## Overview

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### Abstract
This course reviews the presenting signs and symptoms of acute demyelinating optic neuritis and its mimics, diagnostic tests to order in patients with this diagnosis, who should be treated, how this affects disease outcomes, patients at risk for multiple sclerosis, and treatment of patients at highest risk for multiple sclerosis.

### Objectives
On completing this course, the ophthalmologist should be able to
- Diagnose the presenting signs and symptoms of acute demyelinating optic neuritis and its mimics.
- Manage treatment of acute demyelinating optic neuritis.
- Manage treatment of patients at risk for multiple sclerosis.

### Disclosures
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S = grant support

### Audience
Comprehensive ophthalmologists and medical residents

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### Hardware/Software Requirements
This learning activity requires a current web browser with Flash Player 10 installed. A broadband connection is recommended.
Acute Demyelinating Optic Neuritis

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Introduction

Objective:
On completing this section, the ophthalmologist should be able to describe the learning goals for this online course.

A 26-year-old Caucasian woman presents to your office with a 3 day history of vision loss OS. She has had a left-sided periorbital headache for 5 days, and she now reports pain with eye movement on top of the constant ache. She is otherwise well, although she did have a mild fever and runny nose 2 weeks ago which she attributed to “flu.”

Is this patient likely to have optic neuritis? If so, what further tests are needed to confirm the diagnosis and proceed with treatment?

In this course, we will review the presenting signs and symptoms of acute demyelinating optic neuritis and its mimickers. What diagnostic tests do you need to order in patients with this diagnosis? How do you decide if they have the real disease or a masquerading syndrome? Once you have made the diagnosis, who should be treated, and how does this affect disease outcomes? Finally who is at risk for multiple sclerosis, and should we treat those patients who are at highest risk?

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Clinical Evaluation: Clinical Signs

Objective:

On completing this section, the ophthalmologist should be able to describe the typical patient profile and common presenting symptoms of optic neuritis.

Optic neuritis typically affects women aged 18-45 and is often associated with pain or discomfort around the eye that worsens with eye movement (in the Optic Neuritis Treatment Trial, pain with eye movement was present in 92% of patients). Patients also note a decrease in central vision that may be as mild as fogginess to their vision or as severe as no light perception. The vision tends to diminish in a crescendo-like manner over a couple of days, and the visual nadir is at two weeks from onset of vision loss. Patients can also note flashes of light (phosphenes) that do not indicate vitreoretinal pathology.

Systemically, some patients feel run down and have other prodromal symptoms such as a flu-like illness. Both dullness of certain colors such as red and overall reduction of brightness and contrast also occur frequently. Almost any type of visual field defect may be found, and thus while perimetry is a crucial part of the evaluation of patients with suspected optic neuritis, it cannot be used to distinguish optic neuritis from other optic neuropathies. Patients may also experience Uhthoff's phenomenon, which is non-specific but consists of blurred vision when body temperature rises during hot showers or strenuous exercise. Patients may avoid hot saunas or always keep cold water with them to maintain visual clarity.

Optic neuritis typically is unilateral; however, when it occurs bilaterally, one must consider other atypical diseases such as neuromyelitis optica (Devic disease), sarcoidosis, and infectious agents. In Devic disease, transverse myelitis may develop concurrently or occur later in the disease. Rarely will transverse myelitis precede the optic neuritis. Neurosarcoidosis is also an uncommon disease that can occur in Scandinavian or African-American patients with or without pulmonary symptoms. Anterior or posterior uveitis may occur as well. Tuberculosis and syphilis also may lead to optic neuritis, and other neurological manifestations may be present.

In children, optic neuritis presents differently with approximately 50% of cases being bilateral. There tends to be more of a prodromal illness found in the history and tend to have worse vision upon onset. Frequently, there are other findings systemically such as seizures or peripheral sensory or motor neuropathies. When accompanied with an altered sensorium and irritability, acute disseminated encephalomyelitis may be entertained as a diagnosis of exclusion.

