

CASE REPORT

Optic pathway infarct after Onyx HD 500 aneurysm embolization: visual pathway ischemia from superior hypophyseal artery occlusion

Jeremiah Nicholas Johnson,¹ Mohamed Elhammady,² Judith Post,³ Joshua Pasol,⁴ Koji Ebersole,¹ Mohammad Ali Aziz-Sultan²

¹Department of Neurosurgery, University of Miami, Miami, Florida, USA

²Department of Neurosurgery, University of Miami Miller School of Medicine, Miami, Florida, USA

³Department of Neuroradiology, University of Miami, Miami, Florida, USA

⁴Bascom Palmer Eye Institute, University of Miami, Miami, Florida, USA

Correspondence to

Dr M Ali Aziz-Sultan, Department of Neurosurgery, University of Miami Miller School of Medicine, 1095 NW 14th Terrace (D4-6), Miami, FL 33136, USA; brainsurg@gmail.com

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ABSTRACT

We report a case of visual deterioration after Onyx HD 500 embolization of a left 7 mm superior hypophyseal artery (SHA) aneurysm. After the procedure, the patient experienced a right incongruous homonymous hemianopia, and MRI showed an infarct of the ipsilateral optic chiasm/tract but no evidence of aneurysm mass effect or embolic cortical infarcts. The optic pathway ischemia is believed to be secondary to Onyx penetration and occlusion of an SHA branch near the aneurysm neck. Caution is advised when using liquid embolic agents to treat SHA aneurysms as SHA occlusion may lead to visual deficits.

BACKGROUND

Superior hypophyseal artery (SHA) aneurysms arise from the medial wall of the internal carotid artery (ICA) just distal to the ophthalmic artery origin and comprise up to 50% of aneurysms arising from this segment.¹ Treatment of ophthalmic segment aneurysms can produce visual loss by direct mass effect on the optic pathways, retinal artery occlusion, or iatrogenic injury of the optic apparatus during surgery.^{1 2} However, we report an unusual case of acute visual deterioration after aneurysm treatment by occlusion of an SHA irrigating the optic apparatus. To our knowledge, this report is the first published case of visual loss after aneurysm embolization attributed to SHA occlusion. Of note, the patient described in this report was treated prior to the widespread availability of flow diverting stents in the USA.

CASE PRESENTATION

A 60-year-old presented with an incidentally discovered left, wide necked, 7 mm SHA aneurysm (figure 1A, B). On examination, the patient was neurologically intact, visual fields were full, and baseline corrected visual acuity was 20/20 in the right eye and 20/25 in the left.

TREATMENT

The patient had been premedicated with dual antiplatelet agents as an outpatient. Informed consent was obtained. After induction of general anesthesia, arterial access was established and systemic therapeutic heparinization was maintained throughout the procedure. An Echelon-10 microcatheter (eV3, Irvine, California, USA) was maneuvered into the aneurysm, and a 4 mm×30 mm Hyperglide

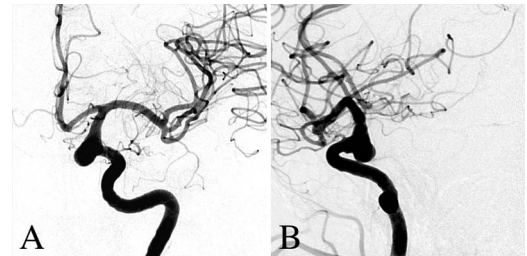


Figure 1 (A, B) Digital subtraction angiography of the left internal carotid artery, anterior and lateral projections, showing a wide necked 7 mm superior hypophyseal aneurysm.

balloon (eV3) was positioned across the aneurysm neck. The balloon was inflated and a seal was confirmed. A framing coil was delivered and then the aneurysm was filled with Onyx HD 500. When there was no aneurysm filling on post-embolization angiographic runs, the balloon and microcatheter were removed. Due to a small residual neck, an Enterprise stent (Cordis Neurovascular, Miami, Florida, USA) was deployed across the aneurysm neck. Final images showed aneurysm obliteration, a patent ophthalmic artery, and no evidence of intravascular thrombus formation or distal cerebral emboli (figure 2A, B).

