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A Review of Toxocariasis in man and animal

Abdel-Rahman M.A.M and Soheir M. El-Menyawe

Parasitology Department, Animal Health Research Institute,

Agriculture Research Center, Giza, Egypt.

Summary

Toxocariasis is an important parasitic zoonosis caused by larval stages of Toxocara species. The dog ascarid, *T.canis* is the probable cause of the human toxocariasis although two other species, *T.cati* and *T.vitulorum* are also possible causes of the disease. Larval migration through soft tissues in the humans generates several clinical entities in the patient such as visceral larval migrans (VLM), ocular larval migrans (OLM) and neuro-toxocariasis. VLM is primarily the disease of children who are more likely to ingest eggs of *Toxocara* species. Generally human become infected by ingestion of embryonated eggs either from soil, dirty hands, raw fruits and vegetables or larvae from under cooked meat of paratenic hosts. It is a public health problem appears in variable frequencies depending in factors related to childrens, hygienic and behavioral habitats. The diagnosis and control of *T. vitulorum* is not easy as the larvae migrate in the tissues, remaining as dormant or hypobiotic parasites. These issues are summarized in this review, with emphasis on the history, epidemiology, clinical signs, molecular aspects, treatment and control of spread to both humans and animals. The preventive measures required through public health initiatives, employing treatment of pets and environmental intervention strategies that limit the areas where dogs and cats are allowed within the confines of urban centers.

Introduction

Toxocariasis is a highly prevalent zoonotic parasitic infection world-wide that affects dogs and cats as well as cattle and water buffaloes. A wide range of animals, including mice, rabbits, monkeys and humans, act as paratenic hosts **(Lescano et al., 2004).** It is caused primarily by *Toxocara canis*, in addition to *T. cati*, *T. vitulorum* and *Toxascaris lemonina*. Both of these are ascarid nematodes in the order Ascaridida, superfamily Ascaridiodea, family Anisakidae. Their definitive hosts are the domestic dog, cat, cattle and buffaloes in which they live as adults within the lumen of the small intestine. Dogs and cats play an important role in societies throughout the world. They are important companions in many households, contributing to the physical, social and emotional developments of children and their owner. On the other hand, stray dogs and cats are neglected and live in streets of urban and suburban areas. In Egypt, stray dogs are about 2

million found in and around Cairo (General Organization of Veterinary Services, Ministry of Agriculture, Cairo, pers. Comm.) where they fed scavenged animal offal's and refuse. Dogs constitute a vast reservoir of *T. canis* worldwide, although two other species, *T.cati* and *T.vitulorum* are also possible causes of the disease **(Soulsby, 1983).**

Larval migration through soft tissues in the humans generates several clinical entities in the patient such as visceral larval migrans (VLM), ocular larval migrans (OLM) and neuro-toxocariasis.

VLM is primarily the disease of children who are more likely to ingest eggs of *Toxocara* species. Generally human become infected by ingestion of embryonated eggs either from soil, dirty hands, raw fruits and vegetables or larvae from under cooked meat of paratenic hosts and unpasteurized milk **(Borecka et al., 2010)**. The contamination of soil and water with eggs of *Toxocara* from faeces of dogs and buffaloes represents the main source of infection.

Migration of *Toxocara* larvae occurs through blood to various organs where they cause inflammation and damage. Most larvae pass through liver and lungs and this may be associated with signs of decreased appetite, abdominal pain, fever, hepatomegaly, coughing and asthma **(Ehrhard and Kernbaum, 1979)**, where migration of larvae to eye causes blindness, retinal granulomas and endophthalmitis. There is eosinophilia, leukocytosis and hypergamma-globinaemia. The severity of the disease ranges from asymptomatic to fatal infection. **(Glickman et al., 1979).**

The diagnosis and control of Toxocariasis is not easy as the larvae migrate in the tissues, remaining as dormant or hypobiotic parasites. Several serological tests such as Indirect Haemagglutination Test (IHA), Counter-current Immuno Electrophoresis (CIEP) and Enzyme Linked Immune-Sorbent Assay (ELISA) have been applied for immunodiagnosis of *T. vitulorum* **(Singh et al., 2003)** by using crude somatic antigens with variable results.