Clinical Evaluation: Physical Exam

Objective:

On completing this section, the ophthalmologist should be able to identify clinical signs of optic neuritis and associated findings of demyelinating disease.

Documentation of Snellen visual acuity is important to follow patient's progress and expected improvement. Low contrast acuity charts are even more sensitive for assessing vision loss in optic neuritis but may not be widely available. Color vision testing (Ishihara, H-R-R, D15, etc.) is also is helpful to determine if optic nerve function is reduced.

In the absence of formal color testing tools, red desaturation testing can be done. A red cap is presented to the patient to one eye at a time. The patient will often perceive the
red as being a dullish orange or brown in the affected eye. This is a relative test, and it is important to compare the saturation to the unaffected eye.

As is the case in any unilateral optic neuropathy, most patients with optic neuritis have a relative afferent pupil defect (RAPD) in their affected eye (Exercise 1) albeit this clinical finding may be absent in cases of bilateral optic nerve involvement. Brightness sense can be tested by shining a bright light source at the patient and asking if the brighter side is 100% intensity, what percent is the other side, 50%, 80%, etc. However, brightness sense can be affected by corneal or lenticular issues such as cataract and is very nonspecific.

Extraocular movements may show a sixth nerve palsy or ocular dysmetria both found in patients with demyelinating disease. A subtle internuclear ophthalmoplegia may become evident when the patient makes large saccades. Dilated fundus exam for detailed evaluation of the optic nerve and macula is mandatory. The fundus examination is often normal in adults, whereas the majority of pediatric patients manifest papillitis. Only 30% of patients in the ONTT presented with disc edema. Optic atrophy should not be noted until several weeks after onset of symptoms. If optic atrophy is seen at presentation, then a compressive process must be suspected rather than optic neuritis.

**Clinical Evaluation: Perimetry**

**Objective:**

On completing this section, the ophthalmologist should be able to explain the different roles that automated and kinetic perimetry play in the diagnostic workup of suspected optic neuritis.

Automated perimetry must be used to document the scotoma that is almost always present. In fact, in the ONTT, a visual field defect was required for patient inclusion. The pattern of visual field loss tends to reflect the topography of the retinal nerve fiber layer, and cecocentral (Figure 1), arcuate and altitudinal defects are common. Up to one third of fellow eyes will have a perimetric defect as well. A large (size V) stimulus may be used if central acuity is very poor. Furthermore, foveal threshold may be used to help identify the degree to which central vision is affected. Kinetic perimetry may allow more accurate diagnosis of central or cecocentral field defects because of its continuous coverage as opposed to testing of discrete points 3 and 9 degrees from fixation (Exercise 1).
Figure 1. Top: Goldmann perimetry in a patient with optic neuritis shows a normal visual field in the left eye and a dense cecocentral scotoma in the right eye. Bottom: Automated perimetry shows a dense central scotoma in the right eye and generalized constriction in the left eye.

Clinical Evaluation: Imaging

Objective:

On completing this section, the ophthalmologist should be able to explain how ophthalmic imaging and neuroimaging tools may be used in the diagnosis of optic neuritis and associated systemic diseases.

MRI of the brain and orbits with gadolinium contrast will be helpful to establish the risk of demyelinating disease. Enhancement of the optic nerve may be seen but is not required to make the diagnosis of optic neuritis (Figure 1). Visual evoked potential latencies are frequently prolonged in the eyes of multiple sclerosis patients, even in the absence of clinically overt optic neuritis events.

Optical coherence tomography (OCT) has been proposed as a potential biomarker for axon loss in multiple sclerosis patients because retinal nerve-fiber layer (RNFL) atrophy in optic neuritis eyes has been shown to correlate well with clinically meaningful visual
outcomes and neurological disability (Figure 2). The RNFL may be normal or even elevated in acute optic neuritis because of axoplasmic stasis and/or edema (Figure 3). Some data indicate that RNFL thinning after typical demyelinating optic neuritis is less severe than after optic neuritis from other causes (Clinical Manifestations), but this finding is controversial. Recent studies indicate that visually evoked potential (VEP) may be more sensitive than OCT in detecting optic neuritis especially after the acute event has resolved, but research in this area is still ongoing. If either VEP or OCT shows abnormalities in the fellow eye, then the possibility of demyelinating disease is increased.

