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Treatment Strategies for Carotid Stenosis in Patients at Increased Risk for Surgery

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Abstract The purpose of carotid revascularization is stroke prevention. The merits of carotid revascularization as well as the type of revascularization are dependent on the "natural risk" and the "revascularization risk." In general, the natural risk of stroke in any patient with carotid stenosis (CS) is dependent on the symptomatic status of the patient and CS severity. Contemporary choices for carotid revascularization include carotid endarterectomy (CEA) and carotid artery stenting (CAS). Anatomical (hostile neck situations, severe bilateral CS, CEA restenosis) and clinical (severe cardiopulmonary diseases, prior cranial nerve injury) factors may increase the risk of CEA. Likewise, anatomical (complex aortic arch and brachiocephalic arterial anatomy, presence of thrombus, and heavy calcification) and clinical (need for heart surgery within 30 days) factors may increase the risk of CAS. Other factors such as the presence of symptomatic CS (transient ischemic attack or stroke within 6 months), decreased cerebral reserve, chronic kidney disease, and age older than 75 years may increase the risk of CEA and CAS. In general, symptomatic patients with severe CS exceed revascularization risk. In contrast, asymptomatic patients who are high risk for CEA should be considered for CAS because the natural risk of stroke should undergo careful assessment of baseline cognitive function, aortic arch and carotid artery anatomy, and likelihood of survival for 3 years. Patients who have normal cognitive function, favorable anatomy, and high likelihood of survival more than 3 years should be considered for CAS, whereas patients with multiple unfavorable features may be treated with optimal medical therapy, without revascularization. (Prog Cardiovasc Dis 2011;54:22-28)

Keywords: Carotid stenosis; Carotid stent; Carotid endarterectomy

Stroke is the third leading cause of death in the United States, and severe carotid stenosis (CS) accounts for at least 20% of all ischemic strokes. Strategies for stroke reduction in patients with CS include medical therapy and carotid revascularization. The indications for revascularization depend on the symptomatic status of the patient, CS severity, and the risk of revascularization.[1] Although carotid endarterectomy (CEA) is considered the criterion standard for revascularization, carotid artery stenting (CAS) is now accepted as a reasonable alternative to CEA. Approximately 25% of patients with severe CS have serious comorbidities that increase the risk of CEA, and these comorbidities may limit their long-term survival. Accordingly, for patients with severe CS who are at high risk for CEA, the optimal therapy should be individualized based on careful risk assessment.

Risk assessment

The purpose of therapies for CS is stroke prevention, which may be achieved with medical therapy and

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Abbreviations and Acronyms

CAS = carotid artery stenting

CEA = carotid endarterectomy

CKD = chronic kidney disease

CMS = Centers for Medicare and Medicaid Services

CS = carotid stenosis

CTA = computed tomography angiography

EPD = embolic protection devices

FDA = Food and Drug Administration

MACE = major adverse cardiovascular events

MRA = magnetic resonance angiography

RCTs = randomized clinical trials

TIA = transient ischemic attack

SAPPHIRE = Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (trial)

revascularization. All patients with CS should be treated with optimal medical therapy (see below) regardless of plans for revascularization. For patients who are receiving medical therapy, the major determinants of the risk of stroke include the risk of unrevascularized CS (natural risk) and the early and late risks of revascularization (revascularization risk) (Table 1). The natural risk is determined primarily by the symptomatic status of the patient and CS severity. Patients are considered symptomatic if they experience a retinal or hemispheric stroke or transient ischemic attack (TIA) in the distribution of the CS within the preceding 6 months. Other factors that influence the natural risk include plaque morphology and microembolization by transcranial Doppler studies.[2,3]

The revascularization risk is determined by assessment of known anatomical and clinical factors.[4] Anatomical factors that increase the risk of CEA include CEA restenosis, the presence of severe bilateral CS requiring revascularizaton, inaccessible CS (stenosis above the mandible or below the clavicle), and hostile neck situations resulting from prior radiation therapy, radical neck surgery, cervical spine immobility, and tracheostomy. Clinical factors that increase the risk of CEA include prior cranial nerve injury and severe cardiopulmonary disease, such as recent myocardial infarction, symptomatic multivessel coronary artery disease, left ventricular ejection fraction less than 30%, and oxygen-dependent pulmonary disease. Anatomical factors that increase the risk of CAS include complex aortic arch (type 3 arch configuration) and brachiocephalic arterial disease (severe tortuosity proximal and/or distal to the CS), severe CS calcification, and visible thrombus. Clinical factors that increase the risk of CAS include the need for heart surgery within 6 weeks after CAS. Importantly, the risks of CEA and CAS are increased in patients with symptomatic CS, impaired cerebral reserve, chronic kidney disease (CKD), and age older than 75 years.

Table 1
Risk assessment for patients with CS

Determinants of Natural Risk
Symptomatic status
Stenosis severity
Plaque composition
Transcranial Doppler
Determinants of the Revascularization Risk
CEA
Anatomical: CEA restenosis, severe bilateral CS,* hostile neck,† inaccessible CS‡
Clinical: prior cranial nerve injury, severe cardiopulmonary disease§
CAS
Anatomical: complex arch/carotid anatomy,‖ lesion calcification, thrombus
Clinical: need for open heart surgery within 6 weeks after CAS
CEA and CAS
Clinical: CKD, age >75 years, symptomatic CS

* Severe bilateral CS requiring revascularization or contralateral carotid occlusion.

† Neck deformity due to radiation therapy or radical neck dissection, cervical spine immobility.

‡ CS below the clavicle or above the mandible.

§ Angina with dynamic electrocardiogram changes or multivessel disease, myocardial infarction within 30 days, severe congestive heart failure, left ventricular ejection fraction less than 30%, need for home oxygen, forced expiratory volume in one second less than 30% of predicted.

‖ Aortic arch type 3, severe carotid tortuosity proximal, or distal to the stenosis.

Finally, the expectation of late survival is an important consideration in evaluating patients with CS for revascularization. Patients with serious cardiopulmonary comorbidities who are at high risk for CEA may have 5-year mortality rates approaching 50%.[5] In these patients, CAS may be justified for symptomatic CS; but the value of CAS (and CEA) compared with optimal medical therapy is less certain for asymptomatic patients because some of these patients may not survive long enough to derive the benefit of carotid revascularization.

Recommendations for medical therapy

In patients with CS, current guidelines provide class I recommendations for medical therapy to achieve blood pressure less than 140/90 mm Hg (<130/80 mm Hg in patients with diabetes or CKD), smoking cessation, and lipid management to achieve low-density lipoprotein less than 100 mg/dL (<70 mg/dL if high-risk coronary artery disease) and non–high-density lipoprotein less than 130 mg/dL. Class II recommendations are provided for diabetes control (hemoglobin level A1C <7%) and weight reduction (waist circumference, <40 cm in men and <35 cm in women). For primary prevention of stroke, daily aspirin (81 mg) is recommended; there are no studies of thienopyridines for primary prevention. For secondary prevention of stroke, aspirin (81-325 mg daily) or