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Ocular toxocariasis: a neglected parasitic disease in Egypt

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Abstract

Background: Ocular toxocariasis is considered a parasitic disease of major socioeconomic importance. In spite of the high prevalence of human toxocariasis (up to 84%) among Egyptian patients, the incidence of ocular toxocariasis is underestimated. The recognition of this neglected disease would be the initial step to overcome it. Thus, this review gave updated information on the pathogenesis, clinical manifestations, diagnosis, and treatment of ocular toxocariasis.

Results: Ocular toxocariasis is an important cause of unilateral vision impairment mostly in children and always in the differential diagnosis of retinoblastoma. This disease exhibits various manifestations such as posterior pole granuloma, peripheral granuloma, or chronic endophthalmitis. Diagnosis of ocular toxocariasis can be carried out by the ophthalmic examination and immunodiagnostic methods to reveal the specific antibodies in serum and ocular fluids. In addition, molecular diagnosis, medical imaging techniques, and histopathologic observation of *Toxocara* larva in the surgically obtained specimens can be performed. Ocular toxocariasis can be treated either medically or surgically. Regarding medical treatment, the ophthalmologists prefer to use steroids and anthelminthic drugs; however, there are no standardized parameters for doses, duration, and route of administration.

Conclusion: Clinical suspicion plays a leading role in the diagnosis of ocular toxocariasis, but always with other diagnostic methods. Accurate diagnosis and prompt treatment can minimize ocular morbidity.

Keywords: Ocular toxocariasis, Pathogenesis, Clinical manifestations, Diagnosis, Treatment, Egyptian studies

Introduction

Ocular parasitic infections cause a significant ocular morbidity not because they are non-treatable, but mostly due to delay or misdiagnosis, frequently from unawareness of the resulting diseases. The parasites attain the eye by means of direct invasion through trauma or surgery, via extension from neighboring infected tissues, or via hematogenous dissemination to the eye. Ocular lesions may be due to direct damage caused by the parasites or indirect pathology caused by toxic products of parasites and also may be due to the immune response to infectious parasitism. Variation in the disease spectrum depends on the geographical location, the hygienic conditions, the living and eating habits of the individuals, and the contact with animals (El-Sayed and Safar 2015).

Toxocariasis is a parasitic infection caused by invading the tissues by larval stages of *Toxocara canis* or *Toxocara cati*. The adult worms of these nematodes parasitize the small intestines of dogs and cats, respectively (Fillaux and Magnaval 2013). The human disease occurs by ingesting the viable *Toxocara* embryonated eggs, commonly from contaminated raw vegetables or from polluted water sources as well as from contact with domestic dogs and cats, especially puppies and kittens, which harbor eggs in their fur (Holland 2017).

According to *Toxocara* larvae migration through tissues, human toxocariasis is classified into visceral, cerebral, ocular, and covert toxocariasis (El-Sayed and Ramadan 2017). Ocular toxocariasis is an important cause of posterior and diffuse uveitis and constantly in the differential diagnosis of retinoblastoma (Cortez et al. 2011). It is more frequent in children with an age group that ranges from 3 to12 years (Fomda et al. 2007; Azira and Zeehaida, 2011; Zibaei et al. 2014). The adult patients were also affected by ocular toxocariasis mostly in Asian populations where eating of raw

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meat is usual (Jee et al. 2016). In 90% of cases, ocular toxocariasis is unilateral (Rubinsky-Elefant et al. 2010). It has been estimated that ocular toxocariasis results in 5 to 20% of blindness secondary to uveitis (Arevalo et al. 2013).

