

Anomalous Course of the Internal Carotid Artery Resulting in Optic Nerve Compression

Danielle Vance Taylor, OD
Rena Cappelli, OD, MPH, FAAO
Leah Wiedemann, OD, FAAO
 Department of Veterans
 Affairs Jacksonville VA
 Outpatient Clinic

Abstract

Compression of the optic nerve by the internal carotid artery can occur due to their proximity as the artery exits the cavernous sinus. This report details a case of unilateral compressive optic neuropathy resulting from an anomalous course of the internal carotid artery. It presents optical coherence tomography scans, Humphrey visual field results, and magnetic resonance images, and reviews other causes of optic neuropathy that can have a similar clinical presentation.

KEYWORDS:

optic neuropathy, internal carotid artery, optical coherence tomography, Humphrey visual field, magnetic resonance imaging

INTRODUCTION

The optic nerve and internal carotid artery (ICA) are in close proximity as the artery exits the cavernous sinus.¹ This can result in compression of the optic nerve, either through an atypical course of the ICA or an increased diameter of the ICA due to systemic vascular comorbidities. Although contact between the ICA and the optic nerve is common, compression of the optic nerve by the ICA resulting in nerve damage is infrequent. Therefore, more prevalent causes of damage are often considered first when a patient presents with signs and/or symptoms suggesting an optic neuropathy. A review of these differential diagnoses will delineate their distinct features and highlight ICA compression as an additional consideration when faced with otherwise unexplained optic nerve damage. This report describes a case of unilateral compressive optic neuropathy resulting from an anomalous course of the internal carotid artery. It presents optical coherence tomography scans, Humphrey visual field results, and magnetic resonance images, and reviews other causes of optic neuropathy that can have a similar clinical presentation.

CASE REPORT

A 59-year-old African American man presented for an annual dilated eye exam with no ocular complaints. Systemic and ocular health were unremarkable. Entering visual acuity with spectacle correction was 20/20 in both eyes. Pupils were equal, round, and reactive to light without afferent pupillary defect. Intraocular pressures were 18 mmHg in both eyes with applanation tonometry. Anterior segment exam with slit lamp was unremarkable. Evaluation of the optic nerve on dilated exam demonstrated the presence of pallor superiorly in the right eye. No disc edema or retinal hemorrhaging was observed. The optic nerve in the left eye was pink without evidence of pallor. Significant cupping was present in both eyes with a cup to disc ratio of 0.7 in the right eye and 0.6 in the left eye. Baseline Humphrey visual field testing and Heidelberg optical coherence tomography of the retinal nerve fibre layer (OCT RNFL) were performed.

A 24-2 visual field test revealed a dense inferior hemifield defect in the right eye and a full visual field in the left eye (Figures 1 and 2). Retinal nerve fibre layer OCT demonstrated thinning superiorly with mild thinning nasally and temporally in the right eye, and mild thinning nasally in the left eye (Figures 3 and 4).

Figure 1: 24-2 Humphrey visual field showing a dense inferior hemifield defect in the right eye

