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Carotid endarterectomy in patients with high plaque

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ABSTRACT

Background: Difficult cephalad exposure during carotid endarterectomy in patients with high plaque (HP) may lead to increased incidence of complications after carotid endarterectomy. We report on our experience of carotid endarterectomy in patients with HP.

Methods: This is a retrospective review of 1,233 consecutive patients who underwent carotid endarterectomy by a single surgeon at 2 teaching hospitals between January 1989 to December 2018. Group A consisted of patients with HP ($n = 100$) diagnosed by computed tomography angiography of the neck in 90, catheter-based arteriography in 8, and an unexpected finding during carotid endarterectomy in 2 patients. Group B consisted of 1,133 consecutive carotid endarterectomies with plaque ending in Zone 1 non-high plaque (nHP).

Results: Both groups were similar in age (70.9 ± 8.7 vs 70.3 ± 9.1, $P = .53$). There was a preponderance of male patients in the HP group (78.0% vs 66.1%, $P = .02$). Associated risk factors, including coronary artery disease, hypertension, diabetes, chronic obstructive pulmonary disease, and hyperlipidemia, were similar in both groups. Indications for carotid endarterectomy in HP patients include recent stroke (<8 weeks) in 15 patients (15.0%), transient ischemic attack in 23 patients (23.0%), and asymptomatic in 62 patients (62.0%). Three patients (3.0%) with HP required shunt placement compared with 10.9% in the nHP group ($P = .009$). Completion carotid arteriogram was performed in 6 patients. Perioperative stroke and mortality were similar in both groups. The incidence of cranial nerve injury was higher in the HP group.

Conclusion: Most patients with HP can be diagnosed with computed tomography angiography of the neck or catheter-based arteriography. Shunt requirement in patients with HP is significantly lower than in the nHP group. Perioperative stroke and mortality in patients with HP undergoing carotid endarterectomy is similar to the nHP group; however, there is a higher incidence of permanent cranial nerve injury. Carotid artery stenting should be considered in cases in which carotid endarterectomy may be challenging, such as in patients with HP. Overall, our results demonstrate that carotid endarterectomy can be safely performed in patients with HP, however, at an increased risk of permanent cranial nerve injury.

Introduction

Stroke is the fifth leading cause of death and is the principal cause of disability in the United States. Approximately 800,000 strokes occur each year, and extracranial atherosclerosis accounts for 10% to 20% of all ischemic strokes.[1,2] Clinical trials have conclusively demonstrated that carotid endarterectomy (CEA) reduces the incidence of stroke in patients with high-grade asymptomatic and moderate- to high-grade symptomatic carotid occlusive disease.[2,3] The combined risk of postoperative stroke or mortality in patients undergoing CEA has been demonstrated to be approximately 3% for previously asymptomatic patients[4,5] and 5% to 6% for those with symptomatic occlusive carotid disease.[5,6]

Although CEA remains the gold standard in the treatment of carotid artery stenosis, carotid artery stenting (CAS) has emerged as an alternative treatment. The Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy trial demonstrated no difference in 1-year stroke, death, and myocardial infarction (MI) in patients who underwent CAS with protective device versus those with CEA.[7] In high-risk patients with severe

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carotid stenosis, long-term follow up showed no significant difference in 3-year outcomes in those who underwent carotid stenting with protective device versus those with CEA.[8] The Carotid Revascularization Endarterectomy Versus Stenting Trial (CREST) study, which enrolled both asymptomatic and symptomatic patients, demonstrated no statistically significant difference between CEA and CAS in the primary end points of stroke, MI, or death from any cause or ipsilateral stroke within 4 years from after randomization.[9] Beyond the advantage of being minimally invasive, CAS is beneficial in patients with carotid artery occlusive disease in which CEA is prohibitive, such as in patients with previous neck radiation or a very high plaque (HP).

Patients with HP within the internal carotid artery (ICA) that extends cephalad to the angle of the mandible toward the base of the skull can be a challenging operation, even for an experienced vascular surgeon. Plaque that extends beyond the angle of the mandible is usually considered to be high. For objective categorization, we define a high carotid plaque as the cephalad end of the plaque extending to the second cervical vertebral body or higher on computed tomography angiography or catheter-based angiography.[10,11] Although a variety of techniques have been described to optimize exposure in patients with HP, the results of CEA in this group of patients have not been described in a large series. We report the results of our experience of CEA in patients with HP as compared with those without.

