Toxocariasis

Toxocarosis,
Visceral Larva Migrans,
Ocular Larva Migrans,
Larval Granulomatosis,
Toxocaral Retinitis

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Importance

Members of the genus *Toxocara* are zoonotic intestinal nematodes (roundworms) that mature in various mammals, including some domesticated species. Parasitized animals can shed large numbers of eggs in the feces, infecting people (particularly children) who ingest these eggs in contaminated soil, or on hands or objects. Although *Toxocara* eggs do not complete their maturation in humans, the developing larvae can migrate through the body for a time. In some cases, they cause symptoms ranging from mild, vague discomfort to ocular disturbances, blindness and neurological syndromes. Human toxocariasis is one of the most common helminth infections in the world, with children living in poverty at the highest risk of infection. In some areas, this disease may also be important in adults who eat undercooked animal tissues containing larvae.

Human toxocariasis is mainly attributed to *Toxocara canis* and *T. cati,* the major roundworm species found in dogs and cats, but other *Toxocara* may also be involved. In particular, the importance of *T. malaysiensis*, a recently recognized species in cats, and *T. vitulorum*, a parasite of cattle and water buffalo, remain to be clarified. In puppies and kittens, *Toxocara* infections can be associated with unthriftiness, diarrhea and poor growth, and in severe cases, may result in death. *T. vitulorum* in bovine or buffalo calves may, similarly, lead to illness, economic losses and increased mortality.

Etiology

Toxocariasis is caused by members of the genus *Toxocara*, nematodes in the family Toxocaridae, superfamily Ascaridoidea. Recognized species include *Toxocara canis*, *T. cati, T. malaysiensis* and *T. vitulorum,* which have domesticated animals as their definitive hosts; and *T. tanuki*, *T. pteropodis*, *T. apodemi*, *T. lyncus*, *T. mackerrasae*, *T. paradoxura*, *T. sprenti and T. vajrasthirae*, which mature and shed eggs in wild animals.

Adult *Toxocara* can cause intestinal illnesses in their definitive hosts, while larvae migrating through the tissues can affect both definitive and paratenic hosts. In paratenic hosts, toxocariasis is often called larva migrans. Visceral larva migrans is a general term used to indicate the presence of larvae in various internal organs, while larvae in the eye are termed ocular larva migrans. Larvae in the brain are sometimes called cerebral (or neural) larva migrans.

Species Affected

Dogs, wild canids and hyaenids are the definitive hosts for *T. canis.* Cats and other felids are the definitive hosts for *T. cati. T. cati* is generally not thought to mature in the intestines of canids, or *T. canis* in cats. Nevertheless, there are a few definitive reports of *T. cati* eggs in canine feces, which could be explained by coprophagy, and *T. canis* eggs in the feces of cats. *T. malaysiensis* has, to date, only been found in the domestic cat.

Water buffalo (*Bubalus bubalis*), cattle and related species, such as yaks (*Bos grunniens, Bos mutus*) and gayal (*Bos frontalis*), are thought to be the primary definitive hosts for *T. vitulorum*. This parasite has also been reported occasionally in sheep, bison (*Bison bison*) and goats. *T. lyncus* occurs in caracals, *T. pteropodis* in fruit bats, *T. apodemi* and *T. mackerrasae* in rodents, *T. paradoxura* and *T. sprenti* in viverrids and *T. vajrasthirae* in mustelids. *T. tanuki* has been detected mainly in raccoon dogs (*Nyctereutes procyonoides*), which are canids, but it has also been found in raccoons (*Procyon lotor*).

Domesticated and wild mammals, birds and some invertebrates (e.g., earthworms, the land snail *Rumina decollata*) serve as paratenic hosts for *Toxocara* spp. Reports of infected birds have included domesticated species such as chickens, quail, ostrich and ducks.

Zoonotic potential

Toxocariasis/larva migrans occurs in humans acting as paratenic hosts for migrating larvae. Most cases are thought to be caused by *T. canis* and *T. cati*.

