



Case Report

Posterior ischemic optic neuropathy with acute monocular vision loss following clipping of anterior communicating artery aneurysm. A case report and review of literature

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Received : 04 June 2021

Accepted : 18 July 2021

Published : 20 September 2021

DOI

10.25259/SNI_551_2021

Quick Response Code:



ABSTRACT

Background: The acute postoperative monocular vision loss following anterior communicating artery aneurysm clipping secondary to posterior ischemic optic neuropathy (PION) a rare presentation.

Case Description: A 32-year old patient presented with a spontaneous holocranial thunderclap headache for 7 days, associated with vomiting. The SAH was diagnosed with a tiny saccular aneurysm arising from the anterior communicating artery. A left pterional craniotomy and clipping of aneurysm were done. On the 3rd postoperative day, he complained of left-sided complete blindness, and on the 5th postoperative day, his GCS dropped to E4V1M5 with right-sided hemiplegia. MRI brain showed normal optic apparatus with bilateral ACA and left MCA territory infarct.

Conclusion: The PION must be kept in the differential diagnosis of post-clipping sudden visual deterioration, especially following anterior communicating artery aneurysm rupture.

Keywords: Acomm aneurysm, Monocular blindness, Optic nerve, Posterior ischemic optic neuropathy

INTRODUCTION

Vision loss in aneurysmal SAH (aSAH) is primarily because of intraocular hemorrhage, direct compression of the optic pathway by the sac of the aneurysm, or iatrogenic trauma optic nerve chiasm during the dissection of clipping of the neck and rarely optic neuropathy following aSAH.

CASE REPORT

A 32-year-old hypertensive man with a history of alcohol intake for 8 years and was abstaining for 1 year; presented with a sudden holocranial thunderclap-like headache 7 days ago. It was associated with 3–4 episodes of projectile vomiting for 2–3 days without any relief in headache intensity. Neurological examination revealed no deficit. Non-contrast CT scan brain showed SAH in the

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interhemispheric fissure and bilateral Sylvian fissures (modified Fischer grade-2) with visible temporal horns suggestive of hydrocephalous [Figure 1]. CT angiography brain [Figure 2] was suggestive of a 3 × 4 mm saccular aneurysm arising from the anterior communicating artery without surrounding hematoma or active bleed. With indocyanine green video angiography (ICGVA), left pterional craniotomy and clipping of aneurysm and fenestration of lamina terminalis were performed. The aneurysm was arising from the right A1 A2 junction directing superiorly and posteriorly, and intraoperative ICGVA showed completeness of sac obliteration and good distal flow, which was also confirmed on postoperative CT angiography. The patient was asymptomatic for 3 days after surgery, complained of inability to see from the left eye and became aphasic, developed right-sided hemiplegia on 5th postoperative day. He had dilated left pupil with an ipsilateral afferent pupillary defect (APFD). MRI Brain [Figure 3] revealed bilateral ICA and left MCA territory infarct, and The fundus examination was normal on post-op day-3 [Figure 4a], and fundus showed attenuated vessels with mild pallor on the 15th day postoperative [Figure 4b]. The decompressive craniotomy was done because of developing infarct and the patient showed improvement in neurological condition but remain aphasic and the left eye APFD persisted.

DISCUSSION

After reviewing the available literature, there are only few case reports of postoperative monocular vision loss in anterior communicating artery aneurysm,^[1-7] but only one

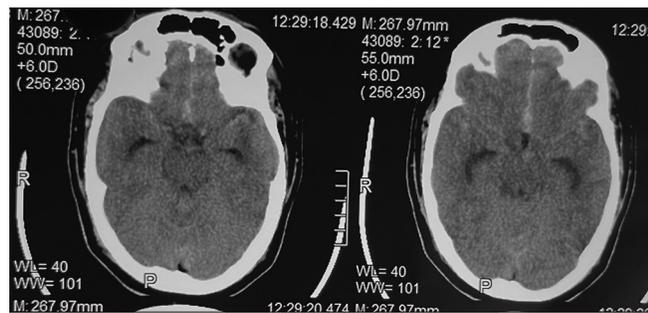


Figure 1: NCCT brain showing SAH in the interhemispheric fissure and bilateral sylvian fissure.

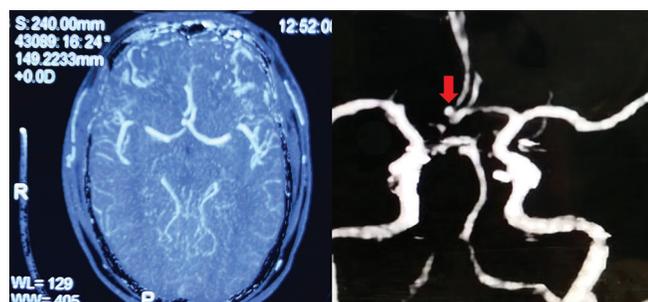


Figure 2: Preoperative CT angiography was showing an anterior communicating artery aneurysm.

case report of ischemic optic neuropathy causing monocular vision loss.^[2] Chung *et al.* proposed saline inflow into the orbit due to iatrogenic injury to the orbital roof leading to the acute compartmental syndrome.^[1] The orbital infarction syndrome has also been reported after the surgery for intracranial aneurysms leading to vision loss.^[3,4,7] Park *et al.* and Noh *et al.*^[4,5] reported proptosis, ophthalmoplegia, and unilateral vision loss after clipping aneurysm, possibly because of direct compression of the eye by the surgical flap leading to vascular insult secondary to hypotension. We can even rule out this particular cause of monocular vision loss. The normal optic apparatus in postoperative MRI rules out the compartment syndrome in our case.

