REVIEW ARTICLE

Symptomatic carotid artery stenosis: what is the preferred treatment?

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KEY WORDS

carotid artery disease, carotid endarterectomy, carotid stent

ABSTRACT

Stroke is a major cause of mortality, morbidity, and disability. Carotid artery disease is the etiology for 15% to 20% of stroke. Carotid endarterectomy (CEA) reduces the risk of ipsilateral stroke and death in symptomatic patients with 50% to 99% carotid artery stenosis when the operative risk of stroke or death is less than 6%. Treatment benefit is greater with earlier surgery, more severe stenoses, and older age. Recently, carotid artery stenting (CAS) has emerged as a treatment option, especially in patients with high surgical risk due to anatomic or clinical variables. Nondisabling stroke risk may be higher with CAS than CEA, but the difference is narrowed with the use of embolic protection devices. The risk for myocardial infarction is lower with CAS than CEA. There is no difference in risk for disabling stroke or death. Worse results with new or low-volume CAS operators is a concern.

CEA and CAS are complementary revascularization strategies. CEA may be preferred in older patients with complex anatomy or bulky plaques. CAS may be preferred in younger patients and those with restenosis, history of neck radiation, surgical contraindications, or surgically inaccessible lesions. The role for optimal medical therapy as an alternative treatment strategy remains to be defined. Nevertheless, all patients should be treated with lifestyle interventions and secondary risk factor control to target levels to reduce the risk of subsequent atherosclerotic events.

Introduction Stroke is a major cause of morbidity and mortality worldwide, ranking as the third leading cause of death in industrialized nations.[1] In the United States, over 6 million people have suffered from stroke and an estimated 795,000 new strokes occur annually.[2] Stroke is the leading cause of long-term disability in the United States, expected to cost over $73 billion in direct and in-direct health care expenses in 2010.[2] Atherosclerotic carotid artery disease accounts for 15% to 20% of stroke.[3] Carotid endarterectomy (CEA), established by randomized trials as an effective treatment for symptomatic carotid artery stenosis 20 years ago, has become one of the most commonly performed vascular surgeries. Recently, carotid artery stenting (CAS) has emerged as an alternative to CEA for the treatment of carotid artery stenosis. Although new data have broadened our knowledge of carotid artery disease and help guide therapeutic decisions, treatment controversies persist.

Pathophysiology Symptomatic carotid artery disease is conventionally defined as the sudden onset of focal neurologic symptoms occurring within 6 months and attributable to a carotid artery vascular distribution. Symptoms may be transient (<24 hours) or permanent and may be due to hemispheric or retinal events. Atherosclerosis affecting the carotid arteries is similar to that in other vascular beds, but tends to be focal with 90% of lesions found within 2 centimeters of the carotid bulb.[4] Proposed mechanisms of stroke or transient ischemic attack include embolism of plaque associated thrombus or other atheromatous debris, acute thrombotic carotid artery occlusion from plaque erosion or rupture, or reduced cerebral perfusion from progressive plaque growth and expansion.

The risk of stroke is significantly higher in symptomatic than asymptomatic patients and increases concordantly with greater degrees of carotid stenosis. Patients with recent nondisabling stroke enrolled in NASCET (North American

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Symptomatic Carotid Endarterectomy Trial) had a 2-year ipsilateral stroke rate of 26% compared with the annual stroke rate of 2.4% seen in asymptomatic patients in ACST (Asymptomatic Carotid Surgery Trial).[5,6] While the stroke rate was noted to be higher in symptomatic patients with more significant stenoses in NASCET (35% in those with stenosis greater or equal to 90% vs. 11% for stenosis 70%–79%), this correlation was not seen in asymptomatic patients. Paradoxically, stroke occurred less frequently in patients with near occlusion. Other clinical features influencing stroke risk are the presence or absence of contralateral disease or established collaterals, silent cerebral ischemia, and medical comorbidities such as hypertension, diabetes, smoking, obesity, and dyslipidemia.[7]