Treatment of Toxocariasis is unsatisfactory due to re-infection which makes assessment of response to therapy difficult, so the control of soil contamination is required. The control of *Toxocara* infection must focus on preventing the contamination by either limiting access to public places by dogs or by strict enforcement of laws requiring owners to clean up after their dogs, immunization could supply one control method.

History of discovery

Human infection with *Toxocara* species was first described by **Wilder (1950)**. He identified a nematode larvae of unknown species within a retinal granuloma of a child. In **1952**, **Beaver et al.** reported on a similar series of children who presented with high

circulating eosinophilia, and suffered from severe, long-term, multisystem disease. From this group of patients, they described most of the clinical features of VLM and, in histopathological sections of tissues obtained at biopsy, correctly classified the causative agents as the larvae of either *T. canis* or *T. cati*. Since that time, the juveniles of these two parasite species have been detected in a variety of lesions of the eye and throughout the body in patients from all corners of the world. Today, the public health community at-large acknowledges that toxocariasis in all its clinical forms constitutes a major health risk, especially among children exhibiting pica **(Magnaval et al., 2001)**.

Epidemiology

The contamination of soil and water with eggs of *Toxocara* from faeces of dogs and buffaloes represents the main source of infection. In sub-urban and rural areas dogs have access to buffaloes, and under these conditions infected hosts are likely to pass considerable numbers of eggs in to the surrounding water. An adult female of *T.canis* worm produces 200.000 egg / day and heavily infected bitch with her puppies can produce about $1.5x\ 10^7$ egg / day. Whereas each female *T. vitulorum* daily produces thousands of eggs ($8x10^6$) egg per day. Egg production ranges from 8000 to as high as 100,000 eggs per gram faeces per day **(Soulsby, 1983)**.

Vegetables in rural areas were washed with contaminated water helping spread of the infection. Furthermore, the farming practice of allowing poultry and other stock to range and mix freely with buffaloes and dogs enhance the transmission of *Toxocara* from the ruminant and canine definitive hosts to paratenic hosts such as chicken, pigeons, turkey, duck and rabbits. Birds that feed primarily on the ground (e.g., pigeons, starlings, and sparrows) can serve as paratenic hosts, carrying eggs from place to place on their feets and beaks, and may be responsible for depositing eggs in places far from the original source **(Lewis and Maizels, 1993)**.

The intake of paratenic host by human being leads to *Toxocara* infection. Furthermore, in both urban and rural areas, the offal of poultry, rabbits and rodents is frequently fed to dogs **(Struchler et al., 1989).**

Eggs of *T.canis* from the dogs, when ingested by buffalo calves and sheep, develop into active larvae in the liver and thoracic muscles, and the larvae are in turn infective to man **(Hafez, 1978)**. **Lee et al. (1976)** recorded that liver is the favorite tissue for migration of nematode larvae; therefore, the possibilities of human visceral larval migrans may be induced due to eating of raw liver of domestic animals. **Lewis and Maizels (1993)** indicated that sero-prevalence of toxocariasis is significantly higher in persons eating under cooked calf liver. They also pointed out that dairy cows experimentally infected with

embryonated eggs of *T.canis* pass out active larvae in the milk. This is particularly prevalent to farming community in Egypt as the children who habitually drink the colostrum from dairy cows, will be continually exposed to a source of active larvae of *T.canis* or *T.vitulorum*. The risk of human community however is greater due to the farming practice of allowing buffalo calves to range and mix freely with dogs which shed billions of eggs in urban and rural areas in Egypt.

Many inhabitants in these regions spray soil surface with water to prevent the dust particles from blowing around, and also, inadequate sewage and drainage systems lead to the over flow of water from the drains on to the streets. These create optimum conditions of temperature and humidity for the development of infective eggs within the soil **(Omar and Lewis, 1993).**

Toxocara eggs were found in all layers of soil due to the inherent habit of cat defecation; also eggs were carried by earth worm castings from deeper layers to the surface of ground up to, at least one year after its contamination. Soil texture influence the recovery of toxocara eggs dusty soil was found to be rich in eggs. Experimental contamination of the soil revealed maximum numbers of *Toxocara* eggs were recovered from clay soil 20%, followed by sawdust 13.4%, and sand soil 10.6% after 6 months incubation under moist condition **(El-Menyawe and Abdel Rahman, 2007**).

