Optimal Perioperative Management of Symptomatic very High Grade Stenosis and near Occlusion of Extracranial Carotid Bifurcation Artery Disease

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Abstract

Background: Carotid artery near occlusion and very high grade stenosis (>95%), is a critical degree stenosis whereby intracranial blood flow is dramatically decreased and the risk of progression to total occlusion or distal cerebral embolisation is very high.

Methods: The current state of knowledge was surveyed in a selective review of pertinent literature retrieved by an electronic search in the PubMed, Scopus and Google Scholar.

Results and conclusions: Treatment of very high grade internal carotid artery stenosis remains a matter of debate between medical and surgical best management. Individualization of each case is imperative to optimal decision making and best perioperative management. It would be advisable for the care of critical symptomatic internal carotid artery stenosis to be centralized in specialized vascular centers implementing defined treatment pathways.

ABBREVIATIONS

TIA: Transient Ischemic Attack; RIND: Reversible Ischemic Neurologic Deficit; ICA: Internal Carotid Artery; CEA: Carotid Endarterectomy; CAS: Carotid Artery Stenting; CHS: Cerebral Hyperperfusion Syndrome

INTRODUCTION

Stroke is the third leading cause of death in the United States and about 160,000 Americans die of stroke each year [1]. Current estimates suggest that 87% of strokes are ischemic and 13% are hemorrhagic, with hemorrhagic strokes being approximately equally divided between subarachnoid and intracranial hemorrhage [2].

The prevalence of cerebrovascular disease is increased with age, total cholesterol, low-density lipoprotein cholesterol, fibrinogen, systolic blood pressure, cigarette use [3], and is much greater in patients with peripheral vascular disease [4].

Carotid artery atherosclerosis predisposes patients to TIA and stroke, and the risk for these events is proportional to the severity of the carotid disease. A linear relationship between the degree of carotid stenosis and the neurologic event rate and correlated clinical outcome with the echogenicity of the carotid lesions is well proven [5,6].

Patients at highest risk for TIA or stroke are those with greater than 80% stenosis secondary to soft, echolucent plaque or those whose plaque progresses from less than 80% to greater than 80% stenosis during follow-up.

A recently published review in invasive treatment of high grade carotid stenosis demonstrated carotid artery near occlusion to be found in 770 patients and typical appearance of string sign in 60% of patients with 92.6% of them being symptomatic [7].

Additionally the decision to treat a very high grade symptomatic carotid stenosis should not be influenced by the presence of high grade contralateral stenosis but the latter may be considered a marker for severe coronary artery disease [8].
CLINICAL DIAGNOSTIC EVALUATION

Transient ischemic attack (TIA)

Transient Ischemic Attack (TIA) and stroke have generally been accepted to represent compelling indications for surgical treatment and cerebral revascularization.

TIAs are defined as neurologic events that are sudden in onset without a preceding aura, are less than 24 hours in duration and resolve to leave the patient at neurologic baseline.

Carotid territory TIAs can involve the eye only (transient monocular blindness or transient monocular field cuts) may also result in speech deficits (dysarthria, dysphasia, or aphasia).

Motor manifestations range from mild clumsiness of a single limb to hemiplegia opposite the carotid lesion and sensory manifestations may include numbness or paresthesias on the side opposite the carotid lesion.

Reversible ischemic neurologic deficit (RIND)

Reversible ischemic neurologic deficit (RIND) is a term used to describe a focal neurologic event lasting longer than 24 hours but resolving completely within 1 week. The duration of symptoms in patients with RIND suggests that some degree of structural damage to the brain must have occurred, although it may be very limited and undetectable by clinical imaging studies.

Strokes

Strokes are infarctions of central nervous system tissue related to hypoperfusion, embolisation or intracranial hemorrhage. The stroke deficits related to carotid disease are similar to the temporary deficits seen with TIA.

Permanent monocular blindness secondary to retinal infarction, aphasia, monoparesis or hemiparesis/hemiplegia, and hemisensory deficits are the most common manifestations of stroke related to carotid disease.

PARACLINICAL DIAGNOSTIC EVALUATION

Duplex color ultrasound

Duplex color ultrasound is the preferred first-line imaging modality for identifying patients with 70% to 99% Internal Carotid Artery (ICA) stenosis [9].

