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Delayed Ischemic Optic Neuropathy After Surgery on Skull Base Meningiomas Successfully Treated with Nimodipine and Rheological Therapy: Report of Two Cases

ABSTRACT—Two patients operated on because of skull base meningiomas experienced delayed ischemic optic neuropathy with loss of vision in one eye on days 6 and 12 after surgery. Treatment with nimodipine and rheological therapy was effective in restoring visual acuity. Possible pathophysiological mechanisms and treatment options are discussed.

Surgery on skull base pathology that involves the optic pathways may be complicated by loss of vision or visual field deficits. Especially in skull base tumor surgery, direct manipulation of the optic nerves or optic chiasm, for example, through retraction, electrocoagulation, or interruption of the vascularization, of these delicate structures may cause optic neuropathy. This adverse effect of surgery will be noticed directly after surgery by the patient.

We report here two cases in which a delayed ischemic optic neuropathy (ION) with diminution or loss of vision occurred after surgery for skull base meningiomas. The neuropathy was successfully treated as part

of the treatment of posthemorrhagic vasospasm with intravenous nimodipine and rheological therapy. We will discuss the possible pathophysiological mechanism and treatment options of this ION.

CASE 1

This 56-year-old-man suffered with headaches for several years until a magnetic resonance (MR) image revealed a meningioma of the tuberculum sellae. Neurological examination revealed no deficits. The patient had received panretinal laser therapy on both eyes

3 years before because of diabetic retinopathy. Before surgery on the meningioma, the visual acuity was 0.8 on both eyes, and the visual fields were narrowed concentrically. The meningioma was radically extirpated through a right pterional craniotomy. The tumor was in part located medial to and beneath the right optic nerve. The right optic canal had to be opened with a high-speed drill under constant fluid irrigation and the dura mater around the optic nerve had to be extirpated. The ophthalmic artery had to be dissected for tumor removal. Bipolar electrocoagulation and the use of fibrin glue may have compromised the vascularization of the right optic nerve.

Postoperatively, the visual acuity was unchanged, but a right temporal hemianopia was found on perimetric examination. A prophylactic therapy with intravenous nimodipine 1 mg/h to prevent eventual vasospastic reactions due to coagulation necrosis or fibrin glue was started immediately after surgery and continued for 5 days. Postoperative computer-assisted tomography (CT) revealed no blood in the operative field. On the 12th day after surgery the patient suddenly reported visual blurring in the right eye. Visual acuity proved to be 0.2. Arterial hypotension had not occurred. Rheological therapy with hydroxy ethyl starch (HES) 6% was started; the patient reported normalization of visual acuity the next day, but rheological therapy was continued. Two days later the patient suddenly became blind in the right eye; nimodipine 2 mg/h was therefore added to the therapy. Within 2 days the patient regained light perception and in 10 more days visual acuity improved to 0.6, but acuity did not return to the preoperative status.

CASE 2

This 48-year-old woman experienced a progressive loss of vision in the right eye over a period of 6 months. Ophthalmologic examination revealed a visual acuity of the right eye of 0.1 and an incomplete right temporal hemianopia. MR imaging showed a meningioma en plaque of the right cavernous sinus, tentorium cerebelli, right anterior clinoid process, and optic canal as well as the temperopolar dura mater. In addition, a nonsymptomatic left temperopolar arachnoid cyst was found (Fig. 1A-C). Tumor removal through a right pterional craniotomy to decompress the right optic nerve was attempted. The right optic canal was opened and parts of pathological dura mater were resected. Surgery was uncomplicated. Postoperatively, prophylactic nimodipine was given 1 to 2 mg/h intravenously. No blood was seen on a postoperative CT scan (Fig. 1D). Visual acuity and visual fields were unchanged after surgery. Sudden blindness of the right eye occurred within 24 hours after discontinuation of nimodipine on the 5th day. Nimodipine was immediately reinstituted, and rheological

therapy with HES 6% was also performed. One day later, visual acuity was 0.1 again. On dismissal from the hospital, the visual acuity was unchanged from preoperative status, and the visual field of the right eye had improved in the upper quadrant. At 1 year follow-up the visual acuity of the right eye had improved to 0.8.

DISCUSSION

Partial or complete loss of vision or visual field deficits may be a complication of skull base surgery, especially when the pathological process involves the optic pathways. The surgeon usually knows what has caused the deficits: direct manipulation, for example; retraction; blunt or sharp traumatization; coagulation of the optic pathways; or interruption of the vascularization toward the optic nerve and chiasm. These deficits can be noticed directly after surgery by the patient and during the ophthalmologic examination.

We have seen two cases in which delayed deficits of the optic pathway of one eye occurred, one after 6 and the other after 12 days. Both patients were operated on because of meningiomas that involved the right optic nerve and optic canal.