Some elements of soil, saprophytic fungi for example (Paecilomyces lilacinus and Paecilomyces marquandii), have been shown under controlled conditions to have larvicidal activity against the juvenile worm within its egg's shell **(Basualdo et al., 2000).** Eggs are long lived and resist both environmental conditions and chemical agents due to thick shell. Infective ascarid eggs of all species can last for months to years outside the host under optimal conditions, due solely to a resistant outer shell composed of ascarosides. This acellular layer enables eggs to withstand various harsh chemicals (e.g., high concentrations of formalin and various inorganic acids), extreme temperature changes, and various degrees of moisture. Future strategies for reducing the number of infectious eggs in soil must find novel ways of breaching the egg shell barrier that protects the infectious eggs from its external environment **(Despommier, 2003).**

The survival time is at least one year. Type of soil, ambient temperature and humidity are the main factors that determine the time it takes to evoke L2. Development occur at 12-18°C within 54 days , while at optimum temperature 25-30°C developed for 14-11 days respectively. The larvae can remain viable during the year inside the egg. Egg remains dormant but viable if covered by snow at -11°C or protected in faeces**.** *T. canis* eggs resist both aerobic and anaerobic conditions of modern sewage processing and remain viable. Only heat and desiccation readily kill the eggs, at 37°C degeneration occurs.

Ultraviolet light and prolonged darkness cause damage of the eggs **(Lewis and Maizels, 1993)**.

Toxocara eggs were recovered from soil samples taken from private yards, public gardens and school playground with high concentrations were found near places where dogs and cats aggregate. The exact prevalence of *T. canis* eggs in the soil depends on the recovery method, the geographical location and the number; volume of the samples taken. Most surveys have found prevalence rates ranging from 1-30% with almost no public places devoid of concentration. **(Lewis and Maizels, 1993).**

Examination of the soil recorded a 13.8% contamination rate with *T. canis* eggs from Cairo and Giza Governorate **(El-Menyawe and Abdel Rahman, 2007**). In some reports, faecal samples taken from parts were only 5% where 16.3% soil samples were positive. It suggests that only few positive faeces may produce wide spread of contamination and the accumulation of *T. canis* eggs from the same stray dogs in different sites. The extreme resistances, long survival time and large number of eggs produced by heavily infected bitch and her puppies; this explains that phenomenon **(Lewis and Maizels, 1993).**

***T.canis* in dog**

Dogs constitute a vast reservoir of *T. canis* worldwide, based on published reports of observation of approximately 42000 dogs, 15% are infected with *T. canis* **(Glickman and Schantz, 1981).**

However, the prevalence is over 90% if one considers only puppies less than 12 weeks of age and it is probably closer to 100% since infection status in some studies was based solely on a single stool exam which lacks sensitivity in lightly infected dogs **(Barriga, 1988).** Thus it can be concluded that nearly all puppies are infected at birth due to trans-placental route and that current available anthelmintic drug are not effective in preventing this phenomenon.

Life cycle: (According to **Lloyd, 1993)**

Infection occurs by ingestion of eggs containing infective 2nd larval stage. Eggs are infective within 10-15 days under optimum condition. Following ingestion hatching occurs in small intestine. After 8 days, 2nd L.S. is found in different tissues (liver, lung and kidney). Somatic migration occurs only in *T. canis* and *T. cati*. At this stage, larvae had undergone no development. They can become resident in somatic tissues of the adult dogs and remain there for many years. Under certain conditions, larvae in the somatic tissues are mobilized to the lung and trachea, where moulting occurs to 3rd L.S. and reach to pharynx, esophagus, stomach and small intestine where adult stage is developed.

