

Ischemic Optic Neuropathy Associated with Subarachnoid Hemorrhage after Rupture of Anterior Communicating Artery Aneurysm

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Key Words

Anterior communicating artery aneurysm · Subarachnoid hemorrhage · Craniotomy · Ischemic optic neuropathy · Vasospasm

Abstract

Two clinical cases in which ischemic optic neuropathy (ION) occurred after subarachnoid hemorrhage (SAH) are reported. Hemorrhage in the proximity of the optic chiasm was confirmed in 2 cases following rupture of an anterior communicating artery aneurysm. Optic disk atrophy with excavation and permanent visual field defect (altitudinal superior hemianopia) occurred in both cases. ION seems to occur in association with the optic nerve coincidental with the hyperdensity side of SAH on head CT scan. The incidence of ION appears to be attributable to an insufficient blood supply to arteries distributed in the posterior part of the optic nerve as a result of SAH.

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Introduction

A case of total blindness following rupture of a small anterior communicating artery (AComA) aneurysm associated with a documented period of reduction in global cerebral blood flow has been reported [1]. There have been reports of cases in which ischemic retinal and optic disk edema occurred due to a ruptured anterior cerebral artery (ACA) aneurysm [2] and cases in which bilateral macular edema and unilateral optic disk atrophy occurred 10 days after ruptured AComA aneurysms [1]. Most current theories focus on the release of factors from the subarachnoid clot [3, 4].

Here are 2 cases with visual field defects and optic disk atrophy associated with subarachnoid hemorrhage (SAH) following a rupture of an AComA aneurysm. These are presumably due to ischemic optic neuropathy (ION).

Case Reports

Case 1

A 66-year-old woman became ill while shopping and lost consciousness on the way to the toilet on July 11, 1998. Although she recovered consciousness several minutes later, a severe, prolonged headache resulted in emergency hospitalization. She suffered from diabetes mellitus, hypertension and left hemiplegia due to a brain

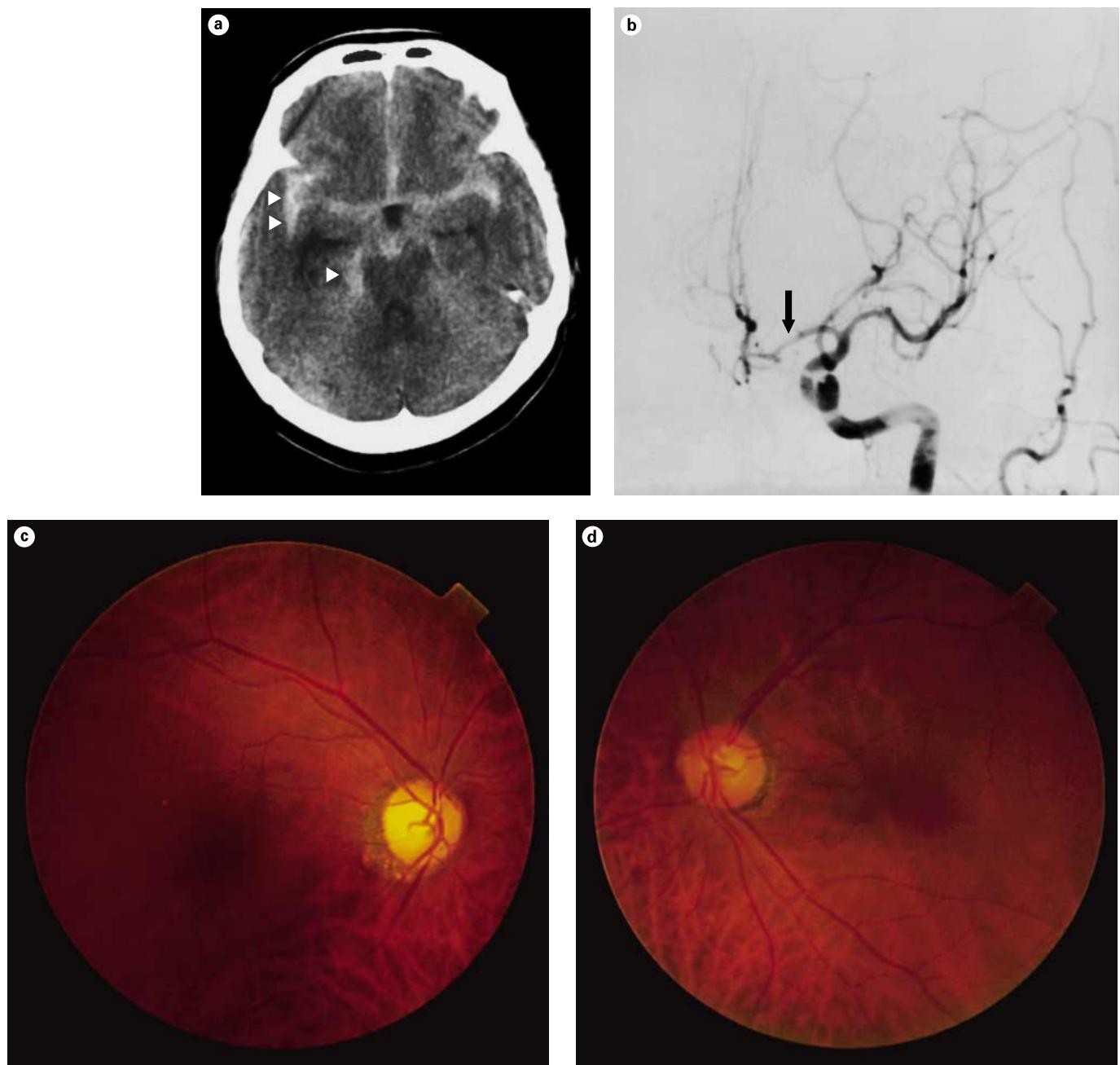
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infarction which had occurred 15 years previously. The neurological findings were as follows: severe headache, nuchal stiffness, nausea and vomiting, an absence of dyskinesia and disturbance of consciousness. GCS (Glasgow Coma Scale) and WFNS (World Federation of Neurological Surgeons Committee) evaluations were both grade I. Diffuse and severe hemorrhage on the right side of the interpeduncular cistern, interhemispheric fissure and sylvian fissure was observed on a CT scan (fig. 1a). A ruptured AComA aneurysm was confirmed by cerebral angiography.

