Case Report: A Troublesome Ophthalmic Artery Aneurysm

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Abstract

Objective and Importance When treating large unruptured ophthalmic artery (OA) aneurysms causing progressive blindness, surgical clipping is still the preferred method because aneurysm sac decompression may relieve optic nerve compression. However, endovascular treatment of OA aneurysms has made important progress with the introduction of stents. Although this development is welcomed, it also makes the choice of treatment strategy less straightforward than in the past, with the potential of missteps.

Clinical Presentation A 56-year-old woman presented with a long history of progressive unilateral visual loss and magnetic resonance imaging showing a 20-mm left-sided OA aneurysm.

Intervention Because of her long history of very poor visual acuity, we considered her left eye to be irredeemable and opted for endovascular therapy. The OA aneurysms was treated with stent and coils but continued to grow, threatening the contralateral eye. Because she failed internal carotid artery (ICA) balloon test occlusion, we performed a high-flow extracranial-intracranial bypass with proximal ICA occlusion in the neck. However, aneurysm growth continued due to persistent circulation through reversed blood flow in distal ICA down to the OA and the cavernous portion of the ICA. Due to progressive loss of her right eye vision, we surgically occluded the ICA proximal to the posterior communicating artery and excised the coiled, now giant, OA aneurysm. This improved her right eye vision, but her left eye was permanently blind.

Conclusion This case report illustrates complications of the endovascular and surgical treatment of a large unruptured OA aneurysm.

Keywords
- ophthalmic artery aneurysm
- endovascular treatment
- neurovascular surgery
- complications

Background

The ophthalmic segment of the internal carotid artery (ICA), running from the distal dural ring to the posterior communicating artery (PCOM), can give rise to aneurysms.[1] The three subtypes are the superiorly pointing aneurysms originating at the ophthalmic artery (i.e., ophthalmic artery [OA] aneurysms), the medially pointing aneurysms originating from the

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superior hypophyseal trunk, and aneurysms originating from a perforator-free part of the ICA, the so-called atypical or blister ICA aneurysms.[2]

The incidence of OA aneurysms is somewhere between 0.5% and 11% of all intracranial aneurysms.[3,4] Unruptured large OA aneurysms frequently present with gradual loss of vision. The goal of their treatment is then twofold: to exclude the aneurysm from the circulation and to preserve or restore visual function. Treatment of OA aneurysms can be surgical or endovascular, depending on the mode of presentation, aneurysm geometrics and size, as well as the patienťs medical condition.[5] In the past, intracranial aneurysm repair was exclusively surgical,[4] and in large unruptured OA aneurysms, clipping is still often the preferred method because surgical decompression of the aneurysm sac may lend the optic nerve a better prognosis.[6,7] However, surgery of OA aneurysms carry inherent risks such as stroke,[8] hemorrhage,[9] or visual loss.[10–12]

Since its introduction in 1991,[13] endovascular aneurysm repair has grown in popularity and versatility.[8,14,15] With the introduction of stents,[16,17] endovascular treatment of OA aneurysms has made important progress. Although this development is welcomed, it also makes the choice of treatment strategy less straightforward than in the past.

In this report, we present a patient with a troublesome unruptured OA aneurysm and some lessons learned.

Case Report

Clinical Presentation

A 56-year-old right-handed woman presented with a 4-month history of retro-orbital headache and progressive decline in visual acuity oculus sinister (OS). Her ophthalmologist (P.L.) found only perception of light OS and normal visual acuity oculus dexter (OD). Magnetic resonance imaging (MRI) detected a left-sided OA aneurysm 20 × 12 × 13 mm in diameter (►**Fig. 1**).

Because of her long history of very poor visual acuity, we considered her left eye to be irredeemable. Because the aneurysm had a wide neck and was of moderate size, endovascular treatment was chosen. The treatment plan was first to place a regular stent within the ICA, running from proximal to the OA to distal to the PCOM artery, with a second semipermeable stent inside the first stent in the aneurysm orifice area to enhance aneurysm thrombosis. Following 1 week of treatment with Plavix and aspirin preceding the endovascular procedure, a LEO (Balt Extrusion, Montmorency, France) 4.5 mm × 40 mm

