Monocular Optic Neuropathy: Case Unsolved

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Case Presentation

Female patient, 58 years old, unremarkable general medical and ophthalmological history, complains about pulsatile headache, with no irradiation, no specific location, and absence of nausea or vomiting. The headache would not aggravate after Valsalva maneuver and faded without medication. Also, refers vision loss in the right eye (RE) since the beginning of the headache, 10 days before. The best corrected visual acuity (BCVA) was hand movement in the (RE) and 9/10 Snellen scale in the left eye (LE). It was observed a relative afferent pupillary defect (RAPD) graded 3+, no color perception on the RE, Ishihara plates fully read in the LE, no eye movement alterations, and no facial proprioception alterations. The anterior segment had no inflammation signs and phacosclerosis was present in both eyes. Posteriorly, there was no signs of disc inflammation, vitritis, macular or vascular alterations. Intraocular pressure (IOP) was 15 mmHg bilaterally.

Investigations

Emergency head Computerized Tomography (CT) and Magnetic Resonance Imaging (MRI) with gadolinium were performed to rule out optic pathway lesions or neuritis. ESR, CRP, blood count were performed to exclude Giant Cell Arteritis, which came negative [1]. Furthermore, testing for HIV, Syphilis, Hepatitis B and C, IGRA Quantiferon-TB (Tuberculosis), ACE and urinary calcium (Sarcoidosis), ANCA PR3 and MPO (Autoimmunity), HLA-B51 (Behçet’s disease) and Anti-NMO Aquaporin 4 antibodies (Devic’s disease) was completed, with no positive results [2]. Optic disc and macular spectral domain Ocular Computerized Tomography (sdOCT) had no acute alterations. Computerized static perimetry (CSP) was unreliable due to a high percentage of false positive and negative responses, but revealed a deep and general loss of visual sensitivity across all right visual field.

Differential Diagnosis

At this time, the main differential diagnosis were:

Posterior optic neuropathy (PON)

Although there was no big abdominal or heart surgery previously, an RAPD graded 3+ and deep visual loss were present [3], without optic disc edema.

Optic neuritis (ON)

The patient was not young enough to fit the diagnosis of multiple sclerosis or Devic’s disease, plus, there were no lesions revealed by the MRI or neurological symptoms, but due to the profoundity of visual loss [4], these options had to be considered.

Anterior Ischemic Optic Neuropathy (AION)

No disc edema was observed, so by definition, this option was excluded [5]. The patient had no risk factors.

Treatment

After exclusion of infectious possibilities, a 1Gr metilprednisone/day was administered for 3 days. After this period [6], she was medicated with oral prednisone 1mgr/Kg/day for 2 months, and then started to halt it in a slow manner.

Outcome

The patient improved greatly after 2 months. RE BCVA was 9/10, no RAPD was present, Ishihara plates were completely read and equally fast [7]. RE CSP, which had initially a Visual Function Index of 2%, presented lastly 65%, with a medium deviation of -15.42dB and pattern stand deviation of 8.45dB. LE was considered unremarkable. sdOCT revealed RE optic disc atrophy, as the peripapillary nerve fiber layer had a thickness of 60μm, and an apparent loss of macular nerve fiber layer thickness [8]. The optic atrophy was observed at the slit lamp.

Discussion

Initially, due to the absence of optic disc edema, the options considered were posterior to the optic disc, which lead us to think of Multiple Sclerosis and Devic’s disease, but the neuro imaging and blood testing ruled out these possibilities. After 2 months, although RE vision had improved, an optic atrophy was evident. It’s curious as there was no optic disc edema observed throughout the follow-up, and no occupying-space lesion was identified. Since there was a clinical response to the steroids, ON is a possible diagnosis, although the study was negative.
References


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