Neurosurgery and Follow-Up. By the left pterional approach, clipping of the aneurysm was performed 2 days after SAH. Slight and diffuse hemorrhage was observed mostly around the sylvian fissure. The hematoma on the right side of the sylvian fissure remained 5 days after surgery on the CT scan. An obvious vasospasm was also detected by cerebral angiography on both right and left sides of the ACA 5 days after surgery (fig. 1b). Eighteen days after surgery, acute epidural edema developed, and hydrocephalus was not cured 2 weeks after surgery, resulting in a ventriculoperitoneal shunt.

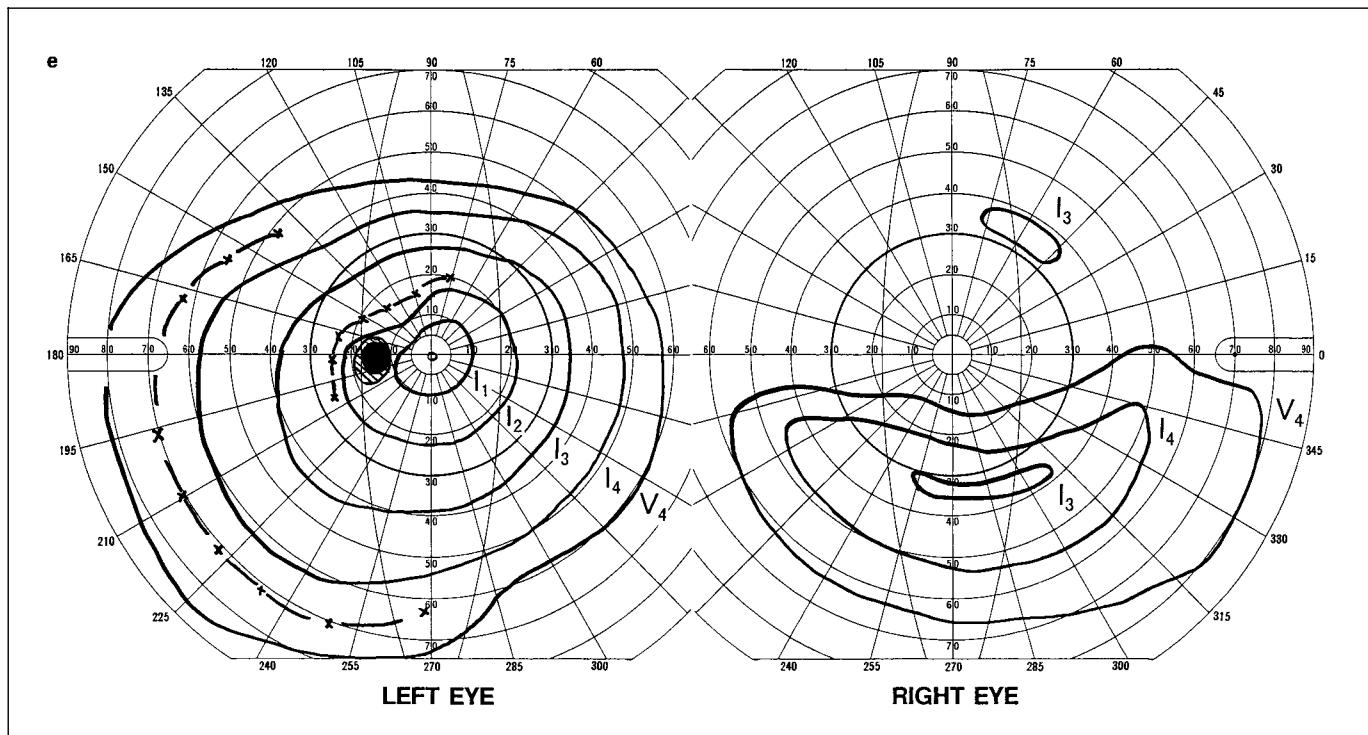


Fig. 1. Case 1. a An image of a head CT scan on the day of the incidence of SAH. Severe and diffuse hemorrhage is shown at the interpeduncular cistern, interhemispheric fissure and sylvian fissure, particularly on the right side (arrowheads). b A cerebral angiogram, obtained 5 days after surgery, shows the spastic appearance at the left A1 segment of the ACA (arrow). c, d Photos of the fundus 60 days after the incidence of SAH. Deep excavation of the optic disk and lamina cribrosa in the right eye can be observed. Optic nerve atrophy is clearly exhibited, with loss of the rim and typical tortuous vessels in and around the disk. Parapapillary chorioretinal atrophy and mild sclerotic changes in the retinal artery are shown in both eyes. e Kinetic visual fields 9 months after surgery. Altitudinal superior hemianopia in the right eye is demonstrated on the Goldman perimeter.

Neuroophthalmological Examination. Neuroophthalmological findings were examined 60 days after SAH. The patient complained of diminished visual acuity of the right eye 30 days after SAH. Visual acuities were 0.02 in the right eye and 1.0 in the left. A relative afferent pupillary defect was observed in the right eye. No abnormality of ocular motility was found in either eye. Optic disk atrophy with excavation was detected in the right eye (fig. 1c, d).

