pneumonia.” There is a higher prevalence of infection in children and young adults (up to 40% in children), and reports of outbreaks are common in closed populations such as those of military recruits and college students.\(^1\) All 3 patients in our series presented to our service within a 2-month period, and 2 of the patients were college students. Although permanent vision loss from optic papillitis has been previously reported, all 3 of our patients recovered their baseline visual acuity within 1 month of treatment.

*Mycoplasma pneumoniae* contains both glycolipid and protein antigens that elicit antibody responses in infected individuals.\(^1\) Circulating immune complexes have been found in the serum of patients with *M pneumoniae*-associated pneumonia, and this type III hypersensitivity reaction is believed to cause microvasculitis from immune complexes depositing along vessel walls in the eye.\(^2,3\) Another mechanism by which *Mycoplasma* infection is thought to cause uveitis is through direct invasion of the bacteria in the anterior chamber.\(^3\) The most common method for diagnosing *Mycoplasma* infection is through serology testing for IgM and IgG antibodies. Direct polymerase chain reaction analysis is still not widely available, and a culture of *M pneumoniae* takes up to several weeks. The enzyme-linked immunosorbent assay test used at our institution has a sensitivity and specificity of 94.5% and 87.5%, respectively.

There have been only 5 reported cases in the literature of anterior uveitis secondary to *M pneumoniae* (Table).\(^2-6\) In 3 of the 5 cases, concomitant findings of bilateral optic disc edema and anterior uveitis were reported, but those patients had normal retinal findings.\(^2,5,6\) Case 2 in our series is, to our knowledge, the first case of uveitis from *M pneumoniae* associated with bilateral macular and peripapillary serous detachments, but perhaps this is a more severe presentation along the same clinical spectrum of inflammatory disease. Of our 3 patients, only the 20-year-old man who presented with a 1-month history of blurry vision (case 1) had pneumonia secondary to his infection, which underscores the fact that extrapulmonary involvement can occur even in the absence of clinically overt respiratory tract disease. We hope that our case series will help highlight this common human pathogen as a cause of uveitis and papillitis, and we believe that *M pneumoniae* should be included in the differential diagnosis when a young and otherwise healthy person presents with uveitis or optic disc edema preceded by a febrile illness.

<table>
<thead>
<tr>
<th>Source</th>
<th>Patients</th>
<th>Ocular Findings</th>
<th>Systemic Findings</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salzman et al,(^2) 1992</td>
<td>Male 15</td>
<td>Bilateral optic disc edema and iritis</td>
<td>Gastroenteritis, fever, and erythema multiforme</td>
<td>Optic disc edema and iritis resolved after 6 wk</td>
</tr>
<tr>
<td>Davidek et al,(^3) 1991</td>
<td>Male 36</td>
<td>Bilateral optic disc edema and iritis</td>
<td>Fever, cough, and macular rash on lower extremities</td>
<td>Recurrence of iritis 1 mo after presentation; resolution after 3 mo</td>
</tr>
<tr>
<td>Di Maria et al,(^4) 1999</td>
<td>Male 38</td>
<td>Bilateral optic disc edema and iritis</td>
<td>Fever, cough, headache, erythematous rash, and pneumonia</td>
<td>Iritis resolved after 2 mo</td>
</tr>
<tr>
<td>Yashar et al,(^5) 2001</td>
<td>Female 15</td>
<td>Bilateral panuveitis, Roth spots, and retinal hemorrhages</td>
<td>Meningitis, fever, vomiting, myalgia, dizziness, and pleuritic chest pain</td>
<td>Panuveitis and Roth spots resolved after 3 wk</td>
</tr>
<tr>
<td>Weinstein et al,(^6) 2006</td>
<td>Female 5</td>
<td>Bilateral optic disc edema and iritis</td>
<td>Fever, arthralgia, abdominal pain, and lethargy</td>
<td>Optic disc edema and iritis resolved after 6 wk</td>
</tr>
</tbody>
</table>

