tum, 70% of isolates were sensitive to amikacin, while 100% of isolates were sensitive to clarithromycin. Intravitreal clarithromycin has been reported to be non-toxic to rabbit eyes up to a dose of 1.0 mg; to our knowledge, the use of intravitreal clarithromycin in humans has not been reported.

In summary, Mycobacterium chelonae is an uncommon cause of endophthalmitis. It is associated with a chronic course of marked intraocular inflammation and, in some cases, a white plaque inside the capsular bag in eyes after cataract surgery. While macrolide antibiotics may show good in vitro efficacy against Mycobacterium chelonae, endophthalmitis caused by this organism has generally poor visual acuity outcomes.

Endogenous Endophthalmitis After Routine Dental Cleaning

Hematogenous dissemination of microorganisms to the eye is an uncommon cause of endophthalmitis. Studies have reported that it accounts for 2% to 8% of all forms of endophthalmitis. For patients with symptoms of uveitis who have a history of systemic or focal infections or evidence of an immunocompromised state, endogenous endophthalmitis falls readily into the differential diagnosis. However, in an immunocompetent individual without evidence of systemic infection, the diagnosis requires a high index of suspicion.

Report of a Case. A 48-year-old woman underwent a routine dental cleaning before development of eye symptoms. She had no history of ginja disease or cavity fillings. At initial examination 10 days later, she had sharp pain and photophobia in the right eye. She had no significant ocular history. Her medical history was remarkable for hypertension, asthma, osteoporosis, and fibromyalgia.

On examination, corrected visual acuity was 20/200 OD and 20/20 OS. Intraocular pressures were 21 and 19 mm Hg, respectively. The anterior segment examination was significant for conjunctival hyperemia, fine keratic precipitates, the absence of iris nodules, and nuclear sclerosis in the right eye. The posterior segment was remarkable for vitreous haze secondary to cellular reaction and 3 areas of intraretinal hemorrhages with marked arteriolar sheathing (Figure 1). The left eye examination was unremarkable.

A tentative diagnosis of uveitis and retinal vasculitis was made, and the patient was started on topical corticosteroids and cycloplegics. At 3 days’ follow-up examination, her vision had worsened to hand motion, she had developed a hypopyon, and there was no view of the fundus (Figure 2). She was then admitted to the hospital and underwent a vitrectomy with biopsy and injection of vancomycin hydrochloride (1 mg/0.1 mL) and amikacin sulfate (400 µg/0.1 mL). Cultures were positive several days later for α-hemolytic streptococci. A thorough medical workup in search of a nonocular site of infection was negative.

She subsequently developed a macular hole, for which she underwent vitrectomy with gas injection. To date, she is free of infection and maintains a best-corrected vision of counting fingers at 0.9 m.
Comment. Ishak and colleagues and May and colleagues each reported a case of endogenous endophthalmitis in patients with gingival disease that progressed to an abscess. In the latter case, the patient also had undergone a cavity filling 7 days before onset of symptoms.

Our case of endogenous endophthalmitis was in an immunocompetent individual who underwent routine dental cleaning 10 days before seeing an ophthalmologist. She did not undergo any procedures such as cavity filling or tooth extraction on that visit. As noted in previous case reports, periodontal disease is well documented as a potential cause of endogenous endophthalmitis. However, as far as we are aware, this is the first reported case indicating that a routine teeth cleaning without evidence of gingival disease or a focal infection, such as a periodontal abscess, can lead to endogenous endophthalmitis. Because α-hemolytic streptococci are known to reside as normal flora in the nasopharynx, we presume that transient bacteremia developed after the dental cleaning, which led to seeding of the organism into intraocular tissues. A lag time of 7 to 10 days before onset of symptoms appears to be consistent with previous reports in the literature. In short, a careful history in any patient with symptoms of uveitis should include inquiries regarding routine dental cleaning.

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Chiasmal Enlargement and Optic Nerve Enhancement on Magnetic Resonance Imaging in Leber Hereditary Optic Neuropathy

Findings on magnetic resonance imaging (MRI) of the brain and orbits are typically normal in patients with Leber hereditary optic neuropathy (LHON). We describe 2 patients with LHON who had abnormalities of the optic nerves and chiasm disclosed by MRI.

Report of Cases. Case 1. A 7-year-old boy had subacute painless decline in vision in both eyes in the middle of July 1999. An evaluation by a local ophthalmologist on August 23, 1999, revealed a visual acuity of 20/60 OD and 20/300 OS. Findings from the remainder of the examination were reportedly normal and the patient was given the diagnosis of functional visual loss. He was otherwise healthy with no other neurologic or systemic symptoms and no medical illnesses. Family history was unremarkable for ophthalmologic or neurologic disease.

Neuro-ophthalmologic evaluation was first performed on September 15, 1999, approximately 2 months after the onset of his visual decline. On examination he had a visual acuity of 20/100 OD and 20/400 OS. Kinetic perimetry showed central scotomas in both eyes. With Ishihara color testing, he identified 8 of a total of 14 plates with the right eye and 3 of a total of 14 plates with the left eye. Pupillary examination showed 4-mm, 2+ reactive pupils with a left relative afferent pupillary defect. Dilated fundus examination showed optic nerve pallor, greater in the left eye. The remainder of the ophthalmologic and neurologic examination findings were unremarkable.

Results from additional tests, including a complete blood cell count, erythrocyte sedimentation rate, blood chemistry, angiotensin-converting enzyme level, antinuclear antibody titer, syphilis serology, and chest radiograph, were normal.

An MRI of the brain and orbits with orbital fat suppression techniques following the administration of intravenous gadolinium was obtained on the day of evaluation (approximately 2 months after the onset of visual loss) and showed enlargement of the optic chiasm and enlargement and enhancement of the intracranial optic nerves (Figure 1, A and B). An optic pathway glioma and optic neuritis were initially considered as potential causes of this patient’s visual decline. However, mitochondrial DNA testing disclosed a mutation at nucleotide position 11778 confirming the diagnosis of LHON. A lumbar puncture showed an opening pressure of 100 mm H2O and normal cerebrospinal fluid contents. Myelin basic protein and oligoclonal bands were not present. Results from the cerebrospinal fluid IgG index and cerebrospinal fluid cytology were normal.

Prior to obtaining the results of mitochondrial DNA testing, the patient was treated with high-dose systemic steroids (intravenous methylprednisolone followed by oral...