Methods

Data from consecutive patients undergoing CEA from January 1989 to December 2018 at 2, midsized teaching hospitals were retrospectively reviewed. Institutional review board approval was obtained at both hospitals.

The ICA in the neck can be divided into 3 zones based on its relationship to the vertebral body (Fig 1)[10,11]: zone 1, where most non–high plaque (nHP) terminate corresponding to upper end of C3 vertebral body; zone 2, plaque extending to the level of C2 vertebral body but below C1 vertebral body; and zone 3, plaque extending above the level of C2 vertebral body. We define HP as the cephalad end of the plaque at the level of the C2 vertebral body or higher, thus, zones 2 and 3 constitute HP. The patients' cohort was divided into 2 groups. Group A consisted of patients with HP ($n = 100$) diagnosed by computed tomography angiography (CTA) of the neck in 90, catheter-based arteriography in 8, and an unexpected finding during CEA in 2 patients. All CTA were reviewed by a neuroradiologist and not blinded. Group B consisted of 1,133 consecutive CEA's with plaque ending in zone 1, nHP. All CEA operations were performed by the senior author.

Demographics and all other pertinent data such as age, sex, history of nicotine abuse, associated comorbidities, current medications, and duration of symptoms before emergency room visit in symptomatic patients were recorded. All symptomatic patients were evaluated by a neurologist and, after 2006, by a stroke neurologist. Both hospitals have primary stroke certification. Cervical block anesthesia was preferred in most patients except those with undue anxiety, a poor command of the English language, or hearing loss. Under general anesthesia, continuous EEG monitoring and monitoring of median nerve somatosensory evoked potentials was performed. After carotid artery clamping, any EEG waveform changes suggesting ischemia (ipsilateral slowing, attenuation, or loss of a signal) resulted in a shunt being placed. Our preferred shunt is the Sundt shunt (Integra LifeSciences, Plainsboro, NJ).

Study endpoints included postoperative stroke and mortality, MI, neck hematoma requiring re-exploration, and permanent cranial nerve palsy. A new postoperative neurological deficit was determined as minor (NIH stroke scale <5).[12] A major stroke was categorized when the NIH stroke scale was >5 after CEA. Cranial nerve injury was permanent if hoarseness of the voice, swallowing difficulties, tongue deviation, or lip deformity persisted longer than 3 months after CEA. The patients with suspected permanent cranial nerve injury had an ENT consultation. Postoperative MI was suspected in patients with unexplained hypotension, cardiac arrhythmias, and shortness of breath with or without chest pain, and a cardiology consult was obtained. Appropriate studies such as 12-lead EKG, 2D echocardiogram (in selected cases), and serum biomarkers, such as creatine phosphokinase and troponin levels were obtained.

CEA was performed through an oblique neck incision with patch graft used in a vast majority of the patients. In most cases, additional dissection by extension of the skin incision cephalad and posterior to the ear provided adequate additional exposure of the distal cervical ICA. No special retractors were utilized in our CEAs. In 2 patients (2.0%), the upper end of the plaque extended above the middle of the second cervical vertebral body and required mandibular subluxation to be performed by an oral or maxillofacial surgeon with nasotracheal intubation.

A follow up was conducted in surviving patients with carotid duplex imaging at 1 month, 6 months, and yearly intervals. Statistical analysis was performed using SPSS 12.01 software (SPSS Incorporated, Chicago, IL). Fisher exact test and χ^2 test was used for comparison.

