(100 mg). He was placed on intravenous ceftazidime (1.0 g/8 hours), topical vancomycin (35 mg/ml), and amikacin (20 mg/ml). Culture of material obtained from the initial vitreous tap grew *Neisseria meningitidis* group Y. On postvitrectomy day 1, the eye was less inflamed. Oral prednisone (80 mg/day) and 1% prednisolone acetate drops every 2 hours were begun. He continued to improve, and the antibiotic eyedrops and corticosteroids were slowly tapered. He was discharged on day 5 on 1% ciprofloxacin drops, 1% prednisolone acetate drops, oral ciprofloxacin (500 mg, twice a day), and a tapering dose of oral prednisone. Two months after vitrectomy, his vision improved to hand motions. The patient declined further intervention to remove vitreous opacities. There were no cases of meningococcal disease in any patient contacts.

N meningitidis, a gram-negative diplococcus, is the second most common cause of bacterial meningitis in patients over 1 year of age.[1] The bacteria is a rare intraocular pathogen. Although several cases of endogenous *N meningitidis* endophthalmitis have been previously reported,[2,3] only one case of exogenously derived endophthalmitis has been previously reported.[4]

The nasopharynx is the natural reservoir of *N meningitidis*. Systemic disease, including meningitis, occurs when the meningococcus crosses the nasopharyngeal mucosa and enters the bloodstream. The factors that lead to invasive disease are incompletely understood but include the virulence of the *N meningitidis* strain as well as host factors such as antibodies and intercurrent respiratory tract infections. The rare case reports of endogenous meningococcal endophthalmitis without signs of systemic infection[5] probably result from ocular seeding during transient bacteremia.

Individuals who have had close contact with patients carrying invasive meningococcal disease are 500 to 800 times more likely to become infected than the general population.[1] Consequently, antibiotic chemoprophylaxis, usually rifampin, is recommended to eradicate the carrier state in patient contacts. In contrast, *N meningitidis* can be isolated from the oropharyngeal secretions of up to 10% of the normal healthy population without exposure to meningococcal infection.[1] These colonized individuals and the individuals they come into contact with are not at increased risk of developing invasive disease and do not require chemoprophylaxis.

It is likely that our patient was colonized by *N meningitidis* and that his infection was a consequence of direct inoculation through his leaking filtering bleb. There was no evidence of bloodstream invasion, and thus the organism did not demonstrate the virulent properties associated with invasive disease nor the capacity to cause secondary cases. Consequently, we did not recommend antibiotic prophylaxis to the patient's close contacts. We think it is reasonable to consider exogenous *N meningitidis* endophthalmitis as no more virulent or communicable than incidental *N meningitidis* pharyngeal colonization. However, administering chemoprophylaxis to close contacts of patients with *N meningitidis* endogenous endophthalmitis may be a prudent preventative measure.

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Chiasmal Optic Neuritis in Lyme Disease

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PURPOSE: To report Lyme disease as the cause of chiasmal optic neuritis in a 10-year-old girl.
METHODS: The patient underwent ophthalmologic, laboratory, and imaging examinations.

RESULTS: **The patient's history and clinical course were consistent with Lyme disease. Laboratory studies disclosed increased serum Lyme immunoglobulin G titer, which improved after antibiotic treatment.**

CONCLUSION: **Lyme disease should be considered in the differential diagnosis of chiasmal optic neuritis.**

CHIASMAL OPTIC NEURITIS IS OFTEN IDIOPATHIC BUT has been associated with multiple sclerosis and with varicella and mononucleosis infections. We report chiasmal optic neuritis in a patient with Lyme disease.

Three months before initial examination, a previously healthy 10-year-old girl noted decreased peripheral vision in her right eye. At that time, pediatric consultation disclosed a visual acuity without correction of BE, 20/20. Right-eye vision gradually declined, and the patient developed sharp pain over the right temple. Review of systems showed a 3-month history of fatigue and ankle and wrist pain. The patient had been camping in Oregon 4 months earlier; she did not recall receiving insect bites or having skin rashes.

On examination, best-corrected visual acuity was RE, light perception and LE, 20/15 with a full field. There was a marked right afferent pupillary defect. Extraocular movements, intraocular pressures, and slit-lamp examination were unremarkable in both eyes. Dilated ophthalmoscopic examination showed severe optic disk pallor diagnostic of optic atrophy in the right eye and no abnormality in the left eye.

A complete blood cell count, rapid plasma reagin test, antinuclear antibody titer, angiotensin I-converting enzyme level, Epstein-Barr and varicella virus antibody titers, and chest x-ray were within normal limits. Lumbar puncture disclosed normal opening pressure, no cells, and normal chemistry. Cerebrospinal fluid immunoglobulin (Ig) G myelin basic protein, cryptococcal antigen, Venereal Disease Research Laboratories test, and Lyme antibody were within normal limits. Serum Lyme immunofluorescent testing demonstrated a negative IgM titer, but IgG titers were positive to 1:512, and four of 12 bands were positive on Western blot analysis. Fluorescent treponemal antibody testing was also positive.

