Human ophtalmologic infections caused by visceral larva migrans

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Abstract

Zoonotic Toxocara eggs are excreted by dogs and cats. A ubiquitous distribution and resistance may lead to human, mainly children, accidental ingestion of embryonated eggs and infection. In humans the infection remains occult, often resulting in disease caused by the migration larval stages. Human infection by Toxocara canis or Toxocara cati may progress to systemic and ocular toxocariasis. Specifically, it has been observed that systemic toxocariasis, also called visceral larva migrans, tends to affect children younger than 3 years of age, whereas ocular toxocariasis typically presents in older children and young adults. Most ocular damage results from the inflammatory response that occurs following larval death that eventually may progress to blindness. The diagnosis of ocular toxocariasis can be challenging since it’s a rare disease with prevalence less than 1% with variable clinical presentation.

PARASITE BIOLOGY

Toxocara canis accomplishes its life cycle in dogs, with humans acquiring the infection as an accidental host [1, 2]. Unembryonated eggs are shed with the feces of the dog [1, 2]. Eggs embryonate and become infective in the environment [2]. Following ingestion by dogs, the infective eggs hatch and larvae penetrate the gut wall [2]. In younger dogs, the larvae migrate through the lungs, bronchial tree, and esophagus becoming sexually mature adult worms in the small intestine [2]. In older dogs, patent infections can also occur, but larval cysts in tissues are more common [2]. Encysted stages are reactivated in female dogs during late pregnancy and pass to the offspring through placenta, and less by lactation, to the puppies small intestine where adult worms become established. Puppies are a major source of environmental egg contamination. Toxocara canis can also be transmitted to dogs when they eat paratenic hosts like rabbits with parasite cysts [3]. Humans are accidental hosts who become infected by the ingestion of infective eggs in contaminated soil or infected paratenic hosts [1, 3-5]. After ingestion, the eggs hatch and larvae penetrate the intestinal wall and are carried by the circulation to a wide variety of tissues like liver, heart, lungs, brain, muscle and eye[3]. The two main clinical presentations of toxocariasis are visceral larva migrans and ocular larva migrans [6-10]. Diagnosis is usually made by serology or the finding of larvae in biopsy or autopsy specimens [3]. Toxocara cati has a similar life cycle but vertical transmission is essentially by lactogenic route from the queen to kittens [4, 11].

CLINICAL ASPECTS

Ocular toxocariasis is unilateral in 90 percent of patients [12]. The condition has traditionally been taught to affect primarily infants and young children, but recent surveys have reported Toxocara infections in a number of teenagers and young adults, as well [12]. Common symptoms that bring patients to the clinic include blurred vision and floaters [12, 13]. Pain and photophobia may also be present but are typically mild [12]. In young patients, the eye infection may not be noticed until they fail a school vision screening test, or develop strabismus or leukocoria [8, 12]. The most serious consequence of the infection is invasion of the retina, leading to granuloma formation, which occurs typically peripherally or in the posterior pole [12]. These granulomas drag the retina and create a distortion, heteropia, or detachment of the macula [3]. This pathology can also cause diffuse endophthalmitis or papillitis with secondary glaucoma and chorioretinitis [3]. Less than 25 percent of eyes with toxocariasis present with dense vitreous inflammation mimicking endophthalmitis. Interestingly ocular toxocariasis present with minimal or absent pain and photophobia and anterior or posterior synechiae [8]. Fundus visualization through the vitreous inflammation is usually difficult. Severe cases may develop an exudative retinal detachment. As the inflammation subsides, vitreous membranes may organize into a retrolental mass, which in severe cases can produce ciliary body detachment, hypotony and irreversible damage [8]. Infection with T. cati was implicated in a further case where decreased visual acuity was caused by a lesion in the macula of the eye [11]. Prevention of toxocariasis and control strategies addressing the dog and cat population
Toxocara spp. eggs are very resistant to adverse environmental conditions and remain infective for years [1, 5, 10, 14]. Since no practical methods exist for environmental egg reduction, prevention of initial contamination of the environment is the most important approach [2]. This can be achieved by taking several measures such as eliminating patent infections in dogs and cats, preventing defecation of pets in public areas, hygiene, and educating the public [6, 15, 16]. Methods to decrease contamination include: restriction of free-roaming dogs and cats, cleaning up feces from soil and on pavements by dog owners, preventing access of dogs and cats to public places, especially children’s playgrounds, and the use of strategic anthelmintic treatment of dogs and cats [1, 2, 4, 5]. Despite the proven association of household pet dogs and human toxocariasis, only a third (148/450) of veterinarian respondents routinely discussed the potential zoonotic hazards of canine roundworms with their clients and a large majority start anthelmintic on puppies after 4 weeks of age, when they already contaminate the environment [10, 17]. All dogs and cats should be screened for intestinal parasites, including ascarids, at least four times in the first year of life and at least twice every year in adults [2, 4]. Puppies and kittens should be given anthelmintic starting at 2 weeks of age and repeating every 2 weeks until regular broad-spectrum parasite control begins, and adult pets should receive year-round broad-spectrum parasite control with efficacy against ascarids [10].

REFERENCES