Gradual Facial Palsy and Intrapetrous Internal Carotid Aneurysm: A Case Report

Abstract—A case of aneurysm of the horizontal intratemporal carotid artery with compression of the facial nerve is presented. Exclusion of the aneurysm with an intraluminal balloon did not relieve the facial nerve compression. Surgical resection of the aneurysm and repair of the facial nerve continuity was successfully performed through a combined transmastoid supralabyrinthine approach, after permanent balloon occlusion of the internal carotid artery. (*Skull Base Surgery*, 3(3):164–169, 1993)

Gradual facial paralysis without other clinical symptoms and with normal ear findings most often arises from supralabyrinthine tumors, such as cholesteatomas, facial nerve neurinomas, hemangiomas, and meningiomas. Intrapetrous internal carotid aneurysms as the cause of facial palsy are very rare.^{1,2} These lesions are usually situated at the carotid foramen or in the proximal vertical carotid segment. Brandt et al³ described the only case of aneurysm of the horizontal segment of the carotid artery inducing a facial paralysis in a 16-year-old boy with multiple aneurysms of the petrous apex.

The purpose of this article is to describe another case of gradual facial palsy caused by an aneurysm of the horizontal segment of the petrous internal carotid artery (ICA) and to discuss guidelines for the management of these lesions.

CASE HISTORY

A 47-year-old woman was referred to the Ear, Nose, and Throat Department of the University Hospital of Zürich for the evaluation of right-sided gradual facial palsy of 7 months duration. Her subjective complaints were headache and intermittent dysgeusia. The patient

denied hearing loss, tinnitus, and vertigo. On examination, she had a right total peripheral facial paralysis but no anomaly of the other cranial nerves and no nystagmus (Fig. 1A,B). Her tympanic membranes showed some tympanosclerotic plaques but were otherwise normal. There was a 20 dB conductive loss in the right pure-tone audiogram.

Because of the progressive evolution of the palsy and the conductive hearing loss, the presence of a supralabyrinthine tumor was suspected. The high resolution computed tomography scan revealed bone destruction at the geniculate ganglion, consistent with the diagnosis of facial neuroma. Magnetic resonance imaging disclosed a 1.3 cm aneurysm of the horizontal segment of the ICA at the level of the geniculum of the facial nerve (Fig. 2). This was subsequently confirmed by carotid angiography (Fig. 3). In the course of angiography, a balloon was selectively placed into the aneurysm, inflated, and detached. In this way, the aneurysm was excluded from the carotid flow. A repeat angiogram, 7 days later, showed a normal carotid flow and a thrombosed aneurysm.

Two weeks later, the facial function slightly improved in the mouth and the conductive hearing loss had disappeared. However, 6 months later, the patient still had a complete facial palsy. Repeat angiography disclosed partial recanalization of the aneurysm (Fig. 4). It was there-

Figure 1. A, B: Patient before surgery with right total facial paralysis. C, D: Patient 1 year after surgery showing 65% recovery of facial movements.

Figure 2. Preoperative axial T1-weighted magnetic resonance image after administration of paramagnetic contrast material (gadolinium-DTPA). There is a round, low-signal intensity lesion at the proximal horizontal segment of the carotid canal (arrow), compatible with an aneurysm.

fore decided to occlude the ICA permanently by balloons and to explore surgically the facial nerve.

Preoperatively, the tolerance to acute occlusion of the ICA was tested angiographically and clinically. During temporary balloon occlusion of the ipsilateral ICA at the level of the cavernous segment, contralateral carotid angiography showed sufficient supply of the ipsilateral hemisphere through the anterior communicating artery. No neurologic deficit was observed during 20 minutes of occlusion. Therefore the ICA was permanently occluded

by two balloons, one detached at the cavernous segment and the second at the level of the carotid foramen, thus trapping the aneurysm. Surgery was performed 1 month later using a combined transmastoid and supralabyrinthine (middle cranial fossa) approach.

The occluded aneurysm was found to compress the distal part of the labyrinthine segment of the facial nerve as well as to be nearly in contact with the malleus head. A large scar neuroma had developed proximal to the compression site. The thrombosed aneurysm was opened, the

Figure 3. Preoperative lateral carotid angiography showing a medium-sized irregularly shaped aneurysm with a broad base on the proximal part of the horizontal segment of the petrous ICA.

Figure 4. Lateral carotid angiography 6 months after selective balloon occlusion of the aneurysm showing partial recanalization of the aneurysm.

partially inflated balloon removed, and the greatest portion of the aneurysm excised. No bleeding occurred from the thrombosed ICA.

The fibrotic facial nerve was resected from the proximal labyrinthine to the proximal mastoid segment. This wide neural resection is routinely performed in facial nerve lesions requiring meatotympanic rerouting with grafting, since it has yielded better functional results than the use of shorter grafts.⁴ The meatomastoid rerouting of the fallopian canal was performed by drilling the new fallopian canal along the superior ampulla.

