CASE REPORT

GIANT INTRACRANIAL ANEURYSM PRESENTING AS TEMPORAL HEMIANOPIA: WHEN THE EYES REVEAL A HIDDEN DANGER

Annisa Nindiana Pertiwi[1], Syntia Nusanti[2], Marsha Rayfa Pintary[1], Sita Paramita Ayuningtyas[2], Salmarezka Dewiputri[2], Muhammad Sidik[2]

[1]*Residency Program in Ophthalmology, Faculty of Medicine Universitas Indonesia – Cipto Mangunkusumo General Hospital, Jakarta, Indonesia*

[2]*Neuro-Ophthalmology Division, Faculty of Medicine Universitas Indonesia – Cipto Mangunkusumo General Hospital, Jakarta, Indonesia*

Email: annisa.nindiana@gmail.com

ABSTRACT

Introduction: *Giant intracranial aneurysms (GIA) are rare and deadly diseases due to the high risk of rupture. The purpose of this report is to describe a case of GIA presenting as temporal hemianopia.*

Case Report: *A 52-year-old male presented with worsening blurry vision three weeks before admission. Visual acuity was light perception in the right eye and 6/18 in the left eye. Neuro-ophthalmic examination revealed a relative afferent pupillary defect in the right eye and bilateral optic atrophy. At the next visit, the visual acuity of the right eye recovered to 6/18. Visual field testing showed temporal hemianopia in the left eye and generalized depression in the right eye. Magnetic resonance imaging demonstrated a 0,4 cm x 2,5 cm x 1,9 cm saccular aneurysm, on the medial side of the left internal carotid artery (ICA).*

Discussion: *A giant (diameter ≥2.5 cm) ICA aneurysm may compress the optic chiasm, leading to various stages of visual loss. Several factors are known to delicate balance between thrombogenesis and thrombolysis within the aneurysmal sac. Spontaneous intra-saccular thrombosis in an unruptured GIA may be induced by calcification within the atherosclerotic wall of the aneurysm and loss of elastic lamina. It is prone to occur in a narrow aneurysm neck (<0.4 cm). Thrombosis reduces the size of the aneurysm sac, in which the accumulated fluid is reabsorbed. This may explain the decompression effect on optic chiasm and spontaneous visual recovery.*

Conclusion: *Intracranial aneurysms are a rare cause of optic chiasm compression but can still be considered in cases of temporal hemianopia.*

Keywords: *Intracranial aneurysm, optic chiasm compression, temporal hemianopia*

INTRODUCTION

Giant intracranial aneurysms (GIAs), which are vascular abnormalities over 25 mm in diameter, represent a small percentage (3%-13.5%) of all intracranial aneurysms, with a typical occurrence of around 5%. They pose a significant risk of rupture, making them rare but life-threatening conditions.[1-4] In a recent case, a GIA mimicked a space-occupying lesion and caused temporal hemianopia.

CASE ILLUSTRATION

A 52-year-old male presented with worsening blurry vision three weeks before admission. He had no complaints of headaches or a history of significant head trauma. Visual acuity was light perception in the right eye and 6/18 in the left eye. Neuro-ophthalmic examination revealed a relative afferent pupillary defect in the right eye, and bilateral optic atrophy (Figure 1 & Figure 2).

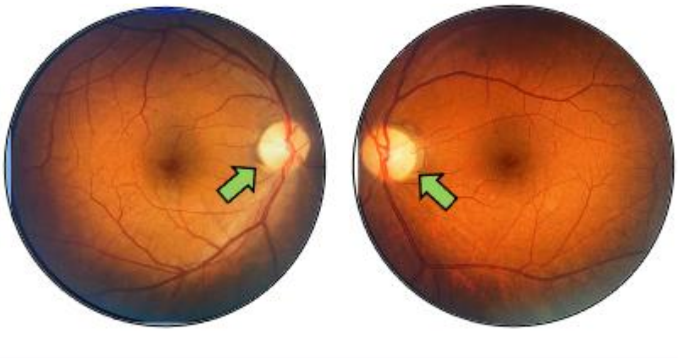

Figure 1. Fundus photographs showed bilateral optic disc atrophy (green arrows)

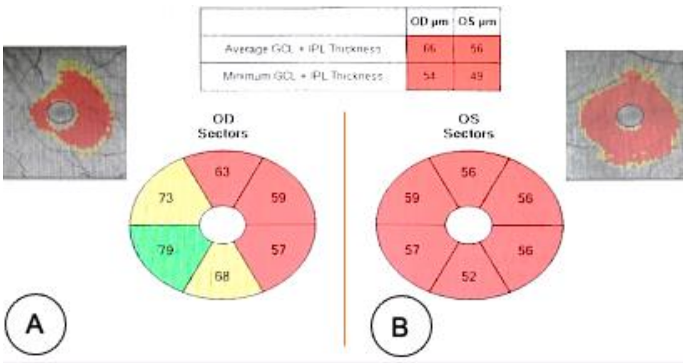

Figure 2. Optical coherence tomography of macular ganglion cell-inner plexiform layer (GC-IPL) showed (A) GC-IPL thinning predominates in the heminasal retina in the right eye, (B) GC-IPL thinning extends beyond midline crossing to the temporal hemifield in the left eye.

