Carotid artery disease: knowing the numbers

Abstract

Ischaemic stroke and transient ischaemic attack are of particular interest to the vascular surgeon as over one-third of all strokes are caused by thromboembolism from a stenotic carotid artery, making carotid artery stenosis the leading cause of stroke. If detected early, stenosis can be managed medically, surgically or endovascularly. However, treatment decisions depend on the timing of the transient ischaemic attack and the degree of stenosis, and must be balanced against procedural risk. This article discusses the evidence outlining the epidemiology, measurement and surgical management of carotid artery stenosis that inform national guidelines. Vascular and non-vascular trainees should understand these guidelines because of the potentially debilitating or fatal consequences of untreated carotid stenosis.

Key words: Carotid; Endarterectomy; Stenting; Stroke; Surgery; Transient ischaemic attack; Vascular

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Adam Heyes[1]

Alexander Crichton[2]

Sriram Rajagopalan[3,4]

Author details can be found at the end of this article

Correspondence to:
Adam Heyes;
adam.heyes@nhs.net

Introduction

Over 100 000 people experience a stroke in the UK every year (National Institute for Health and Care Excellence, 2019) and this figure is predicted to rise by 27% by 2047 (Wafa et al, 2020). Approximately 85% of strokes are ischaemic, with the remaining 15% being haemorrhagic. Around 80% of ischaemic strokes will affect the carotid territory, with 20% affecting the vertebrobasilar system (Naylor, 2015). Of the carotid territory ischaemic strokes, 50% result from thromboembolism from the internal carotid artery, while 25% are lacunar, 20% cardioembolic and 5% miscellaneous (Naylor, 2015). Atherosclerosis of the cervical internal carotid artery is thus the leading cause of all strokes, accounting for approximately 34% of cases. Stroke caused by carotid stenosis can be prevented with prompt intervention but this carries its own risks, so the decision to treat must be balanced against conservative management. Treatment of stroke in the UK is largely based on the Royal College of Physicians 2016 National Clinical Guideline for Stroke (Royal College of Physicians, 2016). This review examines the evidence supporting surgical and endovascular intervention in patients with carotid artery stenosis mentioned in the Royal College of Physicians (2016) guidelines. All patients with symptomatic stenosis should also be placed on best medical therapy, but the evidence for this is beyond the scope of this review.

Definition

Carotid artery stenosis is defined as a reduction in the luminal diameter of the common carotid artery or its divisions as a result of build-up of atheromatous plaque. According to the 2016 European guidelines on cardiovascular disease prevention, a carotid intima media thickness of >1.0 mm is considered abnormal (Piepoli et al, 2016).

Pathophysiology

Physical or metabolic endothelial injury triggers an inflammatory cascade that results in lipid accumulation and smooth muscle proliferation, forming an atherosclerotic plaque. This most commonly occurs at the bifurcation of the common carotid artery as a result of the abnormal shear stress on the wall caused by the turbulent blood flow (Dhawan et al, 2010). The plaque can extend into the lumen of the internal carotid artery causing narrowing

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and disrupting normal laminar flow. Progressive narrowing leads to stenosis and, if left untreated, sub-plaque haemorrhage, plaque rupture, thrombosis and embolisation. Embolic fragments compromise cerebral perfusion, manifesting as sudden onset neurological symptoms in the form of transient ischaemic attacks or stroke.

Presentation

The majority of patients with carotid artery stenosis present to clinicians with one or more transient ischaemic attacks or an ischaemic stroke. Transient ischaemic attacks in the carotid territory can present with sudden onset, temporary symptoms such as unilateral muscle weakness, loss of vision (amaurosis fugax), dysarthria or dysphasia. A minority of patients experience no symptoms and are detected after auscultation of a carotid bruit or as an incidental radiological finding during routine dental care, cerebrovascular or cardiovascular disease or trauma. While patients with a carotid bruit have a higher risk of cerebrovascular disease (Pickett et al, 2010) and auscultation of carotid bruit has a reasonably high specificity, it has a low sensitivity and so should not negate the requirement for ultrasound assessment of the carotid artery in those without a bruit. There is also no relationship between the degree of carotid stenosis and the likelihood of a carotid bruit (McColgan et al, 2012).

