Abstract: The current management of acute optic neuritis (ON) is focused on expediting visual recovery through the use of high-dose intravenous corticosteroids. The recent identification of specific autoantibodies associated with central nervous system inflammatory disorders has provided novel insights into immune targets and mechanisms that impact the prognosis, treatment, and recurrence of ON. Therefore, neurologists and ophthalmologists need to be aware of clinical, laboratory, and imaging findings that may provide important clues to the etiology of ON and the potential need for aggressive management. Moving forward, rapid and accurate diagnosis of inflammatory ON will likely be critical for implementing clinical care that optimizes short-term and long-term therapeutic outcomes.

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Inflammatory optic neuropathy, or optic neuritis (ON), is the most common cause of optic nerve injury in young adults. ON has multiple etiologies, including demyelinating, infectious, and autoimmune causes. Multiple population studies estimate an incidence of 1.5–5.1 cases per 100,000 person-years (1–3); however, epidemiologic studies may fail to capture cases of ON that are combined with other demyelinating events. Although the recovery of high-contrast visual acuity after ON is generally considered to be a favorable outcome (4), many patients complain of persistent visual problems (5). Since the seminal Optic Neuritis Treatment Trial (ONTT), there have been prospective and retrospective studies of acute ON therapy in a variety of autoimmune conditions. Initial reports suggested that ON associated with aquaporin-4 (AQP4) and myelin oligodendrocyte glycoprotein (MOG) autoantibodies are associated with poor (6,7) and good (8,9) visual outcomes, respectively. These observations, however, may be skewed by recurrent injury, the timing of therapy, and the choice of acute treatment (10,11). Nevertheless, these findings suggest that early diagnosis and disease-specific therapeutics may be key to minimizing injury, improving recovery, and preventing future vision loss after ON. This review will focus on formulating a modern rationale for the evaluation and treatment of acute inflammatory ON.

OPTIC NEURITIS: DIFFERENTIAL DIAGNOSIS

Inflammation of the optic nerve can arise from diverse pathologies. Although this review will focus on the expanding array of autoimmune disorders associated with ON, infectious (syphilis, Lyme, and cat-scratch disease) and noninfectious (sarcoidosis and paraneoplastic) causes of optic nerve inflammation should be carefully considered in the differential diagnosis under the appropriate clinical circumstances. Rapid diagnosis of an infectious cause of ON may direct antimicrobial and antiviral therapies, inform on visual prognosis, and mitigate the use of inappropriate immunosuppression. Conversely, the identification of a specific autoimmune-mediated pathology may heighten the need for aggressive immune suppression or the initiation of therapies to prevent recurrent ON or central nervous system (CNS) damage. Although it is not always easy to distinguish infectious from inflammatory causes of ON, Table 1 provides a brief list of infectious and inflammatory causes of ON along with their common clinical features and therapeutic options.

Due to the frequent presence of uveitis, retinitis, and chorioretinitis in infectious causes of ON, ON associated with ocular inflammation, optic nerve granuloma, or severe disc edema should raise clinical concern for acquired infection (Table 1). Fever, meningitis, cranial nerve palsies, and encephalopathy require a more detailed infectious workup based on endemic risks, exposures, and travel history.
### TABLE 1. Infectious and inflammatory causes of optic neuritis