Echographically, the right optic nerve-sheath complex measured 4.2 mm anteriorly and decreased to 3.2 mm with the eye abducted 30 degrees, indicative of increased subarachnoid fluid (positive 30-degree test) as opposed to solid thickening of the nerve. The left optic nerve was normal. Brain magnetic resonance imaging demonstrated thickening and gadolinium enhancement of the right optic nerve and anterior chiasm (Figure 1).

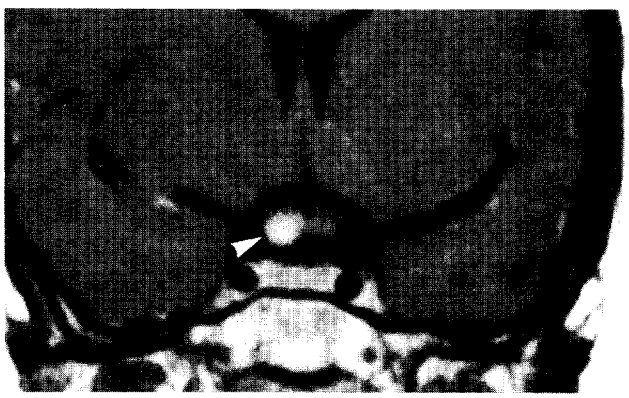

Figure 1. Coronal T_1-weighted gadolinium-enhanced magnetic resonance imaging reveals marked enlargement and enhancement of the right intracranial optic nerve and chiasm (arrowhead).

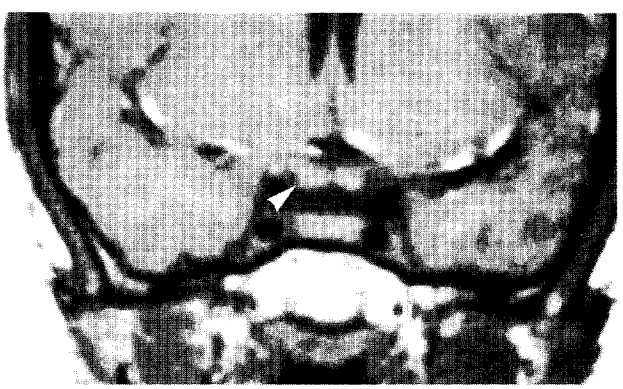

Figure 2. Posttreatment study reveals dramatic improvement in the thickening and gadolinium enhancement of the right intracranial optic nerve (arrowhead).

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The patient was treated for 3 days with intravenous methylprednisolone (125 mg every 6 hours), followed by oral prednisone (60 mg daily, tapered over 4 weeks). After her positive serum Lyme titer was known, she received intravenous ceftriaxone for 14 days (1 g daily) and oral doxycycline for 4 weeks (100 mg 3 times per day). There was no change in visual function. Her right temple pain, fatigue, and arthralgias resolved. After treatment, the patient's serum Lyme IgG titer decreased to 1:64, serum fluorescent treponemal antibody was negative, and brain magnetic resonance imaging disclosed only minimal enlargement of the right side of the optic chiasm, with virtually no gadolinium enhancement (Figure 2).

The patient's history, clinical course, and laboratory findings were consistent with Lyme disease. Idiopathic childhood optic neuritis generally manifests as acute visual loss, with excellent prognosis for recovery. Lyme borreliosis may be seronegative in up to 50% of cases,[1,2] and a high proportion of patients with suspected neuroborreliosis, in whom *Borrelia burgdorferi* was detected by polymerase chain reaction, had negative cerebrospinal fluid Lyme antibody tests.[3] Furthermore, nearly 25% of patients with Lyme disease have been shown to have false-positive fluorescent treponemal antibody tests.[4] Many neuro-ophthalmologic manifestations of Lyme disease have been described, including bilateral optic neuritis.[5] The case described in this report suggests that Lyme disease should be considered in the differential diagnosis of chiasmal optic neuritis; delayed treatment may result in permanent visual loss.

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Identification of Anterior Uveal Tumor Border by Transscleral Transillumination and an Ophthalmic Endoscope

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PURPOSE: To report a method for identifying the borders of anterior uveal tumors in deeply pigmented eyes.

METHOD: The borders of the anterior uveal tumors were marked by transscleral transillumination with a fiberoptic light and observed from the vitreous cavity using an ophthalmic endoscope.

RESULTS: The ophthalmic endoscope disclosed a bright orange spot by transscleral transillumination of a normal uvea in deeply pigmented eyes. Two anterior uveal melanomas and one adenocarcinoma of the nonpigmented ciliary epithelium did not transmit light through the tumors.

CONCLUSION: With an ophthalmic endoscope and transscleral transillumination, the borders of ciliary body tumors can be identified and clearly demarcated, even in deeply pigmented eyes.

ONE POSSIBLE CHOICE FOR MANAGING CHOROIDAL and ciliary body malignant melanoma is surgical resection.[1,2] Although small and localized residual tumors can be successfully treated by laser therapy, complete resection at the time of initial surgery is important. Transscleral transillumination using a bright fiberoptic light is useful in identifying the margin of a uveal melanoma, especially in the anterior section; in deeply pigmented eyes, however, demarcation of the tumor margin may not be possible, although recently, ophthalmic endoscopy has been widely used for observing the ciliary body.[3-5]

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