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Management of Bilateral Uveitis Secondary to Intraocular Filariasis

Management of uveitis secondary to filariasis has been inadequately described as these cases are rare. We report a case of bilateral uveitis due to intraocular filariasis and discuss its medical and surgical treatment. We also document the use of doxycy-

cline hydrochloride, which sterilizes adult worms by eliminating their symbiotic bacteria and may prevent recurrences of uveitis.



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Report of a Case. A 49-year-old Asian Indian man had decreased vision in both eyes for 3 months, with pain and redness in the right eye for 1 month. Both eyes had a best-corrected visual acuity of counting fingers at 1 m with nongranulomatous anterior uveitis, posterior synechiae, cataracts, and dense vitritis. In addition, the right eye had an iris bombe configuration with an intraocular pressure of 44 mm Hg. Systemic history and examination results were unremarkable. Antiglaucoma medication, topical steroids, and cycloplegics were started. A motile worm (approximately 200 μ m long) was subsequently observed on the anterior capsule of the left eye. Peripheral blood smear showed microfilariae of *Wuchereria bancrofti*. Stool examination was negative for cysts or ova of parasites.

Oral prednisone (1 mg/kg/d) was started, and antifilarial treatment (diethylcarbamazine, 6 mg/kg/d for 3 weeks, and albendazole, 400 mg once daily for 1 week) was initiated 3 days later. Treatment with oral doxycycline hydrochloride, 100 mg twice daily, was given for 4 weeks to eradicate *Wolbachia*. Glaucoma was unresponsive to medical management. Therefore, a trabeculectomy with phacoemulsification and intraocular lens implantation was performed on the right eye within 1 week of commencing treatment with steroids. The postoperative period was uneventful. Six months later, phacoemulsification with intraocular lens implantation was performed on the left eye. Steroids were gradually tapered and stopped. Both eyes have been quiet for 6 months, with a visual acuity of 6/12 J2 due to an epiretinal membrane over the macula. Peripheral blood smear 1 year after doxycycline treatment did not show any microfilaria.

Comment. Uveitis secondary to intraocular filariasis in the Indian subcontinent is mainly due to *W bancrofti* and *Brugia malayi*.¹ Intraocular filariasis is caused more commonly by microfilariae than by adult worms.² This is an unusual case of microfilariae causing bilateral uveitis, which to our knowledge has been reported in only 1 other article.³

The role of antifilarial drugs is controversial because of the possibility of increased uveitis due to the killing of microfilaria as seen in the Mazzotti reaction. However, several reports have used these successfully under steroid cover without untoward effects.²⁻⁴ Diethylcarbamazine and ivermectin clear the microfilaria from the blood but do not act on the adult worm, which lives in the lymphatic system for 10 to 15 years.^{2,5} Therefore, repeated treatments may be necessary to prevent recurrent episodes of uveitis.^{2,3} Albendazole can reduce the microfilaria possibly due to its embryotoxic effect on the adult worms.⁵ Recently, an endobacterium of the *Wolbachia* species that belongs to the family Rickettsiaceae

was found in some of these nematodes.⁶ These are mutualistic symbionts. Treatment with tetracyclines clears the *Wolbachia* from the worm, affecting embryogenesis and resulting in worm sterility.⁶ This may prevent future episodes of uveitis. Doxycycline treatment showed no effect on *Loa loa* infections in humans because they do not possess *Wolbachia*.⁶ Therefore, accurate identification of the nematode is essential when planning treatment.

In conclusion, we report a case of bilateral uveitis due to intraocular filariasis treated with corticosteroids and antifilarial drugs (diethylcarbamazine, albendazole, and doxycycline) to prevent recurrences. Also, cataract extraction with intraocular lens implantation performed under steroid cover did not increase the postoperative uveitis in this patient and resulted in quicker visual rehabilitation.