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Common Clinical Features</th>
<th>Treatments</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syphilis (Treponema)</td>
<td>Uveitis, chorioretinitis, vasculitis, and papillitis (varied)</td>
<td>Penicillin</td>
<td>(87,88)</td>
</tr>
<tr>
<td>Intracellular pathogens (Bartonella, Rickettsia, Toxoplasma, and Coxiella)</td>
<td>Neuroretinitis (Bartonella and Toxoplasma), chorioretinitis (Bartonella and Toxoplasma), uveitis (Bartonella and Toxoplasma), fever (Coxiella and Rickettsia), encephalopathy (Coxiella), and CNS abnormalities (Coxiella and Rickettsia)</td>
<td>Corticosteroids; antibiotics: azithromycin, ciprofloxacin, doxycycline, tetracycline, and sulfamethoxazole-trimethoprim</td>
<td>(69,89–92)</td>
</tr>
<tr>
<td>Lyme disease (Borrelia)</td>
<td>Optic disc edema and reports of intermediate uveitis or papilledema</td>
<td>Ceftriaxone and doxycycline</td>
<td>(88,93,94)</td>
</tr>
<tr>
<td>Tuberculosis (Mycobacteria)</td>
<td>Papillitis, uveitis, neuroretinitis, scleritis, meningitis, optic nerve tubercle, and orbital apex syndrome</td>
<td>Isoniazid, rifampicin, pyrazinamide, and ethambutol</td>
<td>(95)</td>
</tr>
<tr>
<td>Viral (WNV, HIV, and VZV)</td>
<td>Variable: mild optic disc edema, chorioretinitis, and vitritis (WNV); normal, mild microangiopathy (HIV); hemorrhagic optic disc edema and cotton wool spots (VZV)</td>
<td>HAART (HIV); acyclovir (VZV)</td>
<td>(96–98)</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>Optic disc edema; granuloma, uveitis, neovascularization, CNS abnormalities; multisystem disease</td>
<td>Corticosteroids and TNF-α blocker</td>
<td>(12,13)</td>
</tr>
<tr>
<td>NMOSD</td>
<td>Recurrent; MRI–optic nerve enhancement/longitudinal lesions; chiasm, bilateral optic tract lesions; and AQP4-IgG</td>
<td>IVSM and PLEX</td>
<td>(20,22,23,57)</td>
</tr>
<tr>
<td>MOG</td>
<td>Recurrent; MRI–optic nerve, sheath, and orbital enhancement; longitudinal nerve lesions; disc edema; and MOG-IgG</td>
<td>Corticosteroids—may require prolonged treatment</td>
<td>(10,23,31,99)</td>
</tr>
<tr>
<td>GFAP</td>
<td>Optic disc papillitis; MRI–perivascular enhancement; and GFAP-IgG</td>
<td>Corticosteroids</td>
<td>(15,18,100)</td>
</tr>
<tr>
<td>Paraneoplastic</td>
<td>Bilateral disc edema; uveitis; vascular leakage; and paraneoplastic antibodies</td>
<td>IVlg; PLEX; corticosteroids; and identify and remove inciting neoplasm</td>
<td>(14,68,101)</td>
</tr>
<tr>
<td>Seronegative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple sclerosis (MS)</td>
<td>Mild disc edema; MRI–optic nerve enhancement, MRI findings consistent with MS, CSF OCBs</td>
<td>IVSM and PLEX</td>
<td>(4,23)</td>
</tr>
<tr>
<td>Other (CRION and AON)</td>
<td>Recurrent, isolated; MRI–optic nerve enhancement/T2 signal; IgG on skin biopsy</td>
<td>Corticosteroids</td>
<td>(84–86)</td>
</tr>
</tbody>
</table>

AON, autoimmune optic neuropathy; CNS, central nervous system; CRION, chronic relapsing inflammatory optic neuropathy; CSF OCBs, cerebrospinal fluid oligoclonal bands; GFAP, glial fibrillary acidic protein; HAART, highly active antiretroviral therapy; IgG, immunoglobulin G; IVlg, intravenous immunoglobulin; IVSM, intravenous solumedrol (methylprednisolone); MOG, myelin oligodendrocyte glycoprotein; NMOSD, neuromyelitis optica spectrum disorder; PLEX, plasmapheresis; TNF-α, tumor necrosis factor-alpha; VZV, Varicella-Zoster virus; WNV, West Nile virus.

Sarcoid ON and paraneoplastic ON also may demonstrate significant ocular inflammation. Sarcoidosis is a multisystem disorder that commonly involves the eye and rarely involves the CNS in isolation (12). As a result, the majority of patients with sarcoid ON have concurrent ocular inflammation; cranial neuropathies, meningeal granulomas, and endocrinopathies also may be evident (13). Elevated serum angiotensin converting enzyme levels are notoriously insensitive (13), and the identification of affected tissue for biopsy confirmation may be challenging. Patients with
paraneoplastic ON associated with collapsin response-mediated protein-5 autoantibodies (CRMP-5-IgG) also may present with prominent disc edema, vitreous cells, and retinal vascular leakage. Therefore, CRMP-5-IgG paraneoplastic ON should be considered in the differential diagnosis of patients with bilateral, subacute ON with disc edema and vitreous cells, particularly in association with progressive neurologic deficits (14).