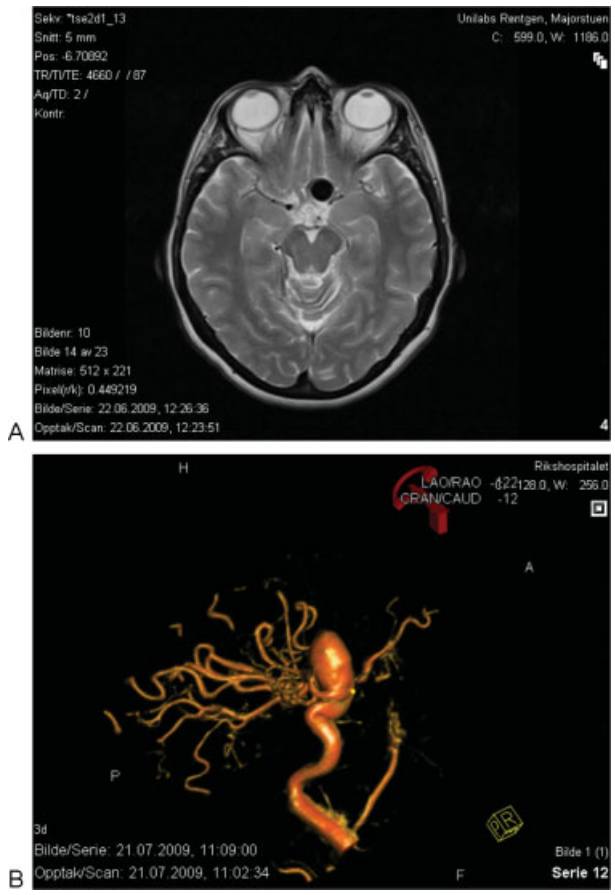

Fig. 1 Magnetic resonance imaging (MRI) of a 56-year-old right-handed woman who presented with a 4-month history of retro-orbital headache and progressive visual loss demonstrating a left-sided ophthalmic artery aneurysm 20 × 12 × 13 mm in diameter. (A) Axial T2-weighted MRI demonstrating the relationship to the left optic nerve. (B) Three-dimensional digital subtraction angiography demonstrating the aneurysm sac.

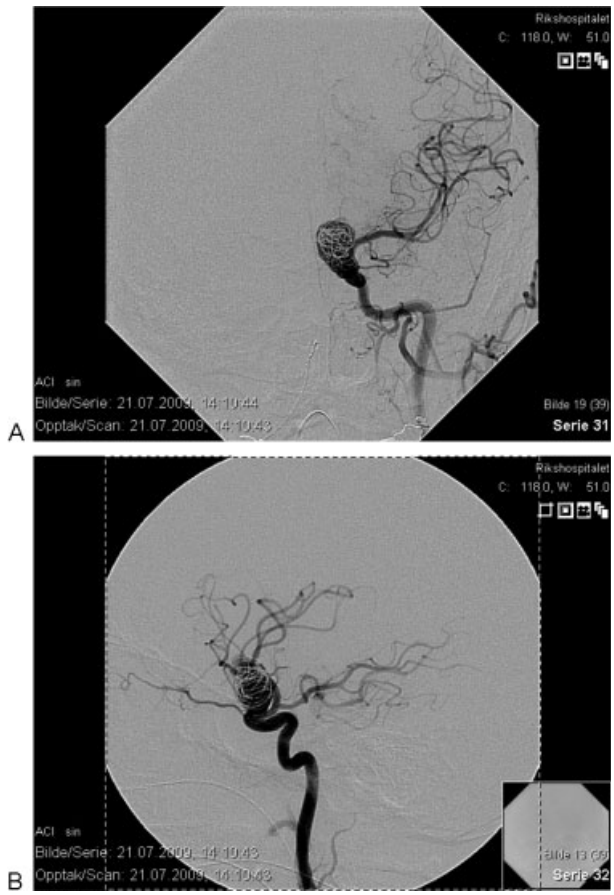

Fig. 2 Digital subtraction angiography after a stent was placed in the interior carotid artery and the aneurysm sac coiled using three matrix coils. (A) Anteroposterior view. (B) Lateral view.

stent was placed in the ICA (S. J. B. and E. A. J.). Attempts to place additional stents, however, were unsuccessful. To promote aneurysm thrombosis, three Matrix coils (Boston Scientific, Fremont, California, United States), of 24 mm × 40 cm, 30 mm × 16 cm, and 14 mm × 30 cm, respectively, were put into the aneurysm sac (►**Fig. 2**). This led to stagnation of the contrast media in the aneurysm sac, but because of the optic nerve compression, the aneurysm was filled loosely so as not to cause further optic chiasm compression.

