Parasitic Zoonoses Of The Brain: Another Challenger?
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Citation

Abstract
We found a high prevalence of dogs among animals kept at home in some villages at the former Transkei where the prevalence for cysticercosis is also high. From our personal experience on the field of epilepsy and neurocysticercosis (NCC) and its radiological signs on CT scan of the brain we were able to identified some nodular and hyperdense lesion on the cerebral hemisphere which not resemble NCC at any stage but we could not confirm any other parasitic zoonoses of the brain because our lack of resources for a proper laboratory diagnoses. Because we have a strong suspicion about Toxocariasis among our patients we reviewed the available medical literature to conclude that we have more suspicion about the presence of Toxocariasis in our population as another challenger to consider.

INTRODUCTION
BACKGROUND
The Zoonoses include a large number of infectious diseases of lower animals that are transmissible to humans. The infectious agent may be transmitted in a variety of ways, namely:

- Direct contact with animal flesh (Tularemia)
- Drinking of cows or goats milk ((TB and Brucellosis)
- Inhalation of dust particles contaminated by animal excreta or products (Q Fever & Anthrax)
- Eating of insufficiently cooked infected flesh (Anthrax, Trichinosis)
- A bite by insect vectors (Plague, Scrub Typhus and Equine Encephalomyelitis) or a bite from a diseased animal (Rabies)
- Ingestion of animal flesh (Taeniosis-T solium)
- Others ways

From 1999 to 2008, we studied clinical, therapeutic, and radiological aspects on neurocysticercosis (NCC) at the former Transkei in South Africa [1-16] and we also conducted some neuroepidemiological studies about NCC, epilepsy, and HIV/AIDS; all results obtained are fully available on Internet [17-25]. Nevertheless, in spite of our personal dedication we could not get accurate and sustainable scientific results as we wished but we learned that almost all brain disorders presenting with an associated HIV/AIDS, have something different and its represent a new challenger.

Former Transkei was one of the three administrative authorities of the so-called independent homelands which include Ciskei and the Cape Provincial Administration (under different apartheid governments). At the present moment, former Transkei corresponds to regions D and E of Eastern Cape Province (ECP) of South Africa (SA). Mthatha is the capital for the former Transkei which is one of the poorest region countrywide, and serves as a labour reservoir for other wealthier provinces, with men leaving behind women and children whilst they seek and find employment elsewhere [17-25]. In our previous studies, we found that our region is plagued by a high proportion of unemployment, low socio-economic level and poverty. In an effort to ensure the highest level of privacy and confidentiality, a realistic approach to research was adopted [17-25]. A mutual relationship based on trust and transparency was first established, so as to make the community open up and give truthful information that would give a true reflection of several locations [17-25]

South Africa is both a first and third world country with a population of about 40 million of which 76% is black. Before 1994, health services were fragmented along the racial lines, with the white population receiving First World
Health care, while black people, especially in the homeland areas, received care below standard. Since 1994, the new democratic government identified, amongst its challenges, provision of equal services to all South Africans, as described by basic elements of Primary Health Care and Declaration of Alma-Ata.

The Eastern Cape is one of the two poorest province of SA, and the third most populated province countrywide with a population of about seven millions individuals. Transkei is the less developed, mainly composed of rural areas and illiterate people [17-25]. This area is not very good for farming as droughts frequently cause reductions in the amount of both harvesting and livestock production. This leads to a constant economical and nutritional challenge in the region. This is the picture that from our previous studies was obtained. About 60% of our population is not economically active and most of the males from here earn their living by being migrant workers in bigger cities, (being its other way for spreading cysticercosis) and this is reflected in the rate of increase in HIV/AIDS in this vicinity in young adults and adults who are married and those in ‘steady’ relationships, because the men tend to have multiple partners away from home and come back to infect their wives.

Taenia solium cysticercosis’ life cycle starts when humans become infected by eating undercooked pork containing cysticerci and later they develop taeniosis. People with taeniosis pass eggs with their faeces which are ingested by humans and pigs. Eggs develop into larval cysts causing human and porcine cysticercosis. Risk factors for taeniosis include the consumption of undercooked infected pork meat and inadequate meat inspection. Risk factors of cysticercosis include free-range pig farming, poor sanitation, close contact of humans and pigs and inadequate hygiene from food handlers. Cysticercosis is thus strongly associated with poverty [17-26]. In both humans and pigs, cysts migrate mostly to the subcutaneous tissue, skeletal muscle, the eye and the central nervous system [27]. Currently, NCC is the major cause of acquired epilepsy in developing countries, and is also of growing concern in northern/western countries due to globalization and immigration of infected people [28-30]

Pig farming has increased considerably during the past decade in Eastern and Southern Africa (ESA); especially in rural, resource-poor, smallholder communities where sanitation is poor [31]. Hence, it is highly suspected that the frequency of NCC in the region may further increase in the foreseeable future.

