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Case report

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## Two case reports of toxocariasis mimicking endophthalmitis in immunosuppressed adults

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### ABSTRACT

This a case of 2 immunocompromised patients with a surprising ocular toxocariasis infestation. The first is a 23-year-old girl who is a known case of systemic lupus erythematosus, presenting with both eyes progressive blurring of vision for the past 3 months. Examinations showed bilateral panuveitis with posterior pole granuloma. She was treated as endogenous endophthalmitis as per protocol, but with limited improvement. To our surprise and with high index of clinical suspicion, *Toxocara* serology was positive. She was then started on oral albendazole for 4 weeks. The second case is a 51-year-old lady with end stage renal failure undergoing regular dialysis and on immunosuppressive therapy. She was warded for long duration for sepsis secondary to candidal fungal infection. Examinations revealed right peripheral and posterior pole granulomas almost exactly similar to the first patient. She was treated for endogenous endophthalmitis and again in close succession a positive serology was confirmed. Both cases since then showed gradual clinical improvement.

## 1. Introduction

Immunocompromised individuals are at high risk of opportunistic infections to ocular structures. A number of organisms are capable of causing infection in these patients. Albeit parasites such as *Toxocara*, have been rarely reported.

This case report pertained to 2 patients seen in our hospital. The first was a 23-year-old female with systemic lupus erythematosus. The second is a 51-year-old female who underwent renal transplantation for end stage renal failure. Both of the patients were being treated with high dose oral steroids for a prolonged period of time. Subsequently, they were found to develop *Toxocara* ocular infection which was confirmed serologically.

This case report is being presented for the rarity of such case reports in published literature. This article also emphasized the importance of public education and awareness of hygiene among immunocompromised individuals in *Toxocara* endemic areas. These measures may help to reduce the infection of these parasites which can lead to irreversible progression of the disease. Intensive anti-parasitic treatment should also be instituted to prevent the reactivation of these larvae in tissues[1].

## 2. Case report

### 2.1. Case 1

A 23-year-old female suffering from systemic lupus erythematosus complicated with lupus nephritis had been on long term steroid therapy. She presented to our ophthalmology clinic with slowly progressive, painless blurring of vision in both eyes for the past 3 weeks. There was also a history of interaction with various animals, including cats and dogs living near her home.

On presentation, her visual acuity was counting fingers in both eyes, with no improvement with pinhole. Her intraocular pressures were 14 mmHg bilaterally. Ocular examination revealed +2 cells in the anterior chamber and +3 cells in the vitreous in both eyes. Fundus examination showed the presence of granulomas in the posterior pole in both eyes. A peripheral granuloma with a band of fibrosis running from the disc to the superior periphery in the right eye developed a few days after initial presentation (Figure 1).

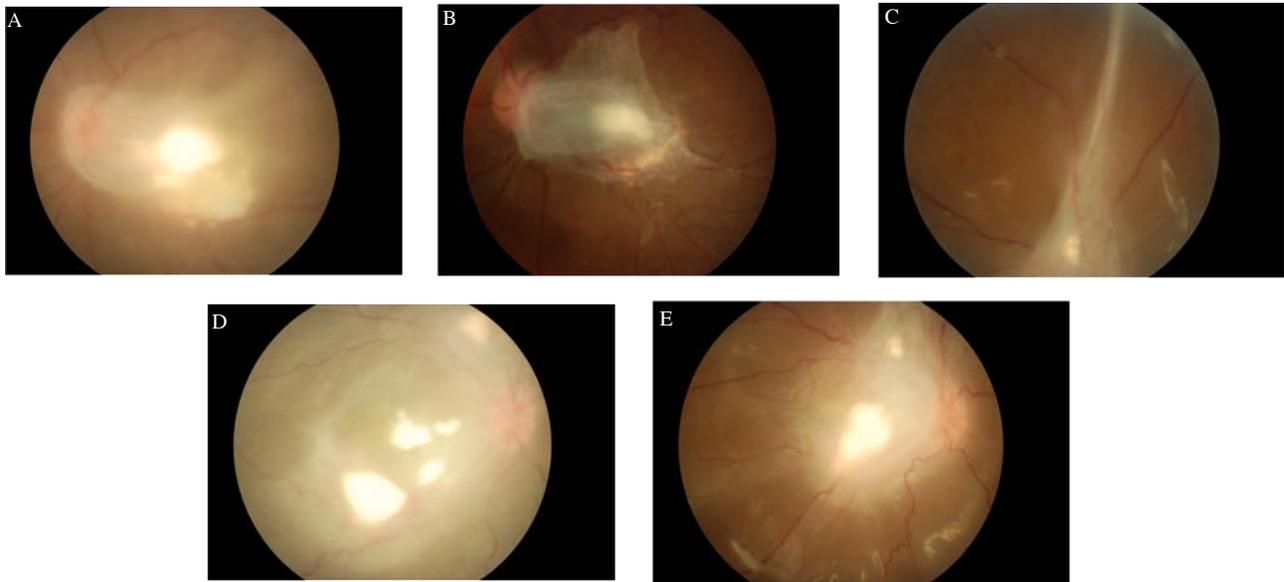
A full blood count, including eosinophil count was normal. Serological testing was subsequently conducted to identify the underlying cause for the condition and included analysis for toxoplasmosis, *Toxocara*, syphilis, sarcoidosis, tuberculosis, rheumatoid factor, antinuclear antibody, anti-neutrophil cytoplasmic antibodies, and Lyme disease. Serological *Toxocara* testing was performed using ELISA technique and the result came back positive. The other tests were negative. Vitreous taps of both eyes for cultures and cytology were negative. Fundus fluorescein angiography showed leakage from vessels extensively.

