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CLINICAL INVESTIGATION

Following the Migration of a *Toxocara* Larva in the Retina by Optical Coherence Tomography and Fluorescein Angiography

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Abstract

Background: The *Toxocara* larva is known to migrate across the retina, but the layer in which it migrates and its effect on the retina has been unknown.

Case: An ocular *Toxocara* infection was diagnosed by an immunological test on a vitreous sample from a patient with a retinal lesion that had migrated. Optical coherence tomography (OCT) and fluorescein angiography (FA) were used in this investigation.

Observations: Many small lesions were first detected in the peripheral retina, and vitrectomy was performed because of vitreous haze. Two peripapillary lesions were found during the vitrectomy. OCT of one lesion demonstrated a highly reflective mass located in the nerve fiber layer, and FA showed dye leakage from the lesion as well as hyperfluorescence of the disc. Three weeks later, another lesion was found in the macular area, and OCT and FA findings were the same as for the first lesions. Fluorescein leakage was also observed along the presumed path of the migrating larva.

Conclusions: The movement of the lesion from the peripapillary area to the macular area suggested that a *Toxocara* larva had migrated across the retina. OCT images indicated that the larva moved in the nerve fiber layer, and FA showed that it caused severe inflammation along its pathway. **Jpn J Ophthalmol** 2005;49:159–161 © Japanese Ophthalmological Society 2005

Key Words: fluorescein angiography, migration, ocular toxocariasis, optical coherence tomography, *Toxocara* larva

Introduction

The *Toxocara* larva commonly migrates across the retina,[1,2] but in which retinal layer the larva migrates has not been clearly determined. We report a case of antibody-confirmed toxocariasis in which the migration was followed by optical coherence tomography (OCT) and fluorescein angiography (FA).

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Case Report

A 51-year-old man complained of blurred vision in the left eye. Ophthalmoscopy demonstrated lesions in the peripheral retina of the left eye, and the best-corrected visual acuity was 20/20 OS. Many white masses were present in the superior peripheral retina, with cells in the vitreous. Ocular toxocariasis with peripheral granuloma was suspected, and the patient was treated with oral prednisone (30 mg daily, tapered over 2 months) and diethylcarbamazine (100 mg/day for 3 days, 300 mg/day for 3 days, followed by 300 mg/week for 8 weeks).

The vitreal turbulence increased, and endophthalmitis developed. Vitrectomy was recommended, and informed consent was obtained. Pars plana vitrectomy was performed with cryocoagulation of the peripheral lesions. Intraopera-

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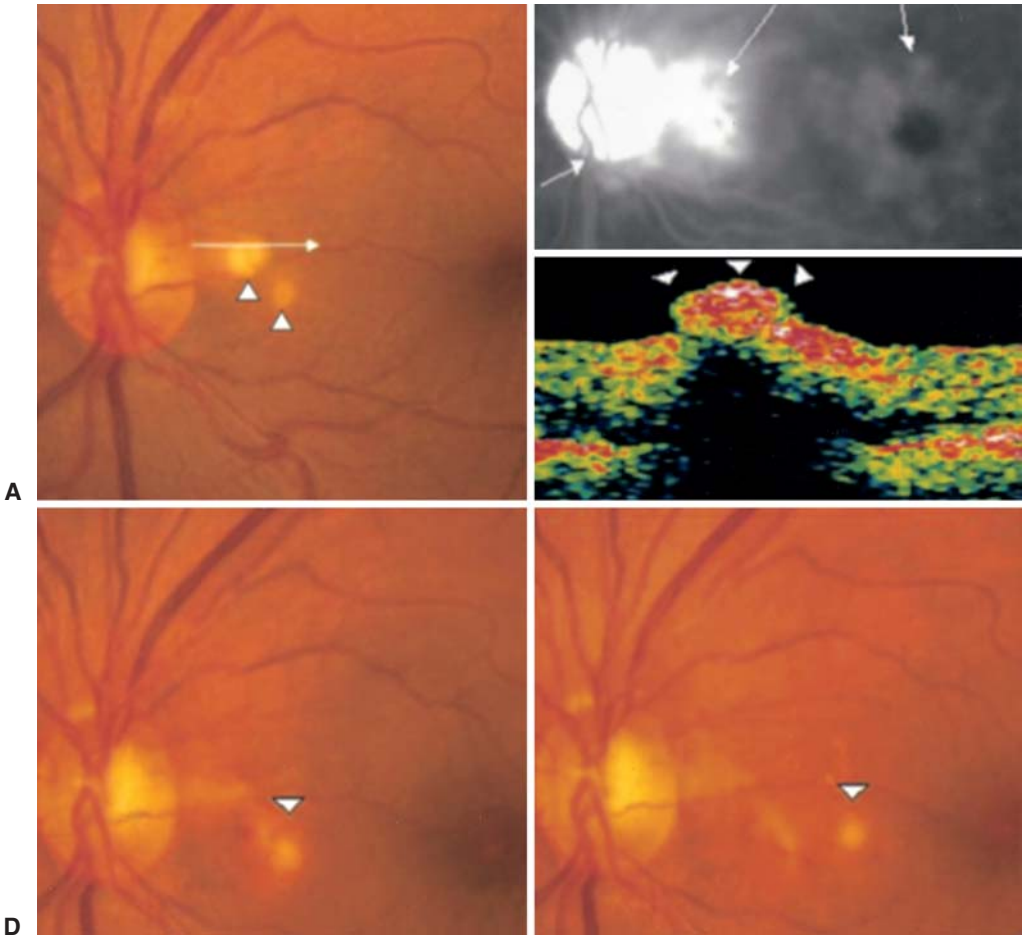

