causes facial deformities and abnormalities of dentition. The disease generally progresses until puberty and may regress in adulthood. Radiologically, the radiolucent bony lesions are cystic and multiloculated in nature and are bilateral. These findings in conjunction with the clinical presentation are usually enough to make a diagnosis. Unless surgical intervention is needed for genetic or reconstructive purposes, a confirmatory biopsy is not obligatory. If a biopsy is performed, the histopathological differential diagnosis of lamellar bone formation with fibrous stroma and giant cell formation includes fibrous dysplasia, aneurysmal bone cyst, central giant cell granuloma of bone, and brown tumor of hyperparathyroidism. However, on the basis of clinical presentation and laboratory data, alternative diagnoses can be ruled out. Indications for treatment include functional problems, such as dental abnormalities and visual compromise, and the need for cosmesis. Treatment of choice is surgical curettage and contouring. Radiation therapy is not recommended because of risks of osteonecrosis and sarcoma transformation.

We present a case of cherubism with documented visual loss secondary to optic neuropathy and macular chorioretinal folds/scarring directly attributable to compression from the fibro-osseous growth within the orbit. To our knowledge, only 3 other cases of cherubism have been described in the ophthalmic literature. None of these cases presented with a relative afferent pupillary defect indicative of optic neuropathy. Although Hawes presented a patient with visual loss secondary to macular scarring, he did not attribute this to globe compression from the fibro-osseous mass. Since most reports on this disease are presented in the oral maxillofacial and otorhinolaryngology literature (MEDLINE search of the past decade reveals 2 articles in ophthalmology journals and 37 in the two aforementioned fields), the focus is not on ophthalmic manifestations of disease and etiology of visual loss. It is therefore quite possible that visual loss from optic neuropathy and/or maculopathy secondary to cherubism is underestimated. As such, we feel that routine examination by an ophthalmologist be recommended in the management of a patient with cherubism.

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Endophthalmitis Caused by Mycobacterium chelonae: Selection of Antibiotics and Outcomes of Treatment

Mycobacterium chelonae is a rapidly growing acid-fast bacterium that has been reported to cause keratitis, interface infection after laser in situ keratomileusis, scleral buckle infection, keratitis in a corneal graft, and periorcular infection after dacryocystorhinostomy and ptosis repair. In a review of the literature, a total of 4 cases of endophthalmitis caused by M chelonae have been reported. The current study reports 5 cases of culture-proven endophthalmitis caused by M chelonae at Bascom Palmer Eye Institute, Miami, Fla, between January 1, 1980, and December 31, 2001.

Report of Cases. Case 1. A 62-year-old man received an intravitreal injection of triamcinolone acetonide (4 mg/0.1 mL) in October 2001 for clinically significant macular edema reducing visual acuity to 20/70 OD.
In November 2001, the patient returned with blurry vision and pain in the right eye. Results of examination demonstrated a visual acuity of hand motions, anterior chamber cells, and dense vitritis in the right eye. The eye was treated with a vitreous tap and injection of intravitreal antibiotics. Vitreous cultures were negative. Because of persistent vitreous inflammation, a pars plana vitrectomy was performed in the right eye in December 2001. Vitreous cultures were again negative.

On initial examination by our group, the patient had a visual acuity of hand motions, 2+ corneal edema, 2+ cell and flare in the anterior chamber, and opacification of the vitreous cavity in the right eye. Ultrasonography showed diffuse vitreous opacities, 360° choroidal detachment, and no retinal detachment. A pars plana vitrectomy and lensectomy were performed. Injections of amphotericin B, 0.01 mg, ceftazidime, 2.25 mg, and vancomycin, 1.0 mg, were given. Cultures of the vitreous cavity yielded *M chelonae*, sensitive to clarithromycin and amikacin; intermediate sensitive to tobramycin, imipenem, and cefoxitin; and resistant to ciprofloxacin, doxycycline, and sulfamethoxazole-trimethoprim. Despite 3 months of treatment with topical fortified amikacin sulfate and 4 intravitreal injections of 0.4 mg of amikacin sulfate, the visual acuity progressed to no light perception in February 2002. In each of 4 separate vitreous cultures, growth of *M chelonae* was documented, but the final culture, taken when the visual acuity was no light perception, showed no growth. The eye was enucleated in April 2002, and histopathologic examination demonstrated persistent acid-fast organisms in multiple intraocular tissues.

**Case 2.** A 76-year-old man underwent uncomplicated extracapsular cataract extraction and intraocular lens (IOL) implantation in the left eye in September 1992. In May 1994, the patient was referred with a complaint of 4 months of pain and decreased vision in the left eye.

