Ischemic Optic Neuropathy in Ruptured Anterior Communicating Artery Aneurysm

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A case of persistent monocular blindness probably resulting from the post-subarachnoid hemorrhage ischemic injury of the posterior optic nerve is reported. On admission, the patient was assessed as Hunt-Hess classification grade III, Fisher group IV. Uneventful surgery for clipping the neck of her small saccular anterior communicating artery aneurysm was performed on second hospital day via left pterional approach. She complained of total blindness of her left eye as she recovered from drowsy consciousness to be lethargic on first post-operative day; her left eye showed sluggish direct pupillary light reflex and normal optic fundoscopy. Her ensuing hospital course had been complicated by symptomatic vasoospasm, bleeding tendency, subacute epidural hematoma, and hydrocephalus. She was independent on discharge. Ophthalmologic evaluation on 60th post-subarachnoid hemorrhage day showed total blindness and optic disc atrophy of her left eye. Probable ischemic optic neuropathy is another cause of the post-subarachnoid hemorrhage visual disturbance, especially after the rupture of anterior communicating artery aneurysms.

KEY WORDS: Subarachnoid hemorrhage - Anterior communicating artery aneurysm rupture - Ischemic optic neuropathy.

Introduction

Visual disturbances complicating aneurysmal subarachnoid hemorrhage(SAH) are not uncommon, being caused mostly by various types of ocular hemorrhages(subhyaloid, preretinal, and vitreous hemorrhages).

The authors report a patient with monocular total blindness without any ocular hemorrhages following SAH due to a ruptured anterior communicating artery(ACoA) aneurysm, and discuss on its pathogenesis.

Case Report

This 61-year old Korean woman had inadequately controlled hypertension; she had been healthy otherwise till the day of admission when she suddenly complained of severe headache and vomiting followed by deterioration of consciousness. On admission, she was drowsy and showed high blood pressure(210/100) and neck stiffness.

Her bilateral pupils were isocoric with prompt light reflex. Her brain computed tomography(CT) revealed diffuse thick subarachnoid hemorrhage as well as a small intracerebral hematoma in right gyrus rectus (Fig. 1). At that time cerebral angiography was not available transiently in our hospital. So an emergency operation for the direct neck clipping of the probable AC-oA aneurysm was performed on the second hospital day.

Operation

Under general anesthesia, a percutaneous lumbar cerebrospinal fluid(CSF) drain was installed. After left pterional craniotomy and a curvilinear dural opening, ca 30cc of bloody CSF was drained through the lumbar drain to make the tense brain ‘slack’. Usual left pterional approach was performed to the ACoA complex. Briefly, as a small volume of the posteromedial corner of left gyrus rectus was removed,
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ipsilateral distal A1, Heubner’s artery, fronto-orbital branch and proximal A2 came into vision. Further careful dissection posterior to the ACoA complex disclosed three thin hypothalamic branches and the contralateral proximal A2. Right Heubner’s artery and hypoplastic A1, and the neck of the aneurysm were identified anterior to the ACoA complex. The dome of the aneurysm was found to be embedded within the contralateral gyrus rectus. A Sugita’s fenestrated aneurysm clip with 6mm straight blade was applied across the neck of the aneurysm parallel to the ACoA; ipsilateral A2 was preserved patent within the fenestration. While the aneurysm was dissected and clipped, bilateral distal A1’s were temporarily clipped twice (for 5 minutes respectively).

After the brain retraction was removed, close observation confirmed that bilateral optic nerves and the optic chiasm were not compressed by any parts of the aneurysm clip. Throughout the operative procedure, the patient’s medical conditions were smooth and uneventful; particularly her blood pressure was stable within normal range without any remarkable spontaneous or induced hypotension. There were neither evident direct surgical injury of the optic apparatus nor electrocoagulation of any vessels concerned with the blood supply of the optic nerves.

Post-operative course

Immediate postoperative recovery was smooth. On first post-operative day (POD) her consciousness improved to be somnolent, and she complained that her left eye was totally blind. She had very mild left upper eyelid swelling; she could open her left eye widely, and showed no conjunctival edema. Her left pupillary direct light reflex was sluggish. Her eyes were normal on bedside optic funduscopy. Her ocular motility was normal. She was alert around POD 7, but aggravated again to be lethargic around POD 10; transfemoral cerebral angiography (Fig. 2) revealed vasospasm at the right posterior communicating artery, left A1, and bilateral distal anterior cerebral arteries.

