Visual dysfunction and neurodegeneration caused by severe inflammatory optic neuropathy after coil embolization of a paraclinoid aneurysm: illustrative case

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BACKGROUND Visual impairment due to delayed optic neuropathy is a rare complication of the endovascular treatment of paraclinoid aneurysms. An inflammatory response induced in the treated aneurysm wall is a known mechanism underlying this pathophysiology. The authors describe a case with severe and progressive optic neuropathy leading to neuronal degeneration and severe visual dysfunction.

OBSERVATIONS A 42-year-old female with a history of surgical clipping for a paraclinoid aneurysm presented with a recurrence. Although the patient was unaware of any visual dysfunction, a preoperative ophthalmological examination revealed mild inferior quadrantanopia in the left eye. The coil embolization procedure was uneventful; however, the following day, the patient experienced progressive visual impairment, which worsened despite the initiation of steroid therapy. Ophthalmological examination revealed a severe decrease in visual acuity and further deterioration of the visual field. Magnetic resonance imaging showed remarkable swelling and edema of the left optic nerve adjacent to the treated aneurysm. Despite continued steroid therapy, the patient’s visual function did not recover well due to subsequent optic nerve degeneration.

LESSONS Optic neuropathy after endovascular procedures can lead to severe visual dysfunction. Careful management is essential, particularly when treating a symptomatic paraclinoid aneurysm, even if symptoms are only apparent on detailed examination.

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KEYWORDS visual impairment; optic neuropathy; cerebral aneurysm; paraclinoid aneurysm; coil embolization

Aneurysms of the internal carotid artery (ICA) that arise near the origin of the ophthalmic artery, a so-called paraclinoid aneurysm, can cause visual dysfunction because of their proximity to both the optic nerve and the ophthalmic artery.1,2 Traditionally, either surgical clipping3 or endovascular coiling4 has been performed to treat these aneurysms. More recently, flow diversion (FD) therapy5 has emerged as an attractive and compelling treatment alternative.

Among these treatment modalities, endovascular approaches, including coiling and FD, are generally considered more protective of the optic nerve and visual function given their minimally invasive nature compared to surgical clipping.6 Theoretically, endovascular procedures have a lower risk of causing any injury or even minor stress to the optic nerve, as these approaches do not necessitate direct exposure or manipulation. In support of this theory, a recent meta-analysis2 showed a higher incidence of iatrogenic visual impairment—in terms of both worsening and new onset—after clipping surgery (worsening 11%, new onset 1%) than after coil embolization (worsening 9%, new onset 0%) or FD treatment (worsening 5%, new onset 0%). Endovascular approaches appear to have a protective advantage over surgical clipping in terms of postoperative complications of visual impairment.

Despite these advantages, there have been sparse case reports1,4,7–9 of delayed visual impairment after endovascular treatments, including coil embolization1,4,8,9 and FD treatment10 of paraclinoid aneurysms. The delayed onset of this complication (more than 24 hours after surgery) and the fact that it typically responds well to steroid therapy suggest that the underlying mechanism goes beyond simple physical compression of the optic nerve by the aneurysmal mass. An inflammatory response,

ABBREVIATIONS CISS = coronal constructive interference in steady state; D/N = dome/neck; FD = flow diversion; ICA = internal carotid artery; MRI = magnetic resonance imaging; OCT = optical coherence tomography.

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possibly induced by the aneurysm treatment, may contribute to the development of inflammatory optic neuropathy and this visual dysfunction complication. \(^6\),\(^9\) Therefore, systemic steroid therapy is commonly used to treat this complication and appears to be effective.\(^7\),\(^9\)

Here, we present a case of significant visual dysfunction after coil embolization of a paraclinoid aneurysm. The severe clinical course, in addition to the characteristic radiological and ophthalmological findings, provides important insights for a better understanding of this complication. We believe that the knowledge gained from this case will help to establish an optimal management strategy to prevent or mitigate this complication.