OUTCOME AND FOLLOW-UP

Post-procedure, the patient had a right incongruous homonymous hemianopsia. MRI/MR angiography of the brain was unremarkable. The following day, an ophthalmological examination confirmed the visual field defect but found no retinal or structural eye damage. On day 3, follow-up MRI of the brain showed no cortical abnormalities but the diffusion

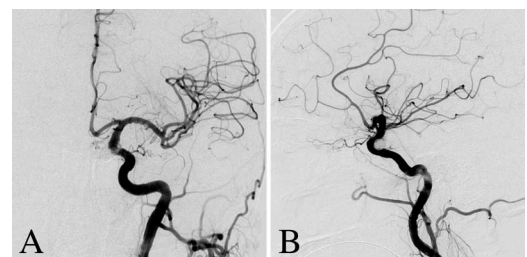


Figure 2 (A, B) Anterior and lateral subtracted angiographic images post-treatment showing an obliterated aneurysm and a patent ophthalmic artery.



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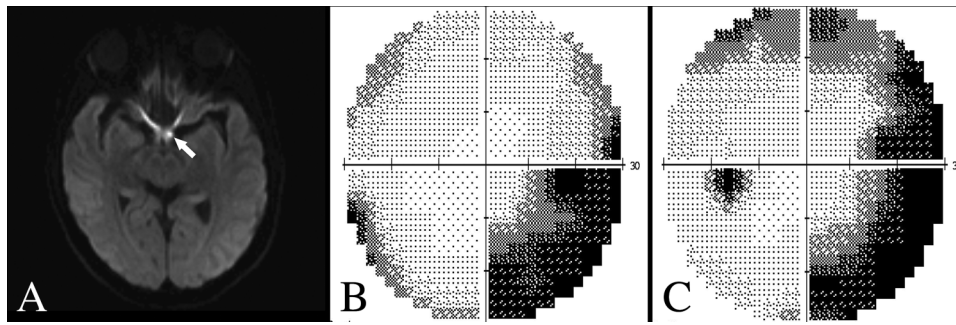


Figure 3 (A) Post-embolization diffusion weighted MRI showing an area of restricted diffusion in the left optic chiasm/tract (arrow). The high signal in the optic nerves is interpreted as a magnetic susceptibility artifact from the base of the skull bone/air interface; however, the signal more posteriorly in the chiasm/tract matched on apparent diffusion coefficient map and is felt to indicate restriction caused by ischemia. (B, C) Automated Humphrey visual field 30-2 of the left and right eyes demonstrating a right incongruous homonymous visual field deficit.

weighted sequence demonstrated a hyperintense signal with matching low signal intensity on apparent diffusion coefficient map in the left optic chiasm/tract, indicative of an acute infarct (figure 3A). The 1 month follow-up ophthalmological examination showed a persistent right incongruous homonymous hemianopsia (figure 3B, C). At 1 year, a visual field examination showed minimal improvement of the right visual field deficit.

DISCUSSION

Ophthalmic segment (C6) aneurysms arise between the distal dural ring and the origin of the posterior communicating artery. They account for 5–11% of all intracranial aneurysms.³ There are multiple classification systems but three basic subtypes of ophthalmic segment aneurysms are ophthalmic, superior hypophyseal, and anterior (dorsal) wall.¹ Due to their deep location and proximity to critical neural structures, aneurysms in this region are technically challenging to treat with open surgery and carry a significant risk of surgical morbidity.¹ Aneurysms in this region can also be challenging to treat with endovascular methods.

Recent advances in device technology such as balloon assisted embolization with Onyx HD 500 and flow diverting stents have made it possible to treat even very difficult aneurysms in this region with endovascular techniques.⁴ An advantageous property of Onyx HD 500 is its liquid nature, which allows it to conform to the shape of irregular aneurysms and exclude them from the circulation; however, this also makes the use of Onyx in aneurysms with nearby perforating arteries or critical small branches hazardous as it may lead to vessel occlusion.⁴

Superior hypophyseal aneurysms arise from the medial wall of the ICA ophthalmic segment and form adjacent to the origin of the SHAs.¹ These arteries are small direct branches from the medial ICA wall that are angiographically occult, traverse under the optic chiasm to supply the infundibulum, but also send branches to the pre-chiasmatic optic nerve, optic chiasm, and retrochiasmatic optic tract.^{5, 6} Goto *et al* demonstrated the importance of the SHA blood supply to the human visual pathway when, during an open SHA aneurysm clipping, visual evoked potentials were monitored while a prominent SHA was temporarily clipped. After 3 min of ischemia, there was a sudden drop off in visual evoked potential response in the ipsilateral eye, which returned to baseline after reperfusion.⁷

