Split optic nerve penetrated by a carotid-ophthalmic aneurysm another instance of a rare presentation: A case report

Caleb E Feliciano, Duke Samson

ABSTRACT

Introduction: Penetration of the optic nerve by aneurysms of the paraclinoid carotid artery is rare. We present the seventh aneurysm-related split optic nerve case reported in the literature, review the pathophysiologic mechanisms and discuss the technical aspects for successful clip ligation and complication avoidance. Case Report: A 64-year-old male with history of hypertension, chronic obstructive pulmonary disease and polycystic kidneys presented with headache and dizziness. Magnetic resonance imaging and angiographic work-up confirmed the presence of a right-sided partially thrombosed carotid-ophthalmic aneurysm. There was no evidence of significant visual dysfunction. A right pterional craniotomy with frontal extension was performed. The aneurysm dome was noted to extend subfrontally after passing through a split optic nerve. Aneurysm neck was clipped using a combination of standard ophthalmic and fenestrated clips. During immediate post-operative period the patient had worsening of visual acuity, yet showed progressive visual improvement on clinical follow-up. Conclusion: The pathophysiologic mechanisms for aneurysm development range from congenital to mechanical/hemodynamic. Our patient’s clinical presentation suggests a slowly growing lesion that may have penetrated through a weak spot in the overlying optic nerve. This may have been caused by either congenital fenestration of the optic nerve, persistent vestigial artery or an old unrecognized hemorrhage. Optic nerve penetration by carotid-ophthalmic aneurysms occurs rarely, yet is being increasingly reported and recognized. Knowledge of this potential configuration can help avoid inadvertent damage and improve results by novel application of fenestrated and standard clips.

Keywords: Carotid-ophthalmic aneurysm, Optic nerve fenestration, Paraclinoid aneurysm, Split optic nerve, Surgical technique

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INTRODUCTION

Carotid-ophthalmic (CO) aneurysms constitute 0.3–1% of intracranial aneurysms and 0.9–6.5% of aneurysms of the internal carotid artery (ICA) [1]. Although they commonly present with subarachnoid hemorrhage, presentation with visual dysfunction due to mass effect on the optic pathways can also occur [2, 3], usually after reaching a size of more than 1 cm [4, 5].
In Yasargil series [6], 18% of patients presented with visual symptoms. Day [4] reported that 30% cases with CO aneurysms can present with visual dysfunction. CO aneurysms usually displace the optic nerve (ON) superomedially against the falciiform ligament as they grow. Penetration of the optic nerve by aneurysms of this region occurs rarely. Only six case reports were found in literature showing this occurrence (seven after including a case of de novo ON splitting without associated aneurysm [7], one of them by a ruptured anterior communicating artery aneurysm, and the rest by paraclinoidal ICA aneurysms [1, 5, 7–10, 13]. To this instance, we present the seventh case reported in literature and review the proposed pathophysiologic mechanisms and the technical aspects for successful clip ligation and complication avoidance.

CASE REPORT

The patient was a 64-year-old male with history of hypertension, chronic obstructive pulmonary disease (COPD) and polycystic kidney disease (PKD) who presented with an episode of headache and dizziness. On physical examination the patient was fully alert and oriented, as well as able to provide his own history. Cranial nerve evaluation, motor, sensory and cerebellar examinations revealed no gross neurologic deficits. There was no evidence of significant visual dysfunction, except for the fact that the patient reported using glasses. No formal visual field testing (perimetry) was done. Due to a significant history of protracted smoking and family history of subarachnoid hemorrhage, magnetic resonance imaging/magnetic resonance angiography (MRI/MRA) work-up were done which confirmed the presence of a right-sided partially thrombosed carotid-ophthalmic aneurysm measuring approximately one cm in its largest diameter (Figure 1).

In view of the aneurysm findings, the patient was offered endovascular treatment with stent-assisted coiling in the referring hospital, however he had significant tortuosity in the access vessels and the procedure was deemed unsafe. At our clinic evaluation, he was offered surgical exploration for clip ligation due to the high estimated risk of hemorrhage and possibility for development of significant mass effect symptoms.

