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Optic Neuropathy Secondary to Dolichoectatic ICA Compression

Madeline Abrams, Deepak Khatri*, Randy D Amico and John A Boockvar

Department of Neurosurgery, Lenox Hill Hospital, New York, USA

Abstract

Optic neuropathy, manifesting as a myriad of visual disturbances due to pathology of the optic nerve, is common. The list of possible etiologies is expansive and includes: ischemic, infectious, inflammatory, demyelinating, compressive, toxic-metabolic, and traumatic etiologie.

Keywords

Dolichoectatic intracranial vessels; Optic neuropathy; Pituitary tumors; Ischemic injury

Description

Optic nerve compression secondary to dolichoectatic intracranial vessels is a rare cause of optic neuropathy, with limited information regarding its natural history [1]. Thus, an individual case-based approach to treatment is of paramount importance, especially in the setting of an adjacent coexisting common pathology.

Our group previously presented a case of compressive optic neuropathy due to a dolichoectatic Internal Carotid Artery (ICA) in a 52-year-old man with post-traumatique headaches. His radiological workup demonstrated a flattened right optic nerve with a dolichoectatic ICA and a co-existing pituitary microadenoma on the left side of the stalk [2]. After 5 months of clinical follow-up, the patient developed progressive bilateral superior altitudinal field defects with normal optic discs on examination. Interestingly, the pituitary adenoma demonstrated no progression. This posed a treatment dilemma to surgical decision making as both, vascular decompression and tumor resection remained viable options.

Pituitary tumors comprise between 10%-15% of all tumors of the Central Nervous System (CNS) and frequently result in compressive visual disturbances, most common being a bitemporal hemianopsia due to compression of the optic chiasm [3]. However, several types of field defects have been reported including both superior and inferior altitudinal field loss [4].

However, in this case we favored the diagnosis of optic compression secondary to the dolichoectatic right Internal Carotid Artery (ICA) as the pituitary tumor was located toward the left of midline and there was no progression of tumor size noticed on the repeat scan despite worsening clinical symptoms.

Early studies suggested that ischemic injury to the optic nerve as a causative factor in optic neuropathy more so than mechanical compression due to dolichoectatic vasculature [5]. However, the underlying mechanism of compressive optic neuropathy by dolichoectatic intracerebral vessels remains a matter of debate [6]. A previous study described the neuro-ophthalmologic features in patients with MRI-confirmed optic nerve compression by the intracranial carotid artery, of which several were dolichoectatic [7]. Similar to pituitary tumors, several patterns of visual field defects were identified, including altitudinal defects. Interestingly, altitudinal field defects are more commonly associated with anterior ischemic optic neuropathy, providing further support for the role of regional hypoperfusion in optic neuropathy secondary to compressive intracranial vasculature [8].

We believe that a combination of both the mechanical compression by the abnormal vessel and ischemic changes in the optic nerve contribute to progressive neuropathy. The finding that not every case with dolichoectatic compression becomes symptomatic suggests that the underlying mechanism may represent a multi-step process whereby chronic compression by the vessel leads to ischemia and subsequent symptoms. Furthermore, vascular decompression can result in immediate improvements in vision arguing against permanent ischemic injury [9,10].

Regardless of the etiology of the compressive injury, it remains a general consensus that dolichoectatic vascular compression is a reasonable addition to a differential diagnosis for optic neuropathy when more common etiologies have been ruled out [6,7]. Importantly, the findings from our case and subsequent improvement after microvascular decompression without resection of the pituitary tumor establishes that a co-existing pathology should not dogmatically exclude the diagnosis of vascular compression as a possible diagnosis. Each case must be evaluated individually and treated accordingly.

Ideal management of this condition still remains ambiguous, due to limited knowledge regarding the natural history of this pathology Purvin et al. suggested conservative management in mild and slowly progressive cases, however surgical intervention may be pursued for severely symptomatic or rapidly progressive cases [9]. A significant improvement in the vision following microvascular decompression has established the role of surgical intervention [9,10]. Following the decompression, our patient had vision improvement with recovery of the field deficits on formal testing [2]. It is important to note that the recovery following surgical decompression may not be complete if the primary optic nerve insult was ischemic in nature, whereas a compressive mechanism of injury would more likely result in near-total or total recovery [7].

In the setting of a co-existing pathology, physicians should be wary of immediately relegating intracranial vascular compression to a last-resort diagnosis. Based on our experience, we recommend a case-based approach and suggest surgical intervention in progressive, symptomatic cases of optic neuropathy secondary to dolichoectatic ICA compression.

***Corresponding author:** Deepak Khatri, Department of Neurosurgery, Lenox Hill Hospital, 130 E 77th ST, 3 Black Hall, New York, USA 10075, E-mail: dkhatri@northwell.edu

Received February 08, 2021; **Accepted** February 22, 2021; **Published** March 01, 2021

Citation: Abrams M, Khatri D, Amico RD, Boockvar JA (2021) Optic Neuropathy Secondary to Dolichoectatic ICA Compression. Optom Open Access 6: 1-2.

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