

Toxocariasis: Visceral and ocular larva migrans

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INTRODUCTION

Toxocariasis (also called visceral larva migrans [VLM]) refers to human infection caused by roundworms that are not natural human parasites. Toxocariasis occurs as a result of human infection with the larvae of the dog ascarid, *Toxocara canis*, or, less commonly, the cat ascarid, *Toxocara cati*. Another form of VLM is caused by human ingestion of eggs of the pig ascarid, *Ascaris suum*. Clinical presentations consist of VLM and ocular larva migrans (OLM); infection may also be subclinical.

EPIDEMIOLOGY

Toxocariasis occurs globally; approximately 1.4 billion people are infected worldwide [1,2]. Infection tends to occur more frequently in tropical regions with high humidity than in temperate regions, more frequently among rural populations with inadequate water supply and poor housing than among urban populations, and more commonly in areas affected by poverty [3]. *Toxocara* larvae can develop at temperatures <50°F, although efficiency decreases as the temperature decreases [3].

One meta-analysis estimated the global seroprevalence of *Toxocara* to be 19 percent (95% CI 16.6-21.4 percent) [4].

In this study, seroprevalence was highest in the African region, at 37.7 percent (95% CI 25.7-50.6 percent); this finding aligns with another seroprevalence study, which demonstrated antibodies

to be common in most African countries [5]. In Southeast Asia, seroprevalence ranges from 3.9 to 84.6 percent [6], with a pooled seroprevalence of 34.1 percent (95% CI 20.2-49.4 percent) [4]. Seroprevalence in the Western Pacific was 24.2 percent (95% CI 6.0-33.5 percent), in the Americas 22.8 percent (95% CI 19.7-26.0 percent), in the European regions 10.5 percent (95% CI 8.5-12.8 percent), and in the Eastern Mediterranean region 8.2 percent (95% CI 5.1-12.0 percent) [4]. In the United States, the seroprevalence of *Toxocara* was estimated at 13.9 percent based on data from 1988 to 1994 [1]; in a subsequent study based on data from 2011 to 2014, the seroprevalence was estimated to be 5 percent [7]. Rates are increased among individuals living in poverty and among certain under-represented groups (especially African Americans) [7,8].

Life cycle — The life cycle of *T. canis* occurs in dogs and the life cycle of *T. cati* occurs in cats; the prevalence of *Toxocara* infection is highest among puppies and kittens. In North America, it is estimated that about 5 percent of dogs are infected; puppies are more commonly infected than older dogs [9].

Humans acquire the infection as accidental hosts ([figure 1](#)). Eggs are shed in the stool of the definitive host. In the environment, eggs embryonate and become infective after about three weeks. Soil contamination with infectious eggs occurs most readily in relatively warm climates, and these embryonated eggs can remain infective in the environment for several years [10]. Following ingestion of infective eggs by dogs or cats, the eggs hatch and larvae (0.5 mm in length) penetrate the gut wall. The larvae then migrate through the lungs, bronchial tree, and enter the esophagus; adult worms develop in the small intestine, where they lay eggs that are shed in the stool.

In most older animals, larvae penetrate the gut wall and, subsequently, larvae encyst in tissues. Encysted larvae can reactivate in female dogs during late pregnancy and infect puppies via transplacental and transmammary routes; adult worms can subsequently become established in the small intestines of puppies.

Toxocara can also be transmitted through ingestion of paratenic hosts (a host that is not required for the parasite life cycle but nonetheless can serve to maintain the life cycle). Eggs ingested by small noncanine mammals (such as rabbits) can hatch and larvae can penetrate the gut wall with subsequent migration into various tissues where they encyst. The life cycle is completed when dogs eat larvae encysted in the tissues of these paratenic hosts and the larvae develop into egg-laying adult worms in the small intestine.

Humans are accidental hosts who become infected by ingesting infective eggs in contaminated soil or food or via ingestion of encysted larvae in the tissues of infected paratenic hosts. Direct contact with infected puppies and kittens is not classically considered to be a risk factor for

human infection since the eggs must embryonate before becoming infective, although sometimes pets carry embryonated eggs in their fur. Following ingestion, the eggs hatch and larvae penetrate the intestinal wall and are carried by the circulation to a variety of tissues (liver, heart, lungs, brain, muscle, eyes). The larvae do not undergo any further development in these sites, but the host inflammatory response against the migrating larvae can cause both mechanical and immunopathologic damage to tissues, which leads to local reactions that are the basis of clinical toxocariasis.

Transmission and risk factors — Individuals who ingest embryonated eggs (in soil or on vegetables or fruits) are at risk for developing infection [11]. Infection can also be acquired via ingestion of raw liver or other undercooked meat from an infected intermediate host (rabbit, chicken, cattle, or swine) containing encapsulated larvae [12].

VLM is principally a disease of young children, especially those with exposure to playgrounds and sandboxes contaminated by dog or cat feces [13]. Having a dog as a pet is a recognized risk factor [14]; however, infection is often acquired in settings outside of the home (such as in public parks and playgrounds) [15].

Toxocara infection does occur in travelers, but the incidence is low [16-18].

CLINICAL MANIFESTATIONS

Clinical manifestations range from asymptomatic infection to severe organ injury; they occur as a consequence of damage caused by migrating larvae in addition to the host eosinophilic granulomatous response. Migration of larvae can cause eosinophilic infiltration, granuloma formation, or eosinophilic abscesses [12].

