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Neurovascular Compression of the Optic Nerve Causing Peripheral Vision Loss

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An 85-year-old man presented to an outpatient clinic with concern for a progressive loss of peripheral vision in his left eye.

History. The patient's history was remarkable for hypertension, hyperlipidemia, aortic valve stenosis, and cataracts. The patient has no history of alcohol, tobacco or drug use. He was currently on aspirin, benazepril, and simvastatin.

Physical examination. Physical examination findings were prominent for a left carotid bruit but were otherwise unremarkable. Likewise, neurological examination findings were unremarkable. However, considering the possibility for a cerebrovascular accident, carotid Doppler ultrasonography was ordered, followed by magnetic resonance imaging (MRI) of the brain and the orbit with and without contrast.

Diagnostic tests. Carotid Doppler ultrasonography findings were unremarkable for blockage or severe stenosis bilaterally. Brain MRI showed cortical atrophy and mild white-matter T2 signal changes that were consistent with mild chronic small-vessel ischemic disease, but findings were otherwise normal.

MRI of the orbit was significant for a flattening of the left optic nerve anterior to the optic chiasm, with a prominent flow void from the left anterior cerebral artery (ACA) crossing over the superior aspect of the nerve. Additionally, a mild mass effect was seen inferiorly from the internal carotid artery (ICA), contributing to the compression of the left optic nerve (**Figures 1-3**)

artery (ICA), contributing to the compression of the left optic nerve (**Figures 1-3**).

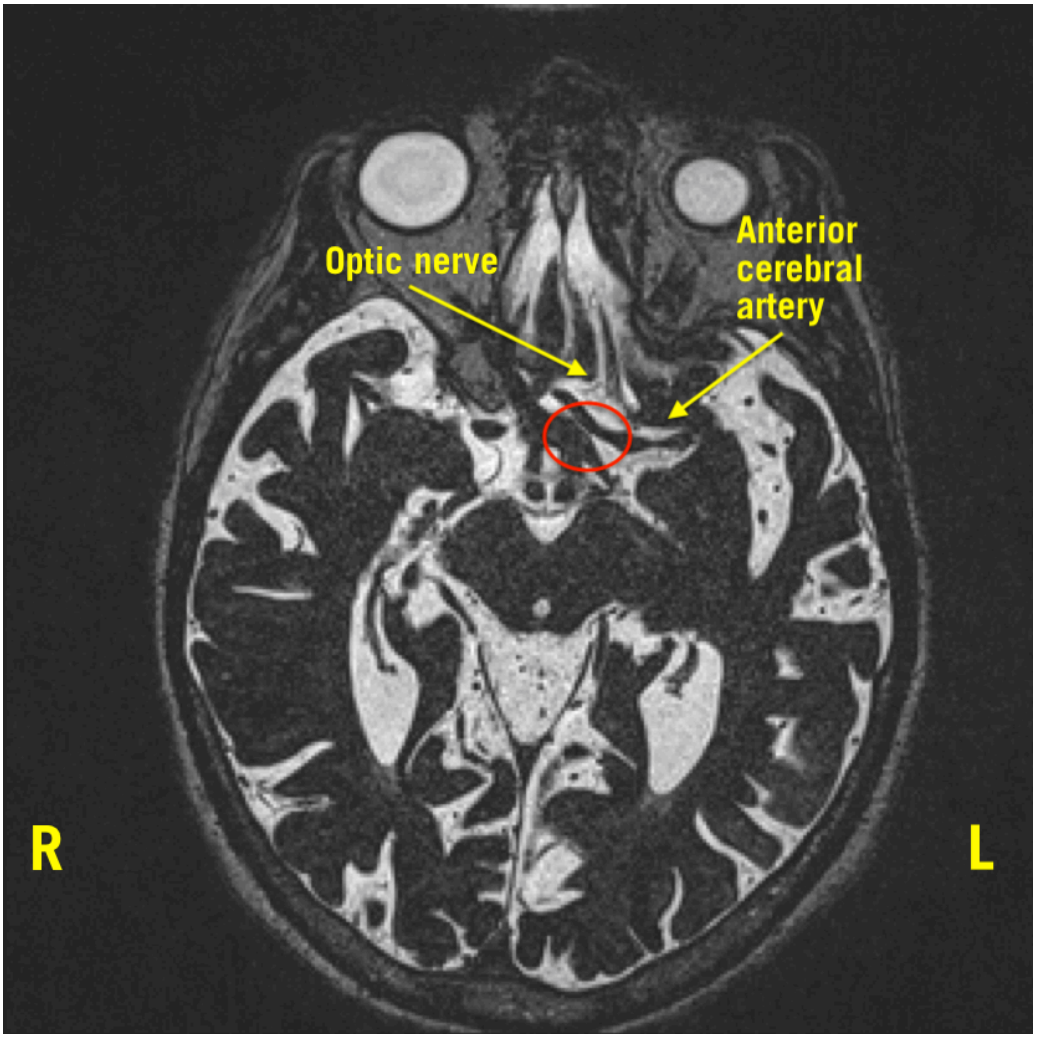

Figure 1. *Thickened left ACA crossing on top of the optic nerve, obliterating its visualization. The red circle notes compression.*