Previous classifications of acute idiopathic ON have centered on clinical presentation, probability of multiple sclerosis (MS), risk of recurrence, and dependency on chronic corticosteroids. Diagnostic categories included chronic relapsing inflammatory ON (CRION), autoimmune optic neuropathy (AON), relapsing isolated ON (RION), MS-ON, neuromyelitis optica ON (NMO-ON), and single isolated ON (SION). Several of these categories, particularly CRION, RION, AON, and SION, lack both diagnostic specificity and sensitivity. As a result, they are not practical for directing ON patient management.

Recent advances in serologic testing have broadened the spectrum of CNS autoimmune disorders associated with acute ON. Currently, 3 disease-specific serologic markers, aquaporin-4-IgG (AQP4-IgG), MOG-IgG, and glial fibrillary acidic protein-IgG (GFAP-IgG), have been identified in patients with isolated and recurrent ON (10,15,16). Retrospective case studies suggest that visual recovery may differ significantly between these cohorts (17–20). Therefore, serologic categorization of patients with ON may offer important information on visual prognosis, treatment response, and risk of recurrence. The immediate evaluation of patients with acute ON should focus on acquiring clinical and paraclinical data that may help to distinguish one of these etiologies.

**EVALUATION OF THE PATIENT WITH ACUTE OPTIC NEURITIS**

The patient with acute ON needs a detailed neuro-ophthalmologic examination that carefully evaluates for the presence of ophthalmologic, neurologic, and systemic disease. The neuro-ophthalmologic examination may be accompanied by a number of ancillary laboratory and imaging studies based on history and examination findings including optical coherence tomography (OCT) (21), infectious and autoimmune serologies, MRI, and cerebrospinal fluid (CSF) analysis.

MRI of the orbits and brain may provide valuable diagnostic and prognostic information (Fig. 1 and Table 2). The distribution and appearance of inflammatory lesions on orbital MRI have been reported to show significant differences between ON associated with AQP4-IgG-seropositive neuromyelitis optica spectrum disorders (NMO-ON), MOG-IgG encephalomyelitis (MOG-ON), and seronegative MS-ON. Bilateral involvement is more common in NMO-ON and MOG-ON than in MS-ON, and chiasmal and optic tract lesions are more frequent in NMO-ON (22,23). Perineural enhancement is notable in MOG-ON (24). Although longitudinally extensive lesions are frequent in both NMO-ON and MOG-ON (22,23,25–27), the retrobulbar optic nerve is predominantly involved in MOG-ON, whereas the intracranial optic nerve is principally involved in NMO-ON (23). In contrast to MS-ON and MOG-ON, optic nerve lesion length correlates with visual outcome in NMO-ON (28,29). The presence and pattern of brain MRI lesions also may provide valuable diagnostic clues (15,23,30). A unique pattern of linear perivascular radial enhancement, extending outward from the ventricles, is observed on postcontrast gadolinium MRI in many patients with ON associated with glioblastoma acidic protein autoantibodies (GFAP-ON).

Autoimmune serology and CSF analysis may provide important clues to focus the differential diagnosis. Coexisting autoantibodies are more common in patients with NMO-ON and MOG-ON patients than in patients with MS-ON (31–33). In patients with GFAP-ON, serum and CSF neural autoantibodies are identified in approximately 40% of patients, with antibodies against N-methyl-D-aspartate receptor and glutamic acid decarboxylase 65 being the most common (15). In the CSF, significant pleocytosis (≥100 cells/mL) is observed more frequently in patients with MOG-ON than in patients with NMO-ON (28% vs 6%), with both disorders showing significant numbers of polymorphonuclear cells. Oligoclonal banding and intrathecal IgG synthesis are rare in MOG-ON and NMO-ON (31,34). CSF pleocytosis is frequently observed in patients with GFAP autoimmunity, and the presence of GFAP autoantibodies may be restricted to the spinal fluid (15).

In cases where there is concern for infection, patients with ON should be tested for syphilis, Lyme disease, Bar- tonella, and West Nile virus. In endemic areas, or with a history of exposure, quantiferon-gold testing for tuberculosis is warranted. Significant suspicion for sarcoidosis necessitates computed tomography of the chest or whole-body positron emission tomography because serum angiotensin converting enzyme and lysozyme testing are insensitive.

