Toxocarosis, an important zoonosis

P.A.M. Overgaauw(1)  F. van Knapen(2)

SUMMARY

This paper reviews the current knowledge about an important zoonotic dog and cat parasite, Toxocara. A good understanding of the epidemiology is required so that effective prevention of infection in man, dogs and cats can be possible. Education of the dog and cat owner will be significant in prevention. In this review, the epidemiology, clinical symptoms, diagnosis, prevention and control in the dog, the cat and the human will be discussed. Uniform guidelines for deworming dogs and cats will be given.

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Introduction

Toxocarosis of dogs and cats

Toxocara canis and Toxocara cati are roundworms of dogs and cats and the reported infection rates in Western Europe vary from 3.5% to 17% for T. canis in dogs and 8% to 76% for T. cati in cats [8, 15, 22, 35, 39, 41, 52]. The prevalence of patent Toxocara infections is highest in young dogs and cats and much less common in adult animals. Toxocara infection follows ingestion of embryonated Toxocara eggs or larvae in a paratenic host. Migration of larvae can lead to (overt) clinical disease (toxocarosis) in the (paratenic) host.

Epidemiology

Infection of the dog and cat

Adult worms in the intestinal tract of infected dogs and cats shed large numbers of eggs into the environment (Fig. 2) via the faeces where they embryonate (Fig. 3) and maybe ingested by natural hosts as well as paratenic hosts. In the intestine the larvae hatch (Fig. 3) and migrate throughout the body via blood vessels. This is called visceral larva migrans or VLM (Fig. 4 and 5). In young animals larvae migrate from the lungs up the trachea and after swallowing, mature in the intestinal tract. In paratenic hosts and most adult dogs and cats that have some degree of acquired immunity, the larvae undergo somatic migration and remain as somatic larvae in the tissues. After predation of Toxocara infected paratenic hosts by dogs or cats, larvae will be released and in most cases develop directly into adult worms in the intestinal tract. This may explain the higher Toxocara prevalence rate in adult cats, which catch prey animals more often than dogs. In the pregnant bitch, ‘dormant’ somatic larvae are reactivated and migrate in the bitch across the placenta to infect the fetuses. New-born puppies and kittens also acquire infection through ingestion of larvae in the milk [49, 59, 60].

Environment

Toxocara eggs are unembryonated and not infectious when passed into the environment in the faeces of dogs and cats. Within a period of 3 weeks to several months, depending on soil type and climatic conditions such as temperature and humidity, eggs will develop to an infectious stage that can survive for at least one year under optimal circumstances. Studies from all over the world demonstrated high rates (10-30%) of soil contamination with Toxocara eggs in backyards, sandpits, parks, playgrounds, lake beaches, and other public places [37]. In a survey in the Netherlands, the presence of T. canis eggs in public parks was comparable with reports from other European cities, but most of the investigated sand-boxes were polluted with T. cati eggs [27].

Infection routes

Tracheal migration

After young dogs ingest infective Toxocara eggs, larvae migrate through the liver, the vascular system and the lungs to the trachea. After swelling, they complete their development in the stomach and small intestine. Eggs first appear in the faeces 4 to 5 weeks post-infection [49]. Depending on previous exposure to infection, the migratory pathway and deworming
Toxocarosis, an important zoonosis - P.A.M. Overgaauw, F. van Knippen

bitch during pregnancy. Within hours of birth, the larvae that were present in the liver of the neonate, migrate to the lungs and undergo a tracheal migration. Adult worms can be found at two weeks of age and large numbers of eggs may be passed in the faeces after a minimum period of 16 days [32].

Transmammary transmission

After activation, somatic Toxocara larvae in dogs and cats will also be transmitted via the colostrum and the milk (transmammary transmission, lactogenic or milk-borne infection). Following ingestion by the offspring, the larvae undergo development without tracheal migration. Larvae are found to pass in the bitch’s milk for at least 38 days after parturition [72]. This route is less important than intra-uterine transmission in the puppy, but it is the primary mode of infection in the kitten. Kittens infected by lactogenic transmission will show faecal egg excretion after 7 weeks.

