Peripheral Facial Paralysis and Bilateral Carotid Pseudoaneurysms of Petrous Localization: A Case Report

Justine Lerat, M.D.,[1] Stéphane Orsel, M.D.,[1] Charbel Mounayer, M.D., Ph.D.,[2] Roberto Riva, M.D.,[2] Pierre-Yves Roudaut, M.D.,[3] Vincent Patron, M.D.,[1] Jean-Pierre Bessede, M.D., Ph.D.,[1] and Karine Aubry, M.D., Ph.D.[1]

ABSTRACT

Carotid pseudoaneurysms of petrous localization are rare. They are mostly due to trauma, tumoral or infectious diseases, or a result of iatrogenic complications after skull base surgery. Symptoms such as facial paralysis are exceptional and have rarely been described in the literature until now. We report the case of a 64-year-old woman, who developed left peripheral facial paralysis induced by two carotid pseudoaneurysms in their intrapetrous section. The treatment is endovascular, despite the high morbidity rate. She was first put on antiplatelet medications, before the left carotid aneurysm was bypassed thanks to a self-expanding pipeline-type stent with flow diversion. The left peripheral facial paralysis was due to the compression exerted by the left carotid aneurysm, probably a congenital malformation. The progressive palsy recovery was fist due to the aneurysmal thrombosis, then to the secondary fibrosis.

KEYWORDS: Peripheral facial paralysis, carotid pseudoaneurysm, petrous localization, endovascular treatment, self-expanding stent

Carotid pseudoaneurysms of petrous localization are rare. They are mostly due to trauma, tumoral or infectious pathologies, or a result of iatrogenic complications after skull base surgery. These aneurysms may enlarge and have such complications as thromboembolism or rupture. Symptoms such as facial paralysis are exceptional and have rarely been described in the literature until now. The treatment is endovascular, despite the high morbidity rate. We report the case of a 64-year-old woman who developed left peripheral facial paralysis induced by bilateral carotid pseudoaneurysms in their petrous section.

CASE REPORT

A 64-year-old woman presented with sudden brutal and isolated left peripheral facial paralysis (grade V in the House-Brackmann classification). It had appeared the day before her admission to the emergency department of our teaching hospital. Her medical history was noncontributory and she was on no current treatment. She had had no recent trauma or infectious disease. Nor did she suffer from any vertigo, tinnitus, or deafness.

The clinical examination revealed grade V left peripheral facial paralysis, whereas the rest of the neurological examination proved strictly normal. The ENT

Departments of [1]Otolaryngology, Head and Neck Surgery, [2]Interventional NeuroRadiology, and [3]Radiology, Dupuytren Teaching Hospital, Limoges, France.

Address for correspondence and reprint requests: Justine Lerat M.D., Department of Otolaryngology, Head and Neck Surgery, Dupuytren Teaching Hospital, Limoges, 2 Avenue Martin-Luther-King, 87042 Limoges Cedex, France (e-mail: justine.lerat@gmail.com).

Skull Base Rep 2011;1:133–138.

Received: April 27, 2011. Accepted: May 6, 2011. Published online: July 22, 2011.
DOI: http://dx.doi.org/10.1055/s-0031-1284209.
ISSN 2157-6971.

examination did not highlight any cervical adenopathy, and the oropharynx showed no abnormality. Otoscopy found two normal eardrums. The Ramsay-Hunt zone did not reveal any sign of cutaneous eruption, and there was no sign of parotitis or mastoiditis.

The biological results did not show any inflammatory or infectious syndrome (CRP: 1, white blood cells: 7000/mm^3, hemoglobin: 13.4 g/dL). A first computed tomography (CT) scan without injection did not find any sign of intra- or extra-axial hemorrhage and no expanding process was detectable.Tests for HIV 1 and 2 and Lyme disease proved negative, whereas tests for Epstein-Barr virus, cytomegalo-virus, varicella-zoster virus, and herpes simplex virus-1 and -2 found traces of previous infections. The patient was discharged with a prescription for a combination of steroids, such as prednisolone per os 1 mg/kg and acyclovir, together with ophthalmologic care and facial physiotherapy.

On the 8th day, the neuronography (ENoG) showed a left facial paralysis with good prognosis (with a 62% of denervation), which did not prompt intervention for facial decompression.

