Carotid endarterectomy for non-hemispheric cerebrovascular symptoms: an unusual indication

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We report on an 80-year-old man who presented with non-hemispheric cerebrovascular symptoms in the form of daily multiple syncope. The left common carotid artery, its two main divisions, and the right vertebral artery were completely occluded. There was high-grade stenosis in the right carotid artery (82%) and left vertebral artery (60%). After excluding other causes of syncope such as postural hypotension, hypoglycaemia, cardiac arrhythymia, and epileptic seizure, a diagnosis of global ischaemia was made. The patient subsequently underwent carotid endarterectomy and the symptoms were relieved. This case represents an unusual indication for carotid endarterectomy.

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Introduction

Non-hemispheric cerebrovascular symptoms or vertebrobasilar insufficiency remain a difficult clinical problem. These symptoms are often difficult to assess and their presentation ranges from dizzy spells, syncope, and drop attacks, to specific cranial nerve deficit. The cause of vertebrobasilar insufficiency is thought to be posterior circulatory insufficiency and a low-flow state. The aim of the treatment is to increase cerebral perfusion globally. The choice of surgery and its indication, however, are still controversial because patient selection remains the most crucial determining factor in relation to the outcome. Furthermore, the outcome of carotid endarterectomy for circulatory insufficiency is generally less satisfactory than that for the prevention of an embolism.[1-3]

Case report

An 80-year-old man presented to the Department of Geriatrics at the Princess Margaret Hospital in early 1998. He had a history of ischaemic heart disease, myocardial infarction, hypothyroidism, gouty arthritis, and gastro-intestinal bleeding induced by non-steroidal anti-inflammatory drugs. At presentation, he had attacks of syncope, which had progressively deteriorated over a 3-year period. The syncopal attacks usually occurred after meals and lasted up to 10 minutes. There were signs of postural hypotension but no signs of neurological deficit.

Results of Holter monitoring and electroencephalography (EEG) were normal, and the computed tomography (CT) scan showed small infarcts within the left side of the corona radiata. Repeated Holter monitoring showed only sinus bradycardia, which was related to the patient's usage of beta-adrenoceptor blocking drugs. His haemodynamic state improved after the dosages of beta-adrenoceptor blocking drugs and nitrates had been reduced.

The patient was subsequently admitted to the Princess Margaret Hospital for repeated episodes of daily syncope. There was no postural hypotension, and the blood pressure was normal during the syncopal attacks. The tilt table test (to detect postural hypotension) gave normal results. Hypoglycaemia was excluded by performing the Haemoglucostix test (Boehringer, Mannheim, Germany). The patient had no seizures, but faecal incontinence, dribbling of saliva, and a transient period of drowsiness after the syncopal attack were occasionally reported. Thus, despite the normal

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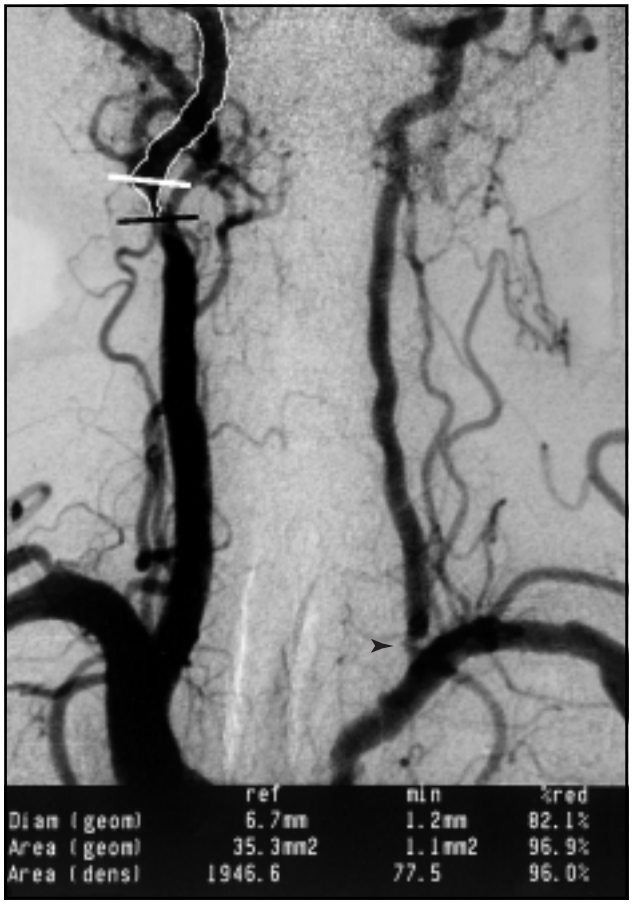

Fig. Carotid angiogram showing tight stenosis in the proximal right internal carotid artery
The left vertebral artery has a 60% narrowing near its origin (arrowhead); the left common carotid artery, its two div-isions, and the right vertebral artery are occluded

EEG results, anticonvulsant therapy was started. After being discharged home, the patient continued to experience disabling syncope several times a day despite taking phenytoin.