Carotid endarterectomy CEA was first introduced as a treatment for symptomatic carotid artery stenosis in 1954,[8] but only became established as a safe and effective therapeutic option in 1991 when the results of 2 pivotal trials were published.[5,9] In NASCET,[5] 659 symptomatic patients with carotid artery stenosis of 70% to 99% were randomized to CEA or medical therapy. Those undergoing CEA had a significant reduction in the primary endpoint of death or stroke at 2 years (15.8% vs. 32.3%) and a 65% relative risk reduction in stroke. Further analyses suggested this benefit did not pertain to those with mild stenosis (<50%), but was extended to patients with moderate stenosis (50%–69%) with an absolute reduction in the ipsilateral stroke rate of 6.5% at 5 years.[10] Similarly, of the 2518 symptomatic patients with severe stenosis >70% in ECST (European Carotid Surgery Trial), CEA reduced stroke by 14% at 3 years compared with medical therapy.[11] Unlike NASCET, initial analyses of patients with moderate stenosis in ECST did not reveal benefit with CEA; however, different definitions in calculating percent diameter stenosis were used in the trials. Once adjusted, the disparities disappeared and CEA for symptomatic carotid stenosis above 50% was shown to be beneficial.[12] Data pooled from these 2 trials, and the VA Affairs Cooperative Study,[13] using consistent definitions for percent diameter stenosis and clinical endpoints further confirmed the reduction in stroke risk at 5 years with CEA. Again, treatment benefit was greater with greater degree of stenosis (48% relative risk reduction with 70%–99% stenosis and 28% with 50%–69% stenosis), but not in those with mild stenosis or near total occlusion.[14] It is worth noting that the medical therapy in these early trials was limited, with treatment largely confined to aspirin alone, and clinical endpoints were poorly defined.

Additional analyses suggest greater clinical benefit in patients older than 75 years.[15] Treatment benefit is greatest within 2 weeks of the qualifying clinical event and rapidly declines over time.[16] Interestingly, compared with men, women had a higher operative risk in ECST, and higher rates of surgical mortality, neurological morbidity, and recurrent stenosis in NASCET.[11,17] Additionally, patients who presented with retinal symptoms had a more benign clinical course than those with hemispheric symptoms and did not experience a significant benefit with CEA unless other high-risk features were present.[14,18]

Risks associated with CEA were highest in the first 30 days after surgery, reaching 7.1% for death and stroke, and were more likely to occur in symptomatic patients presenting with hemispheric symptoms, in an urgent manner, or for restenosis.[14,19] Additional perioperative complications include hemodynamic instability, myocardial infarction (MI), bleeding, and cranial nerve palsy. High surgical risk may be defined by both anatomic and clinical variables.[20] Anatomic high-risk features include lesions above the second cervical vertebra or below the clavicle, prior neck radiation, contralateral occlusion, or previous ipsilateral CEA. High-risk medical comorbidities include recent MI, left main and/or 2 vessel coronary artery disease (CAD), congestive heart failure or angina, left ventricular ejection fraction less than 30%, age greater than 80 years, and severe renal or pulmonary disease. CEA has an American Heart Association Class I indication for the treatment of symptomatic carotid artery disease with 50% to 99% diameter stenosis if the perioperative risk of stroke or death does not exceed 6%.[7,21]

Carotid artery stenting With the evolving success of percutaneous coronary intervention for the treatment of CAD, attempts to apply similar technology to carotid artery stenosis began in the mid-1990s, offering a procedure that is less invasive and potentially applicable to high surgical risk populations. The first carotid balloon angioplasty was performed in 1979[22] and the first stent was deployed in 1989[23]; yet early attempts at endovascular therapy were beset by an unacceptably high embolic stroke rate. As stent design improved and embolic protection devices (EPD) were developed, interest in CAS was renewed. Early CAS trials were limited to small, single-center observational studies with inconsistent EPD and stent use, high procedural success rates, and variable clinical outcomes.[24,25] With further technologic advances and increased operator proficiency, several multicenter CAS registries were developed to assess CAS in high surgical risk populations.[26-29] These more contemporary registries focused on safety endpoints including the 30-day MI rate in addition to standard death and stroke rates, and defined primary efficacy as death and stroke occurring at 30 days to 1 year after CAS. Given the generally favorable results, studies were then developed to compare CAS directly with CEA as opposed to historical controls.