At the next visit, the visual acuity of the right eye recovered to 6/18. Visual field testing showed temporal hemianopia in the left eye and generalized depression in the right eye (Figure 3). Magnetic resonance imaging (MRI) of the brain demonstrated a saccular aneurysm, measuring 0,4 cm x 2,5 cm x 1,9 cm, on the medial side of the left internal carotid artery (ICA) (Figure 4).

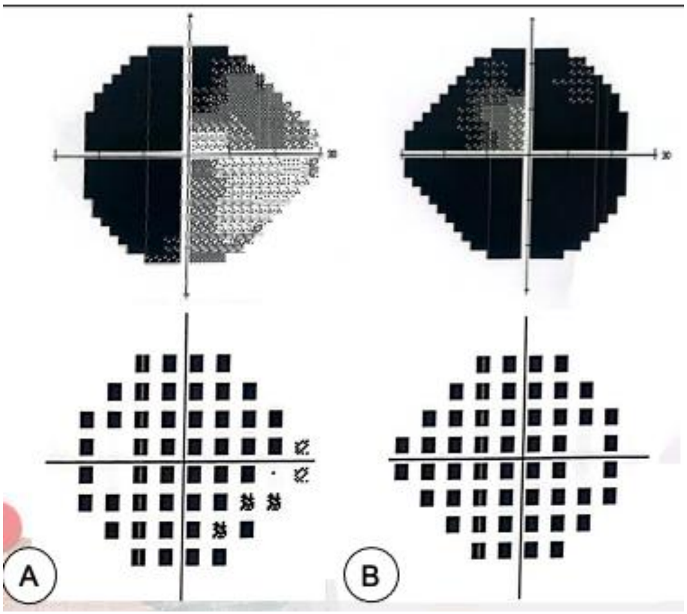

Figure 3. **Humphrey visual field 24-2 test showed (A) temporal hemianopia in the left eye (VFI 28%; MD -21.69 dB), (B) generalized depression in the right eye (VFI 6%; MD -31.15 dB).**

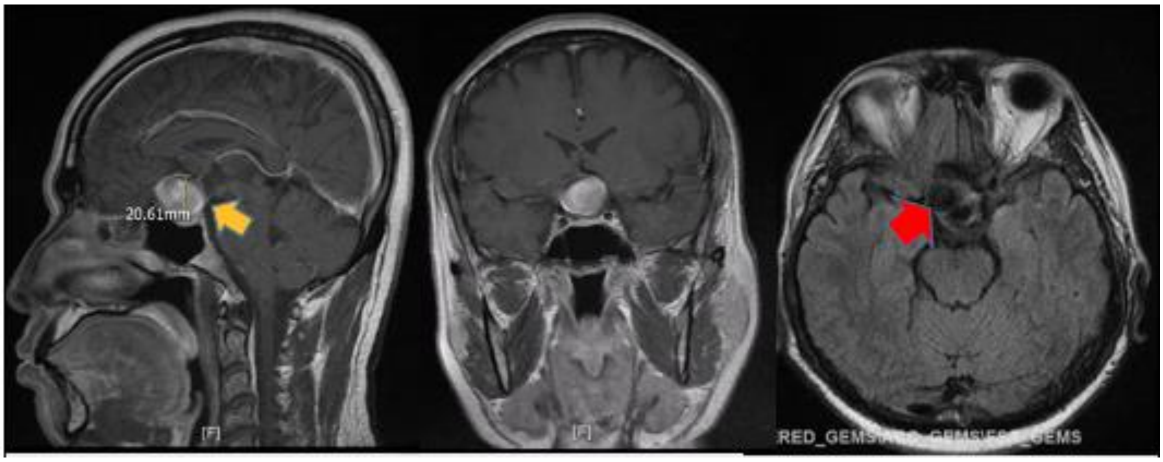

Figure 4. **Magnetic resonance imaging of the brain revealed a saccular aneurysm on the medial side of the left ICA pars C7 (neck width: 0,4 cm; dome height 2,5 cm; dome width: 1,9 cm) (yellow arrow), compressing the optic chiasm (red arrow).**

The patient was then immediately consulted for neurosurgical treatment. He was planned to undergo digital subtraction angiography (DSA) and installation of a flow diverter device (FDD).

