Neurology Asia 2025; 30(1) : 353 – 358

IMAGING HIGHLIGHTS

How common are unruptured intracranial aneurysms misdiagnosed as a serious ocular disease? – An illustrative case

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Unruptured intracranial aneurysm rarely present with visual dysfunction and could be misdiagnosed as serious ocular disease when there is associated vision abnormality. Anterior communicating artery (ACOM) aneurysm is a rare cause of non-glaucomatous optic neuropathy and could cause anterior visual pathway compression due to its proximity to optic nerve.[1] We hereby present a case of co-existing ACOM aneurysm and meningioma with unilateral fully cupped disc mimicking glaucomatous optic neuropathy, with literature review of intracranial aneurysms that had been misdiagnosed as ocular pathology.

Keywords: Intracranial aneurysms, anterior communicating artery aneurysm, vision dysfunction, glaucoma, optic neuritis

CASE REPORT

A 75-year-old female was referred to our centre for right eye (RE) advanced glaucoma. She presented with RE progressive painless blurring of vision for the past few years. There was no headache or neurological symptoms such as slurred speech, gait imbalance or tremors. She denied family history of glaucoma, or previous ocular trauma and was not myopic. The vision was light perception and 6/9 on the right and left eyes (LE) respectively. Right relative pupillary afferent defect was positive. On primary gaze, there was RE exotropia of 15 degrees. The anterior segment examination was unremarkable. The intraocular pressure (IOP) was 26mmHg and 19mmHg over right and left eyes, respectively. Shaffer's gonioscopy revealed open angles bilaterally with no hyperpigmentation seen. Fundus examination showed right pale fully-cupped disc while the left disc was pink with cup-to-disc ratio 0.5 (Figure 1). Optical coherence tomography (OCT) showed generalized thinning of the right retinal nerve fibre whereas the left was of normal thickness. Humphrey visual field was unable to be performed over the RE due to poor vision. The LE visual field was normal.

Neuroimaging was warranted in view of asymmetrical disc cupping. Contrast-enhanced computed tomography (CECT) brain showed co-existence of right ACOM saccular aneurysm measuring 0.6x0.6cm with right tentorium cerebelli meningioma measuring 3.4x4.6x4.0cm, with local mass effect and perilesional oedema (Figure 2). Cerebral angiogram and magnetic resonance imaging of the brain confirmed the finding of ACOM aneurysm with a nipple measuring 0.6x0.7x0.7cm with neck of 0.2cm. The haematological and coagulation profiles were normal. Neurological examination revealed dysdiadokokinesia and past-pointing test positive on the right side. Other cranial nerves examinations were normal, with full power over bilateral upper and lower limbs.

She was referred to neurosurgical team for co-management and was offered the option of clipping of the aneurysm with excision of meningioma versus conservative management. She opted for conservative management. Gutt Simbrinza and Ganfort was continued for neuroprotective effect. Her IOP during subsequent follow-up ranged between 14-17mmHg in both eyes. The ACOM aneurysm and meningioma size remained static at one year of surveillance.

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Date of Submission: 7 September 2024; Date of Acceptance: 26 December 2024