Infection of the dam by the offspring

Infection of the lactating bitch will occur mainly by ingestion of immature fourth-stage larvae from vomit or faeces from the puppies. Larvae develop to adults without a tracheal migration. Toxocara eggs shed in the faeces of puppies or kittens can be ingested by the mother, where they pass through the digestive tract, causing a false-positive diagnosis of Toxocara infection upon faecal examination. There is no development of intestinal infection with T. canis from somatic larvae at other times during gestation and the bitch is not at higher risk of Toxocara infection during metoestrus [44].

Transmission through paratenic hosts

Paratenesis is the mode of infection of some larval nematodes like Toxocara, ensuring its continuing survival by its distribution in prey species [21]. This route of infection exists because of the development of somatic larvae in paratenic hosts, including vertebrates such as rodents and birds or invertebrates such as earthworms and insects (e.g. flies). After ingestion of an infected paratenic host, the larvae develop directly in the intestine. Cats catch and eat more prey animals than dogs and this may be the explanation why higher infection rates are found.

Although the prevalence of T. canis is highest in young dogs, a certain proportion of the adult canine population can also be infected [12, 35, 43, 53], mainly by ingestion of infective Toxocara eggs from contaminated soil.

Somatic migration

After ingestion of infective Toxocara eggs, larvae will migrate actively by penetration of the tissues and invasion of all parts of the body. Gradually somatic larvae accumulate in the tissues, persisting for long periods in a manner similar to that seen in paratenic hosts. Larvae of T. cati prefer to migrate to the muscles, while T. canis larvae were more commonly found in the central nervous system.

Transplacental migration

Nearly 100% of puppies are infected by somatic larvae in utero from day 42 of the gestation [33]. This so called transplacental migration or intra-uterine infection is the most important mode of transmission in dogs. In cats, prenatal infection via the placenta does not occur. The larvae in pregnant bitches are probably reactivated by the changing hormonal status of the bitch during pregnancy.

[12], in pups of one to two months, the probability that newly hatched T. canis larvae will develop into adult ascarids may fall to a lower level, while the probability of somatic migration progressively increases. This is called age resistance and the mechanism operates partly within the lungs. Age resistance seems to be low in older cats. An explanation is not known [69].

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Toxocarosis, an important zoonosis - P.A.M. Overgaauw, F. van Knippen

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and remain infective for years. Since no practical methods exist for reducing environmental egg burdens, prevention of initial contamination of the environment is most important. This can be achieved by taking measures such as eliminating patent infections in dogs and cats, preventing defecation by pets in public areas (Fig. 8), taking hygienic precautions and education of the public [19]. Household garden soil was found to be a potentially greater source of *Toxocara* infection than soil in public green areas [25]. A decrease in contamination can be achieved by methods including: restriction of uncontrolled dogs and cats, cleaning up faeces from soil and on pavements by dog owners, preventing access of dogs and cats to public places (especially children’s playgrounds) and by use of strategic anthelmintic treatment of dogs and cats with emphasis on worming puppies, kittens, nursing bitches and queens.

**Clinical symptoms**

The clinical symptoms depend on the age of the animal and on the number, location and stage of development of the worms. *Toxocara* infection is highest in puppies and kittens up to 6 months of age. After birth, puppies can suffer from pneumonia associated with the tracheal migration and die within 2 to 3 days. At an age of 2 to 3 weeks, puppies can show emaciation and digestive disturbances, caused by mature worms in the stomach and intestine (Fig. 6 and 7). Diarrhoea, constipation, vomiting, coughing and nasal discharge can be found at clinical examination. Distension of the abdomen (‘potbelly’) can occur, probably as result of gas formation caused by dysbacteriosis [41].