In 2004 and 2005, we conducted two pilot studies one at tertiary Nelson Mandela Academic Hospital (NMAH) in Mthatha, and other one at Saint Elizabeth rural hospital in Lusikisiki both at the former Transkei. The second one included 296 consecutive patients consulting the neurological clinic for suspected new-onset seizures or existing epilepsy cases. Each week, four randomly selected, consenting patients with confirmed seizure disorder were transported to Mthatha for CT scan of the brain. The prevalence of seropositivity to antigens of T. solium was 8% (95%CI: 4.5%-13%). A total of 92 patients with recurrent seizures and who also completed a questionnaire were referred to Mthatha for a CT-scan. Of these, 34 (37.0%, 95%CI: 27.1%-47.7%) had a definite diagnosis of NCC, 14 of whom had active lesions visible on CT, 39 (42%) had no CT abnormality, and 19 (21%) had other, undefined non-NCC calcifications. Our results showed that serology alone cannot be used to diagnose NCC in this population.

HIV status was available from 50 patients with confirmed seizures or epilepsy. Among the 47 patients with antibody ELISA results available, the antibody seroprevalence of T. solium was 30.0% among HIV positive patients and 48.1% among HIV negative patients. Interestingly, among the 33 patients with antigen ELISA results, the antigen seroprevalence of T. solium was 16.7% among the HIV positive patients but only 9.5% among the HIV negative patients. These preliminary results suggest that HIV patients may be less able to mount a detectable antibody response to cysticercosis and might be more likely to be infected with active cysts. A total of 22 of these patients (13 HIV negative and 9 HIV positive) were referred for a CT-scan. Of these, 5 HIV negative and 7 HIV positive patients had CT evidence of NCC with 2 HIV negative and 5 HIV positive patients harboring active cysts. These very preliminary and imprecise results do suggest that there may be an association between NCC and HIV infection.

The pilot study conducted at the NMAH consisted of asking 57 epilepsy patients with confirmed NCC, 52 epilepsy patients without NCC and 61 patients from the dermatology and ophthalmology clinics to answer a questionnaire interview about epilepsy and pig raising and management. Very preliminary analysis suggests that, using dermatology and ophthalmology clinic patients as the reference group, the POR of owning pigs and having NCC was 6.8 (95% CI: 2.1-21.7) and the POR for consuming pork compared to never consuming pork and having NCC was 2.1 (0.7-6.2). Using the epileptic patients without NCC as the reference
group, the POR of consuming pork in those with NCC was 14.2 (5.1-39.5) and of owning pigs was 17.5 (5.4-56.2). Why the association between owning pigs and NCC was stronger when the epilepsy patients were used as a reference group needs further investigation, but it is possible that the NCC and non-epilepsy groups came from rural areas whereas the epilepsy non-NCC group came from more urban areas with less exposure to infected pigs. Further analyses will determine whether these results are confounded by the age of the patients or where they live.

There are many issues still not clarified about NCC, and far to be eradicated when new suspicions related to other parasitic infections of the brain are gradually increasing being its another challenger for neurologist, other specialist and general practitioners.

In our door to door neuroepidemiological survey [17-25] we found a big number of animals kept at home and we could count up to 8 dogs per house (Figure 1) and the percentage of dog kept at home was around 30-40% (Table I-II) however no other parasitic zoonoses of the brain at the former Transkei have been found.

**Figure 2**
Table I: Prevalence of animals kept at home in Sidwadweni location (ECP) South Africa
The surveyed sample was at risk to a number of infections ranging from parasitic infections to Zoonoses, due to their close association with domestic and commercial livestock. These infections can be transmitted as explained above or via unhygienic practices such as not washing hands adequately. Failing to control insect vectors would also aid in the transmission of these infections.

The following observations were made:

By far, the most common vector for disease was mosquitoes (96%). This is of no particular significance here, as malaria and yellow fever; etc is not endemic to this region. They may play a role if an infected person brings a disease into the area.

Flies present in the kitchen and toilets inside homes stood at 69%. This is of medical importance as they are vectors for a wide variety of diseases. These include trachoma (Chlamydia trachomatis), amoebic dysentery (Entamoeba histolytica), shigellosis (Shigella spp.) and Salmonella (Salmonella spp.), etc.

Rodents were reported present in 37% of households, predisposing the occupants to a wide variety of vector borne diseases. Examples include: Listeriosis (Listeria monocytogenes); Flea vector - Bubonic Plague (Yersinia pestis); Mite vector - scrub typhus (Rickettsia tsutsugamushi); Bite of an infected animal - rat bite fever (Spirillum minus); direct contact with food – Salmonellosis; etc.