OCT may prove useful by identifying subtle retinal inflammation, macular edema, microcystic change, or neo-vascularization. OCTs performed on patients with acute ON have documented retinal nerve fiber layer (RNFL) edema, ganglion cell plus inner plexiform layer (GC+IPL) thinning, and peripapillary RNFL loss (10,35–40). Optic disc edema, as measured by OCT and fundus examination, is more common in MOG-ON than in NMO-ON (9,23). In the acute setting, however, the correlation between OCT metrics, visual prognosis, and therapeutic response is unclear; therefore, OCT is not recommended for guiding acute ON treatment. In cases of recurrent ON, however, the extent and pattern of RNFL loss and retinal
thinning in a previously affected optic nerve may be informative (10,38,39).

**ACUTE THERAPIES FOR OPTIC NEURITIS**

Since the completion of the ONTT in 1992, high-dose intravenous methylprednisolone (IVMP) has been the treatment of choice for immediate therapy of acute ON. IVMP (1,000 mg daily for 3 days) followed by oral prednisone (1 mg/kg/d for 11 days) accelerated visual recovery and improved short-term, but not long-term, functional outcomes (4,41). Additional studies have failed to demonstrate any effect of high-dose IVMP or oral corticosteroids on long-term function or the subsequent development of optic nerve atrophy (42–44). Nevertheless, the benefit of accelerated visual recovery combined with an acceptable level of side effects has prompted the routine adoption of IVMP for acute ON treatment. Intramuscular or subcutaneous adrenocorticotropic hormone is also approved for the treatment of ON and MS-related relapses, providing an alternative option for hypothalamic–pituitary–adrenal axis modulation (45). Given the excellent bioavailability of orally administered corticosteroids (46), various oral regimens are now available to administer.

**FIG. 1.** MRI of optic neuritis. **A.** Postcontrast axial T1 scan with fat suppression shows a longitudinally extensive lesion involving the intraorbital left optic nerve in a patient with optic neuritis and MOG-IgG. **B.** Postcontrast coronal T1 fat-suppressed image demonstrates prominent optic nerve sheath and nerve enhancement. Axial (C) and coronal (D) T1 views of a lesion involving the right intracranial optic nerve and optic chiasm in a patient with optic neuritis and NMOSD. **E.** Postcontrast coronal T1 fat-suppressed image reveals enhancement of both optic nerves and surrounding sheaths in a patient with optic neuritis and MOG-IgG. MOG-IgG, myelin oligodendrocyte glycoprotein autoantibodies; NMOSD, neuromyelitis optica spectrum disorder.
corticosteroids at bioequivalent doses to high-dose IVMP, providing added therapeutic flexibility (47–49). Oral prednisone at lower doses, however, should be avoided because it increases the risk of ON relapse (4).

Despite extensive clinical data supporting the benefit and safety of high-dose corticosteroid administration for acute ON treatment, it remains unclear whether early treatment with high-dose corticosteroids is optimal for all causes of ON, and whether other treatments should be substituted or combined with corticosteroids to improve visual outcome. Visual outcomes after individual attacks of NMOSD-ON are significantly worse than MS-ON and MOG-ON (6,17,19,20). In patients seropositive for GFAP-IgG, optic nerve papillitis without changes in visual acuity is reported (18). Peripapillary RNFL loss and GC+IPL thinning are correspondingly increased in NMOSD-ON (9,10,22,38,39); however, repeated events of ON in MOG-IgG-seropositive patients often leads to RNFL and retinal OCT measures that are comparable with NMOSD-ON patients (9,10). Interestingly, when serum samples from 177 of 448 patients enrolled in the ONTT were assayed for AQP4- and MOG-IgG, only 4 MOG-IgG-seropositive patients were identified (50). Therefore, the results of the ONTT are not informative on the impact of high-dose corticosteroids on visual recovery in NMOSD-ON and MOG-ON. A retrospective analysis of treatment responses in NMOSD attacks indicates that therapeutic benefit from intravenous solumedrol (IVSM) is often incomplete, and repeated administration does not improve the rate of complete remission (11). Among 232 isolated NMOSD-ON attacks, only 77 patients (33%) showed complete remission after treatment with high-dose IVSM; repeat courses did not increase the number of complete responders but only reduced the number of nonresponders. Response rates were even more discouraging in NMOSD-ON patients with concurrent myelitis.