https://doi.org/10.54029/2025rrj

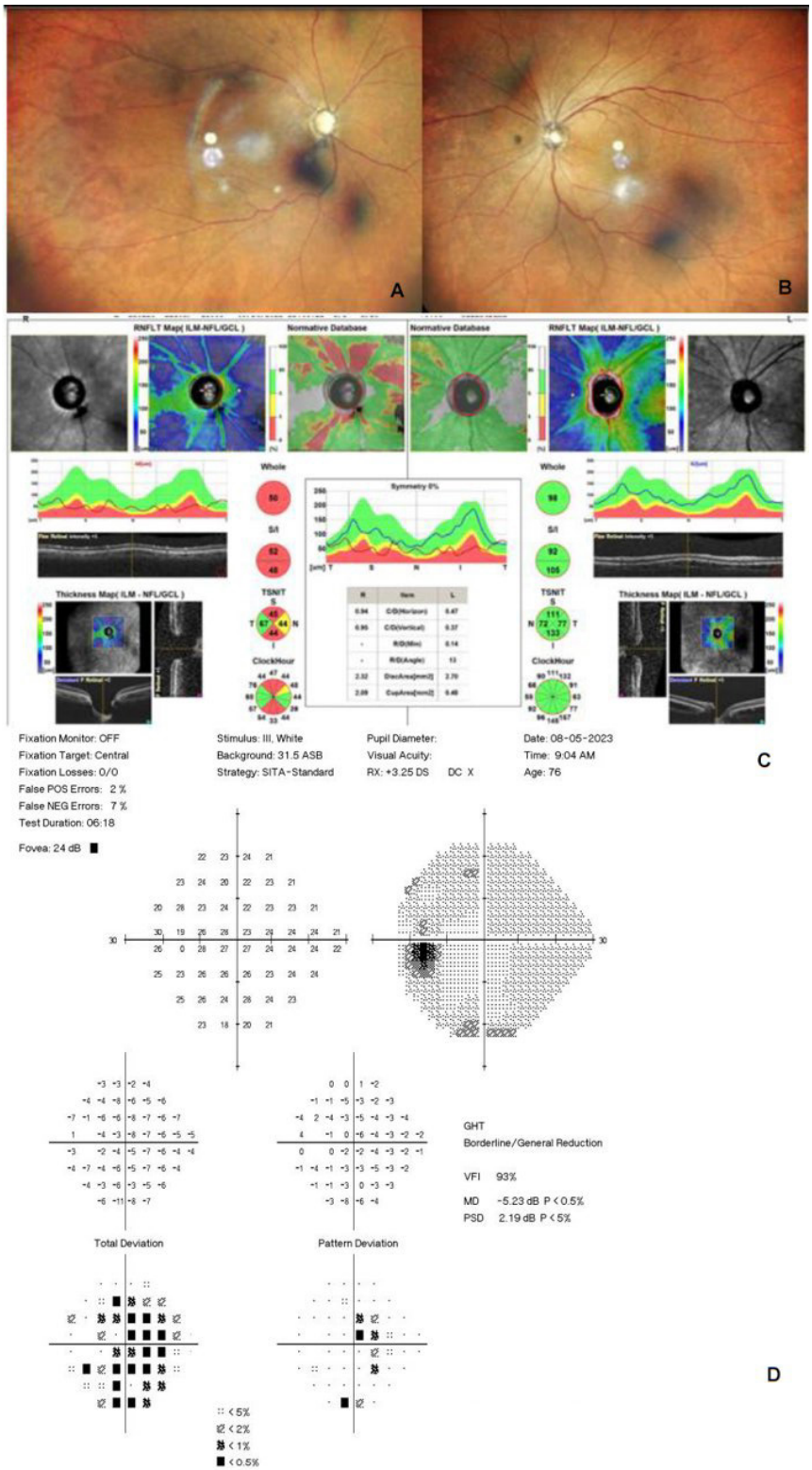

Figure 1. Colour fundus photography of (A) right eye showing pale cupped optic disc, and (B) left eye showing pink optic disc with cup-to-disc ratio 0.5. Optical coherence tomography (OCT) of retinal nerve fibre layer (C) showing right eye generalized retinal nerve fibre layer thinning and left eye normal thickness. Humphrey visual field (D) of left eye showing no obvious field defect