Figure 1. Axial T1 MRI with gadolinium contrast and orbital fat suppression shows L optic nerve enhancement.
Figure 2. OCT of RNFL several months after optic neuritis diagnosed in right eye, showing a marked thinning of the RNFL in all sectors. The left eye is normal.
Acute Demyelinating Optic Neuritis

Figure 3. RNFL OCT (Stratus) in a case of acute left eye optic neuritis. The RNFL in the left eye is elevated, especially inferotemporally.

Clinical Evaluation: Optic Disc Swelling

Objective:

On completing this section, the ophthalmologist should be able to explain how the presence of a swollen optic disc changes the evaluation of a patient with possible optic neuritis.

To help rule out other optic neuropathies that can cause disc edema, one should check for elevated blood pressure. Malignant hypertension can cause the optic nerve to be swollen. Anterior ischemic optic neuropathy presents with sectoral disc edema but can also progress to a total disc edema. Sectoral disc edema has not been to my knowledge reported when patients get optic neuritis. Patients with AION present with vision loss often upon awakening due to possibly postural hypotension. Risk factors include hypertension and sleep apnea. An MRI of the brain is indicated in pediatric optic neuritis due to the high association with other central nervous system dysfunction. An MRI of the cervical and thoracic spine is indicated if Devic's is considered as a possibility. NMO serum blood titers may is specific and may help to diagnose Devic's disease.
Clinical Evaluation: Mimics

Objective:

On completing this section, the ophthalmologist should be able to discuss signs and symptoms that differentiate typical demyelinating optic neuritis from other retinal and optic nerve disorders.

Atypical clinical features should prompt investigation for possible optic neuritis mimics (Table 1, Table 2). Failure of clinical improvement, for example, may indicate an underlying compressive lesion, such as an optic nerve sheath tumor or suprasellar mass. In some cases of compressive optic neuropathy, the patient may report acute-onset awareness of vision loss when, in fact, visual dysfunction in the affected eye has progressed insidiously over time. It is also important to exclude a potential compressive lesion when optic disc pallor is seen at the onset of presumed optic neuritis. Optic disc pallor takes weeks to develop after optic neuritis, and should not be apparent during the acute event. As mentioned, bilateral simultaneous vision loss may occur in children with optic neuritis; but in adults, this occurrence may herald the diagnosis of Leber's Hereditary Optic Neuropathy.

Posterior ischemic optic neuropathy (PION) may be challenging to distinguish from optic neuritis; yet optic neuritis patients are generally younger, have less abrupt onset vision loss, and tend to lack vascular risk factors as compared to PION patients. Systemic clinical symptoms and signs, including fever, weight loss, rash, joint pain, alopecia, and hematologic abnormalities should raise concern for possible infectious, neoplastic or vasculitic conditions.

Patients presenting with vision loss, an enlarged blind spot on visual field testing, and a normal fundus examination should be evaluated for possible retinal mimics of optic neuritis. In these cases, multi-focal ERG testing can reveal a primary retinopathy including multifocal evanescent white dot syndrome (MEWDS), or acute zonal occult outer retinopathy (AZOOR) as the underlying diagnosis.

Neuromyelitis optica (NMO) represents a severe inflammatory process of the optic nerves and spinal cord, which is associated with poor clinical recovery. In addition to optic nerve involvement, typical features of NMO include episodic myelitis (with spinal cord MRI lesions that extend three or more spinal segments) and absent brain MRI lesions. For patients considered to be at high risk for NMO, testing for the NMO-IgG antibody might help expedite the necessary treatment regimen for this distinct clinical syndrome.