Results

The patient characteristics are shown in Table I. Both groups were similar in age (70.9 ± 8.7 vs 70.3 ± 9.1, $P = .53$). There was a preponderance of male patients in the HP group (78.0% vs 66.1%, $P = .02$). Associated risk factors including coronary artery disease, hypertension, diabetes, chronic obstructive pulmonary disease, and hyperlipidemia were similar in both groups. Contralateral ICA stenosis of <50% was present in 52 patients, 50% to 70% in 29, 70% to 99% in 15 patients, and contralateral ICA occlusion in 4 patients. Indications for CEA in HP patients include recent stroke (<8 weeks) in 15 patients (15.0%), transient ischemic attack in 23 patients (23.0%), and asymptomatic in 62 patients (62.0%) (Table II). Of those with HP, 71 patients (71.0%) had CEA with regional block anesthesia (Table III). Three patients (3.0%) with HP required shunt placement compared with 10.9% in nHP group ($P = .009$). Completion carotid arteriogram was performed in 6 patients. Perioperative stroke and mortality were similar in both groups. The incidence of cranial nerve injury was higher in the HP group (Table IV).

Postoperative stroke and mortality

Three patients (3.0%) had a major postoperative stroke in group A and 11 (1.0%) in group B ($P = .10$). Group A had no patients with a minor postoperative stroke, whereas group B had 7 patients (0.6%) who suffered a mild postoperative stroke. All perioperative strokes were ipsilateral to the CEA. There were 2 (2%) mortalities in group A and 7 (0.6%) mortalities in group B, postoperatively (Table IV).

Cranial nerve injury

Two patients in group A (2.0%) and 3 patients in group B (0.3%) ($P = .06$) had permanent cranial nerve injury. Permanent cranial nerve injury was defined as neurological deficit persisting greater than 3 months post operation. Four patients had ipsilateral motor dysfunction of the tongue, which were well compensated by the contralateral tongue with minimal disability. One patient had dysphagia requiring a percutaneous gastrostomy tube to be inserted; however, the dysphagia resolved at 4 months and the percutaneous gastrostomy tube was discontinued.