T. vitulorum is postulated to be a minor cause of larva migrans, mainly affecting children, in the tropics. *T. malaysiensis* might also be zoonotic. One report suggested that *T. pteropodis* may have caused an outbreak of hepatitis associated with feces-contaminated fruit in Australia, but other authors consider this to be doubtful. There is currently no evidence for the involvement of other *Toxocara* species in human illness, but whether this is due to limited exposure, the lack of definitive parasite identification in most clinical cases or other factors is not known.

Toxocara are generally thought to be incapable of maturing to adult nematodes in humans. Although there are rare reports of intestinal infections with adult *T. canis* and *T. cati*, the accuracy of some of these diagnoses has been questioned. Some cases were later identified as immature *Ascaris* worms rather than *Toxocara*, and others may have resulted when young children ingested whole worms that had been expelled by pets.

Geographic Distribution

T. canis and *T. cati* are cosmopolitan, but they are more common in some areas than others. *T. malaysiensis* has been described in Malaysia, China and Vietnam, and may occur in other regions, especially in Asia. *T. vitulorum* has been documented mainly in tropical and subtropical areas, but it has also been reported infrequently from temperate parts of Europe, Australia, North America and Japan. Relatively little is known about the distribution of the various *Toxocara* species found in wildlife; however, all reports of *T. tanuki*, to date, are from Japan.

Transmission and Life Cycle

Toxocara canis in canids

Mature *T. canis* nematodes live in the intestines of canids and hyaenids, the definitive hosts, and produce large numbers of unembryonated eggs, which are excreted in the feces. These eggs are not immediately infectious; they develop to the infective stage, containing third stage larvae, in the environment. Survival and development are affected by both temperature and the availability of moisture. Under laboratory conditions, some eggs can reach the infective stage as quickly as 5-9 days at optimum temperatures (25-30ºC; 77-86°F) and humidity. In most natural environments, embryonation usually takes 3 weeks to several months. Colder temperatures delay development, and larvae die when soil temperatures are below -15°C (5°F). Survival and development are also inhibited by very dry conditions, and high temperatures may result in loss of viability. Although many eggs may no longer be viable after 6 months, some can survive in the soil for a year or more.

Dogs can become infected by ingesting embryonated eggs from the environment, or larvae in their dam's milk or the tissues of paratenic hosts. Canine fetuses can also be infected by migratory larvae before birth. The paths that *T. canis* larvae take after different routes of exposure are still incompletely understood, but seem to be influenced by the animal's age and immunity. If a very young puppy (e.g., < 4-5 weeks of age) eats embryonated eggs, the larvae hatch in the intestines, penetrate the intestinal wall, and migrate through the liver to the lungs, where they enter the alveoli and travel up the airways. Larvae in the pharynx are swallowed. When the parasites reach the intestines a second time, they develop into adults, mate and release eggs. When older puppies and adult dogs ingest eggs, some of the larvae may fail to penetrate the intestinal wall, possibly due to local immunity, and some others penetrate but fail to complete the migration from the lungs to the intestines. Instead, they travel to the muscles, kidneys, liver and other viscera, where they eventually become dormant 'hypobiotic' larvae. Experiments suggest that adult dogs are more likely to develop a patent infection if they only eat small numbers of eggs, possibly because they stimulate a less intense immune response. Dogs can also become infected by eating dormant larvae in the tissues of paratenic hosts. Some experiments have suggested that these larvae may mature in the dog's intestines without further migration; however, migration did occur in other studies. The prepatent period after ingesting *T. canis* eggs is usually 4 to 5 weeks in puppies (with a similar period reported in red fox cubs, *Vulpes vulpes*), but it can be as long as 8 weeks in older dogs. Most of the adult parasites in the intestines are expelled within 6 months.

In a pregnant dog, hypobiotic larvae become reactivated during the last third of the pregnancy and many of them enter the uterus or mammary gland. This can occur repeatedly during each pregnancy, without reinfection. Some of the migrating larvae enter the fetal liver, where they remain until birth. After the puppy is born, they complete their migration through the lungs, and develop into adults. Puppies infected *in utero* begin to shed eggs after approximately 3-4 weeks. Puppies can also be infected from milk during the first few weeks after birth. As with larvae from paratenic hosts, most of these larvae may complete their development in the intestines, without tissue migration. Some bitches develop patent infections during lactation, either from the movement of hypobiotic larvae to their intestines or from exposure to their puppies' feces. These infections disappear spontaneously 4 to 10 weeks after parturition.

