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Endovascular treatment for a symptomatic dissecting ophthalmic artery aneurysm occurring in the orbit: illustrative case

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BACKGROUND Peripheral ophthalmic artery aneurysms (POAAs) arising from the main trunk or branches of the ophthalmic artery (OphA) are extremely rare. However, their epidemiology and optimal management remain poorly understood. The authors report a rare case of a symptomatic POAA caused by arterial dissection that was successfully treated using endovascular therapy, leading to favorable visual recovery.

OBSERVATIONS A 77-year-old woman presented with sudden-onset visual impairment in the right eye. Ophthalmological examination revealed a defect in the right visual field. CT angiography revealed a fusiform aneurysm in the right intraorbital OphA. Digital subtraction angiography revealed a pearl and string sign, consistent with a dissecting aneurysm. A balloon test occlusion (BTO) of the OphA origin confirmed collateral circulation from the external carotid artery. Internal trapping of the OphA was performed under general anesthesia. Postoperatively, the patient's visual function gradually improved, and complete recovery was achieved within 3 months.

LESSONS Although POAAs are exceptionally rare, they may lead to significant visual dysfunction owing to optic nerve compression. When visual symptoms are present, prompt intervention may reverse the symptoms. Preoperative assessment of collateral circulation using BTO is essential for treatment planning. Internal trapping may be an effective strategy when sufficient collateral flow is confirmed.

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KEYWORDS ophthalmic artery; dissecting aneurysm; visual impairment; endovascular treatment

Peripheral ophthalmic artery aneurysms (POAAs) typically refer to aneurysms occurring at the junction of the internal carotid and ophthalmic arteries and account for approximately 5% of all intracranial aneurysms.[1] Aneurysms arising from the main trunk or branches of the ophthalmic artery (OphA) are classified as POAAs,[2] which are extremely rare and lack established epidemiological data and standardized treatment protocols. Herein, we present a case of a POAA secondary to arterial dissection that was successfully treated using endovascular intervention.

Illustrative Case

A 77-year-old woman presented to an ophthalmology clinic with sudden-onset visual loss in her right eye, without any precipitating factors. Her right eye exhibited only light perception, and perimetry revealed a visual field defect extending from the central to the superonasal region despite a normal funduscopic examination. Contrast-enhanced MRI revealed a nodular lesion in the right orbit (Fig. 1A), which was initially suspected to be an orbital tumor. She was referred to our institution approximately 4 months after symptom onset. CT angiography of the head demonstrated a 5.0-mm fusiform dilation at the orbital apex of the right OphA (Fig. 1B and C), suggestive of an aneurysm rather than a tumor. Constructive interference in steady state MRI revealed that the right optic nerve was compressed and deformed inferolaterally by the aneurysm (Fig. 2), which was consistent with the patient's visual symptoms. Digital subtraction angiography showed a pearl and string sign in the OphA on the right internal carotid artery angiogram (Fig. 1D),

ABBREVIATIONS BTO = balloon test occlusion; OphA = ophthalmic artery; POAA = peripheral ophthalmic artery aneurysm.
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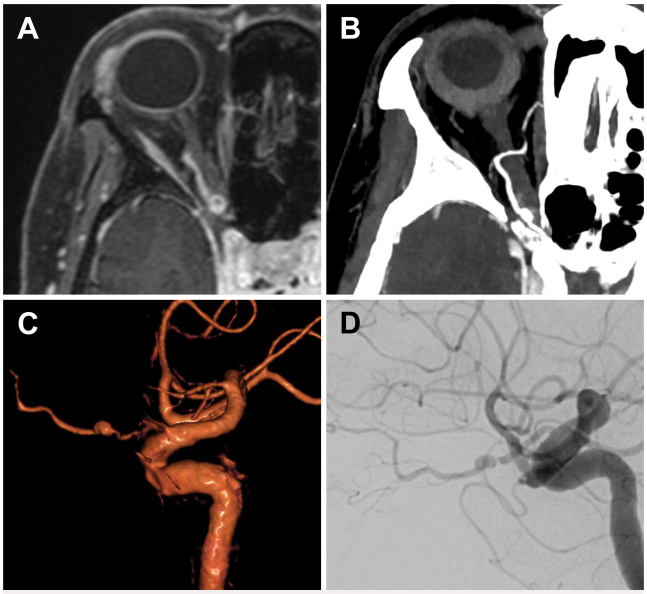