Carotid artery stenting vs. carotid endarterectomy The first randomized controlled trials comparing the safety and efficacy of CAS with CEA in symptomatic patients were stopped prematurely due

to the high rate of neurologic complications with CAS; however, these studies were limited by inconsistent EPD use and poor stent design.[30,31] Subsequently, the SAPPHIRE trial (Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy) compared routine stenting with EPD use to CEA in patients deemed high surgical risk by either clinical or anatomical features.[32] This trial included both symptomatic and asymptomatic patients and was designed to test the hypothesis that CAS was noninferior to CEA. The primary endpoint of death, stroke, and MI at 30 days plus ipsilateral stroke or death between 31 days and 1 year occurred in 12.2% of CAS patients and 20.1% of CEA patients (P = 0.004 for noninferiority). Not surprisingly, patients in the CAS arm suffered less cranial nerve injury at 1 year, but the rates of stroke or target vessel revascularization were no different at 3 years. While critical to establishing CAS as a viable treatment alternative to CEA in high surgery risk patients, this study has been criticized for its inclusion of MI as a clinical endpoint (most of which were asymptomatic, enzymatic events) and its high complication rate given the large number of asymptomatic patients (roughly 70%) enrolled.

Contemporary trials of carotid artery stenting vs. carotid endarterectomy Recently, the results of 4 large randomized trials comparing the safety and efficacy of CAS with CEA in standard surgery risk symptomatic patients were published. The SPACE trial (Stent-Protected Percutaneous Angioplasty vs. Carotid Endarterectomy) showed no significant difference in outcomes at 30 days or 2 years between CAS and CEA, but was underpowered to prove noninferiority and limited by inconsistent EPD use in the CAS arm.[33] Further analysis suggested not only an increase in restenosis rates with CAS, but also an increased risk in death or stroke in the elderly population.[34] In the Endarterectomy Versus Angioplasty in Patients with Symptomatic Severe Carotid Stenosis (EVA-3S) trial, more patients with CAS suffered stroke at 30 days (9.6% vs. 3.9%) and the study was stopped prematurely amid safety concerns.[35] As with the SPACE trial, limitations included incomplete EPD use and inexperienced CAS operators, placing any conclusions in doubt. The larger International Carotid Stenting Study (ICSS) randomized patients with carotid stenosis over 50% to CAS or CEA. The published interim safety data revealed a higher rate of death, stroke, or MI at 3 months in patients treated with CAS (8.5% vs. 5.2%, P = 0.006).[36] Whereas higher death or stroke rates at 120 days were noted in the CAS group, no difference in MI rates was seen, perhaps explained by the lack of protocol driven periprocedural cardiac biomarker or electrocadiographic (EKG) assessment. CREST (Carotid Revascularization Endarterectomy vs. Stenting Trial) enrolled 2502 patients, 53% of whom were symptomatic, and found no significant difference in the primary endpoint between the 2 procedures.[37] Distinct from other trials, proceduralists in CREST were rigorously trained, routine cardiac enzyme measurement and EKGs were performed, and a high rate of EPDs were successfully deployed (96%). Controversy surrounds the inclusion of MI in the primary endpoint as it deviates from previous CEA trials (other than inclusion as a marker of safety in ICSS) and would be expected to favor CAS. As such, stroke and death rates were higher in the CAS arm (4.4% vs. 2.3%), whereas the incidence of MI was higher with CEA (2.3% vs. 1.1%). Interestingly, as opposed to stroke, MI and cranial nerve palsy did not appear to effect quality of life and a trend favoring CAS in patients younger than 70 years was reported.

More recently, a preplanned meta-analysis of the 3 largest trials limited to symptomatic patients only (SPACE, EVA-3S, and ICSS) was published.[38] Higher short-term rates of death and stroke with CAS were seen in the analysis of 3433 patients and a prespecified subgroup analysis indicated a 2-fold increased risk of stroke and death in patients older than 70 years, but no difference in outcomes between CAS and CEA for those younger than 70 years. It has been suggested that the poorer outcomes associated with CAS in older patients may be related to increased atheromatous burden, vessel tortuosity, or greater plaque instability.

