**Photophobia as the Visual Manifestation of Chiasmal Compression by Unruptured Anterior Communicating Artery Aneurysm**

—Case Report—

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**Abstract**

A 37-year-old woman presented with photophobia without visual loss associated with chiasmal compression by an unruptured anterior communicating artery (AcomA) aneurysm. She had suffered progressive photophobia for one year. Neuroimaging indicated an AcomA aneurysm attached to the chiasm. Photophobia was resolved following clipping of the aneurysm. AcomA aneurysm should be considered in patients who experience photophobia without visual loss.

Key words: photophobia, chiasmal compression, anterior communicating artery aneurysm

**Introduction**

Photophobia is defined as visual discomfort suffered during exposure of the eyes to light, and is generally accepted in the etiology of headache, particularly in relation to migraines. Photophobia is most commonly associated with anterior segmental disease of the eye. The mechanism causing light intolerance associated with corneal disruption, iritis, meningitis, and migraines remains unclear, and is presumably related to irritation of the trigeminal afferent pathway. Anterior segmental disease of the eye, subarachnoid hemorrhage, unruptured cavernous carotid artery aneurysm, and retrochiasmal demyelination are all well known causes of photophobia. A small number of reported cases have suggested that photophobia is a rare symptom of chiasmal compression. In general, unruptured large anterior communicating artery (AcomA) aneurysm is well known cause of visual loss (loss of visual acuity and visual field defect).

Here we describe a case of photophobia before onset of visual loss with chiasmal compression by an unruptured AcomA aneurysm.

**Case Report**

A 37-year-old woman presented with complaints of photophobia. She was slightly overweight and habitually drank alcohol and smoked. Laboratory data on admission showed no abnormal findings except a high neutral fat level. She claimed that she had suffered from headache since middle school. She started having severe pain in her right eye and the right side of her head following a sense of discomfort, which appeared as soon as she saw light, one year previously. Her headache was not classical type of migraine. The reaction was most obvious when she saw light reflected from celluloid products or the surface of a river. She also claimed that she had trouble looking at mobile telephone screens. Her symptoms had recently worsened, so she visited our clinic.

Neuro-ophthalmic examination (Vd 0.4, corrected 1.0; Vs 0.7, corrected 1.2) detected nothing abnormal in light perception or fundus oculi, and Goldmann visual field results were normal. Magnetic resonance (MR) imaging showed ischemic change for her age. MR angiography revealed an AcomA aneurysm, and angiography revealed the aneurysm (8 mm in size) projecting inferiorly, and the aplastic left A1 segment of the anterior cerebral artery (Fig. 1). Heavily T2-weighted MR imaging showed that the
Fig. 1 Right internal carotid angiograms, anteroposterior view (A) and lateral view (B), showing an anterior communicating artery saccular aneurysm.

Fig. 2 Coronal (A) and sagittal (B) heavily T2-weighted magnetic resonance images revealing an aneurysm attached to the right side of the chiasm.

Fig. 3 Intraoperative photographs obtained before aneurysm clipping (A) showing attachment of the aneurysm to the chiasm, and after aneurysm clipping (B) showing obvious indentation of the chiasm by the aneurysm. A1: A1 segment of the anterior cerebral artery, AN: aneurysm, II: optic nerve.

The right fronto-temporal craniotomy and trans-sylvian approach was performed. AcomA aneurysm, right A1 segment and bilateral A2 segments of anterior cerebral artery were identified. The patient underwent neck clipping of the AcomA aneurysm. Intraoperative findings revealed chiasmal compression with indentation by the aneurysm (Fig. 3). Postoperatively, the patient noted improvement of her headache and photophobia.

Discussion

The present patient suffered photophobia associated with chiasmal compression caused by an AcomA aneurysm without other visual symptoms. The symptoms of positive visual phenomena can be divided into two groups (photopsia and photophobia). Symptoms such as sparks, flashes, or colored lights constitute photopsia. Demyelinating optic neuritis is a well-known cause of photopsia. Sensations of dazzling or glaring light, general visual discomfort in bright light, and awareness of improved visual function in dim illumination constitute photophobia. Photophobia is frequently associated with diseases of the anterior structures of the eye or meninges. The causative mechanism is thought to involve the trigeminal pathway with possible input from the pretectal nuclei, occipital lobe, and thalamus.

Interestingly, photophobia may have been caused by chiasmal compression in a small number of patients. Photophobia was observed in one patient with a hypophyseal tumor, and in another patient with recurrent craniopharyngioma. Photophobia was detected in five patients, three with pituitary adenomas, one with craniopharyngioma, and one with clivus chordoma. Among them, only one patient with pituitary adenoma suffered onset of photophobia before onset of visual loss. Photopsia or photophobia was found in nine patients with chiasmal compression by a mass lesion. Among these, only three patients, two with pituitary adenomas and one with perioptic meningioma, suffered onset of photopsia or photophobia before onset of visual loss. Therefore, only four patients, three with pituitary adenomas and one with perioptic meningioma, have had photophobia caused by chiasmal compression before onset of visual loss. Our patient suffered photophobia caused by chiasmal compression by an aneurysm before onset of visual loss.

The mechanism of photophobia caused by chiasmal compression by tumor may involve release of blood or cystic components from the suprasellar tumor, causing meningeal irritation, or chemical meningitis causing meningeal irritation, thus producing photophobia and headache. Another possibility is stretching of pain-sensitive structures at the base of the brain, associated with meninges or
blood vessels, transmitted via the trigeminal afferents and processed centrally. These pathophysiology presumably cause irritation of the basal meninges around the diaphragma sellae and thus lead to photophobia.

In the present case, we detected indentation of the chiasm, so both compression and degeneration of the chiasm could have occurred. Rapid growth of aneurysm could damage to the chiasm and cause the visual loss. We speculate that gradual compression of the chiasm by aneurysm resulted in photophobia. This is same mechanism on photophobia in cases of tumor. Certainly neurosurgeons have encountered many aneurysms like our case, but few cases complaining of photophobia have been experienced. Compression of the optic chiasm by an aneurysm should be considered in patients who experience persistent photophobia without visual loss explained by ocular abnormalities.

References


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