The angiogram also showed a well obliterated ACoA aneurysm without any residual neck or any parent artery stenosis, poor visualization of right A1, good visualization of the ophthalmic arteries, and a fetal type right posterior cerebral artery. For chemical angioplasty, 30cc of 0.3% papaverine was injected slowly into the distal part of the left internal carotid artery.

On POD 14, small left frontotemporal epidural hematoma (EDH), scanty left frontal intracerebral hematoma and marked bleeding tendency were detected. The EDH increased in size and she became stuporous on POD 15 when she was also affected by acute hospital acquired pneumonia at her right lower lung. Her EDH was removed surgically on the next day, and post-operatively her consciousness improved to be slightly drowsy.

On post-SAH day 37, the third operation was performed to remove a left frontal liquified subdural hematoma and to install a ventriculoperitoneal shunt; 2 days later she could be transferred to the general ward.

She showed persistent blindness, sluggish direct light reflex, normal optic funduscopic finding of her left eye, but further ophthalmologic examination was hindered by her mental confusion and poor cooperation. She had mild mental confusion and Grade VI+ right leg weakness on discharge (post-SAH day 52). Ophthalmologic evaluation on post-SAH day 60 showed totally blind left eye (no light perception) and left optic atrophy (Fig. 3).

Discussion

Visual disturbances in the patients with the aneurysmal SAH are caused in most occasions by post-SAH intraocular hemorrhages, by direct compression of the visual pathway by the aneurysm sac, or due to the surgical trauma of the optic apparatus during the operative clipping of the aneurysmal neck. The authors believe those causes can be ruled out in this case. In reviewing literatures, the authors found other unusual etiologies of the optic neuropathy following aneurysmal SAH. Park & Shin (1987) reported a case of

Fig. 2. Transfemoral left internal carotid arteriography on 10th postoperative day revealing vasospasm at the anterior cerebral artery as well as a well secured anterior communicating artery aneurysm without any residual neck or any parent arterial stenosis.

Fig. 3. Optic funduscopon on the 60th day after subarachnoid hemorrhage showing optic disc atrophy in the upper half, deep excavation of disc and loss of the rim and lamina cribrosa of her left eye.
bilateral blindness subsequent to an ACoA aneurysm neck clipping operation; they speculated that binocular blindness in their case resulted from the vascular insufficiency caused by induced hypotension and the compression of the eyeballs during the operation. Chung et al reported a case of combined ophthalmic artery occlusion, proptosis, compression optic neuropathy, and palsy of the third nerve following an anterior choroidal aneurysmal neck clipping operation; the authors suggested intra-operative saline in-flow into the orbit through an iatrogenically made hole at the orbital roof as the pathologic mechanism of the acute compartment syndrome of their case.

Zimmerman et al, Kang, and Noh et al reported cases of the orbital infarction syndrome after the surgery for intracranial aneurysms. Noh et al described that their 5 cases of the orbital infarction syndrome probably due to the direct compression of the eyes by the surgical scalp flap manifested proptosis, ophthalmoplegia and unilateral blindness immediately after operations. The above-mentioned unusual event seems hardly suitable to explain the pathophysiology of the monocular blindness in this case.

Ruben & Afshar illustrated a peculiar case with complete and permanent monocular blindness following SAH from the rupture of a small ACoA aneurysm. They suggested that ischemic damage to the anterior visual pathway secondary to post-SAH vasospasm played a role in the visual failure. Hara et al reported two cases of ischemic optic neuropathy following SAH. They attributed the optic disc atrophy with excavation and the permanent visual field defect in both cases to insufficient blood supply to the posterior part of the optic nerve which occurred as a complication of SAH, i.e. vasospasm. Blood supply to optic nerves depends on the thin arterial branches of internal carotid and anterior cerebral arteries. Those branches can be identified in microsurgical operation fields, but hardly visualized on angiograms. Therefore, it is impossible to prove vasospasm of those tiny arterial branches angiographically.

It is clinically difficult to determine the etiology and pathogenesis of the monocular blindness of this case. Based on the neurologic and ophthalmologic findings, such previously known causes as the surgical injury of the optic nerve, post-SAH ocular hemorrhages, the optic nerve injury by an aneurysm itself, or the orbital infarction syndrome can be ruled out. The authors think this case’s monocular total blindness was most probably caused by post-SAH ischemic posterior optic nerve injury.

**Conclusion**

Probable ischemic optic neuropathy should be taken into consideration in the differential diagnosis of post-SAH visual disturbances, especially following the rupture of the anterior communicating artery aneurysm.

**References**