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Toxocarosis: An Enigmatic & Neglected Parasitic Zoonoses

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1. INTRODUCTION

Toxocarosis is the zoonotic disease caused by the nematode species of the parasite called *Toxocara canis* or *Toxocara cati*. Dogs and cats acts as their definitive hosts in which parasites live as adults within the lumen of the small intestine. Infection can occur by the host ingesting viable, embryonated eggs from contaminated sources (e.g. soil and earthworms etc.), or they can acquire the infection in- utero (i.e., transplacentally) from the infected mother when she ingests more infective eggs.

Humans are considered as an accidental host, as the parasites cannot completely mature in their body. Instead, the invasive larvae migrate aberrantly for months through different organs until they are overcome by the host inflammatory reaction and die. The larvae can survive in tissues for at least 9 years and possibly, for the life of the host. Infective larvae hatch after ingestion of eggs, but the juvenile stages fail to develop to mature adult worms. Instead, they wander throughout the body for months or up to several years, causing damage to whatever tissue they happen to enter. The ability of a eukaryotic parasite to survive in any mammal for that length of time is unusual. Only a few others have evolved long-term survival strategies; namely, the adult stage of Schistosomes live for 10 to 25 years, the first-stage larva of *Trichinella spiralis* lives for the life span of the host, some species of adult filarial nematodes live 10 to 15 years, and the juvenile stage of most species of tapeworms survive for 5 to 10 years. To accomplish this daunting feat, all of these parasites have acquired unique mechanisms for evading the host's immune system. *Toxocara* spp. is no exception.

2. BRIEF HISTORY

H.C. Wilder was the first to describe toxocarosis in humans, when he published a paper in 1950 describing ocular granulomas in patients thought to have retinoblastomas. Two years later, Beaver and co-workers in 1952 published the presence of *Toxocara* larvae in granulomas removed from patients with symptoms similar to those in Wilder's patients.

3. TOXOCAROSIS IN THE DEFINITIVE HOSTS

The adult worms live in small intestine of definitive hosts, canids and felids, respectively. Female worms may produce up to 200,000 eggs per day. Eggs passed in the feces are not infective and require an incubation period in the soil to embryonate. Swallowing of infective eggs by adult dogs or cats rarely results in the presence of adult Toxocara worms in the digestive tract. The embryonated eggs hatch in the small intestine where the released larvae perforate the wall. They then enter a blood vessel and go through the liver and lungs to the left heart where they are disseminated by the systemic circulation (somatic migration). Eventually, these larvae penetrate through capillary vessels and migrate to surrounding tissues where they may survive for years without undergoing further development ("hypobiosis"). When a bitch becomes pregnant, the dormant larvae can be reactivated by hormonal stimuli and migrate transplacentally to the fetus. This explains why new-born puppies are likely to be infected. In contrast, transplacental migration of larvae does not occur in cats.

The primary route of infection of kittens is by transmammary transmission of larvae that are found in the milk, whereas this type of transmission is less important in dogs. Infective eggs that are ingested by non-canid and non-felid species follow a somatic cycle similar to that described above. This leads to the presence of larvae in tissues where they are potentially infective to predators on these animals. This type of transmission is called "paratenesis". When a dog or a cat preys upon an infected paratenic host, the larvae are liberated from the tissues during the digestion process and then complete their development in the intestinal tract.

The prevalence of infection tends to decrease with increasing age of the animal and is lower in well-cared pet dogs than in stray or pound dogs. This high prevalence together with the high fecundity of *Toxocara*, and the increasing number of pet animals as well, explain the high level of soil contamination with *Toxocara* eggs in parks, playgrounds, and other public places. Toxocarosis in dogs has worldwide distribution. Its prevalence is 39.5% in Pakistan, 31.5% in Spain, 4.3% in China, 17.4% in Brazil, 7.7% in Turkey. In India, several

surveys revealed prevalence in dogs ranging from 4.95-38.13%.

