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

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Hiccups and bilateral carotid artery dissection

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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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left internal carotid artery occlusion and absence of flow in the distribution of the left internal carotid artery distal to the occlusion. Echocardiography was normal with no evidence of a patent foramen ovale on contrast injection. Autoimmune and thrombophilia screens were negative. Laboratory studies of haematology and blood biochemistry, including fasting glucose, were unremarkable except for a serum cholesterol of 6 mmol/L.

24 hours after thrombolysis the patient was put on antiplatelet and lipid-lowering therapy. Anticoagulation was started one week after admission and the antiplatelet treatment was discontinued when the international normalized ratio became satisfactory. Despite the severity of his initial deficits he made a good recovery and was discharged from hospital after two months of intensive rehabilitation.

COMMENT

Arterial dissection begins with an intimal tear and usually proceeds to intramural haematoma formation. Internal carotid artery dissection is a recognized cause of stroke, the mechanism for which may be either embolism from the site of dissection or haemodynamic compromise secondary to arterial occlusion.² Some dissections occur in patients with heritable connective tissue disorders such as Ehlers–Danlos syndrome type IV and Marfan's syndrome. In other cases, dissections seem to be precipitated by direct neck trauma or events involving neck or head movement such as coughing, sneezing, vomiting, dental examination and therapeutic neck manipulation.^{3,4}

The onset of neurological symptoms was preceded in our patient by hiccups (a precipitant not previously described) and carotid dissection was bilateral, which is unusual. A hiccup consists of involuntary spasmodic contraction of the diaphragm and external intercostal muscles, which abruptly ends with glottic closure. This manoeuvre generates sudden and high negative intrathoracic pressure.⁵ Hiccups might therefore cause internal carotid artery dissection either by mechanical stretching and subsequent arterial dissection during sudden head or neck movements, or by inducing high shear stresses upon the arterial wall because of abrupt pressure changes, leading to tearing. We cannot rule out the possibility that the hiccups were effect rather than cause. They are a common initial symptom in patients with relative ischaemia into the posterior circulation; indeed, hiccups were among the symptoms in a man whose vertebral artery dissection was attributed to neck rotation during golf.⁶ However, in our patient the bilateral nature of the injury at the same level suggests that the two carotid arteries were subject to similar pathological processes and support hiccups as the cause of stroke.

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Epilepsia partialis continua stopped by insulin

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When epilepsia partialis continua develops late in life, likely causes are brain tumours, cerebral infarction and encephalitis.¹ But a benign and treatable cause should not be forgotten.

CASE HISTORY

A man aged 75 had for five days experienced tonic–clonic seizures involving the right side of the face and right upper and lower limbs at a frequency of 20–30 per day. He was conscious during and between episodes. He had noted mild weakness of the right upper and lower limbs. There was no history of fever, exposure to toxins or drugs, alcohol abuse or head injury and he had never previously had a seizure. On examination he was conscious but exhausted. He had a mild right hemiparesis including a right upper motor neuron facial paresis. Power in the right upper and lower limbs was grade 3/5. Sensory and cerebellar systems were normal, and there were no signs of meningeal irritation.

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