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Transvitreal Migration of a *Toxocara* Larva Resulting in a Second Chorioretinal Granuloma

Ocular toxocariasis usually manifests with a solitary chorioretinal granuloma, uveitis, and/or tractional retinal detachment.[1] Although rare, accounting for 1% of total uveitis cases,[1] it is one of the leading causes of posterior uveitis in children wordwide.[2] We present a child with a temporal granuloma that released a larva into the vitreous when treated with cryotherapy.

Case Report

A 14-year-old otherwise healthy boy presented with redness, photophobia, and blurred vision in the right eye for 3 days. His best-corrected visual acuity (BCVA) was 6/12 in the affected eye and 6/6 in the fellow eye. On biomicroscopy, there were fine keratic precipitates centrally and an anterior chamber activity of 2+ with cells and flare. However, no posterior synechiae or lens opacification was seen. Examination of the fundus revealed a temporal chorioretinal granuloma with a vitreoretinal tractional band extending to the optic disc and diffuse vitritis of 1+ cells. There were no abnormalities seen in the left eye. The clinical appearance was consistent with ocular toxocariasis. The enzyme-linked immunosorbent assay serology for *Toxocara canis* was positive for IgG, with a titer of 0.8 optical density, and there was no eosinophilia. He was treated with intensive topical prednisolone 1% and oral prednisolone 0.5 mg/kg body weight daily for 2 weeks, followed by a 5-day course of oral albendazole 400 mg twice daily, but did not obtain remission. Anticipating the possibility of a viable larva in the granuloma, provoking the inflammation, we performed cryotherapy to kill the organism. Vitrectomy was not attempted, in view of the limited vitreoretinal services available in our center and the volatile uveitis status. During the procedure, a small larva was seen by indirect ophthalmoscopy evacuating the granuloma into the vitreous cavity, where it settled inferiorly. In the immediate postoperative period, there was a severe flare-up of the panuveitis, and he developed an exudative retinal detachment (Fig. 1). This was contained with oral prednisolone 1 mg/kg body weight daily and intensive topical prednisolone 1%, which was tapered gradually. He recovered fully in 4 months, regaining a BCVA of 6/6. However, inspection of the fundus revealed another granuloma inferiorly at the site where the larva had settled (Fig. 2). He is being closely monitored for recurrence of the panuveitis and tractional retinal detachment, but his eye has remained quiescent for the past 2 years.

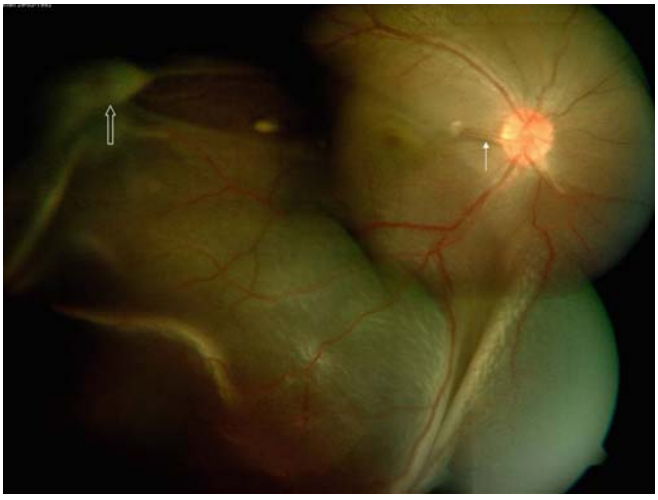

Figure 1. Color fundus photograph of the right eye showing severe exudative retinal detachment following cryotherapy and intravitreal migration of *Toxocara* larva. The primary *Toxocara* granuloma (*thick arrow*) is seen temporally, and vitreal traction (*thin arrow*) is seen on the optic disc.

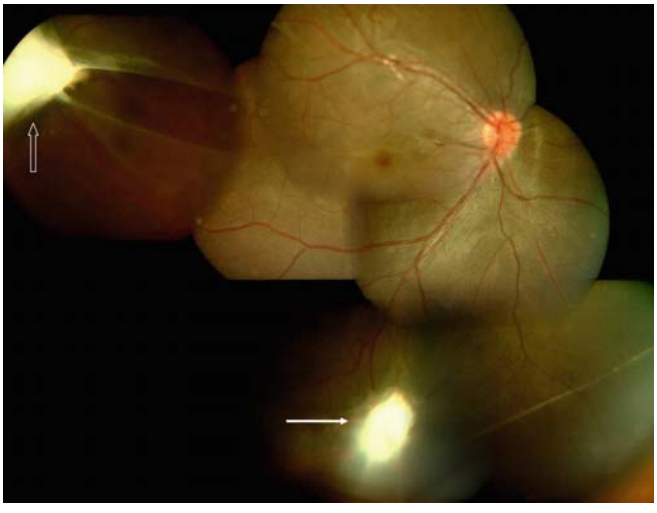

Figure 2. Color fundus photograph of the right eye showing a second *Toxocara* granuloma inferiorly (*thin arrow*) after the inflammation has resolved. The primary *Toxocara* granuloma *(thick arrow*) is still seen temporally.