Operative Technique: Under general endotracheal anesthesia, the patient was positioned supine and his head turned and fixed 45° to the left using the Mayfield-Kees head holder. After prepping and draping, a curvilinear skin incision was made beginning anterior to the tragus and ending midline behind the hairline. Separate skin and temporalsis muscle flaps were elevated and secured with fish-hook retractors. A rectangular bone flap centered at the pterion with frontal extension was performed. Subsequently, the sphenoid wing was drilled until visualization of the lateral superior orbital fissure. The dura was opened and the optico-carotid cistern reached and opened for CSF drainage. A dense arachnoiditis was noted between the optic nerve and the gyrus rectus. This was sharply dissected, and the aneurysm dome was noted to extend subfrontally below the gyrus rectus. It protruded through a split optic nerve (Figure 2A). Attention was then directed to the neck of the aneurysm and after adequate proximal and distal control, it was clipped with two large Yasargil ophthalmic clips that spanned the entire atherosclerotic portion of the neck. The aneurysm dome was subsequently opened and decompressed by removing the atherosclerotic debris and old thrombus. On reaching the proximal neck of the aneurysm brisk arterial bleeding was noted. A thin part of the medial aneurysm neck was not adequately sealed by the clips due to significant luminal irregularity. A third large straight fenestrated clip was then placed, including the lateral half of the optic nerve in the fenestration and effectively occluding the neck of the aneurysm (Figure 2B). Patency of the surrounding vessels was confirmed by Doppler insonation. Postoperative visual examination revealed worsening of visual acuity on the right eye to finger counting and mild right inferior visual field loss. The patient was seen one month after surgery and visual acuity was noted to be steadily improving.

DISCUSSION

Optic nerve penetration by carotid-ophthalmic aneurysms occurs rarely, yet is being increasingly reported and recognized. The pathophysiologic mechanisms proposed in the literature range from
that direction, splitting the nerve longitudinally along
the nerve fibers. A similar mechanism was proposed by
Fujita et al. for a patient with rapidly progressive visual
loss accounted for by a rapidly growing CO aneurysm
[14].

Table 1 illustrates the important aspects of the cases
that have been reported so far, as well as the
pathophysiologic mechanisms proposed. Other similar
cases have been commented on, yet not reported.
Therefore, this condition may be more common than
expected. In our case, there was no obvious
intraoperative evidence of hemorrhage. In addition, a
vestigial remnant of an arterial perforator or congenital
anastomotic artery was not found. Taking into account
that visual deterioration was not the chief medical
complaint of the patient, it suggests a slowly growing
lesion that may have penetrated through a weak spot in
the overlying ON. The weakened spot may have been
caused by any of the prior mentioned mechanisms,
whether congenital fenestration of the ON, persistent
vestigial artery or even an old unrecognized
hemorrhage. The exact pathophysiologic mechanism is
difficult to point out, and every mechanism discussed
may in fact play a role in different cases. Increasing
knowledge of this potential configuration can help avoid
inadvertent damage and improve results through
careful surgical technique and novel application of
fenestrated and standard aneurysm clips.

CONCLUSION

In published literature the pathophysiologic
mechanisms proposed for development of carotid-
ophtalmic aneurysms range from congenital to
mechanical/hemodynamic. Optic nerve penetration by
carotid-ophtalmic aneurysms rarely occurs, yet is
increasingly reported and recognized. Knowledge of this
potential configuration can help avoid inadvertent
damage and improve results by novel application of
fenestrated and standard clips.

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Author Contributions
Caleb E Feliciano – Substantial contributions to
creation and design, Acquisition of data, Drafting the
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Revising it critically for important intellectual content,
Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of
submission.

Conflict of Interest
Authors declare no conflict of interest.
Table 1: Clinical, radiologic, operative findings and proposed mechanisms for split optic nerve cases reported in the literature.