Intravenous immunoglobulin (IVIg) and plasma exchange (PLEX) are alternative immunomodulatory therapies that may offer additional benefit for acute ON treatment. In a placebo-controlled trial of acute ON, IVIg (0.4 g/kg) did not improve contrast sensitivity or visual function at 6 months after injury (51). The identical dosage did not improve refractory vision loss in patients with MS-ON (52). Patient response in both studies, however, may have been limited by delayed IVIg administration: 4 weeks in the acute ON trial and an average of 4 years in the refractory ON trial. In an experimental rat model of NMOSD lesions (53), human IVIg reduced lesion formation by inhibiting complement-dependent and cell-mediated cytotoxicity. Therefore, it remains to be determined whether IVIg may benefit patients with NMOSD-ON when delivered in conjunction or after a round of high-dose corticosteroids.

PLEX has been used successfully in the treatment of steroid refractory ON and NMOSD-ON (54–58). Depending on the study, improvement in visual function has been noted in 45%–55% of treated patients. Unfortunately, due to their retrospective design, these investigations failed to define criteria for the optimal use or timing of PLEX (59). In many instances, the short interval between completion of IVMP and institution of PLEX makes it unclear how much clinical benefit is due to delayed effects of IVMP. Male sex, lower baseline disability, rapid initiation of treatment, and shorter relapse duration have been associated with greater response to PLEX (11,60–62). Although early initiation of PLEX correlates with treatment response,
delayed PLEX therapy may still be a reasonable treatment option for patients with acute ON who may not have immediate access to facilities with the necessary equipment. Deschamps et al (56) found that half of the patients with poor visual recovery (visual acuity worse than or equal to 20/200) after high-dose IVSM improved to visual acuity of 20/30 or better after PLEX (mean time to PLEX: 30 days). Because PLEX incurs significant cost and may result in serious side effects such as hypotension, infection, hypocalcemia, and coagulopathy, a randomized, prospective study of PLEX versus IVMP for the treatment of acute NMOSD-ON is warranted.

Immunoadsorption (IA) is an alternative form of therapeutic apheresis that allows for selective removal of antibodies from plasma using modified membranes. Therapeutic apheresis offers the potential advantage of removing pathogenic autoantibodies while sparing other plasma proteins, therefore eliminating the need for protein replacement and potentially minimizing complications. Immunoadsorption has been reported to benefit steroid refractory ON and NMOSD-ON (63,64). The relative efficacy and safety of PLEX and IA are yet to be directly evaluated. Currently, IA is not approved in the United States.

For individuals who are unresponsive to IVMP and PLEX, immunosuppression with intravenous cyclophosphamide may represent an avenue of final resort. Although no clinical studies have been published on the response of severe ON to intravenous cyclophosphamide, a subset of patients with acute transverse myelitis have benefited from this approach (65). Given the risks of high-dose IV cyclophosphamide, however, careful patient selection and an experienced hospital team are advised.

Recent prospective studies have evaluated novel therapeutic approaches for neuroprotection and remyelination in acute ON. In a Phase 2 randomized controlled trial, phenytoin was shown to ameliorate RNFL loss in acute ON. Treatment with phenytoin, however, had no effect on visual outcomes or visual evoked potentials (VEPs) (66). Opicinumab, a human monoclonal antibody against leucine-rich repeat and immunoglobulin domain-containing

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**FIG. 2.** Flowchart outlining a prospective approach to acute ON treatment. “Recurrent,” repeat event of acute ON in a previously affected eye. “High-dose IVMP,” high-dose intravenous methylprednisolone, subcutaneous/intramuscular adrenocorticotropic hormone, or oral high-dose corticosteroid bioequivalent. “High-dose IVMP + PLEX/IA,” high-dose IVMP with plasma exchange/immunoadsorption performed concurrently or within 5 days. “High-dose IVMP or previous Rx,” previous successful acute therapy. “Antibiotics,” appropriate antimicrobial or antiviral agent. AQP4, aquaporin-4; IA, immunoadsorption; IVMP, intravenous methylprednisolone; MOG, myelin oligodendrocyte glycoprotein; MS, multiple sclerosis; ON, optic neuritis; PLEX, plasmapheresis.
neurite outgrowth inhibitor receptor-interacting protein-1 (anti-LINGO-1), was recently investigated as a potential remyelinating therapy in acute ON. Treatment with opicinumab produced no significant change in the VEP latency at 24 weeks in the intention-to-treat population; however, significant improvement in VEP latency delay was observed at 24 and 32 weeks in the prespecified per-protocol patient population (67). There was no effect of anti-LINGO-1 treatment on RNFL or GC+IPL thickness in either the intention-to-treat or per-protocol patient population at 24 weeks. Nevertheless, the addition of phenytoin or opicinumab to more potent immunomodulatory therapies may still benefit visual outcomes in patients with acute ON. Larger clinical trials that are powered to determine clinical efficacy will be necessary.