Follow-Up. Altitudinal superior hemianopia was present in the right eye 9 months after surgery (fig. 1e).

Case 2

A 63-year-old woman suffered from nausea and vomiting while working as a cleaner and consulted a medical practitioner on September 9, 1998. Because drug-induced poisoning was suspected, her stomach was irrigated at the clinic. Since disturbance of consciousness continued the next day, she consulted a neurological surgeon at Higashiyamato Hospital and was subsequently admitted. She had no diabetes mellitus or previous history of hypertension. Neurological symptoms were as follows: severe headache, nuchal stiffness, nausea and vomiting and dyskinesia were not observed. The GCS score was grade IV while that of the WFNS was grade II. Diffuse hemorrhage

on the left side of the sylvian fissure was observed on a CT scan (fig. 2a). A ruptured AComA aneurysm was confirmed by cerebral angiography.

Neurosurgery and Follow-Up. By the right pterional approach, clipping of the aneurysm was performed 2 days after SAH. Moderate hemorrhage was observed mostly around the left side of the sylvian fissure. Vasospasm was not noted 17 days after SAH by cerebral angiography.

Neuroophthalmological Examination. The patient became aware of horizontal diplopia 20 days after SAH. Visual acuities were 1.2 in the right eye and 0.9 in the left. The pupils measured 4 mm in the right eye and 5 mm in the left eye, and reaction to light was sluggish in the left eye. A relative afferent pupillary defect was observed in the left eye. Left oculomotor paralysis with limited adduction was found. Slight atrophy of the optic nerve head with excavation was detected in the left eye (fig. 2b, c). Altitudinal superior hemianopia in the left eye and peripheral constriction in the right eye were present (fig. 2d).

Follow-Up. Altitudinal superior hemianopia existed in the left eye, but peripheral constriction in the right eye disappeared 10 months after surgery (fig. 2e).

