The clinical course of ocular toxocariasis and the chronological development of peripheral retinal and macular granulomas are reported. Removing the epiretinal as well as subretinal component of the granuloma via pars plana vitrectomy and retinotomy techniques yielded an excellent clinical result. Clinicopathologic correlation of the specimen confirmed the diagnosis with histological evidence of degenerated larval structures in granulomatous inflammation.

In 1950, Wilder\(^1\) first described nematode endophthalmitis. The results of her histological examination revealed eosinophilic abscesses and larvae distributed subretinally and within vitreous membranes.

Modalities in the treatment of toxocariasis include corticosteroids,\(^2\) anthelmintics,\(^3\) laser photocoagulation,\(^4\) and pars plana vitrectomy.\(^5\) Indications for pars plana vitrectomy have included epiretinal membranes, tractional retinal detachment, impending tractional macular detachment, chronic inflammation, and dense vitreous membranes. We report perhaps the first case of ocular toxocariasis managed with subretinal surgical techniques in combination with pars plana vitrectomy.

**REPORT OF A CASE**

A 12-year-old boy was evaluated in August 1995 for floaters and decreased visual acuity in his left eye. At 4 years of age, he sustained penetrating trauma to his right eye that required corneal laceration repair, lensectomy, and vitrectomy. An isolated episode of HLA-B27 iridocyclitis in the left eye occurred at age 10.

His visual acuities were 20/400 OD and 20/40 OS. Examination of the right eye revealed quiet media, a corneal scar, aphakia, and optic atrophy. In the left eye, the cornea was clear, the aqueous had a trace cell and flare, the lens was clear, and the iris was normal without nodules. Moderate inflammatory cells were present in the mid and posterior vitreous. An intraretinal infiltrate nasalily was present in a perivascular distribution with an overlying concentration of vitreous debris. The laboratory evaluation findings are summarized in the Table. Treatment with oral prednisone improved visual acuity to 20/30 OS with resolution of inflammation.

In November 1995, he developed recurrent symptoms and a decline of visual acuity OS to 20/100. He had aqueous and vitreous cell and flare. Adjacent to the nasal retinal infiltrate, new white satellite lesions appeared in a multifocal pattern (Figure 1). An empiric antitoxoplasmosis regimen of oral prednisone, sulfadiazine sodium, and leucovorin calcium resulted in improved visual acuity to 20/50 OS and a reduction in inflammation with mild cystoid macular edema.

One month later, the visual acuity OS dropped to 20/80. The nasal retinal lesion was now raised and its surface was contiguous with a band of vitreous inflammatory membranes. A chalky appearing intraretinal lesion appeared preretinal and temporal to the fovea.

His vision deteriorated to 20/200 OS. The macular lesion enlarged into a solid, elevated mass with subretinal hyperpigmentation. A partial posterior...
vitreous detachment was noted over the nasal retina with vitreous inflammation. The clinical presentation was now consistent with toxocariasis. A course of thiabendazole was added. He improved until macular traction developed in July 1996. A pars plana vitrectomy, membrane stripping, retinotomy, and removal of the epiretinal, retinal, and subretinal granuloma were done.

At vitrectomy, the macular lesion was dumbbell shaped: an intraretinal core connected a round epiretinal component and subretinal component (Figure 2). During membrane stripping the nasal white nodules peeled off the retinal surface.

Histopathologic examination findings for the fibrocellular membrane revealed plasma cells, lymphocytes, and eosinophils. The epiretinal component of the dumbbell-shaped lesion was fibrocellular tissue with a granuloma (Figure 3). An encapsulated eosinophilic cystic structure measuring 22.5 μm with an amorphous core of 10.5 μm within a central area of necrosis was consistent with a degenerated *Toxocara canis* larva. Eosinophilic material encapsulating the structure may represent antigen-antibody precipitate (Splendore-Hoeppli phenomenon) (Figure 4). Surrounding this was a zone of epithelioid histiocytes.

![Figure 1](http://archopht.jamanetwork.com/)

**Figure 1.** Nasal intraretinal infiltrate with adjacent satellite lesions.

![Figure 2](http://archopht.jamanetwork.com/)

**Figure 2.** Preoperative photograph. Intraretinal macular lesion with epiretinal membrane (black arrow) and subretinal granuloma (white arrow). Adjacent fluffy infiltrate in papillomacular bundle.

![Figure 3](http://archopht.jamanetwork.com/)

**Figure 3.** Epiretinal granuloma with eosinophilic structure in central necrotic zone (arrow) (hematoxylin-eosin, original magnification ×188).

![Figure 4](http://archopht.jamanetwork.com/)

**Figure 4.** The central area of necrosis contains an encapsulated eosinophilic structure consistent with a degenerated *Toxocara* larva with the surrounding Splendore-Hoeppli material (arrow) (hematoxylin-eosin, original magnification ×752).
and multinucleated giant cells and an outer zone of plasma cells, lymphocytes, and eosinophils. The subretinal fibrocellular tissue had a granulomatous infiltrate composed of plasma cells, lymphocytes, epithelioid cells, eosinophils, multinucleated giant cells, and hyperplastic retinal pigment epithelium. A degenerated structure compatible in size with a larva was found (Figure 5).

Postoperatively, the patient’s eye has remained quiet with a visual acuity of 20/50 OS (Figure 6).

**COMMENT**

The clinician must differentiate toxocariasis from other causes of uveitis, particularly retinoblastoma, reduce ocular inflammation, and prevent loss of vision and amblyopia. The initial clinical appearance was reminiscent of toxoplasmosis in the way it exhibited multiple satellite lesions with vitreous inflammation. The diagnosis of toxocariasis, supported by a positive titer, was made later in this patient’s course as the retinal lesions evolved into granulomas and vitreous bands formed.

Subsequently, the patient was managed with oral prednisone and thiabendazole. The rationale for the use of corticosteroids involves suppression of the destructive inflammatory response to the parasite. Anthelminthics have been used to destroy viable nematodes and eliminate further migration of the larvae. However, the parasite may persist despite anthelmintic treatment. Vision began to deteriorate because of a combination of inflammation, macular traction, and the development of a macular granuloma. To clear vitreous debris, relieve vitreomacular traction, and remove the posterior hyaloid required vitrectomy. Intraoperatively, the vitreous bands inserted into a macular epiretinal membrane. During membrane stripping, a firm stalk was encountered connected to the subretinal mass. This prompted a retinotomy and intact removal of the entire dumbbell-shaped granuloma. Histopathologically, the specimen was consistent with toxocariasis.

Removal of all components of a *Toxocara* granuloma can be successful in treating ocular toxocariasis and is possible with pars plana vitrectomy and subretinal surgical techniques.

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**REFERENCES**