



Case report

Endovascular treatment of an unruptured anterior communicating artery aneurysm presenting with acute altitudinal visual field defect: A case report

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Abstract

Purpose: To describe a case of endovascular occlusion of an unruptured anterior communicating artery aneurysm presenting with acute altitudinal visual field defect is presented here.

Methods: A 52-year-old man was evaluated and treated for altitudinal visual field defect in the right eye.

Results: In ophthalmic and neuro-imaging, an accompanying anterior communication artery aneurysm was detected as a cause of visual field defect. He underwent endovascular procedure, yielding excellent outcome as full recovery of visual field defect was observed one month following the procedure and sustained when followed at month 24.

Conclusions: Visual dysfunction is a rare presentation of unruptured anterior communication artery aneurysm. Endovascular procedure may be a safe treatment in these cases.

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Keywords: Intracranial aneurysm; Anterior communicating artery; Visual field; Endovascular procedure

Introduction

Subarachnoid hemorrhage (SAH) is often the most common presentation for intracranial aneurysms.¹ However, visual dysfunction can also occur as a result of anterior as well as posterior pathways compression. The former includes optic nerve and chiasma, whereas latter encompass optic tract and optic radiation.^{1,2}

The aneurysms originating from carotid-ophthalmic artery, supraclinoid internal carotid artery, and internal carotid artery bifurcation are more likely to present with symptoms of anterior visual pathway compression.³

We report a case of altitudinal visual field defect caused by a large unruptured anterior communicating artery (AcomA) aneurysm treated safely through an endovascular procedure despite prevailing popularity of surgical intervention as the preferred treatment for such cases.

Case report

A 52-year-old man presented and was admitted with blurred vision in the right eye as well as vague pain while moving in the same eye. Baseline ophthalmologic examinations were normal except for positive relative afferent pupillary defect (RAPD) of the right eye. Best corrected visual acuity (BCVA) was 20/20 in both eyes. Medical history was unremarkable except for hypertension.

Inferior altitudinal visual field defect was noted in the static field test (Humphrey visual field test) (Fig. 1-top left and top right). Visual evoked potential (VEP) revealed decreased amplitude in N75-P100 wave. Brain computerized

Conflict of interest: None.

