Case Report

Ocular toxocariasis masquerading as toxoplasmosis: a case report and literature review

Jinghua Chen [1], Sarah Madison Duff [1], Frances Saccoccio [2], Nausheen Khuddus [3], Nazanin Ebrahimiadib [1] and Gibran Syed Khurshid [1]

[1] *Department of Ophthalmology, College of Medicine, University of Florida Gainesville, Florida, United States*
[2] *Department of Pediatrics, College of Medicine, University of Florida Gainesville, Florida, United States*
[3] *Columbia Eye Associates & Family Focus Eye Care, Gainesville, Florida, United States*

ABSTRACT

Background: False-negative and false-positive results in Toxoplasma serology are possible, and this could be misleading. Here, we report the case of a boy with Toxocara-associated panuveitis who was initially treated for toxoplasmosis owing to false-positive Toxoplasma immunoglobulin M (IgM) serology.

Case Presentation: A nine-year-old boy presented with intermittent headaches and blurred vision in the left eye. Close contact with domesticated animals was remarkable in the patient's history. Upon examination, vision was 20/400 in the left eye. Slit-lamp examination revealed anterior chamber cells and flare without keratic precipitates, with vitreous cells and veils, optic disc edema, and a blurred fundus appearance. A systemic investigation revealed the presence of anti-Toxoplasma IgM antibodies. Treatment was initiated using topical cycloplegic and corticosteroid eye drops, in addition to oral trimethoprim/sulfamethoxazole. Oral corticosteroids were also administered. As the inflammation resolved, an inferior tractional detachment was detected on fundus examination, leading to the ultimate diagnosis of ocular toxocariasis. An enzyme-linked immunosorbent assay was positive for serum Toxocara antibodies. A fourteen-day course of oral albendazole was ordered by the pediatric infectious disease service because of the concern for visceral larva migrans, while topical eye drops were continued and oral prednisone was tapered. One month later, visual acuity in the left eye had improved to 20/70. The anterior chamber inflammation resolved; however, some vitreous cells and optic disc edema persisted. The inferior tractional detachment was much better visualized, and a peripheral granuloma was observed. Four months later, without any oral or topical medications, the patient's visual acuity had improved to 20/30 and his eye had no active inflammation. The patient has been followed up for two years and has never developed any other lesions.

Conclusions: False-positive results on Toxoplasma serology and diffuse vitritis from toxocariasis that limited retinal visualization complicated the initial diagnosis in this case. In diagnosing the etiology of uveitis, ocular examination and detailed history taking should be emphasized, as laboratory results may be misleading.

KEYWORDS

toxocaras, toxocara cani, toxoplasma gondii infection, ocular toxoplasmosis, albendazole monohydrochloride, steroid, cycloplegic, uveitides, panuveitis, retinal detachments

Correspondence: Jinghua Chen, Department of Ophthalmology, College of Medicine, University of Florida, Newberry Rd, Gainesville, Florida, USA. Email: jinghuachen@ufl.edu. ORCID iD: https://orcid.org/0000-0002-4092-7009

How to cite this article: Chen J, Madison Duff S, Saccoccio F, Khuddus N, Ebrahimiadib N, Syed Khurshid G. Ocular toxocariasis masquerading as toxoplasmosis: a case report and literature review. Med Hypothesis Discov Innov Optom. 2024 Summer; 5(2): 85-92. DOI: https://doi.org/10.51329/mehdioptometry202

Received: 01 November 2023; Accepted: 22 May 2024

INTRODUCTION

Toxocariasis is an infection caused by the roundworms Toxocara canis and Toxocara cati. It is acquired by the ingestion of soil contaminated with embryonated eggs in the feces of puppies and kittens, the definitive hosts [1]. It can also be transmitted via the ingestion of contaminated water or uncooked meat or liver. Toxocara eggs are not excreted in human feces; therefore, stool testing is not helpful. Eggs hatch in the human intestines and larvae enter the bloodstream and travel to tissues (visceral larva migrans and ocular larva migrans) [2]. Ocular toxocariasis results from the movement of larvae to the optic nerve and their migration to the subretinal space, leading to the development of an eosinophilic granuloma [3].

Serological testing indicates that 5.1–14% of the United States population has been exposed to Toxocara, with a higher incidence in those with lower socioeconomic status [4]. Active infection is uncommon but occurs more frequently in children. Despite the relatively high prevalence of exposure, ocular toxocariasis, which results from migration of larvae into the eye, rarely occurs [5, 6]. A 2009–2010 survey in collaboration with the American Academy of Ophthalmology found that only 68 cases of ocular toxocariasis presented to ophthalmologists in the United States that year [6].

Toxoplasmosis is more common than toxocariasis, with an estimated 11% of the United States population over the age of six years having been infected, 2% of whom develop ocular toxoplasmosis [7, 8]. As in toxocariasis, serology is the primary method used to confirm the diagnosis of systemic disease. However, fundus abnormalities on photographs are pathognomonic in confirming ocular infections. Owing to technical difficulties, Toxoplasma immunoglobulin M (IgM) testing has a high false-positive rate and a positive predictive value of only 22–45% [8, 9]. Herein, we report the case of a boy with ocular toxocariasis who was initially treated for toxoplasmosis because of misleading Toxoplasma IgM serology.

CASE PRESENTATION

A previously healthy nine-year-old boy was referred to our clinic with a diagnosis of uveitis in the left eye. The child reported blurred vision for five months, along with mild intermittent headaches. He denied having any ocular symptoms, including eye pain or light sensitivity, or any previous ocular problems, surgery, or trauma. On presentation (day 1), visual acuity was 20/20 in the right eye and 20/400 in the left eye using a Snellen chart (Nidek Co., Ltd., Gamagori, Aichi, Japan). An afferent pupillary defect [10] was detected in the left eye. Slit-lamp examination (Haag-Streit AG, Bern, Switzerland) of the left eye revealed 1+ cells and 1+ flare in the anterior chamber with no keratic precipitates. The vitreous had 3+ cells with haze and the presence of vitreous veils. Fundus examination revealed severe optic disc edema; however, the view of the peripheral retina was limited because of vitreous haze. Spectral-domain optical coherence tomography (Spectralis™ OCT; Heidelberg Engineering, Heidelberg, Germany) macular cube imaging of a 6 × 6-mm area showed normal foveal contour in both eyes (Figure 1).