Finally, some patients have an orbital inflammatory process with inflammation in the optic nerve sheath, called optic perineuritis or perioptic neuritis. Appropriate optic nerve imaging can help localize the site of inflammation to the optic nerve sheath, and distinguish perioptic neuritis from optic neuritis.
Table 1. Atypical clinical features

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<thead>
<tr>
<th>Feature</th>
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<tr>
<td>Age &gt; 50 years</td>
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<tr>
<td>Optic disc pallor at presentation</td>
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<td>Bilateral simultaneous vision loss in adults</td>
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<tr>
<td>Absence of pain</td>
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<tr>
<td>Pain and/or vision loss that progresses over weeks</td>
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<tr>
<td>Atypical fundus features (abundant vitreous cell, florid optic disc edema, hemorrhages, and exudates)</td>
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<tr>
<td>Poor visual recovery</td>
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<td>Associated systemic signs and symptoms</td>
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Table 2. Clinical mimics of optic neuritis

<table>
<thead>
<tr>
<th>Mimic</th>
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<tr>
<td>Ischemic optic neuropathy</td>
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<tr>
<td>Optic perineuritis</td>
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<tr>
<td>Compressive optic neuropathy (pituitary adenoma, optic nerve sheath meningioma)</td>
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<td>Neuromyelitis Optica</td>
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<td>Leber's hereditary optic neuropathy</td>
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<td>Inflammatory optic neuropathy (sarcoidosis, Wegener's granulomatosis)</td>
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<td>Toxic/metabolic optic neuropathy</td>
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<td>Infections (neuroretinitis, Lyme, syphilis)</td>
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<td>Medication-induced optic neuropathy</td>
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<td>Infiltrative/neoplastic optic neuropathy</td>
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<td>Steroid responsive optic neuropathy</td>
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<tr>
<td>Big Blind Spot Syndromes (MEWDS, AZOOR)</td>
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**Vision Loss: Prognosis**

**Objective:**
On completing this section, the ophthalmologist should be able describe to the long-term visual prognosis of patients with typical optic neuritis.

More than 88% of patients with optic neuritis in the ONTT had final vision of 20/40 or better. Visual recovery may take up to 6 months after the acute onset, and relapses are uncommon.

Despite the return of central acuity, many patients noted that contrast sensitivity remained poor in the affected eye.

Data from the Longitudinal Optic Neuritis Study (LONS) confirmed the stability of the visual recovery 15 years after the acute episode. Permanent vision loss in the fellow eye was also distinctly uncommon. Visual field abnormalities did persist in a number of patients. These visual outcomes were the same in all three ONTT groups (intravenous corticosteroid treatment, oral prednisone treatment, placebo), although the rapidity of visual recovery was greater in the intravenous steroid cohort.

Thus, patients with typical demyelinating optic neuritis should be reassured that, regardless of treatment, they are very likely to have a good visual result.

**Vision Loss: Treatment**

**Objective:**
On completing this section, the ophthalmologist should be able to explain the appropriate indications for and administration route of corticosteroids in patients with optic neuritis.

The decision to treat a patient with corticosteroids should be based on the data gathered from the ONTT and subsequent studies. The ONTT was designed to compare the speed and level of visual recovery between patients treated with oral prednisone (1 mg/kg/d for 14 days); intravenous methylprednisolone (IVMP) (250 mg every 6 hours for 3 days in hospital followed by an oral taper for 11 days); or oral placebo (for 14 days). Patients treated with oral prednisone had an increased incidence of recurrent optic neuritis as compared to patients treated with IVMP or placebo. The IVMP therapy was associated with a decreased the likelihood of developing CDMS after 2 years, although this effect was not sustained after 3 years. In all patients, treatment was started within 8 days of onset of symptoms.