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**Figure 1.** Fundus images (Case 1).

A: Left eye posterior pole on presentation; B: Left eye posterior pole post treatment; C: Right eye periphery fibrous band on presentation; D: Right eye posterior pole on presentation; E: Right eye posterior pole post treatment.

Plain film chest X-ray showed no lymphadenopathy to suggest sarcoidosis and no signs of pulmonary tuberculosis. A CT scan of orbits and brain reported an old lacunar infarct, with no evidence of dural sinus thrombosis or space occupying lesions.

The patient was being treated with a number of medications for many years. They included tablet prednisolone 35 mg daily, tablet hydroxychloroquine 300 mg daily, tablet calcium carbonate 500 mg twice daily, tablet esomeprazole 40 mg daily, tablet mecobalamin 500 mg twice daily and tablet sertraline 25 mg daily.

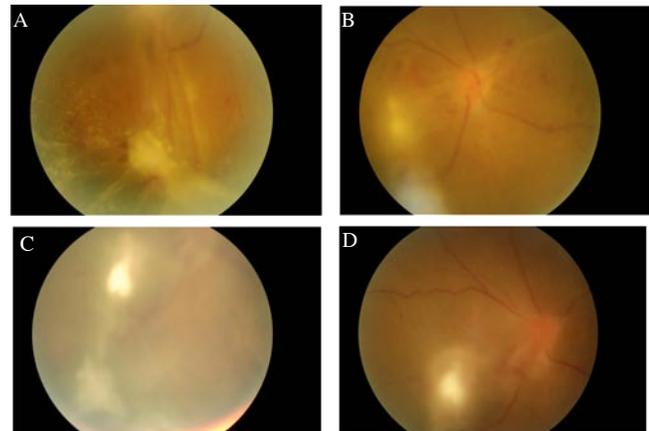
After evaluation in the clinic, she was admitted and treated for bilateral endogenous endophthalmitis. Initially, she was suspected of having fungal infection. Thus, she was given intravitreal amphotericin B, vancomycin, and ceftazidime in both eyes after vitreous samples were taken. She was also started on IV ciprofloxacin 400 mg twice daily, tablet fluconazole 200 mg twice daily, topical steroid-antibiotic eyedrops 2 hourly and homatropine 2% eyedrops three times per day. Subsequently, on receiving the serological test results, the antibiotics and antifungals were tapered off and she was given tablet albendazole 400 mg twice daily for *Toxocara* eradication. The patient's visual acuity improved slightly to 1/60 in both eyes as the posterior pole granulomas progressed to scarring.

## 2.2. Case 2

A 51-year-old lady underwent a renal transplantation for end stage renal failure. Subsequently, she developed chronic candidal sepsis secondary to an infected drain site. The patient was referred to our department with complaints of progressively worsening vision of the right eye associated with pain and redness. Ophthalmological examination showed a large posterior pole granuloma with traction bands in between multiple peripheral granulomas inferiorly (Figure 2).

