Hyperkalaemic ascending paralysis

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J R Soc Med 2004;**97**:330–331

In acute ascending muscle paralysis the most likely diagnosis is Guillain–Barré syndrome, but it is wise to measure the serum potassium.

CASE HISTORY

A man of 61 was admitted after five days of ascending muscle weakness, beginning after an episode of diarrhoea and back pain. He had initially noticed difficulty in walking, and the weakness had advanced to his arms and respiratory muscles. For 2 days he had been breathless at rest and for 12 hours before admission he had been unable to pass urine. On admission he was unable to walk and could not stand unaided. He did not report diplopia or dysphagia. The medical history included type 2 diabetes, complicated by ischaemic heart disease, peripheral neuropathy, nephropathy and amyotrophy. His medication was lisinopril, furosemide, aspirin, clopidogrel, atorvastatin and spironolactone. 12 weeks before admission his creatinine had been 225 mmol/L.

He was breathless at rest, but able to complete sentences. On chest examination there was poor air entry bilaterally. The pulse was 30 beats per minute and blood pressure was 140/70 mmHg, dropping to 110/55 when he sat up. There were no signs of heart failure. He had bilateral lower motor neuron facial weakness but no other detectable cranial nerve abnormalities. Tone was normal in upper and lower limbs; sensation was reduced from his mid-shins downwards, but this was a long-standing complication of his diabetes. He was weak in all muscle groups, with strength graded as 2/5 to 4+/5. The reflexes in both arms were brisk, with no clonus, but no reflexes could be elicited in his legs. Both plantar reflexes were extensor. He was in acute urinary retention, and catheterization showed a

residual volume of 650 mL. Anal sphincter tone was normal.

His electrocardiogram (ECG) on admission showed a nodal bradycardia, but T-wave morphology was normal, without widening of QRS complexes. In addition there was a loss of R-R variation. Forced vital capacity was low at 2.4 L (predicted 4.3). Serum potassium was 9.1 mmol/L, urea 41.4 mmol/L and creatinine 419 µmol/L. pH was 7.246 and serum bicarbonate 14.3 mmol/L, consistent with a metabolic acidosis.

Initial treatment was with insulin and dextrose, calcium gluconate and sodium bicarbonate but the acidosis and hyperkalaemia persisted and six hours after admission he was transferred to the intensive care unit for continuous venovenous haemofiltration. After six hours of filtration the serum potassium had decreased to 6.0 mmol/L and his weakness had completely resolved. The neuromuscular studies and lumbar puncture that had been planned were therefore not done. On discharge 4 days later, the only abnormal signs were those caused by chronic diabetic neuropathy. On follow-up he remained well. He was advised to avoid dehydration and drugs that might worsen renal function.

COMMENT

Hyperkalaemic ascending paralysis has been described several times previously but this case had unusual features. Could the true diagnosis have been Guillain–Barré syndrome (GBS), as originally suspected? The initial back pain and diarrhoeal illness, leading on to an ascending flaccid paralysis with respiratory muscle involvement but no sensory signs, would all be consistent with GBS. However, the resolution on correction of the hyperkalaemia goes against this diagnosis. Hyperreflexia is also not typical of GBS, but has been reported.¹

Autonomic disturbance is not a recognized feature of hyperkalaemic ascending paralysis, although described in other situations with a high serum potassium² and in GBS itself. The present patient may have had autonomic neuropathy, as evidenced by acute urinary retention, a postural blood pressure drop, a loss of R-R variation on the ECG and a nodal bradycardia. However, all these features could be explained by longstanding diabetes together with immobility (urinary retention), dehydration (postural drop), and a direct effect on the conduction system by hyperkalaemia (bradycardia and loss of R-R variation). Nodal bradycardia is seen in GBS³ but again resolved purely on reduction of his serum potassium. Hyperkalaemia is a rare but recognized cause of bradycardia.⁴

The mechanism of secondary hyperkalaemic paralysis remains unclear. Some workers suggest that potassium directly influences the muscle cell membrane or muscle

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fibre,^{5,6} as in episodic familial hyperkalaemic paralysis; others suggest a functional peripheral neuropathy.^{7,8} In one case, nerve conduction studies showed greatly decreased motor and sensory nerve conduction velocities and very low amplitudes, suggesting that the high serum potassium causes abnormal depolarization of the nerve membrane.