Despite the ONTT experience, the management of acute optic neuritis continues to be highly variable and somewhat controversial among clinicians. Many ophthalmologists choose to forgo treatment with corticosteroid therapy because of the lack of proven long-term benefit to the patient, and the potential for adverse effects. Repeated episodes of optic neuritis with use of IV steroids can cause cumulative effects such as osteoporosis and make patients more vulnerable to peptic ulcers.

Although the ONTT did not address the efficacy of high dose oral prednisone, many physicians use an equivalent dose of oral prednisone (1250 mg) daily to treat multiple sclerosis relapses, including optic neuritis. A Japanese study provides data that 500 mg prednisone may be a sufficient substitute in optic neuritis. Because of its association with increased relapse risk, oral prednisone in doses of 1mg/kg/day should not be used in the treatment of acute optic neuritis. Higher-dose oral corticosteroids or IVMP may hasten the speed of visual recovery, but there is no evidence of long-term benefit for visual recovery.
function. Hence, the decision to use these medications should be with the intention to increase the speed recovery but not to improve eventual visual outcome.

**Multiple Sclerosis: Role of MRI**

**Objective:**

On completing this section, the ophthalmologist should be able to characteristic MRI findings that increase multiple sclerosis risk in patients with optic neuritis and the limitations of these results in predicting future multiple sclerosis.

Approximately 20% of multiple sclerosis patients present with optic neuritis as their initial demyelinating event, which means the ophthalmologist is often the first physician to raise the possibility of this diagnosis. The most useful investigation to consider in this context is a cranial magnetic resonance imaging (MRI) study.

In multiple sclerosis patients, the most common abnormalities visualized on MRI are described as "white matter lesions," referring to the appearance of T2 and FLAIR hyper-intense signal changes that are observed in the periventricular brain regions (Figure 1). These lesions are often ovoid in shape, and have their longest axis oriented perpendicular to the ventricular surface ("Dawson's fingers"). T1-weighted images also may show "black holes" that may represent edema and axonal loss (Figure 2). Other common sites of involvement on MRI include the cerebellum, brainstem, and spinal cord. With time, the white matter lesions may become very extensive (Figure 3), and both brain and spinal cord atrophy may occur (Figure 4). Gadolinium-enhancing lesions are believed to reflect active inflammation.
Acute Demyelinating Optic Neuritis

**Figure 1.** Cranial MRI scan (axial view) showing white-matter lesions (arrows), including one in the anterior corpus callosum consistent with a diagnosis of multiple sclerosis.

**Figure 2:** A cranial MRI scan (axial view) showing white-matter lesions (arrows), including one in the corpus callosum consistent with the diagnosis of multiple sclerosis.
Figure 2. T1-weighted noncontrast image demonstrates a number of dark areas with the normal white matter in both hemispheres, representing chronic disease.
Figure 3. Sagittal FLAIR MRI of brain shows numerous periventricular, occipital, and infratentorial (cerebellar) white matter lesions that are highly characteristic of advanced multiple sclerosis.
Multiple Sclerosis: Risk of MS

Objective:

On completing this section, the ophthalmologist should be able to distinguish patients at high-risk for multiple sclerosis from others who present with optic neuritis as a possible sign of demyelinating disease.

The ONTT and subsequent LONS have shown that the baseline MRI study is the best predictor of future clinically-definite multiple sclerosis after optic neuritis. At 10-years, ONTT patients with no lesions on their initial MRI had a 22% risk of multiple sclerosis, which increased to 56% for patients with one or more white matter lesions. At 15-years, the overall risk of multiple sclerosis was 50%; patients with one or more lesions on their baseline MRI had a 72% risk of developing of multiple sclerosis, as compared to a 25% risk for patients with no lesions.

The true risk in patients with a normal baseline MRI may be even lower than implied by these studies, since modern MR imaging is much more sensitive to the presence of white matter lesions. There are many areas of the brain (such as subcortical regions) in which FLAIR lesions are found but are not as characteristic of multiple sclerosis. In addition, patients with optic neuritis often undergo future "surveillance" MRI scanning.
The later development of white matter lesions does not confer the same risk of multiple sclerosis that baseline lesions do.