Epidemiology

A meta-analysis of 59 population-based studies estimated the global prevalence of carotid stenosis ≥50% to be 1.5% (Song et al, 2020). This prevalence increases with age from 30–79 years and is significantly higher in men than women (Song et al, 2020). Smoking significantly increases the prevalence of internal carotid artery stenosis, with smokers being 2.3 times more likely to develop moderate stenosis and 3 times more likely to develop severe stenosis than non-smokers (de Weerd et al, 2014). Population screening shows that approximately 5% of current male smokers over the age of 65 years have an internal carotid artery stenosis >50% (Högberg et al, 2014). As a result, smokers are 1.6 times more likely to experience an ischaemic stroke than non-smokers (Pan et al, 2019). Conversely, individuals who engage in high levels of physical activity have a 25% lower risk of stroke than less active individuals (Lee et al, 2003). Total cholesterol:high density lipoprotein ratio, diabetes mellitus, systolic and diastolic blood pressure level and a history of previous vascular disease (coronary and/or cerebrovascular disease) are also predictors of moderate and severe carotid stenosis (de Weerd et al, 2014).

Measurement of stenosis

Duplex ultrasound is the first-line imaging modality to measure stenosis because of its accessibility, low cost, reliability and ability to measure flow velocity. Computed tomography angiography (Figure 1) and magnetic resonance angiography have the benefit of simultaneously capturing the aortic arch, aortic trunks, distal internal carotid artery and cerebral circulation, details of which are required before intervention. Meta-analysis has shown that all three modalities are equivalent at detecting significant stenosis (Wardlaw et al, 2006). The primary method for measuring the percentage of internal carotid artery stenosis in the UK has been adopted from the North American Symptomatic Carotid Endarterectomy Trial (Figure 2). This method compares the residual luminal diameter at the most stenotic section of the artery with the diameter of the normal internal carotid artery distal to the stenosis. Using this measurement, 50–69% stenosis is classed as moderate and 70–99% stenosis is classed as severe. Guidelines state that carotid endarterectomy duplex ultrasound findings should be corroborated by computed tomography angiography or magnetic resonance angiography or, if centres rely on duplex ultrasound, a second scan should be performed by a different operator. If endovascular intervention is to be considered, follow up with computed tomography angiography or magnetic resonance angiography is recommended to assess the carotid, as well as aortic, extra and intracranial arterial anatomy (Naylor et al, 2018).