A preliminary diagnosis of unilateral panuveitis was established. The differential diagnoses included ocular toxoplasmosis, ocular toxocariasis, ocular bartonellosis, tuberculosis, syphilis, acute retinal necrosis, sarcoidosis, and conditions mimicking retinoblastoma [11]. B-scan ultrasonography was deferred at the initial presentation because of an apparently attached retina on fundus examination. The posterior segment view was excessively blurred; however, the retina was still observed as flat. We did not include this image in this article because of its blurriness. This finding was later confirmed by ultra-widefield (UWF™) fundus imaging using a non-contact camera (Optos P200DTx ICG; Optos, Marlborough, MA, USA) (Figure 2) [12]. Treatment with topical corticosteroid and cycloplegic drops was initiated while laboratory results were pending. Findings of an ocular examination of the right eye were within normal limits. An infectious disease consultation confirmed that this nine-year-old boy lived in rural Florida and had been exposed to numerous animals, including cats, cows, pigs, chickens, horses, and dogs. His parents recalled that he had played with a litter of hunting puppies and was subsequently covered with puppy stool. The boy had a history of eczema and seasonal allergies. He hunted and camped in the area and swam in local bodies of fresh water. The patient's travel was limited to Texas and Georgia, and the family house was supplied with well water. The patient's history increased the likelihood of ocular toxoplasmosis, ocular toxocariasis, and ocular bartonellosis [11, 13].

By the second visit (day 5), systemic investigation revealed a negative Toxoplasma IgG test result, Toxoplasma IgM antibody level was high equivocal [14], negative rapid plasma reagin, and a blood absolute eosinophil count of 883 cell/μL (normal range, 15–500 cell/μL) [15]. Angiotensin-converting enzyme [16] and QuantiFERON-TB Gold test results were negative [17, 18]. Based on these results, treatment for Toxoplasma gondii infection was initiated. A course of oral trimethoprim/sulfamethoxazole (TMP/SMX) [19, 20] was commenced together with topical corticosteroid and cycloplegic agents [21]. Systemic corticosteroid treatment was initiated four days afterward. However, the infectious disease consultant suspected Toxocara infection based on the abnormal eosinophil count.

At the next visit (day 14), as the inflammation had cleared, fundus examination revealed an inferior tractional retinal detachment, leading to further testing and the ultimate diagnosis of ocular toxocariasis. Based on the examination findings, previously high blood eosinophil count, and history of playing with puppies, a Toxocara antibody test and repeat Toxoplasma serology were ordered. Toxoplasma IgG and IgM results were negative, excluding toxoplasmosis [22, 23]. The serum Toxocara antibody result was positive using enzyme-linked immunosorbent assay (ELISA) [24, 25].

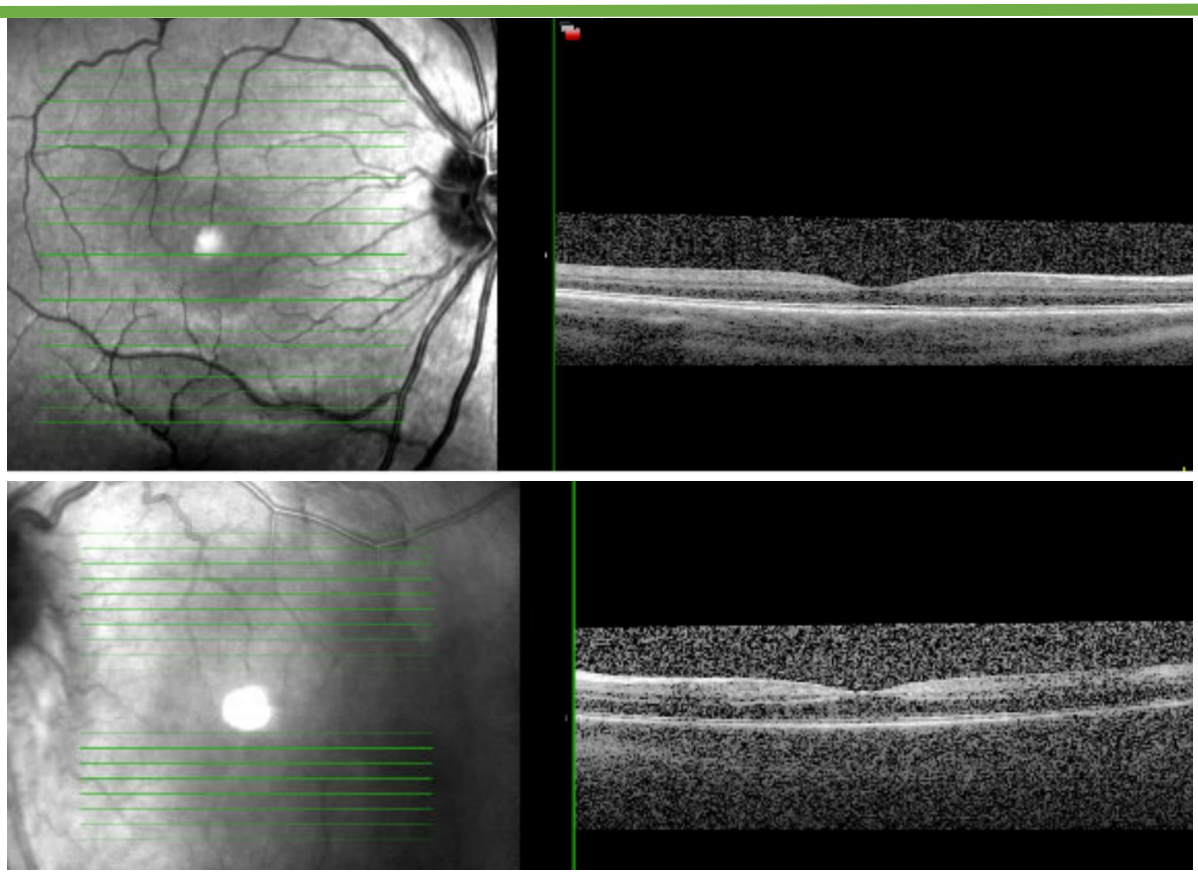

Figure 1. Spectral-domain optical coherence tomography (Spectralis™ OCT; Heidelberg Engineering, Heidelberg, Germany) macular cube imaging of a 6 × 6-mm area demonstrates that the right eye (top photo) has normal macular contour and clear media and the left eye (bottom photo) has hazy media without apparent macular thickening or fluid.