Optic Nerve Involvement From *Pseudomonas aeruginosa*-Associated Skull Base Osteomyelitis

Skull base osteomyelitis (SBO) is an uncommon but life-threatening condition caused by invasive bacterial or fungal infection.\(^1\) Diabetes mellitus, chronic otitis externa, and immunosuppression are frequently associated with SBO.\(^1,2\) More than 60% of patients with SBO are men, and *Pseudomonas aeruginosa* is the most common bacterial pathogen identified in SBO.\(^1,2\) Cranial nerve palsies most frequently involve cranial nerve VII, followed by the lower cranial nerves (IX-XI).\(^1,2\) We report 2 cases of SBO with rare unilateral optic nerve in-
volvement resulting in no light perception (NLP). These cases highlight the importance of suspecting SBO based on magnetic resonance imaging (MRI), the necessity of a biopsy, and prompt treatment with appropriate intravenous antibiotics.

Report of Cases. Case 1. On February 3, 2009, a 65-year-old healthy man was evaluated for painful visual loss in his right eye for 8 days. The right retro-orbital and scalp pain were unchanged 5 days after treatment with oral prednisone (80 mg/d). Best visual acuity was 20/50 OD and 20/25 OS with a right afferent pupillary defect. The results of the ocular and fundus examinations were otherwise normal. Blood analysis showed a normal erythrocyte sedimentation rate and a normal C-reactive protein level.

The visual acuity in his right eye decreased to 20/400 the next day. An orbital MRI scan showed mild enhancement of the right superior orbital fissure and planum sphenoidalis (Figure 1). The patient was given 1 g/d of methylprednisolone sodium succinate intravenously for 3 days followed by treatment with oral prednisone for 11 days. The patient still reported having extreme pain without visual improvement. The results of a lumbar puncture were normal.

By February 23, 2009, the patient’s visual acuity was NLP OD, and he developed right third and sixth cranial nerve palsies. The results of another MRI showed mild, stable enhancement of the right optic nerve sheath with increased enhancement and prominence of soft tissues at the right orbital apex and cavernous sinus (Figure 1). The results of computed tomography of the chest, abdomen, and pelvis were normal.

The results of a biopsy of the right ethmoid sinuses were unrevealing, leading to an equally nondiagnostic optic nerve and sheath biopsy. Empirical antifungal therapy resulted in no improvement in pain, vision, or ophthalmoplegia. A transcranial biopsy of the swollen, posterior optic canal dura showed that P aeruginosa grew on the tissue sample, which was observed with hematoxylin-eosin staining (Figure 1). Six weeks of treatment with intravenous meropenem decreased the patient’s pain level and decreased his symptoms of ophthalmoplegia, but his visual acuity was NLP OD. The results of an MRI of the brain and orbits 1 week later showed only postsurgical changes.

Case 2. A 56-year-old man with uncontrolled diabetes and hypertension was hospitalized on June 18, 2011, for excruciating right facial pain and decreased hearing for 1 month. The results of an initial ophthalmic examination were unremarkable, but 1 week later, his visual acuity was NLP OD with total ophthalmoplegia. Plosis of the right eye and a small nonreactive right pupil were present, with no optic disc edema in either eye. Sensation of the right face was decreased 50% and was worse in the second and third trigeminal divisions. The muscles of mastication were atrophic on the right. The patient had difficulty chewing, and his smile was asymmetric. He developed severe pharyngeal dysphagia, an ineffective cough reflex, and an inability to swallow, which led to the placement of a percutaneous endoscopic gastrostomy tube. In all, there was right optic neuropathy with ipsilateral palsy of the cranial nerves III through XII. The results of an MRI revealed leptomeningeal enhancement of the right temporal skull base and a cavernous sinus extending through the foramen ovale into the infratemporal fossa (Figure 2).