A TREATMENT RATIONALE FOR ACUTE OPTIC NEURITIS

As the clinical landscape of acute ON becomes increasingly complex, affected patients may benefit from a therapeutic rationale that emphasizes early use of PLEX or IA in clinical circumstances that are concerning for poor visual prognosis (Fig. 2). As noted previously, NMOSD and recurrent ON (ON in a previously affected optic nerve) have poorer visual outcomes (10,17,19,20). Nevertheless, in the absence of prospective clinical data, such an approach would need to be tempered by clinical judgment. In cases of known seropositive ON (e.g., MOG-ON, NMOSD-ON, and GFAP-ON), the treatment decision may be rather straightforward; however, in seronegative ON cases, laboratory and imaging data must be balanced against clinical history (e.g., previous ON treatment response) to gauge whether initial high-dose IVSM with or without PLEX is warranted. If high-dose IVSM is used as monotherapy, it is important to predetermine criteria for therapeutic success because the timing of PLEX may be critical for the treatment of conditions such as NMOSD-ON (61). Seropositive MOG-ON and GFAP-ON often are responsive to high-dose corticosteroids (8,9,15); therefore, forbearance in anticipation of clinical improvement is warranted. For paraneoplastic CRMP-5-ON, the use of intravitreal triamcinolone may be worthwhile (68).

In cases of suspected infectious ON, such as Lyme disease, syphilis, and tuberculosis, it is prudent to begin appropriate antibiotics as soon as possible. Unless otherwise contraindicated, antibiotic therapy should be initiated with symptomatic therapy if suspicion for infection is high. Antibiotic therapy may be suspended or tailored based on subsequent imaging or diagnostic studies. In Bartonella infection, however, the benefit of antibiotic therapy is unclear. In Bartonella ON, antibiotic treatment might be limited to individuals with profound visual loss, systemic infection, or immunocompromised status (69).

A treatment rationale for ON would be incomplete without consideration of the potential for recurrent vision loss. As ON is routinely associated with RNFL and GC+IPL loss, the ideal method for optimizing long-term visual outcome is the prevention of future attacks, particularly with NMOSD-ON and MOG-ON. Although there are no Food and Drug Administration–approved therapies for the treatment of NMOSD, there are studies supporting the off-label use of azathioprine, mycophenolate mofetil, methotrexate, rituximab, and tocilizumab (70–74). Differentiating NMOSD from MS is important because worsening disease has been reported after treatment with several approved MS therapies: beta-interferon (75), natalizumab (76), dimethyl fumarate (77), alemtuzumab (78), and fingolimod (79). Recurrent events of ON and transverse myelitis have been reported in patients seropositive for MOG-IgG (80–82). Optimal steroid and steroid-sparing regimens for relapsing adult and pediatric patients are yet to be determined but may comprise a mixture of those used in MS and NMOSD (80,83). AON (84) and CRION (85,86) are defined as cases of seronegative recurrent ON that are steroid-responsive and steroid-dependent. Whether it is prudent to maintain this historical designation is debatable because it is likely that these labels will diminish in use as continual improvements in immunologic and molecular testing are used to define ON nosology.

SUMMARY

Acute ON is caused by an increasingly complex number of disorders that may be differentiated by history, funduscopy, imaging, and serology. Recent immunologic studies have demonstrated that visual recovery after autoimmune ON is not uniformly promising, and the routine use of high-dose corticosteroids to accelerate recovery may be insufficient in certain circumstances. Acute ON treatment is likely to benefit from a focused evaluation designed to identify imaging and laboratory data that favor certain infectious, paraneoplastic, and autoimmune etiologies. Using these results, treatment may be tailored to respond to the mechanism of injury and visual prognosis. Prospective treatment trials designed to investigate aggressive treatment regimens in acute inflammatory ON are likely to support novel treatment rationales that accelerate recovery and preserve visual function and structural components of the optic nerve.

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