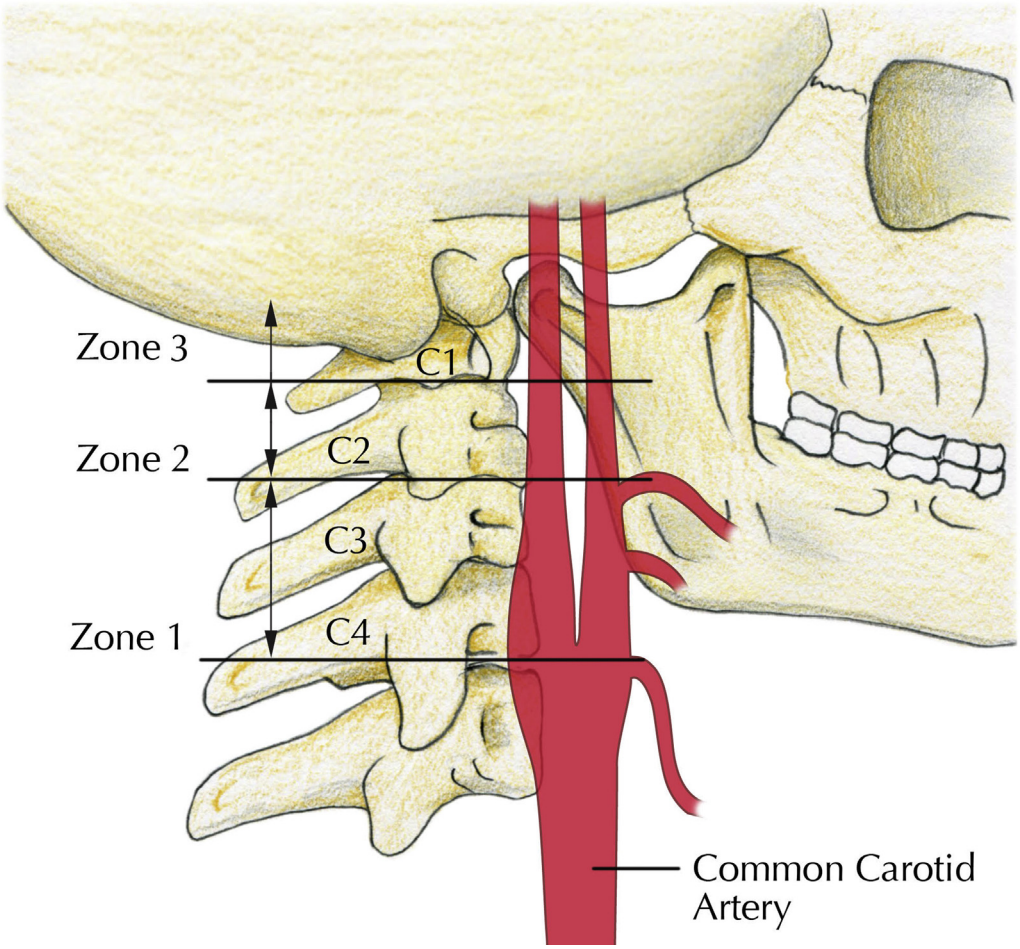

Fig 1. (C1–4) Cervical vertebral body levels. Zone 1: the usual location of plaque in patients with carotid occlusion disease, considered as non-high plaque. Zones 2 and 3: constitutes high plaque location.

Table I
Associated risk factors: High plaque versus non–high plaque

	HP, No. (%) n =100	Non-HP, No. (%) n = 1,133	*P* value
Age, years ± STD	70.9 ± 8.7	70.3 ± 9.1	.53
Sex			
Male	78 (78.0)	749 (66.1)	.02
Female	22 (22.0)	384 (33.9)	.02

Associated diseases and risk factors	HP, No. (%) n = 100	Non-HP, No. (%) n =1,133	*P* value
CAD	53 (53.0)	556 (49.1)	.41
HTN	83 (83.0)	935 (82.6)	1.00
DM	31 (31.0)	394 (34.8)	.51
COPD	19 (19.0)	207 (18.3)	.89
HLD	68 (68.0)	798 (70.4)	.65
CKD	7 (7.0)	79 (7.0)	1.00
Nicotine abuse	68 (68.0)	497 (43.9)	< .001

CAD, coronary artery disease; *CKD,* chronic kidney disease; *COPD,* chronic obstructive pulmonary disease; *DM,* diabetes mellitus; *HLD,* hyperlipidemia; *HTN,* hypertension; *STD,* standard deviation.

Postoperative MI

In group B, MI was diagnosed in 2 patients (0.2%) as compared to none in group A (P = 1.00) as depicted in Table IV.

Table II
High plaque carotid occlusive disease presentation versus non–high plaque

	Group A No. (%), n = 100	Group B No. (%), n = 1,133	*P* value
Acute stroke, <8 wk	15 (15.0)	89 (7.9)	.02
Remote stroke, >8 wk	0 (0.0)	34 (3.0)	.11
TIA	23 (23.0)	338 (29.7)	.17
Asymptomatic	62 (62.0)	672 (59.3)	.67

TIA, transient ischemic attack.

Hematoma requiring re-exploration

Eight patients in group B (0.7%) required re-exploration for hematoma compared to none in group A.

Discussion

CEA for HP can present as a very challenging operation for a vascular surgeon. Difficulty with CEA for HP is exacerbated in patients who have short necks or high carotid bifurcation. Proponents of carotid artery stenting proclaim that high carotid bifurcation or HP are anatomical risk factors for CEA, and CAS should be considered. We generally agree with CAS being preferable to CEA in patients with HP; however, in select patients with severe ICA tortuosity or heavy calcification, CAS may not be a good option. In

Table III
Anesthesia type and shunt requirement during high plaque CEA

	High plaque, No. (%), n =100	Non-high plaque, No. (%), n =1,133	P value
Anesthesia type			
GA	29 (29.0)	602 (53.1)	< .001
CBA	71 (71.0)	531 (46.9)	< .001
	Patients (n = 1,233)	Shunt placement, No. (%)	P value
High plaque	100	3 (3.0)	.009
Non-high plaque	1133	123 (10.9)	

CBA, cervical block anesthesia; *GA,* general anesthesia.