Kittens are older when worms are maturing (adult from day 28 and egg producing from day 49 after birth) and tracheal migration with related symptoms does not occur. Therefore, kittens have a better chance to grow and in the meantime develop better bodily condition before problems may be seen. For this reason clinical symptoms similar to those in puppies are usually inapparent.

**Diagnosis**

*Patent* *Toxocara* infection in dogs and cats can be tentatively diagnosed from the medical history, particularly the use or otherwise of an appropriate anthelmintic schedule, and the clinical symptoms. Confirmation of the diagnosis can be obtained by finding dark brown coloured eggs with thick pitted shells in faecal samples (Fig. 1). The direct faecal smear technique is not a sensitive test and generally should never be used for recovering eggs from faecal samples. Examination of faeces by a floatation technique is a useful method for detecting helminths [7].

**Control measures**

There are two reasons for *Toxocara* control: to prevent human infection and to reduce the risk of infection to pets. *Toxocara* eggs are very resistant to adverse environmental conditions and remain infective for years. Since no practical methods exist for reducing environmental egg burdens, prevention of initial contamination of the environment is most important. This can be achieved by taking measures such as eliminating patent infections in dogs and cats, preventing defecation by pets in public areas (Fig. 8), taking hygienic precautions and education of the public [19]. Household garden soil was found to be a potentially greater source of *Toxocara* infection than soil in public green areas [25]. A decrease in contamination can be achieved by methods including: restriction of uncontrolled dogs and cats, cleaning up faeces from soil and on pavements by dog owners, preventing access of dogs and cats to public places (especially children’s playgrounds) and by use of strategic anthelmintic treatment of dogs and cats with emphasis on worming puppies, kittens, nursing bitches and queens.

**Anthelmintic treatment strategy**

The most serious and concentrated sources of infection are found in bitches nursing a litter and puppies aged between 3 weeks and 6 months. A major aim of long-term prophylactic treatment programmes is to suppress *T. canis* egg-output throughout the whole of puppyhood using a multidose schedule. Puppies should be treated with appropriate anthelmintics at the age of 2 weeks and because milk transmission occurs continuously for at least 5 weeks post partum, repeated treatments are necessary [3]. Larvae that reach the intestine need at least 2 weeks to mature and start passing eggs, therefore the treatment should be repeated every 14 days. The treatment schedule therefore requires deworming at 4, 6 and 8 weeks of age and then monthly until 6 months of age. Because prenatal infection does not occur in kittens, fortnightly treatment can begin at 3 weeks of age. Nursing bitches and queens should be treated concurrently with their offspring since they may develop patent infections along with their young.

Control in older dogs and cats can be achieved by periodic treatments of dogs and cats with anthelmintics, or by treatments prescribed based on the results of periodic diagnostic faecal examinations. Annual or twice annual treatments have been shown not to have a significant impact on preventing patent infection within a population, so a treatment frequency of at least 4 times
Toxocarosis, an important zoonosis - P.A.M. Overgaauw, F. van Knapen

Owners should routinely collect and safely dispose of their pets’ faeces, especially from children’s play areas and finally advice for children not to play in potentially contaminated environments. Although veterinarians should be the most appropriate sources of information for their clients regarding the dangers and the control of toxocarosis, surveys have demonstrated that client education on this issue is lacking [23, 42].

Human toxocarosis

Infection routes

Toxocarosis is a public health problem. Man acts as an unnatural host in which Toxocara larvae will not develop but will migrate and survive for a long time. The mode of transmission to humans is by oral ingestion of infective Toxocara eggs from contaminated soil (sapro-zoonosis), from unwashed hands or consumption of raw vegetables [19]. Some infections may occur from ingestion of larvae in under-cooked organ and muscle tissue of infected paratenic hosts such as chickens, cattle and sheep [2, 38, 55, 61, 62].