In cases with meningiomas in and around the optic canal and optic pathways, we routinely used nimodipine intravenously after surgery, because surgical manipulation of the small feeding arteries of the optic nerves and chiasm, the release of possible vasoconstrictive factors because of extensive coagulation of dura mater with induced necrosis, or the use of fibrin glue, or a combination of these, might cause vasospasm of the arteries. We did not, however, have any proof of our theory.

In both cases discontinuation of nimodipine led in a relatively short time to loss of vision on the side of surgical manipulations. CT scans revealed no blood in the operative field in either case. Resumption of intravenous nimodipine together with rheological therapy restored vision successfully, although incompletely in one case, in a period of several days.

Cerebral vasospasm is most frequently associated with subarachnoid hemorrhage (SAH) after rupture of an intracranial aneurysm but has also been reported after head injuries and head and neck surgery.¹ Although the exact pathophysiological mechanism of vasospasm is not known, breakdown products of blood and other substances have been connected with the occurrence of vasospasm after SAH.² Direct manipulation of arteries may also lead to a vasospastic reaction of vessels, which may be observed by the surgeon during operation.^{3,4} Nimodipine, a calcium antagonist, is widely accepted as an effective prophylactic drug; nimodipine together with hypervolemic or hypertensive therapy is the standard for delayed ischemic deficits due to cerebral vasospasm.⁵⁻⁸

Symptomatic vasospasm after brain tumor resection is rare but has been reported on occasion.⁹⁻²⁴ In

Figure 1. Case 2. (A) T1 axial MR image showing a left temporopolar arachnoid cyst (large arrow) and an enlargement of the right anterior clinoid process indicative of tumor. (B) T1 contrast-enhanced coronal MR image showing a tumor en plaque of the right lateral cavernous sinus wall. (C) The T1 contrast-enhanced sagittal MR image more clearly delineates the meningioma en plaque around the anterior clinoid process, growing into the optic canal and reaching the internal carotid artery. (D) Postoperative CT scan revealing the bony defect of the roof of the optic canal obliterated with fat, without evidence of the presence of cisternal blood.

many of these cases, the vasospasm was associated with significant intraoperative bleeding or skull base tumors, or both, and postoperative CT scans revealed blood in the basal cisterns. The neurological deficits were often transient, although of the four cases that were reported by Aoki et al,¹⁰ two died due to vasospasm and of the five cases reported by Cervoni et al,¹¹ three died.

In a case of pediatric primitive neuroectodermal tumor, an autoimmune or paraneoplastic response was suggested to be the etiology of a severe, delayed, generalized vasospasm that eventually led to the death of the patient because there was no substantial intraoperative hemorrhage.⁹ There have been several earlier reports of vasospasm after surgery of pituitary tumors in which a possible relationship with postoperative hypothalamic dysfunction has been discussed.^{17,22} In the end, we can only speculate about the exact etiology of this kind of postoperative vasospasm, but it seems to be multifactorial.²⁴

Our patients revealed no other delayed neurological deficits so we did not consider vasospasm of major cerebral arteries. Therefore, no cerebral angiography was performed and vasospasm could not be proven or ruled out. Other possible explanations for delayed but reversible visual loss were taken into consideration but seemed unlikely. Segmental demyelination of the optic nerve due to direct mechanical force on the nerve as well as an optic nerve deprived of blood supply would have resulted in direct postoperative loss of vision.²⁵ Postoperative edema that develops gradually could explain a delayed deficit.²⁵ However, we did not perform any antiedematous therapy. On the contrary, our successful therapeutic regimen consisted in part of hypervolemic therapy and improvement came rather early, so that edema was an unlikely explanation. Arterial hypotension, an occasional cause of ION, could be ruled out in our two patients.^{26,27}

A possible explanation might come from the field of ophthalmology. It was found that retinal and optic nerve vessels bear endothelin receptors.²⁸ Endothelin is a potent, long-acting, vasoconstricting peptide that is synthesized by vascular endothelial cells to regulate vascular tone and local blood circulation.²⁹ Its vasoconstrictive properties are mediated by an influx into the intracellular space from extracellular Ca^{2+} . Endothelin-induced vasospasm impairs retinal and optic nerve function but can be antagonized by calcium channel blockers, as has been proven for nicardipine³⁰ and nifedipine.³¹ Endothelin levels are elevated in diabetes mellitus and might be involved in the pathogenesis of diabetic retinopathy. Endothelin, however, is also elevated after SAH and trauma and therefore can also be elevated after skull base surgery. Intraoperative rinsing may have cleared vasoactive substances out of the subarachnoid space, but the use of fibrin glue around the optic nerve may have increased local concentration of vasoactive substances directly on the optic nerve. This might explain why only ocular symptoms occurred, and no other signs of cerebral vasospasm were noticed.

Our clinical observations do not render definite scientific proof of arterial vasospasm as the cause of loss of vision in our cases. Our successful treatment regimen with nimodipine intravenously and rheological or hypervolemic therapy, however, adds to the probability of such a mechanism. Neurosurgeons should be aware of the possibility of delayed ischemic optic neuropathy after skull base surgery around the optic nerves and of how to treat this condition successfully.

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