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CASE REPORT

Possible Optic Nerve Compression by a Normal-Appearing Internal Carotid Artery: A Three-Patient Case Series

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ABSTRACT

Neurovascular contact between the internal carotid artery (ICA) and the optic nerve is common on neuroimaging in asymptomatic individuals. However, clinically significant compressive optic neuropathy remains rare and under-recognised with limited supporting evidence. We describe three cases of painless and progressive visual loss possibly associated with ICA optic nerve compression and propose a two-hit pathophysiological framework in which neurovascular contact becomes clinically relevant in the presence of secondary susceptibility factors.

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Optic nerve compression; internal carotid artery; neurovascular conflict; neuroimaging; two-hit model

Introduction

Neurovascular compression syndromes comprise a group of neurological disorders where cranial nerves are compressed by adjacent vascular structures.[1] While trigeminal neuralgia and hemifacial spasm are well-established entities, optic nerve compression by the internal carotid artery (ICA) presents a clinico-radiological discordance that remains poorly understood.

Jacobson et al.[2] identified ICA-optic nerve contact in 70% of asymptomatic individuals and compression in 30%. While arterial contact of the anterior visual pathway is frequently observed on neuroimaging, its clinical relevance remains controversial.[3]

Why only a minority of individuals with ICA-optic nerve contact develop optic neuropathy is unknown. Proposed mechanisms include direct mechanical compression, chronic ischemia from compromised perfusion and repetitive pulsatile trauma.[3–5] Symptomatic compression can occur with a normal appearing ICA on routine imaging and without aneurysmal dilatation or dolichoectasia. This suggests that factors beyond apparent arterial morphology contribute to the pathogenesis.[4]

The clinical presentation often mimics other optic neuropathies and is frequently misdiagnosed as normal tension glaucoma.[5] Magnetic resonance imaging (MRI) evidence of optic nerve deformation supports the diagnosis once common etiologies are excluded.

This study aims to improve recognition of possible ICA-related compressive optic neuropathy and proposes a pathophysiological framework integrating neurovascular contact with secondary susceptibility factors, without establishing a definitive causal relationship.

Methods

We retrospectively reviewed patients diagnosed with compressive optic neuropathy attributed to ICA-optic nerve contact between 2018 and 2024. Contact was defined as simple apposition. Deformation required contour abnormalities, specifically indentation, flattening or distortion. Displacement and thinning referred to positional deviation and reduced nerve caliber respectively.

Inclusion required: (1) painless and progressive visual loss without an alternative ocular or systemic cause; (2) MRI evidence of supraclinoid ICA apposition with nerve deformation; (3) absence of aneurysms, dolichoectasia or mass lesions. We excluded patients with demyelinating, inflammatory,

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ischemic, nutritional, toxic or hereditary optic neuropathies.

Clinical data were extracted from medical records. We reviewed all MRI studies to assess the degree of contact and recorded management and follow-up outcomes. All patients provided written informed consent. The study adhered to the principles of the Declaration of Helsinki and was approved by our institutional review board.

Results

Three patients (mean age 66 years, range 63–72) were included in this series. All presented with progressive visual loss previously unrecognised or misattributed to other causes.

Case 1 A 65-year-old woman was referred in 2018 with bilateral visual loss and optic atrophy. Right optic atrophy was noted incidentally in 2005. Visual acuity was hand movements in the right eye

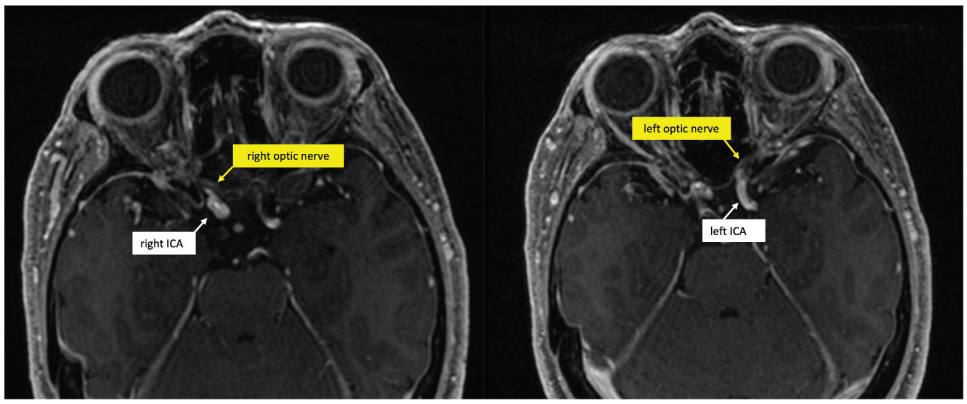

Figure 1. Case 1 axial T1-weighted MRI shows medial displacement and thinning of both optic nerves due to contact with the corresponding internal carotid arteries (ICA).

