CASE REPORT

Distal Cervical Internal Carotid Artery Stenosis

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Abstract—This report describes a case of distal cervical internal carotid artery (ICA) occlusive lesion managed by saphenous interposition vein graft after exposure of the ICA up to the skull base. The skull base approach described herein emphasizes the mobilization of the mandible without mandibular osteotomy or disarticulation. Instead, the stylomandibular and stylo-sphenoidal ligaments are resected to allow mandibular mobilization. In addition, the distal ICA lesion represented a form of diffuse intimal thickening without any plaque formation. This required the resection of the thickened, stenotic segment and reconstruction of the distal cervical ICA with a saphenous interposition graft. The surgical management implications of diffuse intimal thickening are discussed. (*Skull Base Surgery*, 4(2):110–115, 1994)

Carotid artery stenosis, usually a result of atherosclerotic lesions, has a predilection for the bifurcation of the common carotid artery (CCA), sinus portion of the internal carotid artery (ICA), and the curved terminal portion of the ICA.^{1–3} Rarely, the distal cervical segment of the ICA is involved by atherosclerotic lesions causing stenosis. More commonly, however, this segment is a site for fibromuscular dysplasia, dissection, or aneurysms.⁴ Difficulty in gaining access to this segment of the ICA has been a major obstacle to effective operation for lesions at this level. Several authors have described successful approaches to this segment.^{4–10} Some of these techniques require mandibular osteotomies or disarticulation, or both.^{5,6,10} We used a modification of the technique described by Sundt et al⁴ to approach a distal cervical ICA lesion. This lesion was unusual in that it represented a tandem lesion and did not show the typical changes of atherosclerosis. The diffuse intimal thickening (DIT) in the distal cervical ICA segment resulted in a severe stenosis and required a saphenous vein reconstruction of the ICA. The surgical implications and histopathologic aspects of this case are described.

CASE REPORT

History

A 55-year-old white woman presented to the emergency room with right hemisphere transient ischemic at-

tacks (TIAs). These events were described by the patient to involve mainly her left hand and leg: transient (12 to 24 hours) paresthesias and weakness. These events had been progressively increasing in frequency over the previous 3 months prior to admission. A duplex ultrasound of the cervical carotid arteries revealed bilateral stenosis of the cervical ICA at the bifurcation: right > left. Patient's medical history was significant for hypertension. The neurological examination on admission did not reveal any focal deficits. There were no carotid bruits.

Radiological Studies

The cranial computed tomography scan revealed no evidence of an infarct. Cerebral angiographic study revealed four lesions (Fig. 1): (1) right middle cerebral artery (MCA) bifurcation aneurysm; (2) 80% stenosis at the origin of the right proximal cervical ICA; (3) 90% stenosis of the right distal cervical ICA, approximate level of C1–2; (4) 50% stenosis at the origin of the left proximal cervical ICA.

Operative Technique

Although the right carotid bifurcation lesion was most likely the cause of this patient's TIAs, we thought that the distal lesion was significant enough to contribute

Figure 1. Preoperative right carotid angiogram, anteroposterior (A) and oblique (B) views reveal three lesions: (1) 80% stenosis at the origin of the proximal cervical internal carotid artery (ICA) (arrow); (2) 90% stenosis of the distal cervical ICA (arrowhead); (3) middle cerebral artery bifurcation aneurysm (curved arrow).

to the decrease of blood flow and that it may not reopen after a proximal endarterectomy. Thus, a skull base approach to the distal cervical ICA was planned in addition to a proximal endarterectomy.

The procedure was performed under general anesthesia and with intraoperative electroencephalography (EEG). The standard carotid endarterectomy incision paralleling the anterior border of the sternocleidomastoid muscle was extended superiorly in front of the tragus (in the pretragal skin crease) and up to the zygoma (Fig. 2). The initial dissection was started in the posterior border of the parotid gland, which was mobilized anteriorly and the "pointer" cartilage identified (Fig. 3). The temporoparotid fascia was incised between the mastoid process and the parotid gland, and the facial nerve trunk was found below the fascia at the posteromedial surface of the parotid gland, using the "pointer" as a guide. After the main trunk of the facial nerve was identified, it was dissected in the parotid tissue up to its main division. The cervicofacial division and specifically the marginal mandibular nerve form the upper limit of the deeper dissection. This dissection is essential in gaining access to the deeper ligaments and

muscles that need to be divided to approach the skull base as well as to protect the facial nerve.