Recently, studies indicate the fur of as an important source of Toxocara eggs for infection after direct contact [1, 54, 71]. Direct contact with dogs and cats that harbour a patent Toxocara infection is usually not considered a risk, since the eggs need to mature several weeks before they are infective [41, 45, 46]. Moreover, Toxocara eggs are very sticky and therefore difficult to remove from the coat of a dog or cat and this makes ingestion of a sufficient number of eggs unlikely. A low percentage of these eggs were embryonated in the studies and even in the worst case scenario of highly contaminated fur, it is necessary to ingest several grams of heavily contaminated hair to get infected [48].

The importance of Toxocara cati in human toxocarosis

The role of T. cati as a zoonotic parasite is not always clearly recognised. Despite the fact that differentiation between T. canis and T. cati infections is not performed in surveys, the majority of reported human cases of toxocarosis in the past have been associated with T. canis and not with T. cati [13]. The greater

Fig. 6 Patent Toxocara infection resulting in death of 6 months old Border Collie (Photo Paul Overgaauw).

Fig. 7 T. canis adult worms in intestine (Photo RVC).
serum IgE concentration, the presence of allergen-specific IgE and eosinophilia is established. The occurrence of asthma or recurrent bronchitis and hospitalization due to asthma were significantly related to seroprevalence, while eczema tended to be more frequent. It was concluded that allergic phenomena in children who are predisposed to asthma, are more frequently manifested after Toxocara infection [4]. Toxocara is not a causative agent but contributes to the development of atopic diseases and the allergic manifestation of asthma [51].

General clinical symptoms often include malaise, fever, abdominal complaints (vague upper abdominal discomfort attributed to hepatomegaly), wheezing or coughing [17]. Toxocara infection should be considered in the differential diagnosis of any child with a persistent and unexplained eosinophilia or recurrent abdominal pain. Chronic ‘idiopathic’ urticaria, chronic pruritus, and miscellaneous eczema in adults and children are found strongly associated with toxocarosis [16, 50].

Severe clinical symptoms are reported including life-threatening pneumonia after massive infection [28], eosinophilic meningoencephalitis in children [36], and thrombosis of the aorta [67].

Infection risk to children
Children are more frequently infected than adults and VLM with more severe clinical symptoms is mainly found in children of 1 to 3 years of age. This can be explained because young children play and have closer contact with potentially contaminated soil in yards and sand-pits. In addition, children may often put their fingers into their mouth and sometimes eat dirt.

Clinical symptoms

Visceral larva migrans
After ingestion of infective Toxocara eggs by a human, Toxocara larvae hatch in the stomach and migrate into the mucosa of the upper small intestine and disperse throughout the body via blood and lymphatic vessels. A more marked, inflammatory, immune response is called visceral larva migrans syndrome or VLM. This multisystem invasion can be associated with varied, non-specific clinical symptoms as a result of the host’s immune response.

VLM is mainly diagnosed in children between 1 to 7 years of age (mean age 2 years) and is characterised by persistent eosinophilia, leukocytosis, an elevated GTγ level and hypergamma-globulinaemia [56].

Eosinophilia is seen more often in children than in adults [68] and a relationship between Toxocara seroprevalence and the incidence of chronic airway disorder (asthma), elevation of serum IgE concentration, the presence of allergen-specific IgE and eosinophilia is established. The occurrence of asthma or recurrent bronchitis and hospitalization due to asthma were significantly related to seroprevalence, while eczema tended to be more frequent. It was concluded that allergic phenomena in children who are predisposed to asthma, are more frequently manifested after Toxocara infection [4]. Toxocara is not a causative agent but contributes to the development of atopic diseases and the allergic manifestation of asthma [51].

Ocular larva migrans
Migrating Toxocara larvae can induce granulomatous retinal lesions, which are characterised by complaints of loss of visual acuity, squint and ‘seeing lights’ (Fig. 9). This is called Ocular Larva Migrans syndrome (OLM) [6, 63]. In a minority of cases, total blindness of one or both eyes can result. The mean age of patients with OLM is 8 years, but it is diagnosed in adults as well [31, 56]. Ocular larva migrans is usually caused by no more than a single larva.