-Trans-placental migration of larvae: Infection of bitch before pregnancy leads to somatic migration of larvae, activation of these larvae under hormonal effect and mobilization to the placenta and the fetus giving rise to prenatal infection. Some larvae remain in the somatic tissues of bitch to undergo the process at subsequent pregnancies. When the larvae reach to the liver of fetus become 3rd L.S. at birth larvae moult to 4th L.S. in the lung during the 1st week of life. By the end of the 2nd week after birth, 5th L.S. is formed and adult worm developed at the end of 3rd week.

-Trans-mammary transmission of larvae: Activation and mobilization of larvae from the somatic tissues of bitch and migration to mammary gland (postnatal transmission) when bitch is infected in late pregnancy or during lactation, and reach to the puppies through the ingestion of infected milk from their mothers. Development occurs within 16 days.

-Oral infection from soil: Infection of pups, 1-3 weeks old by ingesting the infective eggs from the soil per os leads to tracheal migration and to the intestine. Whereas pups 5 weeks old when infected per os no somatic migration of larvae occurs and larvae fail to reach the intestine.

-Infection of the bitch after parturition: Eggs of *T. canis* are found in faeces of bitch shortly after parturition due to weakening of the immunity which permits larvae to pass through the lung and complete their development in the intestine. Also, infection is acquired due to the habit of bitch in cleaning after her pups and ingesting immature stage of *T. canis* from their faces which undergo no migration in bitch and become mature in her intestine. This infection is eliminated in few weeks.

-Infection of adult dogs:

Oral infection occurs with eggs or infection of paratenic host. Adult dogs were considered resistant to infection and tracheal migration of larvae.

Symptoms and pathological lesions

Heavy infection occurs under conditions of poor hygiene. Pneumonia is found in newly born puppies due to migration of larvae through the lung tissues. In less severe infections, vomiting, diarrhea, anemia and poor growth occur in puppies of few weeks old. Death occurs due to severe infection and presence of 300-400 worm / puppy.

Nervous symptoms were due to absorption of substances secreted by adult worm to the general circulation. Animal show pot-belly or the abdomen is tucked up, dull harsh coat with emaciation, diarrhea or constipation and acute intestinal obstruction. Adult dogs carry

only small number of worms, somatic migration of larvae produces granuloma in kidney while migration of larvae in the eye produces ocular lesion **(Lewis and Maizels, 1993).**

Diagnosis

About one third of infections are not detected by faecal examination. Some factors affect infection of dogs with *T. canis* e.g. age and sex of animal, dose of eggs ingested, breed of dogs (mixed breed more than single), environmental housing (stray more than pet), hormonal effect (♀ in met oestrus is more infected), method of diagnosis, seasonal variation (temperature, humidity and rain), habitat (rural more than urban sites), and population composition of dogs.

***T. vitulorum* in buffaloes**

Toxocara vitulorum is a parasite of small intestine of ruminants, particularly buffalo calves, one to three months of age **(Patnaik and Pande, 1963).** It is responsible for high morbidity and mortality rates resulting in serious economic losses and zoonotic importance **(Enyenihi, 1969).** The adult worms of *T. vitulorum* are encountered principally in suckling calves. The transmission of *T. vitulorum* is both trans-placental (prenatal) and trans-mammary (postnatal). More recently, **Roberts (1990)** concluded that the route of infection is mainly through colostrum and milk, causing disease (severe anemia, diarrhea, weight loss and anorexia) particularly in buffalo calves **(Wickramasinghe et al., 2009).**

The dam sheds no *T. vitulorum* eggs in faeces even she thought to be the source of infection to the calves. This is because the larvae in the cow do not develop to adults but remain in third stage. When the cow is pregnant the larvae migrate from the liver to the mammary gland (prior to parturition), then to the milk through which the calf is infected (**Roberts et al., 1990).**

It is clearly evident that adult animals are refractory to the intestinal infection but under stress factors adult worms are developed in the intestine. **Souza et al. (2004**) found relationship between a marked immunosuppression due to stress of pregnancy and lactation with the development of adult worms. Suppression of mitogen-induced lymphocyte and decrease of antibody titer were reported also by **Amerasinghe et al. (1994).**On the other hand, the highest rate of infection in young calves (1-3 month), might be attributed to prenatal and postnatal routes of infection (from the infected mothers and the contaminated environment).