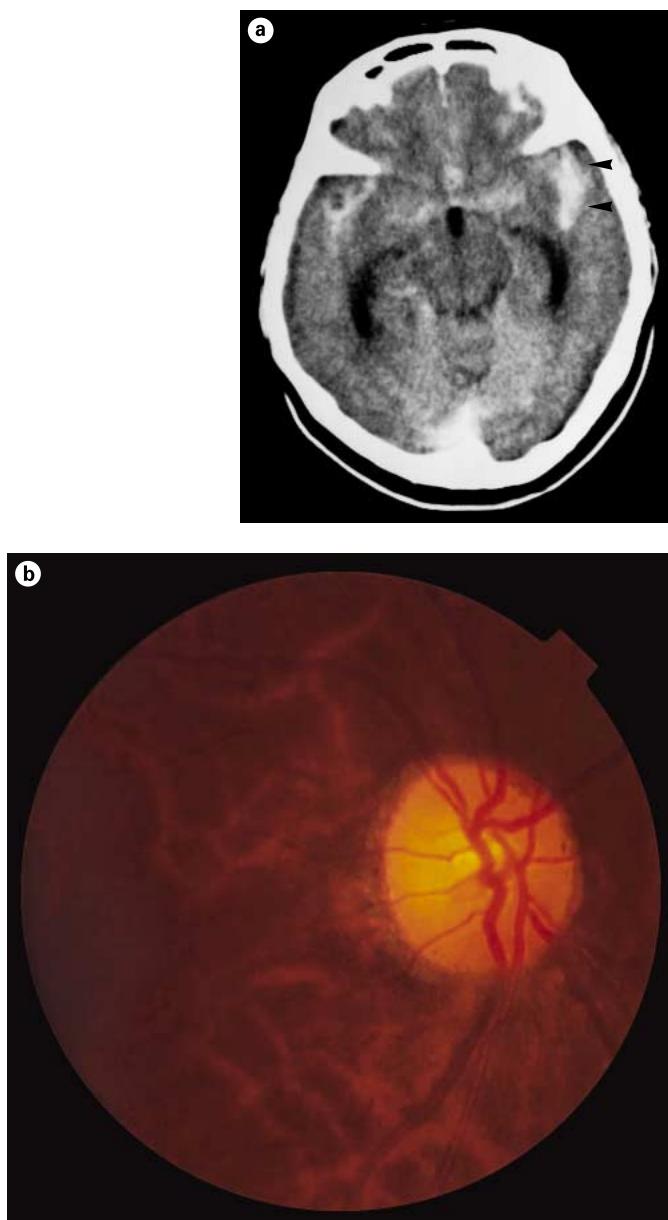


Fig. 2. Case 2. a An image of a head CT scan on the day of the incidence of SAH. Diffuse hemorrhage is shown on the left side of the sylvian fissure (arrowheads). b, c Photos of the fundus 20 days after the incidence of SAH. Temporal pallor of the optic disk appears in the left eye. Deep excavation of the optic disk, loss of the rim and lamina cribrosa can also be observed. d Automated visual fields 20 days after the incidence of SAH. Altitudinal superior hemianopia and inferior peripheral constriction in the left eye and peripheral constriction except on the inferotemporal side in the right eye are exhibited on the Humphrey perimeter (strategy: 30-2). e Automated visual fields 10 months after surgery. Altitudinal superior hemianopia in the left eye remains, but peripheral constriction has disappeared in the right eye on the Humphrey perimeter (strategy: 30-2).