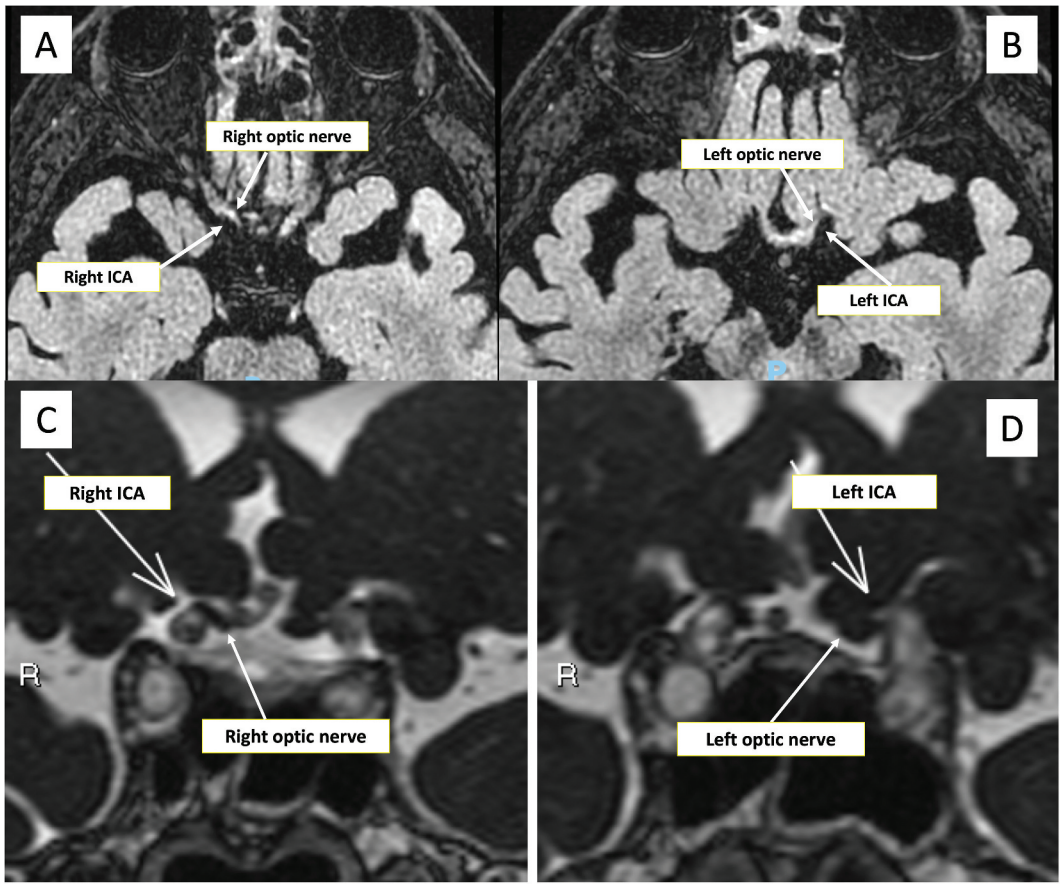

Figure 2. Case 2 MRI. Axial flair (A, B) shows bilateral ICA-optic nerve contact and deformation (arrows). Coronal CISS (C, D) confirms bilateral contact. The right optic nerve is displaced superiorly and medially. The left optic nerve shows contact without displacement.