Toxocara cati in cats

Cats can develop patent infections after ingesting embryonated *T. cati* eggs from the environment, and larvae from paratenic hosts or their dam's milk. The life cycle of this parasite is thought to resemble that of *T. canis* in dogs, and fewer larvae complete tracheal migration in adult cats than in kittens. However, hypobiotic larvae occur mainly in the muscles of cats, and do not appear to contribute to vertical transmission. *T. cati* is not thought to be transmitted *in utero,* and larvae are shed in milk or colostrum only if the queen is acutely infected in late pregnancy. Kittens can also be infected via paratenic hosts or eggs from the environment. The prepatent period is approximately 6.5 weeks after eating larvae in milk or the tissues of paratenic hosts, and 8 weeks after ingesting eggs.

Toxocara vitulorum in ruminants

The life cycle of *T. vitulorum* is similar to other *Toxocara* species; however, mature *T. vitulorum* are usually found only in the duodenum of 3- to 10-week-old calves, which become infected with larvae while nursing. When a pregnant cow ingests embryonated eggs, the larvae do not mature. Instead, they migrate through the liver, lung, other viscera and muscle, as well as the mammary gland, where they enter the milk and colostrum (although water buffalo colostrum seems to contain very few larvae). Hypobiotic larvae also become reactivated and can enter the mammary gland for up to 3 pregnancies. The greatest numbers of larvae occur in milk during the first week after calving, but small numbers can be found up to 18 days. The prepatent period for *T. vitulorum* in calves is usually 21 to 28 days. Most calves stop shedding eggs by the time they are 2-4 months old, but a minority may continue up to 6 months of age. *In utero* transmission of *T. vitulorum* is thought to be insignificant or absent. In a recent field study from Laos, however, eggs were found in the feces of 17% of calves too young to be infected in milk. This finding remains to be confirmed, as other possibilities (e.g., coprophagia or inaccurate age estimates for the calves) could not be ruled out.

In the laboratory, *T. vitulorum* eggs can develop to the infective stage in 7 to 12 days at 28-30°C (82-86°F). Embryonated eggs have been shown to survive in the environment for several months and possibly up to 2 years.

Toxocara infections in paratenic hosts including humans

Embryonated *Toxocara* eggs will release their larvae in the intestines of most mammals, and in birds and some invertebrates. In species other than the definitive host, the larvae do not complete their migration into the intestines. Instead, they continue to migrate through the tissues after penetrating the intestinal wall. Eventually, most or all probably become encapsulated as hypobiotic larvae. How long these larvae can persist in different hosts is incompletely understood. Dormant larvae have been found for at least 9 years in experimentally infected macaques, and small rodents can be infected for life. However, hypobiotic larvae did not seem to persist long-term in experimentally infected pigs. Little is known yet about the potential for vertical transmission in paratenic hosts; however, mice were recently found to transmit larvae to their offspring in milk, and less frequently, *in utero*.

Larvae can be transmitted between paratenic hosts, with no growth or development, if the tissues where they reside are eaten. In an early study, some *T. canis* larvae remained motile for several weeks in frozen mouse carcasses. In recent studies, temperatures of 0-4°C (32-39ºF) reduced the number of viable larvae in mouse liver (*T. canis*) or chicken meat (*T. cati*), but did not eliminate them, while freezing at -20ºC (-4ºF) or -25ºC (-13ºF) appeared to kill the larvae.

Humans can be infected by ingesting embryonated eggs, typically from dirt (e.g., on unwashed hands) or in contaminated food or water; or by eating larvae in raw or undercooked tissues of other paratenic hosts. Uncooked liver seems to be a particularly common source of parasites. Embryonated *T. canis* or *T. cati* eggs on the fur of dogs and cats could also represent a source of infection for people. However, some authors consider the risk to be low, as the vast majority of these eggs do not seem to be embryonated, and *Toxocara* eggs also adhere strongly to the fur. Some researchers have suggested that people might become infected by drinking *T. vitulorum* larvae in unpasteurized milk, although others consider this to be unlikely. In addition, there are a few reports of infections probably acquired from unusual sources, such as eating raw snails or earthworms. One instance of apparent congenital transmission, with ocular toxoplasmosis in a premature neonate, was reported in 2006.