Toxocara spp larvae are unable to grow or replicate in the human host, but parasites can remain viable for at least seven years after infection [19]. Most infections are self-limited as larvae become encapsulated (usually in the musculature and liver).

There are two major categories of clinical manifestations: VLM and OLM. These have also been reported to occur simultaneously [15,20].

Visceral larva migrans — VLM occurs most commonly in young children and results in hepatitis and pneumonitis as the larvae migrate through the liver and lungs, respectively. Heavy infection may result in fever, anorexia, malaise, irritability, hepatomegaly, respiratory symptoms, pruritic urticaria-like cutaneous lesions, and eosinophilia.

Larvae frequently localize in the liver; hepatic manifestations may include hepatomegaly or nodular lesions. Pulmonary involvement may cause dyspnea, wheezing, and a chronic nonproductive cough in 20 to 80 percent of patients [21,22]. Rales are common on physical examination. The chest radiograph demonstrates abnormalities in ≥ 40 percent of patients with symptomatic illness. Bilateral peribronchial infiltration is most common; parenchymal infiltrates can also occur [21,22]. Computed tomography may demonstrate multifocal subpleural nodules with halo or ground-glass opacities and ill-defined margins [23]. Severe respiratory tract involvement is an uncommon complication of heavy infection. Migration of larvae through the intestinal wall can cause eosinophilic enteritis and peritonitis [24].

Toxocariasis can be mistaken for metastatic disease [25,26]; the presence of eosinophilia and radiologic findings may be helpful differentiating features. (See '[Diagnosis](#)' below.)

Larvae can also travel via the systemic circulation to muscles, the heart, the eye, or the central nervous system (CNS) [27,28]. Cardiac involvement in *Toxocara* spp infection is a rare but potentially life-threatening complication. The clinical presentation may consist of myocarditis, pericarditis, Loeffler endocarditis (eosinophilic myocarditis), or pericardial effusion; heart failure or cardiac tamponade can occur and can be fatal [29,30]. (See "[Endomyocardial fibrosis](#)", [section on 'Eosinophilia'](#).)

CNS manifestations include eosinophilic meningo-encephalitis, space-occupying lesions, myelitis, and cerebral vasculitis causing seizures [31-33]. In a meta-analysis including 11 case-control studies and more than 4700 patients with toxocariasis, an association between epilepsy and *Toxocara* spp seropositivity was observed (pooled odds ratio [OR] 1.69, 95% CI 1.42-2.01) [34]. Studies have also found an association between infection and cognitive delay, neurodegeneration, or psychiatric illness; however, it is uncertain whether *Toxocara* infection plays a causal role in pathogenesis [35-37]. Manifestations of the peripheral nervous system include radiculitis, affection of cranial nerves, or musculoskeletal involvement [38]. Death due to myocardial or CNS involvement has been described but is rare.

Ocular larva migrans — OLM involvement may occur as the sole manifestation of VLM; it often presents in individuals without antecedent history of symptomatic VLM [39]. OLM occurs most commonly among older children and adolescents [13]. In one review of ocular toxocariasis in the United States, the median patient age was 8.5 years (range 1 to 60 years) [8].

The ocular lesion is due to larval localization in the eye and the granulomatous response around the larva. Common symptoms are unilateral visual impairment, blurry vision, photophobia, floaters, leukocoria, subsequently causing failing vision and strabismus. The typical lesion is a posterior pole or peripheral whitish elevated granuloma. Occasionally, OLM may present as

uveitis (often intermediate and posterior uveitis), papillitis, endophthalmitis, scleritis, or chronic endophthalmitis [40]. Ocular lesions may resemble retinoblastoma ([table 1](#)) [41]. The most serious consequence of infection is invasion of the retina with granuloma formation in the periphery or posterior pole, leading to dragging of the retina and eventual retinal detachment, which can lead to blindness [42]. Other complications include ocular hypertension, cataract formation, macular cyst edema, and persistent vitreous opacity [43].

OLM is discussed further separately. (See "[Approach to the child with leukocoria](#)", section on '[Ocular toxocariasis](#)'.)

Other presentations — *Toxocara* can also present with other manifestations. Mild infection may present with eosinophilia only. Other symptoms may include fever, headache, behavioral disturbances, anorexia, abdominal pain, rash, hepatomegaly, nausea, vomiting, as well as wheezing and pulmonary infiltrates [15,44].

Wheezing and pulmonary infiltrates, together with eosinophilia, are also the hallmark features of childhood asthma. It has been postulated that the presence of *Toxocara* larvae in lungs may be an underlying factor in the onset of allergic pulmonary disease, perhaps because of the host response to the parasite. In a meta-analysis including 17 studies (11 case-control studies and six cross-sectional studies), an increased risk for asthma among children with *Toxocara* infection seropositivity was observed (OR 1.91, 95% CI 1.47-2.47) [45]. However, a causal association remains uncertain, as both positive associations and negative epidemiologic studies have been reported [46,47].

Cutaneous manifestations are relatively common, either alone, together with eosinophilia, and/or in conjunction with other clinical manifestations of toxocariasis. Chronic urticaria is the most dermatologic manifestation; others include chronic pruritus, transient rash, different forms of eczema, hypodermic nodules, eosinophilic panniculitis, and vasculitis [48,49].