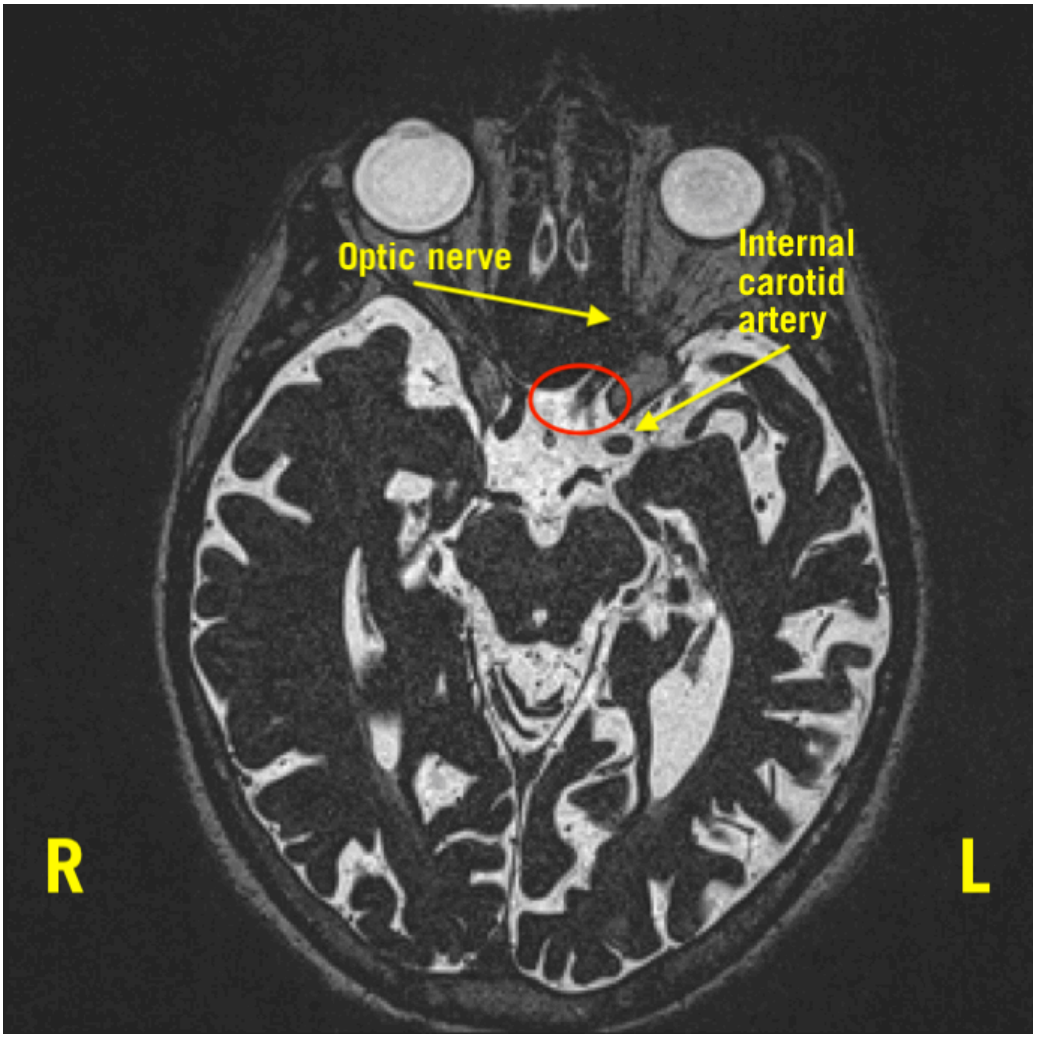

Figure 2. *Harder to appreciate, the ICA was also compressing the optic nerve inferiorly. The red circle notes compression.*