In Egypt, toxocariasis occurs widely in the poor communities with low standard of hygiene and sanitation. Both stray and domestic dogs and cats play a pivotal role in the transmission of *Toxocara* species providing environmental contamination, which perpetuates the spreading of the infection among Egyptian populations. It was estimated that soil contamination with *Toxocara* eggs is up to 30% (Farghly et al. 2016). Several studies were conducted to determine the prevalence of human toxocariasis among the Egyptian population with a significantly high proportion of children because of their playing habits and hygiene standards. The seroprevalence of anti-*Toxocara* antibodies was 10.7% among children with renal troubles (Nada et al. 1996), 6% among children with hepatomegaly in Zagazig City (Hassan et al. 1996), 6.2% among children with respiratory symptoms or pyrexia of unknown origin, 18% among adults with pyrexia of unknown origin in Tanta City (Antonios et al. 2008), 23.3% among adult schizophrenic patients (El-Sayed and Ismail 2012), 84% among asthmatic children in Mansoura City (El-Tantawy et al. 2013a), and 48.5% in children with cryptogenic epilepsy (El-Tantawy et al. 2013b). In spite of the high prevalence of human toxocariasis (up to 84%) among Egyptian patients, the incidence of ocular toxocariasis is undetermined. The potential under-reporting of ocular toxocariasis is related to the wide variation in its clinical manifestations and the complexity in its diagnosis which necessitates clinical, radiological, and immunodiagnosis, many of which are not available for routine diagnostic service for suspected patients. Also, there are some difficulties in the serological diagnosis concerning *Toxocara* antigen production or the highly expensive commercial diagnostic kits which are utilized only for research studies. The consequence of ocular toxocariasis and the potential under-reporting cases, based on seroprevalence studies, make ocular toxocariasis worthy of more medical awareness (Hare and Franco-Paredes 2014). This review discussed the pathogenesis, clinical manifestations, diagnosis, and treatment of ocular toxocariasis. The recognition of this parasitic disease would be the initial step to overcome this neglected infection.

Pathogenesis of ocular toxocariasis

Ocular toxocariasis is caused by the migration of *Toxocara* larvae into the posterior segment of the eye. Based on studies executed in animal models, the migration route of *Toxocara* larvae may follow several pathways. The most common one is via the brain to the cerebrospinal fluid and then to the choroid. The other possible pathways are via the brain to the optic nerve or via the arteries from the internal carotid artery to the ophthalmic artery, retinal central artery, or ciliary artery (Hayashi et al. 2003). Migrated larvae secrete enzymes, waste products, and cuticular components, which cause tissue damage, necrosis, and marked inflammatory reaction, with eosinophils as the most component. These eosinophils release toxic proteins which contribute to the pathology and symptomatology of toxocariasis. The typical pathological finding in ocular toxocariasis is eosinophilic granulomas with central necrosis and remnants of *Toxocara* larva surrounded by a mixed inflammatory infiltrate with numerous eosinophils (Rubinsky-Elefant et al. 2010; Fillaux and Magnaval 2013; Das et al. 2016).

Ocular toxocariasis is usually due to a single *Toxocara* larva invading the eye. This results in a low-level immune response which is insufficiently activated to kill the larva and allows its persistence in the eye for years. During this long period, it can migrate through ocular tissues causing mechanical and immunopathological damage (Magnaval et al. 2001). Thus, the pathogenesis of ocular toxocariasis in the human depends on the inflammatory reactions activated by the existence of larva in the eye, the host immune response, the number of the larvae present in the eye, the incidence of reinfection, and the host sensitization to the secreted or excreted products by the larvae (Fan et al. 2013).

Clinical manifestations of ocular toxocariasis

Clinical manifestations and severity of ocular toxocariasis rely on the primary anatomical site implicated, the number of the larvae existing in the eye, and the immune reaction of the host (Azira and Zeehaida, 2011). The most common symptoms of ocular toxocariasis include photophobia, floaters, leukocoria, strabismus, white pupillary reflex, bloodshot conjunctiva, mild ocular pain, vitreous inflammation, and blindness of one eye which is recorded in about 80% of cases and is permanent in the most patients. Occasionally, the retinal symptoms may be associated with those due to involvement of the central nervous system. These symptoms range for about 12 months. In young cases, the eye infection may not be observed until they fail a school vision screening test (Rubinsky-Elefant et al. 2010; Azira and Zeehaida 2011).

Ocular toxocariasis exhibits various manifestations such as posterior pole granuloma, peripheral granuloma, or chronic endophthalmitis, and occasionally, it may present with atypical manifestations.