Two additional toxocariasis syndromes have been described: "covert toxocariasis" (seen mainly in children) and "common toxocariasis" (seen predominantly in adults); these are probably variations of the same clinical entity with manifestations varying according to the site and intensity of infection and the age of the host [50,51]. Covert toxocariasis refers to nonspecific symptoms and signs including fever, abdominal pain, anorexia, nausea, vomiting, hepatomegaly, cough, headache, lethargy, behavioral and/or sleep disturbances, skin symptoms, limb pains, and lymphadenitis associated with high titers of anti-*Toxocara* antibodies, with or without eosinophilia [52]. Common toxocariasis refers to a syndrome comprised of chronic weakness, shortness of breath, abdominal pain, rash, itch, urticarial and

arthralgia, often with eosinophilia, high immunoglobulin (Ig)E levels, and high titers of *Toxocara*-specific antibodies [53].

Laboratory tests — In general, VLM should be suspected in the setting of compatible clinical manifestations, together with leukocytosis, eosinophilia, and hypergammaglobulinemia (elevated serum levels of IgE and IgG). Marked leukocytosis with eosinophilia occurs in more than 30 percent of cases of VLM, and elevated titers of anti-A or anti-B isohemagglutinins are commonly observed in about 50 percent of patients [15].

There is no peripheral blood eosinophilia in most cases of OLM, presumably because of the low larval load [54]. Eosinophilia may also be absent in patients with long-standing infection and in those with cutaneous symptoms only [50]. An eosinophilic granulomatous hepatitis may develop, leading to abnormalities in liver function tests including elevated transaminases and/or alkaline phosphatase.

DIAGNOSIS

In general, the diagnosis of toxocariasis should be suspected based on history, clinical examination, and laboratory findings of leukocytosis and eosinophilia; the diagnosis is confirmed via serology (or molecular methods if available) [55].

Laboratory assays — There are several commercial enzyme-linked immunosorbent assay (ELISA) antibody assays that detect human IgG antibodies to *Toxocara* excretory/secretory antigens of the third-stage larvae of *T. canis*. The test can detect subclinical or mild infection, though it cannot differentiate between *T. canis* and *T. cati* infections. The reliability varies by assay and clinical presentation. As an example, for VLM and some forms of covert toxocariasis, the sensitivity and specificity of the *Toxocara* enzyme immunoassay (titer 1:32) are estimated at about 78 percent and 92 percent, respectively [13,15,56].

A positive ELISA result does not necessarily demonstrate the presence of active *Toxocara* infection or prove that clinical symptoms are attributable to toxocariasis; the result must be interpreted in the setting of compatible clinical symptoms and epidemiologic exposure. However, a negative test can help to rule out VLM, although ocular and neurologic toxocariasis can occur with negative serology. Cross-reactivity of the ELISA assay with other parasite antigens is common, and the test may remain positive for several years even following treatment. In some settings, positive ELISA results can be confirmed by western blot [57], which has better sensitivity and specificity compared with ELISA [58] but is also more expensive and labor intensive [59].

Future improvements in *Toxocara* serodiagnosis will likely include the use of recombinant antigens, simpler assay formats, IgG4 subclass detection, and antigen detection tests [60,61]. Polymerase chain reaction-based methods for detecting *Toxocara* in clinical samples have been described but are not commercially available [62]. Definitive diagnosis of VLM may also be established via detection of larvae in biopsy tissue, which will show *Toxocara* larvae within eosinophilic granulomatous lesions. However, biopsy is rarely indicated.

The sensitivity of ELISA for OLM is considerably lower than for VLM; the diagnosis of OLM generally relies on the findings on ophthalmologic examination ([picture 1](#)) [63]. Negative serology can occur with a positive vitreous titer [64]. It is possible to compare the antibody levels between the serum and aqueous humour; if the (level of specific IgG in aqueous humour/level of specific IgG in serum)/(total IgG in aqueous humour/total IgG in serum) is greater than 3.0, this can be considered diagnostic [65].

Stool examinations are not helpful since the parasite does not complete a full life cycle involving the human gastrointestinal tract.

Pulmonary involvement may result in eosinophilia that is detectable in bronchoalveolar lavage (BAL) fluid. One case of marked pulmonary infiltration demonstrated 64 percent eosinophils in the BAL analysis [58].

In the setting of central nervous system involvement, the cerebrospinal fluid may show eosinophils [66]. (See "[Eosinophilic meningitis](#)".)

Imaging studies — Hepatic and cerebral lesions may be observed with ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) [67,68]. In one review of radiographic changes associated with hepatic VLM, CT and MRI showed multiple, ill-defined lesions, usually measuring 1.0 to 1.5 cm in diameter, scattered throughout the liver parenchyma [12]. Lesions are usually oval but may be angular or trapezoid. The lesions differ from metastatic nodules in that they are usually uniform in size, nonspherical in shape, and are best seen on portal venous phase ([image 1](#)). Pulmonary VLM appear on CT as multifocal subpleural nodules with halo or ground-glass opacities (GGOs) and ill-defined margins ([image 2](#)) [23].

In one review of radiographic changes associated with pulmonary VLM, four patterns were observed: GGOs, solid nodules, consolidation, and linear opacities; lower lung involvement was predominant [69].

Ultrasound biomicroscopy has been used as a diagnostic technique for the evaluation of patients with ocular toxocariasis [70].

