CASE REPORT

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A Case of Sensory Exotropia Caused by Ocular Toxocariasis

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ABSTRACT We report a case of sensory exotropia caused by ocular toxocariasis. A four-year-old boy was admitted to our clinic after his family noticed a new outward shift in the left eye for about a month. The visual acuity was 10/10 in the right eye and 3/10 in the left eye. A 30 prism diopter exotropia was observed in the left eye without alternation. Fundus examination revealed condensed vitreous, subretinal deposits, peripheral retinal exudates, tractional retinal detachment and a vascularized fibrotic membrane. Clinical and laboratory findings confirmed Toxocara infection. The patient underwent lensectomy and vitrectomy. In childhood, in cases of unilateral, non-alternating strabismus, especially exotropia, sensory strabismus should always be considered, and a careful fundus examination should be performed. Early diagnosis and treatment are critical to prevent monocular vision loss and systemic complications.

Keywords: Ocular toxocariasis; sensory exotropia; vitritis

Toxocariasis is a parasitic infection prevalent worldwide and a significant cause of blindness in childhood. Environmental contamination can lead to the transmission of larvae from *Toxocara canis* or *Toxocara* cati.¹ The disease can manifest as systemic toxocariasis (also known as visceral larva migrans) with major organ involvement or as an ocular infection.² Ocular toxocariasis (OT) typically presents as a unilateral retinal granuloma or inflammatory mass in the posterior pole. Severe cases involve vitreous inflammation, macular edema and retinal detachment, leading to vision loss.³

Diagnosis of OT relies on clinical findings from ophthalmic examination. Serological tests can support the diagnosis by detecting antibodies to the *Tox*-

ocara parasite. Treatment is typically planned according to symptom severity.⁴

Persistent unilateral or bilateral visual deprivation can compromise sensory fusion and lead to sensory strabismus. Conditions such as severe anisometropia, congenital unilateral cataract, corneal opacity, retinal diseases, and optic nerve anomalies can cause visual impairment.

Herein, we present a four-year-old boy with sensory exotropia caused by OT.



A four-year-old boy was admitted to our department after his family noticed a new outward shift in the left

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eye. Written parental consent was obtained. Visual acuity was 10/10 in the right eye and 3/10 in the left eye using the E chart. Anterior and posterior segment examinations of the right eye were normal. A 30 prism diopter exotropia was observed in the left eye without alternation. The left eye revealed normal anterior segment findings. Fundus examination showed condensed vitreous, subretinal deposits and peripheral retinal exudates, vascularized fibrotic membranes, macular ectopia and tractional retinal detachment (Figure 1A). Optical coherence tomography showed subretinal deposits and subretinal fluid in the inferior quadrant (Figure 2A). Cycloplegic refraction values were +1.00 (+0.50x115) in the right eye and +1.00 (-1.25x135) in the left eye.

Laboratory tests revealed positive *Toxocara* antibodies with serum enzyme-linked immunosorbent assay (ELISA), elevated immunoglobulin (Ig) E levels (1,200 IU/mL, reference range 10-180 IU/mL), normal white blood cell count (8.7x10³/micL, reference range 5.5-15.5) and normal eosinophil levels (0.28x10³/micL, reference range 0-0.7). Abdominal ultrasound findings were normal.

The patient was started on albendazole and systemic prednizolone (4 mg) treatment by the pediatric clinic. Along with systemic treatment, lensectomy and vitrectomy were performed at a tertiary clinic due to the extension of the tractional membranes to the lens. Tractional membranes were removed and silicon oil was used to reattach the retina (Figure 1B).

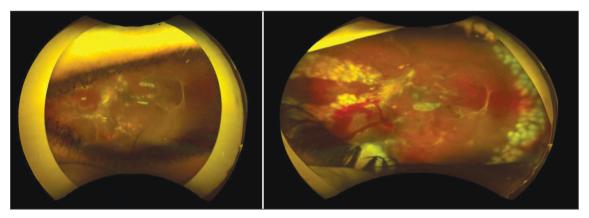


FIGURE 1: The fundus photography taken before and after operations. A) Wide-field fundus photography shows dense vitreous, subretinal deposits, proliferative membranes and tractional retinal detachment. B) After the lensectomy and vitrectomy procedure, attached retina and fibrotic membranes under silicon oil are shown. Retinal granuloma at the inferior retinal quadrant can be visualized. Images were presented with the courtesy of Prof. Dr. Şengül Özdek.

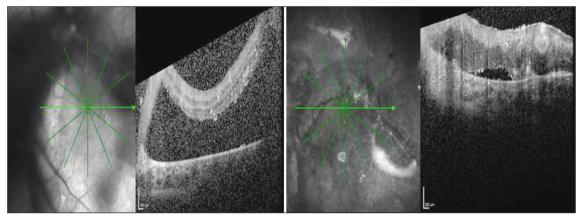


FIGURE 2: Optical coherence tomography images taken before and after the lensectomy and vitrectomy procedure. A) Tractional retinal detachment and subretinal fluid at the posterior pole are present, along with posterior shadowing of the choroid. Intraretinal hyperreflective inflammatory dots are seen and the signal intensity is low due to vitreous activity. B) Attached retina under silicone oil is shown. Diffuse thickening and subretinal fluid can be visualized at the subfoveal area. Images were presented with the courtesy of Prof. Dr. Sengül Özdek.