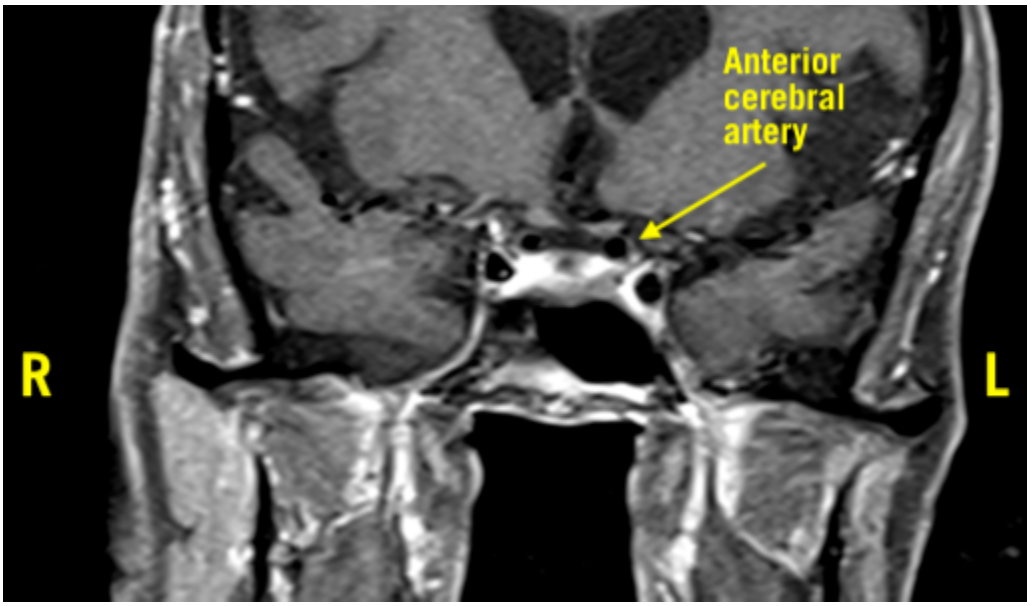

Figure 3. *Prominent flow void from the left ACA crossing over the superior aspect of the optic nerve.*

Follow-up. The results of the workup were discussed with the patient at a follow-up visit, during which his peripheral vision loss was attributed to pressure from the left ACA on his left optic nerve. Prior to the follow-up visit, based on the results of the orbit MRI, a consultant neurosurgeon advised against intervention due to the increased risk of an iatrogenic stroke secondary to a stiff ACA. The patient declined an offer of a referral for a second opinion. At this follow-up visit, the possibility of worsening vision loss without intervention was discussed with the patient.

Discussion. The visual conduction pathway involves the movement of light past the corneal surface, terminating on the retina in the posterior eye.[1] The structural components between the 2 include the aqueous humor, lens, and vitreous humor, contributing to light refraction that stimulates retinal transformation of the visual signal. Transduction of the electrochemical signal is mediated by a G protein-coupled receptor mechanism, which is then transmitted by the optic nerve through the optic chiasm to the occipital lobe for integration.

Progressive vision loss in the elderly population has been well attributed to pathological sequelae of diabetes (retinopathy) and glaucoma, as well as age-related macular degeneration. Oxidative stress has been implicated in eye-related pathology due to aging.[2] Additionally, acute vision loss can result from ischemic changes secondary to a cerebrovascular accident, which in many cases becomes permanent.

In comparison, neurovascular compression of the optic nerve is a rare presentation of vision loss in the elderly. Trigeminal neuralgia, hemifacial spasm, vestibulocochlear neuralgia, and glossopharyngeal neuralgia are more common compression occurrences.[3] Typically, the neurovascular compression involves the cisternal segment of the cranial nerve and localizes at the transitional zone between central myelination with oligodendrocytes and peripheral myelination with Schwann cells.

Optic nerve compression has rarely been reported, with sequelae of vision loss and neuropathy secondary to unruptured aneurysmal lesions of either the ACA or ICA, as well as ectatic or elongated manifestations of these vessels.[4-12] The supraclinoid portion of the ICA and A1 segment of the ACA have been shown to have a greater pathological role in such cases. Rarities in the vasculature at the skull base have also been implicated in compression of both the optic nerve and optic chiasm.[13] A rarer anomaly is compression by both the ICA and the ACA, sandwiching the optic nerve,[14] such as in our patient's case.

The etiology of the underlying mechanisms behind neurovascular compression is controversial, lacking objective evidence even in the common cranial nerves affected, such as in trigeminal neuralgia.[15] However, within trigeminal neuralgia, the most popular theories are attributed to either disease (vascular disease, multiple sclerosis, diabetes mellitus, and others), direct injury

at the root entry zone (eg, arteriovenous malformation or due to a schwannoma), or polyetiological origin (multiple factors evoking demyelination and dystrophy).

In the case reported here, the patient's history was remarkable for hyperlipidemia and hypertension, both of which have been implicated in causing arteriosclerosis.[16] The pathogenesis of arteriosclerosis primarily affects the intimal coat, with lipid accumulation and fibrous thickening leading to calcification and impairment of blood supply. Nevertheless, local vessel wall composition and focal mechanical or hydrodynamic forces contribute to susceptibility of arteriosclerosis in one area over another. Given that the patient's neuroanatomy was otherwise unremarkable, arteriosclerotic changes of the ACA and ICA may explain their contribution to the compression of the optic nerve. The unpredictable nature of where arteriolosclerosis may occur can help explain the vision loss in this patient, especially its unilateral focal component. Compression of the optic nerve in this case, due to progressive stiffening of the left ACA, led to compression on the portion of the nerve conveying information from the left temporal field.

Considering the patient's age and the delicate anatomical relationship of the optic nerve, optic chiasm, ACA, and ICA, surgical intervention carried the risk of iatrogenic stroke and thus was not pursued. However in younger patients with optic nerve compression due to aneurysmal lesions, surgical clipping methods have been pursued.[17,18] The mixed success in these cases further highlights the high risk associated with intervention and its utility only in an ill-defined group of patients.

In summary, neurovascular compression of the optic nerve should be considered with presentation of progressive vision loss. Further workup must elucidate the predisposition to such an occurrence, given that intervention is complicated, especially among the elderly.

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