Magnetic resonance angiography

Magnetic Resonance Angiography (MRA) is playing an increasingly important role in the evaluation of patients with carotid artery occlusive disease presenting high sensitivity and specificity for diagnosing 70% to 99% stenosis but poor for diagnosing 50% to 69% stenosis.

Computed tomography angiography

Computed Tomography Angiography (CTA) is headed toward being considered the overall new gold standard and has the advantage of being less susceptible than MRA to the risk of overestimating the severity of stenosis.

Digital subtraction angiography (DSA)

Its easily interpreted images of the arch, carotid arteries, and vertebral arteries (VA), as well as the siphons and intracranial circulation remains the undisputed gold standard for imaging of the extracranial and intracranial circulation.

CAROTID BIFURCATION DISEASE - INTERNATIONAL TRIALS

In the Asymptomatic Carotid Atherosclerosis Study (ACAS), patients met eligibility criteria as long as they had no previous symptoms in either the ipsilateral cerebral hemisphere or the vertebrobasilar circulation [10].

In the European Asymptomatic Carotid Surgery Trial (ACST), patients were eligible for entry as long as the “stenosis had not caused any stroke, transient cerebral ischemia, or other relevant neurological symptoms in the past 6 months” [11].

Both the ACAS and the ACST demonstrated a benefit of Carotid Endarterectomy (CEA) with medical therapy (aspirin and statin) over medical therapy alone for patients with carotid stenosis in the 60% to 99% range.

Furthermore, the European Carotid Surgery Trial (ECST) and the North American Symptomatic Carotid Endarterectomy Trial (NASCET) demonstrated the benefit of CEA in patients with recent ipsilateral carotid territory symptoms and moderate to severe carotid stenosis to be much greater than the benefit of CEA in asymptomatic patients [12,13].

MEDICAL TREATMENT OF CAROTID DISEASE

Recommendations for lifestyle changes include weight loss, increased exercise, limitation of alcohol use, and smoking cessation.

The goals of medical therapy are to prevent atheromatous plaque progression to arterial occlusion and atheromatous debris distal embolisation. Aggressive medical management of hypertension, hyperlipidemia, and diabetes is paramount.

Antiplatelet agents are the desired antithrombotic medications for prevention of thromboembolic events in patients with atherosclerotic extracranial or intracranial disease [14].

Statins besides the fact that have been effective in lowering the risk for ischemic stroke in patients with coronary artery disease, have also been demonstrated the ability to stabilize the arterial wall and prevent more embolic events [15].

SURGICAL TREATMENT OF CAROTID DISEASE

Neurologically asymptomatic patients with ≥ 60% diameter stenosis should be considered for CEA for reduction of long-term risk of stroke, provided the patient has a 3- to 5-year life expectancy and perioperative stroke/death rates can be ≤ 3% [16].

In most patients with carotid stenosis who are candidates for intervention, Carotid Endarterectomy (CEA) is preferred to Carotid Artery Stenting (CAS) for reduction of all-cause stroke and periprocedural death. [16]
For patients with severe stenosis and a recent TIA or non-disabling stroke, CEA should be performed without delay, preferably within two weeks of the patient’s last symptomatic event [17].

In symptomatic patients with moderate to severe carotid stenosis (>50%), carotid Endarterectomy is recommended plus optimal medical therapy [18].

Symptomatic NASCET patients with stenosis of 70% to 99% who underwent endarterectomy had a cumulative risk of any ipsilateral stroke at 2 years of 9% compared with 26% for those who were treated medically [18].

CEA is preferred over CAS in patients aged >70 years of age, with long (>15-mm) lesions, preocclusive stenosis, or lipid-rich plaques that can be completely removed safely by a cervical incision in patients who have a virgin, nonradiated neck [16].

CAROTID ENDARTERECTOMY (CEA) - SURGICAL TECHNIQUE

Carotid endarterectomy may be performed under general anesthesia (GA), under regional anesthesia (RA) with deep or superficial cervical block, and even under pure local anesthesia (LA).

Through a longitudinal or transverse incision after systemic heparin administration the internal, common and external carotid arteries are sequentially occluded with atraumatic vascular clamps.