FIG. 1. A: Axial contrast-enhanced MR image showing a nodular lesion at the right orbital apex. **B:** Axial CT angiogram of the head showing fusiform dilation of the right OphA at the orbital apex. **C:** Three-dimensional reconstruction of CT angiogram of the head showing a fusiform aneurysm in the OphA. **D:** Lateral view of digital subtraction angiogram showing a pearl and string sign in the OphA.

confirming the diagnosis of a dissecting OphA aneurysm. A balloon test occlusion (BTO) at the internal carotid artery at the origin of the OphA demonstrated adequate collateral flow from the external carotid artery, with preserved retinal blush (Fig. 3A–C). No visual deterioration occurred during temporary occlusion. Based on these findings, internal trapping of the dissecting aneurysm was planned, and dual antiplatelet therapy (aspirin and clopidogrel) was initiated.

Two weeks after the initial angiography, the patient was admitted for treatment. At this time, she had developed new right-sided ptosis without ophthalmoplegia. Preoperative angiography revealed an enlarged aneurysm, likely compressing the oculomotor nerve and causing ptosis. Under general anesthesia, a microcatheter was advanced into the OphA, and coils were placed from the distal aneurysm to the origin of the OphA, achieving internal trapping (Fig. 3D and E). Postoperative angiography confirmed OphA occlusion with collateral retinal blush via external carotid circulation (Fig. 3F). Ptosis began to improve immediately after the procedure, and MRI performed the following day showed no ischemic or hemorrhagic complications. Antiplatelet therapy was discontinued on the day after the procedure. She was discharged on postoperative day 5, and by 1 month, ptosis had resolved completely. Although no early improvement in visual function was noted, gradual recovery began at 2 months, and full restoration was achieved 3 months after treatment.

Informed Consent

The necessary informed consent was obtained in this study.

Discussion

Observation

POAAs are extremely rare, with only 46 reported cases identified in our literature review.[3–43] These aneurysms were classified into four types based on their anatomical location: intracranial, intracanalicular, intraorbital, and terminal branch (Table 1).[2] Among these, 12 cases were intracranial,[3–13] 4 were intracanalicular,[14–17] 22 were intraorbital,[9,18–37] and 7 were terminal branch types.[38–43] Of the terminal branch type, 6 cases originated from the anterior ethmoidal branch[39–43] and 1 from the lacrimal branch.[38] Morphologically, saccular aneurysms accounted for the majority of cases (39 cases, 84.8%). However, in the intracanalicular type, 3 of 4 cases (75%)[14–16] were fusiform, suggesting a different pathophysiology.

The etiology of peripheral OphA aneurysms is multifactorial. Trauma-induced vascular injury resulting in a pseudoaneurysm or dissecting aneurysm,[23,32] as well as hemodynamic stress due to feeder artery involvement in coexisting arteriovenous malformations,[28] dural arteriovenous fistulas,[9,16,22,31] or other high-flow lesions, has been reported. The frequent association with multiple intracranial aneurysms suggests the presence of underlying vascular fragility within the intracranial arteries, and a genetic predisposition is also considered. However, in approximately half of the reported cases, including the present case, no definitive etiology has been identified. Recent genome-wide association studies have demonstrated that single nucleotide polymorphisms in genes encoding proteins involved in endothelial function and angiogenesis, such as *SOX17* and *CDKN2A/2B*, as well as posttranscriptional regulation of gene expression, are associated with the development of intracranial aneurysms.[44] Therefore, in cases without an identifiable cause, these genetic factors may contribute to aneurysm formation. Although the elucidation of genetic predisposition in intracranial aneurysm development holds promise for clinical applications such as identifying high-risk individuals and developing pharmacological therapies, its translation into routine clinical practice has yet to be achieved.[44]

The clinical presentation varies with the aneurysm location. Subarachnoid hemorrhage has been reported in the intracranial, intracanalicular, and terminal branch (particularly the anterior ethmoidal branch) types.[10,17,43] In the terminal branch type, intraparenchymal hematomas in the frontal lobe base may also occur.[43] In contrast, the intraorbital and intracanalicular types often present with intraorbital hemorrhage, visual dysfunction, ophthalmoplegia, and proptosis.[31] Notably, significant visual impairment is frequently observed even in unruptured aneurysms, especially at locations other than the terminal branch type. This is thought to be due to direct compression of the optic nerve by the aneurysm.[14] In the intracanalicular type, where the OphA runs in close proximity to the optic nerve, all reported cases demonstrated severe visual dysfunction regardless of the rupture status, supporting this hypothesis.