Posterior pole granuloma

It represents 25–50% of cases in ages between 4 and 14 years old. Posterior pole granuloma appears as an oval, white lesion in the posterior pole of the retina (Fig. 1). The cause of this predilection to the posterior pole has been proposed by the hematogenous spread of larvae that may lodge in small, perifoveal end arteries. In the acute stage, *Toxocara* retinochoroiditis manifests clinically as a hazy, ill-defined white lesion with overlaying

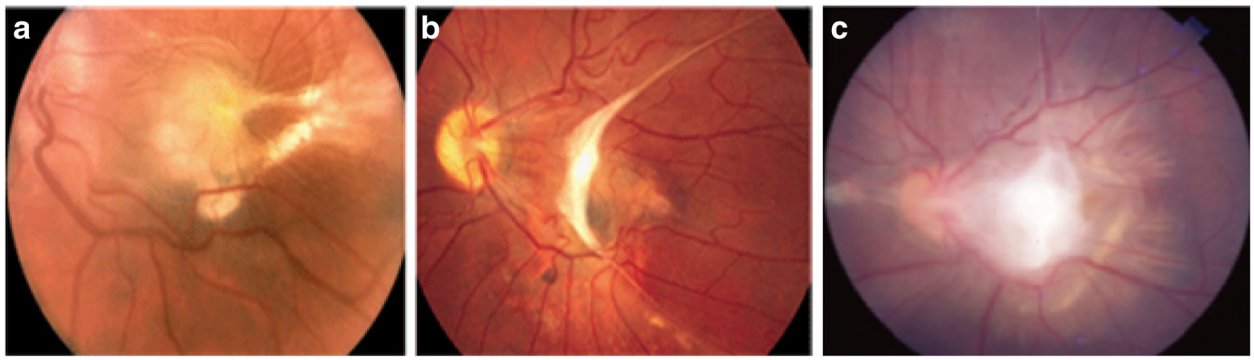

Fig. 1 Posterior pole granuloma due to ocular toxocariasis with prominent retinal folds (**a**), epiretinal membrane formation (**b**) (Singh et al. 2007), and a traction band extending from the lesion to the optic disc and retinochoroidal anastomosis (**c**) (Cortez et al. 2011)

inflammatory cells in the vitreous. When the inflammatory reaction subsides, the lesion appears as a well-defined elevated mass ranging from one half to four disc diameters in size. A retinal pigment epithelium disturbance often surrounds the lesion with retinal folds extending from the lesion (Fig. 1a) and epiretinal membrane formation (Fig. 1b) (Singh et al. 2007; Ahn et al. 2014). Close examination may detect a dark gray center, which has been suggested to be remnants of the larva as well as retinal blood vessels entering the granuloma. In some cases, traction bands may extend from the lesion to the optic disc or to the macular area. In the case of chronic granulomatous inflammation, large retinal vessels may infiltrate the mass and disappear into its substance, probably representing retinochoroidal anastomosis (Fig. 1c) (Cortez et al. 2011).

The causes of vision loss may be by direct involvement of the macula or optic disc, by the secondary formation of retinal folds or epiretinal membranes, or rarely by the development of choroidal neovascularization (Singh et al. 2007; Cortez et al. 2011; Ahn et al. 2014).

Peripheral granuloma

Ocular toxocariasis may present as an acute inflammation in the peripheral retina and ciliary body. About 20–40% of the infected eyes with toxocariasis manifest as a peripheral granuloma. It is observed in cases with ages between 6 and40 years old. The peripheral granuloma presents as a hazy, white, elevated mass in the peripheral fundus. It can be associated with retinal folds that may extend from the peripheral mass to the optic nerve head or to other areas of the fundus. In some cases, the traction may lead to heterotopia of the macula (Fig. 2) (Cortez et al. 2011). Reduced vision in patients with peripheral ocular toxocariasis is due to macular involvement by posteriorly extending falciform folds or exudate. In addition, amblyopia may develop in young patients with media opacities and/or macular involvement (Singh et al. 2007).

Chronic endophthalmitis

Endophthalmitis is a panuveitis manifesting as a red, painful eye with diffused intraocular inflammation and no apparent nodular lesion. It is observed in 25% of cases, between 2 and 8 years old, presenting with leukocoria, strabismus, and hypopyon. In the fundus, there are granulomatous vitritis, cyclitic membranes, and retinal detachment (Fig. 3) (Cortez et al. 2011).

Toxocara endophthalmitis usually results from the prolonged and profound course of the inflammation secondary to the intraocular larva. It is associated with extensive vitritis (Fig. 4) and the formation of traction membranes in the retina and vitreous which lead to severe visual morbidity. When the inflammatory reaction subsides, vitreous membranes may transform into a retrolental mass, which in severe cases can induce ciliary body detachment, hypotony, and phthisis bulbi. Diminished vision in patients with *Toxocara* endophthalmitis results from inflammatory media opacities, cystoid macular edema, and/or cataract formation (Singh et al. 2007).