A 6 cm graft was obtained from the right greater auricular nerve and cut in two 3 cm segments. The epineurium was removed for 3 mm for each end of the grafts. The proximal and the distal stumps of the nerve were split to accommodate double nerve graft. The continuity of the nerve was reconstructed with two nerve grafts. The proximal anastomosis was fixed with one perineural 10-0 Vicryl suture. No suture was used for the distal anastomosis. Both anastomoses were covered with a collagen membrane (Cargile) to prevent early ingrowth of fibroblasts and to promote direct axon growth. The incus was removed to allow placement of the nerve grafts.

The pathology report revealed that the wall of the ICA was fibrotic but without inflammatory infiltration. The specific staining for elastin fibers showed a pronounced splitting of the elastic layer of the tunica interna (Figs. 5, 6). The presence of a large scar neuroma at the geniculum of the facial nerve was confirmed histologically. The mastoid segment of the facial nerve presented with extensive wallerian degeneration.

The patient had an uneventful recovery, in particular no neurologic deficit in relation to the permanent occlusion of the ICA was observed. She was discharged from the hospital after 11 days. At the last checkup, 1 year after surgery, a 65% (House grade III) return of facial function

was visible (Fig. 1 C,D). An incus interposition was performed with local anesthesia at this date. The air-bone gap was closed at the 20 dB level 3 month postoperatively.

DISCUSSION

Pathology

There are two types of aneurysms: pseudoaneurysms (false or traumatic aneurysm), which result from a disruption in the three layers of the arterial wall, and true aneurysms, which have continuous walls with the unaffected portion of the parent vessel. True aneurysms at the cervical course of ICA usually have a fusiform shape and are frequently associated with atherosclerosis. Atherosclerosis is rarely found within the petrous segment of the ICA and therefore is not the major factor of origin of aneurysm in this region.¹

Congenital aneurysms are a particular form of true aneurysms. They originate from developmental defects in the middle muscular layer of the artery. Such defects have been found at the acute angle of vascular branches of the petrous ICA, the caroticotympanic, the cavernous, and the ophthalmic arteries.¹ The caroticotympanic artery is at the origin of most congenital intrapetrous aneurysms.

In our case, the final diagnosis was of a true congenital saccular ICA aneurysm. The histologic appearance of the carotid wall excludes a mycotic or autoimmune origin. Chronic infection or surgical trauma may induce carotid aneurysms, which erode the bony lamella of the carotid canal and facilitate the formation of an aneurysm.^{2,3,5-7} If one compares the high incidence of chronic otitis and the low occurrence of aneurysms, this association seems unlikely. Furthermore, no chronic otitis or surgical trauma occurred in our patient.

Figure 5. Overview of resected aneurysm. The wall is continuous and irregularly thickened by fibrotic tissue; there is no inflammatory infiltration. (H&E with elastin; $\times 65$.)

Figure 6. High-power view of the resected aneurysm wall showing splitting of the elastic layer of the tunica interna. ($\times 200$.)

Treatment

In the past, expectant observation was the treatment of choice of intrapetrous aneurysms. However, the mortality was as high as 70%.² Ligation of the ICA at the neck was performed to promote aneurysmal thrombosis by reducing the pressure along the proximal carotid segment, when angiography with cross-compression and electroencephalographic monitoring established the existence of valid contralateral circulation.

The complications of proximal ligation of the internal carotid artery were not insignificant.¹ Roski et al⁸ published their experience with 16.6% transient ischemic attack, 16.6% cerebrovascular accident, and 5.5% subarachnoid hemorrhage. The rate of mortality ranges from 0 to 15%.⁹

Cerebral protection by hypothermic anesthesia with or without barbiturates has reduced morbidity and mortality rates from 22 to 36% and 4 to 7% to 0.3% and 0 to 3%.² Aneurysmal resection through an infratemporal fossa approach with restitution of the arterial continuity by graft (saphenous vein or prosthetic material) or by rerouting and end-to-end anastomosis has been successfully used for lesions situated close to the carotid foramen.² Utilization of an internal shunt protects against brain ischemia during the invention. Reconstruction of the horizontal carotid artery after aneurysmal resection is possible, but technically more difficult. Selective introduction of a balloon into the aneurysm permits exclusion of the aneurysm from the main carotid flow, maintaining normal blood circulation in the main vessel. The disadvantage of this technique resides in the persistence of the mass effect of the thrombosed aneurysm. When this mass effect is responsible for functional disorders such as facial paralysis, conductive hearing loss, or obstruction of eustachian tube, the excision of the aneurysm becomes mandatory.

This sequence of events is well documented in the present patient.

Preoperative endovascular permanent balloon occlusion (PBO) of the ICA, according to the technique described by Valavanis and Fisch,⁹ permits aneurysmectomy and functional reconstructive surgery at low risk.