Future directions As previously noted, medical therapy in the initial trials of CEA was quite limited. Well-known modifiable risk factors for stroke include hypertension, dyslipidemia, diabetes, tobacco use, and obesity. As of yet, a third arm of optimal medical therapy alone has not been included in the published randomized trials of CEA vs. CAS, but may ultimately prove to be the best strategy to prevent stroke and MI in patients with asymptomatic carotid disease.[39] Newer antiplatelet agents have become available and optimal medical therapy has been shown to be an effective treatment for stable CAD.[40] Furthermore, HMG-CoA reductase inhibitors (statins) significantly reduced the recurrence of stroke compared with placebo in the SPARCL study (Stroke Prevention by Aggressive Reduction in Cholesterol Levels).[41] Data from the ongoing SPACE-2 and ICSS-2 trials will eventually clarify the role of optimal medical therapy compared with revascularization therapy in patients with carotid artery disease.

Recently published trials indicate improved outcomes with both CAS and CEA compared with the original NASCET and ECST studies. The low rates of death and disabling stroke in both arms of the ICSS and CREST trials support significant improvements in procedural success and patient outcomes. Whether improved stent technology or EPD designs will lower periprocedural event rates with CAS will be the subject of continued research. Results with new or low-volume CAS operators remain a concern.

Conclusion CEA for severe symptomatic carotid artery stenosis reduces long-term stroke risk. The exact role for CAS in the treatment of symptomatic carotid artery stenosis has yet to be defined because of the increased procedural risk for nondisabling stroke. Current data indicate that CAS may be a viable alternative to CEA in younger patients and in those with anatomical and clinical high surgical risk (restenosis, postradiation, surgical contraindications, surgically inaccessible sites, women, symptomatic coronary disease). In addition to optimal medical therapy, a tailored approach may ultimately prove to be ideal with CAS being the preferred treatment for patients at increased cardiac risk, whereas CEA may be preferable in older patients with increased stroke risk. Ongoing studies will inform these controversies.

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Jakie jest optymalne leczenie objawowego zwężenia tętnicy szyjnej?

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endarterektomia tętnicy szyjnej, stentowanie tętnicy szyjnej, zwężenie tętnicy szyjnej

STRESZCZENIE

Udar mózgu jest jedną z głównych przyczyn zachorowalności, umieralności oraz niesprawności. Zwężenie tętnic szyjnych powoduje około 15–20% udarów. Endarterektomia tętnicy szyjnej (*carotid endarterectomy* – CEA) u chorych objawowych ze zwężeniem 50–99% zmniejsza ryzyko udaru mózgu po stronie zwężenia oraz zgonu, jeżeli okołooperacyjne ryzyko udaru lub zgonu wynosi <6%. Większą korzyść z leczenia obserwuje się w przypadku wykonania operacji we wcześniejszym okresie, cięższego zwężenia oraz starszego wieku chorego. Wewnątrznaczyniowa angioplastyka z wszczepieniem stentu (*carotid artery stenting* – CAS) jest nowym sposobem leczenia, zwłaszcza pacjentów z grupy wysokiego ryzyka operacyjnego z przyczyn anatomicznych lub klinicznych. CAS w porównaniu z CEA wiąże się z większym ryzykiem udaru niepowodującego niesprawności, jednak stosowanie urządzenia zabezpieczającego przed zatorowością zmniejsza różnicę między tymi procedurami. Z drugiej strony CAS wiąże się z mniejszym ryzykiem zawału serca. Ryzyko udaru mózgu powodującego niesprawność lub zgonu jest podobne w obu metodach leczenia. Obawy związane są z gorszymi wynikami leczenia prowadzonego przez niedoświadczonych operatorów lub tych, którzy wykonują niewielką liczbę zabiegów rocznie.

CEA i CAS są strategiami rewaskularyzacyjnymi, które wzajemnie się uzupełniają. CEA jest metodą preferowaną u chorych w starszym wieku ze złożoną anatomią naczyń lub blaszką miażdżycową. CAS może być zalecana u pacjentów młodszych, z nawrotem zwężenia, po radioterapii okolicy szyi, z przeciwwskazaniami do zabiegu operacyjnego lub ze zmianami niedostępnymi dla chirurga z przyczyn anatomicznych. Należy określić rolę optymalnego leczenia zachowawczego jako alternatywnej strategii terapeutycznej. U wszystkich chorych wskazane są interwencje modyfikujące styl życia oraz wtórna kontrola czynników ryzyka w celu osiągnięcia docelowych wartości i zmniejszenia wystąpienia ryzyka dalszych zdarzeń wynikających z miażdżycy.

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