DISCUSSION

Aneurysms typically develop in regions of high hemodynamic stress due to factors such as endothelial dysfunction, turbulent blood flow, and arterial wall damage. They can be categorized into saccular, fusiform, and dissecting types based on appearance and pathogenesis. Saccular GIAs, the most common type, resemble round or oval malformations with a connecting neck to the vessel. Fusiform aneurysms involve the entire arterial circumference and lack a distinct neck. Carotid artery aneurysms can manifest symptoms through rupture, mass effect on adjacent structures, or compression of nearby vessels, resulting in varying clinical presentations depending on their location and size.[1,4]

Internal carotid artery (ICA) aneurysms, depending on their size and location, can cause various visual pathway disturbances such as hemianopia or loss of acuity. These effects often stem from direct compression on the visual pathway, leading to neurological deficits. Visual impairments linked to intracranial aneurysms typically result from increased pressure on the anterior optic pathway near the Circle of Willis. Symptoms may include unilateral scotoma, blindness, bitemporal hemianopsia, or homonymous hemianopsia, all resulting from compression on the optic nerve, chiasma, or tract. Notably, these symptoms tend to progress gradually. Compressive optic neuropathy from aneurysms can present with diverse visual field defects, often featuring hemianopia visual field defects and progressive visual decline. In our case, temporal hemianopia was attributed to optic nerve compression by an unruptured ICA aneurysm.[1,5-7]

Diagnostic imaging such as computed tomography (CT) scans can reveal calcification and skull base erosion in large or giant aneurysms with mass effect, while MRI aids in assessing ischemia, perianeurysmal hemorrhage, and the aneurysm's relationship with surrounding neurovascular structures. Three-dimensional digital subtraction angiography (3D-DSA) is crucial for surgical planning, providing comprehensive visualization of the aneurysmal sac, neck, parent vessel, and aneurysm itself, facilitating understanding of hemodynamic characteristics related to aneurysm initiation, growth, and rupture. Intraoperative CFD imaging assists surgeons in identifying weaker or thrombosed aneurysmal walls to avoid or handle them with caution. Patients with visual field defects but no apparent ophthalmological abnormalities should undergo intracranial vessel studies like MRI, magnetic resonance angiography (MRA), or CT angiography (CTA).[4,6]

Relief of optic nerve compression through tumor removal or decompression surgery for aneurysms can restore visual function, even in cases of blindness. However, the recovery timeline varies, with some patients experiencing rapid improvement while others require longer periods due to remyelination. Successful decompression doesn't always guarantee full visual recovery, potentially due to vascular insufficiency of the optic nerve. In our case, complete recovery occurred after four weeks of visual deterioration to blindness, although spontaneous visual recovery is rare in compressive optic neuropathy from aneurysms.[7]

Spontaneous partial thrombosis of giant aneurysms is common, occurring in up to 60% of cases, with complete thrombosis being rarer and potentially associated with parent vessel occlusion. Thrombosed aneurysms likely arise from inflammatory mechanisms triggered by chronic dissections, hematomas of the aneurysmal wall, and the proliferation of vasa vasorum.[1,2] Several factors, such as geometrical configurations, hemodynamics, and biological conditions of the blood vessels around an aneurysm, are known to delicate balance between thrombogenesis and thrombolysis within the aneurysmal sac. Spontaneous intra-saccular thrombosis in an unruptured GIA may be induced by calcification within the atherosclerotic wall of the aneurysm and loss of elastic lamina. Other factors contributing to spontaneous thrombus formation are blood stagnation, increased viscosity, and slow flow. Thrombosis especially in giant aneurysms is prone to occur in narrow aneurysm neck size (<4 mm), as observed in our case. Thrombosis reduced the size of the aneurysm sac, and the fluid accumulated in the sac was reabsorbed. This may cause the decompression effect on optic chiasm and explain the phenomenon of spontaneous visual recovery in our case.[8-9]

Treating giant intracranial aneurysms (GIAs) poses significant challenges due to their high rupture risk and disabling symptoms. Management options include neurosurgical techniques (open surgery), endovascular procedures, or a combination of both. Endovascular methods are increasingly favored for ICA GIAs due to challenging neurosurgical access. Survival and complication rates between the two approaches are comparable, although evidence from large clinical trials is lacking. Treatment decisions hinge on factors like patient age, comorbidities, aneurysm size, location, morphology, neck-to-dome ratio, presence of thrombosis and calcification, collateral circulation, and critical perforating vessels. [1,4,6,10]

Endovascular decisions consider aneurysm localization, anatomy, size, dome-to-neck ratio, thrombus presence, patient health, and material availability. Therapeutic techniques include embolization with platinum coils or flow-diverting stents, aiming to occlude the aneurysm while maintaining parent vessel flow. Parent vessel occlusion (OPV) is another option, associated with reduced aneurysm growth and retreatment rates but requires sufficient collateral circulation.[1,2]

Surgical options involve aneurysmal clipping with thrombectomy, trapping with or without revascularization. Careful surgical handling is crucial to prevent thrombus dislodgment and embolism. Wide necks and intraluminal thrombus can complicate clipping, necessitating revascularization in unfavorable embolization conditions or poor collateral circulation. Patient factors, aneurysm characteristics, and team expertise guide treatment decisions for GIAs.

CONCLUSION

Intracranial aneurysms are a rare cause of optic chiasm compression but can still be considered in cases of temporal hemianopia. Prompt diagnosis and immediate treatment are recommended to prevent serious complications.

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