A month later, the patient presented with grade IV left peripheral facial paralysis, while the rest of the clinical examination was unchanged. The tonal and vocal audiograms were normal. CT scan of the petrous bone then highlighted lytic images of the right and left petrous apices, but no abnormality in the middle or internal ear. The posterosuperior part of this picture seemed to be located in front of the left geniculate ganglion fossa. The lesion was hypervascular and was enhanced soon after injection. Mass effect was also found on the posterior part of the petrous course of the left internal carotid artery. Mild osteolysis of the right petrous bone was also found with regular peripheral osteosclerosis together with the same hypervascular characteristics as on the left side (Figs. 1 and 2).

MRI of the petrous bones revealed heterogeneous pictures, prevalent on the left, hypointense on T1 sequence, isointense on T2 sequence, and definitely enhanced after injection; they were 19 × 16 mm on the left side and 11 × 13 mm on the right side (Fig. 3). In addition, there was neither deformation of the petrous apices nor any enhancement of the various portions of the left or right facial nerve.

An arteriography was performed to determine the nature of these hypervascular lesions. Two important pseudoaneurysms were found on the petrous segment of the two internal carotids; they were 2 and 1.5 cm in diameter, respectively, on the left and on the right side (Figs. 4 and 5). In addition, there was no arterial or arteriovenous intracranial malformation, and the renal arteries arteriography did not find any sign of dysplasia.

A two-step endovascular treatment was then performed to bypass these aneurysms; thanks to self-expanding pipeline-type stents with flow diversion so as to avoid carotid occlusion. After the administration of antiplatelet medication (clopidogrel), the left aneurysm was first treated as it was responsible for facial paralysis. A 4.5-mm diameter and 20 mm long stent was inserted through the left carotid aneurysm. The postinterventional care was then uneventful and the patient was discharged on the 6th day.

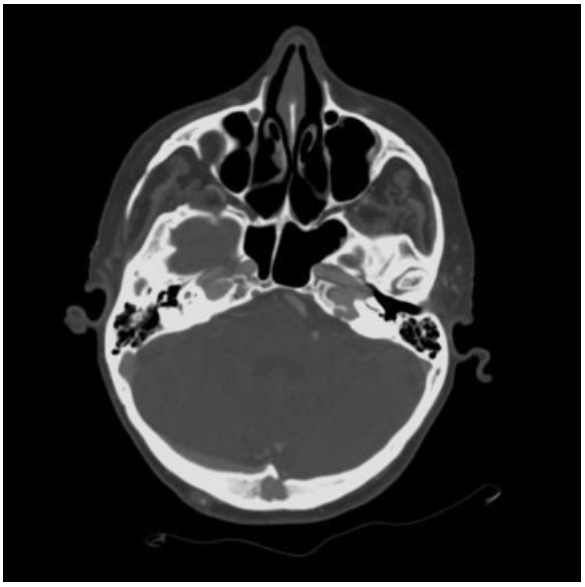

Figure 1 CT scan of the horizontal section of the petrous bone: Lytic images of the right and left petrous apices. There is a lacunar picture with peripheral osteosclerosis of the left petrous apex, located in front of the internal auditory meatus, inside the posterior part of the carotid canal without cortical osteolysis.

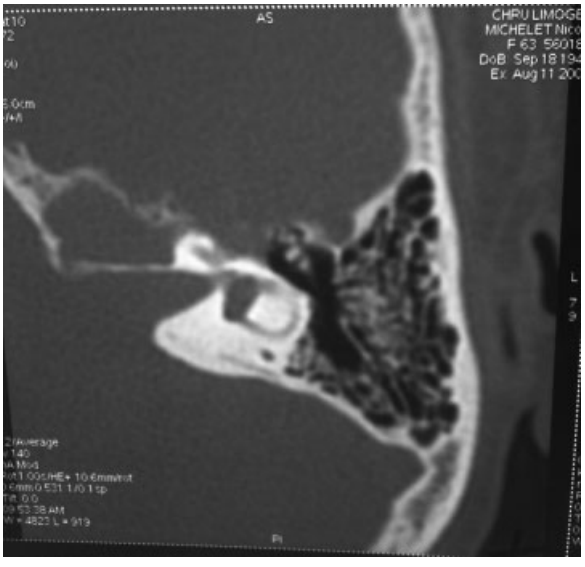

Figure 2 Axial CT scan: The lesion formed an angle on the level of the infratemporal fossa and the anteroexternal pericochlear bone showed signs of lysis, with a quasidenudation of the external bone wall of the basal turn of the cochlea in its anteroinferior part. Axial slide close to the geniculate ganglion.