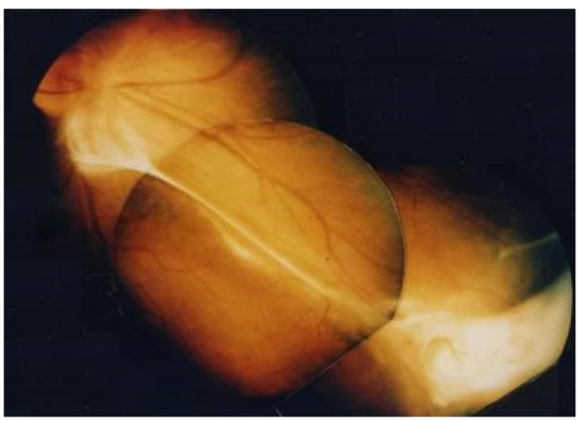

Fig. 2 Ocular toxocariasis with peripheral granuloma and vitreoretinal traction (Cortez et al. 2011)

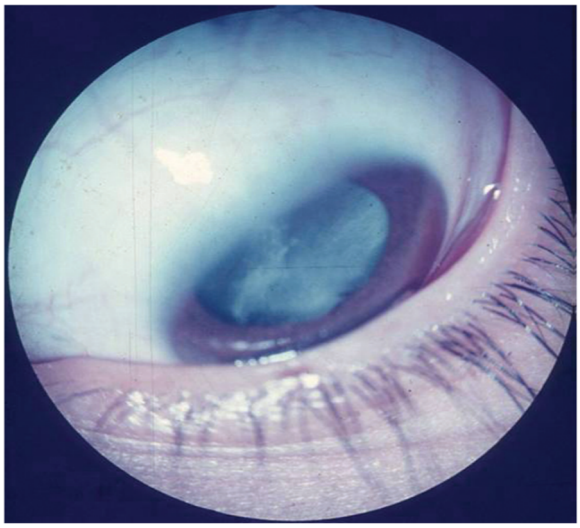

Fig. 3 Chronic toxocara endophthalmitis with partial retinal detachment (Cortez et al. 2011)

Atypical manifestations

Sometimes, ocular toxocariasis represents with atypical manifestations such as bullous retinal detachment (Tou et al. 2006), subacute unilateral neuroretinitis (Cortez et al. 2011), multifocal granuloma (Lakshmi 2013), cataract formation (Singh et al. 2007; Woodhall et al. 2012), and bilateral scleritis (Pak et al. 2016).

Diagnosis of ocular toxocariasis

Owing to the fact that *Toxocara* larva does not develop into the adult stage in humans, the parasitological examination of fecal samples is not beneficial for the laboratory diagnosis (Rubinsky-Elefant et al. 2010). Also, eosinophils detected in the aqueous or vitreous fluid are more direct indicators of ocular infection, but such fluids are rarely available for testing (Singh et al. 2007). Therefore, the diagnosis of ocular toxocariasis can be carried out by the conjunction of patients' history (contact with dogs or cats, geophagia, eating of undercooked or raw meats), distinguishing the typical manifestations by ophthalmic examination and immunodiagnostic methods to reveal the specific antibodies in serum and ocular fluids. In addition, molecular diagnosis, medical imaging techniques, and histopathologic observation of *Toxocara* larva in the surgically obtained specimens can be performed (Fillaux and Magnaval 2013; Ahn et al. 2014).

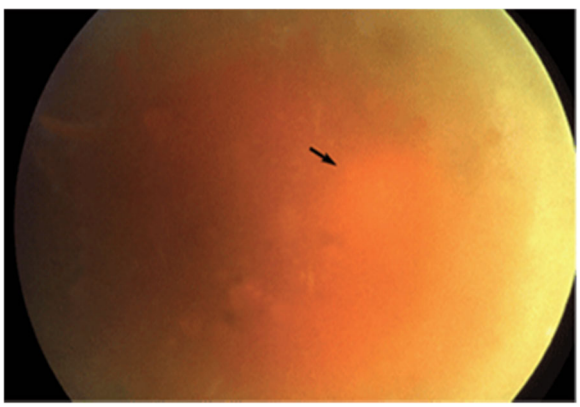

Fig. 4 Toxocara endophthalmitis with extensive vitritis obfuscates fundus details as the optic disc (arrow) is hardly visible (Singh et al. 2007)