Bone marrow aspirate culture and blood culture were performed in the general medicine department. Both grew candida. Hence, she was treated for fungal endophthalmitis in view of her history and investigative findings. A vitreous biopsy was taken for cultures and she was given intravitreal amphotericin, ceftazidime and vancomycin. Blood and serological investigations were taken as in the first case. There was no peripheral eosinophilia, and the vitreous cultures were negative. However, the blood serology was positive for

*Toxocara*. She was started on antihelminthic treatment along with the systemic antifungal and antibacterial treatment that she was already receiving. Clinical improvement was attained, as the granulomas became progressively well defined and became scars. The pain and redness subsided with initiation of treatment. However the final visual acuity of the patient did not improve much due to the macular scar formation.



**Figure 2.** Fundus images (Case 2).

A: Right eye post treatment-periphery; B: Right eye post treatment-posterior pole; C: Right eye on presentation-periphery; D: Right eye on presentation-posterior pole.

## 3. Discussion

Infectious uveitis in the immunocompromised individuals is rapidly progressive and potentially blinding. It is necessary to identify the organism and start the correct treatment in order to prevent recurrences and involvement of the unaffected eye in this vulnerable population. While a large number of organisms like cytomegalovirus, *Toxoplasma* spp. and fungi have been reported as the common etiologic agents, yet opportunistic infection with *Toxocara* spp. is seldom reported[1].

Toxocariasis refers to human infection due to parasitic roundworms. Humans are paratenic hosts, who are infected accidentally by ingestion of invasive eggs. Toxocariasis results from

human infection with the larvae of the dog ascarid, *Toxocara canis*; the cat ascarid, *Toxocara cati*; and rarely the pig ascarid, *Ascaris suum* or some new species being reported recently. The clinical presentation of this infection can vary from visceral larva migrans to ocular infection or even occult or covert toxocariasis. Ocular larva migrans (OLM) usually occurs unilaterally in children and young adults. The commonest presentation is reduction of vision occurring over a period of days to weeks and is usually observed in older children below the age of sixteen[2,3]. Another article mentions the average age of presentation as 7.5 years (ranging from 2–31 years) [4]. Our second case is unique in having infection at an older age.

Once ingested, circulating larval antigens of *Toxocara* species stimulate Th0 lymphocytes to develop into active Th2 cells, which initiate the synthesis of interleukin (IL)-4 and IL-5, responsible for the production of anti-parasitic immunoglobulin E antibodies by plasma cells and accelerated maturation of eosinophils[3]. The ability of the larvae to survive and migrate within the host's tissues for months or even years provokes a stable stimulation of Th2 lymphocytes and a persistent production of immunoglobulin E for a long period of time[5].

OLM syndrome is a localized manifestation of a *Toxocara* eye infection, usually caused by a single second-stage larva[5]. Despite the low intensity of invasion and unilateral location of the migrating parasite, infection may cause severe inflammation and progressive ocular damage, leading to retinal detachment, cataract formation, endophthalmitis, strabismus and blindness[2,6].

OLM is commonly seen unilaterally. Among the common presentations include posterior pole granuloma, peripheral granuloma or *Toxocara* endophthalmitis[7]. In this regard, however, case 1 showed features of toxocariasis in both eyes. She had posterior pole granulomas in both eyes and a peripheral granuloma in the right eye.

The major determinants for vision loss in *Toxocara* infection are: (1) vitritis; (2) cystoid macular oedema; (3) tractional retinal detachment. The inflammation and granuloma formation is apparently due to the immunogenetic antigens in the dead larva. These granulomas drag the retina and create a distortion, heterotropia, or detachment of the macula[8]. OLM may lead to endophthalmitis, papillitis or secondary glaucoma as a sequelae[9]. Choroidal neovascular membrane formation has also been reported following chronic *Toxocara* infection[9].

Diagnosis of OLM is largely on the basis of ocular examination. The clinical features are usually characteristic enough to reach a reliable diagnosis. An ELISA test is available for diagnosis of *Toxocara*. However, the test is hampered by unusually low or absent antibodies in the serum[5]. In one study, the immunodiagnostic test had titers higher than 1:32 in only 45% of the patients diagnosed clinically with OLM[9]. An ELISA test with *Toxocara* excretory-secretory antigen is highly specific for *Toxocara* infection. Ocular toxocariasis is mainly a pediatric disease that serological screening is not informative for the diagnosis of intraocular *Toxocara* infection[10]. *Toxocara* Goldman-Witmer coefficient analysis, however, can be of value when diagnosing patients with posterior focal lesions or vitritis of unknown etiology[10]. Visceral larva migrans and "covert toxocariasis" often show eosinophilia which might be lacking in cases of OLM, making diagnosis difficult[5]. Thus, the immunodiagnosis of OLM can be accomplished by obtaining suitable aqueous or preferably, vitreous samples in order to perform the ELISA tests. In cases where the media is hazy, an ultrasound B-scan or CT scan may be of benefit to assess the posterior segment.