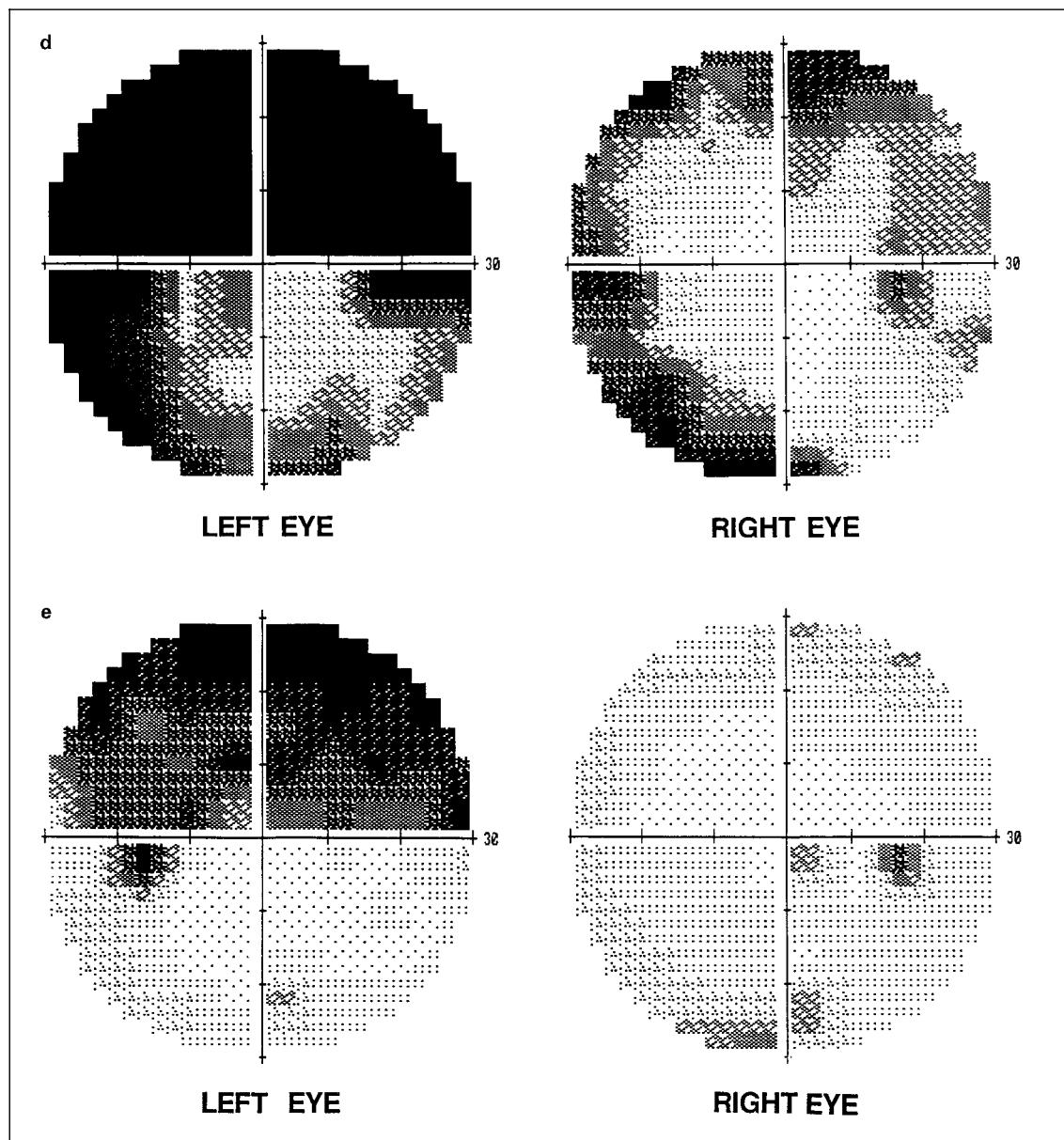
Discussion

The occurrence of optic disk edema at the initial phase, i.e. a period of 4–6 weeks from the onset, is one of the characteristic symptoms of anterior ION. Posterior ION, however, is characterized by a normal optic disk without such edema and none of the fundus abnormalities at the onset, and by the development of optic atrophy 5–6 weeks later. After 2–3 months, both anterior ION and posterior ION result in optic atrophy with optic-nerve-related vi-

sual field defects [5]. Neuroophthalmological examinations in case 1 and case 2 were carried out 60 and 20 days after SAH, respectively.

These times seem to exceed the initial phase when optic edema must have occurred as a characteristic episode. Therefore, optic nerve atrophy cannot be conclusively attributed to posterior ION in either case.

The intensity of SAH is an important risk factor for cerebral vasospasms, particularly when a thick hematoma is located in the subarachnoid space. This suggests that



ischemia at the optic chiasm and optic nerve is affected by cerebral vasospasms after SAH. The occurrence of cerebral vasospasms may be time dependent. Vasospasms were most frequently seen angiographically after 4–17 days [3, 4, 6–9]; for the AComA aneurysm: 23 out of 45 cases (51.1%), in cases of internal carotid artery (ICA) aneurysms 11 out of 25 cases (44%) [8, 9]. In our case 1, an obvious vasospasm was also detected by cerebral angiography at the ACA 5 days after surgery, but in case 2 no vasospasm was noted 17 days after SAH. Based on these

findings, the presence or absence of vasospasm may be dependent upon the time difference, 5 days after surgery being coincident with the day of the increasing incidence of angiographic vasospasm and 17 days after surgery being equivalent to the day of decreasing incidence [3, 4, 6–9]. Although angiographic evidence fails to show any changes in the majority of arteriole vasospasms around the posterior part of the optic nerve and optic chiasm in case 2, we cannot exclude the possibility of arteriole vasospasms.

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