Ophthalmic examination

Fundoscopic examination is a significant tool either for direct observation of a movable larva beneath the retina (Magnaval et al. 2001) or for recognition of typical features of ocular toxocariasis such as peripheral chorioretinal granuloma, posterior pole granuloma, endophthalmitis, and pars planitis which is an inflammation in the narrow area between the iris and the choroid (Singh et al. 2007; Cortez et al. 2011; Ahn et al. 2014). Clinical suspicion should be confirmed by the detection of *Toxocara* antibodies in serum and/or intraocular fluids using immunodiagnostic techniques (Magnaval et al. 2001).

Immunodiagnosis

Serodiagnosis

Several immunodiagnostic techniques have been used for the diagnosis of ocular toxocariasis, such as enzyme-linked immunosorbent assay (ELISA) (Fillaux and Magnaval 2013; Jin et al. 2013), Dot-ELISA test (Paller et al. 2017), and western blot (Magnaval et al. 2002; Jin et al. 2013). The antigens used in these immunoassays include somatic extracts of adult worms, embryonated eggs, intact or sectioned larvae, and *Toxocara* excretory-secretory antigen (TES). However, due to the complexity of TES antigen which includes specific and non-specific protein fractions, the assay using this antigen possesses low specificity as it cross-reacts with *Ascaris* and other helminths, especially in tropical areas where such parasites are endemic (Magnaval et al. 2001). Therefore, the development of high specific recombinant TES antigens may give supplemental solutions for serologic diagnosis by increasing sensitivity and specificity (Mohamad et al. 2009).

The selected test for routine diagnosis of toxocariasis is ELISA test which possesses both sensitivity and specificity ranging between 73–100% and 90–91%, respectively (Fillaux and Magnaval 2013; Jin et al. 2013). However, this assay has limitations, as cross-reactions with other helminths and the prolonged survival of larvae within the eosinophilic granuloma in tissues cause a chronic process and the existence of antibodies does not detect the parasitic activity, leading to difficulty in discrimination between recent

and chronic infection (Magnaval et al. 2001). Assays based on the detection of IgG avidity have the ability to distinguish between the active and chronic infection (Dziemian et al. 2008).

Currently, the best choice serodiagnostic methods are ELISA-IgG as a screening test and confirm the positive samples by western blot test. Immunoblot analysis is usually used in research for separation and identification of proteins. Western blot using crude antigen prepared from *Toxocara canis* larvae revealed antigenic proteins of typical seven bands (24, 28, 30, 35, 132, 147, 200 kDa). The high specificity of this assay is related to its ability to discriminate between high molecular weight bands (not specific and suggestive of cross-reactions with other helminths) and low molecular weight bands (24–35 kDa), which have a high level of specificity (Magnaval et al. 2002; Jin et al. 2013).

A remarkable issue when trying to evaluate serological tests for human toxocariasis is that there is no reference test or parasitological method to conclusively diagnose this parasitic disease. Many authors observed that *Toxocara* antibodies may be undetectable or the titer may be less than the cutoff level in the sera of several patients having clinical manifestations of ocular toxocariasis (Elefant et al. 2006), probably due to the presence of low parasite loads in them. Hence, the absence of serum antibodies does not exclude the diagnosis of ocular toxocariasis. In this condition, intraocular assay could be useful for confirming *Toxocara* infection diagnosis (De Visser et al. 2008).

Intraocular assay

Intraocular fluids include aqueous humor which is a clear liquid found in the space present between the cornea and the lens, and vitreous humor which is present in the space between the lens and the retina. Detection of specific *Toxocara* antibodies in aqueous and/or vitreous samples using different immunological methods assists the confirmation of ocular toxocariasis diagnosis (Fonseca et al. 2019) and distinguishes it from retinoblastoma (De Visser et al. 2008). Some researchers reported cases of ocular toxocariasis via the detection of specific anti-*Toxocara* IgG antibodies only in aqueous and/or vitreous samples and not in sera (Fomda et al. 2007). These findings underscore the hazards in diagnosing ocular toxocariasis based on the clinical and serological assays alone. Therefore, it is recommended to detect *Toxocara* antibodies via ELISA in both serum and aqueous/vitreous samples to increase the sensitivity of ocular toxocariasis diagnosis (De Visser et al. 2008).