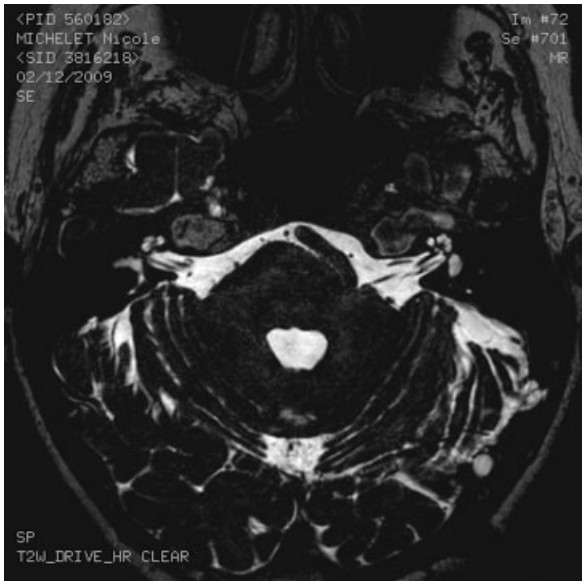

Figure 3 MRI of the petrous bones on T2 sequence with Gadolinium injection: Important enhancement of the petrous apices.

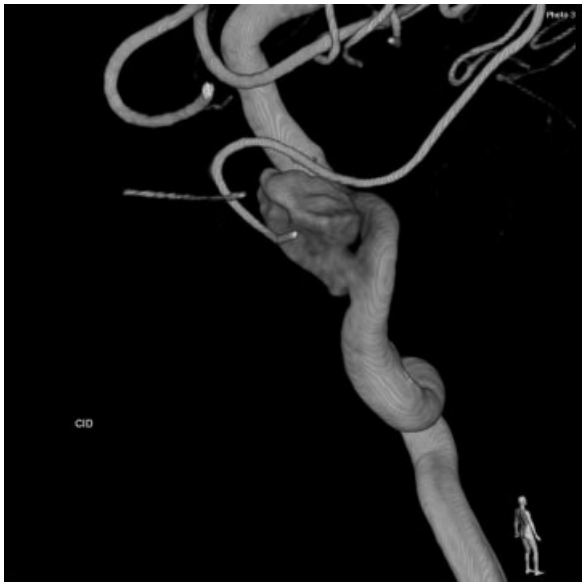

Figure 5 Three-dimensional reconstruction of this right intrapetrous aneurysm.

Angiographic control confirmed on-going left stent thrombosis 2 months later. The right internal carotid arteriography control confirmed the presence of a 14-mm large aneurysm in its petrous segment. Moreover another 5.3 mm dissecting aneurysm was identified upstream. Two 4.75 × 20 mm and 4.5 × 14 mm pipeline stents were thus positioned (Fig. 6). Postinterventional care was uneventful and the patient was discharged on the 4th day with a three-month prescription for clopidogrel. Thereafter, the left peripheral facial paralysis decreased to grade III, then to grade II. Antiplatelet medication with salicylic acid was maintained as a long-term treatment.

A new angiographic control was performed 6 months later and confirmed the total occlusion of the right petrous aneurysm and the persistence of a 5-mm neck permeability of the left carotid aneurysm, which confirmed the continuing aneurysm thrombosis. An angiographic control remained to be scheduled a year later.