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of VLM includes:

- Strongyloidiasis – Transpulmonary migration of *Strongyloides* larvae can produce dry cough, throat irritation, dyspnea, wheezing, and hemoptysis. Similarly, pulmonary involvement of toxocariasis may cause dyspnea, wheezing, and a chronic, nonproductive cough. *Strongyloides* larvae may be detected in stool, sputum, bronchoalveolar lavage fluid, or pleural fluid. (See "[Strongyloidiasis](#)".)
- Schistosomiasis – Schistosomiasis can cause periportal fibrosis and eosinophilia; in general, there are no apparent signs of liver dysfunction. This is in contrast with VLM, which may be associated with abnormal liver function tests including elevated transaminases and/or alkaline phosphatase. The diagnosis of schistosomiasis is established with stool microscopy and/or serologic testing. (See "[Schistosomiasis: Epidemiology and clinical manifestations](#)" and "[Schistosomiasis: Diagnosis](#)".)
- Ascariasis – Ascariasis may be associated with pneumonitis (known as Loeffler syndrome) in sensitized individuals during larval migration through the lungs. Pulmonary symptoms are less common in regions with continuous transmission of *Ascaris lumbricoides*. Ascariasis may also involve the biliary tree but is not associated with hepatitis as with VLM. The diagnosis of ascariasis is usually established via stool microscopy. (See "[Ascariasis](#)".)
- *Echinococcus* – *Echinococcus* is associated with cystic lesions in the liver and lung that may be demonstrated with ultrasonography or computed tomography; VLM may be appreciated on computed tomography as nodular lesions. The diagnosis of *Echinococcus* is established with a combination of imaging and serology. (See "[Echinococcosis: Clinical manifestations and diagnosis](#)".)
- Allergic bronchopulmonary aspergillosis – Allergic bronchopulmonary aspergillosis is a complex hypersensitivity reaction that occurs when bronchi become colonized by *Aspergillus* species. Clinical manifestations include recurrent episodes of bronchial obstruction, fever, malaise, cough, and peripheral blood eosinophilia. Chest radiography may demonstrate parenchymal infiltrates and bronchiectasis. (See "[Clinical manifestations and diagnosis of allergic bronchopulmonary aspergillosis](#)".)

The differential diagnosis of OLM includes:

- Retinoblastoma – Both retinoblastoma and OLM may present with strabismus, poor vision, and leukocoria ("white pupil") ([table 1](#)). The diagnosis of retinoblastoma can usually be made with dilated ophthalmoscopic examination. Pathology is necessary to confirm the diagnosis. (See "[Retinoblastoma: Clinical presentation, evaluation, and diagnosis](#)".)
- Toxoplasmosis – Toxoplasmosis can cause chorioretinitis (a posterior uveitis). It is suspected based on a characteristic scarring appearance ([picture 2](#)); the diagnosis is supported by serology. (See "[Toxoplasmosis: Ocular disease](#)".)
- Ocular tuberculosis – Ocular tuberculosis may be presumed in the presence of suggestive ocular findings (such as choroidal granuloma, broad-based posterior synechiae, retinal vasculitis with or without choroiditis, or serpiginous-like choroiditis) in combination with systemic findings consistent with tuberculosis.

Other parasitic infections can cause eosinophilia in the setting of focal lesions; examples include capillariasis (liver lesions) and gnathostomiasis (brain lesions).

TREATMENT

The treatment approach varies according to symptoms and location of the larvae; data are limited. Treatment is generally based on clinical experience and expert opinion. Most individuals with mild symptoms due to toxocariasis do not require anthelmintic therapy; symptoms are usually self-limited and resolve within a few weeks [71]. Eosinophilia may resolve much more slowly over many months, likely due to ongoing antigenic stimulation from dead larvae. In the setting of protracted symptoms, the possibility of reinfection (such as from continued ingestion of contaminated soil) should be considered.

For individuals with moderate to severe symptoms due to VLM, treatment consists of [albendazole](#) (400 mg orally with fatty meal twice daily for five days) [72]. In cases of severe respiratory, myocardial, or central nervous system involvement, concomitant [prednisone](#) (0.5 to 1 mg/kg/day) is warranted. Some favor treatment in the setting of moderate eosinophilia and positive serology even in the setting of minimal symptoms, given risk of larval localization to the brain during the course of infection [50,54].

For treatment of OLM topical or systemic corticosteroids are used to control intraocular inflammation. If there is sight-threatening ocular inflammation, we favor treatment with anti-inflammatory therapy with corticosteroids (eg, [prednisone](#) 0.5 to 1 mg/kg/day with slow taper). The extent of benefit on initial clinical symptoms achieved by adding in [albendazole](#) therapy is uncertain but recurrences are reduced [73,74]. In addition to prednisone, we therefore

recommend albendazole 400 mg orally twice daily for two weeks, given with a fatty meal. The optimal dosing for albendazole is uncertain; data are limited to retrospective studies [75]. In complicated cases, surgical intervention may be warranted [76].

Mebendazole (100 to 200 mg orally twice daily for five days) is an alternative to **albendazole** [77], but albendazole is preferred (particularly in OLM and neurologic disease) since it crosses the blood-brain barrier. **Ivermectin** does not appear to be effective for toxocariasis [78].

Diethylcarbamazine (3 to 4 mg/kg/day for 21 days, starting at 25 mg/day for adults) has been found to be effective in a small number of cases but has greater side effects than albendazole so it is rarely used.

Follow-up consists of monitoring the eosinophil count, which usually decreases within one month of treatment [79]. Serology is not a good follow-up tool because IgG decreases very slowly. Studies have suggested that IgE decreases after therapy [80], but this is not reliable [59].