Molecular diagnosis

Molecular-based methods for *Toxocara* detection in clinical and environmental samples have been described by several investigators (Fogt-Wyrwas et al. 2007; Tian and O'Hagan 2015). However, these methods are not widely available as *Toxocara* organisms do not replicate inside the human host and sequestration of *Toxocara* larvae within various tissues. Tian and O'Hagan (2015) reported a case of clinically suspected ocular toxocariasis with a negative result via serological test; however, the diagnosis was proven by using polymerase chain reaction (PCR) on ocular fluids. Although the detection of *Toxocara* deoxyribonucleic acid (DNA) is a very sensitive method, it may be negative in patients who have a very low larval burden, or if the larvae are sequestrated or destroyed within granulomas and did not shed DNA-containing tissues into the aqueous or vitreous humor (Schneier and Durand 2011). Subsequently, recombinant DNA and mitochondrial markers can offer new insight for the diagnosis of toxocariasis and may be helpful for epidemiological studies.

Diagnostic imaging techniques

Medical imaging techniques such as ocular ultrasound, computed tomography, optical coherence tomography, fluorescein angiography, and magnetic resonance imaging are used to detect granulomatous lesions related to *Toxocara* larva migration within the eye (Magnaval et al. 2001; Arevalo et al. 2013) and may be helpful in the differential diagnosis of ocular toxocariasis from other ocular diseases, particularly retinoblastoma.

Optical coherence tomography is a non-invasive imaging technique which uses coherent light to obtain cross-sectional images with high resolution. In ocular toxocariasis, posterior pole granuloma appears as a highly reflective mass above the retinal pigment epithelium layer. Also, optical coherence tomography can reveal the contributing factors which lead to vision loss, as the presence of intraretinal and subretinal fluids (Hashida et al. 2014).

B-scan ultrasonography uses the reflections of high-frequency sound waves to construct a two-dimensional, cross-sectional view of the tissue. In toxocariasis, ultrasonography can be utilized to reveal the optic disc granuloma, vitreous bands, retinal folds, and tractional retinal detachment, and also, it is useful in ruling out the presence of ocular tumor (Liu et al. 2017).

Histopathologic examination

In surgically treated cases, the definitive diagnosis of ocular toxocariasis is via the histopathological identification of *Toxocara* larva or its fragments in the vitrectomy specimens (Singh et al. 2007). Otherwise, the obtaining of biopsy materials from the eyes is difficult and risky. This method is a time-consuming, and occasionally, it is difficult to identify *Toxocara* larvae due to their small

size and their extensive distribution (Rubinsky-Elefant et al. 2010; Fillaux and Magnaval 2013).

Treatment of ocular toxocariasis

Ocular toxocariasis can be treated either medically or surgically. Treatment is usually based on the intensity of symptoms, the appearance of intraocular inflammation, the visual impairment, the macular involvement, and the occurrence of ocular damage. It is noticeable that the most significant parameter of cure is the clinical response (Woodhall et al. 2012).

Medical treatment

The goal of medical treatment is to prevent the ocular damage and visual loss. The choice of therapeutic medications relies on the previous experience of the ophthalmologist for toxocariasis treatment. Corticosteroids are the mainstay medical treatment for ocular toxocariasis as they have the ability to decrease the release of local mediators of inflammation leading to the suppression of inflammation, induce cell membranes stabilization, and prevent vitreous opacification and tractional retinal detachment. In spite of that, corticosteroids have limited efficiency to deal with structural complications in the retina (Ahn et al. 2014). Corticosteroids are applied topically or infiltrated into periocular space, and/or given systemically, based on the clinical condition. In most cases, they result in notable amelioration (Cortez et al. 2011). Oral prednisolone is the most commonly used anti-inflammatory drug with an effective dosage of 1 mg/kg/day for a period of 1 month or more when needed; after that, the dose is reduced (Magnaval et al. 2001).