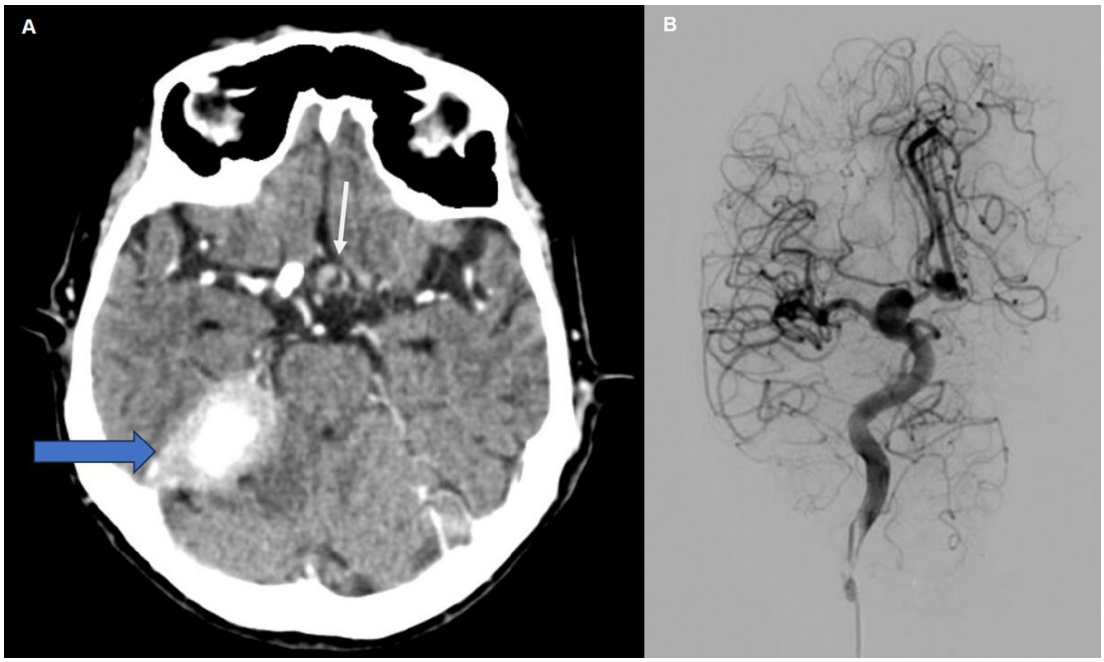

Figure 2. CECT brain imaging (A) showing ACOM saccular aneurysm (white arrow) with coexistence of right tentorial cerebelli meningioma (blue arrow) with local mass effect and surrounding perilesional edema. Cerebral angiogram (B) demonstrates right ACOM aneurysm with nipple measuring 0.6x0.7x0.7cm with neck of 0.2cm

DISCUSSION

ACOM aneurysms tend to rupture while they are small, hence they usually present with subarachnoid haemorrhage rather than visual symptoms. Therefore, it is rare to observe visual symptoms caused by unruptured ACOM aneurysm.[1] Park *et al*. demonstrated that only 3% of patients who had ruptured ACOM aneurysm presented with visual symptoms, and majority had bilateral eye involvement due to proximity to the optic chiasm.[1]

Our literature review included 13 unruptured intracranial arterial aneurysm cases with vision dysfunction (Table 1). The first three cases were unruptured ACOM aneurysm presenting as rapid monocular blindness.[2-4] Two of the cases were misdiagnosed as retrobulbar optic neuritis. Our case is unique as our patient had progressive deterioration of vision over years mimicking a glaucomatous disc cupping, instead of a rapid deterioration. Visual symptoms caused by unruptured ACOM aneurysm could be due to compression of the optic tract, resulting in neuronal dysfunction, and/or poor blood circulation due to vasospasm of the blood circulation to the optic nerve leading to monocular blindness.[1] In our case, the small aneurysm could have caused a slow progressive optic nerve compression mimicking glaucomatous optic neuropathy. The chronicity of the ACOM aneurysm in our case may have compromised the blood supply to the optic nerve as evidenced by the right optic disc pallor, leading to monocular blindness. Compressive optic neuropathy typically causes generalized vision loss with optic disc pallor. This contrasts with glaucoma where the field loss typically starts in the periphery before affecting central vision as it spares the papillomacular bundle[5]. The vision loss out of proportion to optic disc cupping and pallor in our patient fits the picture of compressive optic neuropathy.

We also reviewed four cases of co-existing unruptured ACOM aneurysm with meningioma that presented with reduced vision.[6,7,11,12] The meningiomas were located at suprasellar region compressing optic tract resulting in bilateral eye visual deterioration. However, our patient's meningioma was located at the tentorium cerebelli. Therefore, vision loss in our patient was more likely to be caused by ACOM aneurysm rather than meningioma. The incidence of co-existing intracranial aneurysms and meningioma is only 1.1%.[8] It is postulated that growing meningioma increases intracranial pressure, leading to rise in arterial blood pressure to regulate cerebral perfusion. This stresses the cerebral arteries and

Table 1: Literature review of unruptured intracranial aneurysms presenting with visual disturbance