After the procedure, the patient woke up with an expressive dysphasia and a right-sided hemiparesis. MRI demonstrated several punctate lesions in the left parieto-occipital region secondary to emboli and perfusion MRI showed a small hypoperfusion in the left lentiform nucleus region. She subsequently recovered completely from her neurologic deficits. Her visual acuity improved to perception of hand movement ad ocolum OS and acuity OD remained at 1.0.

Over the following months, the aneurysm continued to grow, measuring 22 × 13 × 14 mm (►**Fig. 3**). It now caused significant compression of both optic nerves, and her ophthalmologist recorded a reduction of visual acuity to 0.8 OD and an upper quadrant anopia. A course of prednisolone 20 mg twice daily was tried for 5 days without effect.

Six months after her presentation, her visual acuity remained at light perception OS and 0.8 OD with an upper quadrant anopia. A digital subtraction angiography (DSA) demonstrated coil compaction and further aneurysm growth. The patient failed an ICA balloon test occlusion (W.S.O.) and was therefore referred to high-flow extracranial-intracranial (EC-IC) artery bypass grafting in Berlin, Germany. Preoperatively, her visual acuity OD was down to 0.7 with an upper quadrant anopia, whereas OS remained at light perception. She underwent a high-flow venous graft bypass between the left external carotid artery and the left middle cerebral artery (P.V. and T.R.M.) (►**Fig. 4**). Because of the combined effects of stent within the ICA and the large coil mass in the aneurysm, it was difficult to put an occluding clip on the ICA just distal to the OA take-off, and the ICA was therefore occluded proximally in the neck.

Postoperatively, she had expressive dysphasia, right-sided hemiparesis, and short-term memory loss. She was also completely blind in the left eye. Postoperative T1-weighted MRI demonstrated some encephalomalacia with peri-Sylvian cortical laminar necrosis in the temporal and frontal lobes (►**Fig. 5**). Over the next months she recovered well from these neurologic deficits, with the exception of blindness OS.

Contrary to what we anticipated, the aneurysm continued to expand (27 × 19 × 20 mm); her right eye visual acuity

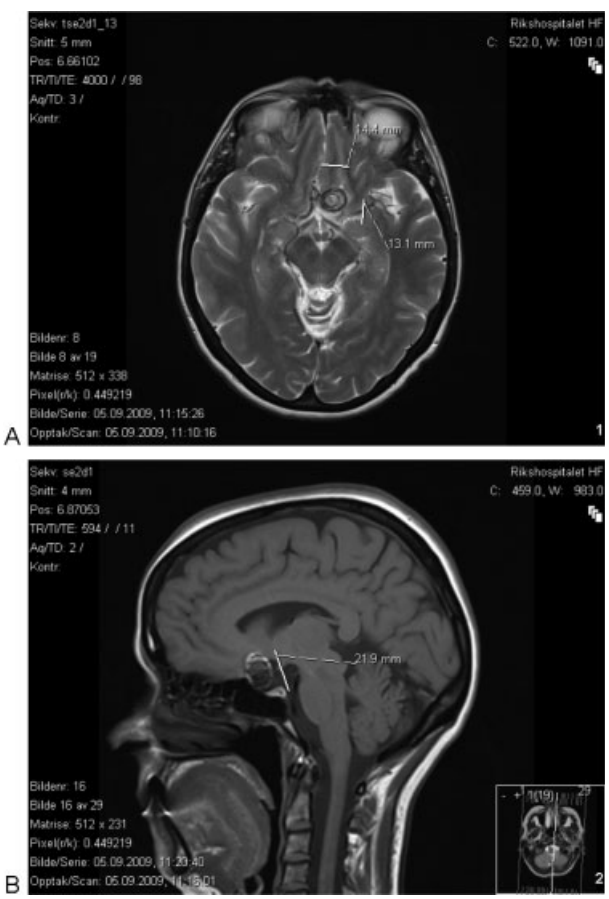

Fig. 3 The aneurysm continued to grow, measuring 22 × 13 × 14 mm 2 months post-coiling. (A) Axial T2-weighted magnetic resonance imaging (MRI). (B) Sagittal T1-weighted MRI.