Disinfection

Like other ascarid eggs, *Toxocara* eggs are very resistant to chemical disinfectants, and environmental disinfection is considered to be unreliable. The effects of various chemicals may differ depending on whether or not the eggs are embryonated. Cresol or phenol disinfectants are thought to be among the more effective disinfectants for the eggs of other ascarids (e.g., *Ascaris suum*). In one experiment, the larvae inside embryonated *Toxocara* eggs were completely inactivated when they were immersed in aqueous iodine (2.5-10%) for 40 minutes to several hours, depending on the concentration of iodine, However, immersion in povidone iodine (1% iodine) for 2 hours had no effect on nonembryonated eggs of *Ascaris suum,* suggesting that this concentration may also be ineffective against nonembryonated *Toxocara* eggs. Nonembryonated *Toxocara* eggs were destroyed by immersion in 70% ethanol for approximately 1 week, or 2% sodium hypochlorite for more than 2 weeks. Some commercial disinfectants (including the current formulation of Lysol™, which no longer contains phenol or cresol) had little or no effect on ascarid eggs, even after exposure for 2-3 weeks. Physical means, such as ultraviolet light (direct sunlight), prolonged drying, and high temperatures (e.g., 37°C [99°F] under arid conditions) may be effective, given sufficient exposure. Although conditions to inactivate ascarid eggs in sewage have been published, they are not always effective in practice. *Toxocara* eggs can also survive composting.

Infections in Animals

Incubation Period

Puppies infected *in utero* can develop clinical signs associated with tissue migration within a few days of birth, and enteric signs within the first 2 to 3 weeks of life. In kittens, *T. cati* begins to mature in the intestines starting 4 weeks after birth. In experimentally infected calves, the incubation period for *T. vitulorum* ranged from 8 to 21 days.

Clinical Signs

Toxocara canis in dogs

Intestinal *T. canis* nematodes are usually symptomatic only in puppies, especially when they are young. The clinical signs can include a reduced growth rate, loss of condition and sometimes an enlarged abdomen (potbelly). There may also be diarrhea, constipation, vomiting and flatulence, as well as signs associated with larval passage through the lungs and other tissues. Worms are sometimes passed in the feces or vomited. Chronic enteritis can result in the thickening of the intestinal walls or even intussusception. Other uncommon but serious complications include obstruction of the gall bladder, bile duct or pancreatic duct, or rupture of the intestine and peritonitis. Intestinal infections with small numbers of parasites can be subclinical.

Larvae migrating through the liver and lungs can cause inflammation and respiratory signs of varying severity. Pneumonia can develop rapidly in some puppies that were infected *in utero,* and they may die within 2 or 3 days after they were born. Severe infections can also result in ascites, anemia, fatty degeneration of the liver, or rare complications involving other organs, such as myocarditis.

Symptomatic infections are rare in adult dogs. However, high levels of liver enzymes may be seen during larval migration, and ocular signs, including orbital cellulitis and multifocal retinal disease, have been described.

Toxocara cati in cats

Because kittens are not infected *in utero*, they are more mature when the parasite burden becomes heavy, and the clinical signs tend to be less severe than in puppies. Many infections are subclinical. In more severe cases, there may be abdominal distension, a rough coat, diarrhea, vomiting, anemia, anorexia and possibly dehydration.

Some experimentally infected older kittens and cats that were fed embryonated eggs had pathological and radiological evidence of pulmonary disease, without clinical signs, during larval migration.