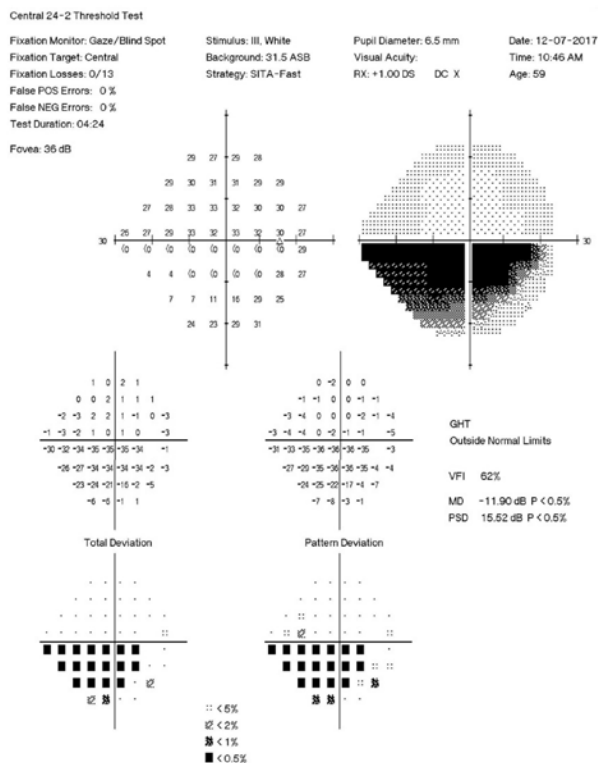


Figure 2: 24-2 Humphrey visual field showing a full visual field in the left eye

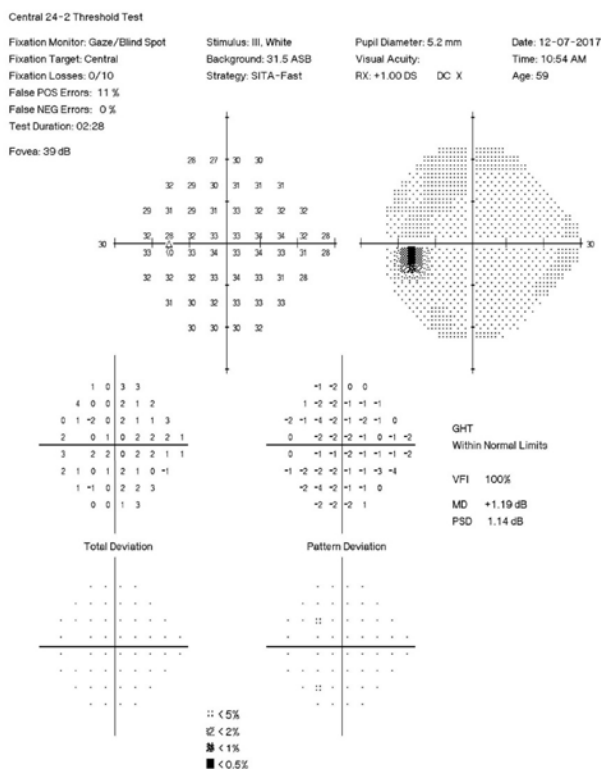


Figure 3: Retinal nerve fibre layer OCT demonstrating thinning superiorly with mild thinning nasally and temporally in the right eye

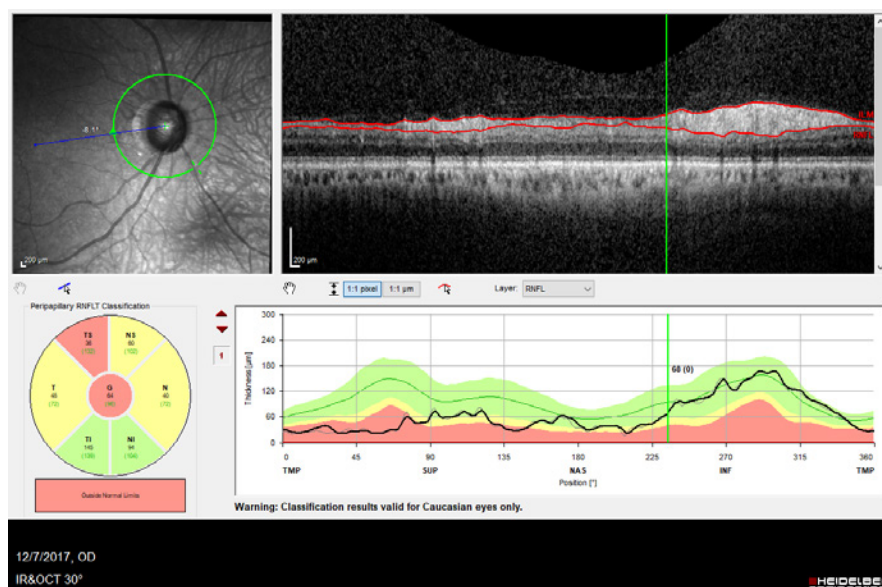
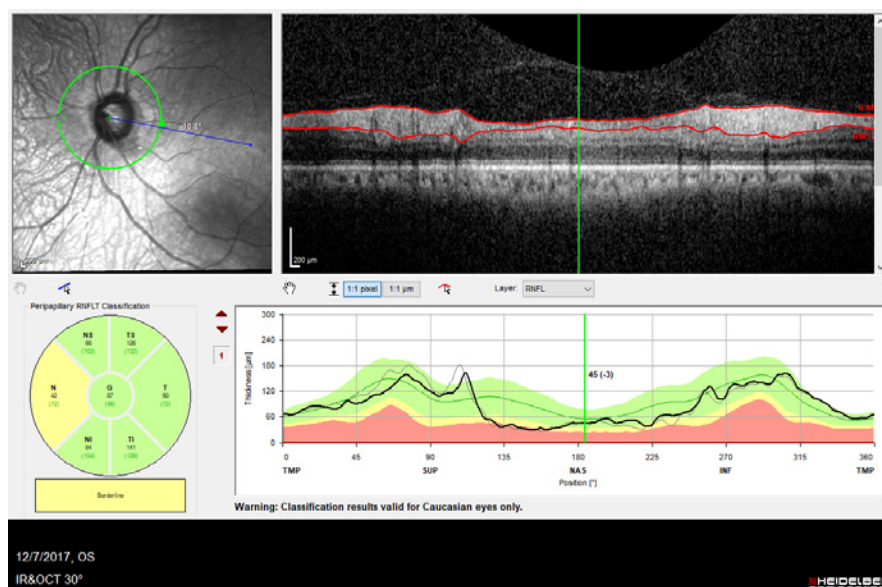


