

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

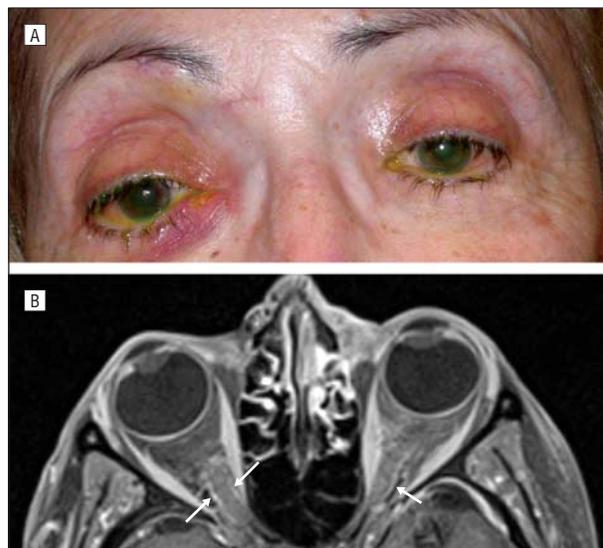


Figure 1. Clinical photograph demonstrating 2 firm, nontender subcutaneous masses below the medial end of the right eyebrow with right lower eyelid erythema (A), and an axial T1-weighted magnetic resonance image of the orbit with gadolinium infusion and fat suppression showing smooth perineural enhancement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