In June 1998, carotid Doppler ultrasonography suggested that there was a severe bilateral carotid lesion. Haemodynamic insufficiency was diagnosed to be the most likely cause of the patient's repeated syncopal attacks. He was referred to the surgical unit at the Kwong Wah Hospital for surgical investigation. As the symptoms were unusual, the patient was admitted to hospital for a period of observation, which confirmed the syncopal attacks. Carotid angiography showed that the patient had multivessel disease (Fig): the left common carotid artery and its two divisions were completely occluded; the right vertebral artery was also occluded. The right internal carotid artery and external carotid artery were affected by a reduction in diameter of 82% and 63%, respectively. The left vertebral artery also showed 60% stenosis at its origin. There was no obvious intracerebral abnormality. However, the circle of Willis could not be well visualised in the angiogram or in the scan from subsequent magnetic resonance imaging. Global hypoperfusion became the definitive diagnosis. Although surgery in the presence of such extensive disease and co-morbidity carries a high risk of stroke, the patient still opted for carotid endarterectomy.

Right carotid endarterectomy was performed on the 20 August 1998. Intravenous heparin 4000 U was given during the operation. There was no discernible intra-arterial pressure waveform and the stump pressure was low (20 mm Hg). In view of the poor backflow, a Javid inlay shunt was inserted to ensure cerebral protection. The operation and postoperative recovery were uneventful. The patient was discharged home 5 days after the operation was and given low-dose aspirin.

During follow-up, the patient was generally asymptomatic. However, he experienced two episodes of dizziness without actual loss of consciousness 3 months after the operation. One of these episodes was associated with a transient inability to speak. On this occasion, he was readmitted to the Kwong Wah Hospital and the CT scan of the brain was found to be normal. Magnetic resonance angiography was subsequently performed and showed a 40% narrowing at the site of the endarterectomy. This problem was treated conservatively; the patient has been otherwise completely relieved of the symptoms up to the time of writing.

Discussion

Both transient ischaemic attack and stroke associated with carotid disease are generally considered to be caused by embolism due to a thrombogenic atheromatous plaque at the origin of the internal carotid artery. Indications for carotid endarterectomy to prevent stroke have been well defined for the majority of affected patients.[4,5] However, for a subset of patients, disease symptoms may be caused by haemodynamic failure. The concept of ischaemic penumbra, which describes the maintenance of cellular viability but with impaired cellular function, is now widely accepted. In this patient, the multiple episodes of a brief loss of consciousness without sustaining a stroke fitted the clinical picture of ischaemic penumbra. The questions at issue are how can cerebral hypoperfusion be recognised and when does it become an indication for carotid endarterectomy.

Patients with non-hemispheric cerebrovascular symptoms or vertebrobasilar insufficiency are often difficult to evaluate clinically, because the symptoms can range from giddiness and syncope to specific

cranial nerve dysfunction. For this patient, taking a thorough history and performing thorough investigations ruled out postural hypotension as the cause of the symptoms. He was not taking any vasoactive drugs and he was not hypoglycaemic. After the bilateral carotid lesions were detected by duplex scanning, a series of special investigations and a therapeutic trial of anticonvulsant therapy ruled out cardiac arrhythmia and epileptic seizure as the underlying cause. Although positron emission tomography, xenon CT, and single-photon emission CT (SPECT) can produce quantifiable measurements of cerebral flow, the first two methods were not available and performing SPECT was not appropriate, given the presence of the deteriorating and distressing symptoms. In addition, the severity of the arterial disease warranted carotid endarterectomy, even though the patient was asymptomatic.

The results of carotid endarterectomy for global ischaemia have been more variable than those of carotid endarterectomy for the management of a hemispheric neurological event. An improvement of up to 80% has been reported; however, 44% of patients had persistent preoperative symptoms.[3-5] It seems logical that the results of surgery would depend on the collateral blood flow through the circle of Willis. The high failure rate may be related to the fact that only slightly more than 50% of people have a complete circle of Wills.[6] Harward et al[7] found that when at least one posterior communicating artery was visible angiographically, 92% of patients subsequently became asymptomatic or had improved carotid haemodynamics. However, two thirds of the patients whose posterior communicating artery was not visible also became asymptomatic or had improved carotid haemodynamics following carotid endarterectomy. Interestingly, the carotid haemodynamic state was improved in 12 patients after vertebral artery reconstruction had been performed, despite the fact that only one third of these patients had an angiographically visible posterior communicating artery.[8] Patients who have hypoperfusion have a higher risk of experiencing postoperative intracerebral hemorrhage and postendarterectomy seizure, which may be related to reactive hyperperfusion.[9,10]

The improvement in the condition of this patient was satisfactory despite the two episodes of dizziness. The transient inability to speak suggested that a contralateral hemispheric event had occurred. Since the left carotid artery was completely occluded, this event would not have been related or amenable to surgery.

In conclusion, symptomatic global hypoperfusion should be considered for elderly patients who have frequent syncopal attacks. The diagnosis may be difficult and is often established by excluding other more common causes. Vascular reconstruction should be offered in the presence of multivessel disease and carotid endarterectomy can provide worthwhile improvements in well-selected patients.

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