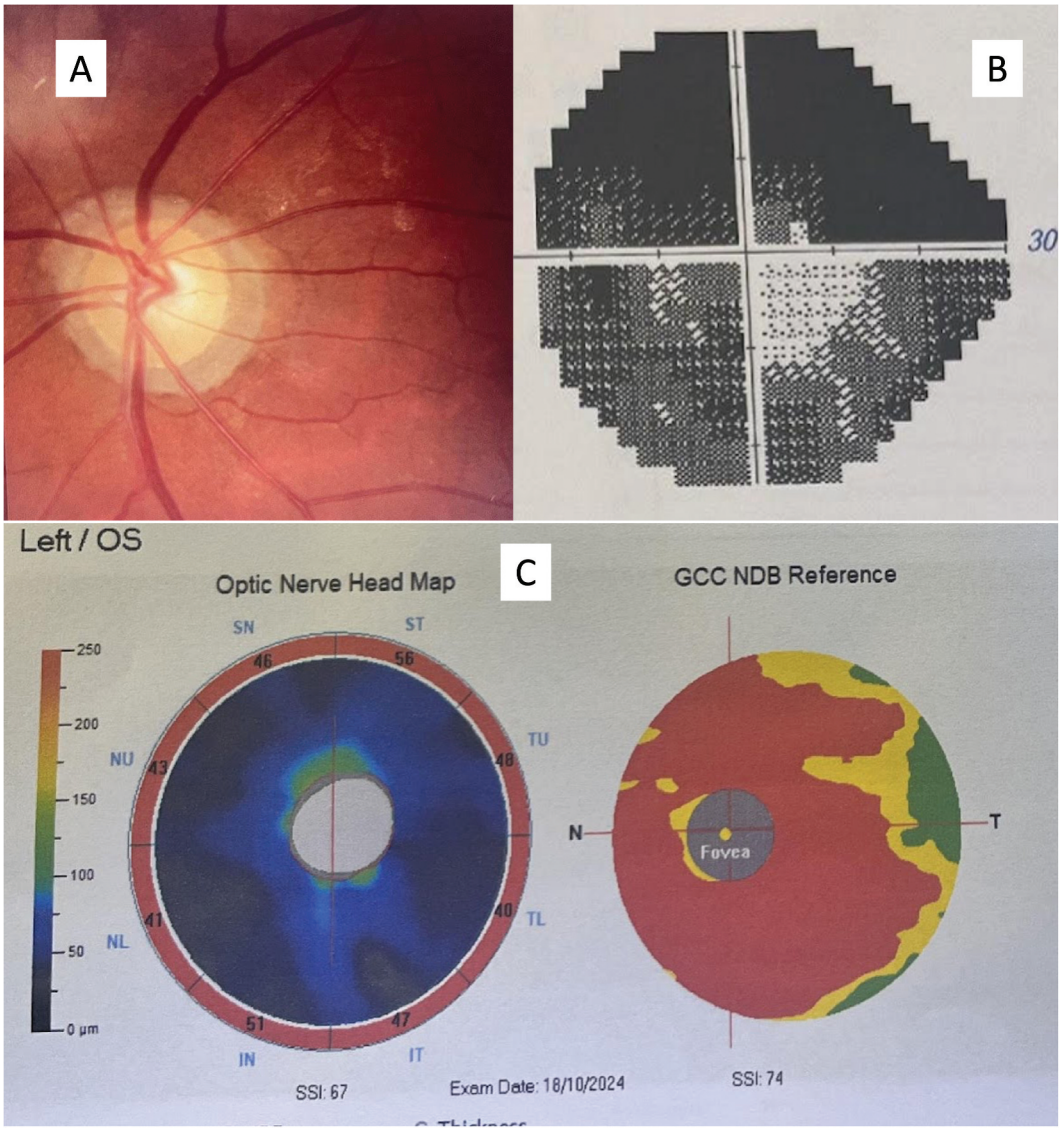

Figure 3. Case 3, left eye structure-function correlation. (A) Color fundus photograph showing optic disc pallor with an increased cup-to-disc ratio. (B) Automated perimetry (Humphrey 24–2) showing peripheral constriction with relative sparing of the inferior nasal paracentral region. (C) OCT demonstrating diffuse peripapillary RNFL thinning and diffuse macular ganglion cell complex loss.

(RE) and 20/25 in the left eye (LE). She had systemic hypertension and hypercholesterolaemia. Painless visual loss in the LE progressed from 2005. By 2018 visual acuity was hand movements in both eyes. Fundoscopy showed bilateral optic disc pallor with enlarged cups. MRI revealed supraclinoid ICA-optic nerve apposition with displacement and thinning (Figure 1). No dolichoectasia, aneurysm or mass was detected. Conservative management was adopted.

Case 2 A 63-year-old man presented in August 2023 with difficulty reading and reversing his vehicle. Medical history included diabetes mellitus and hypercholesterolaemia. Examination revealed count fingers (RE) and 20/80 (LE). Fundoscopy demonstrated bilateral optic disc pallor. Optical coherence tomography (OCT) confirmed retinal nerve fibre layer (RNFL) and ganglion cell complex thinning. Axial MRI showed contact and deformation of both optic nerves (Figure 2A,B). Coronal CISS images confirmed bilateral ICA-optic nerve contact (Figure 2C,D). The right optic nerve was displaced. The left optic nerve showed contact without definite displacement. No dolichoectasia, aneurysm or mass was present. Observation was adopted.