The patient in the present case exhibited a pearl and string sign and showed rapid growth over a short period, leading to the diagnosis of a dissecting aneurysm. Including the current case, only 4 cases of dissecting intraorbital OphA aneurysms have been reported[18,21,35] (Table 2), highlighting their extreme rarity. The patients presented with severe visual impairment despite unruptured aneurysm. Treatment was administered in 3 cases, with visual improvement achieved in 2 cases, including the present case. In both cases, internal trapping was performed after preoperative BTO, which confirmed adequate collateral flow to the OphA. In contrast, visual improvement was not achieved in 1 case in which ligation of the ipsilateral common carotid artery was performed, likely because of the simultaneous loss of both aneurysmal flow and collateral circulation to the OphA. Direct surgical trapping may be considered as an alternative treatment, but it requires removal of the superior orbital wall or optic canal roof, posing a risk of iatrogenic optic nerve injury.[15] In 2 previously reported cases of

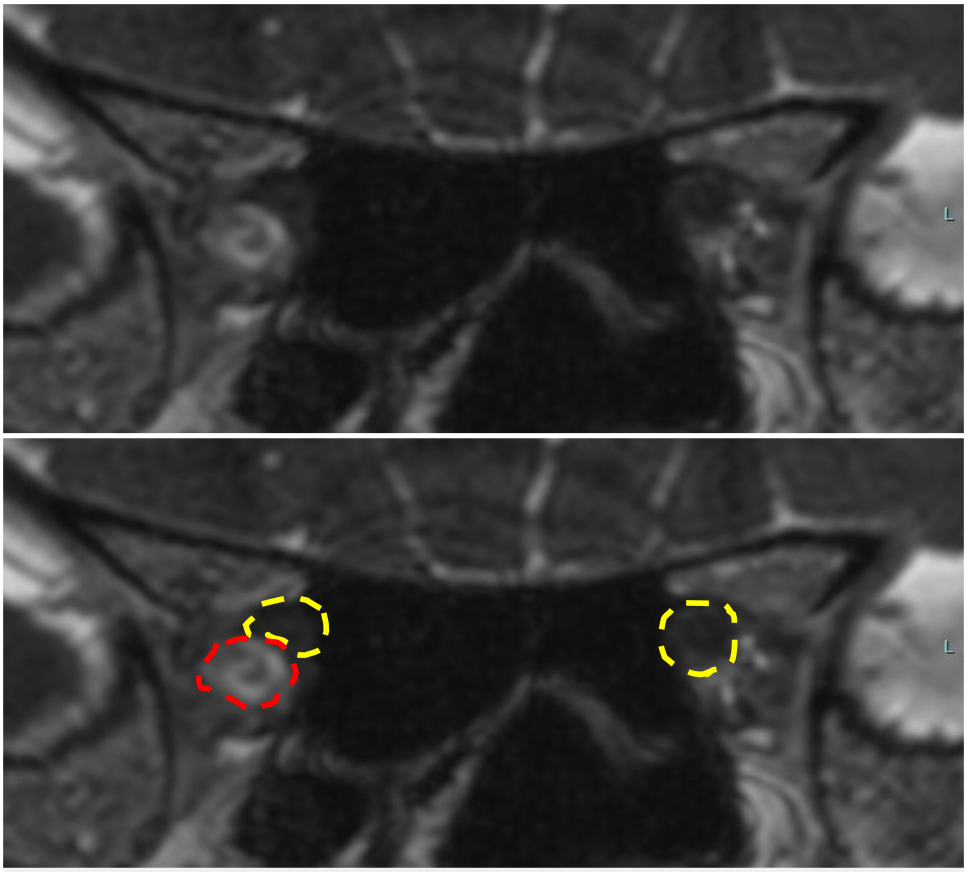

FIG. 2. Constructive interference in steady state MR image showing compression and deformation of the right optic nerve from the inferolateral side by the aneurysm.

intraorbital saccular aneurysms treated with direct trapping,[24,27] aneurysm obliteration and retinal blush via the external carotid collateral pathways were confirmed on postoperative digital subtraction angiography; however, no visual recovery was achieved, suggesting the possibility of intraoperative nerve damage. In contrast, endovascular treatment avoids the direct manipulation of the optic nerve and is minimally invasive.[15] Furthermore, performing BTO at the internal carotid artery at the origin of the OphA allows for real-time evaluation of collateral circulation. Despite these advantages, endovascular treatment is associated with a risk of thromboembolic complications, requiring appropriate perioperative and intraoperative antithrombotic management. Additionally, catheter navigation to the lesion can be technically demanding depending on the anatomical variation of the OphA origin and the presence of atherosclerotic changes. Thus, meticulous preoperative planning and simulation are essential. Considering these factors, this strategy may be particularly advantageous in treatment planning aimed at preserving visual function.