At the 6-month postoperative visit, the best-corrected visual acuity remained at light perception. Fundus examination showed fibrotic membranes and an attached retina under silicon oil. Optical coherence tomography images showed diffuse thickening and subretinal fluid in the macular area (Figure 2B).

DISCUSSION

OT primarily affects young patients with no gender predilection and is mostly unilateral. Diagnosis is based on typical clinical features, including peripheral or posterior granuloma, vitreous inflammation and tractional band s wit h a n inflammatory mass. The posterior pole granuloma is the most commonly presented form of OT with a visual acuity less than 20/40.5

Diagnosis of OT is based on ophthalmological findings, with serological tests supporting, such as serum ELISA titers and antibodies to Toxocara. Standard diagnostic tests include indirect ELISA based on the excretory-secretory antigens of T. canis. The TES (Toxocara excretory-secretory antigens)-based ELISA for IgG antibodies detection has been reported to be 78% sensitive and 92% spesific in diagnosing Toxocara infection. Western blotting is a confirmatory test that can be used to verify ELISA results by distinguishing spesific *Toxocara* antibodies from those that might cross-react with antigens from other helmints. However, it is less commonly used due to its higher cost and the complexity of the procedure. Diagnostic methods including optical coherence tomography, fundus florescein angiography, co mputed tomography and ocular ultrasonography are also useful. There is usually no eosinophilia in OT, as in our case. In unexplained chorioretinitis or vitritis, evaluating the Goldman-Witmer coefficient of T. canis is also beneficial.6

Standard treatment of OT includes corticosteroids for active ocular inflammation. Although the role of antihelminthic therapy is unclear, combined corticosteroid and albendazole therapy (800 mg/day for adults and 400 mg/day for children, for 2-4 weeks) is preferred for eyes with active ocular inflammation. Vitreoretinal surgery is useful to treat retinal detachment and removes vitreous opacities and retinal membranes.⁷

Differential diagnosis includes primarily retinoblastoma, retinopathy of prematurity, congenital cataract, persistent fetal vasculature, infectious endophthalmitis, and exudative or hemorrhagic retinopathies. As retinoblastoma is the most prevalent malignant intraocular neoplasm in childhood, distinguishing it, especially the sporadic, unilateral variant, from OT is crucial. The presence of eosinophils and the absence of malignant cells in cytologic examinations of the aqueous or vitreous may aid in differentiation. Intraocular calcification, though less common, can be seen in OT with significant ocular disruption or phthisis.8 However, calcifications are present in approximately 85% of retinoblastoma cases and are considered a crucial feature for distinguishing it from simulating lesions.9 In cases of suspected retinoblastoma, interventional procedures like biopsy should be avoided because of the possible risk of tumor dissemination. 10 Peripheral granulomas in OT may cause retinal tissue pulling, leading to a "dragged disc" appearance, a characteristic feature seen in conditions such as familial exudative vitreoretinopathy disease and hereditary retinal vascular anomalies. Retinopathy of prematurity is bilateral and affects preterm infants with low birth weight. Although proliferative changes and membrane formation are seen in these pediatric diseases, they are not accompanied by inflammation. Persistent fetal vasculature is a unilateral congenital disorder associated with microphthalmia. Coats' disease is another unilateral disorder typically affecting young males, characterized by peripheral vascular telangiectasia and lipid exudation without epiretinal membrane formation.3 Our patient had no history of preterm birth. The family was from a rural area and the patient had a history of puppy contact. However, direct contact with infected puppies is usually not a risk factor for human Toxocariasis, as the eggs shed by such animals must undergo embryonic development before becoming infectious.¹¹

In this case, we believe the sensory exotropia developed due to reduced vision in one eye. When visual acuity is lost in one eye, it impairs sensory fusion and may lead to strabismus as a consequence of primary sensory impairment. An organic cause, such as retinal problem or refractive anisometropia, induces loss of fusion and horizontal deviation. The angle of

deviation varies from 10 to 70 PD.¹² Several factors influence the direction of sensory strabismus, including age at the onset of visual impairment, refractive error in the unaffected eye, degree of visual acuity and anatomical factors. In the literature, sensory exotropia is more common when vision impairment occurs after the age of 5 years and involves acquired vision loss.¹³

OT is a rare parasitic infestation, primarily affecting young children. Sanitary education in childhood is crucial to prevent parasitic infections. In cases of unilateral, non-alternating strabismus, sensory strabismus should always be considered, and a careful fundus examination should be performed. Early diagnosis and treatment is essential due to monocular vision loss and systemic involvement.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm

Authorship Contributions

All authors contributed equally while this study preparing.

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