Figure 4: Retinal nerve fibre layer OCT demonstrating mild thinning nasally in the left eye



No vascular risk factors, such as diabetes mellitus or hypertension, were present. All symptoms associated with giant cell arteritis were denied. Similarly, any history of trauma was denied.

Lab work was ordered to exclude alternate inflammatory, autoimmune, and nutritional etiologies. Lab work included complete blood cell count, C-reactive protein, erythrocyte sedimentation rate, platelets, Lyme titer, anti-nuclear antibodies, angiotensin converting enzyme, folate, vitamin B12, IGG/IGM antibodies, and rapid plasma reagin. All lab results were unremarkable.

Magnetic resonance imaging (MRI) of the brain and orbits was ordered with and without contrast to evaluate for optic nerve compression or a space-occupying lesion. The brain MRI revealed right optic nerve flattening due to a tortuous course of the internal carotid artery (Figures 5 and 6).

Figure 5: Brain MRI revealed right optic nerve flattening from contact with the internal carotid artery

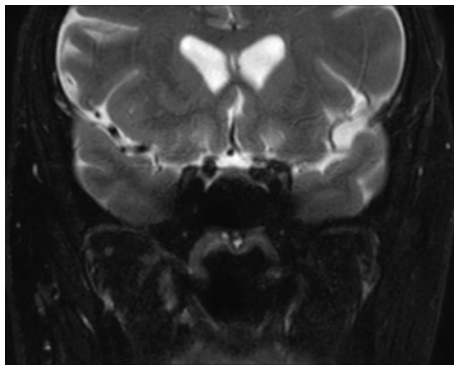
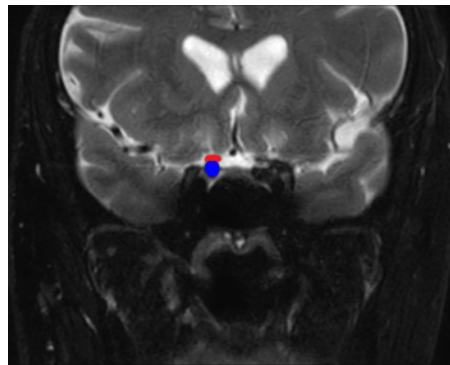


Figure 6: Brain MRI with the right optic nerve in red and the internal carotid artery in blue

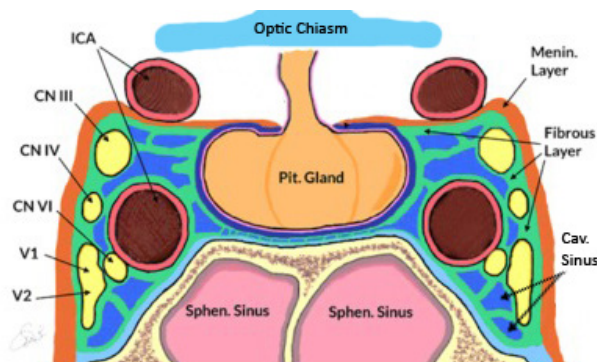


DISCUSSION

Compressive optic neuropathy is most often attributable to a space-occupying lesion such as a tumor or aneurysm.² Common compressive lesions include pituitary adenoma, intracranial aneurysm of the anterior communicating artery or the internal carotid artery, orbital or intracranial meningioma, craniopharyngioma, and glioma.^{3,4} Though uncommon, vascular compression of the optic nerve by the internal carotid artery can result in optic nerve damage and visual field defects.^{2,5} Presenting visual field defects are most often arcuate scotomas and nasal steps, representative of optic nerve fibre bundle injuries, but central scotomas can also be associated.⁵ The mechanism of damage is theorized to be either direct compression of the nerve fibres or ischemia due to occlusion of the vessels supplying the nerve.²