Ruben and Afshar reported a case of small Acomm. Artery aneurysm leading to complete and permanent monocular vision loss.^[6] They suggested the post-SAH vasospasm causing ischemic injury to the anterior visual pathway as a cause of visual loss. Similarly, the ischemic optic neuropathy following SAH was also reported by Hara *et al.*^[2] in their two cases. They hypothesized that the posterior optic nerve's insufficient perfusion following SAH due to vasospasm caused the optic nerve atrophy with excavation and permanent visual field defect in both cases. The blood supply to posterior optic nerve relies on the pial vascular plexus formed thin perforators of the ophthalmic artery, peri-papillary choroidal, Circle of zinn

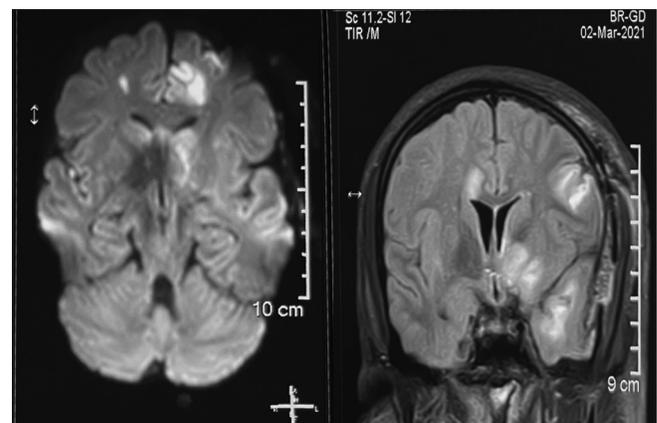


Figure 3: MRI brain showing multiple focal infarcts.

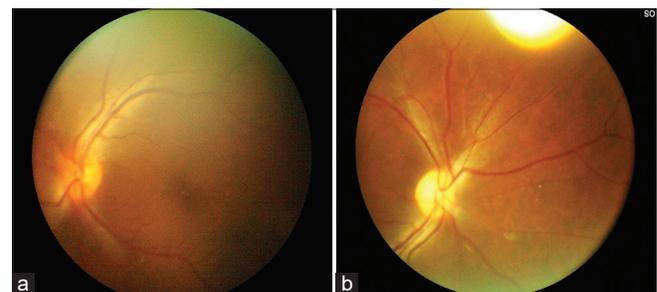


Figure 4: (a) Fundus on postoperative day 3: Normal vessels, disc margins (b) well-defined disc with attenuation of blood vessels fundus picture on 15th postoperative day.

Authors	No of patients	Vision loss mono/ binocular	Aneurysm location	Proposed cause
Chung <i>et al.</i> ^[1] (1999)	Case report	Monocular	Anterior communicating artery.	Acute orbital compartment syndrome secondary to saline inflow from iatrogenic orbital roof injury
Noh <i>et al.</i> ^[4] (2003)	5 cases	Binocular	Anterior communicating artery	Orbital Infarction Syndrome Caused by Compression of Surgical Scalp Flap
Park <i>et al.</i> ^[5] (1987)	Case report	Binocular	Anterior communicating artery	Orbital Infarction Syndrome Caused by Compression of Surgical Scalp Flap
Ruben and Afshar ^[6] (1991)	Case report	Monocular	Anterior communicating artery	post-SAH vasospasm causing ischemic damage to the anterior visual pathway as a cause of visual loss
Zimmerman <i>et al.</i> ^[7] (1995)	4 cases	Monocular	Anterior communicating artery	Orbital infarction syndrome due to hypoperfusion of the ophthalmic artery
Hara <i>et al.</i> ^[2] (2003)	2 cases	Monocular	Anterior communicating artery	Ischemic optic neuropathy post-SAH.
Our case	Case report	Monocular	Anterior communicating artery	Posterior Ischemic optic neuropathy secondary to vasospasm

and Haler arteries, other orbital arteries, superior hypophyseal arteries, anterior communicating, and anterior cerebral arteries. The perforators are well visualized intraoperatively over the optic nerve but are very difficult to visualize on angiograms. Hence, it is almost impossible to document the vasospasm of these thin perforators on angiography. The etiology and pathogenesis of monocular complete vision loss are difficult to ascertain in this case. Based on the clinic-radiological and intraoperative findings, we can easily rule out the earlier reported known causes such as iatrogenic injury to the optic nerve, intraocular hemorrhage post-SAH, and the optic nerve injury by aneurysm itself, or the orbital infarction syndrome. We report this particular case of ipsilateral acute monocular blindness as a sequela of posterior ischemic neuropathy of the posterior part of the optic nerve due to the vasospasm of perforators forming the crucial pial vascular plexus following SAH.

CONCLUSION

The possibility of posterior ischemic optic neuropathy must be kept in the differential diagnosis of post-SAH sudden visual deterioration, especially following anterior communicating artery aneurysm rupture.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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How to cite this article: Sharma AK, Dholakia B, Jagetia A, Singhal GD, Bodeliwala S, Srivastava AK, *et al.* Posterior ischemic optic neuropathy with acute monocular vision loss following clipping of anterior communicating artery aneurysm. A case report and review of literature. *Surg Neurol Int* 2021;12:471.