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Neurovascular compression of the oculomotor nerve presenting with aberrant reinnervation

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ABSTRACT

Purpose: To report a case of neurovascular compression in a patient presenting with ophthalmic evidence of aberrant reinnervation.
Observation: A 68-year-old woman diagnosed with right partial third nerve palsy with aberrant regeneration. Suspicion was based on isolated clinical features of the right eye, including ptosis, upper eyelid elevation on adduction, mydriasis, exotropia, and hypotropia. Magnetic resonance imaging revealed atrophy of the right oculomotor nerve secondary to neurovascular compression from a prominent right superior cerebellar artery.
Conclusion and importance: This case highlights the importance of utilizing Fast Imaging Employing Steady-state Acquisition (FIESTA) for the diagnosis of oculomotor nerve palsy presenting with evidence of aberrant reinnervation.

1. Introduction

Aberrant reinnervation of the oculomotor nerve can be the sole presentation of neurovascular compression, especially in a patient without a history of trauma. We report a case of neurovascular compression in a patient presenting with ophthalmic evidence of aberrant reinnervation secondary to a prominent superior cerebellar artery.

2. Case report

A 68-year-old woman presented to our clinic with a two-year history of diplopia and ptosis of the right upper eyelid. She denied any prior trauma, hypertension, or diabetes, but had a history of Hashimoto's thyroiditis and took synthroid. She also had a history of minimally atypical smooth muscle tumor of the uterus and had undergone total abdominal hysterectomy six years prior. She denied having any localized pain, headaches, vision loss, balance issues, weakness, facial spasms, or dysesthesia. An MRI scan of the brain and orbits performed 18 months prior at an outside facility was reportedly normal.

Her examination revealed uncorrected visual acuities of 20/20 OD and 20/25 OS. Ptosis was present on the right side, marginal reflex distance-1 (MRD-1) was 0 mm on the right and 4 mm on the left. The levator function was 17 mm bilaterally. The pupils were 4 mm on the right and 2 mm on the left and the right pupil showed minimal reactivity to light or accommodative effort but no afferent defect. The right pupil diameter remained unchanged under bright and dim light. There were 35 prism diopters (PD) of exotropia in primary gaze, 60 PD in left gaze, and 5 PD of right gaze. As well, a 6 PD of right hypotropia in primary gaze, 10 PD in right gaze, and 4PD in left gaze (Fig. 1). Elevation of the right eye was limited more than depression, and adduction was markedly reduced. The right upper eyelid was noted to elevate on attempted adduction of the right eye (Fig. 1). The ductions of the left eye were normal. There was no evidence of blepharospasm or hemifacial spasm. Anterior segment and funduscopic examination were unremarkable bilaterally except for mild incyclotorsion of the right fundus.

Repeat MRI scanning with and without contrast was performed, including Fast Imaging Employing Steady-state Acquisition (FIESTA), and showed no evidence of tumor or aneurysm. However, the right oculomotor nerve, normal in caliber as it emerged from the brainstem, appeared atrophic after passing by a prominent right superior cerebellar artery (Fig. 2). The atrophy was particularly apparent when comparing the dimensions of the right and left oculomotor nerves as they entered the cavernous sinus (Fig. 3). There was no radiological evidence of compression of other cranial nerves. Consultation with neurosurgery

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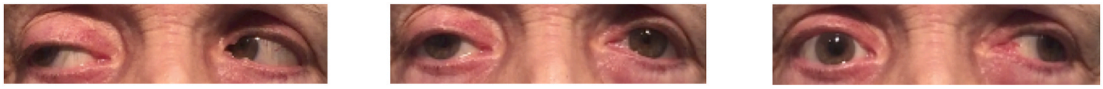

Fig. 1. Composite photos showing primary, right, and left gazes. The ptosis of the right upper eyelid is greatly reduced in left gaze, consistent with aberrant reinnervation of the right levator muscle from fibers intended for the right medial rectus.