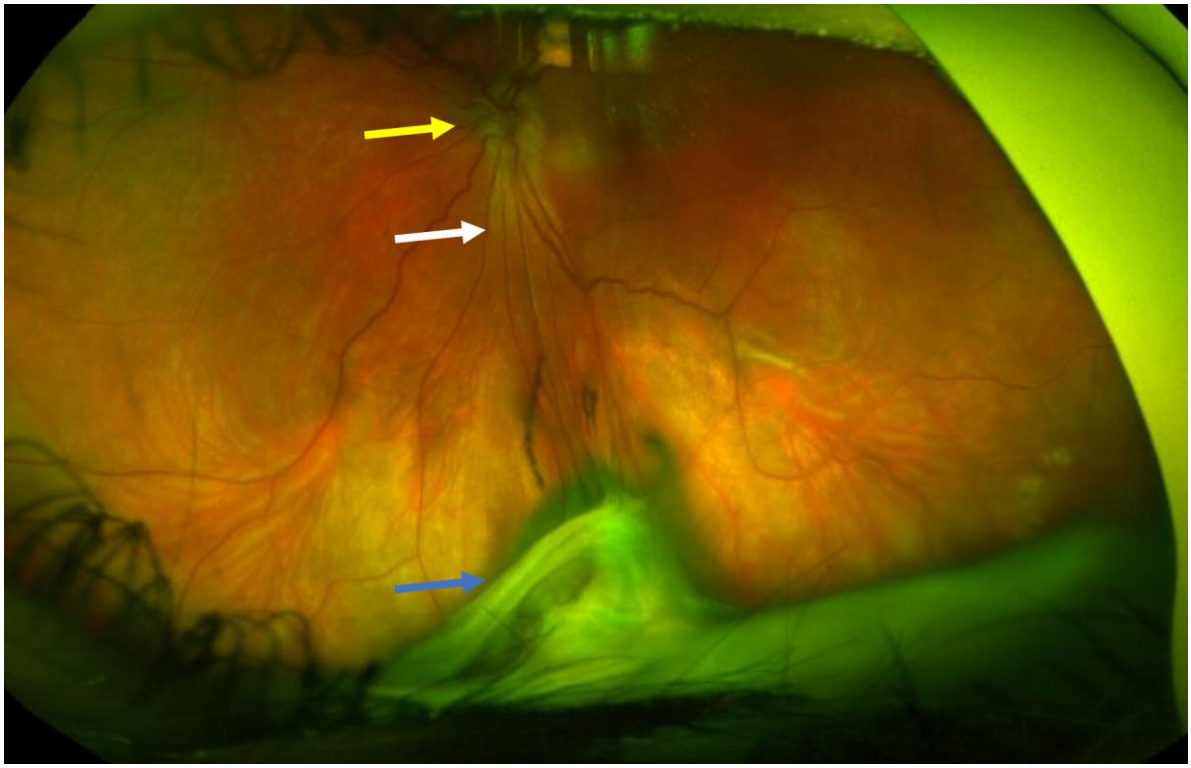

Figure 2. Ultra-widefield (UWF™) fundus image of the left eye using a noncontact camera (Optos P200DTx ICG; Optos, Marlborough, MA, USA) four months after treatment for ocular toxocariasis. A peripheral granuloma (blue arrow) with residual optic disc elevation (yellow arrow) is present. A tractional retinal fold (white arrow) extends from the peripheral granuloma (blue arrow) to the optic disc (yellow arrow). Brown pigmentation is observed in the inferior retinal folds.

Therefore, ocular toxocariasis was diagnosed. A fourteen-day course of oral albendazole was ordered by the pediatric infectious disease service because of concerns for visceral larva migrans, while topical eye drops were continued and oral prednisone [5, 26] was tapered. One month later, visual acuity in the left eye had improved to 20/70. The anterior chamber inflammation had resolved; however, some vitreous cells and optic disc edema persisted. The inferior tractional detachment was much better visualized, and a peripheral granuloma was observed. Four months later, without any oral or topical medications, the patient's visual acuity had improved to 20/30 and his eye showed no active inflammation, as shown in Figure 2. The patient has been followed up for two years and has never developed any other lesions.

This study was performed in accordance with the Declaration of Helsinki and was compliant with the Health Insurance Portability and Accountability Act. This retrospective review of patient data did not require ethical approval in accordance with institute's guidelines. The patient's mother provided written informed consent to publish this case, including the publication of images. Information revealing the participant's identity was avoided.

DISCUSSION

The boy in this case lived in rural Florida and was exposed to numerous animals, including cats and dogs. His blood eosinophil count was elevated, suggesting a parasitic infection. The serum Toxocara antibody result was positive, and clinical examination revealed panuveitis in the left eye. After partial resolution of the initial inflammation, there was evidence of inferior granuloma, inferior retinal folds, and tractional retinal detachment extending from the granuloma toward the optic nerve. Streaks of brown pigment were observed along the inferior retinal folds. Based on these findings, a diagnosis of ocular toxocariasis was established and managed accordingly.

The relevant case reports, retrospective studies, and reviews are summarized in Table 1 [27-37]. Typically, ocular toxocariasis is unilateral and can present in three forms, including peripheral granuloma, posterior granuloma, or rarely, chronic endophthalmitis [28-30, 33-38]. Granuloma is a retinochoroiditis that usually appears as a poorly demarcated yellow-white lesion. It develops in response to subretinal larvae and frequently results in intraocular inflammation and retinal traction [27-30, 34-38]. Necrosis may occur in the center of the granuloma and in the black center, which may consider of larvae or blood vessels. Ocular toxocariasis is a differential diagnosis of leukocoria [27]. Our patient had an inferior granuloma, inferior retinal folds, and tractional retinal detachment extending from the granuloma toward the optic nerve in the affected eye, which was evident after the inflammation subsided.

Although ocular toxoplasmosis can be diagnosed by serological testing, fundus photography revealing focal retinitis adjacent to an old, pigmented scar is pathognomonic for diagnosis [39]. Severe panuveitis is usually present; therefore, the hue of retinitis through many vitreous cells mimics a "headlight in fog". The diagnosis can be further confirmed by molecular testing of ocular fluids, and for a higher yield, with vitreous sampling [40]. Exposure to both Toxoplasma and Toxocara is common in the United States and can be explained by the common route of exposure to contaminated soil [41]. Among school children in the city of Jataizinho, Parana, 27.4% (113/412) were reactive for these two species [42]. In a rural population in the Pelotas municipality, Brazil, concomitant seropositivity for Toxoplasma gondii and Toxocara canis was 38.3% [43]. The seroprevalences of Toxoplasma gondii, Toxocara spp., and their co-infection in pregnant women were 39.7%, 21.2%, and 9.5%, respectively. Regarding risk factors, contacts with cats and dogs were significantly associated with seropositivity for Toxoplasma gondii and Toxocara spp., respectively [44]. Multivariate analysis controlling demographic and risk factors showed that persons infected with Toxocara spp. were more likely to be infected with Toxoplasma gondii (odds ratio, 1.93; 95% confidence interval, 1.61–2.31); similarly, persons infected with Toxoplasma gondii were more likely to be infected with Toxocara spp. [45]. However, our patient was repeatedly seronegative for toxoplasmosis, excluding the possibility of coinfection.