Toxocara vitulorum in ruminants

Clinical signs that have been reported in naturally infected calves include anorexia, signs of abdominal pain, diarrhea of varying severity, constipation, dehydration, steatorrhea, unthriftiness, weight loss or poor weight gain, poor hair coat, and a butyric odor on the breath. Coughing has been described in experimentally infected calves. Uncommon sequelae may include intestinal obstruction, volvulus or perforation and intussusception, and some infections can be fatal. Subclinical cases have also been described, and may be common in some herds, particularly in calves that are otherwise healthy and well-fed.

In adult cattle and water buffalo, moderate experimental infections, probably comparable to natural infections, are asymptomatic. Feeding large doses of eggs can cause fever, diarrhea and coughing, while very large doses have resulted in paralysis, conjunctivitis and opisthotonos.

Toxocara in paratenic hosts

There are few reports of larva migrans syndromes in animal paratenic hosts. Disseminated granulomatous disease due to *T. canis* was reported in a cat with a 19-day history of fever but no other clinical signs. Neurological and/or ocular signs have been documented in some experimentally infected mice and nonhuman primates, and infections in mice can be fatal. Cats experimentally infected with *T. canis*, pigs inoculated with *T. canis*, and chickens inoculated with *T. canis* or *T. cati* had gross lesions at necropsy, but remained asymptomatic.

Post Mortem Lesions   📑 **Click to view images**

Lesions caused by intestinal worms

The most obvious finding in enteric disease is the presence of nematodes in the intestinal lumen. There may also be mucoid enteritis, thickening of the intestinal walls, or complications such as intussusception, obstruction of the gall bladder, bile duct or pancreatic duct, intestinal perforations, peritonitis or blood loss into the peritoneal cavity.

Lesions caused by Toxocara larvae

Migrating larvae can result in petechial hemorrhages and multifocal, circumscribed white to gray foci (eosinophilic granulomas or accumulations of inflammatory cells) during their migration through the lungs. More extensive hemorrhages, congestion and/or evidence of inflammation may be seen in some animals. Larvae can sometimes be detected in the pleural cavity and diaphragm. Lesions such as interstitial pneumonitis, eosinophilic arteritis and bronchiolitis, and hypertrophy and hyperplasia of the pulmonary arteries may be apparent on histopathological examination. Secondary bacterial pneumonia can also be present, especially in young puppies. White to gray foci and hemorrhagic (e.g., petechial) lesions may also be found in other organs, particularly the liver, and there may be other evidence of tissue damage (e.g., edema, areas of necrosis or fibrosis, ascites). Granulomas containing larvae are occasionally found in the renal cortex of young dogs, often as an incidental finding. Ocular lesions including retinal disease and orbital cellulitis have been described.

Diagnostic Tests

Patent infections in the definitive host can be diagnosed by detecting eggs with various fecal flotation techniques or other assays. In adult dogs, eggs may be excreted intermittently or sporadically. In fresh fecal samples, *Toxocara* eggs (approximately 85 µm x 75 µm) contain a single dense cell mass within a thick, brown outer shell. The shell contains a distinctive finely stippled, brownish-yellow, proteinaceous coat, best detected by moving the fine adjustment on the microscope. Eggs with an aberrant shape, size, or coat may be found. Although certain morphological criteria may suggest a particular *Toxocara* species, morphologies overlap. Definitive identification requires more specific tests, such as PCR, which is available mainly in research laboratories. Some infections can be missed, due

the presence of only immature worms or small numbers of worms. Conversely, false positive tests are possible in dogs, due to coprophagy. Immature worms may occasionally be voided in feces or vomitus.

Treatment

Anthelmintics eliminate worms in the intestines, but their efficacy against larvae in different hosts, at different body sites, or at different stages of larval development, is still incompletely understood. Hypobiotic larvae are thought to be difficult to kill in dogs and cats.

Control

Disease reporting

Veterinarians who encounter or suspect toxocariasis should follow their national and/or local guidelines for disease reporting. This disease is not usually reportable in dogs and cats.