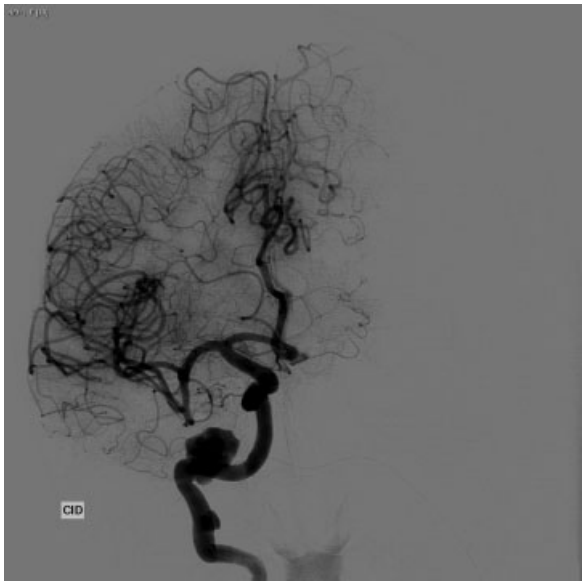

Figure 4 Angiography: Right carotid aneurysm in the petrous segment (1.5 cm in diameter).

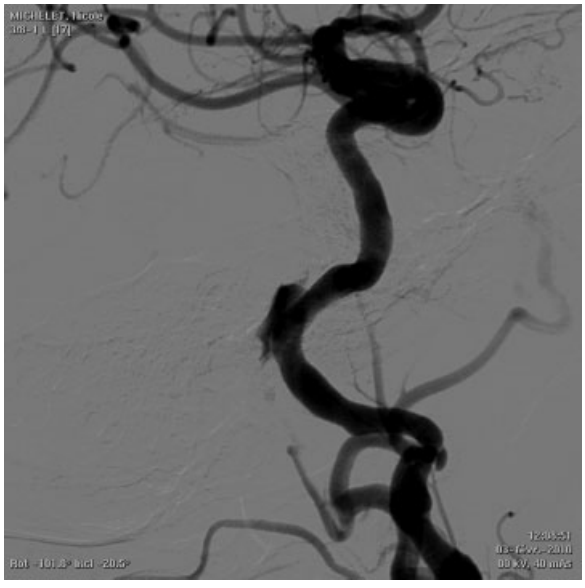

Figure 6 Angiographic control after right carotid aneurysm occlusion.

DISCUSSION

Intrapetrous carotid aneurysms usually sit in the carotid foramen or in the proximal vertical segment.[1]

Pseudoaneurysms, whether false or traumatic, result from an interruption in the three layers which constitute the artery wall.[1] Arteriosclerosis is rarely found within the petrous segment of the carotid artery and is not a major risk factor for aneurysm in this area.

Pseudoaneurysms of the internal carotid artery in its petrous portion can occur after trauma, infections, dissection, invasive tumors, or surgical complications. They can also be of unknown etiology, as was the case with our patient.[2]

A case of intrapetrous pseudoaneurysm has been described in fibromuscular dysplasia. It is a noninflammatory and nonatherosclerotic arteriopathy which affects medium arteries with multifocal stenoses and microaneurysms. It is intracranial in 20% of the cases.[2]

Petrous aneurysms can also be of mycotic origin, as in chronic otitis media. Because of the closeness of the middle ear and the Eustachian tube, the infection of the arterial adventice weakens the artery wall and allows the aneurysm to form.[3]

Brandt et al described a case of aneurysm of the horizontal segment of the carotid which induced a facial paralysis in a 16-year-old patient with multiple aneurysms of the petrous apex.[4] Bergés et al reported the case of a 47-year-old patient who presented a progressive peripheral facial paralysis due to a 1.3-cm aneurysm of the horizontal portion of the internal carotid artery near the geniculate ganglion.[1] These are the only two cases of peripheral facial paralysis induced by an aneurysm in the intrapetrous portion of the internal carotid artery reported in the literature.

Sherman and Thompson described one case of isolated facial paralysis due to a pontine hemorrhage in a 37-year-old patient, caused by a cavernous angioma.[5]

Of all cranial nerve paralysis 3 to 12% are related to dissection of the internal carotid artery and 0.5% are isolated. This can be due to the nerve compression or to the stretching by the aneurysmal artery, but also to the disturbances undergone by the parent artery of the nerve. The latter can be caused by distal embolizations, pressure changes in collateral support, or the abnormal origin of these feeder vessels.[6,7]

Isolated facial paralysis can also be induced by vascular tumors of the facial nerve, as described by Ray with a venous angioma in the geniculate ganglion. The cavernous hemangiomas of the seventh cranial pair have also been described and constitute the most frequent vascular lesion of cranial nerves.[8]

