

CASE REPORT

Richard C. Kho, MD

*Department of Ophthalmology
and Visual Sciences
University of the Philippines
Philippine General Hospital
Manila, Philippines*

Optic-nerve compression by the internal carotid artery as a cause of “unexplained” optic atrophy

ABSTRACT

Objective

To present a case of bilateral optic neuropathy secondary to optic-nerve compression by the internal carotid artery (ICA).

Methods

Observational case report; single patient seen in private practice.

Results

Compression of the optic nerves by the ICAs as the etiology of unexplained progressive optic neuropathy is reported. The patient refused further invasive procedures after diagnosis.

Conclusion

Compressive optic neuropathy due to direct pressure by the adjacent ICA should be considered as a possible etiology of occult, progressive visual loss in an otherwise healthy patient. Although rare, the clinical picture of progressive optic neuropathy backed by evidence of magnetic resonance imaging (MRI) should clinch the diagnosis early and spare the patient from further unnecessary ancillary work-up.

Keywords: *Compressive optic neuropathy, Internal carotid artery, Dolichoectatic artery, Fusiform enlargement*

Correspondence to:
Richard C. Kho, MD
15 General de Jesus St., Heroes Hill
1106 Quezon City, Philippines
Tel: +63-2-6360762
Fax: +63-2-6385837
E-mail: rkho@i-manila.com.ph

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PROGRESSIVE loss of vision secondary to optic-nerve compression by the supraclinoid portion of the internal carotid artery (ICA) became known before the age of computed tomography.¹⁻³ Neurosurgical exploration was frequently utilized to evaluate the etiology of occult progressive optic neuropathy.² With the advent of and subsequent advances in neuroimaging techniques, interest in this clinical entity was revived with noninvasive methods of assessing the anatomic relationship between the optic nerve and the supraclinoid ICA.^{4,6} This clinical condition as a cause of unexplained chronic, progressive visual loss has not been reported in the Philippines. Clinical and neuroradiological correlation of these cases is crucial in correctly making the initial diagnosis before otherwise healthy patients presenting with “unexplained” visual loss are either subjected to various unnecessary work-up, or worse, left in the dark about the etiology of their progressive blindness.

The author presents a case in which the diagnosis of optic-nerve compression by the intracavernous ICAs as the cause of slowly progressive visual loss eluded clinicians for several years before a review of the patient’s old magnetic resonance imaging (MRI) plates and repeat scans confirmed the diagnosis.

CASE REPORT

A 79-year-old hypertensive male was referred for bilateral optic atrophy. The patient had undergone phacemulsification with posterior chamber intraocular lens (IOL) implantation on the left eye (OS) in 1997. Best-corrected visual acuity on this eye was 20/40 at the time. The right eye (OD) underwent the same procedure in 2001. Optic-nerve pallor was initially noted on the right eye at the time after the patient complained of gradual and progressive blurring of vision. Ancillary procedures conducted revealed the following: MRI of the orbits and optic nerves (Figure 1) was read as within normal, fluorescein angiogram (FA) was normal, visual-evoked response (VER) showed severe conduction defect in OD and mild conduction defect in OS, electroretinography (ERG) revealed reduced cone (macula) responses and normal rod responses in both eyes (OU), automated achromatic perimetry showed diffuse depression greater in OD compatible with advanced optic-nerve damage. Routine cardiovascular and blood chemistry did not yield any insights as to the etiology of the visual loss.

