and ocular pain had resolved. His visual acuity and visual field deficit were unchanged when examined 3 months after onset.

The pathophysiology of orbital inflammation with bisphosphonates is uncertain but may involve the release of acute-phase reactants and cytokines by these drugs.  

Ischemic optic neuropathy is suggested by the sudden, nonprogressive visual loss that persisted despite resolution of the orbital inflammation with steroid therapy. Ischememia may have been caused by orbital or ocular inflammation contiguously affecting the posterior ciliary arteries that supply the optic disc, creating a local small-vessel vasculitis. Though unusual, ischemic optic neuropathy has been described in association with uveitis or choroiditis in other conditions. For example, Anterior Ischemic Optic Neuropathy has been described in association with local orbital inflammation, infections such as herpes zoster, Posner-Schlossman syndrome, and systemic inflammatory disorders such as Crohn's disease or ankylosing spondylitis.

Rifabutin-associated uveitis in an immunocompetent individual with Mycobacterium simiae

A 54-year-old, HIV-negative, white male was referred to the uveitis service at the University of Texas Health Science Center at San Antonio for evaluation of pain, photophobia, and redness in the left eye.

The patient's medical history was significant for severe emphysema and a Mycobacterium simiae pneumonia diagnosed 5 months before presentation. His condition had been treated with clarithromycin 500 mg bid, ethambutol 900 mg qd, and rifabutin 300 mg qd over the preceding 3 months. His ocular history included cataract extraction and intraocular lens placement in both eyes 4 years earlier. Review of systems disclosed left ankle pain. Visual acuity was 20/40 in the right eye and 20/30 in the left. Slit-lamp examination of the right eye revealed moderate conjunctivitis, and 3+ anterior chamber cells with no hypopyon, posterior synechiae, or capsular plaques. Slit-lamp examination of the right eye was normal. Dilated fundus examination disclosed a cup-to-disc ratio of 0.6 in both eyes with no evidence of posterior uveitis. Acute anterior uveitis possibly associated with rifabutin was diagnosed. Rifabutin was discontinued, and treatment with topical prednisolone acetate 1% every hour and atropine once daily was started in the left eye. Additional workup included negative serologic tests for syphilis and HLA-B27, and normal complete blood count.

Within 1 month of treatment the uveitis had resolved, the visual acuity had improved to 20/25, and prednisolone acetate 1% was tapered off. Follow-up has continued for 4 years with no evidence of recurrence while the patient has been off steroids. Pulmonary treatment was continued with ethambutol and clarithromycin alone.

Rifabutin-associated uveitis has been described in immunosuppressed patients (most commonly those with AIDS) and rarely in immunocompetent individuals receiving therapy or prophylaxis against Mycobacterium avium complex (MAC) infection. Rifabutin-associated uveitis can be severe, causing a hypopyon. It has been associated with concomitant use of clarithromycin or fluconazole, which is believed to raise serum levels through inhibition of the hepatic cytochrome p-450 system that metabolizes the drug. It has been suggested that the risk of rifabutin-associated uveitis can be decreased by maintaining the dose of rifabutin at or below 300 mg/day if clarithromycin is concomitantly used. It has been associated with certain MAC strains and possibly with rifabutin doses greater than 1900 mg qd, as in other reported cases. His ocular symptoms resolved upon discontinuation of rifabutin and initiation of topical steroids. The patient's eyes have remained quiet for 4 years after discontinuation of topical steroids. As shown by our case, rifabutin-associated uveitis may occur regardless of the immunologic status of the patient and in patients treated for mycobacteria other than MAC. It is important to recognize this association in patients with uveitis who are taking rifabutin, regardless of the patient's immunologic status and organism for which the rifabutin is prescribed.

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Protean manifestations of pediatric neurosarcoidosis

Sarcoidosis is a multisystem, granulomatous disease involving the central nervous system in 5% of patients. Neurosarcoidosis may present with protean manifestations in children, resulting in permanent morbidity if treatment is delayed. We describe a 15-year-old patient who presented with bilateral anterior uveitis and audiovestibular dysfunction as signs of neurosarcoidosis.

A 15-year-old African-Caribbean male was referred for an evaluation of bilateral uveitis, hearing loss, and ambulation difficulties. Five months earlier he had experienced unsteady gait with subsequent left facial weakness. The facial weakness and gait instability improved without therapy. One month later, he complained of bilateral blurred vision, followed by hearing loss and tinnitus. The condition was diagnosed as bilateral anterior uveitis, treated with bilateral periocular triamcinolone injections (40 mg) and topical prednisolone acetate ophthalmic 1%, and subsequently referred to the uveitis service.

Visual acuities were 20/20 OU. Slit-lamp examination showed inferior keratic precipitates and rare anterior chamber cells OU. Funduscopic examination was normal. General physical examination demonstrated bilateral axillary and inguinal lymphadenopathy, and abnormal tandem gait. Audiometric testing showed severe sensorineural hearing loss in the right ear (AD) and the absence of speech and pure tone hearing in the left ear (AS).

Tests for Lyme antibody and rapid plasma reagin, chest x-ray, erythrocyte sedimentation rate, complete blood count, and purified protein derivative testing were negative. The angiotensin-converting enzyme level was elevated at 75 U/L. MRI of the brain was normal. MRI of the internal auditory canals (IACs) showed hyperintense signals in the vestibule, cochlea, and semicircular canals bilaterally with enhancement on postcontrast fluid-attenuated inversion recovery sequences, indicating active inner ear inflammation (Fig. 1). There was also subtle bilateral enhancement of the labyrinths on T1-weighted images. Examination of the cerebrospinal fluid showed reactive lymphocytosis.

CT scan revealed hilar, mediastinal, axillary, and inguinal lymphadenopathy (Fig. 1). Right inguinal lymph node biopsy showed noncaseating granulomas consistent with sarcoidosis (Fig. 2).

Prednisone (1 mg/kg) was administered and tapered over 3 months. Bilateral transtympanic methylprednisolone injections were given. At 6 months the uveitis remained quiescent. His hearing improved partially AD, but no improvement in hearing was observed AS. Repeat MRI of the IACs 6 weeks after the initial MRI was normal. The family declined corticosteroid-sparing immunosuppression.

The differential diagnosis for uveitis and hearing loss in a child includes syphilis, tuberculosis, Cogan’s syndrome, Vogt-Koyanagi-Harada syndrome, Behçet’s disease, and sarcoidosis. Pediatric neurosarcoidosis is extremely rare and commonly presents with seizures. Ocular involvement occurs in 25%–50% of patients with sarcoidosis, but over 60% of neurosarcoidosis patients demonstrate ophthalmic involvement.

The precise pathogenesis of sarcoidosis is thought to involve the interaction of T cells and macrophages with...