Case 3 A 72-year-old man with a normal examination in November 2023 noticed progressive

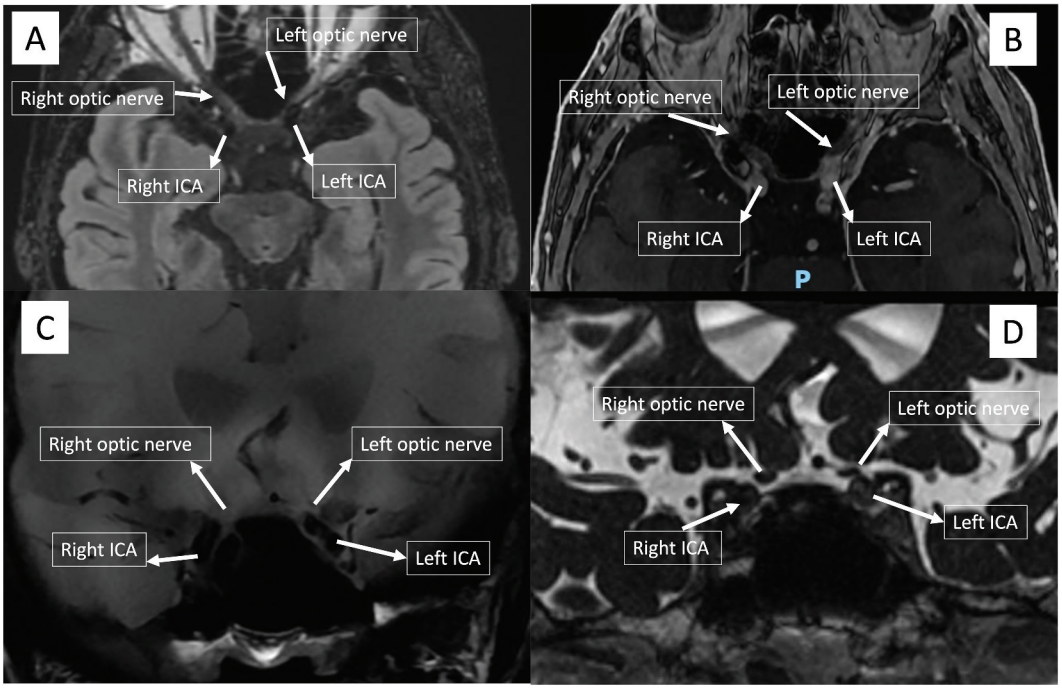

Figure 4. Case 3 MRI (A) axial flair and (B) axial T1 SPGR post-contrast images show bilateral ICA-optic nerve contact, with visible deformation of the left optic nerve only (arrow). (C) Coronal T1-weighted image confirms bilateral contact with distortion of the normal oval contour of the left optic nerve (arrow). (D) Coronal fiesta image demonstrates contact and slight superior displacement of the left optic nerve, with no contact of the right optic nerve.

blurring in the LE in October 2024. He had diabetes mellitus and hypercholesterolaemia. Visual acuity was 20/20 bilaterally with normal intraocular pressures. A left relative afferent pupillary defect was present. The left optic disc showed pallor disproportionate to the degree of cupping (Figure 3A). Automated perimetry (Humphrey 24–2) showed peripheral constriction with relative sparing of the inferior nasal paracentral region (Figure 3B). OCT demonstrated diffuse RNFL thinning and marked macular ganglion cell loss (Figure 3C).

MRI revealed bilateral ICA-optic nerve contact (Figure 4A–D). Left-sided indentation was evident on axial images (Figure 4A,B) with coronal sequences confirming distortion of the normal oval contour (Figure 4C). No dolichoectasia, aneurysm or mass was present. Observation was initiated for a provisional diagnosis of unilateral compressive optic neuropathy.

