Pupil-Sparing Oculomotor Nerve Paresis as an Early Symptom of Unruptured Internal Carotid-Posterior Communicating Artery Aneurysms
—Three Case Reports—

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Abstract

Oculomotor nerve paresis caused by internal carotid-posterior communicating artery (IC-PC) aneurysm usually manifests with pupillary dysfunction. Recently, we treated three patients with unruptured IC-PC aneurysms initially manifesting as pupil-sparing oculomotor nerve paresis, which resolved after clipping of the aneurysms. Review of the 56 patients admitted to our hospital with unruptured IC-PC aneurysms between January 2000 and December 2006 identified 6 patients with oculomotor nerve disturbances, and the 3 present cases with pupil sparing. The incidence of IC-PC aneurysms manifesting as pupil-sparing oculomotor nerve paresis may be increasing with improved accessibility to medical services and wider awareness of oculomotor nerve paresis as a symptom of cerebral aneurysms. Cerebral angiography should be performed in patients with pupil-sparing oculomotor nerve paresis.

Key words: pupil sparing, oculomotor nerve paresis, unruptured cerebral aneurysm, posterior communicating artery

Introduction

Oculomotor nerve paresis is associated with intracranial aneurysm, especially at the internal carotid-posterior communicating artery (IC-PC) junction. Pupillary dysfunction is important for differentiating between aneurysmal and diabetic oculomotor nerve paresis, in which pupillary function is usually normal.2,5) However, a case of pupil-sparing oculomotor nerve paresis caused by an IC-PC aneurysm has been reported.6) Seven patients initially had normal pupils among 51 patients with IC-PC aneurysms manifesting as oculomotor nerve involvement, suggesting that the incidence of pupil sparing may be more common than previously appreciated in patients with IC-PC aneurysms and oculomotor nerve involvement.

Recently, we treated 3 patients with unruptured IC-PC aneurysm initially manifesting as pupil-sparing oculomotor nerve paresis.

Case Reports

Case 1: A 47-year-old female suffered onset of right orbital pain on June 20, 2004. She developed diplopia and visited our hospital on June 28. Computed tomography angiography failed to detect the IC-PC aneurysm at this time, whereas the edrophonium test was positive. She underwent treatment for myasthenia gravis, but her symptoms worsened. She developed right lid ptosis on June 30, although her pupillary function was still intact. She finally developed pupillary dysfunction on July 4. Digital subtraction angiography (DSA) detected the right IC-PC aneurysm (Fig. 1A). She underwent surgical clipping of the aneurysm and her symptoms had fully resolved 3 months later.

Case 2: A 66-year-old female realized diplopia at the beginning of July 2004. She developed right lid ptosis on July 20, and visited a local doctor. She was referred to our hospital on July 23. Right oculomotor nerve paresis with pupil sparing was detected. Angiography revealed right IC-PC aneurysm (Fig. 1B).
Fig. 1 Digital subtraction angiograms, oblique views, showing aneurysms (arrowheads) arising from the internal carotid artery at the origin of the posterior communicating artery in Cases 1 (A), 2 (B), and 3 (C). Note the narrow and long aneurysm body, especially in Cases 1 and 2.

Fig. 2 Case 3. Photographs of the ophthalmologic examination demonstrating left lid ptosis (A) and normal pupil size (B).

She underwent surgical clipping via the right pterional approach. Her symptoms had fully resolved before December 2004. 

Case 3: An 82-year-old female developed left lid ptosis and diplopia without pupil dysfunction in October 2006 (Fig. 2). She visited our hospital on October 26. The edrophonium test was negative. DSA revealed left IC-PC aneurysm on October 27 (Fig. 1C). Surgical clipping was performed. Her diplopia disappeared and lid ptosis is still improving after surgery.

Discussion

Pupil-sparing oculomotor nerve paresis may be an early symptom of enlarging IC-PC aneurysm that is unstable with impending rupture. Symptomatic unruptured aneurysms carry relatively high risk for future rupture, and early treatment improves the prognosis for patients with IC-PC aneurysms manifesting as oculomotor nerve paresis. Therefore, accurate and early diagnosis is required when treating those patients. Anatomically, parasympathetic fibers supplying pupillary functions pass along the dorsal medial surface of oculomotor nerve, so are easily compressed by IC-PC aneurysms projecting laterally from the carotid artery. If the enlarging aneurysm begins to compress the oculomotor nerve at a point where the parasympathetic fibers are not present, the pupil functions may be preserved. Therefore, the shape of the aneurysm may also contribute to the pupil-sparing presentation. The narrow and long aneurysm body in our Cases 1 and 2 may have compressed the oculomotor nerve at a point without parasympathetic fibers. Therefore, we need to pay more attention to the occurrence of pupil sparing in patients with enlarging IC-PC aneurysms.

The differential diagnosis for oculomotor nerve paresis includes diabetic neuropathies, myasthenia gravis, brain stem infarction, and cerebral aneurysms. Pupil dysfunction has long been considered important for the diagnosis of aneurysmal oculomotor nerve paresis. In a series of 84 patients with IC-PC aneurysms, seven patients had pupil-sparing oculomotor nerve paresis among the 51 patients presenting with oculomotor nerve paresis. These findings suggest that cerebral angiography should be performed in patients with pupil-sparing oculomotor nerve paresis. We identified six patients with oculomotor nerve paresis, including the present three patients with pupil sparing, among 56 patients with unruptured IC-PC aneurysms who visited our hospital between January 2000 and December 2006. Recently, cases of pupil-sparing oculomotor nerve paresis caused by basilar artery or anterior temporal artery aneurysms, or dissection of the intracranial carotid artery have also been reported. Accessibility to medical services has been greatly improved. In addition, many primary physicians have recently become aware of oculomotor nerve paresis as a symptom of cerebral aneurysm. The increased incidence of pupil-sparing oculomo-
tor nerve paresis caused by cerebral aneurysms may be a reflection of these improvements. The present cases illustrate the need to consider this presentation as an initial symptom of cerebral arterial aneurysm.

The incidence of cerebral aneurysms manifesting as pupil-sparing oculomotor nerve paresis seems to be increasing. Early treatment is required for symptomatic cerebral arterial aneurysms, so cerebral angiography should be performed in patients presenting with oculomotor nerve paresis regardless of pupil involvement.

References


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