On initial examination by our group, the patient had visual acuity of 20/300, a hypopyon smaller than 1 mm with anterior chamber cellular reaction, a posterior capsular plaque, and diffuse vitreous cells in the left eye. Pars plana vitrectomy and injection of intravitreal antibiotics were performed in May 1994. A second pars plana vitrectomy and injection of intracocular antibiotics, as well as an IOL exchange, were performed in August 1994. In October 1994, the patient returned with visual acuity of 1/200 and a hypopyon in the left eye; a fluid-air exchange was performed, and vitreous cultures yielded *M chelonae*, which was sensitive to erythromycin, intermediate sensitive to ciprofloxacin, and resistant to tetracycline, sulfamethoxazole-trimethoprim, cefoxitin, and amikacin. Treatment with topical amikacin and oral rifampin was initiated, and smoldering inflammation with intermittent hypopyon formation persisted. In April 1995, the patient underwent a third pars plana vitrectomy, with further removal of residual lens capsule, vitreous culture, and injection of intravitreal erythromycin lactobionate, 0.5 mg, and imipenem, 0.5 mg. Vitreous cultures showed growth of *M chelonae*. Intraocular inflammation resolved. In July 1995, final visual acuity was 5/200 OS.

**Case 3.** A 77-year-old man underwent intracapsular cataract extraction without IOL in the left eye in 1979. A secondary IOL was placed in the left eye in December 1985 without complication. Four weeks postoperatively, pain and blurry vision developed in that eye.

On initial examination by our group, the patient had a visual acuity of 20/100 OS with a 10% hypopyon, and fluffy white infiltrates on the anterior hyaloid face (Figure, A). Possible fungal endophthalmitis was diagnosed. The patient was treated with an anterior chamber tap, anterior vitrectomy, and intravitreal injection of gentamicin sulfate, 0.1 mg, and amphotericin B, 0.01 mg. Anterior chamber cultures yielded *M chelonae* sensitive to clarithromycin, amikacin, kanamycin, and tobramycin, and resistant to ofloxacin and ciprofloxacin. Despite 3 intravitreal injections of amikacin sulfate (0.25 mg) and 2 months of topical fortified amikacin and prednisolone acetate, in December 1986, final visual acuity was hand motions and the intraocular pressure was 0 mm Hg by applanation tonometry (Figure, B).

**Case 4.** A 70-year-old woman underwent complicated cataract extraction in the right eye in February 1986.
Table 1. Endophthalmitis Caused by Mycobacterium chelonae: Source, Treatment, and Outcomes

<table>
<thead>
<tr>
<th>Study</th>
<th>Source</th>
<th>Treatment</th>
<th>Final Visual Acuity</th>
</tr>
</thead>
<tbody>
<tr>
<td>El-Asrar and Tabbara*</td>
<td>ECCE/IOL</td>
<td>PPV × 2</td>
<td>Hand motions</td>
</tr>
<tr>
<td>Ramaswamy et al*</td>
<td>Phaco/IOL</td>
<td>PPV</td>
<td>No light perception</td>
</tr>
<tr>
<td>Ambler et al*</td>
<td>Endogenous</td>
<td>PPV</td>
<td>Data not provided</td>
</tr>
<tr>
<td>Roussel et al* (case 3, present study)</td>
<td>Secondary IOL</td>
<td>Anterior vitrectomy, intravitreal antibiotics</td>
<td>Hand motions</td>
</tr>
<tr>
<td>Present study</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case 1</td>
<td>Corticosteroid injection</td>
<td>PPV × 2</td>
<td>No light perception</td>
</tr>
<tr>
<td>Case 2</td>
<td>ECCE/IOL</td>
<td>PPV × 3</td>
<td>3/200</td>
</tr>
<tr>
<td>Case 4</td>
<td>Bleb related</td>
<td>Anterior vitrectomy, intravitreal antibiotics</td>
<td>Light perception</td>
</tr>
<tr>
<td>Case 5</td>
<td>Corneal ulcer</td>
<td>Penetrating keratoplasty</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ECCE, extracapsular cataract extraction; IOL, intraocular lens; Phaco, phacoemulsification; PPV, pars plana vitrectomy.

