Ocular Toxocariasis Masquerading as Toxoplasmosis

Sarah Madison Duff (duffsm05@gmail.com)
University of Florida https://orcid.org/0000-0002-2851-093X
Frances Saccoccio
University of Florida
Nausheen Khuddus
University of Florida
Gibran Syed Khurshid
University of Florida
Jinghua Chen
University of Florida

Brief report

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Abstract

Purpose

To report panuveitis due to Toxocara, with positive Toxoplasma serology.

Observation

A nine-year-old boy presented with a panuveitis, intermittent headaches, and a visual acuity of 20/400 in his left eye. Slit lamp examination showed anterior chamber cells and flare without keratic precipitates (KPs), vitreous cells and veils, and optic disc edema with a blurred fundus view. Initial lab values were positive for toxoplasmosis. Topical cycloplegic and steroid eye drops, in addition to trimethoprim/sulfamethoxazole (TMP/SMX) treatment, was initiated. Four days later, oral corticosteroids were started. As inflammation cleared, the fundus examination showed an inferior tractional detachment, leading to an ultimate diagnosis of ocular Toxocariasis.

Conclusion and Importance

High false positives on Toxoplasma serology and diffuse vitritis from toxocariasis limiting retinal visualization may confuse the initial diagnosis.

1. Introduction

Toxocariasis is a known, nevertheless uncommon, infection caused by the roundworms *Toxocara canis* and *Toxocara cati*. Active infection is more typical in children than adults, since children may eat contaminated soil containing embryonated eggs in the feces of pregnant or young dogs and cats.[1]. Serological testing shows that 5.1–14% of the United States population has been in contact with Toxocara, with higher percentages in those living below the poverty line.[2–3]. Despite the prevalence of exposure, ocular toxocariasis, the result of the migration of a larva into the eye, is rather infrequent. A survey in collaboration with the American Academy of Ophthalmology found that in 2009–2010 there were only 68 cases of ocular toxocariasis presenting to ophthalmologists in the United States that year.[4].

Unlike toxocariasis, toxoplasma is much more common, with an estimated 11% of the United States population over age six having been infected and with an estimated 2% of those infected having an episode of ocular toxoplasmosis.[5–6]. Like toxocariasis testing, serology is used to confirm a diagnosis. For ocular infection, clinical evaluation is most important. Toxoplasma immunoglobulin M (IgM) testing has a high-false positive rates, (positive predictive value (PPV) of toxoplasma IgM is 22–45%), likely due to technical difficulty with this test.[7]. We report a case of a boy with toxocariasis infection who was treated for toxoplasmosis initially due to a misleading Toxoplasma IgM.

2. Case Report

A 9-year-old previously healthy boy was referred to our clinic with vitritis and visual acuity of 20/400 in his left eye. The child had had blurry vision for 5 months. He denied any fever or ocular symptoms including eye pain or light sensitivity, or any previous ocular problems, surgeries, or trauma. The patient did report mild intermittent headaches. On presentation [i.e. day 1], the patient's right eye was normal, but an afferent pupillary defect (+ APD) was found in the left eye. Anterior examination of the left eye showed 1 + cell and 1 + flare without KPs. The vitreous had 3 + cells with haze and presence of vitreal veils. Grade 4 optic disc edema was visualized, but peripheral retinal examination was limited due to vitreous haze. Spectral-domain optical coherence tomography (SD-OCT) showed normal foveal contour of both eyes. Labs, ordered by the referring ophthalmologist, were pending. A preliminary diagnosis of neuroretinitis and panuveitis was made. Differential diagnoses included ocular toxoplasmosis, ocular toxocariasis, ocular bartonellosis, tuberculosis, syphilis, acute retina necrosis (ARN), Vogt-Koyanagi-Harada syndrome (VKH), and sarcoidosis. B scan was deferred since we did have a blurred view of an attached retina. A topical steroid drop and cycloplegic drop were started.

An infectious disease consult confirmed this 9-year-old lived in rural Florida and had had exposure to many animals, including cats, cows, pigs, chickens, a horse, and dogs. His parents did remember that the children played with a litter of hunting puppies and were covered in puppy stool afterwards. He suffered from eczema and seasonal allergies. He hunted and camped in the area and swam in local fresh water. His travel was limited to Texas and Georgia and the family house was supplied by well-water. His history made the diagnoses of ocular toxoplasmosis, ocular toxocariasis and ocular bartonellosis more likely.

By the second visit, [day 5], initial labs had returned with a negative Toxoplasma immunoglobulin G (IgG), positive Toxoplasma IgM Antibody of 9.20 [high equivocal], negative rapid plasma reagin (RPR), and complete blood count (CBC) with an absolute eosinophil count of 883 [normal 15–500]; angiotensin-converting enzyme (ACE) and QuantiFERON-TB Gold test were negative. Based on these results, treatment was initiated for *Toxoplasma gondii*. Trimethoprim/Sulfamethoxazole (TMP/SMX) 800/160 mg was started. Four days after oral intake of TMP/SMX, oral Prednisone at 0.5 mg/kg daily was added to reduce posterior segment inflammation. At the same time his abnormal eosinophil count raised the suspicion of Toxocara infection.