Illustrative Case

History and Examination

A 42-year-old female underwent clipping surgery for a large left paraclinoid aneurysm at the referring hospital approximately 15 years ago. Follow-up magnetic resonance imaging (MRI) suggested a recurrence of the aneurysm, which was confirmed by cerebral angiography (Fig. 1A). The recurrent aneurysm size was measured as 8.1 mm \(\times\) 6.9 mm and 3.1 mm for the dome and neck, respectively, with a dome/neck (D/N) aspect ratio of 2.61 and an aneurysm sac volume of 0.234 \(\text{cm}^3\). The ophthalmic artery originated proximal to the aneurysm neck with clear separation from the aneurysmal structure. Given the relatively high D/N aspect ratio, a simple coil embolization procedure was planned to treat the aneurysm. Preoperative ophthalmological examination showed normal visual acuity (left vision \(\text{L.V.} = 1.2\), right vision \(\text{R.V.} = 0.9\)) but inferior quadrantanopia in the left eye (Fig. 1B), which was thought to be attributable to mild optic nerve compression by the recurrent aneurysm, as demonstrated on coronal constructive interference in steady state (CISS) MRI (Fig. 1C).

Operation

The coil embolization procedure was performed with the patient under general anesthesia. After the first framing coil (7 mm \(\times\) 20 cm, Axium Prime Frame, Medtronic), 13 bare platinum coils with sizes ranging from 6 mm \(\times\) 10 cm to 1.5 mm \(\times\) 2 cm were deployed (Fig. 1D). Packing density was calculated as 25.3%. The final angiogram (Fig. 1E) showed a favorable coil embolization status within the aneurysm dome, albeit with a small residual neck, which was classified as a Raymond-Roy occlusion class 2.\(^8\) The ophthalmic artery remained patent. Postoperatively, the patient regained consciousness immediately after weaning from general anesthesia and did not complain of any neurological deficits, including visual impairment.

Postoperative Course

The day after the procedure, the patient complained of visual disturbance in her left eye. Because MRI did not show ischemic and hemorrhagic lesions in the brain parenchyma, the symptom was assumed to be due to a postoperative optic neuropathy induced by an evoked inflammatory response involving the adjacent optic nerve. Despite the initiation of steroid therapy (prednisolone 50 mg/d), the patient's visual impairment progressed. On postoperative day 4, ophthalmic examination revealed severe visual dysfunction in

FIG. 1. A: Preoperative cerebral angiogram showing a recurrent paraclinoid aneurysm in the anterior wall of the ICA. The ophthalmic artery (arrow) originates proximal to the aneurysm neck with clear separation from the aneurysmal structure. B: Preoperative ophthalmological examination showed normal visual acuity (left vision \(\text{L.V.} = 1.2\), right vision \(\text{R.V.} = 0.9\)) but inferior quadrantanopia in the left eye. C: Coronal CISS MRI shows that the recurrent aneurysm (dashed line) is slightly compressing the left optic nerve (arrow). D: After the first framing coil, 13 bare platinum coils with sizes ranging from 6 mm \(\times\) 10 cm to 1.5 mm \(\times\) 2 cm were deployed. E: Final angiogram showing a favorable coil embolization status within the aneurysm dome, albeit with a small residual neck.
A L.V = 0.1 R.V = 1.0

![Diagram](image)

**FIG. 2.** A: Postoperative ophthalmic examination revealed severe visual dysfunction in the left eye, with significantly decreased visual acuity (L.V = 0.1, R.V = 1.0) and further visual field deterioration (central scotoma and progression of quadrantanopia). B: Coronal CISS MRI revealed profound thickening of the optic nerve with severe edema extending to the chiasma and right optic nerve (arrows). C: Axial postcontrast T1-weighted MRI displaying surrounding enhancement in the aneurysm wall, with enhancement extending to the adjacent optic nerve.

the left eye, with significantly decreased visual acuity (left vision = 0.1, right vision = 1.0) and further visual field deterioration (central scotoma and progression of quadrantanopia). Coronal CISS MRI (Fig. 2B) focusing on the optic nerve and adjacent structures revealed profound thickening of the optic nerve with severe edema extending to the chiasma and right optic nerve. Additionally, postcontrast T1-weighted MRI (Fig. 2C) displayed surrounding enhancement in the aneurysm wall, with enhancement extending to the adjacent optic nerve, suggesting an inflammatory reaction within the aneurysm wall, resulting in severe optic neuropathy.

Steroid treatment was continued; however, even after 3 months, there was no improvement in visual function, in terms of both visual acuity and visual field defects (Fig. 3A). Optical coherence tomography (OCT) imaging (Fig. 3B) showed thinning of the retinal nerve fiber layer, and follow-up CISS MRI (Fig. 3C) revealed cyst formation within the optic nerve and chiasma. Both findings were consistent with apoptotic degeneration of the injured optic nerve. The systemic steroid therapy was tapered and discontinued after 6 months. The patient remains under observation in our outpatient clinic with persistent visual dysfunction.