At the time of consultation, best-corrected visual acuity was 20/200 OD and 20/40 OS. Slit-lamp examination showed normal anterior chambers with a centrally placed posterior chamber IOL in both eyes. Intraocular pressures were normal and motility examinations were within normal limits. Fundus examination with a 78D lens revealed bilateral optic-nerve pallor, greater in OD. The

vascular arcades and the rest of the posterior pole did not show any gross abnormalities. A review of MRI scans performed two years prior to the consultation showed that the intracavernous portion (Figure 1) of the ICA impinged on the chiasm, with slightly greater indentation on the right side. These scans were sent to a neuroradiologist for review and new MRI scans of the orbit/optic nerves were requested for comparison. Figure 2 shows a coronal section through the chiasm portion of the optic nerves on the repeat MRI. The supraclinoid and cavernous ICAs are bilaterally tortuous and mildly ectatic. Both ICAs demonstrate vascular segments which are more medially and superiorly located than the average patient, abutting the optic chiasm bilaterally, as well as a significant portion of the prechiasmatic segments of both optic nerves. The right side of the optic chiasm/proximal right optic nerve is superiorly displaced and draped around the subjacent



Figure 1. First MRI (October 9, 2002). T1-weighted coronal section through the chiasm (white arrow) and intracavernous internal carotid arteries (black arrows).

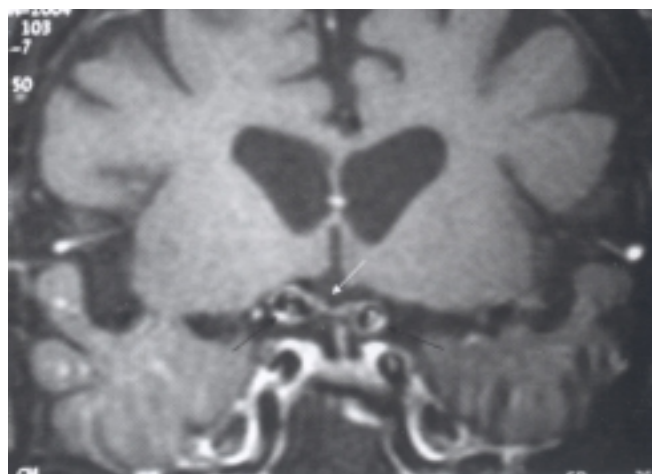


Figure 2. Repeat MRI (August 26, 2004). T1-weighted coronal section shows both ICAs (black arrows) pushed up against the chiasm (white arrow), greater on the right side (right optic nerve literally wrapped over the convexity of the artery).

right intracranial ICA segment. The optic chiasm appears smaller than average and probably represents atrophy from chronic compression by the intracavernous ICAs. The patient refused further invasive treatment, including scant neurosurgical options, which have not been tried in the Philippines.

DISCUSSION

Unilateral or bilateral optic neuropathy secondary to fusiform enlargement or “dolichoectasia” of the ICA as a cause of occult, slowly progressive loss of vision has previously been reported in foreign literature.^{1,3} In this age of high-resolution neuroimaging techniques, renewed interest in the intimate anatomic relationship between the optic nerve and the supraclinoid ICAs has emerged. Golnik and associates analyzed the MRIs of 20 patients presenting with unexplained optic-nerve dysfunction and speculated that the anatomic proximity between the optic nerve and the ICA may be important in the development of optic neuropathy.⁴ Jacobson and associates further elucidated on this anatomic relationship by analyzing the MRIs of 100 patients who underwent the procedure for a variety of reasons unrelated to visual loss or optic neuropathy.⁵ The authors presented a grading scheme to classify their appearance on neuroimaging as follows: Grade 0, no contact; Grade 1, contact but without distortion of the optic nerve (i.e., contact but no compression); Grade 2, contact with distortion of the optic nerve contour (i.e., compression). Surprisingly, contact (no compression) between the two structures occurred at a high frequency (70%), while compression was noted at a much lower frequency (30%) in these asymptomatic patients. It should be noted from this study that neuroradiological evidence of contact or compression of the optic nerves need not be accompanied by clinically evident optic neuropathy. In a follow-up paper, Jacobson described *symptomatic* compression of the optic nerve in 24 eyes of 18 patients with radiologic evidence of contact and compression.⁶ Hypertension (present in our patient) was noted in 10 of 18 patients in this series.