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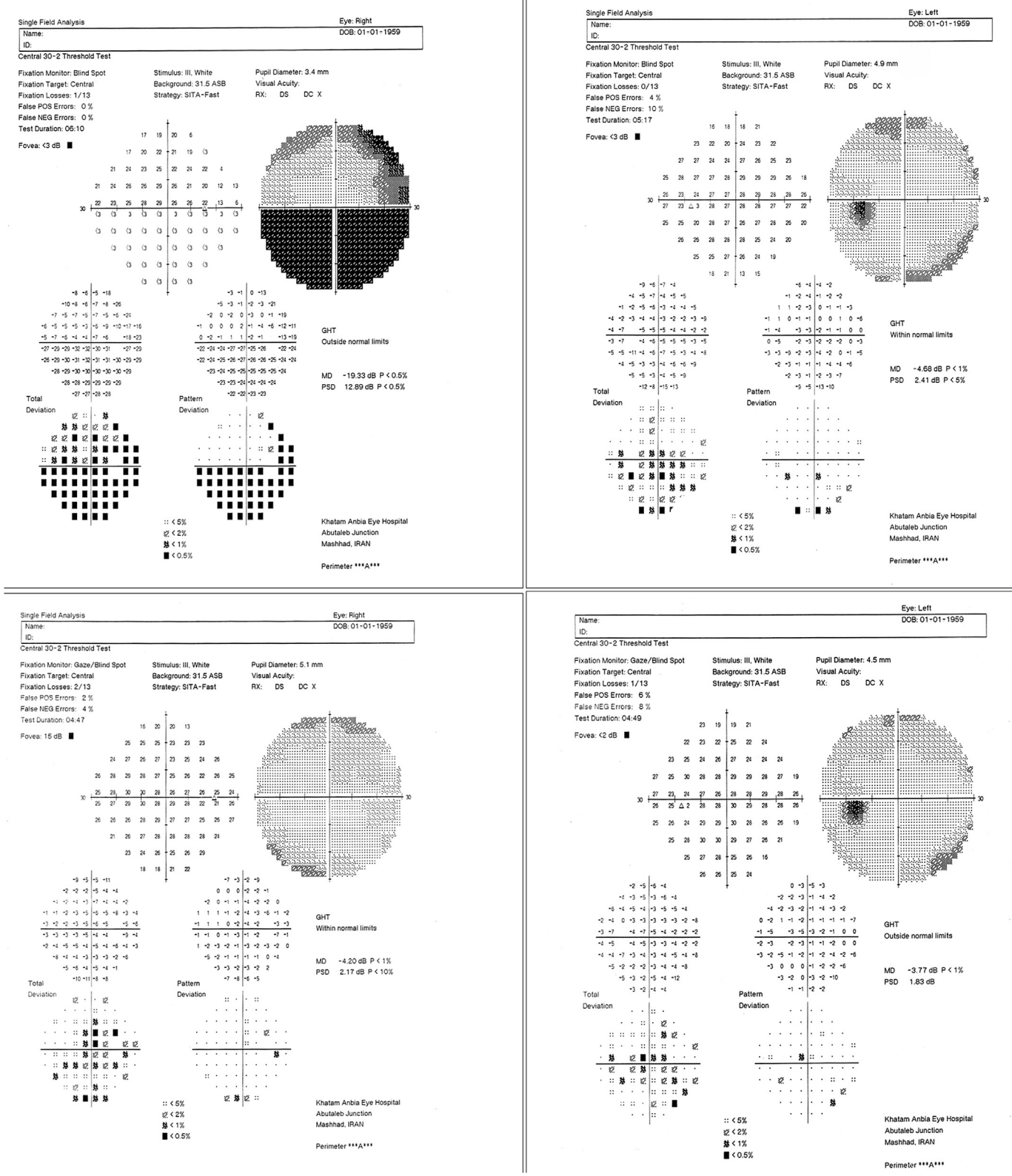


Fig. 1. Top left: Preoperative static visual field test showed inferior altitudinal visual field defect of the right eye. Top right: Preoperative normal left visual field. Bottom left: Postoperative static visual field test showed complete disappearance of visual field defect of the right eye. Bottom right: Normal left visual field.