Progressive peripheral facial paralysis can also be caused by the rapid enlargement of a cerebellopontine arteriovenous malformation combined with an internal carotid artery aneurysm on the level of the meatal loop of the anteroinferior cerebral artery.[9]

In addition, the case of a 10-month-old child with peripheral facial paralysis due to a congenital aneurysm of the middle cerebral artery was described, although the incidence of intracranial aneurysms in childhood is exceptional.[10]

In our case, the progressive aggravation of the isolated peripheral facial paralysis justified a tomodensitometry of the petrous bones. Intrapetrous aneurysms were thus a fortuitous discovery on CT scan. Indeed, in the case of Bergés et al, the patient also presented intermittent dysgueusia associated with headaches, and the aneurysm diagnosis was also confirmed with tomodensitometry.[1]

Isolated palsies of the sixth cranial pair are related to aneurysms of the internal carotid artery in 2 to 4% of the cases, whereas they are of unspecific origin in 26 to 30% of the cases.[11]

In our clinical case, the principal differential diagnosis in front of this mass of the petrous apex was a cholesterol granuloma. Indeed, this aspecific granuloma reaction due to cholesterol crystals is generally hyperintense on T1 and T2 sequences on MRI. The differential diagnoses of the petrous apex pathologies include cholestatomas (there is a hypersignal on diffusion weighted sequences), histiocytosis (on MRI, there is a strong enhancement after gadolinium injection), mucoceles, tumoral pathologies (mesenchymatous tumor metastases, neurinomas), infectious diseases (malignant otitis, apicite), and vascular pathologies such as hemangiomas or aneurysms.[12,13]

These aneurysms may enlarge, leading to thromboembolic complications or rupture.[2]

The treatment of these intrapetrous aneurysms is either to ligate the carotid artery with potential tragic cerebral side-effects (transitory ischemic accidents, cerebral vascular accident, subarachnoid hemorrhage, and a death rate up to 15%),[14] or to exclude the aneurysm with endovascular techniques. Unruptured aneurysms are generally treated electively: observation is an option for very small aneurysms but is not recommended, craniotomy and clipping to the neck of the aneurysm permanently prevents blood flow into the aneurysm. Proximal ligation has also been used with success for giant aneurysms, particularly of the vertebrobasilar circulation. Then, treatment may incorporate high flow extracranial-intracranial bypass to maintain flow distal to the trapped segment.

Thus, the treatment with coils is indicated for the management of small aneurysms. Stents with self-expanding balloon are stiffer and imply more difficult flexibility.[2] They also have the disadvantage of the persistence of the mass effect of the thrombosed aneurysm.[1] In the case of Berges et al, the aneurysm occlusion was performed with the assistance of an endoluminal

balloon but a surgical resection of the aneurysm followed by nervous repair surgery was necessary to remove the nervous compression.[1]

The recent use of covered stents proves to be an effective treatment to occlude aneurysms, pseudoaneurysms, or fistulas, while preserving the parent artery. The principal limitation of these stents rests in their limited longitudinal flexibility, which however, could be improved by the recent use of self-expanding stents.[2] As a consequence, navigation through winding vessels is easier, making these stents the ideal device for the treatment of intrapetrous aneurysms.

Pipeline-type self-expanding stents are new endovascular microcatheter devices designed to treat wide-necked and fusiform intracranial aneurysms. They are a secure, durable, and curative technique for giant and large cerebral aneurysms as the rate of complete occlusion at 12 months of follow-up attains 95%, without major complication or angiographic recurrence.[15] This treatment was finally chosen for our patient.

In the case of fibromuscular dysplasia, the patient was successfully treated with a Symbiot-type self-expanding stent, without any significant postinterventionnal neurological event.[2] As a consequence, endovascular treatment is effective and less invasive,[3] although the complication rate of these self-expanding stents can reach up to 10% and the bleeding rate 29%.[16]

Our patient's paralysis improvement was probably due to the progressive thrombosis of the aneurysm. In addition, the left peripheral facial paralysis could be explained by the pseudoaneurysm of the left intrapetrous carotid artery being close to the left geniculate ganglion fossa.

