ected causative agents including miotic and nonmiotic antiglaucoma drugs, mydriatics, preservatives, antiviral compounds, sulfonamides, penicillin, hydrocortisone, epidermal growth factor, prostaglandins, practolol, and thyroid hormone, none of which have been used by the 2 patients reported herein. In most instances, the prolonged use of a topical medication seems to induce conjunctival epidermidalization, which may resolve over the course of a few months, after the drug is discontinued. Persistent or recurrent inflammation of the conjunctivae, as was observed in case 1, can itself lead to epidermidalization. It has been proposed that the mechanism probably involves a region-specific cellular immune response to the topical application, rather than a drug mechanism—specific reaction, because the compounds that are responsible vary widely in chemical structure and therapeutic effect.3

Conjunctival epidermidalization has been associated with ocular drying related to exposure, Stevens-Johnson syndrome, and avitaminosis A with xerophthalmia.4 Because ocular drying was present in case 2, it is a possible mechanism. Based on the clinical and histopathologic findings in these 2 unrelated patients, we propose that the administration of risendronate sodium is the primary cause of their conjunctival epidermidalization. Although the mechanisms for the development of Actonel-induced epidermidalization are not fully understood, potential mechanisms include conjunctival inflammation and dry eye. Conjunctival epidermidalization constitutes a serious, vision-threatening condition that is likely to resolve after discontinuation of the drug, but it could lead to vision loss if the drug is continued. Therefore, clinicians should be aware that conjunctival epidermidalization is a potentially dangerous adverse effect of risendronate sodium.

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3. Wright P. Squamous metaplasia or epidermidalization of the conjunctiva as an adverse reaction to topical medication. Trans Ophthalmol Soc UK. 1979;99(2):244-246.


Mycoplasma pneumoniae: The Other Masquerader

Mycoplasma pneumoniae is a bacterium in the class of Mollicutes and is a common cause of atypical pneumonia, particularly in children and young adults. A Mycoplasma infection primarily manifests as a respiratory tract disease, but an extrapolmonary manifestation has occurred in up to 25% of infected patients. The organ systems that may be involved include the skin, the gastrointestinal tract, and the musculoskeletal, cardiac, renal, hematopoietic, and nervous systems.5 Ocular disease from M pneumoniae has also been reported, with conjunctivitis being the most frequent finding.2 Less common ophthalmologic manifestations of M pneumoniae include cranial neurapthies, optic papillitis, and anterior uveitis.2 We present herein 1 case of bilateral optic papillitis and 2 cases of uveitis secondary to M pneumoniae infection.

Report of Cases. Case 1. A 20-year-old man presented with a 1-month history of blurry vision. Prior to presentation, he completed a 1-week course of oral levofloxacin for community-acquired pneumonia. His visual acuity was 20/25 in the right eye and 20/30 in the left eye. The ophthalmologic findings were normal, except for bilateral optic disc edema (Figure 1A and B). The results of neuroimaging using magnetic resonance imaging and all the cerebrospinal fluid parameters, including opening pressure level and cell counts, were normal. The results of a laboratory workup for Lyme disease, syphilis, and Bartonella henselae were negative. The results of a chest radiograph were normal, and the levels of angiotensin-converting enzyme and calcium were within normal limits. Because of his recent outpatient treatment for community-acquired pneumonia, serology testing for M pneumoniae was performed, and elevated IgM and IgG antibodies were found. The patient was treated with a 1-week course of oral azithromycin. One month after presentation, the patient’s visual acuity returned to 20/20 in both eyes, and his optic disc edema had resolved.

Case 2. A 14-year-old boy presented with a 1-month history of bilateral eye pain, blurry vision, headache, and subjective fevers. On physical examination, he was found to have an erythematous macular rash on his lower extremities. His visual acuity was 20/40 and 20/50 in the right and left eyes, respectively. An ophthalmologic examination revealed bilateral conjunctival injection, with 3+ cell and flare in the anterior chambers. A dilated fundus examination showed 2+ cell in the vitreous and bilateral optic disc edema and serous macular detachments (Figure 2A and B). There
was no macular star or exudate. The results of a full uveitis workup were negative for antinuclear antibody, rheumatoid factor, antineutrophil cytoplasmic antibody, HLA-B27, angiotensin-converting enzyme, Lyme disease, syphilis, and \( B \) henselae. The erythrocyte sedimentation rate was within normal limits. The results of viral serologic testing for adenovirus, rhinovirus, influenza A and B viruses, and respiratory syncytial virus were also negative. The results of a magnetic resonance imaging/magnetic resonance venography of the brain were within normal limits. A lumbar puncture revealed a normal opening pressure, normal chemistry results, a normal cell count, and a negative culture result. Serology testing for \( M \) pneumoniae showed elevated IgM and IgG antibodies. Macular optical coherence tomography revealed peripapillary and macular serous detachments (Figure 3), and fluorescein angiography revealed bilateral optic disc leakage without evidence of leakage in the macula (Figure 4A and B). In addition to topical steroids and cycloplegic eye drops, he was treated with a 1-week course of oral azithromycin. Because his optic disc edema and macular serous detachments persisted for 2 weeks after presentation, he was subsequently treated with 40 mg of oral prednisone daily. One month after presentation, the uveitis and serous macular detachments resolved.

Case 3. A 27-year-old woman presented with a 1-week history of bilateral eye pain, photophobia, injections, and blurry vision. Her visual acuity was 20/100 with pinhole to 20/30 in both eyes. The results of an examination showed extreme photophobia, diffuse conjunctival injection, and 2+/H11001 cell and flare in the anterior chamber. The results of a full uveitis workup were negative, as in the 2 previous cases. Because she presented within 1 month of the 2 previous patients, \( M \) pneumoniae serology testing was performed and revealed elevated IgG and IgM antibodies. The patient was treated with topical steroids and cycloplegic eye drops, in addition to a 1-week course of oral azithromycin. The uveitis resolved after 2 weeks of treatment, and her visual acuity returned to baseline.

Comment. \( M \) pneumoniae is a common human pathogen responsible for atypical cases of community-acquired pneumonia, or “walking
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. 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.

*M pneumoniae* contains both glycolipid and protein antigens that elicit antibody responses in infected individuals. 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 microvascularitis from immune complexes depositing along vessel walls in the eye. Another mechanism by which *Mycoplasma* infection is thought to cause uveitis is through direct invasion of the bacteria in the anterior chamber. 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). 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. 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.

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### Table. Summary of Cases of Mycoplasma pneumoniae–Associated Uveitis in the Literature

<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</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</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</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</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</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. Diabetes mellitus, chronic otitis externa, and immunosuppression are frequently associated with SBO. More than 60% of patients with SBO are men, and *Pseudomonas aeruginosa* is the most common bacterial pathogen identified in SBO. Cranial nerve palsy most frequently involves cranial nerve VII, followed by the lower cranial nerves (IX-XI). We report 2 cases of SBO with rare unilateral optic nerve in-