Surgery Versus Stenting in Symptomatic Patients

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KEYWORDS

• Symptomatic carotid artery stenosis • Carotid artery stenting • Carotid endarterectomy • Stroke

KEY POINTS

- Carotid artery stenosis is an important cause of ischemic stroke, which carries significant social and economic burden.
- Revascularization with carotid endarterectomy (CEA) decreases the incidence of recurrent stroke in patients with symptomatic carotid stenosis.
- The technique of carotid artery stenting (CAS) has evolved over the past several decades with improvement in patient selection, adjunctive medical therapy, stent design, and embolic protection devices.
- CAS has been demonstrated to be equivalent to CEA in randomized, controlled, prospective clinical trials when considering the composite endpoint of death, myocardial infarction, and ipsilateral stroke in both standard-risk and high-risk patients.
- The choice of revascularization modality (CEA vs CAS) should be an individualized, patient-specific decision based on comorbidities, target vessel and plaque characteristics, and patient preference.

INTRODUCTION

Stroke is the fourth leading cause of death in the United States. It is estimated to account for close to 130,000 deaths per year with a case fatality rate of approximately 16%.[1,2] Moreover, stroke continues to be associated with significant morbidity and economic costs. From the Framingham Heart Study population, it is estimated that 16% of stroke patients are subsequently institutionalized; 20% are dependent for mobility, and 31% are dependent for self-care.[3] In the United States, stroke is associated with an annual economic burden of 312.6 billion dollars in combined direct (eg, medical) and indirect (eg, lost productivity) costs.[2]

It is estimated that more than 80% of strokes are ischemic, and of these, approximately 15% to 20% are thought to be the direct result of carotid atherosclerosis.[4–6] The dominant mechanism for symptomatic carotid artery–related stroke is artery-to-artery embolization from atherosclerotic disease in the carotid bifurcation, although embolization from the intracranial carotid artery, proximal vertebral artery, and the aortic arch are also common.[6] Other mechanisms, such as carotid artery dissection and multivessel occlusive plaque, are uncommon causes of symptomatic carotid

Disclosures: Drs Li and Sakhuja report no disclosures. Dr Parikh serves as a consultant for Abbott Vascular, Boston Scientific, and Medtronic and receives research support from Boston Scientific and Medtronic.

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Intervent Cardiol Clin 3 (2014) 73–90
http://dx.doi.org/10.1016/j.iccl.2013.09.006

disease. With the tremendous societal and personal burden of stroke, carotid stenosis is an important disease to combat.

Revascularization remains the mainstay of therapy for patients with symptomatic carotid stenosis. Carotid endarterectomy (CEA) has traditionally been the strategy for surgical revascularization, with significant reduction in the recurrence of stroke compared with medical therapy alone. In the past 3 decades, the advent of endovascular techniques and carotid artery stenting (CAS) have led to the current paradigm of revascularization with either CEA or CAS, in conjunction with aggressive medical management for the treatment of patients with symptomatic disease. Candidates for revascularization should have a predicted periprocedural stroke or death rate of less than 6%, a benchmark set by early CEA trialists for which surgical benefits outweigh risks.[7–10]

PATIENT EVALUATION

Screening for carotid stenosis is typically triggered by the presence of symptoms or signs on physical examination (eg, abnormal palpation of carotid pulse, presence of a carotid bruit). Symptoms of carotid stenosis include transient ischemic attack (TIA) and stroke. Although an imperfect correlation, severity of stenosis and symptomatic disease remain the most important clinical indicators for embolic potential of carotid atherosclerosis.[7,11]

In symptomatic patients, the association between disease severity and stroke risk are closely related. With greater degrees of stenosis, the risk of recurrent stroke increases substantially. Specifically, data from the North American Symptomatic Carotid Endarterectomy Trial (NASCET) illustrate that patients with greater than 70% stenosis had a stroke rate of 24% at 18 months, versus patients with 50% to 69% stenosis with a stroke rate of 22% at more than 5 years.[12] In addition, the greatest risk of recurrence exists soon after the index event, estimated to be 10% to 25% within the first month.[6,7,13] Thus, timely determination of the degree of carotid stenosis is essential in symptomatic patients. A proposed algorithm for the assessment of patients with symptomatic carotid stenosis is outlined in **Fig. 1**.

There are a variety of modalities for estimating severity of stenosis. Although the gold standard for assessment of the presence and degree of carotid stenosis has been catheter-based digital subtraction angiography, advances in noninvasive imaging techniques have led to their widespread use as the first-line screening test.[14–17] **Table 1** shows the different imaging modalities commonly used, the technique of quantifying degree of stenosis, and their respective strengths and weaknesses. There are 2 methods of measuring the degree of stenosis on angiography, modeled after the NASCET and the European Carotid Surgery Trial (ECST) studies (**Fig. 2**).[7,8,11,12,18] The most commonly used technique in clinical trials and practice has traditionally been the NASCET approach; 70% stenosis by NASCET correlates well with approximately 85% stenosis by the ECST approach.[7,12]

The optimal imaging modality depends on patient-related and practice-related factors. Patient-related factors include age, renal function, prior surgical interventions affecting the imaging field, and importantly, patient ability to comply with necessary testing. Practice-related factors derive primarily from the available expertise in a health care system. For example, if a center has technologists skilled in carotid duplex ultrasound (CDUS) and physicians trained in the nuanced interpretation of carotid duplex, this may be the best imaging modality at that facility.

CDUS is often a simple, sensitive, and inexpensive initial study to evaluate stenosis severity. Patients with severe stenosis and high-risk features for CEA, as shown in **Table 2**, are considered for carotid angiography and CAS as indicated.[19] Patient characteristics that portend a need for further anatomic study before carotid angiography—either with computed tomography (CT) or magnetic resonance (MR)—are listed in **Table 3**.

Imaging in the setting of acute stroke differs, as CT and MR are fundamental to the evaluation of an acute stroke. CT is less time-consuming and more sensitive than MR for the detection of intracranial hemorrhage. However, diffusion-weighted MR is much more sensitive for the detection of infarction in the setting of recent cerebrovascular occlusion.

MEDICAL TREATMENT

The immediate management of a patient with acute ischemic stroke is composed of thrombolytic therapy if deemed appropriate and supportive care to minimize intracranial hypoxemia and hypotension to reduce parenchymal injury.[11,17] Following a first stroke or TIA, modifiable risk factors to treat aggressively include hypertension, tobacco use, diabetes, dyslipidemia, physical inactivity, and dietary choices.[20,21] The role of interventional therapies for restoration of cerebral perfusion remain actively under investigation.

The foundation for secondary prevention of stroke is based on lowering of blood pressure and cholesterol, as well as the use of antiplatelet therapy.[21] The amount of benefit derived from