PREVENTION

Good hygiene practices, timely disposal of pet feces, and routine deworming of pets are important strategies for prevention of toxocariasis in humans [8]. Hand washing should be encouraged after contact with pets or areas at high risk for soil contamination, such as playgrounds and sandboxes [8]. Education regarding behaviors that increase risk for infection is also important, including geophagia and consumption of raw or undercooked meat (particularly liver). Consuming raw vegetables that have been irrigated by contaminated wastewater or grown in soil contaminated with *Toxocara* eggs is also a risk factor for infection; cooking or thorough rinsing in clean water will render foods safe [81].

SUMMARY AND RECOMMENDATIONS

- Toxocariasis (also called visceral larva migrans [VLM]) refers to human infection caused by helminths that are not natural human parasites. Toxocariasis occurs as a result of human infection with the larvae of the dog ascarid, *Toxocara canis*, or, less commonly, the cat ascarid, *Toxocara cati*. VLM is principally a disease of young children, especially those with exposure to playgrounds and sandboxes contaminated by dog or cat feces. (See ['Introduction'](#) above.)
- The life cycle of *T. canis* occurs in dogs ([figure 1](#)). Puppies are a major source of environmental egg contamination. Humans are accidental hosts who become infected by ingesting eggs in contaminated soil or encysted larvae in the tissues of infected paratenic

hosts. Ingested eggs mature into larvae which penetrate the intestinal wall and are carried by the circulation to a variety of tissues (liver, heart, lungs, brain, muscle, eyes). The larvae can cause severe local reactions at these sites. (See '[Life cycle](#)' above.)

- Larvae frequently localize in the liver; hepatic manifestations may include hepatomegaly or nodular lesions. Mild infection may be asymptomatic and only suspected by the finding of elevated blood eosinophilia. Heavy infection may result in fever, anorexia, malaise, irritability, hepatomegaly, and pruritic urticaria-like cutaneous lesions. Ocular larva migrans (OLM) is due to larval localization in the eye and the granulomatous response around the larva. Common symptoms are unilateral visual impairment and subsequent strabismus; complete visual loss can occur. (See '[Clinical manifestations](#)' above.)
- VLM should be suspected in the setting of compatible clinical manifestations together with leukocytosis, eosinophilia, and hypergammaglobulinemia (elevated serum levels of immunoglobulin [Ig]E and IgG). In the appropriate clinical scenario, the diagnosis can be confirmed by enzyme-linked immunosorbent assay antibody assay, which detects human IgG antibodies to *Toxocara* excretory/secretory antigens. (See '[Laboratory tests](#)' above.)
- In general, individuals with mild symptoms may not require anthelmintic therapy as symptoms are usually self-limited. For individuals with moderate to severe symptoms, we suggest treatment with [albendazole \(Grade 2C\)](#). In cases of severe respiratory, myocardial, or central nervous system involvement, we suggest concomitant treatment with [prednisone \(Grade 2C\)](#). (See '[Treatment](#)' above.)
- Good hygiene practices, timely disposal of pet feces, and routine deworming of pets are important strategies for prevention of toxocariasis in humans. Hand washing should be encouraged after contact with pets or areas at high risk for soil contamination, such as playgrounds and sandboxes. (See '[Prevention](#)' above.)

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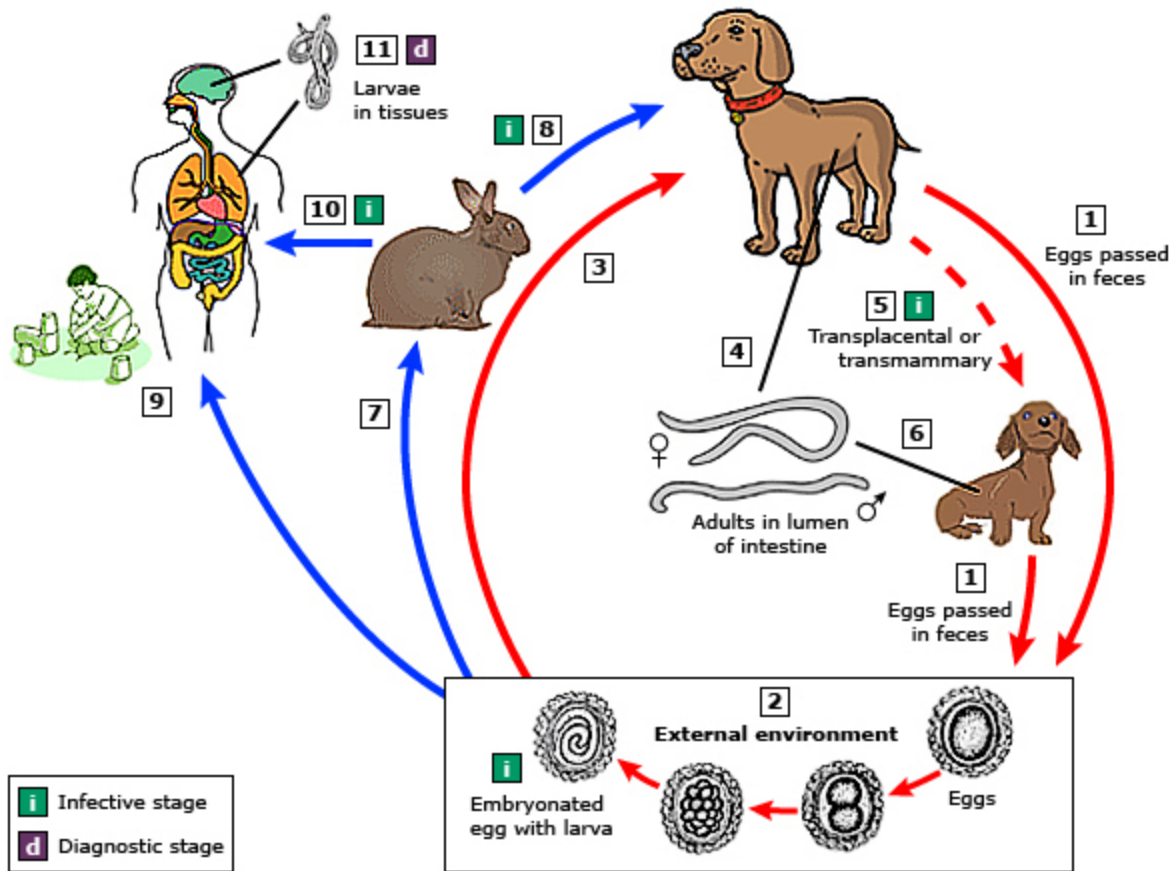
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GRAPHICS