Table IV
Postoperative complications in patients undergoing high plaque CEA

	Group A (n = 100)	Group B (n = 1,133)	P value
Major stroke	3 (3.0)	11 (1.0)	.10
Minor stroke	0 (0.0)	7 (0.6)	1.00
Mortality	2 (2.0)	7 (0.6)	.16
MI	0 (0.0)	2 (0.2)	1.00
Permanent CN injury	2 (2.0)	3 (0.3)	.06
Hematoma requiring re-exploration	0 (0.0)	8 (0.7)	1.00

MI, myocardial infarction.

these circumstances, CEA remains a safe and viable treatment modality.

The safety of CEA in challenging anatomy has been documented previously. In reviewing CEA in high-risk patients, Boules et al found no adverse outcomes in any of the patients (n = 14) with high carotid bifurcation.[13] In addition, Capoccia et al reported the use of mandibular subluxation as a technical adjunct for high carotid bifurcation or challenging carotid lesions (n = 38) with an overall neurologic morbidity rate (major stroke, minor stroke, TIA, peripheral nerve injury) comparable to that of those who underwent a standard CEA.[14] Our results also demonstrate that CEA can be safely performed in patients with HP with similar outcomes compared with patients without nHP.

Several techniques have been described to provide better distal exposure in CEA with challenging anatomy, such as HP. For patients with plaque ending in zone 1, a standard CEA is usually performed without difficulty. Most patients with HP (zone 2) will undergo successful CEA, with additional cephalad dissection and exposure of the ICA above the hypoglossal nerve. In our series, we were able to successfully perform CEA in 98 patients (98.0%) with extension of our dissection cephalad and posterior to the ear. Carotid endarterectomy was performed through an oblique neck incision with patch graft used in vast majority of the patients. In most cases, additional dissection by extension of the skin incision cephalad and posterior to the ear provided adequate additional exposure of the distal cervical ICA. No special retractors were required in our operations; however, the use of special retractors, such as an omnidirectional retractor ring, have been previously described.[15,16] The sternocleidomastoid branch of the occipital artery and vein crossing the hypoglossal nerve are ligated and divided. The hypoglossal nerve is mobilized cephalad. The posterior belly of the digastric muscle is mobilized and, if needed, divided. If additional exposure is required, the styloid process is carefully removed. During removal of the styloid process, care is taken to prevent injury to the glossopharyngeal nerve. In rare cases, the upper end of the posterior tongue of the plaque may reach zone 3. In these circumstances, CAS is preferred if the plaque is not heavily calcified and if the artery is not very tortuous. If CEA is performed, mandibular subluxation may be required. Mandibular subluxation has been proven to be effective, quick, easy to perform, and safe for high carotid exposure.[15,17–19] The use of mandibular subluxation was required in only 2 of our patients (2.0%). In addition, mandibular osteotomy or resection of the mastoid process are alternate strategies to additional exposure. Completion angiography or intraoperative carotid duplex imaging should be performed in cases where the distal end point of the plaque cannot be clearly visualized. If the end point of the CEA remains unclear despite the use of adjunct maneuvers, intraoperative carotid stenting has been previously reported with good outcomes.[20] Intraoperative CAS can be performed by extending the skin incision inferiorly to allow for placement of a 7-Fr sheath in the supraclavicular common carotid artery.

This retrospective analysis has several limitations. Our small sample size may limit our ability to document actual differences between the 2 groups. In addition, all procedures were performed by a single, experienced surgeon. It is unclear whether our outcomes are applicable to low-volume surgeons or settings in which limited availability of supporting services, such as oral or maxillofacial surgery to provide distal exposure maneuvers, if needed. These factors are surely important considerations when contemplating CEA in patients with HP, and CAS should be given strong consideration.

In conclusion, most patients with HP can be diagnosed with CTA of the neck or catheter-based arteriography. Shunt requirement in patients with HP is significantly lower than the nHP group. Perioperative stroke and mortality in patients with HP undergoing CEA is similar to the nHP group; however, there is a higher incidence of permanent cranial nerve injury. CAS should be considered in cases in which CEA may be challenging, such as in patients with HP. However, in patients with highly calcified or tortuous internal carotid artery, CAS may not be the best option. Overall, our results demonstrate that CEA can be safely performed in patients with HP, however, at an increased risk of permanent cranial nerve injury.

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We have nothing to disclose.