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Cutaneous $\gamma\delta$ T-Cell Lymphoma With Bilateral Ocular and Adnexal Involvement

Subcutaneous panniculitis-like $\gamma\delta$ T-cell lymphoma (SPTL-GD) is a rare subtype of primary cutaneous non-Hodgkin T-cell lymphoma caused by a clonal proliferation of mature activated cytotoxic $\gamma\delta$ T cells.^{1,2} It typically manifests with skin nodules or plaques over the extremities and has an aggressive clinical course.² Although rare, reports exist of other metastatic cutaneous T-cell lymphomas with periocular or intraocular manifestations.^{3,4} However, to our knowledge, we report the first case of SPTL-GD manifesting this way. Furthermore, the simultaneous adnexal, intraocular, and neuro-ophthalmic involvement described here has not been reported for any manifestation of ocular lymphoma.

Report of a Case. A 62-year-old woman had a 7-month history of bilateral red eyes with blurred vision. She was diagnosed as having bilateral nongranulomatous anterior uveitis and began treatment with topical steroid eye-

drops. However, her vision continued to slowly deteriorate and she additionally began to experience night sweats, fevers, and weight loss. Two months later, she also noticed 2 firm swellings by her right eyebrow and described sequential bilateral pupillary enlargement.

When examined at this stage, her visual acuity was counting fingers OD and 20/80 OS. Both pupils measured 4.5 mm and were poorly reactive to light with no light-near dissociation but hypersensitivity to pilocarpine, 0.125%. There was no ptosis, eyelid malposition, proptosis, globe displacement, or limitation of eye movements. Anterior segment examination revealed bilateral punctate epitheliopathy with reduced corneal sensation, corneal edema, and no infiltrates. There were multiple small nongranulomatous keratic precipitates with 2+ to 3+ of anterior chamber cells in both eyes but no fibrin or hypopyon. Views of the posterior segments were difficult but showed no abnormalities.

Facial examination revealed 2 right subbrow, subcutaneous lesions that were well circumscribed, firm, nontender, and immobile (**Figure 1A**). No lymph nodes were palpable, and no other systemic abnormalities were detected. However, during the ensuing weeks, 2 other smaller subcutaneous masses developed around the right lower punctum and left cheek.

Her erythrocyte sedimentation rate, C-reactive protein level, full blood cell count, and renal and liver function test results were normal except for an increased platelet count of $679 \times 10^3/\mu\text{L}$ (reference range, $150\text{-}400 \times 10^3/\mu\text{L}$; to convert to $\times 10^9/\text{L}$, multiply by 1.0) and a serum angiotensin-converting enzyme level of 124 U/L (reference range, 8-65 U/L; to convert to nanokatal per liter, multiply by 16.667). Computed tomographic scans of the head, chest, abdomen, and pelvis were normal, but magnetic resonance imaging of the head revealed smooth perineural enhancement of both optic nerve sheaths along their intraorbital portion (**Figure 1B**). An anterior chamber paracentesis was

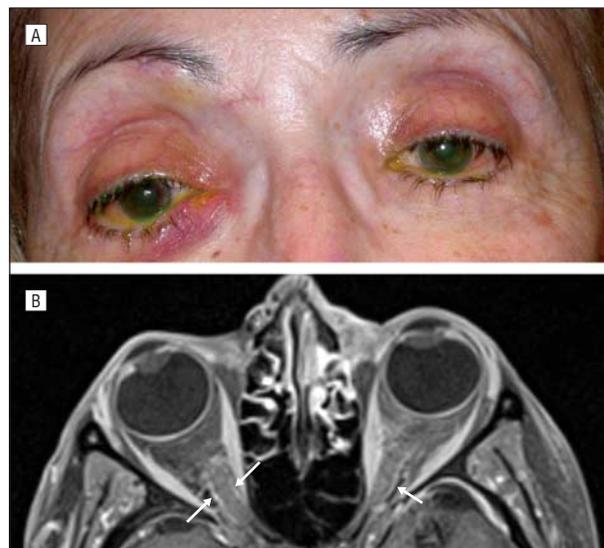


Figure 1. Clinical photograph demonstrating 2 firm, nontender subcutaneous masses below the medial end of the right eyebrow with right lower eyelid erythema (A), and an axial T1-weighted magnetic resonance image of the orbit with gadolinium infusion and fat suppression showing smooth perineural enhancement of both intraorbital optic nerve sheaths (arrows) (B).