Toxocariasis life cycle



Toxocara canis accomplishes its life cycle in dogs, with humans acquiring the infection as accidental hosts. Unembryonated eggs are shed in the feces of the definitive host (1). Eggs embryonate and become infective in the environment (2). Following ingestion by dogs (3), the infective eggs hatch and larvae penetrate the gut wall. In younger dogs, the larvae migrate through the lungs, bronchial tree, and esophagus; adult worms develop and oviposit in the small intestine (4). In older dogs, patent infections can also occur, but larval encystment in tissues is more common. Encysted stages are reactivated in female dogs during late pregnancy and infect by the transplacental and transmammary routes the puppies (5), in whose small intestine adult worms become established (6). Puppies are a major source of environmental egg contamination. *Toxocara canis* can also be transmitted through ingestion of paratenic hosts: eggs ingested by small mammals (eg, rabbits) hatch and larvae penetrate the gut wall and migrate into various tissues where they encyst (7). The life cycle is completed when dogs eat these hosts (8) and the larvae develop into egg-laying adult worms in the small intestine. Humans are accidental hosts who become infected by ingesting infective eggs in contaminated soil (9) or infected paratenic hosts (10). After ingestion, the eggs hatch and larvae penetrate the intestinal wall and are carried by the circulation to a wide variety of tissues (liver, heart, lungs, brain, muscle, eyes) (11). While the larvae do not undergo any further development in these sites, they can cause severe local reactions that are the basis of toxocariasis. The two main clinical presentations of toxocariasis are visceral larva migrans and ocular

larva migrans. Diagnosis is usually made by serology or the finding of larvae in biopsy or autopsy specimens.

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Distinguishing between ocular toxocariasis and retinoblastoma

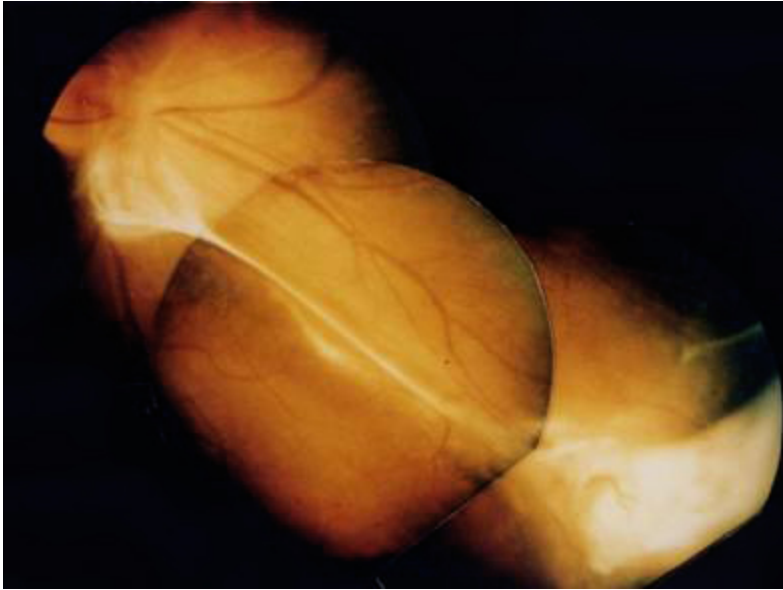
Parameter	Description	
	Ocular toxocariasis	Retinoblastoma
Morphology	Centrally located mass with moderate enhancement revealed by MRI	Nodular in contour and lies along the posterior pole of the globe
Enhancement	Moderate uveoscleral enhancement or enhancement of a granuloma	Usually characterized by moderate or avid enhancement
Calcification	Rare	Punctate or speckled calcification in more than 90 percent of cases
Demographics	Patients usually 5 to 10 years of age	Observed before 5 years of age in more than 90 percent of cases, average age at diagnosis is approximately 18 months
Signs and complications	Usually presents with unilateral visual loss, and pain and redness are common presenting symptoms	Leukocoria in more than half of cases; strabismus is the second most common presentation, occurring in approximately 20 percent of cases

MRI: magnetic resonance imaging.