Preoperative PBO is, of course, only possible if sufficient cross-circulation is present. The patient's tolerance to occlusion of the internal carotid artery is tested by a temporary balloon occlusion of the vessel. Through a transfemoral route, a double-lumen nondetachable balloon catheter is introduced into the ICA and inflated until the artery is completely occluded. A second conventional angiographic catheter is then placed with its tip proximal to the balloon. Contrast material injected into the second catheter is observed to stagnate when the arterial blood flow is completely interrupted. Heparinized saline is continuously injected into the occluded vessel to prevent thrombosis. This test is performed for 15 to 20 minutes in local anesthesia while the patient is monitored clinically and by electroencephalogram (EEG) for the development of neurologic deficits. The development of neurologic symptoms or an abnormal EEG requires immediate deflation of the balloon.

To occlude the ICA permanently, the double-lumen nondetachable balloon catheter is replaced with a thin-walled introducer catheter, placing the tip in the proximal part of the vessel. A detachable latex balloon coupled to a coaxial microcatheter is inserted through the introducer catheter. The balloon is manipulated into the cavernous segment of the ICA just proximal to the origin of the ophthalmic artery and is fully inflated with isotonic contrast material. After angiographic demonstration of complete occlusion of the ICA and patency of the ophthalmic artery, the inflated balloon is detached by gently pulling the coaxial microcatheter under fluoroscopic control. In

similar manner, a second detachable balloon is positioned in the extracranial part of the ICA.⁹⁻¹¹

The patient is kept in the intensive care unit for 24 to 36 hours with monitoring of neurologic signs and blood pressure. The development of ischemic neurologic deficits or a systolic blood pressure less than 100 mmHg is treated with administration of fluids, plasma expanders, and vasopressive drugs.^{9,10} If the patient does not develop neurologic deficits after temporary occlusion, this technique permits the avoidance of an aneurysmectomy with restitution of arterial continuity.¹ Surgical resection of the aneurysm of the horizontal part of the ICA at the pyramid apex would have been an hazardous procedure if the contralateral cerebral blood flow had been insufficient or in the presence of bilateral aneurysms. In our case, the aneurysm exclusion through a balloon did not induce sufficient nerve decompression. Aneurysmectomy became necessary for facial nerve reconstruction.

SURGERY

The progressive facial palsy was induced by aneurysmal compression of the distal labyrinthine segment of the facial nerve. A large scar neuroma developed proximal to the aneurysm. Resection of the traumatized nerve segment with meatotympanic rerouting along the superior ampulla and double grafting using the greater auricular nerve is a new established technique.⁴ The patient has recovered 65% (Fisch grading) of facial movements. This satisfactory result corresponds to the upper level of a House grade III. The preoperative conductive hearing loss was due to the compression of the aneurysm on the mal-

leus head. Its resolution after balloon occlusion was probably due to the reduction in size of the aneurysm after thrombosis.

REFERENCES

1. Glasscock ME, Smith PG, Bond AG, Whitaker SR, Bartels LJ: Management of aneurysms of the petrous portion of the internal carotid artery by resection and primary anastomosis. *Laryngoscope* 93:1445-1453, 1983
2. Fisch UP, Oldring DJ, Senning A: Surgical therapy of internal carotid artery lesions of the skull base and temporal bone. *Otolaryngol Head Neck Surg* 88:548-554, 1980
3. Brandt TW, Jenkins HA, Coker NJ: Facial paralysis as the initial presentation of an internal carotid artery aneurysm. *Arch Otolaryngol Head Neck Surg* 112:198-202, 1986
4. Fisch U, Lanser MJ: Facial nerve grafting. *Otolaryngol Clin North Am* 24:691-707, 1991
5. Moskowitz D, Lee K-J, Goodrich I: Intrapetrous internal carotid aneurysm presenting as a middle ear tumor. *Otolaryngol Head Neck Surgery* 91:326-330, 1983
6. Shimizu T, Sakakura Y, Yamagiwa M, Hori M, Yuasa H, Murata M: Aneurysm of the extracranial carotid artery. *Arch Otolaryngol Head Neck Surg* 112:203-206, 1986
7. Heesselink JR, Weber AL: Petrous carotid aneurysm. *Ann Otol Rhinol Laryngol* 92:207-208, 1983
8. Roski RA, Spetzler RF, Nulsen FE: Late complications of carotid ligation in the treatment of intracranial aneurysms. *J Neurosurg* 54:583-587, 1981
9. Andrews JC, Valavanis A, Fisch U: Management of the internal carotid artery in surgery of the skull base. *Laryngoscope* 99:1224-1229, 1989
10. Valavanis A, Fisch U: Balloon-occlusion of the internal carotid artery in extensive skull base tumors. In Fisch U, Valavanis A, Yasargil MG, (eds): *Neurological Surgery of the Ear and the Skull Base*. Kugler & Ghedini, 1989, pp 81-86
11. Triebel HJ, Grzyska U, Frehrking M, Zeumer H: Clinical and MRT follow up of carotid aneurysm following balloon embolisation. *Radiologie* 29:427-431, 1989