The treatment of ocular toxocariasis has to be customized according

to the clinical presentation. Patients with a significant inflammatory component need to be treated with steroids. Both periocular and systemic steroids (0.5–1.0 prednisolone mg/kg body weight/ per day) can be given. Inflammation leads to formation of membranes and can lead to complications such as tractional retinal detachment and dragging of the macula or optic disc[11]. Anti-helminthic agents have also been successfully employed to treat OLM. The agents used include thiabendazole (25 mg/kg twice daily for 5 days with a maximum of 3 g/day), albendazole (800 mg twice daily for 6 days), or mebendazole (100 to 200 mg twice daily for 5 days)[12]. In our patients also, there was a dramatic response to antihelminthics and steroids.

Surgery might be required in cases of inflammation induced complications such as retinal detachment, persistent vitreous opacification, and epiretinal membrane formation with vitreomacular or optic nerve traction[11]. Laser can also be used to kill live, mobile larvae seen in the retina[5]. Since our patients responded well to medical treatment despite being immunocompromised, surgery was not required.

In conclusion, it is rare to have *Toxocara* ocular infection in immunocompromised individuals. However, this opportunistic organism should be kept in the differential diagnosis of patients with posterior uveitis.

### Conflict of interest statement

We declare that we have no conflict of interest.

### References

- Westeneng AC, Rothova A, de Boer JH, de Groot-Mijnes JD. Infectious uveitis in immunocompromised patients and the diagnostic value of polymerase chain reaction and Goldmann-Witmer coefficient in aqueous analysis. *Am J Ophthalmol* 2007; **144**: 781-5.
- Magnaval JF, Glickman LT, Dorchie P, Morassin B. Highlights of human toxocariasis. *Korean J Parasitol* 2001; **39**: 1-11.
- Paul M, Stefaniak J, Twardosz-Pawlik H, Pecold K. The co-occurrence of *Toxocara* ocular and visceral larva migrans syndrome: a case series. *Cases J* 2009; **2**: 6881.
- Azira NMS, Zeehaida M. A case report of ocular toxocariasis. *Asian Pac J Trop Biomed* 2011; **1**: 164-5.
- Figueiredo SD, Taddei JA, Menezes JJ, Novo NF, Silva EO, Cristóvão HL, et al. [Clinical-epidemiological study of toxocariasis in a pediatric population]. *J Pediatr (Rio J)* 2005; **81**: 126-32. Portuguese.
- Holland CV, Smith HV. *Toxocara: the enigmatic parasite (Cabi)*. Wallingford: CABI Publishing; 2006, p. 127-44.
- Ahn SJ, Ryoo NK, Woo SJ. Ocular toxocariasis: clinical features, diagnosis, treatment, and prevention. *Asia Pac Allergy* 2014; **4**: 134-41.
- Small KW, McCuen BW 2nd, de Juan E Jr, Machermer R. Surgical management of retinal traction caused by toxocariasis. *Am J Ophthalmol* 1989; **108**: 10-4.
- Despommier D. Toxocariasis: clinical aspects, epidemiology, medical ecology, and molecular aspects. *Clin Microbiol Rev* 2013; **16**: 265-72.
- de Visser L, Rothova A, de Boer JH, van Loon AM, Kerkhoff FT, Canninga-van Dijk MR. Diagnosis of ocular toxocariasis by establishing intraocular antibody production. *Am J Ophthalmol* 2008; **145**: 369-74.
- Cortez RT, Ramirez G, Collet L, Giuliani GP. Ocular parasitic diseases: a review on toxocariasis and diffuse unilateral subacute neuroretinitis. *J Pediatr Ophthalmol Strabismus* 2011; **48**: 204-12.
- Stephanie L, Harper, Louis JC, Foster CS. Ocular toxocariasis. In: Foster CS, Vitale AT, editors. *Diagnosis and treatment of uveitis*. Philadelphia: WB Saunders; 2001, p. 428-36.