4. TOXOCAROSIS IN HUMANS

Human toxocarosis is primarily a soil transmitted zoonosis. Geophagia or soil eating is a specific type of pica that increases the risk of toxocarosis, especially in children living in homes with puppies that have not been dewormed. Poor personal hygiene as well as consumption of raw vegetables grown in contaminated kitchen gardens may result in chronic low-dose of infection. Less commonly, zoonotic toxocarosis infection is associated with consumption of raw meat from potential paratenic hosts, such chickens, lambs or rabbits.

In humans, after ingestion of embryonated Toxocara eggs, the larvae follow the same somatic route of migration as for paratenic hosts. The hatched larvae have been found in the liver, lungs, heart, eye, and brain. They are often associated with migratory tracks characterized by hemorrhage, necrosis, and inflammation, with eosinophils predominating. Larvae may become encapsulated within granulomas where they are either destroyed or persist in a viable state for many years. In the eye where the migration of a single larva can be observed, the inflammatory response can lead to partial or total retinal detachment with visual loss.

The prevalence of human toxocarosis in tropical regions has been found to be higher than in the temperate regions. The highest seroprevalence ever recorded was in a village of Santa Lucia, West Indies, where the prevalence of 86% in children aged 6 months to 6 years was reported. In India studies on the prevalence of human toxocarosis shows that the children who were in the habit of eating raw vegetables were more prone to infection (36.48%) than those who were not of such (20.31%) and children with the habit of geophagia were more vulnerable to this infection (36.48%). Contact or close association with dogs or presence of pet in house was the high risk for *Toxocara* prevalence and was found significant factor for Toxocarosis. Prevalence of *Toxocara* infection was more in people using water from streams, rivers, ponds and wells than those using water from public supply in piped water.

5. CLINICAL ASPECTS

The dog show signs of unthriftiness, dullness, pale mucous membrane, rough and harsh body coat, emaciation, anaemia, diarrhoea and constipation. The worms absorb the nutrition from what the dog eats and can damage the intestinal lining. The dog reluctant to move, groan when touched or lifted and is either pot-bellied or the abdomen is tucked up. Due to irritation of intestinal mucosa by mature worms vomiting is seen. Cough, nasal discharge, noisy breathing may be seen in pups due to the migration of the larvae through the respiratory system. In pups nervous symptoms are shown known as “ascaris toxaemia”. Death may occur due to obstruction of intestine by the worms and also due to ulceration and perforation of the intestinal wall. Visceral larva migrans (VLM), Ocular larva migrans (OLM), Covert toxocariasis are the three main clinical forms of toxocarosis that are traditionally described. The classic VLM syndrome consists of episodes of fever, coughing and wheezing, anemia, eosinophilia, hepatomegaly, and positive *Toxocara* titers. Skin lesions, such as urticaria and nodules, wheezing to eosinophilic pneumonia and respiratory failure are common signs of VLM that have been described. Ocular larva migrans (OLM) refers to eye (usually retinal) involvement during *Toxocara* infection where the larvae migrate to the eyes, causing eosinophilic inflammatory reaction which is unilateral most of the time. It can be confused and misdiagnosed with a retinoblastoma. Covert toxocarosis refers to asymptomatic form which is less specific syndrome that was recognized with the wider use of serodiagnostic assays for *Toxocara* infection. Eosinophilia is less frequent and less pronounced with this form than with VLM and *Toxocara* antibody titers are lower.

6. DIAGNOSIS

In dogs and cats diagnois can be done with the help of clinical signs and symptoms like pale mucous membrane, pot-bellied or tucked up abdomen etc. Presence of adult worms usually 3-4 inches long upto 7 inches in faeces or in dogs vomit and faecal examination for presence of brown and pitted *Toxocara canis* eggs when observed under microscope.