Cockroaches were reported in unacceptable numbers in 13% of households. These have not been proved to be vectors of infectious diseases but they are indicators of poor hygiene, especially in cooking areas. They could perhaps be indicators of the risk to infections relating to unhygienic practices favouring the spread of disease for example by flies.

Domestic pets such as cats (20%) and dogs (37%) also present the danger of many parasitic infection as well as vector borne diseases associated with ticks, fleas and mites. Examples of infections are: Cats – Toxoplasmosis by Toxoplasma Gondii; Cat-scratch fever; Cat Liver Fluke. Dogs – Dog tapeworm; Hydatid cysts from Echinococcus Granulosus; and Rabies. Toxocariasis is also transmitted by both cats and dogs.

Pigs (23%), cattle (5%), Goats /sheep (18%) made up most commercial livestock. Horses and donkeys were not reported in the survey but were seen in the community. Tape worm infestations (Pigs and Cattle) play a major role in rural communities and most epileptic cases can be attributed to neurocysticercosis in rural South Africa. Anthrax, Brucellosis, Tuberculosis and vector borne diseases (By Ticks and fleas) would be a possible risk where these animals are kept.

Chickens and ducks/geese were found in 42% of the households. Anthrax, brucellosis, salmonellosis and...
listeriosis are examples of infections associated with these animals.

**TOXOCARIASIS AND OTHERS?**

The prevalence of helminthic infections in most of the developing countries is overwhelming, and almost a quarter of the world’s population is infected by them. As summarized in two more recent reviews, some authors have demonstrated that chronic immune activation with a dominant T helper cell 2 profiles, and anergy, are indeed the hallmarks of chronic helminths infections [32,33]. These immune changes are characterized by several modulations in the normal immune response, particularly those of the cellular immune response, which together could account for possible profound effects of the chronic helminthic infection on the host's ability to handle HIV as well as other infections. Importantly, most of these modulations are reverted almost completely following treatment of the helminthic infections.

Parasitic zoonoses affecting the brain area summarized below (Table I)

![Figure 6](image)

**Figure 6**

Graphic 2: Prevalence of animals kept at home in Nkalueni.(ECP) South Africa

Baylisascaris procyonis brain infections have only been reported in North America where raccoons, the definitive host, are found. Typically, B. procyonis neural larva migrans presents as acute fulminant eosinophilic meningoencephalitis. Once invasion of the central nervous system has occurred, the prognosis is poor with or without treatment. To date, despite anthelminthic treatment of cases of B. procyonis neural larva migrans, there are no documented neurologically intact survivors. Epidemiologic study of human cases of neural larva migrans demonstrate that contact with raccoon feces or an environment contaminated by infective eggs and geophagia or pica are the most important risk factors for infection. [34, 35].

Cerebral schistosomiasis has been reported to occur with Schistosoma japonicum when the parasite produces granulomatous lesions in the brain [36].

Three well-defined syndromes caused by schistosomiasis mansoni have been described: the first, ‘swimmer's itch’, occurs in response to cercarial penetration of the skin, Katayama fever a second clinical entity, classically occurs 4–6 weeks later, and is thought to be precipitated by the onset of ovulation by maturing schistosomula and chronic schistosomiasis. Complications of the acute and chronic syndromes have also been reported: pulmonary hypertension, neuroschistosomiasis, association with Salmonella, association with Staphylococci, viral hepatitis B, glomerulonephritis. Unlike the other three schistosome species affecting humans, S. japonicum is a true zoonosis, infecting at least 40 species of mammals [37, 38].

Despite of the schistosomula of S. mansoni and S. haematobium has similar migratory patterns through human skin. Cerebral schistosomiasis has not been reported in our region and the geographical distribution of this infection remains limited to the People’s Republic of China, the Philippines and parts of Indonesia [39].

Paragonimiasis occurs in Asia (principally Korea, Japan, Taiwan, the highlands of China, and the Philippines), West Africa and in parts of South and Central America. Although more than 30 species of Paragonimus can infect humans, P. westermani is the most frequent cause of disease. Cerebral paragonimiasis has not been reported in SA. Humans, a definitive host as well as other carnivores, acquire infection when they consume insufficiently cooked infected freshwater crab or crayfish. Cerebral paragonimiasis (CP) presents symptoms similar to NCC and has been reported to occur in 0.8% of symptomatic human cases [40].

Another parasitic zoonosis which shows similar symptoms to NCC is coenurosis, due to invasion of the brain by the larval stage knowing by Coenurus cerebralis (CC) of the tapeworm Multiceps multiceps. Watson and Lurie from Edendale Hospital in PieterMarisberg (SA) reported five cases from 1951 to 1956 and described their .anatomopathological finding during their postmortem investigations [41].

