CASE REPORT

Acute Retrobulbar Optic Neuropathy as the Sole Manifestation of Subarachnoid Haemorrhage from a Ruptured Anterior Communicating Artery Aneurysm

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ABSTRACT

Anterior communicating artery aneurysm is the most common form of intracranial aneurysm and subarachnoid haemorrhage (SAH) is the common presenting feature of anterior communicating artery aneurysms. In general, patients with SAH from anterior communicating artery aneurysm present with neurological deficit. We report an interesting case of a 60-year-old man who presented with acute monocular vision loss accompanied by periorbital pain without any neurological deficit, finally diagnosed with SAH from ruptured anterior communicating artery aneurysm. Five months after immediate craniotomy with aneurysm neck clipping, his visual acuity was improved to 20/63 with a pale optic disc appearance. Acute retrobulbar optic neuropathy may be the sole manifestation of SAH from ruptured anterior communicating artery aneurysm. Unilateral decrease of visual acuity with periorbital pain, in the absence of other neurological change, may be the initial and isolated sign.

Keywords: Anterior communicating artery aneurysm, retrobulbar optic neuropathy, subarachnoid haemorrhage

INTRODUCTION

Anterior communicating artery aneurysm is the most common form of intracranial aneurysm, accounting for 25–38% of total cerebral aneurysm cases.¹ Subarachnoid haemorrhage (SAH) is the common presenting feature of anterior communicating artery aneurysms.² In general, patients with SAH from anterior communicating artery aneurysm are presented with neurological deficit.² Since aneurysm of the anterior communicating artery is located closely to the optic nerve, it can also cause visual symptoms by compressing the optic nerve or rupture of aneurysm with focal haematoma formation.¹ We here present an unusual case of a patient confirmed as SAH from a ruptured anterior communicating artery aneurysm, initially manifested as an acute retrobulbar optic neuropathy without any other neurological change.

CASE REPORT

A 60-year-old man presented with decreased visual acuity in his right eye for 2 days. He had underlying disease of hypertension controlled with medication. He showed no apparent neurological symptoms. He also complained of periorbital pain in his right eye and headaches for 3 days. His best-corrected visual acuity was hand motion in the right eye and 20/20 in the left eye. He was orthophoric without any limitation of eye movement, and slit-lamp findings of the anterior segment were nonspecific in both eyes. He showed positive signs of relative afferent pupillary defect and sluggish light reflex in his right eye. The fundusoscopic findings were also nonspecific (Figure 1A). Orbital magnetic resonance imaging (MRI) showed increased perineural signal intensity of the right optic nerve (Figure 1B). Intravenous

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FIGURE 1 (A) Fundus photography of the initial presentation showing nonspecific findings in both eyes. (B) Orbital magnetic resonance imaging (MRI) showing enhancement of the right optic nerve sheath (white arrows) in this fat-suppressed, postcontrast, T1-weighted image.

steroid pulse therapy (methylprednisolone 250 mg every 6 hours) was started with the impression of retrobulbar optic neuritis of the right eye. On the second day, we studied the cerebrospinal fluid (CSF) analysis, and it showed a suspicious subarachnoid haemorrhage. On the subsequent brain computed tomography (CT) angiography (Figure 2A) and four-vessel angiography (Figure 2B), a saccular aneurysm in the anterior communicating artery was detected, measuring about $4.8 \times 4.4 \times 7.3$ mm with 2.1-mm-sized neck. Immediate craniotomy with aneurysm neck clipping was performed. On the postoperative CT angiography, there was no remnant pathologic lesion. Five months after the operation, his visual acuity of the right eye was improved to 20/63 with a pale optic disc appearance.

DISCUSSION

The anterior communicating artery is located closely to the optic nerve. Aneurysm arising from anterior communicating artery can produce visual symptoms according to the direction and size.¹ Patients may

complain of vision loss with transient headaches.³ Other visual symptoms include bitemporal or homonymous hemianopsia due to the compression of the optic chiasm or tract.¹ Ruptured anterior communicating aneurysm with SAH generally is accompanied by focal neurological deficit.²

Acute retrobulbar optic neuropathy due to anterior communicating artery aneurysm is rare, and there have been a few case reports of acute retrobulbar optic neuropathy related to ruptured anterior communicating artery aneurysm.^{3,4} In these reports, the patients showed abnormal neurological signs such as stuporous mentality or disorientation before and after the development of visual symptoms. Our patient presented with acute monocular vision loss accompanied by periorbital pain and headaches, but unlike the previous reports, there was no neurological deficit or mental change. A relative afferent pupillary defect was present in the affected eye, and the orbit MRI findings were also suspected of optic neuritis. Subsequent CSF analysis and brain angiography examinations revealed SAH from a ruptured anterior communicating artery aneurysm. The increased perineural signal intensity of the right optic nerve on

FIGURE 2 (A) Computed tomography angiography showing saccular aneurysm in anterior communicating artery. (B) Right internal carotid artery angiogram showing an anterior communicating artery saccular aneurysm (white arrow).

orbital MRI (Figure 1B) could be the finding compatible with perineural haematoma around the optic nerve resulting from the spread of rupture of anterior communicating artery aneurysm toward the optic nerve. Direct optic nerve compression from perineural haematoma could be the possible mechanism responsible for retrobulbar optic neuropathy in our case.

In cases of sudden unilateral visual loss combined with signs of optic nerve dysfunction, vascular and compressive lesions should be considered for the differential diagnosis.⁴

In case of SAH related to anterior communicating artery aneurysm, emergent surgical decompression with clipping of the aneurysm would have the best chance to preserve any potential vision.⁵ The associated focal neurological deficits may remain as sequelae.² In our case, the patient underwent a prompt neurosurgical treatment after the diagnosis of SAH from a ruptured anterior communicating artery aneurysm, on the third day after onset of visual symptom. His visual acuity of the affected eye recovered to 20/63, but some deficits such as visual field defect and pale optic disc remained.

In conclusion, acute retrobulbar optic neuropathy may be the sole manifestation of SAH from ruptured anterior communicating artery aneurysm. Unilateral decrease of visual acuity with periorbital pain, in the absence of other neurological change, may be the only first isolated sign.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

Note: Figures 1 and 2 of this article are available in colour online at www.informahealthcare.com/oph.

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