Penetration of the optic chiasm by a ruptured anterior communicating artery aneurysm

Case report

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Although anterior communicating artery (ACoA) aneurysms are one of the most frequent causes of subarachnoid hemorrhage, they are less frequently a cause of visual disturbances. Most of the previous reports of visual deterioration related to aneurysms have described large or giant aneurysms, and the mechanism of visual loss has been attributed to compression of the optic pathways either by aneurysm or by an associated hematoma. We recently encountered a case of a ruptured ACoA aneurysm that penetrated the right side of the optic chiasm causing left homonymous hemianopsia and right visual acuity disturbance.

Case Report

This 40-year-old man presented with an abrupt onset of bilateral blurred vision and slight headache. There was no alteration of his level of consciousness.
History and Examination. Because the patient noted a gradual deterioration of right visual acuity, he consulted an ophthalmologist 1 week after the onset. The patient was found to have left homonymous hemianopsia and right visual acuity disturbance (finger counting). A non-contrast-enhanced computerized tomography (CT) scan revealed a high-density mass in the suprasellar region (Fig. 1A) and a contrast-enhanced CT scan revealed the lesion to be an ACoA aneurysm (Fig. 1B). Although the arteries around the aneurysm were enhanced, the mass itself was not, suggesting that part of the aneurysm had thrombosed. The patient was referred to the department of neurological surgery where neurological examination revealed the visual symptoms mentioned previously. There were no other neurological deficits. Right internal carotid angiography disclosed a 7-mm ACoA aneurysm that was projecting inferiorly (Fig. 2).

Operation. The patient underwent a right frontotemporal craniotomy 2 weeks after the onset of blurred vision. A small amount of subarachnoid clot was observed around the chiasmal region. The aneurysm originated from the junction of the right A1 and A2 segments and protruded downward, penetrating the right side of the optic chiasm (arrows). After clipping the neck of the aneurysm, the dome of the aneurysm was resected, and it was confirmed that the aneurysm penetrated the right half of the optic chiasm (black arrows) and the thrombosed aneurysm compressed the right optic tract (white arrows). An = aneurysm; IC = internal carotid artery; II = optic nerve.

Postoperative Course. The patient’s postoperative course was uneventful. A postoperative angiogram showed complete clipping of the aneurysm (Fig. 4). The patient’s visual disturbance persisted after the operation, suggesting that the damage to the optic apparatus was irreversible in this case.

Discussion

Although ACoA aneurysms may be located close to the visual pathways, they rarely cause visual disturbances. Most previous reports of ACoA aneurysms causing visual disturbance are those that describe large or giant an-
eurysms. The mechanism of visual disturbance in these cases is ascribed to compression of the visual pathways either by the aneurysm itself or an associated hematoma. The present case is the first report of a ruptured ACoA aneurysm causing visual disturbance by penetrating the optic chiasm.

There have been two reported cases of a fenestrated oculomotor nerve associated with an internal carotid–posterior communicating artery aneurysm. Beatty reported a case in which the optic nerve was split by a carotid-ophthalmic artery aneurysm. There were no preoperative symptoms of visual disturbance in that case, and the aneurysm was considered to have developed from a vessel already penetrating the optic nerve. In the present case, we believe that the aneurysm penetrated the optic chiasm when it ruptured, causing the patient to experience a sudden onset of blurred vision and slight headache. The subsequent gradual deterioration of visual acuity was the result of either optic nerve edema or the increased size of the thrombosed aneurysm.

The pattern of visual disturbance in reported cases of ACoA aneurysms is highly variable. Although some reports describe homonymous or bitemporal visual loss, most mention predominantly monocular visual loss, indicating pressure on a single optic nerve. The type of visual loss is commonly determined by the position of the aneurysm in relation to the optic nerves and chiasm, and this is consistent with the present case. Ruben and Afshar presented the case of an ACoA aneurysm that caused visual failure 6 days after the patient suffered a subarachnoid hemorrhage precipitated by a cerebral vasospasm. This case demonstrates that ischemia resulting from arterial spasm, as well as direct compression of the visual pathways, may play a role in visual failure.

In summary, we present the case of a ruptured ACoA aneurysm that penetrated the optic chiasm. The pattern of visual symptoms corresponded to the portion of the optic apparatus that had been penetrated by the aneurysm. Although the aneurysm was successfully clipped, the visual symptoms did not remit after surgery, suggesting that the damage to the visual pathways caused by this aneurysm was irreversible.

Acknowledgments

The authors thank Dr. Massimo S. Fiandaca for his helpful suggestions. We also thank Mr. Hideki Wakimoto for his editorial assistance.

References


Manuscript received November 25, 1996. Accepted in final form March 5, 1997.

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