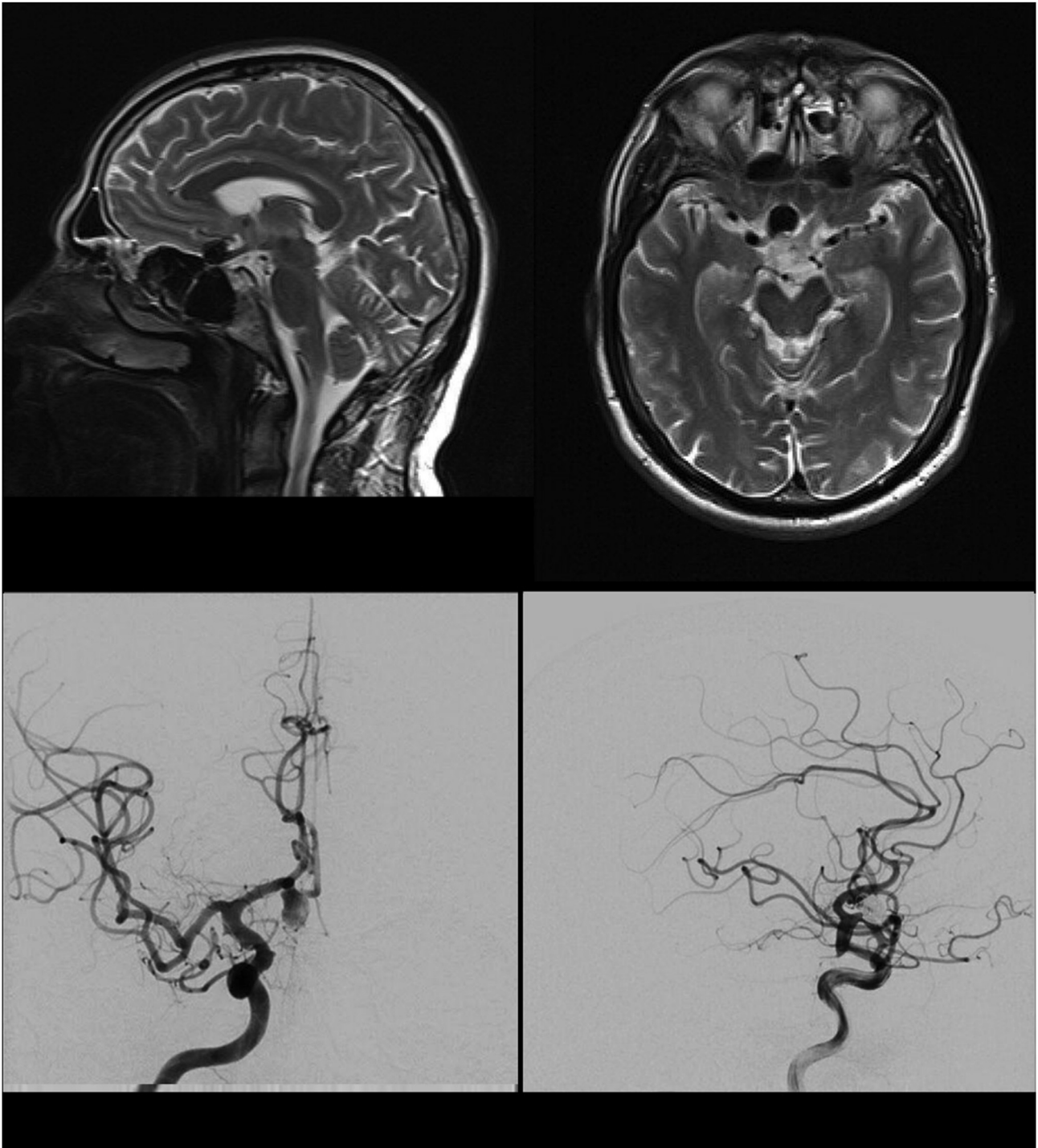


Fig. 2. Top left: Preoperative axial T2-Weighted magnetic resonance imaging (MRI). Top right: Sagittal T2-Weighted MRI revealing a heterogeneous flow void in the suprasellar region. Bottom left: Preoperative brain angiography demonstrated an anterior communicating artery aneurysm. Bottom right: Postoperative brain angiography showed occluded aneurysm after one year.

tomography (CT) and CT angiography (CTA) demonstrated a heterogeneous lesion in anterior fossa. Brain magnetic resonance imaging (MRI) showed a partially thrombosed large aneurysm with greater than 10 mm in diameter (Fig. 2-top left and top right). Digital subtraction angiography (DSA)

indicated a partially thrombosed large unruptured AcomA aneurysm with projection toward right optic nerve (Fig. 2-left bottom).

Endovascular coiling resulted in complete obliteration of the aneurysm with visual defect disappearing in the static field

test one month after (Fig. 1-left bottom and right bottom). The patient remained problem-free in the same tests at month 12 of the follow-up.