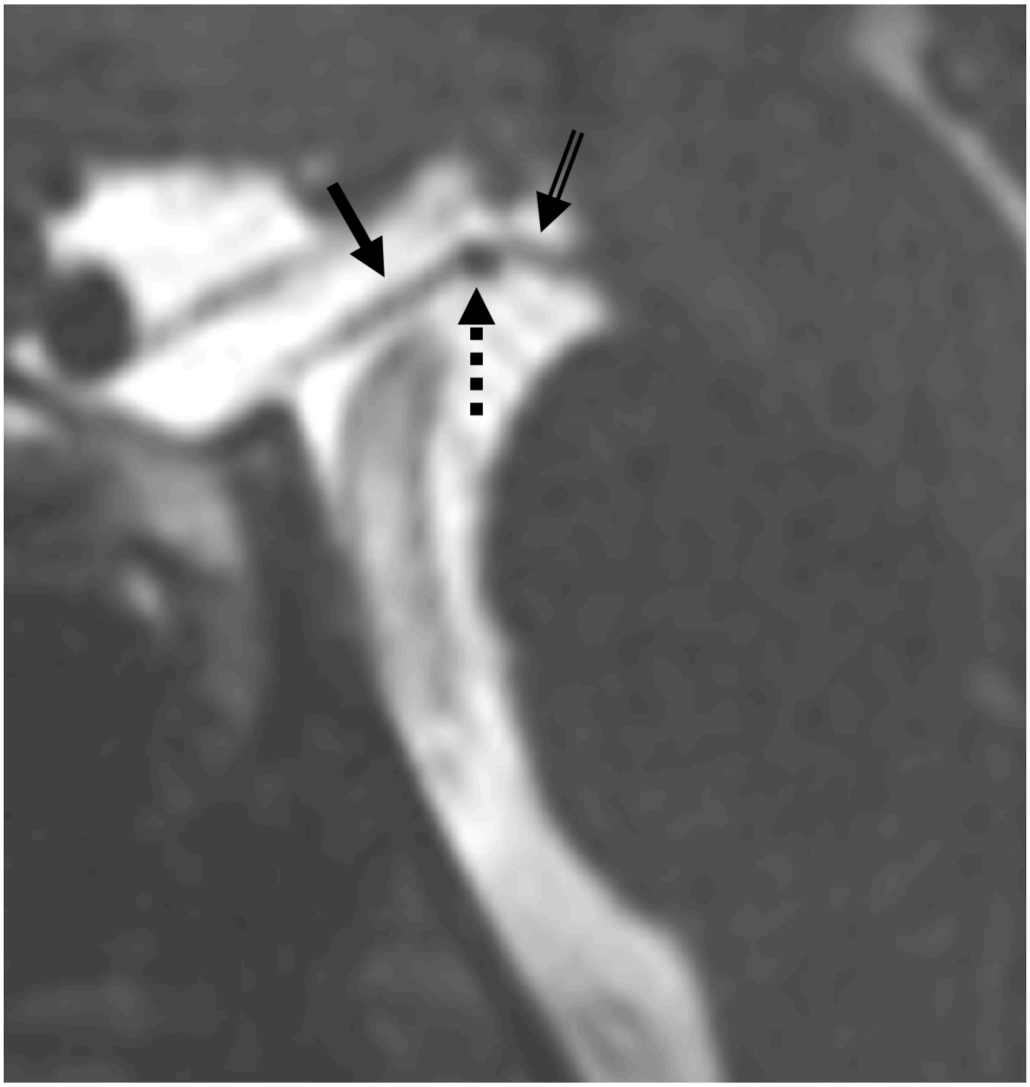

Fig. 2. A reformatted sagittal image of a steady-state gradient echo sequence demonstrates the right CN III before (double line arrow) and after (solid arrow) contacting the superior cerebellar artery (dotted arrow)..

was requested and it was felt that surgical intervention to reduce neurovascular impingement would likely cause further injury to the nerve with little potential for improving its function.

3. Discussion

Neurovascular compression is a rare and easily overlooked cause of oculomotor nerve palsy. In the absence of trauma, this finding strongly suggests a chronic compressive lesion rather than an ischemic, demyelinating, inflammatory, or infectious etiology. The acquisition of high-resolution MRI images from a balanced steady state gradient echo sequence was particularly helpful to make the diagnosis.[1,2] Vendor-specific names for these sequences include FIESTA (Fast Imaging Employing Steady-state Acquisition) by General Electric, CISS (Constructive Interference Steady State) by Siemens, and balanced-FFE (Fast Field Echo) by Philips.[1] This mode of imaging is especially sensitive for visualizing the cranial nerves due to high contrast resolution between the cerebrospinal fluid (CSF) cisterns and nerves.

