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Neurovascular Compression of the Optic Nerve

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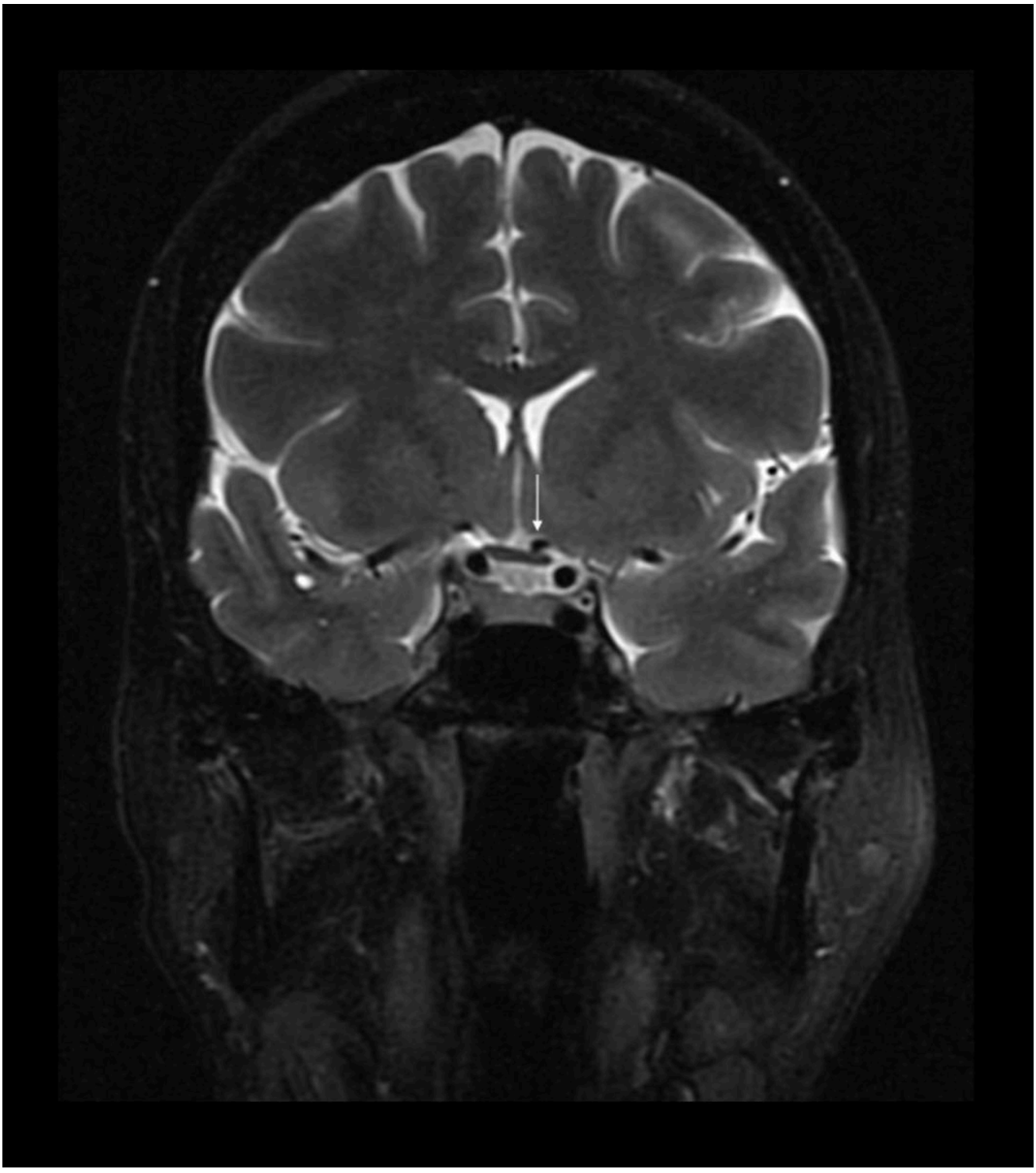
A 52-year-old right-handed woman presented to an ophthalmologist after referral from an optometrist for abnormal findings on an optometric examination. She denied symptoms of visual loss, eye pain, or headaches. She also denied focal neurologic symptoms other than occasional stumbling.

