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SHORT COMMUNICATION

Optociliary shunt vessels in compressive optic neuropathy by the intracranial internal carotid artery

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PURPOSE. *To report a clinical case of optic nerve compression by supraclinoidal internal carotid artery associated with optociliary shunt vessels.*
METHODS. *A 78-year-old woman with the clinical triad of left visual loss, ipsilateral optic disc pallor, and retinochoroidal (optociliary) shunt vessels is reported. She complained of loss of vision in the left eye of 2 years' duration.*
RESULTS. *A diffuse depression of the visual field was found in the affected eye. Magnetic resonance imaging revealed left optic nerve compression by the supraclinoidal internal carotid artery.*
CONCLUSIONS. *The occurrence of optociliary shunt vessels, visual loss, and optic atrophy is a non-specific sign of chronic optic nerve compression and in some instances may be falsely localized. (Eur J Ophthalmol 2008; 18: 316-9)*

KEY WORDS. *Compressive optic neuropathy, Supraclinoidal intracranial carotid artery, Optociliary shunt vessels*

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INTRODUCTION

Optociliary shunt vessels are classically described to be associated with optic nerve sheath meningioma, with the triad symptoms of optociliary veins, disc pallor, and visual loss (1). Other clinical settings include optic nerve glioma, meningocele of the optic nerve, other less common orbital lesions, glaucoma, and chronic papilledema.
Noninvasive evaluation of the relationship between the optic nerve and the intracranial carotid artery (ICA) by the new neuroimaging devices has raised the question of whether vascular compression can lead to occult visual loss.
A patient with slowly progressive monocular visual loss and optic atrophy with optociliary shunt vessels is reported. Magnetic resonance imaging (MRI) revealed compression of the optic nerve by the ICA.

Case report

A 78-year-old woman was referred to our neuro-ophthalmology department for left visual loss that was insidious at onset, painless, and unassociated with other neurologic symptoms. Past medical history was unremarkable.
Best-corrected visual acuity was 20/40 in right eye (RE) and counting fingers at 50 cm in left eye (LE). A left relative afferent pupillary defect was noticed. Slit-lamp biomicroscopy showed a moderate lens opacification in RE and an uneventful phacoemulsification in LE. Intraocular pressure was 19 mm Hg in both eyes. RE fundus examination was normal. Left optic disc was pale with optociliary shunt vessels overlying it (Fig. 1).
Automated perimetry (24-2 SITA strategy, Humphrey Visual Field Analyzer; Carl Zeiss Meditec, Dublin, CA) of the RE showed a moderate diffuse visual field loss in right

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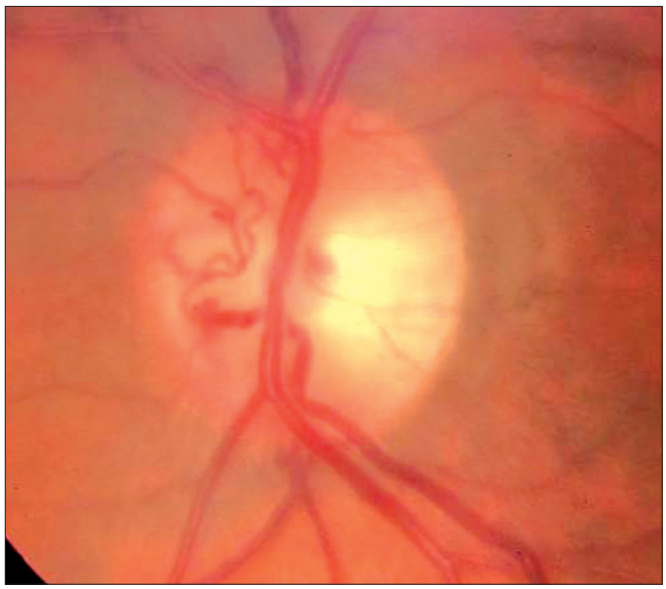

Fig. 1 - *Left eye: Optic disc pallor and optociliary shunt vessels.*

eye, related to cataract. A severe diffuse visual field loss was observed in LE (Fig. 2).

Optical coherence tomography using the peripapillary fast retinal nerve fiber layer (RNFL) program (Stratus OCT-3; Carl Zeiss Meditec) was normal in RE and revealed a significant RNFL thinning in nasal, temporal, and inferior quadrants in LE (Fig. 2).

Blood count and erythrocyte sedimentation rate were normal.

MRI revealed no lesion within the orbit. Surprisingly, T2-weighted, coronal orientation showed left optic nerve compression by the supraclinoid segment of the ICA. Left optic nerve was medially displaced, thinned, and molded over the artery. This finding was more remarkable when comparison was made with normal optic nerve in RE (Fig. 3).

DISCUSSION

Although unusual, intracranial compression of the optic nerve by the carotid artery should be considered in unexplained or progressive optic neuropathy. As the intracranial portion of the optic nerve ascends from the canal to the chiasm, it lies immediately above the initial supracavernous segment of the ICA. This vessel may compress the optic nerve from below, elevating it against the underline periforaminal dura, anterior clinoid, and anterior cerebral artery (2, 3).

CT is less sensitive to evaluate the intracranial optic nerve segment and its relation with the ICA. Several studies have emphasized that thin-section, MRI coronal image is the optimal protocol to evaluate the anatomic relation between intracranial optic nerve segment and the ICA (4-7). Moreover, compressive optic neuropathy is not necessarily related to sclerotic degeneration, and may occur by contact with developmental abnormality of ICA (3), ectatic dilation (dolichoectasia, fusiform enlargement, or fusiform aneurism) (4), and even by normal ICA (4, 5). In fact, Golnik et al (6) studied patients with unexplained optic neuropathy and found a smaller distance between optic nerve and ICA in the affected side, and suggested that the intimate relation between theses two structures could be important in the development of unexplained optic neuropathy.