**Patient Informed Consent**

The necessary patient informed consent was obtained in this study.

**Discussion**

In this report, we present a case of severe inflammatory optic neuropathy that developed after coil embolization treatment of a paraclinoid aneurysm, resulting in significant visual dysfunction and apoptotic degeneration of the optic nerve. Follow-up MRI and ophthalmological examination revealed characteristic findings and their temporal progression, aspects that have not been clearly demonstrated to date. The knowledge gained from this case will help to understand the unclear pathophysiology of this serious complication and emphasize the importance of establishing an optimal therapeutic strategy to prevent or mitigate this complication.

**Observations**

Instances of optic neuropathy as a complication after endovascular treatment of cerebral aneurysms have been rarely documented. Among the cases reported, the majority of aneurysms were located in the paraclinoid region, treated with simple coil embolization, and predominantly classified as large aneurysms measuring more than 10 mm. Previous studies have suggested that the visual impairment observed after coil embolization was mainly due to direct physical compression of the adjacent optic nerve by the enlarged aneurysmal mass. However, recent reports have suggested that other molecular mechanisms may be involved in this complication. Stracke et al. presented a case of delayed optic neuropathy showing remarkable gadolinium enhancement effects in both the treated aneurysm wall and the adjacent optic nerve, similar to those observed in the present case. Based on this characteristic radiological finding, besides a favorable response to systemic steroid therapy, the authors concluded that an inflammatory response induced by the endovascular procedure could contribute to the occurrence of posttreatment optic neuropathy.

Although the precise mechanisms remain under investigation, intra-aneurysmal thrombus formation appears to play a crucial role in triggering inflammatory responses. During the thrombus formation phase after endovascular treatment, which could also be called the “healing phase,” aggregated platelets could be the source of proinflammatory cytokines, leading to the migration of immune cells into the thrombus and aneurysm wall, where they may provoke further inflammatory responses. The rapid accumulation of a large volume of thrombus within the aneurysm may provoke a more excessive inflammatory response, potentially precipitating this complication. In support of this theory, several authors have reported cases of posttreatment optic neuropathy even in patients treated with FD, a modality known for its potential to reduce the size of the aneurysm sac.

A distinctive feature of the present case compared to previous case reports was its resistance to systemic steroid therapy and the severe clinical course that followed, characterized by remarkable optic nerve swelling in the subacute phase and subsequent neurodegeneration. Two possible factors may account for the unusual and severe clinical course observed in the present case. One probable reason is that the patient previously underwent craniotomy and clipping surgery at the same region, which might have resulted in minor damage to the optic nerve. This preexisting vulnerability of the optic nerve could have exacerbated the damage caused by neuropathy, leading to a more severe injury than typically observed. Another considerable factor is the influence of foreign body reactions associated with coil embolization. Complications arising from such allergic reactions typically occur later than 2 weeks postsurgery and generally
reduce the mass signs of the aneurysms. When treating a paraclinoid aneurysm, especially if there is evidence of optic nerve compression, even if mild, as in our case, both prophylactic steroid therapy and FD treatment should be considered.

The use of prophylactic systemic steroid therapy, particularly in the preoperative period, may be beneficial in preventing or reducing this complication. In addition, the therapeutic option of FD treatment compared to conventional coil embolization could potentially reduce the severity of this complication. FD treatment has been reported to reduce the mass signs of the aneurysms. When treating a paraclinoid aneurysm, especially if there is evidence of optic nerve compression, even if mild, as in our case, both prophylactic steroid therapy and FD treatment should be considered.

Lessons
After endovascular treatment of paraclinoid aneurysms, severe optic neuropathy can occur due to an exaggerated inflammatory response in the aneurysm wall and adjacent optic nerve, which is triggered by rapid thrombosis. Although systemic steroid therapy is generally effective, some cases are resistant, which can result in significant visual dysfunction. Therefore, particular care should be taken in the preoperative management of patients at high risk for optic neuropathy, such as patients with any visual symptoms similar to those in the present case. To evaluate the risk of this complication, a detailed ophthalmological examination and adequate review of the medical history would be essential.

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References
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