From: Fan CK, Holland CV, Loxton K, Barghouth U. Cerebral toxocariasis: Silent progression to neurodegenerative disorders? Clin Microbiol Rev 2015; 28(3):663-86. DOI: 10.1128/CMR.00106-14. Reproduced with permission from American Society for Microbiology. Copyright © 2015.

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Ocular toxocariasis with peripheral granuloma

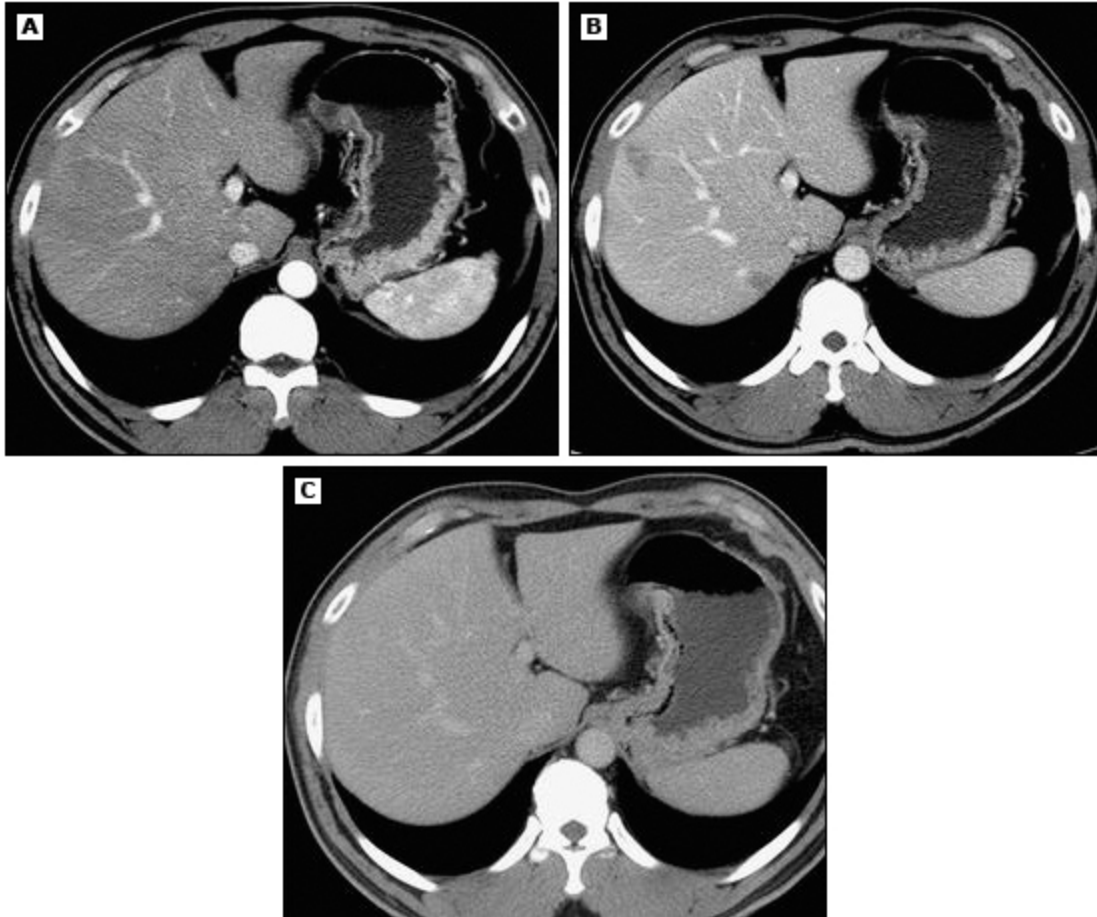


Ocular toxocariasis with peripheral granuloma and vitreoretinal traction.

*Original figure from: Pivetti-Pezzi P. Ocular Toxocariasis. Int J Med Sci 2009; 6:129-130. <http://www.medsci.org/v06p0129.htm>.
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Hepatic toxocariasis