The diagnosis on imaging requires changes in the nerve after contact with the offending vessel. Compression that causes distortion of the nerve's course or contour, splaying of its fibers, or atrophy, is predictive of symptoms.[2–5] Although arteries and veins can both cause neurovascular compression with resulting neuropathy, the higher pressure and pulsatility of arteries more commonly implicates them.[5]

Within the subarachnoid space, central myelination by oligodendrocytes transitions to peripheral myelination by Schwann cells in a segment of the nerve called the root transition zone.[3,5] It is at this position that nerves are particularly susceptible to neurovascular compression,[6,7] perhaps in part due to the fixed location of the nerve root at the brainstem.

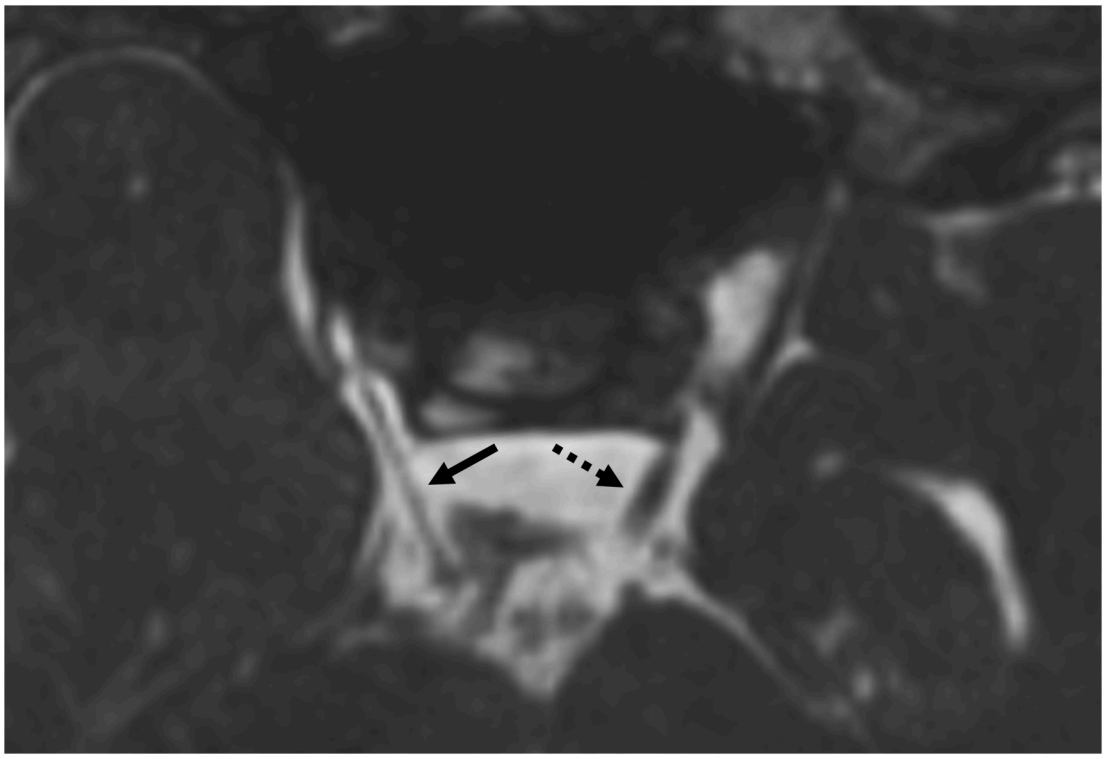

Fig. 3. An axial image of a steady-state gradient echo sequence demonstrates the right CN III nerve is atrophic (solid arrow) distal to the neurovascular contact compared to the left CN III (dotted arrow).

4. Conclusion

In the case of our patient, the finding of anomalous reinnervation was strongly suggestive of a chronic compressive process, and the use of FIESTA imaging provided the key imaging information to establish the underlying etiology.

Patient consent

The patient consented to publication of this case in writing.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship. ‘

Disclosures

None.

Declaration of competing interest

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