Prevention

Routine treatment of puppies, kittens, and nursing dogs and cats is recommended to reduce worm burdens and egg shedding, and prevent illness in young animals. Prenatal treatment regimens may decrease vertical transmission in dogs and cats; however, their efficacy may vary, and some programs are impractical for routine use. Patent infections in older dogs and cats, including adults, should be eliminated by treatment based on fecal examination, or by periodic treatment with monthly preventives or other drugs. Animals allowed to hunt or eat raw tissues have a higher risk of becoming infected. Because patent infections with *T. vitulorum* occur only in 3- to 10-week-old calves, anthelmintic treatments in ruminants are targeted to young animals. A single dose of anthelmintic, given when calves are 14–21 days old, can control *T. vitulorum* and reduce environmental contamination with eggs. Other regimens, which may include multiple treatments, have also been described.

Prompt elimination of feces can help reduce environmental contamination. In kennels, removal of feces should be followed by thorough cleaning. High pressure steam cleaning, followed by a disinfectant that has efficacy against ascarid eggs (e.g., cresols) is recommended by some authors. A solution of 1% sodium hydroxide has also been suggested, as an aid to the removal of *Toxocara* eggs; the sodium hydroxide removes the sticky outer protein coat and makes the eggs easier to remove. However, it does not kill the developing larvae, and these decorticated eggs are more infective than eggs with an intact protein coat. There is no practical method to remove *Toxocara* eggs from soil once contamination has occurred.

Morbidity and Mortality

Toxocara canis and Toxocara cati

The probability that an animal will have a patent intestinal infection varies with its age, role (e.g., pet, working dog, shelter animal or stray) and type of environment.

Infections are most common in young animals up to a year of age, especially those less than 6 months. In dogs, maternal transmission is very efficient: nearly all puppies born to infected bitches are infected. In older dogs and cats, animals allowed to eat paratenic hosts (e.g., cats allowed to roam freely) are more likely to acquire *Toxocara*. There is still limited information about recurrent patent infections in adult dogs, under natural conditions. However, one study from the Netherlands found that repeated infections tended to occur in a minority of dogs not treated with preventive anthelmintics (15%), and some risk factors (e.g., corticosteroid use) suggested that impaired immunity might play a role in susceptibility. Most of the dogs in this study (68%) never shed eggs, and 17.5% had only a single patent infection.

Worldwide, most published reports of *T. canis* and *T. cati* prevalence in dogs and cats range from approximately 1% to 50-60%, with a few studies showing higher prevalence (up to 79% in dogs and 85% in cats), particularly when the study population includes large numbers of young animals and/or strays. The number of infected animals is generally higher in warm, humid areas than in regions where conditions are less favorable for survival and embryonation of the eggs (e.g., hot, dry, sandy soils). Prevalence rates of <10-20% are not uncommon among well-cared-for pets in some temperate areas. Some studies have reported that patent infections were seasonal, and for unknown reasons, were more likely to be seen in winter. Significant numbers of wild canids (e.g., up to 50% of red foxes in some parts of Europe) and felids may also be infected with *T. canis* or *T. cati* in some areas. These populations might play a role in parasite maintenance, and urbanized wild animals could contribute to environmental contamination. Little is known about the prevalence of *T. malaysiensis*, but one study found this parasite in 11% of domesticated cats in Kuala Lumpur.

The severity of toxoplasmosis depends on the parasite burden. Puppies can die occasionally from the effects of larval migration (especially pneumonia) and rarely from intestinal complications. Clinical signs in kittens are generally less severe. However, even uncomplicated infections may have a detrimental effect on the animal's health. Young animals that survive the critical period usually recover fully and expel the adult worms by 6 months. Adult dogs and cats are rarely symptomatic even when shedding eggs. However, one recent experiment in cats suggests that migrating larvae might affect lung health or cause other issues.

Toxocara vitulorum

The prevalence of *T. vitulorum* among cattle and water buffalo calves in the tropics ranged from 4% to 48% in several studies conducted between 2000 and 2013. Older studies suggested that prevalence rates may approach 100% among water buffalo calves in some locations. Mortality rates of 30-40%, and up to 80%, have been described in some uncontrolled infections in the field; however, calf mortality is often multifactorial, and it can be difficult to evaluate the contribution of *T. vitulorum* alone. There are also reports of

outbreaks where most infections were subclinical, and some studies have found no clinical signs even in animals with high egg counts.