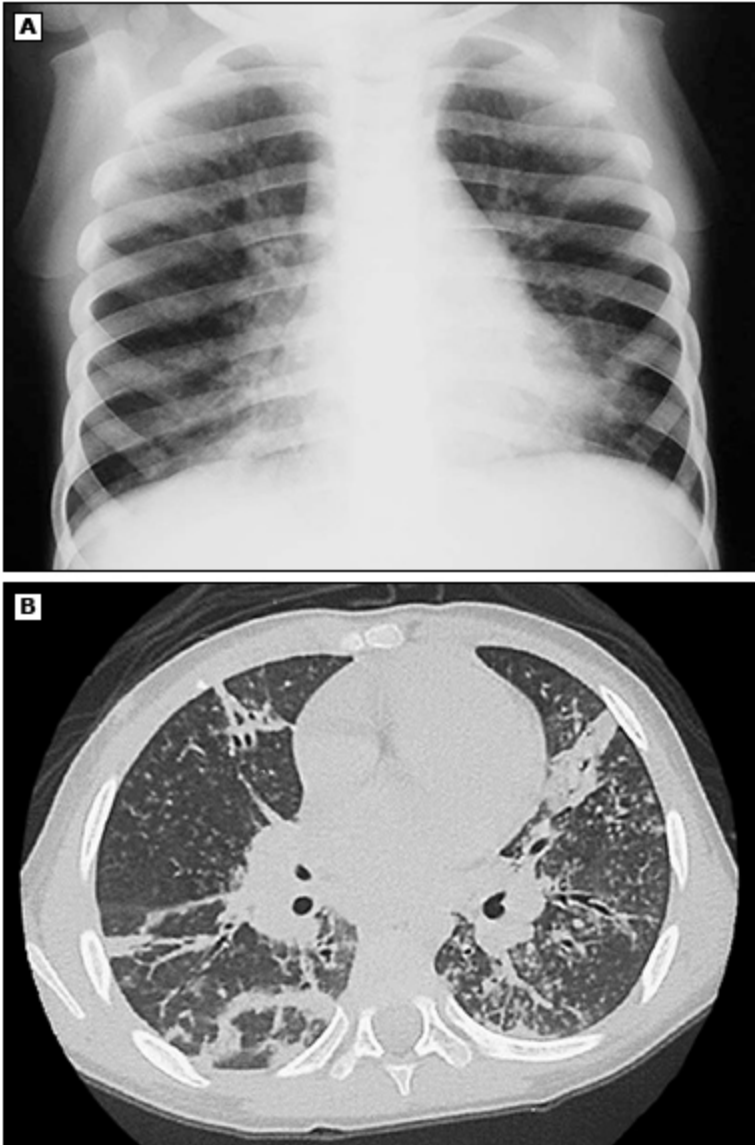


A 43-year-old man with hepatic toxocariasis. Contrast-enhanced arterial (A), portal phases (B), and equilibrium phase (C). Computed tomography images display two small triangular and oval nodules in the periphery of the liver. The nodules are clearly seen on portal venous phase image but not clearly seen on arterial and equilibrium phase images.

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Toxocariasis - Chest images



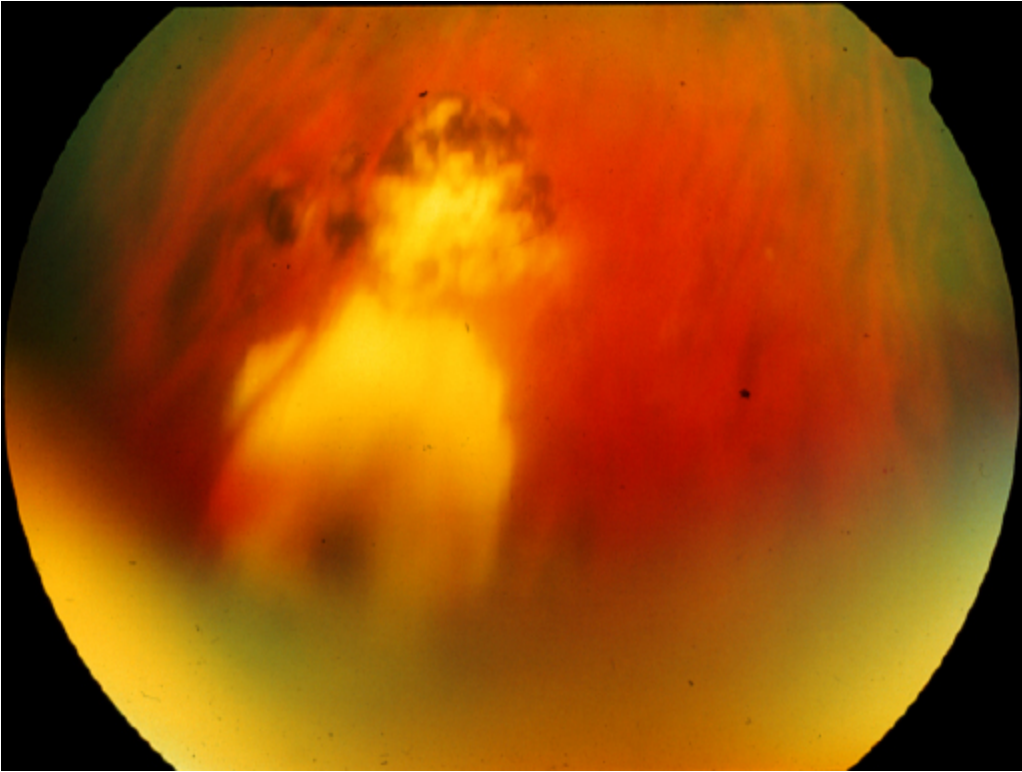
(A) Radiograph of a three-year-old girl with toxocariasis.

(B) Computed tomography scan of the same 3-year-old girl with toxocariasis as shown in panel A.

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Toxoplasma chorioretinitis



Chorioretinitis characteristic of toxoplasmosis. A pigmented scar is seen with an adjacent area of active chorioretinitis. The diagnosis of toxoplasmosis is based primarily on the appearance of the chorioretinal lesion rather than serologic studies.

Courtesy of James T Rosenbaum, MD.

Graphic 69853 Version 3.0

Contributor Disclosures

Peter F Weller, MD, MACP Consultant/Advisory Boards: AstraZeneca [DSMB – Hypereosinophilic syndrome]; GlaxoSmithKline [Eosinophilic diseases]. All of the relevant financial relationships listed have been mitigated. **Karin Leder, MBBS, FRACP, PhD, MPH, DTMH** No relevant financial relationship(s) with ineligible companies to disclose. **Edward T Ryan, MD, DTMH** No relevant financial relationship(s) with ineligible companies to disclose. **Elinor L Baron, MD, DTMH** No relevant financial relationship(s) with ineligible companies to disclose.

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