Standard proximal dissection in the anterior triangle of the neck was performed next to expose the CCA and its bifurcation, the internal jugular vein, the vagus and hypoglossal nerves. The carotid sheath was opened and the CCA, external carotid artery, and ICA as well as the superior thyroid and ascending pharyngeal and occipital arteries were fully dissected. The occipital artery was doubly ligated as it coursed over the superior margin of the hypoglossal nerve. This step is necessary to expose the distal cervical ICA.

The ICA was now dissected distally toward the skull base (see Figs. 3 and 4). The division of the posterior belly of the digastric muscle at its insertion with subsequent resection of the stylomandibular and stylohyoid ligaments is essential for adequate mobilization of the mandible and exposure of the distal cervical ICA. The mandible does not need to be resected or disarticulated. In addition, the styloid process also needs to be partially resected to provide a good exposure to the distal ICA.

The distal cervical ICA lesion was attacked first. This

Figure 2. Diagrammatic representation of proposed skin incision to approach the distal and proximal cervical internal carotid artery. The standard carotid endarterectomy incision paralleling the anterior border of the sternocleidomastoid muscle is extended superiorly in front of the tragus and up to the zygoma.

lesion represented a form of diffuse intimal thickening without any plaque formation or ulceration. Thus, there was no plane of dissection between the thickened area and the underlying intima. This required the resection of the thickened segment and reconstruction of the distal cervical ICA with a saphenous vein interposition graft (4 cm), which was harvested from the right medial lower leg. The procedure was performed under microscope magnification. No EEG changes were observed throughout the reconstruction. Good flow and pulsatility of the graft were noted. Figure 5 shows an intraoperative view of the graft. Subsequent to the reconstruction, a standard carotid endarterectomy of the proximal stenosis was performed without complications.

Postoperative Course and Follow-up

The patient had an uneventful postoperative recovery. She had no cerebral hemispheric deficits. All cranial nerves, except the right recurrent laryngeal nerve, were intact postoperatively. The patient had hoarseness postoperatively, which has shown improvement in the 4-month follow-up examination. In the 4-month follow-up visit, the patient did not report any TIA's. Postoperative angiogram reveals patency of the graft and good flow through the right ICA (Fig. 6). In addition, the patient was offered surgical clipping of the right MCA aneurysm at 6 weeks postoperatively as well as at the 4-month follow-up, but she refused.

Figure 3. Diagrammatic representation of the intraoperative exposure. Note that the parotid gland as well as the facial nerve have been mobilized. The external ear canal cartilage serves as a guide to the facial nerve after its exit from the stylomastoid foramen. The posterior belly of the digastric muscle and the stylomandibular ligament have to be resected to mobilize the mandible and provide good exposure to the distal internal carotid artery.

Figure 4. Intraoperative view showing the dissection of the distal internal carotid artery (black arrow-head) toward the skull base. The posterior belly of the digastric muscle, stylomandibular ligament, and the styloid process have been resected. The white vessel loop is around the facial nerve trunk under the parotid gland (black arrow). Occipital artery has been ligated to aid in the distal dissection (note the black suture). Shown also are the common carotid artery (white arrow), external carotid artery (white arrowhead), and hypoglossal nerve (curved arrow).

DISCUSSION

Distal cervical ICA lesions have usually been treated conservatively or by carotid ligation because of the difficulty of gaining access to this region.⁶ Sundt et al,⁴ however, have emphasized the importance of directly treating certain distal lesions, especially aneurysms. In addition, the rare solitary distal atherosclerotic lesions have been shown to be accessible and should be treated.^{6,11}

Tandem Lesions

Tandem stenotic lesions usually occur in the proximal ICA and in the intracavernous or intracranial portion

of the ICA.^{1,2,12} The management of these lesions is controversial. Most authors believe that carotid endarterectomy of a significant proximal lesion is indicated for TIAs even if the distal lesion is also significantly stenotic.¹² One study did not reveal any relationship between the severity and presence of carotid siphon stenosis and the risks of recurrent symptoms after a proximal carotid endarterectomy.¹³ Another study, however, showed a high risk of cerebral infarction in patients with siphon stenosis.¹⁴ Our case represents an unusual problem of two significant stenotic lesions located on the same vessel with one of the lesions being in the distal cervical ICA. The management of a similar tandem lesion is not clear in the literature. We believe that the distal lesion was significant enough to diminish blood flow despite an open proximal ICA. Thus, an approach to this lesion was planned in addition to a standard proximal carotid endarterectomy.