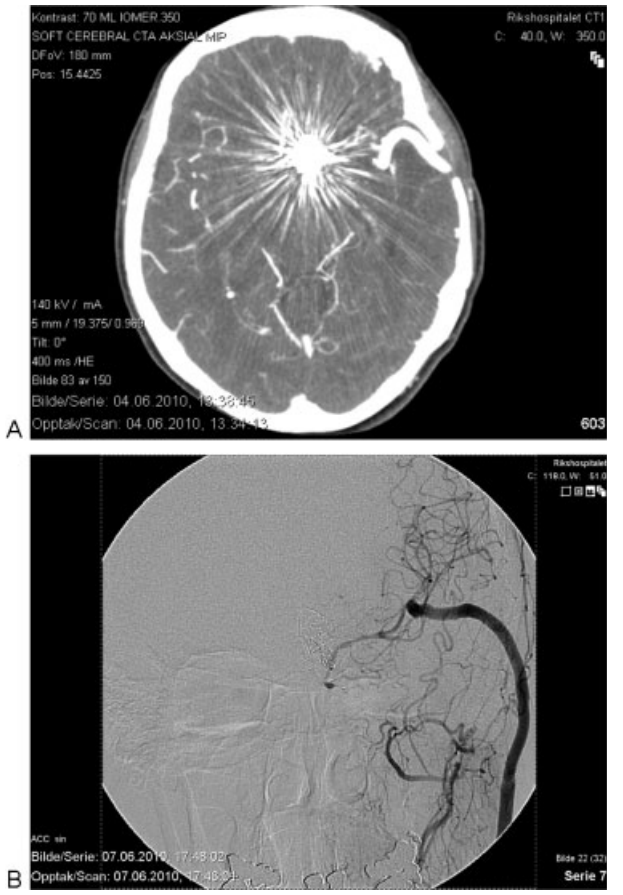

Fig. 4 The patient underwent a high-flow venous graft bypass between the left external carotid artery and the middle cerebral artery (MCA). The internal carotid artery was occluded proximally in the neck. (A) Axial computed tomography angiography demonstrating the patent graft to MCA and coil artifacts. (B) Digital subtraction angiography anteroposterior view demonstrating filling of the extracranial-intracranial bypass.

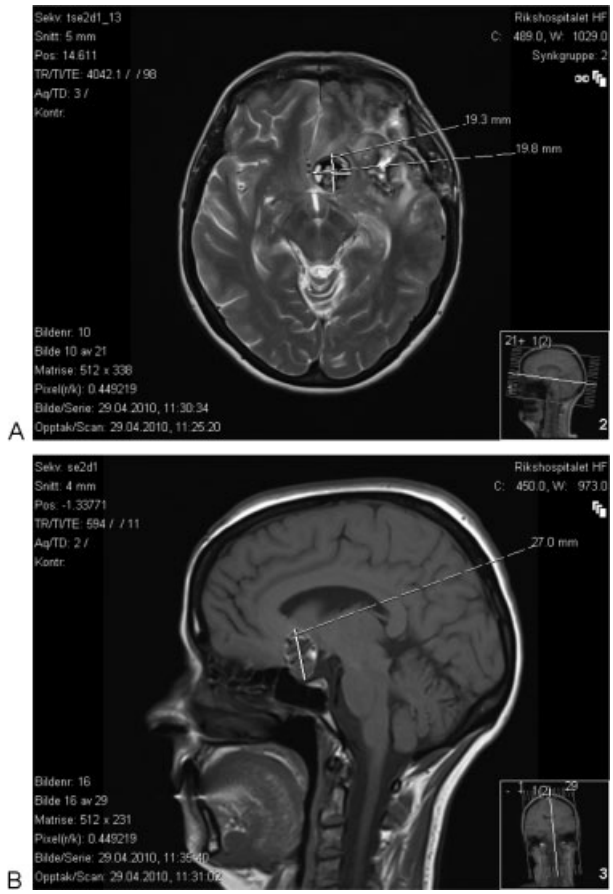

Fig. 5 Postoperative fluid attenuated inversion recovery magnetic resonance imaging demonstrates some encephalomalacia with peri-Sylvian cortical laminar necrosis. She became completely blind in her left eye.