On fundoscopic examination, the ophthalmologist found evidence of low-tension glaucoma, left afferent pupillary defect, and bilateral optic atrophy, with the latter more pronounced on the left. The patient's visual acuity was 20/25 in the right eye and 20/20 in the left eye. Travoprost ophthalmic solution, 0.004%, 1 drop at bedtime, was prescribed for use in her left eye. Travoprost is an analogue of prostaglandin that lowers intraocular pressure by increasing the outflow of aqueous fluid.[1]

The patient was referred to a neurologist for workup of optic nerve atrophy. At the neurology visit, the patient had normal vital signs and normal general medical examination findings. Neurologic examination findings were also normal except for a left afferent pupillary defect. Visual fields were intact to manual confrontation. Visual acuity and cranial nerve function were intact. Motor strength and sensation were normal bilaterally throughout the face and body. Folate and vitamin B_{12} levels were normal.

Diagnostic tests. Magnetic resonance imaging (MRI) of the orbits with and without gadolinium demonstrated the left A1 segment of the anterior cerebral artery to be in contact with and deforming the superior surface of the cisternal segment of the prechiasmatic left optic nerve (**Figure**). The cisternal left optic nerve was also smaller in diameter compared with the contralateral side. The remainder of the optic nerve sheath complexes and intraorbital contents were unremarkable with no acute abnormality. There was no evidence of an underlying mass lesion or other source of compression.



Treatment. Because her visual acuity and visual fields were not impacted, the patient's condition was managed conservatively with periodic ophthalmologic follow-up visits. Travoprost eye drops were continued in her left eye for the treatment of normal-tension glaucoma (NTG).

Discussion. Optic nerve compression (ONC) from normal intracranial internal carotid arteries (ICAs) has been suggested as a cause of visual-field defects in patients with optic neuropathy.[2-7] However, the mechanism of optic nerve injury by ICA compression has been controversial and temporarily lost endorsement for several reasons.[8] The anatomic relationship between the intracranial optic nerve and supraclinoid ICA could not be delineated using imaging techniques prior to MRI. Neurovascular decompression frequently did not improve vision, and neurosurgical exploration was associated with higher rate of morbidity and mortality than conservative management. Many of the cases of occult optic neuropathy were attributed to ischemic optic neuropathy rather than ICA compression. Additionally, Jacobson and colleagues showed that anatomic compression of the intracranial optic nerve by the supraclinoid ICA occurs frequently and by itself may not be clinically important.[9]

However, there are cases of occult or progressive optic neuropathy for which no explanation other than neurovascular compression exists.[10] In particular, in patients with NTG, ONC by the ICA has been suggested as a cause of or a risk factor for optic nerve damage.[11] Compression by a dolichoectatic basilar artery has also been described.[12]

Patients with compressive optic neuropathy and atypical glaucoma can have overlapping symptoms.[2,5,7,13-15] Patients with compressive optic neuropathy show loss of central acuity associated with central and concentric scotomas. Most patients with ONC have symptoms atypical for glaucoma alone, including a decrease in visual acuity, abnormal color vision, central scotoma, pallor of the neuroretinal rim, and unusual optic nerve cupping. Patients with NTG do not routinely undergo neuroimaging unless atypical findings are present, such as poor correlation between the pattern or amount of visual field loss and optic cupping.[11]

Neurovascular decompression of the optic nerve has been rarely reported in the literature.[4,16,17] The patients described in the literature typically did not have NTG, as our patient did, but demonstrated improvement in visual symptoms postoperatively. To date, no study has been performed regarding neurovascular decompression of the optic nerve in the setting of NTG. Ogata and colleagues do not directly advocate for neurosurgical decompression despite their conclusion that ONC is a cause of or a risk factor for optic nerve damage in patients with NTG, since ICA compression of the optic nerve is so common.[11]

Compressive optic neuropathy is usually related to aneurysms, meningiomas, or other types of tumors.[13,14,18] Other etiologies include ischemic optic neuropathy related to hypertension and diabetes, and demyelinating diseases such as multiple sclerosis and neuromyelitis optica.

Neurovascular compression of the optic nerve should be considered in patients with optic nerve injury, particularly in the setting of atypical glaucoma. MRI in the coronal plane through the orbits can be useful for delineating the relationship of the optic nerve with regard to adjacent arterial structures.

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