Carotid artery disease: clinical features and management

A Ross Naylor

Abstract

The most common single cause of ischaemic carotid territory stroke is thromboembolism from stenoses in the extracranial internal carotid artery (ICA). In the majority, embolism is preceded by an acute change in plaque morphology predisposing the patient to overlying thrombus formation and embolization. The management of patients with carotid artery disease mandates risk factor modification, antiplatelet and statin therapy in everyone. There is grade A, level I evidence that recently symptomatic patients with 50–99% NASCET stenoses gain significant benefit from carotid endarterectomy (CEA), despite a small risk of perioperative stroke. Maximum benefit is conferred if the patient undergoes surgery as soon as possible after onset of symptoms. The management of patients with asymptomatic disease remains controversial. The 2018 European Society for Vascular Surgery (ESVS) carotid guidelines now advise that asymptomatic patients with a 60–99% stenosis who have one or more clinical/imaging features that might make them at higher risk for stroke on medical therapy should be considered for CEA, with the remainder being treated medically. The 2018 ESVS carotid guidelines also advise that carotid artery stenting may be an alternative to CEA in 'average risk' symptomatic and asymptomatic patients, although CEA is still the preferred option when treating patients within 14 days of symptom onset.

Keywords Carotid endarterectomy; carotid stenting; stenosis; stroke; thromboembolism

Definitions

Stroke is defined as a focal (occasionally global) loss of cerebral function lasting >24 hours and which is found to have a vascular cause. A transient ischaemic attack (TIA) has a similar definition, but the deficit lasts <24 hours. The term 'crescendo' TIAs is used when the patient suffers repeated neurological events with complete recovery in between. There is no strict definition of this phenomenon, but most clinicians would use this term if the patient suffered more than three TIAs within a 7-day period. Stroke in evolution describes the clinical situation where there is a progressive worsening in the neurological deficit interspersed with transient improvements, but no complete recovery.

Epidemiology of stroke

Approximately 150,000 first-ever strokes occur in the United Kingdom each year. The incidence of stroke is 2:1000, while that of TIA is 0.5:1000. The incidence of stroke/TIA increases significantly with age.[1] Overall, the 20-year risk for a 45-year-old male subject is 3%, but this increases to 25% for a 40-year risk.

Aetiology of stroke

Eighty per cent of strokes are ischaemic and 20% haemorrhagic. About 50% of carotid territory ischaemic strokes follow embolism from internal carotid artery (ICA) stenoses (Figure 1). Intracranial small vessel disease accounts for 25% of strokes, cardiogenic brain embolism (15%) whereas haematological (e.g. polycythaemia, sickle cell disease, myeloma, thrombocytosis) and non-atheromatous disorders (e.g. dissection, fibromuscular dysplasia, radiation arteritis, Takayasu arteritis, giant cell arteritis, aneurysm) account for 5%, respectively. Risk factors include: increasing age, smoking, hypertension, ischaemic heart disease, TIA, diabetes, peripheral vascular disease, hyperfibrinogenaemia and hypercholesterolaemia. Only 15% of stroke patients will suffer a preceding TIA.[2]

Pathology of stroke

The origin of the ICA is prone to atherosclerosis, largely because this is an area of low shear stress. Most plaques remain asymptomatic, but some undergo acute disruption with thrombus formation and secondary thromboembolism (Figure 2). The trigger for causing the acute change in plaque morphology is unknown, but may be related to increased expression of matrix metalloproteinases.

Clinical features

Patients presenting with stroke/TIA require expedited assessment as the highest risk of stroke is within the next 7 days. The history will provide useful information as to whether the event involved the vertebrobasilar or carotid territories.

Carotid symptoms are normally classified as hemispheric or retinal. Hemispheric symptoms include unilateral weakness/numbness of face, arm or leg and/or evidence of higher cortical dysfunction (dysphasia, visuospatial neglect). Retinal TIAs comprise transient monocular blindness (amaurosis fugax). Permanent monocular visual loss follows central retinal artery occlusion and is analogous to a stroke. On rare occasions, symptoms can be precipitated by exercise or emerging from a hot bath. In these situations the clinician should be suspicious that the patient is suffering haemodynamic TIAs.

Typical vertebrobasilar symptoms include: bilateral motor/sensory signs, dysarthria, bilateral visual loss, gait and stance problems, nystagmus and homonymous hemianopia. However, 10% of vertebrobasilar events will present with unilateral hemispheric signs and differentiation from carotid territory events can be difficult.

There is controversy over the significance of 'non-hemispheric' symptoms, including isolated vertigo, isolated diplopia, isolated dizziness, pre-syncope and syncope. Although some advocate surgery, there is no evidence that this either improves symptoms or reduces the late risk of stroke. For example, the majority of patients with dizziness precipitated by lateral head movements (often labelled vertebrobasilar TIAs) have benign positional vertigo. In general, patients with 'non-hemispheric'

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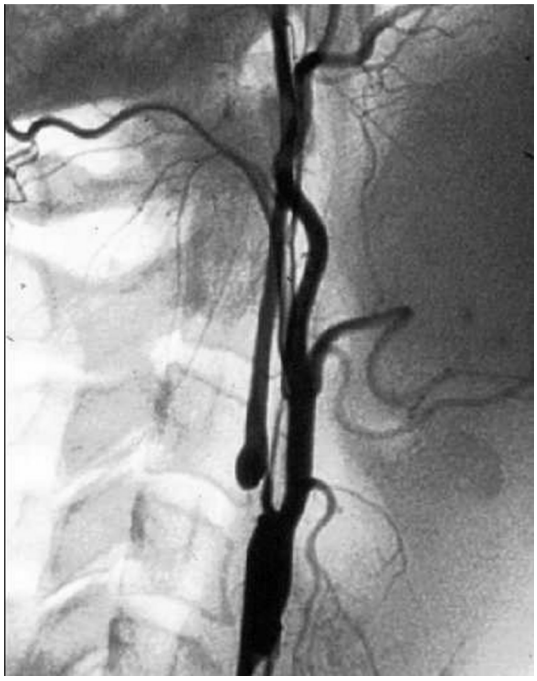

Figure 1 Intra-arterial digital subtraction angiogram showing a severe stenosis at the origin of the internal carotid artery. Evidence suggests that these plaques are liable to acute disruption with overlying thrombus formation. Embolization of the thrombus then causes a stroke.