Anatomically, the OphA is divided into three segments within the orbit: the first segment from the orbital apex to the medial turn, the second segment coursing laterally to medially over the optic nerve, and the more distal third segment.[45] The junction of the first and second segments of the OphA gives rise to crucial branches, including the central retinal artery, lateral posterior ciliary artery, and medial posterior ciliary artery.[45] Occlusion of these branches during treatment can cause significant visual deficits. Therefore, during treatments that may result in occlusion of the OphA, it is essential to perform BTO preoperatively to confirm the presence of collateral circulation to these critical branches[35] and to avoid embolization within this critical segment. In the present case, careful consideration of these anatomical and physiological factors enabled successful treatment, with favorable visual outcomes.

Conservative management is generally preferred in asymptomatic patients. However, prompt intervention is recommended in cases of visual deterioration.[2] In the present case, treatment was performed approximately 5 months after the visual symptoms developed, later than in any previously reported case, but near-complete visual recovery was achieved. In the present case, neither ischemic changes nor optic nerve atrophy was observed on fundoscopic examination, and MRI revealed no signs of optic nerve atrophy or abnormal signal intensity. These findings may be useful in predicting the potential for visual function improvement following treatment.

Lessons

POAAs are exceedingly rare but clinically important because of the high risk of severe visual dysfunction. We report a rare case of an intraorbital dissecting POAA that was successfully treated with internal trapping of the lesion after confirming adequate collateral circulation using BTO, leading to visual improvement. Early treatment