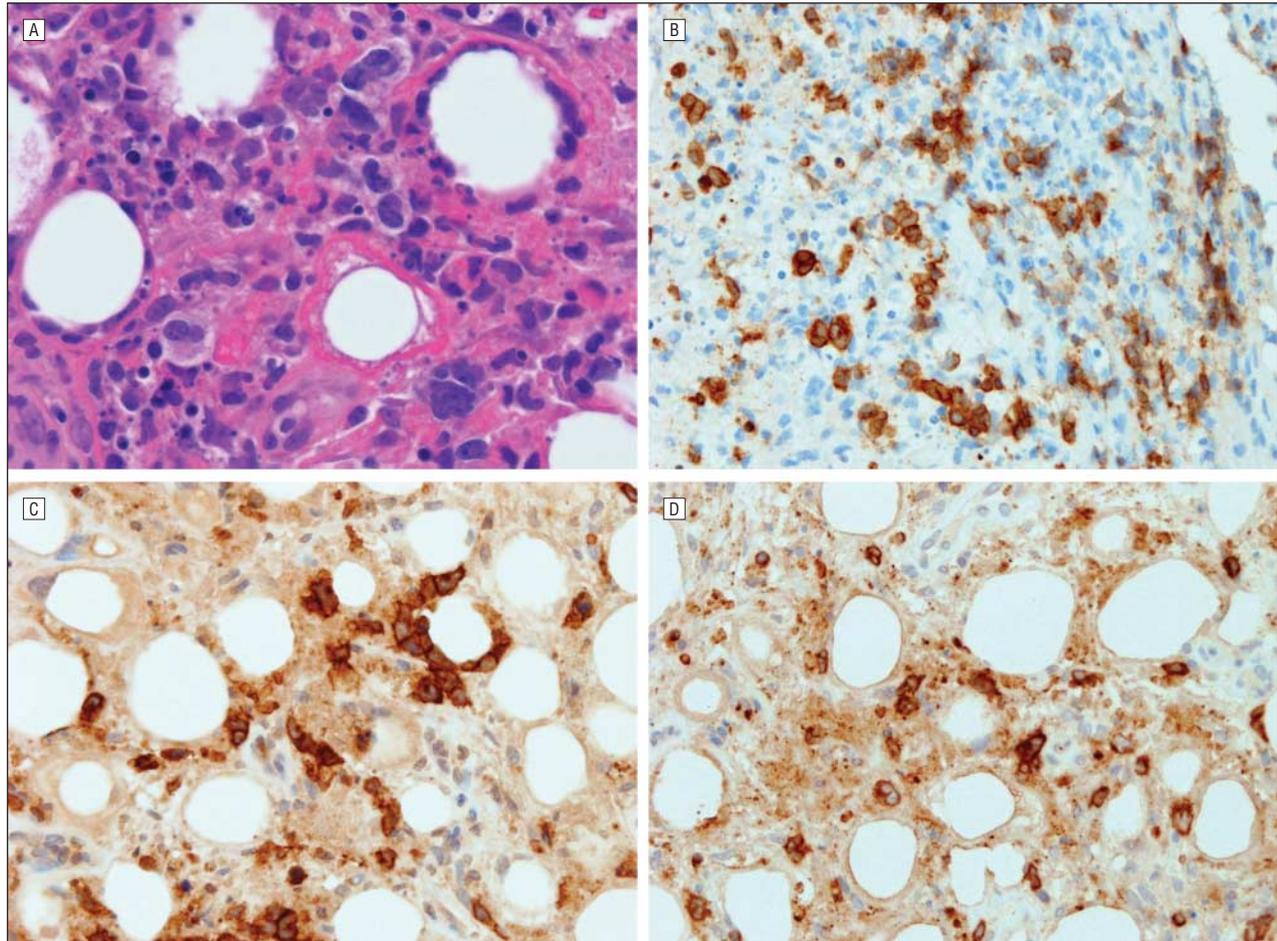


Figure 2. Pleomorphic lymphoma cells with fat rimming and lipomembranous change in a necrotic background (hematoxylin-eosin) (A), with the cells expressing CD3 (B), CD30 (C), and perforin (D) (original magnification $\times 40$).

performed and fluid was negative for herpes simplex virus, varicella-zoster virus, cytomegalovirus, and Epstein-Barr virus DNA on polymerase chain reaction. Insufficient sample was available for cytology.

Excisional biopsies of the right subbrow lesions were performed; they were difficult to interpret because of severe necrosis. However, subsequent specimens from the left cheek and right lower eyelid lesions confirmed SPTL-GD (**Figure 2**). Lesional T cells expressed CD3, CD7, CD30, perforin, TIA-1, and granzyme B and were negative for CD4, CD8, CD56, T-cell receptor α/β (BF-1), and Epstein-Barr virus–encoded small RNA on immunophenotyping/in situ hybridization. The proliferation index as assessed by MIB-1 was 90%.

Comment. Ophthalmic manifestations of lymphoma are broad and include both ocular adnexal and intraocular disease. Most are B-cell non-Hodgkin lymphomas, although the intraocular and adnexal manifestations relate to different subtypes and do not coexist.⁵ T-cell lymphomas, including primary cutaneous ones, have also been reported to involve the eye. Typically, they have eyelid involvement and occasionally have intraocular manifestations such as nonspecific uveitis or retinal infiltrates.³ Our case is unusual owing to the simultaneous manifestation of ocular adnexal, intraocular, and neuro-

ophthalmic pathologic findings. Although the only biopsy-proven site of involvement in our case was the ocular adnexae, both papillitis and infiltrative optic neuropathy have separately been reported with cutaneous T-cell lymphoma, as have anterior uveitis, corneal involvement, and pupillary dilation.^{3,4} Finally, SPTL-GD as a specific subtype of cutaneous T-cell lymphoma has previously been reported only with extraocular muscle involvement.⁶

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Anterior and Nasal Transposition of the Inferior Oblique Muscle for Iatrogenic Superior Oblique Palsy

The reflected tendon of the superior oblique (SO) muscle lies in close proximity to the medial horn of the levator palpebrae superioris aponeurosis in the superomedial aspect of the upper eyelid, placing it at risk for inadvertent damage during surgery in that area. However, although levator resection or excision procedures are commonly performed, this complication is rarely reported.^{1,2} Kushner and Jethani¹ reported a series of 7 patients who sustained SO tendon injury during various types of eyelid procedures, which led to SO palsy in 4 cases and Brown syndrome in 3. We describe a case of iatrogenic SO palsy following congenital ptosis surgery that, because of a large torsional component, we elected to treat with anterior and nasal transposition of the ipsilateral inferior oblique (IO) muscle instead of other conventional procedures.

