Ocular Toxocariasis

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Introduction

Toxocariasis is an infection caused by the parasitic roundworms commonly found in the intestines of dogs (*Toxocara canis*) and cats (*Toxocara cati*). A US study in 1996 demonstrated that 30 % of dogs younger than 6 months deposit Toxocara eggs in their feces; each worm releases 200,000 eggs per day. Once released in the stool, the eggs require 2–4 weeks to develop and become infectious. *Toxocara* embryonic eggs have a tough shell which lengthens their viability once in the stool. If these eggs are subsequently ingested, larvae hatch in the small intestine, then continue through the intestinal wall, entering the bloodstream and migrating to muscles, lungs, liver, central nervous system, and the eyes. Often the infections are asymptomatic however the two most common syndromes are systemic toxocariasis (visceral larva migrans) and ocular toxocariasis (ocular larva migrans). The severity of the organ damage depends upon the parasite load, site of larval migration, and the host's inflammatory response. Visceral larva migrans occurs mostly in children who are at a higher risk of infection due to exposure to the eggs in sandboxes and dirt on outdoor playgrounds. Ocular larva migrans can occur even if only a single larva reaches the eye [1].

Epidemiology

Approximately 13.9 % of the US population have antibodies to Toxocara implying that millions of people have been exposed to the organism [2]. Ocular toxocariasis accounts for 1–2 % of all uveitis in children throughout the world [3]. The organism is not specific to any region of the world or US but occurs at substantially higher rates in Asia and in areas of the US with higher levels of poverty [4]. The increased prevalence of ocular toxocariasis in Asia is generally thought to be caused by food habits and traditional dishes specific to many Asian cultures. Specifically in Korea 80.8 % of ocular toxocariasis patients reported having eaten raw cow liver [5]. The higher levels of ocular toxocariasis in poverty-ridden communities in the US are associated with animal interactions, specifically stray cats and dogs, along with a higher number of pets living in less sterile environments enabling easier transmission of toxocariasis to humans [6, 7].

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Clinical Manifestations

Toxocariasis manifests as two general syndromes: systemic toxocariasis and ocular toxocariasis. Systemic toxocariasis (visceral larva migrans) is typically seen in young children and can be asymptomatic. The severity of the symptoms is dictated by the age of the patient, quantity of larva ingested, distribution of the larva in the body, and host response. The condition can be mild but may be associated with a varied range of symptoms including cough, wheezing, abdominal pain, fever, and fatigue. In more severe cases, patients can develop hepatitis, pneumonitis, and encephalitis [8]. Ocular involvement is usually not present in cases of systemic toxocariasis, and conversely systemic toxocariasis is rarely seen in cases of ocular toxocariasis.

Ocular toxocariasis manifests as granulomatous uveitis commonly involving the posterior pole or in the periphery as the most commonly affected ocular tissue is the retina. Posterior pole toxocariasis lesions are typically white or gray, round, and elevated. The degree of intraocular inflammation can vary from scant to robust depending upon the number of larvae present. The infection alone can result in direct retinal injury but secondary complications from the inflammatory response can similarly induce severe vision loss. The major causes of decreased vision have been attributed to vitreous traction, endophthalmitis, macular involving lesions, retinal detachment, and papillitis [9, 10].

Diagnosis

The diagnosis of toxocariasis is based on the clinical manifestations and supportive serologic testing. The sensitivity and specificity of the serum ELISA assay is approximately 90 % [9]. A more sensitive assay is the detection of Toxocara antibodies in the aqueous humor (calculation of a Goldmann-Witmer coefficient) [4].

Treatment

The paradigm for the treatment of infectious ocular inflammatory disease often combines appropriate antimicrobial therapy with corticosteroids to reduce the propensity for tissue injury from the associated immune response. In patients with ocular toxocariasis, the focus has been directed predominantly toward the destructive inflammatory reaction, and corticosteroids have been the mainstay of treatment administered locally and systemically (alone and in conjunction with systemic antihelminthic agents). There are case reports and limited trials demonstrating efficacy of thiabendazole (25 mg/kg twice daily for 5 days) and albendazole (100–200 mg twice daily for 5 days) in the treatment of ocular toxocariasis [11, 12].

Surgical procedures have also been employed to treat associated complications of the disease including pars plana vitrectomy, retinal cryopexy, and photocoagulation.

Conclusion

Ocular toxocariasis is a common worldwide infection caused by the roundworms *T. canis* and less commonly *T. cati*. The ocular manifestations may include granulomatous uveitis, endophthalmitis, retinal granulomas, intermediate uveitis, vitreous traction, papillitis, and tractional retinal detachment. The diagnosis is established with the appropriate clinical findings and confirmed via serum ELISA or aqueous humor aspirate for detection of *Toxocara* antibodies. The predominant modality for treatment is local and systemic corticosteroids administered alone and in conjunction with appropriate antihelminthic treatment.

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