The serological presence of IgM in the absence of IgG indicates early infection, whereas in newborns, it indicates congenital toxoplasmosis. The presence of both IgM and IgG indicates a current infection, chronic infection, or reactivation [46]. IgM may be present for several months after the infection resolves. Molecular testing, such as polymerase chain reaction (PCR), detects DNA of a parasite or protozoan in body fluids. However, false-negative and false-positive results are possible [22, 47, 48]. A negative test result lessens the likelihood of toxoplasmosis, but does not rule out infection; Toxoplasma may not be present in sufficient quantities to be detected. Evaluation of local specific antibody production against Toxoplasma gondii in ocular fluid and comparison with production levels in blood (Goldmann–Witmer coefficient) is even more reliable for establishing the diagnosis [28, 49]. Thirty-six percent of Toxoplasma gondii cases were detected by PCR, whereas a positive Goldmann–Witmer coefficient was found in 92% of cases [50].

IgG and IgM test results can vary greatly depending on the method and kit used [51]. Furthermore, the specificity of IgM tests varies greatly, even among lots from the same manufacturer [52]. IgM can persist for several years, and commercial Toxoplasma IgM diagnostic test kits can yield false-positive results [52]. Therefore, chronic toxoplasmosis can be erroneously classified as an acute infection, resulting in serious adverse consequences. Repeated testing is accordingly recommended. The interpretation of Toxoplasma serology at a reference laboratory can help differentiate recently acquired infections from chronic infections. An increasing IgM titer on subsequent testing suggests recent infection [7-9]. A positive anti-Toxoplasma IgM test result was initially misleading in our patient, as the fundus was obscured by a heavy cellular reaction, and we relied mainly on serological results. Many experts recommend repeat testing for both IgM and IgG before the diagnosis of toxoplasmosis [9, 14, 19].

Table 1. Literature review on toxocariasis

Author (Year)	Study design	Ocular findings	Laboratory results	Treatment
Xie et al. (2022) [27]	Case report, (n = 1 patient)	Leukocoria, posterior synechiae, cataract, and tractional retinal detachment.	Toxocara IgG was detected in the serum and aqueous humor. Embryonated eggs and worms of Toxocara canis were observed in the feces of the dog.	Underwent cataract surgery and received systemic anti-inflammatory therapy with oral prednisone.
Lin et al. (2022) [28]	Case report and literature review, (n = 1 patient)	Vitreous inflammatory opacity, optic disc elevation with granuloma, and proliferative membrane starting from the optic disc and extending toward the superior temporal retina, expanded around a Toxocara larva.	Anti-Toxocara IgG was detected in vitreous fluid using ELISA with a high Goldmann–Witmer coefficient.	Underwent PPV twice and received oral prednisone.
Wakabayashi et al. (2021) [29]	Case report, (n = 1 patient)	Acute endophthalmitis with hyphema mimicking pink hypopyon; non-perfused white-sheathed retinal vessels mimicking septic emboli.	The anterior chamber tap and vitreous tap culture results were negative. Blood tests revealed an elevated total IgE level. ELISA detected anti-Toxocara IgG in vitreous fluid; however, western blot was negative for serum anti-Toxocara canis IgG.	Underwent an early PPV followed by administration of oral albendazole and prednisone.
Inagaki et al. (2019) [30]	Case report, (n = 3 patients)	Granulomatous chorioretinitis, vitritis, snowbank, macular striae, retinal membranes, and cystoid macular edema.	Eosinophil proportion of 2.1% in peripheral white blood cell count. Blood testing by ELISA was negative for Toxocara antibody; however, ELISA of vitreous sample was positive. Normal MRI findings.	Empiric intravitreal clindamycin without improvement. PPV for vitreous biopsy and cataract extraction with intraocular lens implantation, as well as corticosteroid injection to control inflammation. Topical ophthalmic corticosteroid therapy was also initiated. Oral albendazole.
Fonseca et al. (2019) [31]	Case report, (n = 1 patient)	Hyperemic optic disc edema at presentation, and later, a granulomatous retinal lesion in the superior temporal artery with adjacent vitreous strands.	Serum ELISA detected Toxocara canis IgG. Aqueous humor sample was positive for Toxocara canis antibodies using immunoblot.	Combined treatment with oral albendazole 200 mg two times daily for fifteen days and methylprednisolone 32 mg with slow tapering.
Karaca et al. (2018) [32]	Case report, (n = 1 patient)	Subretinal fluid in the macula, optic nerve head edema with indistinct margins, and star-like macular exudates (neuroretinitis).	Toxocara IgG-seropositive with increased avidity using ELISA and western blot and elevated total IgE level without eosinophilia.	Administration of intravenous methylprednisolone 1 g daily for one week with oral albendazole 400 mg twice daily.
Sahu et al. (2018) [33]	Retrospective case series, (n = 16 patients)	Peripheral granuloma was the most common presentation.	A positive serum ELISA for Toxocara canis antibody occurred in 62.5% (n = 10 patients).	Combined anti-helminthic and corticosteroid administered for 31.2% (n = 5 patients), and 50% (n = 8 patients) underwent vitreous surgery.
Iddawela et al. (2017) [34]	Retrospective descriptive study, (n = 250 patients)	Uveitis (n = 53, 34.19%), vitritis (n = 20, 12.9%), choroiditis (n = 12, 7.74%), retinal lesion (n = 10, 6.45%), and endophthalmitis (n = 3, 1.94%).	Using ELISA, 62% (n = 155) were seropositive.	Not reported.
Ahn et al. (2014) [35]	Retrospective cohort study, (n = 101 patients)	Granuloma was detected in 92.1% (n = 93) (posterior pole granuloma [n = 47, 50.5%], peripheral granuloma [n =41, 44.1%], macular granuloma [n = 16, 17.2%], optic nerve granuloma [n = 2, 2.2%], and combined granuloma [n = 5, 5.4%]), intraocular inflammation in 77.2% (n = 78) (anterior uveitis [n = 2, 2.6%], intermediate uveitis [n = 53, 67.9%], posterior uveitis [n = 13, 16.7%], and panuveitis [n = 10, 12.8%]), and vitreoretinal comorbidities composed of associated retinal nerve fiber layer defect in 31.7% (n = 32), epiretinal membrane in 26.7% (n = 27), vitreous opacity in 21.8% (n = 22), tractional/rhegmatogenous retinal detachment in 12.9% (n = 13), pigmentary scarring in 7.9% (n = 8), macular edema in 4.0% (n = 4), and macular hole in 2.0% (n = 2).	Eosinophilia was detected in 10 of 86 patients (11.6%) in whom complete blood count results were available. Increased serum IgE level was detected in 39 of 56 patients (69.6%). The mean (standard deviation) ELISA titer for serum Toxocara IgG was 0.398 (0.115) (range, 0.254–0.737) (titer ≥ 0.250 was considered seropositive).	Treatment outcomes of combined administration of albendazole and corticosteroids were comparable with that of corticosteroid monotherapy; however, the six-month recurrence rate in combined treatment (n = 4, 17.4%) was significantly lower than that in corticosteroid monotherapy (n = 6, 54.5%).
Stewart et al. (2005) [36]	Retrospective chart review study, (n = 22 patients)	Unilateral inflammation (90.9%), peripheral granuloma (50%), macular granuloma (25%), and moderate to severe vitreous inflammation mimicking endophthalmitis (25%). The primary causes of vision loss were vitritis (52.6%), cystoid macular edema (47.4%), and tractional retinal detachment (36.8%).	Serum ELISA for anti-Toxocara antibodies was positive in 50% (n = 11), negative in 36.4% (n = 8), and unknown in 13.6% (n = 3). In two of these, one had a negative serum ELISA, and aqueous humor ELISA revealed positive results. In two of whom one had an unavailable serum ELISA result, vitreous ELISA was strongly positive. Overall, in 64% (n = 14), antibodies confirmed the diagnosis.	Not reported.
Logar et al. (2004) [37]	Retrospective study, (n = 239 patients)	Posterior or peripheral retinochoroiditis, vitritis, papillitis, or circumscript endophthalmitis.	Using ELISA for Toxocara canis IgG and confirmation by western blot IgG, 72% (n = 172) were seronegative and 28% (n = 67) were seropositive.	Not reported.

