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Optic Neuritis: A Comprehensive Review

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Abstract: ***Optic neuritis (ON) is an inflammatory demyelinating condition affecting the optic nerve, often associated with multiple sclerosis (MS) and other autoimmune diseases. It presents with acute or subacute vision loss, pain on eye movement, and dyschromatopsia. Diagnosis is primarily clinical but may be supported by imaging and electrophysiological tests. Management strategies include corticosteroids and immunomodulatory therapies, with an overall favorable visual prognosis in isolated cases. This review summarizes the epidemiology, pathophysiology, clinical presentation, diagnostic approaches, management, and prognosis of ON, integrating data from recent ophthalmology literature.***

Keywords: optic neuritis, multiple sclerosis, neuromyelitis optica, demyelination, corticosteroids

1. Introduction

Optic neuritis is one of the most common optic neuropathies, particularly in young adults. It is frequently associated with demyelinating diseases, but can also be seen in infectious, inflammatory, and paraneoplastic conditions. The Optic Neuritis Treatment Trial (ONTT) provided pivotal insights into its natural history and treatment response, influencing current clinical practice (1). This review explores recent advancements in the understanding and management of ON.

Epidemiology

ON predominantly affects females (3: 1 ratio) and typically presents between 20–40 years of age (2). The annual incidence is estimated at 1–5 per 100, 000 individuals. It is more common in populations with a higher prevalence of MS, with nearly 50% of MS patients experiencing ON at some point (3).

Pathophysiology

The underlying pathology of ON involves immune - mediated inflammation and demyelination of the optic nerve. In MS - related ON, auto - reactive T - cells target myelin oligodendrocyte glycoprotein (MOG), leading to focal demyelination, axonal loss, and gliosis (4). A subset of ON cases is associated with neuromyelitis optica spectrum disorder (NMOSD), characterized by antibodies against aquaporin - 4 (AQP4) (5).

Clinical Presentation

Patients typically present with unilateral vision loss that progresses over hours to days, accompanied by periocular pain exacerbated by eye movement. Other features include:

- Dyschromatopsia (color desaturation, particularly red desaturation)
- Relative afferent pupillary defect (RAPD) in unilateral cases
- Visual field defects (central scotomas, altitudinal defects)

Systemic symptoms, such as Uhthoff's phenomenon (worsening vision with heat), may indicate an MS - related process (6).

2. Diagnostic Approach

1) **Clinical Examination**:
- Fundoscopy: Often normal in retrobulbar neuritis but may reveal optic disc swelling in papillitis.
- Visual Acuity Testing: Ranges from mild impairment to severe loss.
- RAPD Testing: Confirms asymmetric optic nerve dysfunction.

2) **Imaging:**
- Magnetic Resonance Imaging (MRI) with gadolinium contrast is the modality of choice, showing optic nerve enhancement in active inflammation (7).
- Brain MRI identifies demyelinating plaques, which predict MS risk.

3) **Optical Coherence Tomography (OCT):**
- Demonstrates retinal nerve fiber layer (RNFL) thinning, useful for monitoring chronic damage (8).

4) **Electrophysiology:**
- Visual Evoked Potential (VEP) testing reveals delayed P100 wave latency, supporting demyelination (9).

5) **Serology:**
- Anti - AQP4 and anti - MOG antibodies help distinguish NMOSD and MOG - associated ON from MS - related ON (10).

Differential Diagnosis

- NMOSD
- MOG - antibody associated disease
- Compressive optic neuropathy (tumors, aneurysms)
- Ischemic optic neuropathy

- Infectious optic neuritis (syphilis, tuberculosis, Lyme disease)

Management

1) Acute Phase Treatment:
- Intravenous methylprednisolone (1g/day for 3 days) followed by oral taper reduces recovery time but does not impact final visual outcome (11).
- Plasma exchange therapy (PLEX) is beneficial in severe or steroid - resistant cases, especially in NMOSD (12).

2) Long - Term Considerations:
- MS - related ON: Disease - modifying therapies (DMTs) like natalizumab or fingolimod reduce recurrence (13).
- NMOSD - related ON: Requires immunosuppressants (rituximab, eculizumab) due to high relapse risk (14).
- MOG - related ON: More relapsing - remitting course; corticosteroids and IVIG are beneficial (15).

Prognosis Most patients experience significant visual recovery within 6 months, with 95% regaining 20/40 or better vision. However, recurrent ON increases the risk of permanent axonal loss (16). MS - related ON carries a 50% risk of converting to clinically definite MS within 15 years (17).

3. Conclusion

Optic neuritis remains a critical entity in neuro - ophthalmology, requiring careful differentiation between MS, NMOSD, and MOG - associated ON. Advances in imaging and serologic markers have refined diagnosis and management strategies. Future research should focus on neuroprotective strategies to prevent irreversible optic nerve damage.

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