Figure 1A–E. Fundus photographs, fluorescein angiograms, and optical coherence tomography (OCT) images before and after the migration of a *Toxocara* larva. **A** Fundus photograph before migration showing two yellowish white lesions (*arrowheads*) with slight papillary hyperemia. The *arrow* points to the location and indicates the direction of the OCT scan. **B** Late-phase fluorescein angiogram showing dye leakage at the lesion, optic disc, and macula. The optic disc is hyperfluorescent (*arrows*). **C** OCT image of the nasal lesion. A hyperreflective mass can be seen protruding into the vitreous (*arrowheads*); the retina is thickened. **D** Fundus photograph 1 week after the migration: the nasal lesion has coalesced with the temporal lesion. **E** Fundus photograph 2 weeks after the migration: the lesion is now one disc diameter away from the optic disc.

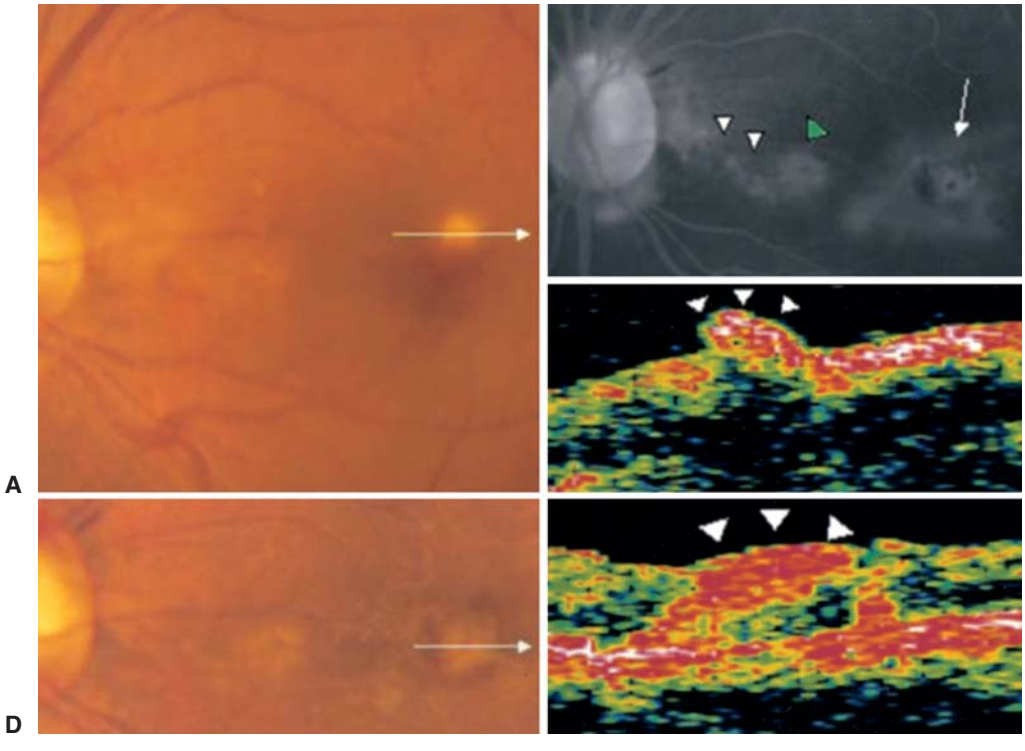

Figure 2A–E. Fundus photographs, fluorescein angiograms, and OCT images after the migration. **A** Fundus photograph taken 3 weeks after the migration showing a yellowish white lesion in the upper foveal area of the macula. The direction of the OCT scan is shown by the *arrow*. **B** Late-phase fluorescein angiogram 3 weeks after the migration. There is dye leakage from the lesion, the macula (*arrow*) and along the track of migration (*white arrowheads*). Window defects are seen at the site of the photocoagulation (*green arrowhead*). **C** Three weeks after the migration, OCT shows a highly reflective protrusion on the retinal surface (*arrowheads*), with thickened retina. **D** Fundus photograph at 6 months after the migration. The scars of healed lesions can be seen at the fovea in the macular region. The direction of the OCT scan is shown by the *arrow*. **E** OCT image 6 months after the migration. A highly reflective mass (*arrowheads*) is seen in the retina.