Abdel Rahman and El-Ashmawy (2013) indicated that a rapid decline of faecal EPG counts with the decrease of antibodies level in the serum of the calves after 6 weeks of age. This result suggests the expulsion of adult worms from the intestine and toxocariasis is self-cure process.

Antibodies, passively or actively acquired, may have an important role during worm rejection by the calves and prevention of intestinal reinfection.

Stevenson et al. (1994) stated that tissue eosinophilia has been also associated with the rejection of worm from the gut. It is found that eosinophils release mediators that could attack nematode cuticle in the lumen of calf infection **(Maria et al., 2003)**.

It should be emphasized that features of *T. vitulorum* larvae including the morphology, patho-physiology, and migratory behavior in the paratenic host are similar to those of *T.canis*. The biological similarity between two Toxocara species is further confirmed by the presence of strong cross reactivity between both species using immunological techniques (**Barriga and Omar, 1992).** In conclusion, it is suggested that zoonotic transmission of toxocariasis is likely to be enhanced by the presence of both canines and ruminant hosts in tropical and sub-tropical communities, where domestic and agricultural practice overlap.

Human toxocariasis

Transmission

Since eggs of *T. canis* are not embryonated when passed in faeces of dogs, they are not directly infective for human. The transmission occurs mainly from the soil contaminated with faeces of infected dogs and cats. The shell of *T. canis* egg is very sticky, adhere to fingers, toys, food with subsequent ingestion is important in transmission of infection.

It should be noted that zoonotic toxocariasis can also be acquired from sources other than soil and the possibility of domestic food animal as sheep, goat and cattle and poultry serving as paratenic hosts for human infection is considered food born infection. Ingestion of raw hen's liver in Japan from chicken confined with dogs was associated with human toxocariasis. Also, in Switzerland, toxocara antibodies in children were associated with consumption of rabbit giblets and lightly grilled meat (**Hafez, 1978**).

Toxocara fail to complete its majority cycle in human and adult stage not develop only 2nd L.S. undergo somatic migration in the internal organs (liver, lung and eye) producing the following pathological conditions (**Despommier, 2003**).

Clinical signs and symptoms

1- Visceral larval migrans

VLM is mainly a disease of young children (<5 years old) due to poor hygiene, close association to puppies, immature system and geophagia or pica (ingestion of non-edible substances as dirts , soil contaminated with eggs of Toxocara. It is a chronic granulomatus

oesinophillic lesion associated with somatic migration of large number of Toxocara larvae in the abdominal organs.

Signs of VLM in the form of abdominal pain, fever, decrease appetite, coughing, hepatomegaly, necrosis of the liver, enlargement of the spleen; lower respiratory symptoms (particularly bronchospasm, resembling asthma); and asthma. There are usually eosinophilia , leucocytosis and hypergammaglobulinemia of immunoglobulin M (IgM), IgG, and IgE classes.

In the last of these instances, symptoms are more pronounced, with increased levels of IgE/anti-IgE immune complexes. Myocarditis, nephritis, and involvement of the CNS have been described. CNS involvement can lead to seizures, neuropsychiatric symptoms, or encephalopathy.

These signs are self-limiting if there is no subsequent exposure to the infection. If the exposure persists or intensified it will lead to sever disease and other organs are invaded, the disease can be life threatening in which myocardium and brain are invaded (**Shetty and Aviles, 1999**).

2- Ocular larval migrans (OLM)

It results from the migration of few number of Toxocara larvae in the eye; the disease is unilateral in older children (5 to 10 years) and found in males more than females.

Signs are in the form of loss of vision, fixed eye pupil or red eye. Using serological techniques, **Khalil (1964)** found that *T. canis* was responsible for causing blindness in 37% of patients examined in Egypt. Antibodies in serum were lower than VLM, there are no eosinophilia, leukocytosis nor hypergamma-globinaemia .