Abbreviations: n, number of patients; IgG, immunoglobulin G; ELISA, enzyme-linked immunosorbent assay; PPV, pars plana vitrectomy; IgE, immunoglobulin E; %, percentage; MRI, magnetic resonance imaging; mg, milligram; g, gram.

Toxocara IgM tests are not commercially available, and Toxocara IgG may be present in otherwise healthy children in the United States. ELISA is the test of choice [1]. The diagnosis of ocular toxocariasis is mainly clinical and based on obtaining relevant social history because the parasitic load and the immune response are low, and serological tests cannot detect the antibody [1, 3]. However, in dubious cases, confirmatory tests may be required, such as histological demonstration of larvae or cytological studies of ocular fluids [27-31].

Albendazole, an antiparasitic agent, was administered in our case because of eosinophilia and a complaint of headache, according to the American Academy of Pediatrics (AAP) recommendations [53]. Eosinophilia is more common in visceral toxocariasis. However, albendazole has not been demonstrated to kill intraocular larvae [35]. Most ocular manifestations are associated with a strong inflammatory response to dead worms in the subretinal space. Most providers recommend anti-inflammatory therapy to limit the intraocular complications of inflammation [3, 35, 54], with topical and oral corticosteroids used in our case. Periocular injection of corticosteroids is an alternative treatment [3, 54].

Surgical therapy is considered in cases with complications of vitreal hemorrhage, extensive tractional retinal detachment, rhegmatogenous retinal detachment, or macular pucker from an epiretinal membrane [28, 29]. Other interventions, although not relevant to this case, may include intravitreal anti-vascular endothelial growth factor therapy to treat the rare secondary choroidal neovascularization associated with inactive Toxocara granulomas, which threaten vision [36, 55].

Our case reveals the importance of re-evaluating serological tests in children with a strong history of exposure, highlights the possibility of toxocariasis- versus toxoplasmosis-induced uveitis, and could be a suitable educational example for eye care practitioners. However, being a case report, its generalizability to all cases of pediatric uveitis with similar manifestations is limited. Further large-scale multicenter studies using a spectrum of laboratory investigations in individuals suspected of having toxoplasmosis- or toxocariasis-induced uveitis may provide strong evidence in this context.

CONCLUSIONS

Toxocariasis should be considered in all cases of unilateral posterior uveitis in children exposed to young animals. Toxoplasma IgM testing has high false-positive rates; thus, repeated testing for both Toxoplasma IgM and IgG is suggested. Notably, diagnosis of the underlying etiology of uveitis mostly relies on history taking, and detailed ocular examination and laboratory testing should be tailored according to this guide.

ETHICAL DECLARATIONS

Ethical approval: This study was performed in accordance with the Declaration of Helsinki and was compliant with the Health Insurance Portability and Accountability Act. This retrospective review of patient data did not require ethical approval in accordance with institute's guidelines. The patient's mother provided written informed consent to publish this case, including the publication of images. Information revealing the participant's identity was avoided.

Conflict of interest: None.

FUNDING

This work was supported in part by an unrestricted departmental grant from Research to Prevent Blindness awarded to the Department of Ophthalmology at the University of Florida.

ACKNOWLEDGMENTS

We thank our photography team: Harry Rosa, Lisa Ellis, Michael Jederlinic, and Jessica Riefkohl.