From the experience of the ONTT and LONS, a number of factors have been shown to predict a decreased risk for the diagnosis of multiple sclerosis in patients with no MRI abnormalities including: male gender, severe optic disc swelling, NLP vision, lack of pain, peripapillary hemorrhages, and retinal exudates. Thus, the baseline ophthalmic assessment can reveal important information regarding the future risk of multiple sclerosis in optic neuritis patients.

**Clinically Isolated Syndrome: CIS**

**Objective:**

On completing this section, the ophthalmologist should be able to describe features of a clinically isolated syndrome that includes optic neuritis, and will understand treatment options for affected patients.

The clinically isolated syndrome (CIS) describes an initial neurological deficit in the setting of possible demyelinating disease. Optic neuritis fits this definition and may be considered in the same spectrum as systemic events. The presence of white matter lesions may be considered the second neurologic event used to indicate possible multiple sclerosis. By definition, patients with CIS do not meet the criteria for multiple sclerosis but must have something (brain MRI lesions, abnormal lumbar puncture with oligoclonal bands, etc) other than optic neuritis alone to suggest increased risk of multiple sclerosis development.

**Clinically Isolated Syndrome: Treatment**

**Objective:**

On completing this section, the ophthalmologist should be able to discuss the use of disease modifying therapies in the treatment of optic neuritis patients at high risk of progression to multiple sclerosis.

The evidence to date indicates that early treatment with disease-modulating multiple sclerosis therapies is both efficacious and safe in CIS patients. For this reason, ophthalmologists who suspect the diagnosis of optic neuritis should refer their patient to a neurologist, so that the role of disease-modifying therapies can be fully explored.

Recently, several published studies have shown that early initiation of disease – modifying therapies is beneficial in patients with optic neuritis and other clinically isolated syndromes (CIS) who are deemed to be at increased risk for developing multiple sclerosis. The first of these was the Controlled High-Risk Subjects Avonex Multiple Sclerosis Prevention Study (CHAMPS), which included 383 patients with CIS who were enrolled if they had two or more clinically silent lesions on baseline cranial MRI scan. After initial treatment with high-dose IVMP, half the patients received weekly interferon beta-1a (30 µg once per week), and half received placebo. The rate of clinically definite multiple sclerosis development was significantly lower (44%) in the treated group, and a relative reduction of new MRI lesions was found in patients treated with interferon versus the placebo group.

A second study, Early Treatment of Multiple Sclerosis (ETOMS), enrolled 308 CIS patients with four asymptomatic white matter lesions (or three lesions if one enhanced with gadolinium) on the baseline cranial MRI scan. Half the patients received subcutaneous interferon beta-1a (22 µg once a week), and half received placebo. After
two years, 45% of the placebo group developed clinically definite multiple sclerosis as compared to 34% of treated patients.

The Betaferon in Newly Emerging Multiple Sclerosis for Initial Treatment (BENEFIT) trial, included CIS patients with at least 2 brain MRI lesions, who were randomized to receive interferon beta-1b 250 µg subcutaneously on alternate days (n = 292 patients), or placebo (n = 176 patients) until the diagnosis of clinically definite multiple sclerosis or a 24-month follow-up point was reached. Treatment with interferon beta-1b delayed the time to diagnosis of multiple sclerosis. The results of the PreCISE trial, which used glatiramer acetate in a placebo-controlled treatment trial of 481 patients with CIS, also showed a 45% risk reduction for progression to clinically definite multiple sclerosis.

Thus, all of the immunomodulatory agents used in the treatment of multiple sclerosis also may be used in CIS. Controversy still exists regarding patient selection for treatment, since in all of the quoted trials, the majority of patients did not progress to multiple sclerosis regardless of treatment strategy. This question is not unlike the one faced by ophthalmologists in managing patients with ocular hypertension; while early treatment delays progression to glaucoma, most patients do not suffer vision loss, treated or not.

References


