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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The routine biochemical tests were normal apart from a serum sodium of 132 mmol/L and a blood sugar of 19.4 mmol/L; serum osmolality was 304 mosm/kg. No ketone bodies were present in the urine. Plain and contrast CT of the brain, and MRI done later, revealed nothing of note and the cerebrospinal fluid was normal. In the emergency room he was given intravenous lorazepam followed by phenytoin sodium but the seizures continued; he was then started on midazolam by infusion. To lower the blood sugar he was given insulin (actrapid), at first in intermittent subcutaneous doses and subsequently by continuous infusion. Seizures that had persisted despite midazolam 3 mg/h ceased when the actrapid infusion brought the blood sugar below 8 mmol/L. Therefore the cause of his seizures was judged to be non-ketotic hyperglycaemia and the antiepileptic drugs were withdrawn. He was discharged on oral hypoglycaemic agents and at follow-up three months later he was symptom-free.

COMMENT

Epilepsia partialis continua (EPC) is spontaneous regular or irregular clonic muscle twitching of cerebral cortical origin, sometimes aggravated by action or sensory stimuli, confined to one part of the body, and continuing for hours, days or weeks.² The clonic jerks can affect any muscle group, the extent ranging from a single muscle or muscle group to widespread involvement. Agonists and antagonists are affected together and distal muscles are more commonly affected than proximal ones. EPC can have many causes (Box 1), and in the present patient the most likely was a cerebrovascular accident. This and a brain tumour were ruled out by CT, and encephalitis was excluded by the normal cerebrospinal fluid. When non-ketotic hyperglycaemia was discovered, the cause was in little doubt.

This is not the first report of EPC as the presenting symptom of diabetes mellitus. In a previous study, EPC led to a diagnosis of diabetes mellitus in 9 of 21 patients;³ and in a group of patients with diabetes mellitus presenting with seizures, 20% had EPC (average duration eight days, seizure frequency 15 per day).⁴ Hyperglycaemia increases the metabolism of gamma-aminobutyric acid and thereby lowers seizure threshold.⁵ Mild hyperosmolality, mild hyponatraemia and absence of ketoacidosis may also contribute.³ These factors were present in our patient.

It is particularly important to remember non-ketotic hyperglycaemia as a cause of persistent focal motor seizures

Box 1 Various aetiologies of epilepsy partialis continua

Children	
Infections	Rasmussen encephalitis Subacute measles encephalitis Viral encephalitis or meningoencephalitis Infective granuloma
Developmental	Cortical dysplasia
Genetic	Mitochondrial cytopathies such as MELAS Alper's disease
Metabolic	Non-ketotic hyperglycaemia Diabetic ketoacidosis
Cerebral tumours	Gliomatosis cerebri
Adults	
Cerebrovascular diseases	Embolic or thrombotic ischaemic stroke Cerebral venous thrombosis Cerebral haemorrhage Arteriovenous malformations
Cerebral tumours	Metastatic tumours Gliomas
Metabolic	Diabetic ketoacidosis Non-ketotic hyperglycaemia Hepatic or uraemic encephalopathy
Infections	Encephalitis Infective granuloma

MELAS = mitochondrial encephalopathy with lactic acidosis and stroke

since administration of phenytoin may do harm by worsening glycaemic control.⁶ These patients do not require long-term antiepileptic drug prophylaxis.⁷

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