At the next visit, [day 14], vitritis had slightly improved and visual acuity had improved to 20/200. A previously undiagnosed inferior tractional detachment was visualized a fibrotic band extending from the ora serrata to the disc, a classic presentation for ocular toxocariasis. Based on examination findings, previous high eosinophil count and history of playing with puppies, a Toxocara antibody test was ordered, as well as repeat Toxoplasma serology. This time, Toxoplasma IgG and IgM were negative and serum Toxocara antibody was positive (enzyme-linked immunosorbent assay (ELISA)). No titer information was available as the result was either positive or negative. The diagnosis of ocular toxocariasis was made. A fourteen-day course of Albendazole was ordered by pediatric infectious disease service because of the concern of visceral larva migraines while oral prednisone was tapered and topical eye drops continued.

One month later, his visual acuity in the left eye improved to 20/70. Anterior chamber inflammation resolved, but some vitreous cells and optic disc edema remained. The inferior tractional detachment was much better visualized and a peripheral granuloma was noticed. Four months later, his visual acuity had improved to 20/30 and his eye had no active inflammation without any oral or topical medications. [Figure 1]

3. Discussion

In this case the diagnosis of ocular toxocariasis was supported by the positive IgG serology, classical presentation of a peripheral granuloma inducing retinal traction extending to the posterior pole, and a supportive social history. No invasive ocular biopsy was needed. Typically, ocular toxocariasis presents in one of three forms. While the most prevalent of these is debated, each form involves a larva in the subretinal space creating an overlying granuloma and frequently resulting in intraocular inflammation and retinal traction. The different subtypes are classified as a peripheral granuloma, posterior granuloma, or chronic endophthalmitis in which inflammation is so severe it mimics an infectious endophthalmitis and limits view of the retina. In this case the patient had tractional retinal detachment and a peripheral granuloma which was obscured by his vitritis.

Toxocara IgM is not commercially available and Toxocara IgG may be present in otherwise normal children in the US. While the definitive diagnosis could be made with histology demonstrating the larva in the affected tissues, a cytologic study of aqueous humor or of a vitrectomy sample, these are rarely performed. ELISA is the typical test of choice.[1]. However, like other immunological testing, a positive IgG only implies previous contact with the infectious agent. It is helpful to obtain a good social history to include contact with kittens or puppies, or consumption of soil. In this case, the serum Toxoplasma IgM testing was misleading and his fundus view was obscured by the severe vitritis. Many experts now recommend repeat testing of both IgM and IgG prior to the diagnosis of Toxoplasma.

Anti-parasitic therapy, Albendazole, was utilized in this case because of an elevated eosinophilia count and complaint of headache, which is the standard of care by the American Academy of Pediatrics (AAP).[8]. Eosinophilia presents more commonly in visceral toxocariasis. The 2018 AAP Red book recommends treatment while acknowledging the parasite is likely already dead. The albendazole is fairly safe, so the benefit of ensuring the parasite is dead is worth the minor side effect of the drug. However, technically Albendazole is unproven to kill intraocular larvae.[9] Most of ocular manifestations are related to the strong inflammatory response against the presence of a dead worm in the sub-retinal space. Most providers recommend anti-inflammatory therapy to limit the intraocular complications of inflammation, with topical and oral steroids being used in this case; periocular injections of corticosteroids are an alternative treatment. Surgical therapy is considered in cases with complications of vitreal hemorrhaging, extensive tractional retinal detachment, rhegmatogenous retinal detachment, or macular pucker from an epiretinal membrane. Other interventions, although not relevant to this case, may include laser photocoagulation of live, motile larvae or intravitreal anti–vascular endothelial growth factor therapy to

treat the rare occurrence of choroidal neovascularization arising in association with inactive Toxocara granulomas. [10].

4. Conclusions

In summary, toxocariasis must be considered in all cases of panuveitis when dealing with children with exposure to young animals, even in cases where Toxoplasma serology is positive. Toxoplasma IgM testing has high-false positive rates, thus we recommend repeat testing of both Toxoplasma IgM and IgG prior to the diagnosis of Toxoplasma, in addition to Toxocara serology.

Abbreviations

(KPs): Keratic Precipiates

(TMP/SMX): Trimethoprim/Sulfamethoxazole

(IgM): Immunoglobulin M

(PPV): Positive Predictive Value

(SD-OCT): Spectral-Domain Optical coherence tomography

(ARN): Acute Retina Necrosis

(VKH): Vogt-Koyanagi-Harada Syndrome

(IgG): Immunoglobulin G

(RPR): Rapid Plasma Reagin

(CBC): Complete Blood Count

(ACE): Angiotensin-Converting Enzyme

(ELISA): Enzyme-linked Immunosorbent Assay

(AAP): American Academy of Pediatrics

Declarations

Ethics approval and consent to participate:

Not applicable.