Comments

Ocular toxocariasis is typically unilateral and even with severe posterior uveitis, there may be little external evidence of inflammation; hence, the patient may present with only blurred vision, mild photophobia, or strabismus.[1] Char-

acteristically, a solitary subretinal granuloma is found either in the peripheral retina or macula or on the optic nerve.[1] Peripheral granulomas are the most common and are associated with the most visual morbidity because of tractional bands inciting cystoid macular edema (37.5%) or tractional retinal detachment (29.2%).[1] Other clinical presentations include vitritis (91.7%), retinal hemorrhage (20.1%), and optic disc swelling (16.7%).[1] Neovascularization of the disc and rubeosis are rare (4.2%).[1] Chronicity and recurrence of the disorder as seen in our patient may be related to a viable larvae within the eye, as has also been demonstrated in primate models, which have shown that in the quiescent form no larva is found.[3] The diagnosis of ocular toxocariasis is usually clinically based, as it may not be associated with eosinophilia or serum antitoxocara antibody positivity.[4] Thus, even when investigating exudative retinal detachment,[5] clinicians should be vigilant. In the literature, there are no reports of ocular toxocariasis with transvitreal migration or multiple granulomas as seen in our patient, who had two granulomas within the same eye.

Key Words: granuloma, intraocular larva, *Toxocara canis*, toxocariasis, transvitreal migration

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Case of Aggressive Posterior Retinopathy of Prematurity with Atypical Neovascular Growth

Fibrovascular proliferation in eyes with retinopathy of prematurity (ROP) usually, but not always, appears at the junction of the vascularized and nonvascularized retina.[1] Aggressive posterior ROP (AP-ROP) occurs in the posterior retina and progresses rapidly to total retinal detachment.[2] We report an atypical case of AP-ROP in which the neovascularization developed in the posterior retina around the optic disc.

Case Report

A female infant was born at 30 weeks' gestation (birth weight, 1670 g) with severe persistent pulmonary hypertension from prolonged premature rupture of the membranes and oligohydramnios. She was treated with nitric oxide (NO) inhalation for 28 days. At 33 weeks postmenstrual age, an ophthalmoscopic examination identified initial signs of zone I AP-ROP bilaterally, including marked dilation and tortuosity of the posterior pole vessels (zone I, stage 1 ROP with plus disease).

Argon laser photocoagulation was performed (duration, 300–400 ms; power, 300–400 mW; 3751 shots OD, 3658 shots OS) under intravenous sedation (fentanyl) with topical anesthesia. However, fibrovascular proliferation and retinal detachment developed bilaterally in the posterior retina around the optic disc 1 week postoperatively (Fig. 1a, b). The patient underwent vitrectomy with lensectomy as a secondary treatment at 35 weeks postmenstrual age. The retina was reattached and the ROP stabilized in the left eye, but the fibrovascular tissue regrew from the posterior retina of the right eye (Fig. 1c). A second vitrectomy stabilized the ROP in that eye (Fig. 1d).

Immunohistochemistry of the fibrovascular tissue collected during vitrectomy was strongly positive for factor VIII over a wide area and locally positive for vimentin but negative for glial fibrillary acidic protein. These findings suggested that the tissue consisted mainly of vascular endothelial cells (Fig. 2).

Comments

We report the successful surgical results of early vitrectomy for AP-ROP.[3] Our findings suggest that when neovascularization develops only at the peripheral end of the developing vessels, the retina can be reattached by removing the vitreous framework around the fibrovascular tissue and the vitreous base. These procedures reduce the tractional forces of the fibrovascular tissue and suppress neovascular growth. Residual vitreous gel did not affect the retinal reattachment, and a regrowth of neovascularization was not observed in a previous study.[3]

In our case, the neovascularization that developed in the posterior retina could have grown along the residual vitreous gel on the retinal surface and around the optic disc. This tissue could not be completely removed during the initial vitrectomy. In cases such as this, another vitrectomy to peel the residual vitreous gel can lead to retinal reattachment, which worked well in our case.