The internal carotid artery (ICA) courses upward through the neck and enters the skull through the carotid canal at the base of the temporal bone.⁶ Once through the canal, it passes through the cavernous sinus.⁶ Although a detailed review of the cavernous sinus is beyond the scope of this paper, a brief review is warranted due to its associated pathological processes. The cavernous sinus is a group of veins located on the lateral sides of the pituitary gland.⁷ The ICA courses medially through the cavernous sinus while cranial nerves III-V are lateral to the ICA (Figure 7).^{7,8} Due to the proximity of the pituitary gland to the cavernous sinus, up to 10% of pituitary adenomas invade the cavernous sinus.⁷ As the ICA exits the top of the sinus through the proximal dural ring, it is bordered medially by the optic nerve and laterally by the oculomotor nerve.^{1,6} This portion of the ICA, as it transitions from the cavernous sinus to the subarachnoid space, is known as the clinoid segment, from which the ophthalmic artery arises.^{1,6} Given the close proximity of the optic nerve and the internal carotid artery, an anomalous course or an enlargement of the ICA could result in compression of the nearby optic nerve.⁹ In a similar fashion, other cranial nerves such as the trigeminal, facial, and abducens have been known to sustain damage from an aberrant course of intracranial blood vessels such as the anterior inferior cerebellar artery and superior cerebellar artery.^{10,11,12} Thus, trigeminal neuralgia and hemifacial spasm are often attributed to vascular compression of cranial nerves.^{12,13}

Figure 7: Illustration identifying the relationship between the cavernous sinus, cranial nerves III-V, ICA, pituitary gland, and optic chiasm.⁸



Magnetic resonance imaging examines soft tissue and allows for noninvasive evaluation of the relationship between the optic nerve and the ICA, while concurrently assessing for the presence of a space-occupying lesion or other source of optic nerve compression. Though not routinely ordered to evaluate optic nerve damage, a magnetic resonance angiogram would more clearly delineate the relationship between the ICA and optic nerve and could be considered if ICA compression is suspected.

A study by Jacobsen et al. demonstrated that the optic nerve and ICA are frequently in contact with each other in asymptomatic patients.¹³ However, actual compression of the optic nerve by the ICA is rare, especially unilaterally.¹³ The risk of compression is directly related to the diameter of the carotid artery; dolichoectasia, or enlargement, of the ICA (correlated with hypertension and diabetes) makes compression more likely.^{9,13}

In cases of progressive vision loss, neurosurgical decompression procedures can be considered in an effort to halt continued visual decline. While not studied in a randomized clinical trial, review of the literature provides examples of decompression through craniotomy relieving the optic nerve from compression by the ICA, resulting in improvement in vision if axonal atrophy has not already occurred.^{9,13}

Compressive optic neuropathy has a presentation similar to other conditions characterized by optic nerve damage, including ischemic optic neuropathy, optic neuritis, traumatic optic neuropathy, and toxic-nutritional optic neuropathy. These were considered and subsequently ruled out during the exam based on the patient's medical and ocular history, symptoms, and lab testing.