tively, two yellowish white lesions were detected near the optic disc with slight papillary hyperemia (Fig. 1A).

Vitreous samples were collected, and an enzyme-linked immunosorbent assay showed a strong positive reaction to the larval antigen of *Toxocara canis*.

Postoperatively, the vitreous cleared, and OCT of one of the peripapillary lesions showed a highly reflective mass protruding from the thickened retina into the vitreous (Fig. 1C). FA demonstrated dye leakage from the mass and the macula, with a hyperfluorescent optic disc (Fig. 1B). This result indicated that the inflammation extended from the mass to the macula and to the optic disc.

The peripapillary lesions coalesced in a week and began spreading toward the macula (Fig. 1D). In 2 weeks, the lesion was one disc diameter from the optic disc (Fig. 1E). It was photocoagulated with an argon laser (Argon Green with 200 mW of power; spot size, 50 μm; and duration, 0.3 s), but, the lesion continued to migrate to the superior foveal area. It stopped moving at 3 weeks (Fig. 2A). The patient's visual acuity was 20/100, and OCT showed that the migrating larva remained in the same intraretinal layer (Fig. 2C). FA showed dye leakage from the lesion and along the trail of migration (Fig. 2B).

With time, the size of the lesion decreased, and the inflammation subsided at the optic disc. There was still dye leakage along the trail of the migration. Without further management, the lesion healed 6 months later (Fig. 2D), and the patient's visual acuity improved to 20/50. OCT showed hyperreflection at the site of the lesion with the disappearance of the protrusion, but the retina was still thickened (Fig. 2E).

Comments

Migration of the *Toxocara* larva has been reported but without details on the intraretinal pathway.[1,2] In our patient, the OCT images obtained before and after the migration showed that the protruding lesions were highly reflective on the retinal surface, more specifically from the nerve fiber layer to the internal limiting membrane. FA showed dye leakage along the track of the migration and from the macular area, with a hyperfluorecent optic disc. Ophthalmoscopy demonstrated that the lesions were probably in the nerve fiber layer. These findings indicate that the *Toxocara* larva most likely migrated in the nerve fiber layer and caused the inflammation to the optic disc and macula.

In a case of posterior pole *Toxcara* granuloma, Higashide et al.[3] demonstrated by OCT that the granuloma was located in the subretinal space and resembled choroidal neovascularization. In our patient, the OCT image did not show any granuloma, perhaps because the larva was migrating. Takayanagi et al.[4] found that in an experimental model, migrating *Toxocara* larvae did not cause any granuloma to form.[4] Perhaps the pathology of a lesion migrating in the retinal surface is quite different from that of a subretinal lesion; thus, the experimental model might correspond to our case.

The peripapillary lesion appeared concurrently with the development of apparent endophthalmitis. However, because of the vitreous haze, we were not able to observe the fundus in detail when the lesion migrated to the peripapillary area, and thus we could not determine whether the larva had migrated from the peripheral retina or from elsewhere. The endophthalmitis apparently developed while the larva migrated in the retina. This finding indicates that the migration of the *Toxocara* larva caused the inflammation of the retina.

In conclusion, our case showed that a *Toxocara* larva in the nerve fiber layer can migrate and cause severe inflammatory reactions in the optic disc and macula. The anthelmintic drug and steroids were ineffective in stopping the migration or in reducing the retinal and papillary inflammation.

References

1. Sorr EM. Meandering ocular toxocariasis. Retina 1984;4:90–96.
2. Shields JA. Ocular toxocariasis. A review. Surv Ophthalmol 1984; 28:361–381.
3. Higashide T, Akao N, Shirao E, Shirao Y. Optical coherence tomographic and angiographic findings of a case with subretinal *Toxocara* granuloma. Am J Ophthalmol 2003;136:188–190.
4. Takayanagi TH, Akao N, Suzuki R, Tomoda M, Tsukidate S, Fujita K. New animal model for human ocular toxocariasis: ophthalmoscopic observation. Br J Ophthalmol 1999;83:967–972.