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Discussion

Dr Karen Ho (Chicago, IL): Thank you. I enjoyed your paper. I agree. I commend you and your senior author, Dr. Hans, on reminding us in this age of carotid stenting and TCAR that open carotid surgery can and should still be performed in patients with challenging anatomy.

Given the relatively high (8%) prevalence of high carotid plaque in your cohort, do you routinely get cross-sectional imaging in patients before surgery for surgical planning?

Dr Steven Vang: Yes, CT angiography is routinely performed at our institution.

Dr Karen Ho (Chicago, IL): It can be hard to see the internal carotid artery when you get close to the skull base. Is there a retraction system that you like to use? In addition, it can be challenging to secure a shunt when you're high up in the neck. What's your preferred shunt and how do you secure it?

Dr Steven Vang: Absolutely. To answer your question, for our 2 patients who required carotid endarterectomy for very high lesions, we have utilized mandibular subluxation. We did not use any kind of special retractors for patients undergoing carotid endarterectomy for high plaque.

To answer your second question about the requirement of shunt, again, it's difficult to find the exact reason why our data shows that patients with high plaque tend to not require or had a lower requirement of shunt placement. That would require further investigation to find perhaps the etiology. We propose that this is possibly because patients with high plaque have chronic extensive disease that allows for collateralization to develop, which mitigates the requirement of shunt placements. In regards of what type of shunts are used, I would have to defer to Dr. Hans.

Dr Sachinder Hans: We prefer not to use a shunt whenever possible, but when we do need to use the shunt, we use Sundt shunt which is very flexible. Some surgeons have used the Pruitt Inahara shunt, but I have never used that shunt. My mentor passed away 2 years ago at the age of 95 years, 9 months, and he never used a shunt. He had excellent results without the use of shunt. We are always concerned that shunt itself can caused neurological deficit by causing dissection or scraping of the plaque with embolization during insertion. We perform most of our carotid endarterectomies under cervical block anesthesia, however, in patients with high plaque, general anesthesia is preferable. Our shunt usage is 10% in patients undergoing carotid endarterectomy under cervical block anesthesia. Importantly, this does not mean that all 10% of patients in whom shunt was indicated (ie, by EEG changes, low stump pressure, or neurological deficit following carotid artery cross-clamping in awake patients) will develop cerebral infarction if shunt was not used, particularly if reperfusion can be established quickly; they may not develop cerebral infarction.

I think without usage of shunt, carotid endarterectomy has been reported with 2% or 3% stroke rate by Dr Baker et al from our past president's, Dr Luchette, institution (Loyola). Our data shows that patients with high plaque rarely require a shunt. I think the most important point in carotid endarterectomy is get a good endpoint and to perform a technically satisfactory operation so that cerebral reperfusion can be established. I think these are far more important factors than the use a shunt in preventing perioperative neurological deficit. I'm sure if you need a shunt that is technically challenging in a patient with high plaque. Thank you very much.

Dr Karen Ho (Chicago, IL): Could you comment on cranial nerve palsy (transient and permanent) in patients who had high carotid plaque. Which nerves were affected and what were the consequences?

Dr Steven Vang: I would have Dr. Hans share the patients' clinical findings, but for permanent cranial nerve palsy, we classify that as patients who have persistent neurological disorder greater than 3 months out from surgery. These patients do follow up with the ENT, so we have documentation for that in our manuscript.

Dr Sachinder Hans: Usually cranial nerve injury is a result of a clamp or stretch injury as opposed to a transection of the nerve.

The most common nerve injured in patients undergoing carotid endarterectomy is the hypoglossal nerve. It is impressive that even patients with hypoglossal nerve injury show recovery from the hypoglossal nerve palsy. Obviously, recovery from cranial nerve palsy is poor, however, the muscles on the contralateral side of the tongue may occasionally compensate for tongue function. Sometimes, the disability from cranial nerve palsy can become compensated over time with minimal disability. For vocal cord paralysis, we had one patient that recovered in nine months and did not require ENT intervention.

I'm not trying to minimize cranial nerve injury. Obviously, during carotid stenting, there is no reason to get a cranial nerve injury.