REFERENCES

1. Rostami A, Riahi SM, Holland CV, Taghipour A, Khalili-Fomeshi M, Fakhri Y, Omrani VF, Hotez PJ, Gasser RB. Seroprevalence estimates for toxocariasis in people worldwide: A systematic review and meta-analysis. PLoS Negl Trop Dis. 2019 Dec 19;13(12):e0007809. doi: 10.1371/journal.pntd.0007809. PMID: 31856156; PMCID: PMC6922318.
2. Choi D, Lim JH, Choi DC, Lee KS, Paik SW, Kim SH, Choi YH, Huh S. Transmission of Toxocara canis via ingestion of raw cow liver: a cross-sectional study in healthy adults. Korean J Parasitol. 2012 Mar;50(1):23-7. doi: 10.3347/kjp.2012.50.1.23. Epub 2012 Mar 6. PMID: 22451730; PMCID: PMC3309047.
3. Ahn SJ, Ryoo NK, Woo SJ. Ocular toxocariasis: clinical features, diagnosis, treatment, and prevention. Asia Pac Allergy. 2014 Jul;4(3):134-41. doi: 10.5415/apallergy.2014.4.3.134. Epub 2014 Jul 29. PMID: 25097848; PMCID: PMC4116038.
4. Jarosz W, Mizgajska-Wiktor H, Kirwan P, Konarski J, Rychlicki W, Wawrzyniak G. Developmental age, physical fitness and Toxocara seroprevalence amongst lower-secondary students living in rural areas contaminated with Toxocara eggs. Parasitology. 2010 Jan;137(1):53-63. doi: 10.1017/S0031182009990874. Epub 2009 Aug 20. PMID: 19691865.
5. Woodhall DM, Fiore AE. Toxocariasis: A Review for Pediatricians. J Pediatric Infect Dis Soc. 2014 Jun;3(2):154-9. doi: 10.1093/jpids/pit066. Epub 2013 Oct 31. PMID: 26625368.

6. US Centers for Disease control and Prevention (2011). 'Ocular Toxocariasis-United States, 2009-2010'. Morbidity and Mortality Weekly Report. Available at: https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6022a2.htm(Accessed: November 30, 2023).
7. McCall J, Rothfeldt L, Giesbrecht K, Hunt A, Bauck L, Scheftel J, Birn R, Buss B, Schroeder B, Haupt TE, Klos R, Straily A. Public Health Surveillance and Reporting for Human Toxoplasmosis - Six States, 2021. MMWR Morb Mortal Wkly Rep. 2022 Jul 15;71(28):889-893. doi: 10.15585/mmwr.mm7128a1. PMID: 35834417; PMCID: PMC9290386.
8. Holland GN. Ocular toxoplasmosis: a global reassessment. Part I: epidemiology and course of disease. Am J Ophthalmol. 2003 Dec;136(6):973-88. doi: 10.1016/j.ajo.2003.09.040. PMID: 14644206.
9. Dhakal R, Gajurel K, Pomares C, Talucod J, Press CJ, Montoya JG. Significance of a Positive Toxoplasma Immunoglobulin M Test Result in the United States. J Clin Microbiol. 2015 Nov;53(11):3601-5. doi: 10.1128/JCM.01663-15. Epub 2015 Sep 9. PMID: 26354818; PMCID: PMC4609698.
10. Glazer-Hockstein C, Brucker AJ. The detection of a relative afferent pupillary defect. Am J Ophthalmol. 2002 Jul;134(1):142-3. doi: 10.1016/s0002-9394(02)01503-9. PMID: 12095831.
11. Leveque TK (2021). 'Pediatric Posterior and Panuveitis', American Academy of Ophthalmology Website. Available at: https://www.aao.org/education/disease-review/pediatric-posterior-panuveitis (Accessed: March 30, 2024).
12. Chen A, Dang S, Chung MM, Ramchandran RS, Bessette AP, DiLoreto DA, Kleinman DM, Sridhar J, Wykoff CC, Kuriyan AE. Quantitative Comparison of Fundus Images by 2 Ultra-Widefield Fundus Cameras. Ophthalmol Retina. 2021 May;5(5):450-457. doi: 10.1016/j.oret.2020.08.017. Epub 2020 Aug 29. PMID: 32866664; PMCID: PMC8501635.
13. Pinelli E, Aranzamendi C. Toxocara infection and its association with allergic manifestations. Endocr Metab Immune Disord Drug Targets. 2012 Mar;12(1):33-44. doi: 10.2174/187153012799278956. PMID: 22214330.
14. Jones JL, Kruszon-Moran D, Elder S, Rivera HN, Press C, Montoya JG, McQuillan GM. Toxoplasma gondii Infection in the United States, 2011-2014. Am J Trop Med Hyg. 2018 Feb;98(2):551-557. doi: 10.4269/ajtmh.17-0677. Epub 2017 Dec 14. Erratum in: Am J Trop Med Hyg. 2018 Jul;99(1):241-242. doi: 10.4269/ajtmh.17-0677err. PMID: 29260660; PMCID: PMC5929212.
15. Mazur-Melewska K, Mania A, Figlerowicz M, Kemnitz P, Służewski W, Michalak M. The influence of age on a clinical presentation of Toxocara spp. infection in children. Ann Agric Environ Med. 2012;19(2):233-6. PMID: 22742793.
16. de Visser L, Rothova A, de Boer JH, van Loon AM, Kerkhoff FT, Canninga-van Dijk MR, Weersink AY, de Groot-Mijnes JD. Diagnosis of ocular toxocariasis by establishing intraocular antibody production. Am J Ophthalmol. 2008 Feb;145(2):369-74. doi: 10.1016/j.ajo.2007.09.020. Epub 2007 Dec 3. PMID: 18061138.