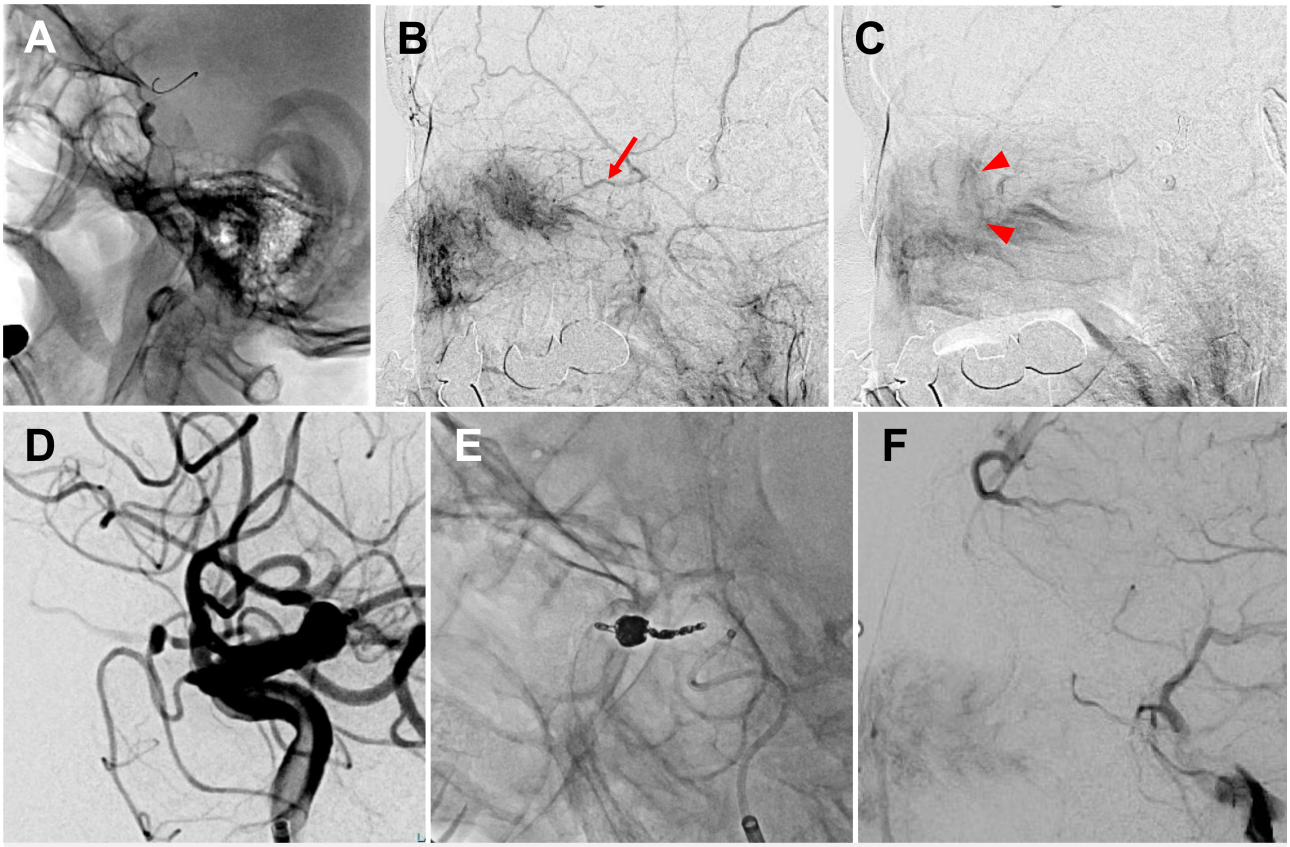

FIG. 3. A–C: BTO at the origin of the OphA. The OphA is visualized in a retrograde fashion (*arrow*), and retinal blush is observed (*arrowheads*). **D:** Preembolization angiogram of the right internal carotid showing that the aneurysm is enlarged compared with the initial digital subtraction angiogram. **E:** Post–internal trapping. **F:** Immediately postoperative angiogram of the right common carotid showing the retinal blush via collateral circulation from the external carotid artery system.

TABLE 1. Reported cases of POAA

Type	No. of Cases	Median Age, yrs (IQR)	Sex, M/F	Ruptured Aneurysm, n (%)	Aneurysm Morphology, Saccular/Fusiform	Clinical Presentation, n (%)
Intracranial	12	43 (29.8–55.2)	7/5	1 (8.3)	12/0	Vision loss: 7 (58)
Intracanalicular	4	43 (33–45)	4/0	1 (25.0)	1/3	Vision loss: 4 (100)
Intraorbital	22	51.5 (44–60)	16/6	2 (9.1)	19/3	Vision loss: 19 (86), proptosis: 9 (41)
Terminal branch	8	56.5 (49.5–64.3)	4/4	6 (75)	7/1	Vision loss: 1 (12.5)

TABLE 2. Reported cases of dissecting intraorbital OphA aneurysms

Authors & Year	Age, yrs/Sex	Ruptured Aneurysm	Clinical Presentation	Treatment of Aneurysm	Visual Outcome
Mortada, 1961[18]	50s/F	No	Vision loss, proptosis	CCA ligation	No improvement
Danziger & Bloch, 1974[21]	30s/M	No	Vision loss	Conservative treatment	No improvement
Sattur et al., 2019[35]	40s/M	No	Vision loss	BTO, internal trapping	Improved
Present case	70s/F	No	Vision loss, ptosis	BTO, internal trapping	Improved

CCA = common carotid artery.

and preoperative evaluation of collateral circulation using BTO are essential in patients presenting with visual decline. Internal trapping appears to be a promising treatment strategy when collateral circulation is preserved.

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Disclosures

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Author Contributions

Conception and design: Haruma, Izumihara, Sugiu, Kimura. Acquisition of data: Izumihara, Hirata, Kimura. Analysis and interpretation of data: Izumihara, Tanaka. Drafting the article: Izumihara. Critically revising the article: Haruma, Izumihara, Baba, Fujita, Hirata, Sotome, Kawakami, Kimura, Hiramatsu, Tanaka. Reviewed submitted version of manuscript: Haruma, Sugiu, Kimura, Hiramatsu, Tanaka. Approved the final version of the manuscript on behalf of all authors: Haruma. Administrative/technical/material support: Sugiu. Study supervision: Haruma, Sugiu.

Supplemental Information

Previous Presentations

Previously presented at the 99th Chugoku-Shikoku Regional Meeting of the Japan Neurosurgical Society, Okayama, Japan, April 6, 2025 (Izumihara K, Haruma J, Tanaka S, et al. A case of endovascular internal trapping for symptomatic dissecting ophthalmic artery aneurysm).

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