blood pressure when first seen but it is possible that a brief sudden surge in arterial blood pressure at the peak of amphetamine blood concentration had caused the haemorrhage. This transient sympathomimetic effect of amphetamines has been documented in both experimental animals⁵ and in man.⁶ Intracerebral haemorrhage may also result from vasculitis in some amphetamine users.⁴ However, vasculitis is unlikely to have had an aetiological role in this case because of the short period between the ingestion of the drug and the stroke onset.

QUESTION 4

Liver damage may result from an idiosyncratic reaction to amphetamines. This suggests the need for regular monitoring of these patients in the first few days after amphetamine consumption.

Final diagnosis

Intracerebral haemorrhage, possibly related to amphetamine use.

Keywords: brain haemorrhage; amphetamines

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Hyperkalaemia following blood transfusion

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A 35-year-old man weighing 63 kg was presented for elective repair of an atherosclerotic thoraco-abdominal aortic aneurysm. He was a known hypertensive on oral nifedipine 20 mg 12 hourly and propranolol 20 mg 8 hourly. He had no known allergies. Routine biochemistry (Na^+ 135 mmol/l, K^+ 3.6 mmol/l, urea 5.1 mmol/l, creatinine 56 $\mu\text{mol/l}$) and haematological investigations were within normal limits. Echocardiography suggested normal left ventricular function and the electrocardiogram (ECG) was normal. An aortogram demonstrated a fusiform aneurysm extending from the descending thoracic aorta (distal to the left subclavian artery) to just distal to the renal arteries.

After anaesthetising the patient, the aneurysm was exposed using a thoraco-abdominal incision. Mannitol 0.5 g/kg and heparin 1 mg/kg were given before aortic cross-clamping. No temporary arterial shunts were used during the aortic cross-clamping and the total cross-clamped time was 65 min. On declamping, the patient developed severe hypotension (a systolic pressure of 40 mmHg), which was treated with rapid transfusion of 1 litre of crystalloids, 1 litre of colloids, 4 units of warmed CPD-A (citrate-phosphate-dextrose-adenine) stored whole blood and 10 ml of 10% calcium gluconate. Despite this, the hypotension persisted; therefore the aorta was re-clamped (supra-coeliac) to maintain the blood pressure and coronary perfusion. This was immediately followed by bradycardia, widening of QRS complexes and ventricular fibrillation (VF). The VF did not respond to two internal DC shocks of 20 J each and one DC shock of 30 J. Internal cardiac massage was started; the heart was found to be very flabby. Intravenous lignocaine (2 mg/kg) and additional doses of calcium gluconate (10 ml of 10%) and 7.5% sodium bicarbonate 100 ml produced no improvement. In view of the VF resistant to DC shock and a 'flabby' heart, hyperkalaemia was suspected. Blood transfusion was discontinued, even though the haemoglobin was 7 g/dl and the hypovolaemia was treated by infusing crystalloid and colloid solutions only. The patient's blood was analysed for serum potassium, which was 7.0 mmol/l. The blood used for transfusion was noted to be 16 days old and biochemical analysis of the unwarmed bag blood showed a pH= 6.803, standard bicarbonate content 7.8 mmol/l, base deficit 20.1 mmol and potassium 16.6 mmol/l.

The hyperkalaemia was treated with 100 ml of 50% dextrose and 20 units of actrapid insulin infused over 15 min along with frusemide 100 mg. Following this the heart recovered its tone and reverted to sinus rhythm with a systolic blood pressure of 80 mmHg. Dopamine 10 $\mu\text{g/kg/min}$ was started to augment the blood pressure with successful declamping of the aorta. At this stage the serum potassium was found to be 5.5 mmol/l, falling to 3.3 mmol/l one hour later. Subsequently only fresh blood was given. Unfortunately, the patient died of multiple organ failure 3 days after the operation in the intensive care unit.

Questions

- 1 What are the causes of hyperkalaemia during surgery?
- 2 What are the causes of hyperkalaemia during blood transfusion?

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