Author, year	Age/ gender	Type of aneurysm	Co-existence of intracranial mass	Vision‡	RAPD‡	IOP‡ (mmHg)	Disc cupping on presentation‡	Visual field defect‡	Initial diagnosis‡
Craenen, 2004[2]	37/ Female	Unruptured anterior communicating aneurysm	No	RE 6/6; LE NPL	NR	NR	BE symmetrical disc	NR	LE retrobulbar optic neuritis
Bhat, 2011[3]	22/ Female	Unruptured anterior communicating aneurysm	No	LE 1/60	NR	NR	NR	LE temporal inferior quadrantanopia	LE retrobulbar optic neuritis
Shukla, 2013[4]	65/ Male	Unruptured anterior communicating aneurysm	No	RE NPL; LE 6/6	RE positive	NR	RE pale disc; LE normal disc	RE unable to perform; LE temporal hemianopsia	NR
Dolenc, 1998[11]	50/ Male	Unruptured anterior communicating aneurysm	Meningioma at tuberculum sellae	RE loss of vision	NR	NR	NR	NR	NR
Javadpour, 2004[12]	61/ Female	Unruptured anterior communicating aneurysm	Suprasellar meningioma	RE 6/12; LE HM	NR	NR	LE pale disc	RE temporal field defect; LE total field defect	NR
Chen, 2015[6]	65/ Female	Unruptured anterior communicating aneurysm	Meningioma at tuberculum sellae	BE loss of vision	NR	NR	NR	RE temporal hemianopsia; LE blindness	NR
Yildirim, 2015[7]	72/ Female	Unruptured anterior communicating aneurysm	Meningioma at tuberculum sellae	BE loss of vision	NR	NR	NR	NR	NR
Portney, 1977[13]	51/ Female	Berry aneurysm arising from the junction of the left ophthalmic and ICA†	No	RE 6/6; LE 6/15	LE positive	BE 18	RE normal; LE temporal pallor and a vertically oval cup elongated superiorly	RE normal field; LE inferior hemianopsia	LE ischemic optic neuropathy
Kulkarni, 2000[14]	41/ Female	Left carotid-ophthalmic artery aneurysm	No	RE 6/5; LE 6/4	LE positive	RE 18-24; LE 21-26	RE 0.3, LE 0.6	RE normal field; LE superonasal arcuate defect	LE secondary glaucoma
Beatty, 2001[10]	70/ Female	Ophthalmic segment aneurysm from ICA†	No	RE 6/12; LE 6/12	LE positive	RE 18; LE 19	RE normal; LE enlarged deep cup-to-disc ratio, loss of neuroretinal rim	RE normal field; LE supratemporal defect	NR

Nucci, 2016[15]	41/ Male	Ophthalmic segment aneurysm from ICA†	No	RE 6/9; LE 6/7.5	No RAPD	RE 16; LE 17	RE 0.5 pink; LE 0.8 with inferotemporal neuronal rim loss and pallor	RE normal field; LE scotoma in the superonasal quadrant	BE normotensive glaucoma
Netto, 2018[16]	48/ Female	Right ICA† aneurysm near emergence of ophthalmic artery	No	RE 6/6; LE 6/6	No RAPD	BE 12	RE 0.5, LE 0.7	RE normal field; LE temporal superior loss of sensitivity	BE normotensive glaucoma
Ashok, 2024[9]	72/ Female	Left carotid-ophthalmic artery aneurysm	No	RE 6/6; LE 6/18	LE positive	RE 06; LE 08	RE 0.6; LE 0.75	RE normal field; LE progressive temporal and paracentral loss	LE normotensive glaucoma

†ICA: internal carotid artery;
‡RE: right eye; LE: left eye; BE: bilateral eye; NR: not reported

result in arterial wall degeneration leading to aneurysm formation. Another possible explanation is that aneurysm formation could result from tumor adhesion to the arterial adventitia causing arterial wall damage.[8] Other hypothesis are hormonal factor such as estrogen influencing aneurysm formation, and genetic factors.[8]

The last six cases in our literature review described presence of intracranial aneurysms mimicking glaucomatous disc cupping. All these cases were secondary to internal carotid-ophthalmic artery aneurysm[9,10,13-16], but none had intracranial mass. Three were diagnosed with normotensive glaucoma, and one with secondary glaucoma initially presenting with unilateral anterior uveitis.[9,15,16] ACOM aneurysm mimicking glaucomatous disc cupping has not been reported before.

Our case is unique in several aspects. It is rare to observe a slow progression of ipsilateral monocular blindness resulting from an unruptured ACOM aneurysm without affecting the contralateral eye. Furthermore, our patient had a large tentorium cerebelli meningioma which has presumably led to ACOM aneurysm formation. To our best knowledge, this is the first reported case of monocular blindness caused by unruptured ACOM aneurysm with co-existence of tentorium cerebelli meningioma mimicking glaucomatous cupped disc.

In conclusion, from our literature review, there have only been 7 cases of unruptured ACA being reported presenting with loss of vision, thus indicating that they are rare. There have been 6 reported cases of internal carotid-ophthalmic artery aneurysm mimicking glaucomatous disc cupping, but ACOM aneurysm mimicking glaucomatous disc cupping has not been reported before. We hereby emphasize the importance of keeping a high degree of suspicion for life-threatening intracranial aneurysms in presence of asymmetrical optic disc cupping or optic disc pallor, especially when the visual dysfunction does not correlate with ocular findings.

DISCLOSURE

Ethics: Informed consent has been obtained from the patient.

Financial support: None

Conflicts of interest: None

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