Table 2. Mycobacterium chelonae: Antibiotic Sensitivities*

<table>
<thead>
<tr>
<th>Class of Antimicrobials</th>
<th>Effectiveness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycopeptides (e.g., vancomycin)</td>
<td>Usually ineffective</td>
</tr>
<tr>
<td>Cephalosporins</td>
<td>Usually ineffective (cefoxitin may be useful)</td>
</tr>
<tr>
<td>Macrolides</td>
<td>Usually effective (azithromycin, clarithromycin)</td>
</tr>
<tr>
<td>Aminoglycosides</td>
<td>Usually effective (amikacin, tobramycin)</td>
</tr>
<tr>
<td>Fluoroquinolones</td>
<td>Sometimes effective</td>
</tr>
<tr>
<td>Amphotericin B</td>
<td>Ineffective</td>
</tr>
<tr>
<td>Sulfonamides</td>
<td>Usually ineffective</td>
</tr>
</tbody>
</table>

*Data from Gilbert et al 11 and Brown and Wallace. 12 All of the classes of antimicrobials listed were used in the present study patients.

tors. In April 1995, with anterior chamber IOL implantation, 2 penetrating keratoplasties for pseudophakic bullous keratopathy and failed graft, and 2 Baerveldt implantations for elevated intraocular pressure. In December 1998, the patient complained of pain and decreased vision in the right eye. On initial examination by our group, the patient’s right eye had a visual acuity of hand motions, an exposed Baerveldt glaucoma drainage device, a flat anterior chamber, and peripheral choroidal detachment. The Baerveldt tube was removed and the fistula tract was closed. Postoperatively, dense intraocular inflammation was noted, and an anterior vitrectomy with vitreous culture was performed. Intravitreal injections of cefazolin, 2.25 mg, cefazidime, 1.0 mg, and dexamethasone sodium phosphate, 0.4 mg, were administered at the conclusion of the procedure. Intraocular cultures yielded Mycobacterium chelonae, sensitive to clarithromycin, intermediate sensitive to ciprofloxacin and amikacin, and resistant to tobramycin, doxycycline, and tetracycline. Postoperatively, a 2-month course of fortified amikacin, atropine, and prednisolone acetate, and oral clarithromycin, resulted in resolution of smoldering intraocular inflammation. At last follow-up in April 2000, visual acuity was 3/200 OD.

Case 5. A 73-year-old man was examined in March 1999 complaining of pain and discharge in the right eye for 1 week. A tarsorrhaphy had been performed in February 1990 for a neurotrophic corneal ulcer.

On initial examination by our group, the patient had a visual acuity of light perception, a 70% lateral tarsorrhaphy, a central corneal infiltrate with perforation plugged by a fibrin plaque, and dense anterior chamber fibrin. A penetrating keratoplasty with extracapsular cataract extraction, peripheral iridectomy, and conjunctival flap was performed, and treatment was initiated with topical amikacin and oral ciprofloxacin. Culture of the anterior chamber yielded Mycobacterium chelonae sensitive to erythromycin, azithromycin, and clarithromycin, and resistant to cefoxitin and ciprofloxacin. One month postoperatively, the patient did not complain of pain in the right eye, and visual acuity was light perception. A vascularized cornea prevented further examination.

Comment. In the present series, several categories of endophthalmitis are represented: (1) acute-onset postoperative endophthalmitis, (2) chronic or delayed-onset postoperative endophthalmitis, (3) endophthalmitis associated with glaucoma surgery, (4) endophthalmitis associated with a perforated corneal ulcer, and (5) intravitreal injection of triamcinolone. The 2 cases that occurred after anterior segment surgery each were notable for the presence of white opacifications of the capsular bag or anterior vitreous face, leading to initial misdiagnosis as Propionibacterium acnes or fungal endophthalmitis. Other authors have noted white opacities of the lens capsule or vitreous in cases of M chelonae endophthalmitis.7,8

Mycobacterium chelonae endophthalmitis is generally associated with poor visual acuity outcomes (Table 1). No patients in previous reports or the current series achieved visual acuity of 20/400 or better. Many eyes developed chronic hypotony or underwent enucleation or evisceration.7,10

The preferred antibiotic for M chelonae remains uncertain, but amikacin is usually considered as the first-line antibiotic of choice7 (Table 2). In the present series, the cultured organism was sensitive to macrolide antibiotics (sensitivity was tested to clarithromycin in 4 patients and to erythromycin in 2 cases). However, the cultured organism was sensitive to amikacin in only 2 cases, was intermediate sensitive in 1 case, and was resistant in 1 case (sensitivity to amikacin was not tested in 1 case). In a recent case series of nontuberculous mycobacterial kera-
M. chelonae. In summary, M. 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 M. chelonae, endophthalmitis caused by this organism has generally poor visual acuity outcomes.

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8. Al-Asrari AM, Tabbara KS. Chronic endophthalmitis after extracapsular cataract extrac-