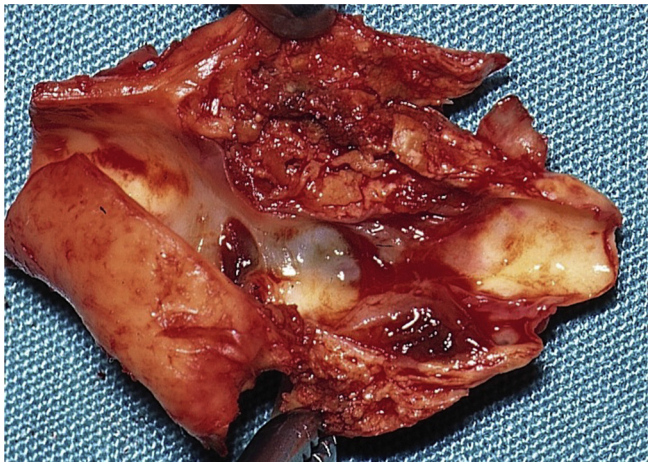

Figure 2 Resected endarterectomy specimen. There is a severe stenosis containing soft, yellow atheromatous debris. Immediately proximal to the stenosis is an ulcer. Immediately distal to the stenosis is recent thrombus.

symptoms should not be considered to have suffered a TIA unless they are associated with other more classical carotid or vertebrobasilar symptoms.

Investigation

Most centres offer single-visit clinics where the patient is seen by a specialist in cerebrovascular disease. Comprehensive risk factor review and imaging of the brain and carotid arteries is undertaken. Carotid angiography is now reserved for rare cases, largely because of the 1–2% risk of procedural stroke. The first-line non-invasive investigation is Duplex ultrasound (DU). An HTA meta-analysis showed DU to have a sensitivity of 89% and a specificity of 84% for diagnosing a 70–99% stenosis (Figure 3). This compares with 77% and 95% for CT angiography (CTA), 88% and 84% for conventional MR angiography (MRA) and 94% and 93% for contrast enhanced MR angiography (CEMRA). Sensitivity and specificity is much less for diagnosing 50–69% stenoses.[3]

When using any of these imaging modalities, it is essential that the measurement method is known. The European Carotid Surgery Trial (ECST) measurement method compares the residual luminal diameter against a 'guestimate' of the diameter of the carotid bulb at the level of the stenosis. The North American Symptomatic Carotid Endarterectomy Trial (NASCET) measurement method compares the residual luminal diameter against the diameter of the ICA about 1–2 cm above the stenosis. The two measurement methods do not give the same results. A 50% NASCET stenosis is broadly equivalent to a 75% ECST lesion, while a 70% NASCET stenosis approximates to an 85% ECST stenosis.[4] This anomaly accounts for confusion regarding whether surgeons should use a 50% or 70% threshold for intervening.

Most carotid endarterectomy (CEA) patients can safely undergo surgery on the basis of DU alone. Although the HTA found DU to be the most practical method for evaluating carotid stenosis, it did suggest that it would be preferable to undertake corroborative imaging with MRA, CTA or CEMRA where possible.[3] This is so that patients do not undergo inappropriate surgery. However, this can introduce unacceptable delays in treating recently symptomatic patients and the HTA advise that a second DU examination performed by a different operator is acceptable. By contrast, carotid artery surgery (CAS) cannot be performed on the basis of DU alone and some other form of corroborative imaging is required in order to evaluate the status of the aortic arch, the presence of tortuosity and calcification in the common carotid artery (CCA) and ICA, the status of the distal ICA (in order to see which embolic protection device should be used), and ascertaining integrity of the circle of Willis. This information can generally be provided by CEMRA or multi-slice CTA.

TIA patients should undergo a chest X-ray (cardiomegaly, lung tumour). An ECG will provide information on arrhythmias, left ventricular hypertrophy and the presence of ischaemic heart

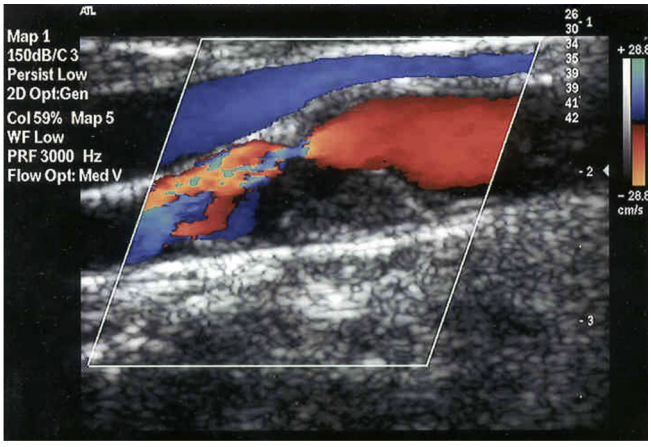

Figure 3 Duplex ultrasound image showing a severe stenosis within the ICA (arrow). The degree of stenosis is determined by morphological measurements and/or by assessment of increases in peak systolic or end-diastolic velocities across the area of narrowing.

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