A right temporal craniotomy with extradural biopsy demonstrated a mixture of CD3-positive T cells and CD20-positive B cells that were not malignant. Acid-fast bacilli and Gomori methenamine silver stains were negative for acid-fast bacilli and fungi, respectively. Immunostains were negative for carcinoma and glial tissue. The results of a subsequent nasopharyngeal biopsy were equally nondiagnostic. The results of a C1–C2 cervical puncture revealed normal opening pressure, and the puncture itself allowed for analysis of cerebrospinal fluid samples. An endoscopic biopsy of deeper pharyngeal tissue displayed a few budding yeast forms without tissue invasion, and the tissue sample eventually grew P aeruginosa.

The patient’s condition was diagnosed as SBO, and he was given cefepime hydrochloride intravenously. Although the patient’s polyneuropathy improved and his visual acuity improved to hand motions after 6 weeks, he was rehospitalized with fever and hyperglycemia, which required that he receive an additional 6 weeks of...
antibiotics to resolve the symptoms. His visual acuity in the right eye remained hand motions beyond 3 months after treatment but returned to NLP by 8 months.

Comment. Skull base osteomyelitis is well reported in the literature, with a few early reports of optic nerve involvement that provide no details of visual function.1 Girkin et al1 described a case of P aeruginosa–associated SBO involving the right optic nerve and multiple bilateral cranial nerve palsies in the setting of pachymeningitis. After 4 weeks of intravenous cefazidime and tobramycin sulfate, the patient’s visual acuity in the right eye improved from NLP to hand motions, with resolution of dysphagia and right third and sixth cranial nerve palsies; the left sixth cranial nerve palsy was persistent, and the patient’s hearing loss progressed to complete deafness.

Our cases had unilateral involvement of the optic nerve from P aeruginosa–associated SBO without pachymeningitis. Visual acuity progressed to NLP despite treatment and improvement in other cranial nerve palsies. In both cases, findings on MRI scans prompted multiple biopsies; however, we were unable to identify the organisms on pathology. Cultures of the samples from the last biopsy of each case eventually revealed P aeruginosa. This underscores the need for persistence in obtaining tissue to determine diagnosis, assess antibiotic sensitivity, and initiate prompt appropriate intravenous antibiotic treatment, which is essential to preserve the uninvolved optic nerve and reduce mortality.3

Suspicion of P aeruginosa–associated SBO is warranted in patients with painful progressive multiple cranial nerve palsies in combination with MRI-detected skull base lesions and/or meningeval enhancement. Although diabetes is a consistent risk factor for SBO, our first patient (case 1) had no diabetes and was not immunocompromised. Vision loss may progress to NLP as early as 1 week after optic nerve involvement, and although empirical therapy is likely beneficial, most cases require at least 6 weeks of appropriate intravenous antibiotics based on antibiotic sensitivity.5 Treatment of P aeruginosa–associated SBO must be based on tissue diagnosis, which may require multiple biopsies for pathologic and/or culture confirmation.

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Conflict of Interest Disclosures: None reported.

Funding/Support: This work was supported by National Institutes of Health grant P30-EY014801 and an unrestricted grant from Research to Prevent Blindness.


Didanosine-Associated Retinal Toxicity in Adults Infected With Human Immunodeficiency Virus

Intraocular toxicity from didanosine was first reported in immunocompromised children who demonstrated peripheral chorioretinal atrophy after taking the drug.1 To date, there have been only 2 reports of didanosine-associated retinal toxicity in adults.2,3

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Figure 2. A, T1-weighted coronal magnetic resonance imaging scan with fat saturation and gadolinium enhancement of the right temporal skull base and cavernous sinus (top arrow), foramen ovale (curved arrow), and subtemporal space (bottom arrow) of a 56-year-old man (case 2). One week later, the right optic nerve was enlarged and thickened with enhancement of the perineural tissue (bottom arrow) and a concurrent superior ophthalmic vein thrombosis (top arrow) (B). Axial computed tomography without contrast demonstrates sclerosis of the petrous bone (long arrow) and apex (short arrow) (C).