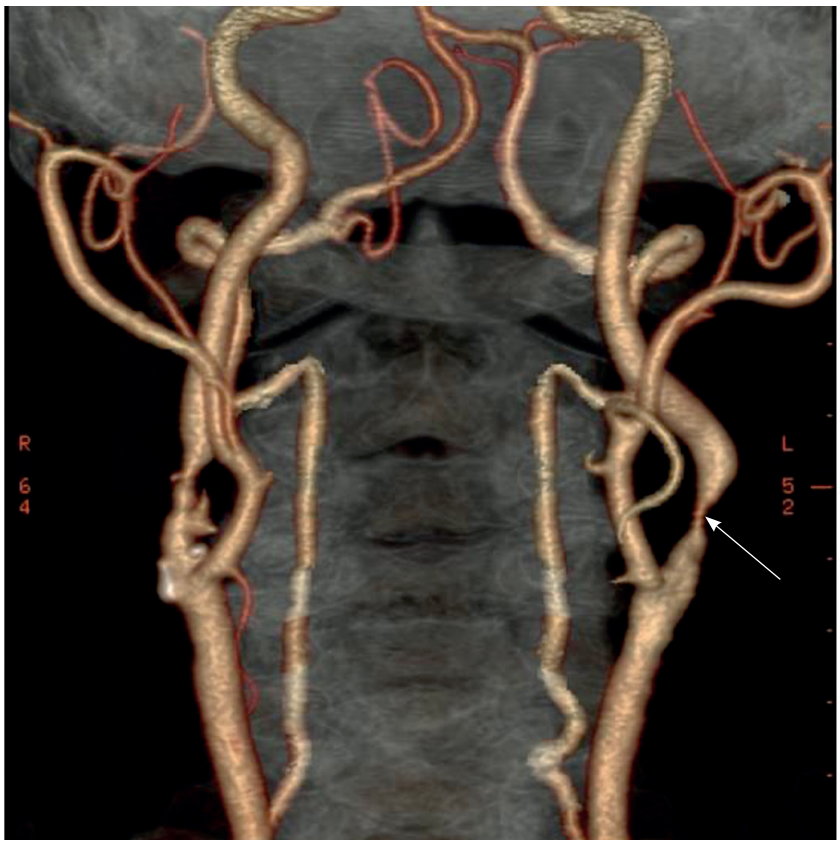

Figure 1. Computed tomography angiography demonstrating a markedly narrowed lumen of the left internal carotid artery (arrow).

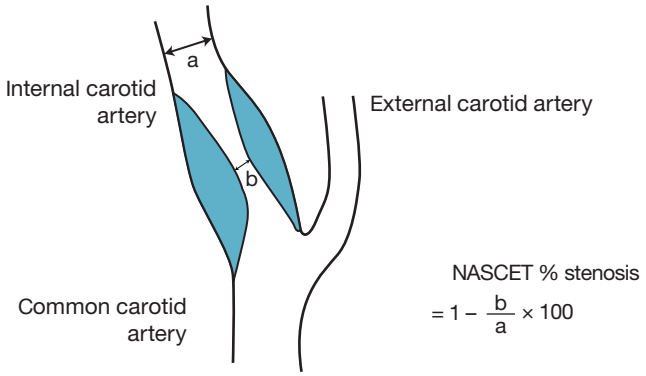

Figure 2. North American Symptomatic Carotid Endarterectomy Trial (NASCET) method of calculating internal carotid artery stenosis. Adapted from Oates et al (2009).

Best medical therapy

The Royal College of Physicians (2016) guidelines recommend that patients with a non-disabling stroke or transient ischaemic attack should be given medical therapy for secondary prevention of further vascular events. This includes addressing lifestyle factors (diet, exercise, alcohol consumption, smoking), and medications including clopidogrel (300 mg loading, 75 mg daily), atorvastatin (20–80 mg daily) and antihypertensive therapy. Patients in atrial fibrillation should also be anticoagulated as soon as haemorrhage is excluded, if this is not contraindicated. Medical therapy alone has been shown to be inadequate in preventing disease progression in all patients. Conrad et al (2013) demonstrated that medical therapy alone fails to prevent disease progression in 40% of patients and fails to prevent the development of ipsilateral neurology in 12% of patients with moderate (50–69%) stenosis over 5 years. Therefore, Royal College of Physicians (2016) guidelines recommend that all patients should also receive carotid imaging within 24 hours.

Revascularisation

Patients with a non-disabling carotid territory stroke or transient ischaemic attack should be considered for carotid artery revascularisation by carotid endarterectomy or angioplasty with or without stenting if the symptomatic internal carotid artery has a stenosis between 50 and 99% (Royal College of Physicians, 2016). Pooled analysis of the European Carotid Surgery Trial, Veterans Affairs trial 309 (VA309) and North American Symptomatic Carotid Endarterectomy Trial showed that carotid endarterectomy reduced the 5-year absolute risk of ipsilateral ischaemic stroke by 4.6% in individuals with 50–69% stenosis and 16.0% in those with 70–99% stenosis, but was of no significant benefit in patients with near-occlusion of the internal carotid artery (Rothwell et al, 2003). Naylor et al (2018) stated that patients with chronic near-occlusion of the internal carotid artery can be considered for carotid endarterectomy if there are recurrent ipsilateral symptoms despite best medical therapy and following multidisciplinary discussion.