These aneurysm exclusions should be followed with arteriography 6 months later, and then a year later, as was the case for our patient. Indeed, the complete efficiency of the stent is better after 6 months.[16] In addition, antiplatelet medication as prescribed for our patient is essential to the long-term treatment.[16]

CONCLUSION

Isolated peripheral facial paralysis may be the first sign of various vascular malformations on the different segments of the internal carotid artery, although it is a rare symptom. The paralysis is in fact due to the compression exerted by the aneurysm. Our case is the first in the literature to present a bilateral pseudoaneurysm of the internal carotid artery in its intrapetrous portion, discovered at the time of an isolated peripheral facial paralysis, independant from fibromuscular dysplasia. The endovascular treatment is the required treatment, particularly with the recent use of self-expanding stents which provide better manageability and satisfactory results with lower morbidity. The progressive improvement of this palsy is first related to the aneurysm thrombosis, then to the secondary fibrosis. A congenital origin is likely, but future research will be necessary to better understand the physiopathology of these vascular malformations.

NOTE

Presented as a poster presentation at the French Society of Otolaryngology, Head and Neck Surgery Meeting, Palais des Congrés, Paris, France, October 16–18, 2010.

REFERENCES

1. Bergés C, Pollak A, Valavanis A, Fisch U. Gradual facial palsy and intrapetrous internal carotid aneurysm: a case report. Skull Base Surg 1993;3(3):164–169
2. Cohen JE, Grigoriadis S, Gomori JM. Petrous carotid artery pseudoaneurysm in bilateral carotid fibromuscular dysplasia: treatment by means of self-expanding covered stent. Surg Neurol 2007;68(2):216–220; discussion 220
3. Mori R, Murayama Y, Irie K, et al. Endovascular treatment of large petrous internal carotid artery aneurysm associated with chronic otitis media: case report. No Shinkei Geka 2006;34(4):415–419
4. Brandt TW, Jenkins HA, Coker NJ. Facial paralysis as the initial presentation of an internal carotid artery aneurysm. Arch Otolaryngol Head Neck Surg 1986;112(2):198–202
5. Sherman SC, Thompson T. Pontine hemorrhage presenting as an isolated facial nerve palsy. Ann Emerg Med 2005;46 (6):555
6. Guidetti D, Pisanello A, Giovanardi F, Morandi C, Zuccoli G, Troiso A. Spontaneous carotid dissection presenting lower cranial nerve palsies. J Neurol Sci 2001;184(2):203–207
7. McCarron MO, Metcalfe RA, Muir KW. Facial nerve palsy secondary to internal carotid artery dissection. Eur J Neurol 2000;7(6):723–725
8. Ray A, Lammie GA, Shone G, Hourihan MD, Vafidis J. Venous angioma of the facial nerve: a case report. Surg Neurol 2004;61(2):198–200
9. Neimat JS, Hoh BL, McKenna MJ, Rabinov JD, Ogilvy CS. Aneurysmal expansion presenting as facial weakness: case report and review of the literature. Neurosurgery 2005; 56(1):190
10. Iglesias S, Hinojosa J, Esparza J, Muñoz A, Otero A. Congenital aneurysm presenting as peripheral facial paresis. Pediatr Neurosurg 2007;43(6):504–506
11. Goodwin D. Differential diagnosis and management of acquired sixth cranial nerve palsy. Optometry 2006;77(11): 534–539
12. Griffith AJ, Terrell JE. Transsphenoid endoscopic management of petrous apex cholesterol granuloma. Otolaryngol Head Neck Surg 1996;114(1):91–94
13. Veillona F, Ramos-Taboada L, Abu-Eid M, Charpiot A, Riehm S. Imaging of the facial nerve. Eur J Radiol 2010; 74(2):341–348
14. Roski RA, Spetzler RF, Nulsen FE. Late complications of carotid ligation in the treatment of intracranial aneurysms. J Neurosurg 1981;54(5):583–587

15. Lylyk P, Miranda C, Ceratto R, et al. Curative endovascular reconstruction of cerebral aneurysms with the pipeline embolization device: the Buenos Aires experience. Neurosurgery 2009;64(4):632–642; discussion 642–643; quiz N6

16. Pavlisa G, Ozretic D, Murselovic T, Pavlisa G, Rados M. Sole stenting of large and giant intracranial aneurysms with self-expanding intracranial stents-limits and complications. Acta Neurochir (Wien) 2010;152(5):763–769