The most serious consequence of the infection is invasion of the retina, leading to granuloma formation, which occurs typically peripherally or in the posterior pole. These granulomas drag the retina and create a distortion, hyperopia, or detachment of the macula. The degree of visual acuity impairment depends on the specific area involved, and blindness is common. OLM can also cause diffuse endophthalmitis or papillitis; secondary glaucoma can follow. In at least one rare instance following long-term infection with Toxocara, a choroidal neovascular membrane formed after presenting earlier as chorio-retinitis (**Monshizadeh et al., 2000**).

Risk factors for toxocariasis

The risk factors for VLM and OLM comes from numerous descriptive and analytic epidemiological studies **(Glickman and Schantz, 1981).** Factors include children with pica (geophagia), rural residence where lower socio-economic states, contact with puppies in home or soil contaminated with *Toxocara* eggs. Also, consumption of raw liver and poor

hygiene predispose to the infection. The relative importance of these risk factors may differ between countries and between geographic regions within countries. A larger dose of larvae is required to produce clinical signs of VLM than OLM. The role of pica, defined as the persistence ingestion of non-edible substance, beyond 18 months of age need further investigation. A common theory is that pica results from deficiency of iron and that it can be corrected by supplemental iron administration **(Crosby, 1976).** Further studies are also needed to define the relative role of dogs in home and the contaminated soil in risk factors for VLM and OLM.

Molecular aspects

Dormant tissue larvae have been recorded as long as 9 years after infection in a mammalian host **(Beaver, 1966).** The ability of arrested-stage larval parasites to survive in the tissues for many years must depend on potent immune-evasive and anti-inflammator mechanisms operated by the parasite**.** Cultured larvae of *T.canis* secrete macromolecules called Toxocara Excreted-Secreted (TES) products (**Maizels, et al., 1987**). These products are the primary candidates for immune evasion mediators, and in deed larvae are found to release large quantities of glycoproteins in vitro. Antibodies aqgainst secreted TES glycoproteins also detected antigens in vivo which appear to have been released from the parasite **(Smith, 1991)**. .

Investigations into the molecular biology of *Toxocara* have mainly focused on the secreted proteins of the migrating juvenile stages. These proteins have proven useful in immunodiagnosis of VLM, and OLM. Presumably, the worms eventually die of old age. The fact that many of the excretory-secretory proteins from the juvenile stages constitute a family of at least six highly antigenic mucins associated with the cuticular surface reinforces this concept **(Loukas, et al., 2000).**

A range of fascinating molecules have emerged, some of which can be predicted from their primary sequence structure to fulfill certain functions: mucins, proteases, enzyme inhibitors, ect (**Maizels et al., 2000**). Secreted mucins temporarily coat the surface of the worm and are shed into the host periodically. It is thought that this shedding behavior represents an attempt on the part of the parasite to confuse the host's immune system, leaving behind it a trail of slime, not unlike that of a snail. In this model, the worm periodically switches its secreted antigenic identity, thus avoiding harm **(Maizels and Holland, 1998).**

Diagnosis

Any pediatric patient with an unexplained febrile illness and eosinophilia should be suspected of having VLM. Hepato-splenomegaly and evidence of multisystem disease and history of pica make the diagnosis of VLM more likely. Since the larvae of *T. canis* fail to complete their migratory cycle in human, remaining as dormant or hypobiotic parasites in the tissues and eggs are not passed in stool, thus a diagnosis of *T. canis* infection is not easy. Indirect diagnosis usually occurs by demonstration of elevated concentration Ab in the serum or other biological fluids e.g. vitreous humor. Several serological tests such as Indirect Haemagglutination Test (IHA), Counter-current Immune Electrophoresis (CIEP) and Enzyme Linked Immune-Sorbent Assay (ELISA) have been applied for immune-diagnosis of Toxocariasis **(Singh et al., 2003)** by using crude somatic antigens with variable results.