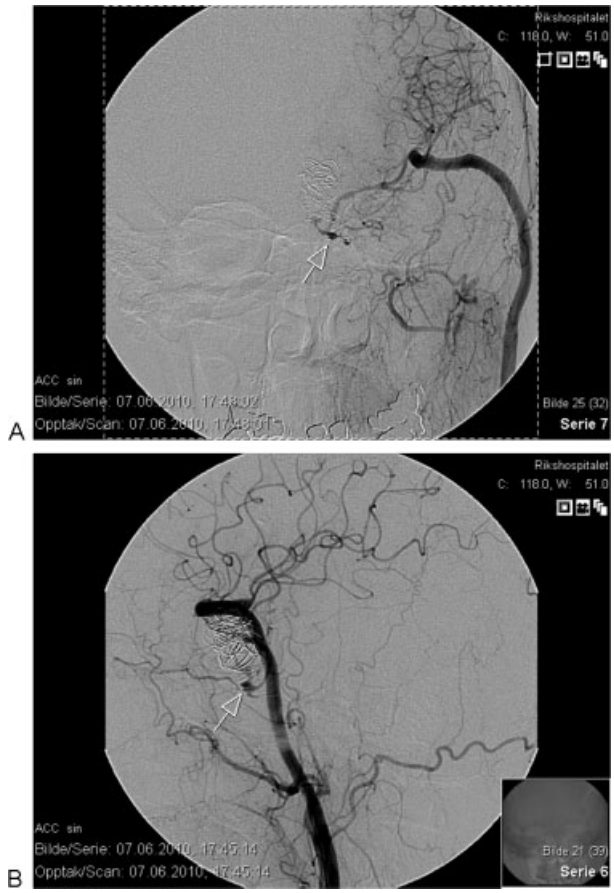

Fig. 6 The aneurysm continued to expand, severely compromising vision of her right eye. Digital subtraction angiography demonstrates good bypass flow, with retrograde filling of the distal internal carotid artery (ICA), antegrade filling of the ophthalmic artery, and some filling in the cavernous portion of the ICA. There is also some filling of the caudal part of the aneurysm (arrow), the latter finding explaining the continued aneurysm growth. (A) Anteroposterior view. (B) Lateral view.

decreased dramatically to 0.3 a month after the surgery. A DSA demonstrated good flow through the bypass, retrograde filling of the distal ICA, antegrade filling of the OA, some filling in the cavernous portion of the ICA, and some filling in the bottom of the aneurysm (►**Fig. 6**), the latter finding explaining the continued aneurysm growth.

Because of progressive visual field defect and acuity loss OD, the patient was operated again a year after her initial presentation via a 30×25 mm supraorbital craniotomy (T.R.M.). The stented ICA was "crushed" using a DeBakey forceps to fit a clip across the ICA proximal to the PCOM. The previously coiled giant aneurysm was thereafter excised (►**Fig. 7**), and the optic chiasm and both optic nerves were decompressed. The patient made an uneventful recovery, and the surgery led to a significant improvement in visual acuity OD to 0.7. Her left eye remained amaurotic.

Discussion

Major neurologic deficits after treatment of giant intracranial aneurysms occur in $\sim$ 10% of patients.[18–22] With paraclinoid giant aneurysms, visual deterioration after treatment is observed in up to 17% of patients.[19,20] Although surgical clipping is often the preferred treatment for aneurysms causing cranial nerve deficits,[6,7] in cases of giant paraclinoid ICA aneurysms, it may not be feasible in $>$ 60% of cases.[18,20–22] Surgery on OA aneurysms is technically demanding because these aneurysms are often large and may extend into the cavernous sinus.[21–26] Thus they are challenging even for the most experienced of neurosurgeons. Over the years, these aneurysms have therefore been treated by common carotid artery ligation,[20–22,27,28] ICA occlusion,[18–20,22,29,30] proximal vessel ligation with EC-IC bypass,[18–20,22] trapping,[18,19,31] intra-aneurysmal balloon occlusion,[32,33] coiling,[34–36] or by stenting.[37] However, because visual deterioration after coil embolization of OA aneurysms has been reported in as many as a third of the patients,[14] the decision whether to clip or coil is often difficult.

