Case Presentation Summary

A 38-year-old Ethiopian man with a history of anterior uveitis in the right eye for 7 months presented to his retina specialist with anterior chamber inflammation, vitritis, optic disc edema, and a yellow focus of chorioretinitis in the right eye. His prior workup was also notable for a history of antinuclear antibody positivity. The patient was promptly referred to the Emory Eye Center for additional workup and management.

On his initial presentation to the Emory Eye Center, the patient’s visual acuities were 20/25 in the right eye and 20/20 in the left eye. Slit lamp examination of the right eye showed 1+ anterior chamber cell, trace flare, and trace anterior vitreous cell. The patient’s dilated funduscopic examination showed a fusiform-shaped yellow lesion in the preretinal space with a surrounding granuloma. There was a ring of exudates surrounding this lesion with extension into the temporal parafoveal region (Figure 1).

A fluorescein angiogram showed leakage corresponding to the area of the chorioretinal lesions and spectral-domain optical coherence tomography (SD-OCT) revealed an area of focal thickening superotemporal to the fovea (Figure 2).

Because of concern for an infectious etiology, a uveitis workup was performed to identify a potential pathogen that could result in the unusual disease appearance.

Chest x-ray, rapid plasma reagin, syphilis immunoglobulin G (IgG), angiotensin-converting-enzyme level, lysozyme, comprehensive metabolic panel, and complete blood count were normal. However, quantiferon-TB was positive, toxoplasmosis IgG antibody was positive at 17.3 IU/mL (reference 0-6.4 IU/mL) and toxocariasis IgG antibody was also positive at 2.76 (normal reference <0.299).

Because of the preretinal granuloma appearance of the lesion, which differed from the distinct appearances of toxoplasmosis (ie, “headlight in a fog” or activation adjacent to a pigmented scar) and tuberculosis (ie, tuberculous choroidopathy, focal granuloma isolated to the choroid) and the similarity of the lesion to peripheral granulomas more commonly seen with ocular toxocariasis, albendazole 200 mg twice daily was initiated.

Three weeks later, the patient complained of increased visual distortion. The visual acuity had declined to 20/100. Funduscopic examination showed interval improvement in the exudation and preretinal fibrosis in the region of the fusiform-shaped yellow lesion. SD-OCT showed moderate epiretinal membrane (ERM) with retinal thickening and intraretinal edema. A repeat serum toxocariasis antibody remained elevated at 3.14. Albendazole was continued.
prednisone 40 mg/day was initiated, and a weekly taper was instituted with judicious follow-up given the patient’s prior serologies, which showed toxoplasmosis and tuberculosis exposure.

Six weeks later, the patient presented with worsening visual acuity to 20/160 and, although the funduscopic examination showed complete resolution of the exudates, significant preretinal fibrosis had developed with VMT. SD-OCT showed progressive thickening of the ERM and broad VMT extending over the posterior pole to the foveal region (Figure 3).

Twenty-three-gauge pars plana vitrectomy with scissors dissection of the preretinal granuloma, ERM and internal limiting membrane (ILM) peeling and fluid-air exchange were performed. (Watch the video of this procedure by either scanning the QR code on page xx or by following the short link provided.) Endolaser and air to 12% C3F8 gas exchange were also performed because of an intraoperative break noted at the time of posterior vitreous detachment induction. Interestingly, the preretinal granuloma was too large to negotiate the 23-gauge cannula system; however, the vitreous fluid was notably positive for toxocariasis antibody at 0.419, consistent with a diagnosis of ocular toxocariasis and subsequent macular preretinal granuloma formation.

Four months postoperatively, the patient’s vision improved to 20/40. SD-OCT showed interval improvement of the patient’s macular edema and confirmed the absence of macular pucker, ERM, and ILM. A subretinal component to the lesion remained, however, and the patient will require long-term monitoring for disease recurrence and secondary complications including choroidal neovascularization (Figure 4).
Vitreoretinal complications of toxocariasis have been reported previously and include both tractional and combined tractional-rhegmatogenous retinal detachments. The peripheral toxocariasis granuloma in a pediatric-aged patient is the most commonly discussed ophthalmic manifestation of this condition, and posterior pole complications have rarely been reported.

Small et al. reported a series of 12 eyes of 12 patients with tractional macular detachment from *Toxocara canis*, and all patients underwent pars plana vitrectomy with membrane peeling while 4 eyes also underwent scleral buckling surgery. A more recent report by Amin et al. described 10 eyes from 10 patients with traction and combined traction-rhegmatogenous retinal detachments. Although all eyes achieved macular attachment following surgery, vision improved in 5 (50%) eyes and was unchanged in the other 5 eyes. The *Toxocara canis* organism was identified in 1 eye, and histologic specimens revealed various degrees of fibrous tissue and inflammatory cells including plasma cells and eosinophils.

Another case report suggested that removal of both the ERM and subretinal components of the granuloma were valuable in achieving a clinical result and allowed the identification of degenerated toxocariasis larval structures within zones of granulomatous inflammation.3

Our case presented a challenging medical dilemma initially because of the multiple serologic tests, which were positive, including tuberculosis, toxoplasmosis, and toxocariasis testing. However, the characteristic clinical appearance of peripheral granuloma observed in toxocariasis, and the features inconsistent with toxoplasmosis or ocular tuberculosis led us to initiate therapy with antiparasitic medication.

The tempo with which the ERM and VMT progressed (ie, over a 6 to 8 week period) despite the resolution of exudates and treatment of presumptive active toxocariasis infection led us to recommend vitreoretinal surgical intervention. Intraoperatively, several questions arose regarding 23-gauge diagnostic pars plana vitrectomy. The specimen was broader than anticipated and, following horizontal scissors dissection of the preretinal component from the subretinal component, we were unable to successfully remove it via the 23-gauge port. Methods of specimen harvesting discussed during the session included the use of a soft tip cannula to aspirate the specimen, enlarging the sclerotomy, or using a different sclerotomy altogether. Fortunately, the antibody testing for toxocariasis was positive from vitreous fluid, although, in the future, consideration of the size of the specimen will be needed for improved preoperative planning.

Besides harvesting the specimen, another question arose as to whether it was necessary to excise the subretinal component, whether it was a potential nidus for additional complications including choroidal neovascularization or infection recurrence, but there was a consensus from the VBS audience that, once the VMT had been relieved and the specimen removed, the subretinal component could be observed closely.

Lastly, audience members brought up the need to use a double-peeling technique with the potential risk of indocyanine green or light toxicity. The ILM was peeled because of concern for reproliferation, while a scaffold remained given the patient’s underlying uveitic disease process. This topic was open for discussion and, while most audience members were comfortable with the additional ILM peeling technique, others recommended judicious observation, which also would have been a reasonable approach.

**CONCLUSION**

Although controversies remain regarding the proper management of VMT from a preretinal granuloma, this case presentation, like many others from the VBS meeting provided a platform for lively and open discussion regarding vitreoretinal surgical disease.

Steven Yeh, MD, is an Assistant Professor of Ophthalmology, Section of Vitreoretinal Surgery & Diseases, at Emory Eye Center in Atlanta. He may be reached at steven.yeh@emory.edu.

Sonia Mehta, MD, is an Assistant Professor in Vitreoretinal Diseases and Surgery at Wills Eye Hospital/Mid-Atlantic Retina in Philadelphia. She may be reached at sonia-mehtamd@gmail.com.