Case report

Acute aphasia and hemiplegia during karate training

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In August, 1999, a 40-year-old karate expert had sudden weakness of the right arm and leg and speaking difficulties as he attempted to free himself from a neck grasp, inflicted by the flexed arm of his opponent. On arrival in the emergency room, neurological examination showed a rightsided sensorimotor hemiplegia and severe motor aphasia. The rest of the physical examination was normal. The history was unremarkable with no cardiovascular risk factors apart from smoking.

Routine laboratory tests, electrocardiography, cerebral computed tomography, and extracranial and intracranial doppler sonography were normal. B-mode ultrasound showed a weakly echogenic plaque in the left internal carotid artery (ICA) with 40% lumen reduction. Left middle cerebral artery (MCA) territorial infarction was diagnosed and intravenous thrombolytic therapy with 0.9 mg/kg recombinant tissue plasminogen activator was begun 125 min after stroke onset.

Magnetic resonance imaging (MRI) confirmed cerebral ischaemia in the territory of the superior division of the left MCA, but showed no evidence of carotid artery dissection (CAD). Microembolic signals were not detected during or after thrombolysis. Cholesterol (9.5 mmol/L) and lowdensity lipoproteins (6.6 mmol/L) were raised; homocysteine, proteins C and S, and antithrombin III were normal, and there were no antibodies to phospholipids. Transoesophageal echocardiography and 24-h electrocardiographic monitoring showed no source of cardiac embolism. A new three-dimensional (3-D) ultrasonographic technique for compounded B-mode imaging¹ detected a punched-out defect at the centre of the plaque surface in the left ICA (figure). Ultrasonographic analysis of plaque surface motion showed a low maximal discrepant surface velocity of 1.2 mm/s.

Following thrombolysis, the patient regained full strength of his right arm and leg. The aphasia also showed pronounced improvement. He was placed on secondary prophylaxis with clopidogrel, given cholesterol-lowering treatment, and referred for ambulatory logopaedic therapy.

The immediate temporal relation between the powerful neck grip during karate training and the acute onset of aphasia and hemiplegia suggested traumatic CAD. However, common symptoms (headache, neck or facial pain) and signs (partial Horner's syndrome) were lacking and MRI techniques, highly sensitive and specific for detection of CAD,² showed no evidence of an intramural

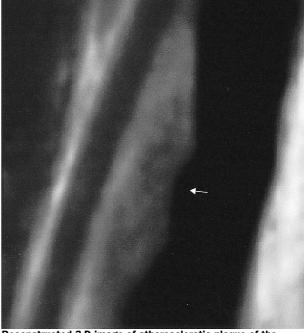
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Reconstructed 3-D image of atherosclerotic plaque of the proximal ICA

B-mode ultrasound, compounded from multiple, arbitrary imaging planes was used; surface defect is shown (arrow)

haematoma. Other causes of stroke were excluded.

Traumatic plaque rupture was the most likely aetiology of the cerebral infarction. Atherosclerotic plaques in the carotid bifurcation with less than 50% lumen narrowing are unlikely to spontaneously embolise and cause stroke. Since weak plaque echogenicity is associated with a high lipid content,³ the plaque is soft and pliable, and therefore susceptible to external compression. The unusual punched-out defect of the plaque surface, visualised with compounded 3-D ultrasonography, was not typical of a plaque ulceration. The results of plaque surface motion analysis⁴ were also not suggestive of symptomatic carotid artery disease. Microembolic signals, often seen in symptomatic plaques which continue to embolise after causing infarction,⁵ were not detected with transcranial doppler monitoring.

Traumatic plaque rupture should be considered in patients who have a stroke while engaged in sports such as karate, judo, wrestling, rugby, or football. As asymptomatic carotid artery stenosis is common, screening for this disease should be considered in those with risk factors participating in contact sports.

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