Discussion

Our patients' clinical profiles align with previously reported series. Mean age was 66 years. Patients presented with painless and progressive visual loss. Intraocular pressure was normal. MRI showed ICA-optic nerve contact with distortion at the symptomatic site. In the Jacobson cohort,[4] mean age was 69 years. Visual decline was typically insidious and associated with saucerlike optic disc excavation. MRI in that study demonstrated optic nerve compression with contour distortion at the supraclinoid ICA contact site.[4] Jain et al.[3] reported a mean age of 71 years and identified deformation of the anterior visual pathway on MRI. Their patients often presented with features atypical of glaucoma.[3]

The causal link between ICA-optic nerve contact and optic neuropathy remains unclear. Jacobson et al.[2] demonstrated that neurovascular contact is a common incidental finding. Contact occurred in 70% of asymptomatic patients and anatomic compression in 30%.[2] This high prevalence in asymptomatic individuals creates a clinicoradiological discordance. Elmalem and Purvin discussed whether this relationship is a true cause of optic neuropathy or merely an associated finding.[6] This discrepancy highlights the need for a pathophysiological model to explain why only a subset of individuals develops visual loss.

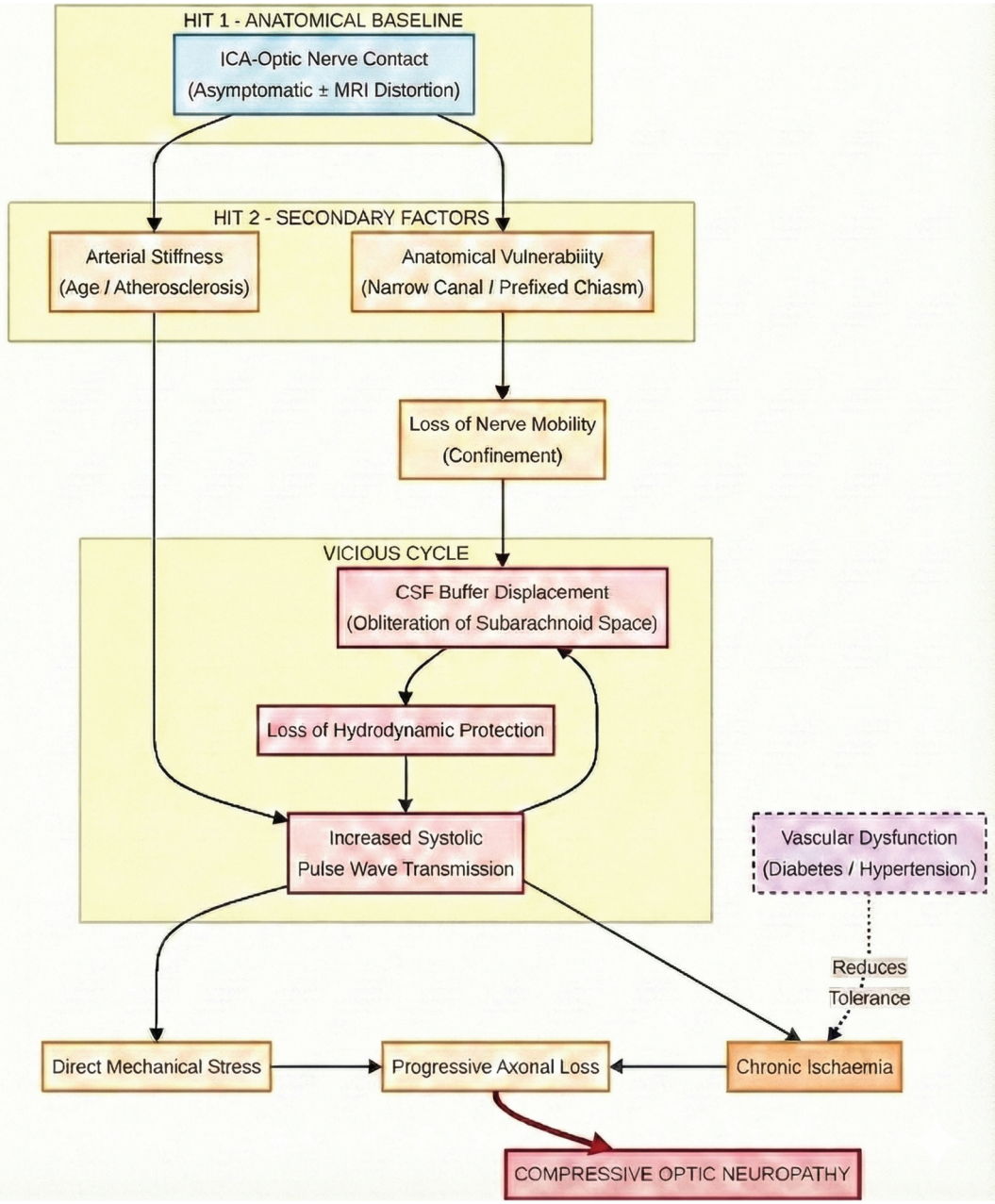

Figure 5. Two-hit framework for ICA-related compressive optic neuropathy. Hit 1 is a preexisting ICA-optic nerve relationship that is often asymptomatic and may progress to MRI distortion. Hit 2 includes arterial stiffening due to age or atherosclerosis and anatomical vulnerability such as a narrow canal or a prefixed chiasm. These factors reduce nerve mobility and may promote CSF buffer displacement with increased pulse-wave transmission in a vicious cycle. Direct mechanical stress and chronic ischemia, amplified by systemic vascular dysfunction such as diabetes or hypertension, may lead to progressive axonal loss and compressive optic neuropathy. ICA, internal carotid artery. MRI, magnetic resonance imaging. CSF, cerebrospinal fluid.

We propose a two-hit model. Hit 1 is the anatomical contact. Arterial wall elasticity and perioptic cerebrospinal fluid hydrodynamic buffering normally mitigate the carotid's pulsatile force.[7] Contact without optic nerve distortion on MRI is likely incidental. Optic nerve distortion may reflect a later stage of contact preceding clinical expression.

The second hit occurs when synergistic insults exceed compensatory reserves. Age and atherosclerosis increase arterial stiffening. This transmits higher mechanical force to the optic nerve with each cardiac cycle.[4,6] Anatomical vulnerabilities like a congenitally narrow canal or a prefixed chiasm immobilise the optic