In our patient, it was evident that optic-nerve contact and compression by the ICA (Grade 2 on the right; Grade 1-2 on the left) was already present when the first MRI was taken almost two years before this consultation (Figure 1). The arterial ectasia and optic-nerve compression in the right eye appear to be worse than in the left eye, clinically evident as worse optic-nerve dysfunction on this side (Figure 2). This diagnosis was missed on the first MRI when the optic nerves were read as within normal limits. Several ophthalmologists who evaluated the patient at the time could not identify the etiology of the optic neuropathy after subjecting the patient to various ancillary procedures. Discussions with local neuroradiologists confirmed that contact between these structures is

occasionally seen on MRI but is neither considered nor reported as a potential cause of progressive “unexplained” optic neuropathy in the local setting. Armed with MRI plates returned as “normal,” attending ophthalmologists consequently do not consider this etiologic possibility for the optic neuropathy. A review of the local literature did not yield any report on this condition. To our knowledge, this is the first local report.

The prognosis for visual improvement in this patient (who refused further invasive procedures) was not encouraging. Although isolated reports of visual improvement with neurosurgical procedures to decompress optic-nerve compression by the ICA have been published,⁷ it is still unclear whether these procedures are beneficial at all in the reversal of existing visual loss or prevention of further optic neuropathy.^{2, 6} These procedures should not be expected to reverse the optic neuropathy if ischemia from compression is its predominant mechanism.

The rate of visual loss in these cases without surgical intervention tends to be very slow.⁶ Subsequent follow-ups should give the clinician an idea of the course of the optic neuropathy, which may be slowly progressive or static. Improvement in the cardiovascular status should theoretically be beneficial, as ageing and hypertension are correlated with an increase in arterial diameter. Indeed, Jacobson et al. noted that the estimated odds of compression were increased by a factor of 2.54 for each millimeter increase in the diameter of the carotid artery.⁵

Thin-section, T1-weighted, coronal cuts constitute the optimal MRI protocol for identifying optic-nerve compression by the ICA (Figures 1 and 2). Presented with clinical findings of progressive optic neuropathy and evidence of compression on MRI (not just contact), the clinician may spare the patient from unnecessary ancillary tests and delay in diagnosis. Certainly, these MRI techniques should be performed on any patient presenting with progressive optic neuropathy of unknown etiology. Though rare and unreported locally, optic-nerve compression by the supraclinoid carotid artery should be kept in mind as a potential cause of “unexplained” progressive optic neuropathy.

References

1. Bergaust B. Unusual course of internal carotid accompanied by bitemporal hemianopsia. *Acta Ophthalmol (Scand)* 1963; 41: 270-274.
2. Mitts MG, McQueen JD. Visual loss associated with fusiform enlargement of the intracranial portion of the internal carotid artery. *J Neurosurg* 1965; 23: 33-37.
3. Manor RS, Ouaknine GE, Matz S, Shalit MN. Nasal visual field loss with intracranial lesions of the optic nerve pathways. *Am J Ophthalmol* 1980; 90: 1-10.
4. Golnik KC, Hund PW III, Stroman GA, Stewart WC. Magnetic resonance imaging in patients with unexplained optic neuropathy. *Ophthalmology* 1996; 103: 515-520.
5. Jacobson DM, Warner JJ, Broste SK. Optic-nerve contact and compression by the carotid artery in asymptomatic patients. *Am J Ophthalmol* 1997; 123: 677-683.
6. Jacobson DM. Symptomatic compression of the optic nerve by the carotid artery: clinical profile of 18 patients with 24 affected eyes identified by magnetic resonance imaging. *Ophthalmology* 1999; 106: 1994-2004.
7. Colapinto EV, Cabeenn MA, Johnson LN. Optic nerve compression by a dolichoectatic internal carotid artery: a case report. *Neurosurgery* 1996; 39: 604-606.