But the examination of stools has no role in the evaluation of human toxocarosis. Finding *Toxocara* larvae within a patient is the only definitive diagnosis for human toxocarosis, however biopsies to look for second stage larvae in humans are generally not very effective and therefore, cannot be relied**.** Radiology Medical imaging techniques can be used to detect and localize granulomatous lesions due to *Toxocara* larvae. Abdominal ultrasound has shown multiple hypoechoic areas in the liver of children who initially presented with hepatomegaly, eosinophilia and a positive *Toxocara* serology. Using computed tomography (CT), hepatic lesions appear as low-density areas. In the CNS, more sensitive magnetic resonance imaging (MRI) has revealed granulomas appearing as hyper-intense areas on T2-weighted images, primarily located cortically or sub-cortically. In patients with OLM, ultrasound has revealed a highly reflective peripheral mass, vitreous bands or membranes, and traction retinal detachment.

Laboratory investigations are leukocytosis and marked eosinophilia, hypergammaglobulinemia (particularly IgM) and elevated isohemagglutinin titers for antigens in blood groups A and B. Serological testing using immunological techniques such as ELISA with a sensitivity of 75% and specificity more than 90% is recognized as an effective approach. However, inherent lacunae are always associated with test, purity of antigens or the antibody titre level. Molecular techniques, such as DNA hybridization, polymerase chain reaction (PCR) amplification and restriction fragment length polymorphism (RFLP), have been, used to diagnose infections.

A number of diagnostic candidates are being evaluated, which include cathepsin L cysteine proteases (Tc-cpl-1), arginine kinase, O-methylated glycans, surface associated glycoproteins of molecular weights 32,55,70,120 KDa , *Toxocara* excretory secretory antigen (TES-57) , recombinant *Toxocara* excretory secretory antigen (rTES-120, rTES-26, rTES-30USM).

7. TREATMENT

In dogs: (a) Piperazine adipate- Dose of 100mg per kg is highly effective against adult worms. Dose of 200mg/kg b.wt removes immature worms from 1-2 weeks of age puppies (b) Diethyl carbamazine- Dose rate of 50 mg/kg is highly effective in a single dose (c) Pyrantel- Dose rate of 7.5mg/kg b.wt (d) Benzimidazole compounds- dose rate of 10-15 mg/kg (e) Ivermectin- Dose rate of 200 mg (total dose)

In humans albendazole is the treatment of choice for toxocarosis. Patients receiving a 5-day treatment course of albendazole (10 mg/kg of body weight/day in two divided doses) improved relative to patients who received treatment with the older anthelminthic drug thiabendazole. A dose of 400 mg of albendazole twice a day for 5 days is the currently recommended therapy. Because the other commonly used benzimidazole, mebendazole, is poorly absorbed outside the gastrointestinal tract, this agent is a second-line treatment, although some success has been reported in patients who ingest 1 g or more for a 21-day course. Symptomatic treatment, including administration of corticosteroids, has been helpful for suppressing the intense allergic manifestations of the infection. OLM is treated by surgery (vitrectomy), anthelminthic chemotherapy, and/or corticosteroids.

8. CONTROL MEASURES.

What is needed in terms of future control programs is the development of radically new approaches, such as effective molecular or DNA-based vaccines that offer the possibility of lifelong protection. Oral baits laced with vaccine would be ideal for dealing with semi wild dog and cat populations, an approach similar to ones that already exist for the large-scale control of rabies in wild animal populations. To augment vaccine development, rapid, highly sensitive and specific diagnostic tests employing recombinant, antigenic peptides that can be executed in the field are essential elements needed to formulate any future control program. Finally, more effective single-dose treatment regimens with safer (over-the-counter?) drugs for paediatric patients would help limit the time of illness, provided of course that adequate medical infrastructure is already in place. Killing *Toxocara* eggs in contaminated soils is seen by most epidemiologists as a nearly impossible task, but if such a strategy could be found and safely implemented, vast numbers of acres of now potentially dangerous city landscape could be rendered *Toxocara*-free.

Since, free access of pet and stray animals to public places is hazardous, keeping in view public health safety, prompt disposal of animal excreta and control of free access of stray animals to public places is recommended. Pet owners must also be educated for regular deworming and the visitors should be made aware of such soil borne helmintic zoonosis. Preferably guidelines must be provided regarding hygiene and environmental sanitation at places of public health importance. The knowledge of the areas contaminated with *Toxocara* eggs will help in the planning of effective measures to prevent the infection.

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