Toxocariasis

**Synonyms:** toxocarosis, visceral larva migrans, ocular larva migrans, covert toxocariasis, Toxocara canis, Toxocara cati

Human toxocariasis is caused by migration of the larvae of *Toxocara canis* (dog roundworm) and less frequently of *T. cati* (cat roundworm) through the human tissues. Toxocara are nematode parasites of animals. Despite cases of illness due to *T. cati* (which can be difficult to distinguish from *T. canis* as they share many common antigens), the importance of this zoonosis has yet to be fully established.[1]

Humans become infected by ingestion of eggs in soil contaminated by dog faeces. Larvae hatch out in the small intestine and migrate via the liver and lungs to other tissues, though they never mature in humans. In most cases the larvae are probably eliminated, but a surrounding granuloma may form in some patients. Rarely, *T. cati* can mature in humans and be transmitted by vomit or faeces. However, patients with adult *T. cati* don't have antecedent symptoms, eosinophilia or antibodies, suggesting that they are acquired by ingestion of adult worms or advanced larval stages from cat vomit or faeces.

**Life cycle**[2]

- The definitive hosts of *T. canis* are dogs, with humans acquiring the infection as accidental hosts. Unembryonated eggs are shed in the faeces of the definitive host. Following ingestion by dogs, the infective eggs hatch and larvae penetrate the gut wall.
- In younger dogs, the larvae migrate through the lungs, bronchial tree, and oesophagus. Adult worms develop and deposit eggs in the small intestine. In older dogs, infections may occur but larval encystment in tissues is more common.
- Encysted stages are reactivated in female dogs during late pregnancy with transplacental and transmammary infection of the puppies, in whose small intestine adult worms become established. Puppies are a major source of environmental egg contamination.
- *T. canis* can also be transmitted by ingestion of other hosts: eggs ingested by small mammals (eg, rabbits) hatch and larvae penetrate the gut wall and migrate into various tissues where they encyst. The life cycle is completed when dogs eat these 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 infected animals such as rabbits. 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). The larvae do not undergo any further development in these sites but they can cause severe local reactions that are the basis of toxocariasis.

**Epidemiology**

- Distribution is worldwide.[3]
- Exposure is common, especially among children from socio-economically disadvantaged populations both in the tropics and sub-tropics and in industrialised nations.[4]
- Seroprevalence studies (particularly tropical and relatively undeveloped regions) show that the vast majority of the population has been exposed at some time.[5]
- Many studies show contamination of children's play areas/sandpits with eggs of *T. canis*, in about 15-25% of those surveyed.[6]
- Prevalence rates for visceral larva migrans are difficult to estimate, as the disease is largely asymptomatic in many, and presents in a variety of ways.[7]

**Risk factors**

- Contact with soil contaminated with dog faeces; children are at greatest risk as they may ingest soil with contaminated eggs in playing areas.
- Cohabitation with dogs and cats.
- Eating without hand washing.
- Socio-economic deprivation.
- Rural dwelling.
- Travel to areas of high prevalence.

**Presentation**[8]

- Studies suggest symptomatic illness may be due to induced autoimmunity associated with the infestation. Affected areas include liver, lungs, skin, joints, eyes, heart and brain.
- Many human infections are asymptomatic, with only eosinophilia and positive serology. The two main clinical presentations of toxocariasis are visceral larva migrans and ocular larva migrans.[8]
Visceral larva migrans:
- Occurs mostly in preschool children.
- The larvae invade multiple tissues and clinical features include fever, anorexia, abdominal pain, weight loss, cough, wheezing, pleural effusion, hepatosplenomegaly, urticaria, subacute meningitis/encephalitis, seizures, monoarthritis, cutaneous vasculitis, myocarditis and hypereosinophilia.
- Death (from severe cardiac, pulmonary or neurological involvement) may occur but is rare.

Ocular larva migrans:[3]
- Often occurs in older children or young adults, with only rarely associated eosinophilia or visceral manifestations.
- Clinical features include uniocular decreased visual acuity or blindness, strabismus, seeing floaters or ‘bubbles’, peripheral retinal lesions, retinal detachment, peripheral retinal exudates, optic neuritis, vitreoretinal bands, uveitis, vitreous abscess.

Differential diagnosis
- Visceral form: other causes of acute hepatitis, adverse drug reactions, pulmonary eosinophilia, other helminth infestations, allergic bronchopulmonary aspergillosis, angio-oedema, hypereosinophilic syndrome, eosinophilic pneumonia, systemic lupus erythematosus.
- Ocular form: this should be investigated by ophthalmologists to exclude other possible causes. Some cases of ocular larva migrans have been misdiagnosed as retinoblastoma, resulting in surgical enucleation.

Investigations[10]
- FBC: leukocytosis with marked eosinophilia (20-80% of total white cell count). Eosinophilia is less common in ocular and occult forms.
- Serology may be positive (in the ocular form, test vitreous fluids for antibodies). The currently recommended serology test for toxocariasis is enzyme immunoassay.[11]
- Ultrasound of the liver may show multiple hypoechoic areas.
- CXR may show nodular infiltrates if there is lung involvement.
- CT/MRI scan of the brain may show meningeal/cerebral involvement.
- Tissue biopsy may be necessary.

Management

Visceral[4, 12]
- Most patients recover without treatment.
- Anthelmintics. Albendazole or mebendazole are usually used in advanced or highly symptomatic cases or where there is organ damage.
- Corticosteroids play a role in suppressing intense allergic manifestations of the disease.

Ocular[3]
- Surgical intervention may be required.
- Corticosteroids have an important role.
Prognosis and complications

- In the ocular form, outcome is variable but uniocular visual loss is not uncommon.
- In the visceral form, outcome is usually good but marked organ damage and even death can occur in extreme cases.
- Epilepsy may be associated with toxocariasis.¹³

Prevention

- Removal of pet faeces (‘poop scoops’, bins, and enforcement fines).
- Personal hygiene.
- Wearing gloves when gardening.
- Vegetables to be eaten raw should be washed.
- Regular worming of pets, especially puppies.

Further reading & references

2. Toxocariasis; DPDx - Centers for Disease Control and Prevention
12. British National Formulary

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Document ID: 2890 (423)
Last Checked: 15/10/2014
Next Review: 14/10/2019

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