Ischemic optic neuropathy (ION) is characterized by a sudden, painless loss of vision accompanied by a relative afferent pupillary defect and visual field defect.¹⁴ There are two subtypes of ION: anterior and posterior, differentiated by the presence of visible optic nerve swelling in the former. Anterior ION is more common, comprising up to 90% of ischemic optic neuropathies.¹⁵ Ischemic optic neuropathy is also divided further based on etiology, the three primary types are non-arteritic, arteritic, and perioperative. Non-arteritic ION is often associated with vascular risk factors such as diabetes mellitus, hypertension, hypercholesterolemia, or obstructive sleep apnea.¹⁴ Additionally, non-arteritic ION occurs most often in optic discs with small or absent cups, suggesting that mechanical risk factors associated with the structure of the optic disc may also play a role.¹⁶ Arteritic ION is usually associated with arterial inflammation from giant cell arteritis (GCA) and is often accompanied by symptoms of jaw claudication, scalp tenderness, headache, fatigue, low-grade fever, polymyalgia, and acute loss of vision.¹⁴ The presence of GCA will also result in elevated erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) levels, and platelet count.¹⁴ Perioperative ION occurs during a non-ocular surgical procedure. Though both anterior and posterior ION have been reported in association with many different types of surgical procedures, perioperative ION is most commonly experienced during spinal surgeries.¹⁷ The risk of perioperative ION is increased if the surgical procedure is of prolonged duration (> 6.5 hours), results in substantial blood loss (44.7% of blood volume), or results in secondary anemia.¹⁷ In our case, the patient did not have any vascular risk factors, symptoms of GCA, or prior spinal surgical procedures. Given the potential vision and life-threatening consequences of untreated GCA, and the possibility of an occult GCA absent of constitutional symptoms, precautionary lab work was ordered. However, the results indicated normal ESR, CRP, and platelet levels. Therefore, ischemic optic neuropathy was ruled out as the cause of our patient's findings.

Optic neuritis generally manifests in females under 50 years of age and is characterized by painful unilateral loss of vision of varying severity.¹⁴ No pain or loss of visual acuity was seen in our patient, and he did not fit the typical demographic profile of patients with optic neuritis.

Traumatic optic neuropathy was also a consideration but was ruled out based on the absence of a history of trauma, particularly to the head, orbit, or eye.

Lab work was ordered to exclude alternate inflammatory, autoimmune, and nutritional etiologies. Lab testing included a complete blood cell count with platelets, C-reactive protein, erythrocyte sedimentation rate, angiotensin converting enzyme, Lyme titer, rapid plasma reagin, anti-nuclear antibodies, IgG/IgM antibodies, folate, and vitamin B12. C-reactive protein and erythrocyte sedimentation rate detect inflammation which, when elevated, could be associated with giant cell arteritis.¹⁴ Angiotensin converting enzyme regulates blood pressure and is increased in sarcoidosis, which can cause inflammatory optic neuropathy.¹⁸ Lyme titer detects antibodies that would indicate infection with the bacteria *Borrelia burgdorferi* associated with Lyme disease.¹⁹ This can result in optic neuritis and ischemic optic neuropathy.¹⁹ Antibodies for syphilis are checked with the rapid plasma reagin because this condition can also be associated with inflammatory optic neuropathy or atrophy.²⁰ The anti-nuclear antibody test detects autoantibodies which could

indicate the presence of autoimmune disease such as systemic lupus erythematosus possibly leading to autoimmune optic neuritis.^{21,22} IgG and IgM antibodies fight infection and reduced levels can indicate immunodeficiencies. Lastly, folate and vitamin B12 levels check for nutritional deficiencies. All lab results were unremarkable.

Drugs, toxins, and nutritional deficiencies can also result in optic nerve dysfunction, often bilateral with simultaneous onset and symmetric presentation.³ The patient had no history of substance abuse or nutritional deficiency. Review of the patient's medical history confirmed no current or prior use of medications commonly associated with optic neuropathy, including ethambutol, amiodarone, and methotrexate.³

The patient in this case was diagnosed with optic neuropathy due to compression by the internal carotid artery. He was also considered a normal tension glaucoma suspect. Monitoring with annual dilated fundus exams, visual field testing, and OCT RNFL has demonstrated stable findings for four years. Repeat MRI may be considered should any progression be suspected. Surgical decompression is not recommended at this time given the stability of the findings and the fact that the patient is asymptomatic.

This case provides an additional consideration when evaluating the source of otherwise unexplained optic neuropathy. The anatomic proximity of the ICA and optic nerve can result in carotid artery compression of the optic nerve. This report presents a patient with unilateral asymptomatic optic neuropathy due to ICA compression, details the clinical presentation, HVF, OCT RNFL, and MRI results, and reviews alternate diagnoses with similar features. ●

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CONFLICTS OF INTEREST

none

CORRESPONDENCE

Dr. Danielle Vance Taylor, Email: Danielle.VanceTaylor@va.gov

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