The most widely used tests, because its high sensitivity and specificity is ELISA in which antibodies against *T. canis* larval security–excretory antigens or larval extracts are measured. The serological prevalence of human *T. canis* infection using ELISA generally varies between 2% and 10% depending on the demographic composition of the population. ELISA has a reasonably high degree of sensitivity, as well (approximately 78%), at a titer greater than 1:32. Liver biopsy may reveal a granuloma surrounding a larva, but a successful diagnosis using this approach is fortuitous at best and not recommended. The immunodiagnostic tests used for VLM are not as reliable for OLM. In one study, only 45% of patients with clinically diagnosed OLM had titers higher than 1:32 **(Schantz, 1989).**

Laboratory Animal

Several studies with laboratory animal models have been developed particularly to investigate larval migration and pathological changes in different organs and to evaluate the effect of drugs as well as to screen the immunological aspects in experimentally infected animals. **Smith (1991)** reported that migration pathway of larvae in humans and mice is similar, and lesions in experimental mouse models and human are comparable. The migratory route in mouse includes two phases: a visceral phase, during 1st week post-infection, the larvae reach to liver and lungs; afterwards, larvae migrate throughout the body and accumulate in the muscles and brain. **Amerasinghe et al. (1992)** found that oral infection of mice with *T. vitulorum* eggs induced protection against a challenge infection with eggs of *T. vitulorum*. The results obtained by **Abdel-Rahman et al. (2013**) indicated that larval antigen of Toxocara when injected to rabbit model induced high level of protection rate in immunized animal, where total number of recovered larvae from infected animal decreased and ELISA optical density values increased as compared with control group. The overall protection rate was 84.9% where the protective immunity was more

effective in liver (89.2%) than in lungs (77.9 %). According to **Barriga and Omar (1992)** the protective immunity was less effective in lungs and muscles**,** where the larvae reach later to muscles not killed during migration but killed in organs and the lethal anti-larval immunity may be cell-mediated immunity rather than antibody-mediated. The acquired humeral immunity plays a minor role in the elimination of the parasite. They believed that antibodies may be adequate probe to determine the specific epitope of parasitic antigen relevant for protection. Isolation of this antigen for assay in vaccination against Toxocariasis may be a worthy undertaking.

It is concluded that peri-enteric antigen of the adult worm is the most potent and could be used for serological diagnosis of VLM and vaccination against Toxocara using larval antigen could play a role in limiting the distribution of the problem in suspected animals.

Treatment

Albendazole is the treatment of choice for toxocariasis. Patients receiving a 5-day treatment course of albendazole (10 mg/kg of body weight/day in two divided doses). 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 **(Dinning et al., 1988).**

Prevention and control

The high prevalence of toxocara infection among dogs and cats and the increased level of environmental contamination could indicate that human in these regions are seriously at risk. Human infection may spread vastly and cause severe damage if preventive measures are not taken into account. In this regard, preventive program is required such as:

- Preventing the contamination by either limiting access to public places by dogs or by stricter enforcement of laws requiring owners to clean up after their dogs.
-Improved hygiene and better parental supervision of children playing with puppies at home and private yards, can reduce the chance of infection (**Omar and Lewis, 1993**).
- Regular treatment of both pets with pregnant and lactating bitch with benzimidazoles helps reduce worm burdens and limits the number of eggs deposited in soil, also to kill larvae in its tissues or prevent trans-placental and trans-mammary infection to puppies **(Despommier, 2003**).
- Buffalo calves are given Ivomac at dose of 200 mg/kg. b.wt.
- Treatment of milk from recently parturated buffalo before human consumption should be a matter of public health importance.

- Control of soil contamination by Toxocara eggs using chemicals such as, ammonia, phenolic acid, lime, formalin and betadine solution **(Capizzi et al., 2004)**.
- Finally, early diagnosis via serological techniques would allow initiation of useful chemotherapy before extensive hepato-pulmonary larval migration with resulted pathologic changes, thus producing a significant impact on the economic aspect of the disease.

Concluding remarks

T.canis presents an attractive model system for parasitic nematodes due to its striking features e.g. tissue-dwelling phase, trans-placental infection, its tropism for neurological tissue in humans and its ability of immune evasion. The elimination of this infection would be a highly desirable goal **(Maizels et al., 2000).** Killing Toxocara eggs in contaminated soils 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 **(Despommier, 2003**).

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