nerve.[2,4] Immobilisation prevents the lateral displacement that would dissipate pulsatile energy. This creates a vicious cycle. Increased pulsatile force displaces the protective fluid buffer. Mechanical stress eventually impairs regional perfusion and leads to chronic ischemia.[4,6] This may be more relevant in patients with diabetes or hypertension.[3,5] Repetitive mechanical trauma and chronic ischemia cause progressive axonal loss resulting in compressive optic neuropathy. Figure 5 illustrates this pathophysiological framework.

ICA-optic nerve contact is common on MRI. Clinically significant compressive optic neuropathy remains controversial. Our patients presented with painless and progressive visual loss, normal intraocular pressure, optic disc pallor disproportionate to the degree of cupping and OCT evidence of axonal loss. MRI confirmed optic nerve deformation at the carotid contact site. All three patients had systemic risk factors consistent with the second hit including hypertension, diabetes and hypercholesterolaemia. While optic nerve compression by a normal-appearing ICA cannot be entirely excluded, it should be presented as a possible rather than confirmed mechanism.

Study limitations include the small sample size and retrospective design. These findings suggest a two hit model. Contact alone appears insufficient. Secondary factors including arterial stiffening, restricted nerve mobility or vascular vulnerability may contribute to clinical expression.

Author contributions

CRediT: **Frederico Castelo Moura:** Conceptualization, Formal analysis, Investigation, Methodology, Writing – original draft, Writing – review & editing.

Disclosure statement

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References

1. Szmyd B, Sołek J, Błaszczyk M, et al. The underlying pathogenesis of neurovascular compression syndromes: a systematic review. *Front Mol Neurosci*. 2022;15:923089. doi: 10.3389/fnmol.2022.923089.
2. Jacobson DM, Warner JJ, Broste SK. Optic nerve contact and compression by the carotid artery in asymptomatic patients. *Am J Ophthalmol*. 1997;123(5):677–683. doi: 10.1016/S0002-9394(14)71080-3.
3. Jain NS, Kam AW, Chong C, et al. Intracranial arterial compression of the anterior visual pathway. *Neuroophthalmology*. 2019;43(5):295–304. doi: 10.1080/01658107.2019.1566383.
4. 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(10):1994–2004. doi: 10.1016/S0161-6420(99)90414-1.
5. Ogata N, Imaizumi M, Kurokawa H, Arichi M, Takahashi K, Matsumura M. Optic nerve compression by normal carotid artery in patients with normal tension glaucoma. *Br J Ophthalmol*. 2005;89(2):174–179. doi: 10.1136/bjo.2004.047035.
6. Elmalem VI, Purvin VA. Does dolichoectasia of the carotid artery cause optic neuropathy? *J Neuroophthalmol*. 2018;38(3):368–374. doi: 10.1097/WNO.0000000000000653.
7. Killer HE. Production and circulation of cerebrospinal fluid with respect to the subarachnoid space of the optic nerve. *J Glaucoma*. 2013;22(5 suppl 1):S8–S10. doi: 10.1097/IJG.0b013e318293498b.