Infections in Humans

Incubation Period

The incubation period in humans can be weeks to months.

Clinical Signs

Toxocara larvae can cause symptoms related to the internal organs (visceral larva migrans), eye (ocular larva migrans) or brain (cerebral larva migrans), or vague, mild syndromes known as covert or common toxocariasis. Small numbers of larvae are usually asymptomatic.

Most cases of visceral larva migrans are subclinical, and are recognized mainly by persistent eosinophilia. Typical signs in more severely affected patients can include malaise, fever, hepatomegaly and upper abdominal discomfort. Some patients may also have nausea, vomiting, or respiratory signs such as wheezing, coughing and dyspnea. Pruritic rashes, chronic urticaria, hypodermic nodules, lymphadenopathy, arthralgia and myalgia have also been described, and there are rare reports of cardiac involvement. Invasion of the CNS seems to be uncommon, but can result in various syndromes including meningoencephalitis, myelitis, cerebral vasculitis, or signs of a space-occupying lesion. Unusual presentations (e.g., a case in a child characterized solely by polyarthritis) are described occasionally. Deaths are rare but have been seen in cases of CNS disease, severe pneumonia or cardiac involvement.

Invasion of the eye (ocular larva migrans) can result in retinal granulomas, endophthalmitis, and/or various other abnormalities such as uveitis (especially posterior uveitis), iritis or focal iris nodules, vitreous mass or haze, optic neuritis, conjunctivitis, keratitis, hypopyon and cataract. The infection is usually unilateral and caused by a single larva; however, bilateral infections have been reported. Common presenting complaints include leukocoria (white pupils), decreased visual acuity, strabismus, ocular injection, eye pain and "seeing lights." Serious consequences, including retinal detachment, are possible. Loss of vision may be progressive or sudden, and can be permanent. Subclinical infections have sometimes been detected during routine eye examination. Concurrent systemic signs are uncommon.

The covert form, described in children, and similar common form, described in adults, are syndromes where antibodies to *Toxocara* are associated with a few nonspecific systemic or localized symptoms. Abdominal pain appears to be the most common sign, but there may also be hepatomegaly, coughing, sleep disturbances, headaches, behavioral changes, weakness, pruritus, rash and respiratory distress. These symptoms can last for months or years, but often resolve spontaneously.

T. canis has also been implicated as a possible cause of idiopathic epilepsy, and some researchers have suggested that there may be a link between *Toxocara* infections and some allergic conditions or exacerbations of symptoms. However, proving a link between relatively common conditions is difficult, and not all studies have shown an association.

Diagnostic Tests

Human infections can be diagnosed by the clinical signs, ophthalmoscopic examination (in eye disease), and clinical pathology findings, often in conjunction with serology. Eosinophilia is common in patients with visceral larva migrans, but absent in many cases of ocular larva migrans. Enzyme immunoassays (ELISAs) and immunoblotting (Western blotting) are often used to detect antibodies to *Toxocara*. There may be cross-reactivity to *Ascaris lumbricoides*, *Strongyloides* or filarial nematodes in ELISAs, particularly in tropical regions where these infections are more common. An initial ELISA test, confirmed by immunoblotting if positive, offers the highest sensitivity and specificity. Serological tests cannot, however, determine whether the antibodies are to *T. canis* or *T. cati*. Antibodies to *Toxocara* can persist for several years, and serological assays are best used in conjunction with other evidence for a current infection. Some patients with ocular or cerebral larva migrans have low circulating antibody titers. In some of these cases, reactivity to *Toxocara* may be detectable in the aqueous or vitreous fluid of the eye (ocular larva migrans) or cerebrospinal fluid (CSF).

Histopathology can sometimes detect larvae in biopsy or autopsy specimens, but it is not routinely used or recommended. It can be difficult or impossible to identify ascarid larvae in tissues by morphology alone. *Toxocara* infections do not become patent in people; however, the presence of other parasite eggs (e.g., *Ascaris* or *Trichuris*) in the feces suggests that a child has been exposed to feces-contaminated soil and supports the diagnosis.