Visual deterioration from aneurysm mass effect on the optic nerve is common in patients with large ophthalmic segment aneurysms and is a feared complication of their treatment. Large endovascular coiling series of ophthalmic region

aneurysms have reported visual deterioration rates of 0–5%.^{8, 9} The etiology of visual decline after coiling these aneurysms has been attributed to cortical embolic stroke, ophthalmic artery occlusion, retinal artery embolus, and aneurysm mass effect on the visual apparatus. Our patient had visual loss in both eyes that was not consistent with unilateral retinal artery ischemia, there was no evidence of cortical ischemia, no evidence of mass effect on the visual pathways, and the progression was not consistent with a contrast reaction.¹⁰ The post-treatment MRI did not show mass effect on the ipsilateral optic nerve; however, it did show an area of ipsilateral posterior chiasm infarction in a distribution consistent with SHA occlusion (figure 3A). The region of chiasm ischemia correlated well with the patient's bilateral visual field deficit.

Ipsilateral visual deterioration was reported in three of the 47 ophthalmic region aneurysms treated with Onyx HD 500 in the CAMEO (Cerebral Aneurysm Multicenter European Onyx)

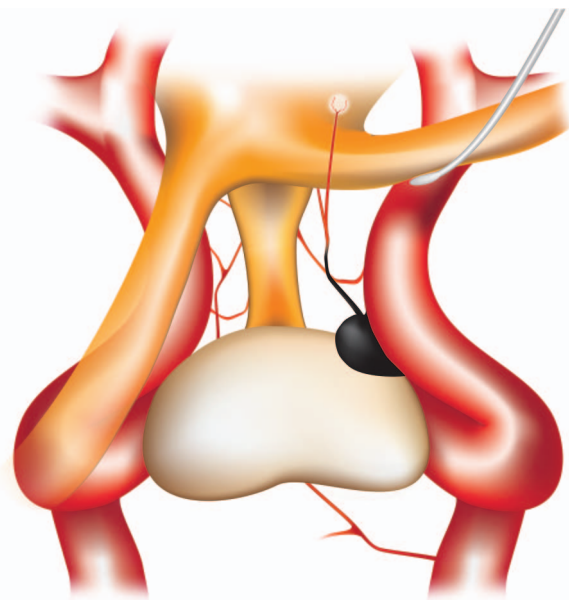


Figure 4 An illustrated anterior view of the sellar and suprasellar region showing branches of the superior hypophyseal artery (SHA) from the medial internal carotid artery traveling to the pituitary stalk, optic nerve, and optic chiasm. The left optic nerve is being pulled aside to expose the SHA vessels traveling to irrigate the hypophysis and inferior surface of the optic nerves and chiasm. The SHA aneurysm post-Onyx embolization is depicted with Onyx (black) material occluding an SHA branch causing ischemia to the optic apparatus.

trial. In two cases, there was ophthalmic artery occlusion, and in the third case, there was a transient visual disturbance that was not discussed in detail.⁴ Other large published series have reported a 0–3% incidence of visual complications after Onyx HD 500 embolization of ophthalmic region aneurysms, but none was attributed to SHA occlusion.^{11 12}

In the case reported here, the mechanism of vision loss is believed to be SHA origin occlusion by Onyx, leading to optic pathway ischemia and visual deterioration (figure 4). The treatment of the patient described in this report occurred before flow diverting stents became widely available in the USA; therefore, at the time of treatment this technology was not an option. In contrast with liquid embolic agents, however, flow diverting stents are designed to preserve branching vessels while inducing aneurysm thrombosis, thus treatment with such devices or stent assisted coiling would have likely avoided this ischemic complication.

In conclusion, Onyx embolization of SHA aneurysms or other aneurysms near small critical arteries should be avoided and alternative treatment techniques pursued.

Key messages

- ▶ Optic pathway ischemia can result from superior hypophyseal artery occlusion.
- ▶ To prevent ischemic complications, alternative embolization techniques, such as flow diverting stents or stent assisted coiling, should be considered for the treatment of superior hypophyseal aneurysms.
- ▶ The use of Onyx HD 500 should be avoided in the treatment of superior hypophyseal region aneurysms or aneurysms near other critical arterial branches.

Contributors All authors contributed to the intellectual design and preparation, and approved submission of the manuscript.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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