For scientific purposes it is unfortunate that nerve conduction studies were not done in the present case since they might have excluded GBS and confirmed neuronal conduction block, the likely cause of the paralysis.⁷

REFERENCES

- 1 Kuwabara S, Nakata M, Sung JY, *et al.* Hyperreflexia in axonal Guillain-Barré syndrome subsequent to *Campylobacter jejuni* enteritis. *J Neurol Sci* 2002;**199**:89–92
- 2 Perez GO, Oster JR, Pelleya R, Caralis PV, Kem DC. Hyperkalemia from single small oral doses of potassium chloride. *Nephron* 1984;**36**:270–1
- 3 Flachenecker P, Toyka KV, Reiners K. Cardiac arrhythmias in Guillain-Barré syndrome. An overview of the diagnosis of a rare but potentially life-threatening complication. *Nervenarzt* 2001;**72**:610–17
- 4 Surawicz B. Relationship between electrocardiogram and electrolytes. *Am Heart J* 1967;**73**:814
- 5 Livingstone IR, Cumming WJ. Hyperkalaemic paralysis resembling Guillain-Barré syndrome. *Lancet* 1979;**ii**:963–4
- 6 Villabona C, Rodriguez O, Joven J, *et al.* Potassium disturbances as a cause of metabolic neuromyopathy. *Intens Care Med* 1987;**13**:208–10
- 7 Evers S, Engelen A, Karsch V, Hund M. Secondary hyperkalaemic paralysis. *J Neurol Neurosurg Psychiatry* 1998;**64**:249–52
- 8 Shinotoh H, Hattori T, Kitano K, *et al.* Hyperkalaemic paralysis following traumatic rupture of the urinary bladder. *J Neurol Neurosurg Psychiatry* 1985;**48**:484–5

Hiccups and bilateral carotid artery dissection

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J R Soc Med 2004;**97**:331–332

Internal carotid artery dissection is a common cause of stroke in young patients without vascular risk factors.¹ Whilst some dissections occur in patients with known genetic predisposing factors, many are either caused by direct neck trauma or precipitated by an event involving head or neck movement.

CASE HISTORY

A man aged 38, right-handed, attended the emergency department after the sudden onset of right-sided weakness and word-finding difficulties. He had no previous history of cerebrovascular events and his only vascular risk factor was occasional cigarette smoking. The other relevant medical history was of intermittent migraine; there was no family history of connective tissue disorders. Before the event there had been no trauma, headache, vomiting, coughing or sneezing but on direct questioning the patient recalled that, five minutes before the onset of symptoms, he had experienced a violent bout of hiccups lasting one to two minutes.

On examination he was not marfanoid. He had a dense right hemiplegia and hemianaesthesia involving the face, arm, and leg, a right-sided hemianopia and neglect, severe expressive dysphasia and moderate receptive dysphasia. CT of the brain showed an area of low attenuation affecting the head of the left caudate nucleus and the lateral lentiform nucleus with cortical sparing. Since the patient had presented within a time-frame for thrombolytic therapy, he received 0.9 mg/kg intravenous tissue plasminogen activator. There were no acute complications. CT brain 24 hours after thrombolysis revealed infarct extension with cortical involvement and sulcal effacement.

Duplex ultrasound of the left internal carotid artery suggested a distal occlusion. Transcranial doppler revealed no middle cerebral artery flow on the left but normal findings on the right. Magnetic resonance angiography (Figure 1) demonstrated bilateral carotid artery dissection,

Figure 1 Axial neck magnetic resonance angiogram showing bilateral carotid arterial dissection and luminal occlusion of the left internal carotid artery, consistent with thrombus

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