17. Masoumi Asl H Md Mph, Alborzi A Md, Pourabbas B PhD, Kalani M MSc. QuantiFERON-TB Gold and Tuberculin Skin Test for the Diagnosis of Latent Tuberculosis Infection in Children. Iran J Med Sci. 2015 Sep;40(5):411-7. PMID: 26379347; PMCID: PMC4567600.
18. Debulpaep S, Corbière V, Levy J, Schelstraete P, Vanden Driessche K, Mascart F, Mouchet F. Contribution of QuantiFERON-TB Gold-in-Tube to the Diagnosis of Mycobacterium tuberculosis Infection in Young Children in a Low TB Prevalence Country. Front Pediatr. 2019 Jul 18;7:291. doi: 10.3389/fped.2019.00291. PMID: 31380325; PMCID: PMC6657736.
19. Schallhorn JM, Gonzales J. Ocular toxoplasmosis: the treatment dilemma. J AAPOS. 2013 Oct;17(5):454-5. doi: 10.1016/j.jaapos.2013.08.001. PMID: 24160961.
20. Feliciano-Alfonso JE, Muñoz-Ortiz J, Marín-Noriega MA, Vargas-Villanueva A, Triviño-Blanco L, Carvajal-Saiz N, de-la-Torre A. Safety and efficacy of different antibiotic regimens in patients with ocular toxoplasmosis: systematic review and meta-analysis. Syst Rev. 2021 Jul 19;10(1):206. doi: 10.1186/s13643-021-01758-7. PMID: 34275483; PMCID: PMC8287816.
21. Kishore K, Conway MD, Peyman GA. Intravitreal clindamycin and dexamethasone for toxoplasmic retinochoroiditis. Ophthalmic Surg Lasers. 2001 May-Jun;32(3):183-92. PMID: 11371084.
22. Kalogeropoulos D, Sakkas H, Mohammed B, Vartholomatos G, Malamos K, Sreekantam S, Kanavaros P, Kalogeropoulos C. Ocular toxoplasmosis: a review of the current diagnostic and therapeutic approaches. Int Ophthalmol. 2022 Jan;42(1):295-321. doi: 10.1007/s10792-021-01994-9. Epub 2021 Aug 9. PMID: 34370174; PMCID: PMC8351587.
23. Robert-Gangneux F, Guegan H. Anti-Toxoplasma IgG assays: What performances for what purpose? A systematic review. Parasite. 2021;28:39. doi: 10.1051/parasite/2021035. Epub 2021 Apr 26. PMID: 33904818; PMCID: PMC8078101.
24. Schoenardie ER, Scaini CJ, Brod CS, Pepe MS, Villela MM, McBride AJ, Borsuk S, Berne ME. Seroprevalence of Toxocara infection in children from southern Brazil. J Parasitol. 2013 Jun;99(3):537-9. doi: 10.1645/GE-3182. PMID: 23738711.
25. Mendonça LR, Figueiredo CA, Esquivel R, Fiaccone RL, Pontes-de-Carvalho L, Cooper P, Barreto ML, Alcantara-Neves NM. Seroprevalence and risk factors for Toxocara infection in children from an urban large setting in Northeast Brazil. Acta Trop. 2013 Oct;128(1):90-5. doi: 10.1016/j.actatropica.2013.06.018. Epub 2013 Jul 8. PMID: 23845771; PMCID: PMC7612831.
26. Magnaval JF, Bouhsira E, Fillaux J. Therapy and Prevention for Human Toxocariasis. Microorganisms. 2022 Jan 22;10(2):241. doi: 10.3390/microorganisms10020241. PMID: 35208697; PMCID: PMC8875715.
27. Xie Y, Sun L, Chen Y, Zhou X, Zhang Z, Ding X. Ocular toxocariasis presenting as leukocoria. Lancet Infect Dis. 2022 Mar;22(3):426. doi: 10.1016/S1473-3099(21)00704-0. PMID: 35218757.
28. Lin S, Han X, Dai R. Recurrent vitreous opacity caused by intraocular Toxocara larva: a case report and literature review. BMC Ophthalmol. 2022 Dec 22;22(1):505. doi: 10.1186/s12886-022-02687-2. PMID: 36550446; PMCID: PMC9773548.
29. Wakabayashi T, Kamuro R, Hashida N, Shiraki N, Sakaguchi H, Ohguro N, Nishida K. Acute endophthalmitis and hyphema mimicking pink hypopyon associated with ocular toxocariasis: A case report. Am J Ophthalmol Case Rep. 2021 Aug 10;23:101188. doi: 10.1016/j.ajoc.2021.101188. PMID: 34430757; PMCID: PMC8365312.
30. Inagaki K, Kirmse B, Bradbury RS, Moorthy RS, Arguello I, McGuffey CD, Tieu B, Hobbs CV. Case Report: Ocular Toxocariasis: A Report of Three Cases from the Mississippi Delta. Am J Trop Med Hyg. 2019 May;100(5):1223-1226. doi: 10.4269/ajtmh.18-0766. PMID: 30860012; PMCID: PMC6493957.
31. Fonseca C, Silva AM, Freire S, Proença R. Ocular toxocariasis: atypical clinical course. BMJ Case Rep. 2019 Apr 4;12(4):e228717. doi: 10.1136/bcr-2018-228717. PMID: 30948413; PMCID: PMC6453420.
32. Karaca I, Menteş J, Nalçacı S. Toxocara Neuroretinitis Associated with Raw Meat Consumption. Turk J Ophthalmol. 2018 Oct;48(5):258-261. doi: 10.4274/tjo.27085. Epub 2018 Oct 31. PMID: 30405949; PMCID: PMC6216536.

