Elongation of fusiform enlargement or dolichoectasia of the ICA has been associated with a variety of visual field deficits including binasal and bitemporal hemianopia as well as both unilateral and bilateral optic neuropathy. Visual field deficits due to compression of the ACA are rare.

We report on a case of progressive visual deficits caused by compression of the optic chiasm by an elongated ACA. To our knowledge, this is the first description of successful MVD for such vascular compression.

Case Report

History and Examination. This 56-year-old man was referred to our neurosurgery department for progressive bitemporal hemianopia. He was known for high blood pressure, smoking, familial hypertriglyceridemia, and chronic pancreatitis with episodes of intravenous hyperalimentation. There was also a family history of glaucoma. He was first seen by the ophthalmologist in 1998 for bilateral blurry vision in the context of a migraine. The ophthalmological examination was normal except for a slightly elevated ocular tension at 19 torr bilaterally. No medication was begun at this time. In 2002, although the ocular tension remained stable, a discrete bilateral nasal step was noticed on visual field examination. In 2006 the ophthalmological follow-up revealed binasal hemianopia, with normal visual acuity and ocular tension.

On cerebral MR imaging, the right proximal ACA revealed a vascular loop compressing the optic chiasm, especially on the right side, and displacing it inferiorly (Fig. 1). The left ACA appeared normal. The verteobasilar system also appeared tortuous. Other than an intrasellar arachnoidcele, no other lesion was visualized. No other cause could explain the progressive visual field deficits. The vascular loop was thought to be responsible for the progressive binasal deficit, and MVD was recommended.

Operation. Through a brow incision, a right supraorbital craniotomy was performed. The right A1 segment was dissected on all its length up to the lamina terminalis cistern. The right ACA was larger than usual and tortuous; however, the vessel did not appear sclerotic. The vascular loop made an indentation in the chiasm (Fig. 2). During surgery, the transmission of vascular pulsations onto the chiasm could be seen. A piece of Teflon was inserted between the right tortuous A1 segment and the chiasm, preventing the transmission of pulsations (Fig. 2).
Postoperative Course. Within the first 48 hours after surgery, the patient’s visual field deficits improved. During hospitalization, he experienced a recurrent episode of chronic pancreatitis for which he required a short period of intravenous hyperalimentation. Four months after surgery, a follow-up ophthalmological examination revealed normal visual fields according to Humphrey testing (Fig. 3), normal macula and optic disks, and stable visual acuity and ocular tension.

Discussion

Compression of the optic nerve or the chiasm by intracranial vessels, most often the ICA, has been reported in the literature. Indeed, enlargement or dolichoectasia of the ICA has been associated with a variety of visual field defects, depending on the site of compression on the visual apparatus.4,5 Microvascular decompression has been performed in few cases, with improvement of visual field defects in some cases.1,3

Compression of the visual apparatus by an elongated ACA has rarely been reported, with only 5 other cases found in the literature (Table 1). This compression by the ACA has been described with a variety of visual field defects including binasal and bitemporal deficits and optic neuropathy.2,8 The patient in 1 case presented with binasal visual defects similar to those in our case.8 As stated by Manor et al.,3 the pathogenesis of nasal visual field defects remains unclear in the presence of unilateral compression.

Of the 5 cases discussed in the literature, 3 were documented in autopsy studies,6,7 1 was suspected on angiography,2 and another was identified during surgical exploration for a brain tumor.8 To our knowledge, this report is the first instance of MVD of the chiasm in such circumstances.

The progressive rather than stable established visual field deficits as well as the absence of optic neuropathy prior to decompression might have contributed to the favorable outcome following MVD. The early postoperative visual improvement supports the hypothesis that the visual field deficits were due to the pulsatile compression of the chiasm by the tortuous ACA. In addition to the dynamic aspect of the compression, the inferior displacement of the optic chiasm by the elongated right ACA might have induced traction on lateral fibers, possibly contributing to the pathogenesis of nasal visual field deficits. Other authors have proposed that visual deficits might result from impaired blood circulation in the prechiasmal anastomotic plexus due to stretching of nutrient vessels.2

In summary, MVD should be considered when facing a progressive and/or severe visual field deficit in the presence of an elongated ACA compressing the optic chiasm and in the absence of other apparent etiology. Minimally invasive supraorbital craniotomy offers good exposure for MVD of the optic chiasm.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Acknowledgement

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**TABLE 1: Literature review of cases of compression of the visual apparatus**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Known Visual Deficits</th>
<th>Method of Diagnosis</th>
<th>Atherosclerosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yaskin &amp; Schlezinger, 1942</td>
<td>61, H</td>
<td>binasal inferior quadrantopia, rt temporal field deficit, decreased visual acuity</td>
<td>intraop</td>
<td>present</td>
<td>none</td>
</tr>
<tr>
<td>Walsh &amp; Gass, 1960</td>
<td>64, F</td>
<td>NS</td>
<td>autopsy</td>
<td>present</td>
<td>none</td>
</tr>
<tr>
<td>Hilton &amp; Hoyt, 1966</td>
<td>68, M</td>
<td>bitemporal hemianopia, mostly confined to superior quadrants, decreased visual acuity, bilat pale optic disc</td>
<td>carotid arteriogram</td>
<td>NS</td>
<td>none</td>
</tr>
<tr>
<td>Sacks &amp; Lindenburg, 1969</td>
<td>NS</td>
<td>NS</td>
<td>autopsy</td>
<td>absent</td>
<td>none</td>
</tr>
<tr>
<td>present case</td>
<td>56, M</td>
<td>binasal hemianopia</td>
<td>MRI, confirmed intraop</td>
<td>absent</td>
<td>MVD</td>
</tr>
</tbody>
</table>

* NS = not specified.
Chiasm compression by the anterior cerebral artery

Fig. 2. Intraoperative photographs showing an indentation (A, asterisk) made in the chiasm by the elongated right ACA and a piece of Teflon (B) inserted between the right tortuous A1 segment and the chiasm.

Fig. 3. Preoperative visual field assessments (upper) documenting the progression of binasal visual field deficits as well as postoperative analyses (lower) revealing normalization of the visual field defects.
References


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