<table>
<thead>
<tr>
<th>Reference (year) (ref. no.)</th>
<th>Age (years) /sex</th>
<th>Clinical presentation</th>
<th>Radiologic findings</th>
<th>Operative findings</th>
<th>Outcome</th>
<th>Mechanism proposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beatty et al. (1986) [10]</td>
<td>66/F</td>
<td>HA, dysphasia and right arm clumsiness, VF full by confrontation.</td>
<td>CT showed a four cm calcified mass with surrounding edema and SAH around anterolateral margin; DSA confirmed a superiorly projecting CO aneurysm.</td>
<td>Giant CO aneurysm with neck passing through left ON, split longitudinally in half. Recent anterolateral blood. Lateral half of ON sacrificed for clipping.</td>
<td>Only light perception postop.</td>
<td>Aneurysm arising from vessel already penetrating ON.</td>
</tr>
<tr>
<td>Date et al. (1997) [7]</td>
<td>40/M</td>
<td>HA and bilateral blurred vision. Found to have left homonymous hemianopsia and finger counting acuity in right eye.</td>
<td>CT w/ and w/o contrast showed partially thrombosed AComMA aneurysm; DSA showed a seven mm AComMA aneurysm projecting inferiorly.</td>
<td>AComMA aneurysm originating from A1/A2 junction and projecting inferiorly through optic chiasm. Small amount of SAH noted.</td>
<td>Patient preoperative visual disturbance persisted in F/U.</td>
<td>Rupture of the AComMA aneurysm against the chiasm and subsequent growth.</td>
</tr>
<tr>
<td>Fujita et al. (2002) [14]</td>
<td>70/F</td>
<td>Progressive left eye visual loss. Finger counting VA in left eye. VF showed nasal hemianopsia in left eye and upper temporal quadrantanopsia in right eye. Pale left optic disk on fundoscopy.</td>
<td>CT normal; MRI w/ flow void in suprasellar area, no intraluminal thrombus; DSA showed 5.5x9.5 mm CO aneurysm projecting superomedially.</td>
<td>Penetration of posterior half of chiasm by aneurysm, extending to posterior part of left ON. Thinning of the perianeurismal chiasm. Aneurysm decompressed. No intraluminal thrombus.</td>
<td>VA on the left improved to 20/50. VF deficit persisted.</td>
<td>Rapid growth of unruptured CO aneurysm causing penetration of optic chiasm and visual symptoms.</td>
</tr>
<tr>
<td>House et al. (2005) [9]</td>
<td>50/F</td>
<td>Refractory tinnitus and HAs.</td>
<td>Eight mm aneurysm at origin of PComMA. No info on modality used.</td>
<td>Split ON with a smaller medial portion coursing separately to optic canal. No embryonic artery, osseous trabeculae or penetrating aneurysm found.</td>
<td>N/A</td>
<td>De novo fenestration of ON.</td>
</tr>
<tr>
<td>Full Name</td>
<td>Age (y)</td>
<td>Gender</td>
<td>Presenting Symptom</td>
<td>Neuroimaging Findings</td>
<td>Clinical Findings</td>
<td></td>
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<td>Jea et al. (2003)</td>
<td>48/M</td>
<td></td>
<td>HA and right visual acuity deterioration to 20/800. Central scotoma on visual fields. Pale ON on fundoscopy.</td>
<td>CT w/ contrast showed a one cm suprasellar mass and mild HCP; MRI revealed piercing of ON by aneurysm and mild HCP; DSA showed a 12x7 mm right CO aneurysm, projecting superomedially and with a “waist” in the ON.</td>
<td>Small protruding mass coming from a large ophthalmic aneurysm inferior to the ON at the chiasmal junction. Unchanged visual function at least resistance in the ON possibly favored by arterial perforator.</td>
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<tr>
<td>Kanamaru et al. (2001)</td>
<td>73/F</td>
<td></td>
<td>HA and abrupt loss of consciousness.</td>
<td>CT showed diffuse SAH; DSA showed a large aneurysm arising from anterior ICA and projecting superiorly.</td>
<td>Saccular aneurysm arising from anterior ICA wall and projecting superiorly through left ON. Poor neurologic outcome due to severe refractory vasospasm. Aneurysm arising from vessel already penetrating the ON.</td>
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<tr>
<td>Wang et al. (2010)</td>
<td>45/F</td>
<td></td>
<td>HA. No visual dysfunction.</td>
<td>CT showed diffuse SAH; CT-A and DSA showed 15 mm CO aneurysm projecting anterosuperiorly.</td>
<td>Aneurysm dome noted superior to ON w/ surrounding clot. ON noted to be split by aneurysm after clipping of neck and further dissection. Dome decompressed. Recovery of aneurysm from uneventful. No visual deficits reported. Remnant/perforat or vessel through de novo fenestration. Slow development</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: HA - headache, VF - visual fields, CT - computerized tomography, SAH - subarachnoid hemorrhage, DSA - digital subtraction angiography, CO - carotid-ophthalmic, ON - optic nerve, w/ - with, w/o - without, AComM - anterior communicating artery, F/U - follow-up, VA - visual acuity, MRI - magnetic resonance imaging, PComM - posterior communicating artery; N/A - not applicable, HCP - hydrocephaly, ICA - internal carotid artery, CT - A-computerized tomography-angiography, postop - postoperative.

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