Regarding antiparasitic medications, some ophthalmologists recommend anthelmintic using in addition to corticosteroids while the others use these medications only when the response to corticosteroids is inefficient. Usage of the anthelmintic treatment for patients with ocular toxocariasis may induce an intraocular inflammation due to a hypersensitivity reaction to larval death inside the eye, leading to the permanent damage of the eye (Schneier and Durand 2011). Anthelmintic treatment can be given, especially with the presence of extraocular toxocariasis symptoms. Albendazole and diethylcarbamazine, the most commonly used drugs, have larvicidal activity and are able to penetrate the blood-brain barrier. Other anthelmintic drugs such as thiabendazole, mebendazole, and tinidazole are highly effective in preventing the progression of *Toxocara* larvae to the neurotropic phase of infection (Magnaval et al. 2001; Schneier and Durand 2011). Seong et al. (2014) presented a case of ocular toxocariasis treated with albendazole (400 mg twice daily) for 1 month, and from day 13 of the treatment, oral triamcinolone was given. Also, Antonowicz et al. (2016) presented a case of a 6-year-old patient with steroid-dependent nephrotic syndrome having ocular toxocariasis. It was observed that the lesions decreased after the treatment of the patient for 7 days with albendazole (15 mg/kg/day) plus concomitant increase of prednisone dose to 1 mg/kg/day.

The effectiveness of anthelmintics in human toxocariasis is difficult to determine because the comparatively small number of the treated cases, treatment is usually started after varying lengths of the disease and different responses of both migrating and trapped *Toxocara* larvae. In addition, the immunopathological response may differ among patients and symptoms and signs may undergo remission in treated and placebo-treated patients. Therefore, the dose rates of drugs and length of treatment differ among patients (Othman 2012).

There is a new promising horizon of potential new drugs for toxocariasis such as nitazoxanide, tribendimidine, and immunomodulators. Usage of glucan with benzimidazoles gave better therapeutic effect in experimental animals (Othman 2012). Nowadays, natural products and medicinal plants are under assessment for the treatment of toxocariasis (Musa et al. 2011; El-Sayed 2017). The value of such medicinal plants is related to their therapeutic effect and their use as template molecules for the production of novel drugs.

Surgical management

Surgical interferences are required in cases with post-inflammatory complications such as vitreous opacification, retinal scars, bands, or detachment and formation of the epiretinal membrane with vitreomacular or optic nerve traction. Pars plana vitrectomy is the most common procedure in ocular toxocariasis, especially for patients who do not respond to the medical treatment or have severe complications (Woodhall et al. 2012; Othman 2012). Successful outcome of surgery is obtained by inducing structural modification such as peeling of the membrane, removal of the vitreous opacification, or retinal reattachment which leads to improvement in visual function (Magnaval et al. 2001; Ahn et al. 2014). Surgery may preserve visual acuity in patients where fovea is not affected (Woodhall et al. 2012; Othman 2012).

Laser photocoagulation is recommended when *Toxocara* larva is directly visualized inside the eye or in cases with choroidal neovascular membrane. This procedure may induce an inflammatory reaction; therefore, it needs to combine with steroid therapy (Azira and Zeehaida 2011; Woodhall et al. 2012; Othman 2012). Cryotherapy is used to treat ocular granulomas. It is applied directly to exudation parts at the pars plana by using a double freeze-thaw procedure followed by steroid therapy administration (Arevalo et al. 2013).

In resistant cases of *Toxocara* endophthalmitis who are not responsive to surgical or medical treatment, cyclosporine A may be effective. It is an immunosuppressive

drug and may relieve signs of uveitis with lower side effects. Local ocular injections result in more drug concentration in the eye and prevent systemic adverse effects (Mora et al. 2006).

Conclusion

In Egypt, ocular toxocariasis is considered a neglected parasitic disease. Research Institute of Ophthalmology (RIO) as a professional Egyptian institution should create website support for Egyptian ophthalmologists and clinicians. This website can be provided with standardized images of ocular toxocariasis lesions, diagnostic criteria, treatment regimens, and monitoring questionnaires to determine disease severity and effectiveness of the treatment. By this manner, Egyptian ophthalmologists, clinicians, and epidemiologists could use uniform protocols to determine the occurrence of ocular toxocariasis and its prevalence more accurately. In addition, consciousness of the public population could assist *Toxocara* parasite control in pets and animals and avoid the environmental contamination.

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NGM collected the scientific data and wrote the manuscript. NME-S revised and edited the manuscript. All authors read and approved the final manuscript.

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Ethics approval and consent to participate
Not applicable

Consent for publication
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Competing interests
The authors declare that they have no competing interests.

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