RETROBULBAR OPTIC NEURITIS

Dr. Krati Gupta
Dr. Saurabh Deshmukh

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Retrobulbar optic neuritis

- Optic neuritis typically occurs in young (mean age, 32 years), female (77%) patients.

**Signs and symptoms**
1. It presents as subacute monocular visual loss that develops over several days.
2. Periorbital pain, particularly with eye movement, occurs in 92% of cases and often precedes vision loss.
3. The retrobulbar form (in which the optic disc appears normal) occurs in 65% of cases.
4. Unless the optic neuritis is bilateral and symmetric, an RAPD is also present.
5. Perimetry testing most often shows generalized reduction of sensitivity (48%) and any pattern of visual field loss may appear.
6. Dyschromatopsia, particularly for red, is nearly universal and is often out of proportion to the visual acuity loss.
7. Optic neuritis shows some improvement within 1 month in the vast majority of cases.
8. Most cases of optic neuritis represent isolated or demyelinating disorders and do not require further workup for another diagnosis.

- **Atypical features** that should prompt further evaluation include
  1. Lack of pain,
  2. Protracted pain or vision loss,
  3. Significant swelling of the optic disc,
  4. Inflammatory ocular features (eg, uveitis, phlebitis, choroiditis, pars planitis),
  5. Bilateral vision loss, involvement of other cranial nerves,
  6. Steroid responsive optic neuropathy,
  7. Lack of any vision recovery by 1 month.

**Investigation**
- Additional hematologic, serologic, and other testing may be of value.
- Such studies may include the following:
  1. Serum and CSF rapid plasma reagin and fluorescent treponemal antibody absorption testing (for syphilis)
  2. Chest x-ray, gallium scan, serum angiotensin converting enzyme testing (for Sarcoidosis)
  3. ESR determination, antinuclear antibody testing, and anti-DNA antibody testing (for systemic lupus erythematosus or vasculitis)
  4. Serum neuromyelitis optica IgG testing and spinal MRI (for neuromyelitis)
  5. Brain and orbit MRI with gadolinium contrast (for compressive, infiltrative disorders)
  6. Testing of antibody titers for Lyme disease (if endemic)
  7. Leber hereditary optic neuropathy testing serum
  8. Quantiferon Gold testing or purified protein derivative skin test (for tuberculosis).
  9. In the absence of a known diagnosis of MS, MRI of the brain is recommended in every case of retrobulbar neuritis.
  10. The evaluation for periventricular white matter lesions consistent with demyelination is the single best test for assessing the risk of future MS and to guide subsequent decisions on the use of immunomodulation therapy.
ONTT

1. The ONTT 10-year follow-up study reported that optic neuritis recurred in the affected or fellow eye in 35% of cases overall and in 48% of patients with conversion to MS.
2. Most eyes with a recurrence regained normal or almost-normal vision.
3. After 15 years of follow-up in the ONTT, 92% of patients with optic neuritis had recovery of visual acuity to 20/40 or better; 3% had final visual acuities of 20/200 or worse.
4. Despite their seemingly excellent prognosis, patients with optic neuritis usually remain aware of visual deficits in the affected eye after recovery.
5. Studies using measures of visual function other than Snellen visual acuity (eg, contrast sensitivity, motion detection, stereopsis) show residual abnormalities in up to 90% of patients with at least 20/30 visual acuity.
6. The 15-year data from the ONTT demonstrate a risk for MS of 25% for patients with zero lesions on MRI versus 72% for patients with at least 1 lesion, with the highest rate of conversion within the first 5 years.
7. Patients with normal MRI results and no conversion to MS by year 10 had only a 2% risk of conversion by year 15.
8. Among patients with normal baseline MRI results, a lower risk of future MS was associated with male sex, optic disc swelling, and atypical features of optic neuritis (absence of pain, no light perception vision, peripapillary hemorrhages, and retinal exudates).

Treatment of optic neuritis

1. The ONTT demonstrated that corticosteroid therapy for optic neuritis had no long-term beneficial effect on vision, although the use of intravenous methylprednisolone, 250 mg every 6 hours for 3 days, followed by oral prednisone, 1 mg/kg/day for 11 days, sped recovery by 1–2 weeks.
2. Patients receiving oral prednisone alone did not have any benefit to vision and incurred a recurrence rate double that of the other groups; therefore, this treatment is not recommended.
3. Intravenous therapy demonstrated a reduction in the rate of development of clinically definite MS after the initial optic neuritis only in the subgroup of patients with MRI scans showing 2 or more white matter lesions.
4. At 2 years, these patients’ risk for MS was 36% untreated, 16% treated. By follow-up year 3 and thereafter, however, this protective effect was lost.
5. With unclear benefits, the value of therapy and of additional diagnostic evaluation for MS must be assessed individually.
6. In cases in which a rapid return of vision is essential (eg, monocular patient, patient with an occupational need), intravenous methylprednisolone on an outpatient basis may be considered; otherwise, treatment for vision recovery is not required.
7. An MRI scan is generally performed to assess MS risk, but additional evaluation, including CSF analysis, is probably best referred to a consulting neurologist.
8. The value of intravenous corticosteroids alone to reduce the long-term risk of MS is unproven.
9. Immunomodulatory therapy is of proven benefit for reducing morbidity in the relapsing-remitting form of MS, and studies have shown that such drugs delay the conversion of patients with acute optic neuritis or other clinically isolated syndrome with high-risk MRI characteristics to definite MS.