The North American Symptomatic Carotid Endarterectomy Trial showed a number needed to treat of 6 in patients with 70–99% stenosis and 15 in those with 50–69% stenosis. Carotid endarterectomy confers maximum benefit if performed <14 days following stroke or transient ischaemic attack with benefit falling rapidly with increasing delay (Rothwell et al, 2004). Surgical delay of >3 months has been shown to bring about net harm in patients with moderate stenosis (50–69%). This is balanced by a reduction in postoperative 30-day risk of death or stroke risk with surgical delay. The 30-day risk of stroke or death is 2.4% if performed <48 hours of symptom onset, 1.8% at 3–7 days, 0.8% at 8–14 days and 0.7% >14 days (Sharpe et al, 2013). However, carotid endarterectomy is not without its drawbacks: dissection of the carotid artery carries the inherent risk of injury to the surrounding structures, namely the cranial nerves. The risk of cranial nerve injury during surgery is low (Table 1) but can be significantly disabling to those affected.

Alternatively, carotid artery stenosis can be managed endovascularly with carotid artery stenting. However, meta-analysis of four randomised control trials showed that patients >70 years of age treated with endovascular stenting or angioplasty are over twice as likely to suffer perioperative stroke or death than those treated with carotid endarterectomy (Howard et al, 2016). Endovascular therapy is also associated with a 15.2% 5-year cumulative risk of procedure-related non-disabling strokes compared to 9.4% in patients who undergo carotid endarterectomy (Bonati et al, 2015). Despite this, endovascular therapy is associated with a 56% lower incidence of myocardial infarct and 92% lower incidence of cranial nerve palsy in the perioperative period compared to carotid endarterectomy (Bonati et al, 2012). Although it is not a first-line treatment, carotid artery stenting is an appropriate alternative in patients deemed too high risk for open surgery or at increased risk of ipsilateral stroke.

Incidentally-detected carotid stenosis should not be treated until the index event has occurred. However, there are cases when it might necessitate treatment of asymptomatic disease if the patient is undergoing high-risk surgery that in itself could cause them to have a perioperative stroke, such as in cardiothoracic cases. These patients should be discussed in a multidisciplinary setting to evaluate the risks and benefits of undergoing prophylactic carotid endarterectomy.

Table 1. Risk of cranial nerve injury following carotid endarterectomy

Cranial nerve	Injury risk
Recurrent laryngeal – vagus (CN X)	4.2%
Hypoglossal (CN XII)	3.8%
Mandibular branch of facial (CN VII)	1.6%
Glossopharyngeal (CN IX)	0.2%
Spinal accessory (CN XI)	0.2%

From Kakisis et al (2017)

Key points

- All patients with carotid stenosis should receive secondary prevention as per Royal College of Physicians guidelines, but this alone is not enough to slow disease progression.
- Patients with symptomatic internal carotid artery stenosis between 50–99% (North American Symptomatic Carotid Endarterectomy Trial criteria) should be considered for revascularisation with either carotid endarterectomy or stenting.
- Carotid endarterectomy carries a lower risk of post-intervention stroke than carotid artery stenting.
- Carotid endarterectomy confers maximum benefit if performed <14 days after stroke or transient ischaemic attack.
- Carotid artery stenting should be considered in patients who are high risk for surgery, re-stenosis or with hostile neck anatomy.
- Patients with carotid occlusion >99% (North American Symptomatic Carotid Endarterectomy Trial criteria) should not be considered for revascularisation.

Conclusions

Intervention in patients with carotid artery stenosis is complex and driven by numbers. Although surgical and endovascular treatments are available, it is important to realise when a given treatment may do more harm than good. The Royal College of Physicians guidelines provide clear, concise recommendations on the management of carotid artery stenosis based on a wealth of clinical data. These guidelines should form the foundation of any multidisciplinary decision on the management of this condition.

Author details

[1]Department of General Surgery, Great Western Hospital, Great Western Hospitals NHS Foundation Trust, Swindon, UK

[2]Department of Vascular Surgery, Russells Hall Hospital, The Dudley Group NHS Foundation Trust, Dudley, UK

[3]Department of Vascular Surgery, Royal Stoke University Hospital, University Hospitals of North Midlands NHS Trust, Stoke-on-Trent, UK

[4]Department of Undergraduate Medical Education, Keele University School of Medicine, Keele, UK

Conflicts of interest

The authors declare that there are no conflicts of interest.

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