Covert toxocarosis
A third clinical syndrome, called ‘covert toxocariasis’ (CT), was found in patients with a vague complex of non-specific clinical symptoms, which do not fall within the categories of VLM or OLM. Symptoms such as hepatomegaly, cough, sleep disturbances, abdominal pain, headaches and behavioural changes have been associated with raised Toxocara antibodies.
The diagnosis ‘Idiopathic Abdominal Pain of Childhood’ is usually made in children.

**Cerebral toxocarosis**

There are some indications that larval involvement in the human brain may have subtle public health implications, such as changes to cognitive function in children [26]. A large study to determine OLM in 120,000 Irish schoolchildren, ranging in age from 3 to 19 years, revealed a strong association between having had a convulsion and ocular toxocarosis [20].

**Diagnosis**

Direct diagnosis of *Toxocara* infection is not easy because patients do not excrete parasite material such as eggs or larvae. Serodiagnostic techniques (ELISA) are the most reliable tools to detect antibodies and circulating antigens [58]. Anti-*Toxocara* antibodies measured by ELISA were found to persist for up to 2.8 years in infected adults and their presence alone does not distinguish between current and past infections. It should therefore be accompanied by other laboratory tests for a blood eosinophil count and total serum IgE [34]. Seroprevalence for *Toxocara* in some reports varied between 4.6 to 7.3% in children in the USA [24], 2.5% in Germany to 83% for children in the Caribbean [65]. In the Netherlands the prevalence was found to be 19% on average: between 4% and 15% in people younger than 30 years and 30% in adults older than 45 years [5]. Regularly re-infection of adults is probably the cause of the higher prevalence. Titres fall gradually over a period of about three years but should be considered as a balance between the fading memory of the immune system and its stimulation by continuing ingestion of viable ova or reactivation of dormant larvae. A review of cases of toxocarosis (VLM and OLM) from all over the world revealed that more than half of the patients were less than three years old, one fifth were adults and 60% were males [9]. For OLM, serum antibodies are not diagnostic but the presence of intraocular antibodies appears more promising as a diagnostic aid [63].

On CT or MR imaging, hepatic lesions may be seen as multiple, ill-defined, oval lesions that measure 1.0-1.5 cm in diameter. On sonography, the lesions appear as multiple, small, hypoechoic lesions in the liver parenchyma [30]. Magnetic resonance imaging (MRI) can be used in patients with neurological syndromes to detect granulomas located cortically or subcortically [34].

**Control measures**

**Preventive measures**

A zoonotic disease like toxocarosis can be prevented for the most part. Control is important from the point of view of welfare, for the quality of human life and also for the economic costs to society [66]. Prevention of toxocarosis is possible by the institution of certain measures: appropriate health care for pets including regular anthelmintic treatments; reducing the number of uncontrolled and stray pets; preventing contamination of the environment with faeces; and promoting responsible pet ownership [40, 57, 70].

To increase the awareness of potential zoonotic hazards, particularly amongst pet owners, veterinary practitioners, general practitioners and public health agencies should provide sufficient information and advice for appropriate measures to be taken to minimize the risk of infection. Several reports, however, have indicated a significant lack of knowledge within the professions [23, 42, 47]. All authors concluded that continuing education with emphasis on the zoonotic risks is still strongly recommended.

Recommendations include the following: be careful when in contact with young dogs and cats; wash hands before eating and after contact with animals; deworm dogs and cats regularly especially puppies and kittens; prevent children from eating earth and from playing on areas soiled with animal faeces; remove pet faeces; keep children’s nails clipped.

**Treatment of patients**

Patients with severe *Toxocara* infections can be treated with systemic acting and larvicidal anthelmintics [34]. Clinicians, however, should balance the risk of therapy with the severity of the disease, because treatment can lead to severe hypersensitivity reactions caused by dying larvae. Especially in OLM cases the anthelmintic dose should be increased gradually over a period of days and accompanied by the concomitant administration of steroids [34].

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