Fig. 2 - *Left: Left eye (LE) perimetry (Humphrey SITA 24-2) showing severe and diffuse visual field defects. Right: Optical coherence tomography testing revealed a significant retinal nerve fiber layer thinning in inferior, nasal, and temporal quadrants of LE.*

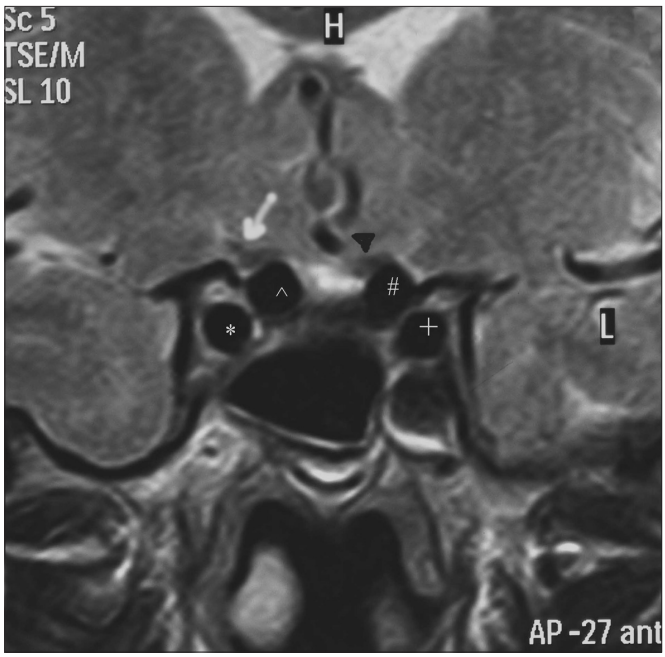

Fig. 3 - *Right ICA (*) and supraclinoid segment of ICA (^). Normal right optic nerve (white arrow) placed in a lateral position. Normal intracavernous segment of left ICA (+). Intracranial left optic nerve (black arrowhead) is compressed by the supraclinoid segment of the ICA (#). Left optic nerve is thin and displaced medially (to view it clearly, compare with the lateral position of noncompressed contralateral optic nerve).*

Jacobson et al (5) showed that anatomic compression of the intracranial optic nerve by the supraclinoid carotid artery is relatively frequent in asymptomatic patients, so that identification of this relationship may not be clinically significant unless no other plausible mechanism of optic nerve injury exists. Factors other than contact between theses two structures must be implicated so that some patients manifest symptomatic compression while others do not.

All types of visual field defects may occur in optic neuropathy caused by compression of the ICA, and a nonspecific visual field generalized depression, as in our clinical case, is not unusual. Longstanding compression of the intracranial optic nerve may also produce a nerve fiber bundle pattern of visual field loss and excavation of the optic disc, which are two signs consistent with glaucoma. However, other features atypical for glaucoma such as normal IOP, pallor of the neuroretinal rim, and impairment of visual acuity alert to the possibility of compressive optic neuropathy (4). Recently, a higher percentage of optic nerve compression by ICA has been reported in normal tension glaucoma patients specially in patients with cup to disc ratio greater than 0.7 suggesting a possible role in this entity (7). In our case, IOP and cup to disc ratio were not increased. Moreover, the slow and painless progressive monocular visual loss and the lack of other plausible mechanisms of optic nerve injury make clear a causal relationship between left optic nerve compression by ICA and ipsilateral visual loss.

In addition to visual loss and optic disc pallor, our patient had ipsilateral optociliary shunt vessels. This triad of signs was described by Frisén et al in a patient with a sphenoorbital meningioma (1), but also occurs in patients with optic nerve gliomas and other orbital lesions that produce chronic optic nerve compression. Other diagnostic implications of optociliary shunt vessels include congenital origin, glaucoma, hyaloid bodies of the optic nerve head, dysthyroid optic neuropathy, papillophlebitis, and chronic papilledema (8-10).

Optociliary veins are a pre-existing shunt system that allows retinal venous blood to bypass the central retinal vein and exit from the orbit via the choroidal circulation and its anastomoses. It is postulated that chronic compression of the intraorbital portion of the optic nerve produces gradual obstruction of the central retinal vein, thus preventing the normal passage of venous blood from the retina through the central retinal vein. However, optociliary shunt vessels are not exclusive for compressive lesions of the optic nerve and they may appear in any condition in which central retinal venous return is disturbed. In fact, the most common cause of retinochoroidal shunt veins is central retinal vein occlusion.

The mechanism explaining optociliary shunt vessels observed in our case remains unclear. We suggest an indirect mechanism of hampering central retinal venous return.

In compressive optic neuropathy by ICA, many patients have pale optic disc when they are first examined, but some patients have optic disc edema. Newman proposed that disc swelling in these patients is probably caused by ectasia-related thrombotic or embolic impair of the short posterior ciliary arteries, thus producing a true anterior ischemic optic neuropathy (11). In other words, chronic compression of the optic nerve may also compromise regional perfusion, producing ischemic injury superimposed on compressive injury of optic nerve fibers.

In addition, anterior ischemic optic neuropathy associated with central retinal vein occlusion has been described. In these cases, authors hypothesize that a compression of

the retinal vein by the swollen optic nerve could be the predisposing factor (12, 13). In a similar way, we hypothesize that in our case disc swelling, occurring before optic disc became pale, disturbed central venous return, explaining optociliary shunt vessels formation.

The treatment approach in this clinical entity generally is conservative. Despite isolated examples of visual improvement, it is not clear that neurosurgical decompression offers reversal of visual loss or any protection from further visual deterioration.

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