A longitudinal incision is made anteriorly in the common carotid artery proximal to the obviously diseased segment, and extended distally along the anterior surface of the internal carotid artery beyond the offending plaque. If a shunt is elected to it is inserted at this time.

The endarterectomy is begun by carefully developing a subadventitial plane with a freer dissector in the common carotid artery, completed circumferentially, feathered to a good end-point proximally and continued distally, evertting the plaque out of the external carotid artery and then completed in the internal carotid artery when the plaque transitions into normal intima.

Most evidence strongly supports arteriotomy closure with an autogenous vein, Dacron or polytetrafluoroethylene patch using a running 6-0 polypropylene suture [16].

CEREBRAL HYPERPERFUSION SYNDROME (CHS) AFTER CEA

Cerebral Hyperperfusion Syndrome is a relatively rare syndrome with significant and potentially preventable clinical consequences.

Impaired cerebral autoregulation and post-revascularization changes in cerebral hemodynamics are the main mechanisms involved in the development of the syndrome.

The early recognition of CHS is important to prevent complications such as intracerebral hemorrhage. CHS consists in the clinical triad headache, convulsions and focal neurological deficit, associated with arterial hypertension and the absence of cerebral ischemia.

Most hemorrhagic strokes result from untreated postoperative hypertension leading to hyperperfusion injury, especially in patients with reestablishment of flow in previously infarcted cerebral tissue [19].

Postoperative CHS is associated with a very high mortality rate that approaches 75% to 100% in some series [20].

DISCUSSION & CONCLUSION

Carotid artery atherosclerosis remains one of the major causes of ischemic stroke. The efficacy of carotid recanalization has already been established and is considered significant for high grade symptomatic stenosis.

Regarding asymptomatic, carotid artery recanalization should be reserved for carefully selected patients with a high grade stenosis and an estimated periprocedural risk at less than 3%.

Long-term mortality in patients with asymptomatic carotid stenosis review by A. Giannopoulos, S. Kakkos and colleagues in 2015, indicated that in patients with >50% ACS, 5- and 10-year all-cause mortality was 23% and 52.5% respectively [21].

Soft, echoluent, ulcerated plaques at carotid bifurcation present higher embolic risk compared with stable, dense non ulcerated plaques and as such should be treated.

The presence of contralateral internal carotid occlusion or very high grade stenosis may greatly compromise cerebral perfusion and postoperative occurrence of cerebral hyperperfusion syndrome is increased.

The ECST and NASCET have also shown that the ubiquitous “string sign” is not associated with a high risk of stroke, and emergency CEA is unnecessary [22].

Additionally no medically treated patients with a string sign in NASCET suffered a stroke within 30days.

There has been a worldwide move towards performing CEA as soon as possible after onset of symptoms due to the highest risk period for recurrent stroke in patients with ipsilateral 50-99% stenosis of the internal carotid artery in the first few days [23,24].

Additionally the CARESS and CLAIR studies showed that early institution of aspirin and clopidogrel significantly reduced rates of embolisation in patients with recent onset TIA and stroke [25,26].

The importance of carefully case selection was evident in the American College of Surgeons National Surgical Quality Improvement Program (ACSNSQIP) Registry, which evaluated patients undergoing CEA between 2007 and 2009[27] in whom AHA/ASA guidelines [28] advised that “only highly selected patients with a predicted 5 year lifespan should be considered for intervention.”

A meta-analysis of pooled European RCT data had clearly
shown that CAS was associated with a significantly higher risk of procedural death/stroke (CAS= 8.9% vs 5.8% after CEA, HR 1.53, 95% CI 1.20-1.95; p= .0006) [29].

Furthermore CAS is associated with a threefold excess risk of procedural stroke if performed within 7 days of the index symptom (compared with CEA) and a twofold excess risk when performed between 8 and 14 days [30].

Finally, in no other clinical area encountered by the vascular surgeon are the quality and breadth of evidence as strong. It is essential that the vascular surgeon be well versed in the details of this evidence base.

Multidisciplinary management and full cooperation between doctors of different specialties (emergency department, neurologists, radiologists, surgeons, vascular surgeons and anesthesiologists) ensures the optimal result in terms of time-brain saving in such life threatening situations.

Proper selection of patients for intervention is as important a contributor to achieving overall patient benefit as technical skill in performance of the procedure.

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