33. Sahu ES, Pal B, Sharma T, Biswas J. Clinical Profile, Treatment, and Visual Outcome of Ocular Toxocara in a Tertiary Eye Care Centre. Ocul Immunol Inflamm. 2018;26(5):753-759. doi: 10.1080/09273948.2016.1249375. Epub 2016 Nov 30. PMID: 27901644.
34. Iddawela D, Ehambaram K, Bandara P. Prevalence of Toxocara antibodies among patients clinically suspected to have ocular toxocariasis: A retrospective descriptive study in Sri Lanka. BMC Ophthalmol. 2017 Apr 24;17(1):50. doi: 10.1186/s12886-017-0444-0. PMID: 28438141; PMCID: PMC5404299.
35. Ahn SJ, Woo SJ, Jin Y, Chang YS, Kim TW, Ahn J, Heo JW, Yu HG, Chung H, Park KH, Hong ST. Clinical features and course of ocular toxocariasis in adults. PLoS Negl Trop Dis. 2014 Jun 12;8(6):e2938. doi: 10.1371/journal.pntd.0002938. PMID: 24922534; PMCID: PMC4055477.
36. Stewart JM, Cubillan LD, Cunningham ET Jr. Prevalence, clinical features, and causes of vision loss among patients with ocular toxocariasis. Retina. 2005 Dec;25(8):1005-13. doi: 10.1097/00006982-200512000-00009. PMID: 16340531.
37. Logar J, Soba B, Kraut A, Stirn-Kranjc B. Seroprevalence of Toxocara antibodies among patients suspected of ocular toxocariasis in Slovenia. Korean J Parasitol. 2004 Sep;42(3):137-40. doi: 10.3347/kjp.2004.42.3.137. PMID: 15381862; PMCID: PMC2717364.
38. Despommier D. Toxocariasis: clinical aspects, epidemiology, medical ecology, and molecular aspects. Clin Microbiol Rev. 2003 Apr;16(2):265-72. doi: 10.1128/CMR.16.2.265-272.2003. PMID: 12692098; PMCID: PMC153144.
39. Ebrahimiadib N, Fadakar K, Hedayatfar A, Soheilian M, Hosseini SM, Zarei M. Expanded Spectrum of Optical Coherence Tomography Findings in Patients with Ocular Toxoplasmosis. Ocul Immunol Inflamm. 2022 Apr 3;30(3):533-540. doi: 10.1080/09273948.2021.1933065. Epub 2021 Jul 8. PMID: 34236280.
40. Garweg JG, de Groot-Mijnes JD, Montoya JG. Diagnostic approach to ocular toxoplasmosis. Ocul Immunol Inflamm. 2011 Aug;19(4):255-61. doi: 10.3109/09273948.2011.595872. Erratum in: Ocul Immunol Inflamm. 2011 Dec;19(6):456. PMID: 21770803; PMCID: PMC3154545.
41. Liu EW, Elder ES, Rivera HN, Kruszon-Moran D, Handali S, Jones JL. Concurrent Seroprevalence of Antibodies to Toxoplasma gondii and Toxocara Species in the United States, 2011-2014. Clin Infect Dis. 2019 Feb 1;68(4):712-713. doi: 10.1093/cid/ciy729. PMID: 30137240; PMCID: PMC10804233.
42. Cabral Monica T, Evers F, de Souza Lima Nino B, Pinto-Ferreira F, Breganó JW, Ragassi Urbano M, Rubinsky-Elefant G, Freire RL, Navarro IT, Mitsuka-Breganó R. Socioeconomic factors associated with infection by Toxoplasma gondii and Toxocara canis in children. Transbound Emerg Dis. 2022 May;69(3):1589-1595. doi: 10.1111/tbed.14129. Epub 2021 May 14. PMID: 33908184.
43. Araújo AC, Villela MM, Sena-Lopes Â, Farias NADR, Faria LMJ, Avila LFDC, Berne MEA, Borsuk S. Seroprevalence of Toxoplasma gondii and Toxocara canis in a human rural population of Southern Rio Grande do Sul. Rev Inst Med Trop Sao Paulo. 2018;60:e28. doi: 10.1590/s1678-9946201860028. Epub 2018 Jun 28. PMID: 29972461; PMCID: PMC6029849.
44. Raissi V, Taghipour A, Navi Z, Etemadi S, Sohrabi Z, Sohrabi N, Getso M, Shamsaei S, Fasihi Karami M, Raiesi O. Seroprevalence of Toxoplasma gondii and Toxocara spp. infections among pregnant women with and without previous abortions in the west of Iran. J Obstet Gynaecol Res. 2020 Mar;46(3):382-388. doi: 10.1111/jog.14184. Epub 2020 Jan 17. Erratum in: J Obstet Gynaecol Res. 2022 May;48(5):1287. doi: 10.1111/jog.15260. PMID: 31953906.
45. Jones JL, Kruszon-Moran D, Won K, Wilson M, Schantz PM. Toxoplasma gondii and Toxocara spp. co-infection. Am J Trop Med Hyg. 2008 Jan;78(1):35-9. PMID: 18187782.
46. Rym BA, Yasmine K, Hedia B, Nesrine I, Olfa S, Rania M, Karim A, Aïda B. Contribution of the Toxoplasma ICT IgG IgM® test in determining the immune status of pregnant women against toxoplasmosis. J Clin Lab Anal. 2021 May;35(5):e23749. doi: 10.1002/jcla.23749. Epub 2021 Mar 15. PMID: 33720427; PMCID: PMC8128306.
47. Bin Dajem SM, Almushait MA. Detection of Toxoplasma gondii DNA by PCR in blood samples collected from pregnant Saudi women from the Aseer region, Saudi Arabia. Ann Saudi Med. 2012 Sep-Oct;32(5):507-12. doi: 10.5144/0256-4947.2012.14.7.1200. PMID: 22796740; PMCID: PMC6080984.
48. Reischl U, Bretagne S, Krüger D, Ernault P, Costa JM. Comparison of two DNA targets for the diagnosis of Toxoplasmosis by real-time PCR using fluorescence resonance energy transfer hybridization probes. BMC Infect Dis. 2003 May 2;3:7. doi: 10.1186/1471-2334-3-7. PMID: 12729464; PMCID: PMC156600.
49. Fekkar A, Bodaghi B, Touafek F, Le Hoang P, Mazier D, Paris L. Comparison of immunoblotting, calculation of the Goldmann-Witmer coefficient, and real-time PCR using aqueous humor samples for diagnosis of ocular toxoplasmosis. J Clin Microbiol. 2008 Jun;46(6):1965-7. doi: 10.1128/JCM.01900-07. Epub 2008 Apr 9. PMID: 18400917; PMCID: PMC2446865.
50. De Groot-Mijnes JD, Rothova A, Van Loon AM, Schuller M, Ten Dam-Van Loon NH, De Boer JH, Schuurman R, Weersink AJ. Polymerase chain reaction and Goldmann-Witmer coefficient analysis are complimentary for the diagnosis of infectious uveitis. Am J Ophthalmol. 2006 Feb;141(2):313-8. doi: 10.1016/j.ajo.2005.09.017. PMID: 16458686.
51. Petersen E, Borobio MV, Guy E, Liesenfeld O, Meroni V, Naessens A, Spranzi E, Thulliez P. European multicenter study of the LIAISON automated diagnostic system for determination of Toxoplasma gondii-specific immunoglobulin G (IgG) and IgM and the IgG avidity index. J Clin Microbiol. 2005 Apr;43(4):1570-4. doi: 10.1128/JCM.43.4.1570-1574.2005. PMID: 15814967; PMCID: PMC1081322.
52. Wilson M, Remington JS, Clavet C, Varney G, Press C, Ware D. Evaluation of six commercial kits for detection of human immunoglobulin M antibodies to Toxoplasma gondii. The FDA Toxoplasmosis Ad Hoc Working Group. J Clin Microbiol. 1997 Dec;35(12):3112-5. doi: 10.1128/jcm.35.12.3112-3115.1997. PMID: 9399504; PMCID: PMC230132.
53. Baker Cj (2017). 'Red Book Atlas of Pediatric Infectious Diseases', third edition (pp. 634-636). Published by the American Academy of Pediatrics. ISBN: 978-1-61002-060-2, eBook: 978-1-61002-061-9.
54. Pivetti-Pezzi P. Ocular toxocariasis. Int J Med Sci. 2009;6(3):129-30. doi: 10.7150/ijms.6.129. Epub 2009 Mar 19. PMID: 19319231; PMCID: PMC2659485.
55. Yoon DY, Woo SJ. Intravitreal Administration of Ranibizumab and Bevacizumab for Choroidal Neovascularization Secondary to Ocular Toxocariasis: A Case Report. Ocul Immunol Inflamm. 2018;26(4):639-641. doi: 10.1080/09273948.2016.1239744. Epub 2016 Oct 24. PMID: 27775459.