Report of a Case. An 11-year-old girl underwent left levator excision and fascia lata brow suspension for Marcus-Gunn jaw-winking ptosis. The SO tendon was inadver-

tently cut when the levator palpebrae superioris was severed above the Whitnall ligament, and repair was attempted during the procedure. She visited 1 week later with vertical and torsional diplopia associated with a right head tilt and face turn to the right side, and she was found to have signs consistent with left SO palsy. Strabismus surgery was performed 1 year after the procedure; preoperatively, she had a left hypertropia of 20 prism diopters (PD), which increased to 25 PD on both right gaze and left tilt (**Figure 1**). This hypertropia increased significantly from upgaze (5 PD) to downgaze (25 PD), and there was a V-pattern with 8 PD of exotropia on upgaze and 8 PD of esotropia on downgaze. Motility examination showed SO underaction of -3 and IO overaction of +4 in the left eye. Testing of subjective torsion using double Maddox rod showed excyclotropia of 8° in primary position, which increased to 14° on downgaze. The left IO was transposed nasal to the inferior rectus muscle, with its posterior border reattached to the sclera 2 mm below the medial rectus insertion and the anterior border reattached 2 mm nasal to this point (**Figure 2C**). Postoperatively, the abnormal head posture was corrected. She was orthophoric in both primary position and downgaze, although there was a 6-PD left hypotropia on upgaze with limitation of elevation, more in adduction (Figure 2A and D). She was diplopia free except in upgaze, and the excyclotropia was eliminated in primary position, with excyclophoria of 3° only in downgaze (Figure 2B). These results were stable at 11 months.

Comment. Anterior and nasal transposition of the IO is a relatively new procedure devised by Stager et al.³ It has been reported to be useful in patients with severe or recurrent SO palsies⁴ as well as those with missing SO tendons.⁵ The IO is transposed not only anteriorly as in standard anterior transposition but also nasal to the inferior rectus insertion. This converts the IO from an extorter and elevator in adduction to an intorter and antielevator in adduction.³⁻⁵ Although the effects are more pronounced in upgaze owing to neurological activation of the IO in attempted upgaze and inhibition in downgaze,

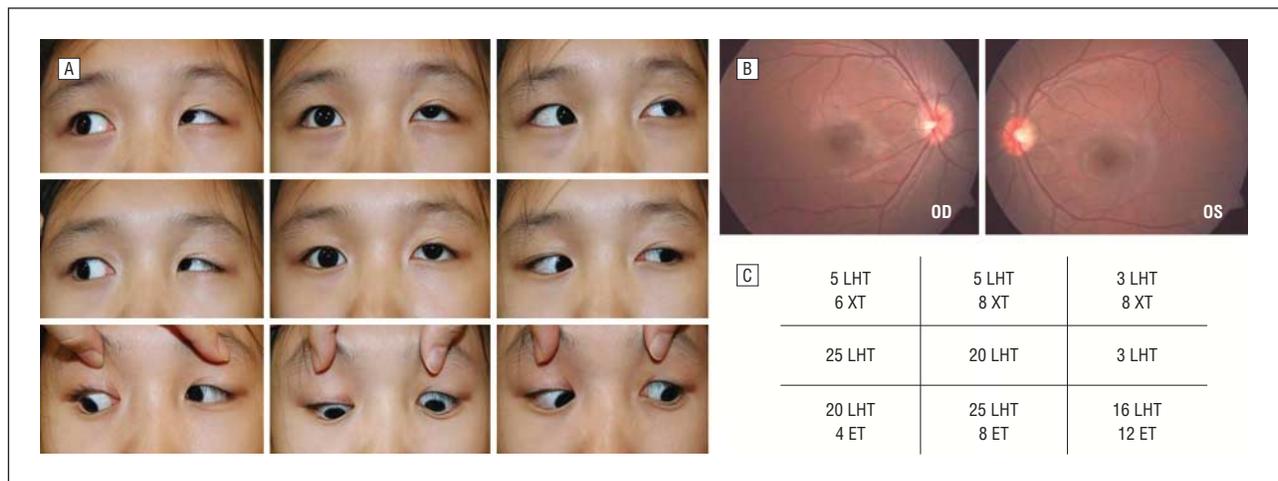


Figure 1. Preoperative results. A, Photographs showing left hypertropia, overelevation in adduction, and underdepression in adduction. B, Fundus photographs showing excyclotropion (left eye [OS] > right eye [OD]). C, Preoperative measurements in prism diopters. LHT indicates left hypertropia; XT, exotropia; and ET, esotropia.