In the present case, the endovascular treatment plan was to first place a regular stent in the ICA from the OA to the PCOM, with a second semipermeable stent inside the first stent in the aneurysm orifice area. Retrospectively, upon failing to place the semipermeable stent, we should have discontinued the endovascular procedure because the

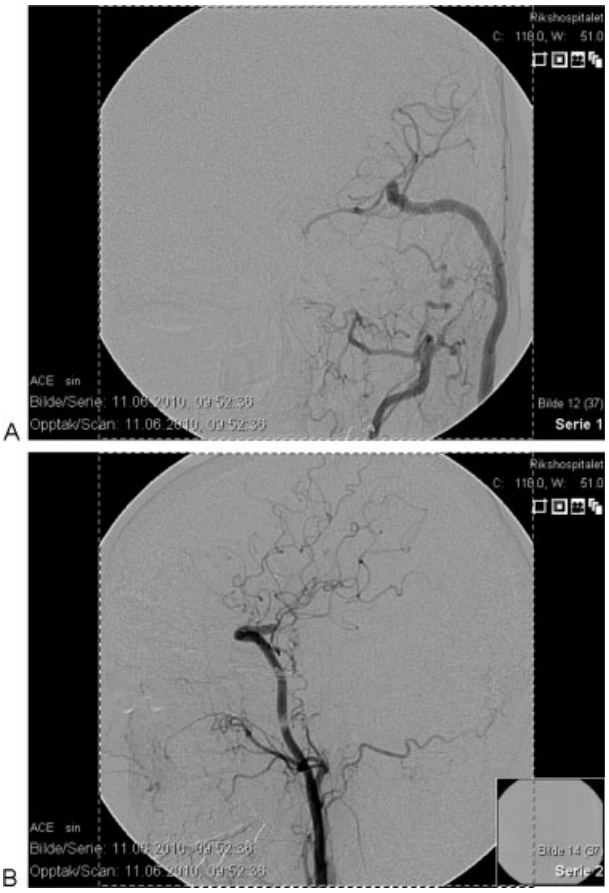

Fig. 7 The patient was reoperated via a 30 × 25 mm supraorbital craniotomy, so as not to compromise the bypass. The stented internal carotid artery (ICA) was "crushed" using a DeBakey forceps to fit a clip across the ICA proximal to the posterior communicating artery, and the coiled giant aneurysm was excised, thereby decompressing the optic chiasm and optic nerves. Surgery led to a significant improvement in visual acuity on the right side; the left remained amaurotic. (A) Anteroposterior view. (B) Lateral view.

subsequent aneurysm coiling would not make the aneurysm shrink and would make aneurysm surgery at a later stage considerably more difficult.

Given a situation with a large and growing coiled aneurysm and with a failed balloon test occlusion (BTO), we opted for a high-flow EC-IC bypass followed by therapeutic ICA closure because ICA occlusion, with or without EC-IC bypass, is a good therapeutic option when treating giant ICA aneurysms.[29,38–44] However, it proved difficult to put an occluding clip on the ICA just distal to the OA take-off due to the combined effects of the stent within the ICA and the large coil mass within the aneurysm. Furthermore, the PCOM was also not in sight due to the previously mentioned limiting structures, but also because of the high-flow vein graft in the surgical field. Consequently, the ICA was occluded proximally in the neck. Retrospectively, after finishing the bypass and realizing that the intracranial ICA closure was impossible, we should have left the ICA open and closed this vessel off endovascularly by placing coils at the origin of the OA/aneurysm because it is known that proximal ICA occlusion does not fully prevent subsequent hemorrhage or necessarily prevent aneurysm enlargement.[10,20,21,38,41,45,46] A more distal ICA closure would have eliminated blood flow in the ICA below the PCOM and triggered thrombosis and shrinkage of the noncoiled portion of the aneurysm.

In retrospect, our patient should have been treated initially with surgical clipping. It is difficult to tidy up the mess, as it were, if you start out wrong. Given the chance again, we would have followed our usual game plan when treating patients with large unruptured OA aneurysms: If there is reduced visual acuity and no or moderate thrombus material in the aneurysm, we opt for surgical clipping via a lateral supraorbital craniotomy with extradural clinoidectomy for proximal control of the ophthalmic ICA segment,[47] aneurysm dissection, suction/decompression of the aneurysm (either direct after temporary trapping or ad modum Hunt Batjer with exposure of the ICA in the neck[48]), and aneurysm clipping. If there is a large thrombotic mass in the aneurysm, with or without reduced visual acuity, we generally manage our patients with semipermeable (flow diversion) stents or therapeutic ICA closure with or without a high-flow excimer laser-assisted nonocclusive anastomosis (ELANA) bypass for flow replacement,[43] depending on the BTO tolerance.[44]

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