Figure 5. Intraoperative view of the saphenous vein graft (curved arrow). Hypoglossal nerve is indicated by an arrowhead.

Figure 6. Postoperative right carotid angiogram, oblique (A) and the lateral (B) views. Saphenous vein graft (curved arrow) appears patent and there is good flow through the internal carotid artery system. Note the proximity of the vein graft to the skull base in B.

Intracranial Aneurysm and Carotid Stenosis

The association of high-grade cervical ICA stenosis with ipsilateral intracranial aneurysm is recognized in the literature.¹⁵ In patients with TIAs, it is thought that the cervical ICA stenosis is the source of these symptoms and resolution of these symptoms is usual after a carotid endarterectomy.¹⁶ However, embolus from an aneurysm may also rarely cause similar symptoms. Moreover, most studies conclude that there was no additional risk in performing a carotid endarterectomy in patients harboring an asymptomatic aneurysm.¹⁶ The only precaution one should take in these cases is to avoid intraoperative hypertension. Thus, we elected to manage the cervical ICA stenosis before addressing the aneurysm.

Approaches to the Distal Cervical Internal Carotid Artery

Several authors have described surgical approaches to the distal cervical ICA. Techniques have included resection of the mastoid process and release of the sternocleidomastoid muscle,¹⁷ resection of the styloid process,¹⁸ division of the styloid and digastric muscles,¹⁹ en block

resection of the mastoid process and portion of the mandible,²⁰ mandibular osteotomies, and disarticulation.^{5,6,10} Of note, Fisch et al,⁸ Purdue et al,⁹ and Sekhar et al⁷ describe more extensive techniques to expose the distal cervical and petrous ICA. Sundt et al⁴ describe a less extensive preauricular approach with mobilization of the parotid gland and the facial nerve.⁴ Our technique is a modification of Sundt et al's approach. The segment of the cervical ICA between the styloid process and entrance to the carotid canal is probably the most difficult to expose.⁷ To facilitate its exposure, certain key steps are necessary. These include mobilization of the parotid gland and facial nerve and resection of the stylomandibular and stylo-sphenoidal ligaments, followed by partial resection of the styloid process. These steps allow mobilization of the mandible without performing osteotomies or disarticulating the mandible.

Distal Internal Carotid Artery Lesions

Vascular lesions of the distal cervical ICA include aneurysm, dissection, fibromuscular dysplasia, and atherosclerotic plaques.^{4,7} Distal cervical ICA aneurysms are usually caused by trauma, spontaneous dissection, fibromuscular dysplasia, atherosclerosis, or infection.⁴ It is this

lesion that usually requires a direct approach to the distal cervical ICA. Stenosis secondary to atherosclerotic disease is unusual at this site. Most studies have shown the cervical carotid bifurcation, siphon, or intracranial ICA bifurcation as the more common sites of stenosis and specifically of atherothrombotic disease.¹⁻³ When a distal cervical ICA stenosis is secondary to atherosclerosis, a typical atheroma or plaque is dissected from the arterial wall.⁶ In our case, there was no plaque formation; instead, the stenotic lesion represented DIT. This process represents physiological growth of the arterial intima that begins at birth and progresses to adult life.²¹ Hypertension and other forms of hemodynamic stress may accelerate and aggravate DIT.³ In addition, there is evidence that DIT may predispose to the development of atherosclerosis. Arteries with minimal DIT are usually not the site of atherosclerotic lesions, whereas those with prominent DIT eventually show atherosclerotic plaques.²² Intimal thickening may, thus, form an early stage of atherosclerosis. In addition, as Lie³ points out, the distinction between DIT and an atherosclerotic lesion can be blurred when the latter lesion consists mainly of fibrocellular proliferation of the intima, without lipid deposits or calcification. Thus, as our case emphasizes, a distal cervical ICA stenotic lesion may not consistently represent an atherosclerotic plaque and the surgeon should be prepared to resect and reconstruct the artery at this level.

CONCLUSION

This report highlights the surgical management of distal cervical ICA lesions. These lesions may not represent typical atherosclerotic plaques, but instead be a form of diffuse intimal thickening. Thus, when approaching distal cervical ICA occlusive lesions the surgeon should be prepared for possible reconstruction of the artery.

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