Treatment

The optimal treatment of larva migrans is incompletely understood, and recommendations for various syndromes can differ between authors. Anthelmintic drugs can be used to destroy at least some of the larvae, but the dying larvae may cause severe hypersensitivity reactions, and anti-inflammatory drugs (e.g., corticosteroids) are often given concurrently. Treatment of ocular disease may include surgery and/or anti-inflammatory drugs to decrease further damage to the eye, with or without anthelmintics. Other therapies such as laser photocoagulation have also been used. Symptomatic treatment alone may be employed in some cases of larva migrans, particularly with syndromes such as covert toxocariasis, where the signs are vague and spontaneous recovery is common. However, some authors recommend treating all cases to reduce the risk of serious complications such as ocular or cerebral disease.

Control

Environmental contamination can be reduced by preventing and/or treating *Toxocara* infections in animals, and by removing feces before the eggs can become embryonated, especially in areas where children play. Dog owners are encouraged to collect their animal's feces in public areas. The feces should be burned, buried, or bagged and disposed of in the trash. Restrictions on uncontrolled dogs and cats, and prevention of animal access to areas such as children's playgrounds may also be helpful. Patent infections are especially prevalent among dogs and cats less than 6 months of age, with 3-week-old to 3-month-old puppies shedding particularly large numbers of *T. canis* eggs. However, the relative contributions of different animal populations, including pet dogs and cats, strays and wild species may differ between locations. There is no practical way to remove eggs from the soil once contamination has occurred.

Good hygiene can help prevent infections or reduce the severity of illness by decreasing the dose of parasite eggs. Hands and raw fruits or vegetables should be washed or peeled before eating. Children should be taught not to eat soil, and to wash their hands after playing with pets or outdoor activities. They should not be allowed to play in areas where animal feces are found. Families may also consider postponing the acquisition of a new pet until children are past the toddler stage.

Tissues from mammals, birds and other potential paratenic hosts should be cooked before eating. Liver seems to be a particularly common source of larvae.

Morbidity and Mortality

Infections with *Toxocara* larvae are thought to be widespread and common in human populations. In serological studies, exposure rates may be as low as 1-20% in some developed countries with temperate climates, and as high as 30-85% in some tropical regions. The prevalence of infection also varies with age, socioeconomic status, sanitation/ hygiene, occupation and other factors. The prevalence of antibodies is generally higher among children than adults, and higher in rural than urban regions, probably due to increased contact with soil.

In most countries, clinical cases tend to occur mainly in children. Symptomatic visceral larva migrans is particularly common between the ages of 1 to 7 years, especially in children who have a history of pica or frequently play in the dirt. The most severe cases are often reported in toddlers 1-3 years of age. Ocular migrans tends to be more common in older children and young adolescents. However, the most significantly affected age groups can differ between countries, most likely due to social factors that influence exposure and its timing during childhood.

Recent studies suggest that a subset of patients develops larva migrans as adults, probably after eating raw or undercooked tissues (e.g., liver) from paratenic hosts. There are some indications that adult patients may comprise the majority of toxocariasis cases in Japan. Adult cases (e.g., with ocular and neurological disease) have also been reported from other Asian countries where this practice is common.

The percentage of infected individuals who develop clinical signs is still unclear; however, many people with antibodies to *Toxocara* have no history of larva migrans. The severity of the symptoms depends on the parasite burden, location of the larvae and duration of the infection. Most cases of visceral larva migrans are asymptomatic or mild and go unnoticed. Fatalities are rare but have occurred in cases with severe pneumonia, cardiac involvement or neurological disease. One report found that 1% of patients with uveitis at a tertiary referral center had ocular larva migrans, and a survey of Irish school-aged children reported 6.6 cases of ocular larva migrans per 100,000 population. Damage to the eye can be permanent.

Internet Resources

Centers for Disease Control and Prevention (CDC)

CDC Guidelines for Veterinarians: Prevention of Zoonotic Transmission of Ascarids and Hookworms of Dogs and Cats

Companion Animal Parasite Council Current Advice on Parasite Control: Intestinal Parasites

The Merck Manual

The Merck Veterinary Manual

Public Health Agency of Canada. Pathogen Safety Data Sheets.

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