Discussion

AcomA aneurysm rarely presents with symptoms of compression of anterior visual pathways.⁴ Visual impairment can follow intracranial aneurysm owing to increased pressure on anterior optic pathway in proximity with circle of Willis, include unilateral scotoma or blindness, bitemporal hemianopsia and homonymous hemianopsia, all occurring as a result of the compression of the optic nerve, chiasma, and tract, respectively. It is worth noting that these symptoms often progress gradually.⁵

A few cases with visual deficits such as monocular blindness have been shown following ruptured AcomA aneurysms. Chiasma compression was the main cause of visual field deficits. Isolated involvement of one optic nerve due to unruptured AcomA aneurysm resulting in monocular blindness has not been reported yet.⁶

Altitudinal field defects are seen commonly in carotid-ophthalmic aneurysm.⁷ In this particular case being reported here, visual field defect seems to have been caused by the pressure exerted from aneurysm on superior portion of the right optic nerve.

Surgery is widely accepted as the standard mode of therapy in unruptured cases. Nevertheless, endovascular treatment prevailing as an alternative in incidental cases of aneurysms, or those appearing as mass effect, has also grown popular for ruptured aneurysms, particularly owing to its safety.^{8,9}

Coil occlusion hypothetically functions in two ways: (A) It alters the viscoelastic properties off the lesion partially, blocking the energy transfer from the pulsatile vessels to adjacent neuronal elements along with reducing tissue edema, (B) Owing to the reorganization and the ultimate resolution of the thrombosis, the aneurysm shrinks in size following the procedure.⁸

Date et al. reported 17 cases of unruptured aneurysm with visual symptoms before surgery, and two aneurysms were located in the AcomA with chiasma compression. There has been no impairment after clipping the aneurysms.¹

Shukla et al. reported a 65-year-old man, who had complete loss of vision in right eye and temporal hemianopsia in left eye due to giant AcomA aneurysm and who did not have any other neurological deficits. The patient was offered surgery for securing aneurysm. As there was uncertainty about visual recovery, he did not opt for any treatment and was discharged against medical advice.⁴

Park et al. reported in their study that out of 10 patients who had visual symptoms, 8 showed improvement in visual symptoms within 6 months after clipping of aneurysms.⁵

Nishino et al. reported improvement in visual field defect following the clipping of AcomA aneurysm.² Likewise, Oliveria et al. has 15 patients with visual field defect accompanying internal carotid artery aneurysms, five of whom

underwent aneurysm clipping procedure due to the involvement of AcomA.⁸

Similar result of surgery was achieved following coiling in 18 patients with cranial nerve symptoms, three of whom presented with optic nerve dysfunction with underlying unruptured aneurysm.¹⁰

A case with sudden altitudinal visual field loss and normal fundus resulting from ruptured aneurysm of the AcomA was reported by Chan et al. Vasospasm was considered the cause of posterior ischemic optic neuropathy.¹¹

In another study, a 40-year-old man was reported with the abrupt onset of left homonymous hemianopsia, right visual acuity disturbance, and slight headache. They clipped the neck of the aneurysm and resected its dome. Although the aneurysm was successfully clipped, the visual disturbance persisted after surgery, suggesting that the damage to the visual pathways by aneurysm penetration was irreversible in this case.¹²

Horiuchi et al. reported three patients who underwent clipping surgery for the AcomA aneurysm infiltrating into the optic nerve or chiasm. They propose that the unruptured AcomA aneurysm protruding inferiorly should be evaluated carefully to exclude a penetration of the optic nerve and chiasm by preoperative MRI.¹³

Given the above mentioned, it can be safely concluded that coiling can be performed as a safe option in cases of optic nerve involvement in the case of unruptured AcomA aneurysm. Nevertheless further studies seem warranted in corroborating this choice of treatment.

In conclusion, visual dysfunction is a rare presentation of unruptured AcomA aneurysm; however, it is considered a potential cause of visual disturbance. Intracranial vessels studies such as MRI, MRA, or CTA are recommended in patients with visual field defect who lack overt pathology in ophthalmologic examinations. Endovascular procedure can be a safe therapeutic modality in patients with unruptured AcomA aneurysms.

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