Consent for publication:

Consent for publication was obtained and documented.

Availability of data and material:

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Competing interests

The authors declare that they have no competing interests: SMD, FS, NK, GSK, JC.

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Authors' contributions

SMD wrote this article. Patient was cared for by: SMD, FS, NK, GSK, JC. Article was edited by: FS, NK, GSK, JC.

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Figures

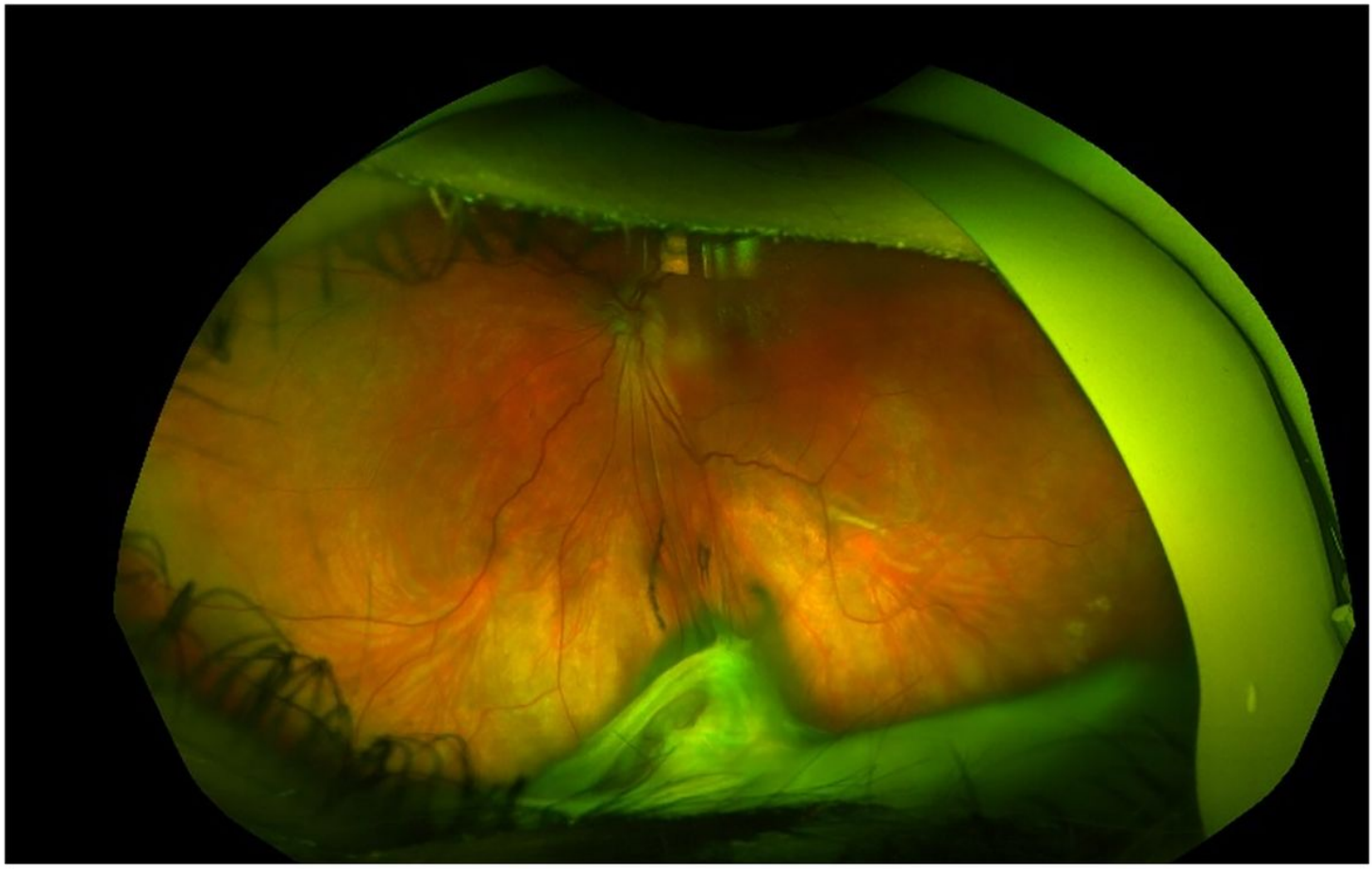

Figure 1

Ultra-widefield retinal imaging (Optos) after 4 months of treatment. There is a peripheral granuloma (blue arrow). Optic disc elevation remains (yellow arrow). A tractional retinal fold (white arrow) extends from the peripheral granuloma to the optic disc.