At the time that they reviewed the available English-language medical literature in 1956, a total 14 cases from SA.
were found. From his anatomopathological report we could not find gross different from racemose NCC from other description done on CC. At the veterinary side, two rare clinical manifestations of coenurusiasis in sheep have been reported.

Angiostrongyliasis is caused by the rat lungworm. Angiostrongylus cantonensis is endemic through South-East Asia and the Pacific Islands [42]. The infection in humans, an accidental host, is associated with eosinophilic meningitis.

Toxocariasis (TC) is a public health problem. The prevalence of infection in different communities is directly proportional to the infection rates among canines and the free access of dogs to public places. Obviously, the higher the rate of infected dogs and the easier their access to public places, the more easily humans are exposed to infective eggs. Because eggs need weeks in the soil to become infective, direct contact with young puppies is not a risk factor for acquiring disease. Young children are at higher risk because of their play habits and tendency to place their fingers in their mouths. Children with pica (geophagia) and children who have contact with puppies are particularly at risk. In tropical climates, the high temperature and humidity favor the embryonization of eggs.[43]

We found a high prevalence of dogs among animals kept at home in Nkalukeni Village, which represent a tiny place in a tiny region in a large continent. Many of the problems faced in this region are common to all mankind and it will take a change of mindset to alleviate these problems. (Graphic 2)

Animals Kept at House: This is also an important aspect of sanitation and hygiene because these animals are also carriers of diseases and carriers of insects and arthropods that also carry diseases. The following table summarizes the animals kept in the houses questioned. Each figure represents a percentage of the total since many houses kept more than one type of animal. It is easy to see that there are a large number of animals kept by people in this community. Dogs are especially prevalent and they carry ticks and fleas which can spread disease to humans, apart from TC.

![Figure 7](image1.png)

Infected dogs by Toxocara canis is a well known problem among veterinary doctors in SA while infected humans infected by the same parasite remains unknown. We strong believe that TC is an important health problem still no identified.

Life cycle of Toxocara canis is show in the following diagram:

![Figure 8](image2.png)

(Courtesy of the Centers for Disease Control and Prevention)

Neurotoxocariasis (NTOX) is caused by Toxocara canis and, less frequently, Toxocara catis. Toxocara species are not the only causes of ocular larva migrans (OLM) and visceral larva migrans (VLM); others include Trichinella spiralis, Angiostrongylus and Anisakis species. Other causes include A suum (especially in Japan); Angiostrongylus cantonensis
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or Angiostrongylus costaricensis; and, much less commonly, ascarids from salt-water fishes, such as members of the genera Phocanema, Anisakis, and Contracaecum apart from those before-mentioned.

T. canis and T. catis are intestinal nematodes (roundworms) found in dogs and cats, respectively. In humans, toxocariasis is considered an aberrant infection because humans are incidental hosts, and the parasites cannot completely mature in the human body. Instead, the invasive larvae migrate for months through different organs until they are overcome by the human inflammatory reaction and die. The larvae can survive in tissues for at least 9 years and, possibly, for the life of the host. [43]

The adult T. canis female worms can excrete as many as 200,000 eggs per day. These eggs need several weeks of optimal environmental conditions (10-35°C, high soil humidity) to develop from noninfective and unembryonated forms to infective embryonated eggs. The embryonated eggs are resistant to freezing, moisture, and extreme pH levels.

When a dog ingests the infective eggs, the larvae hatch in the small intestine, penetrate the intestinal wall, and gain access to the blood and lymphatic circulation. The larvae invade the liver, lungs, and other tissues. In most dogs, the larval maturation process is arrested in most tissues, but in a pregnant female, T. canis resumes development and migrates across the placenta, infecting the fetus. After the birth of the puppies, the larvae continue their maturation process, migrating from the lungs to the GI tract via the trachea; they achieve their mature forms in the puppies’ intestinal tracts. Female dogs then become reinfected while caring for their puppies.

At least, three clinical forms of TC had been reported; these include visceral larva migrants (VLM), ocular larva migrants (OLM), and covert toxocariasis (CoTOX). Numerous disease manifestations have also been attributed to these parasites.

Diagnosis is based on serologic findings. Polymerase chain reaction (PCR) has been introduced as a diagnostic tool. Examination of stools has no role in the evaluation of toxocariasis. Whether or not the infection should be treated and, if so, when and how it should be treated is controversial. Mebendazole, thiabendazole, albendazole, and diethylcarbamazine, among others, are agents used in the treatment. Corticosteroids also have a significant role in therapy. [43] Due to reasons before-cited T. canis infections are more frequently seen in puppies from 0–6 months old (mainly 3 months) and lactating female dogs than adults. The prevalence of T. canis is significantly higher in male than in female dogs and also higher in dogs which are exercised daily than in those without exercise. The highest prevalence is found in Belgian Malinois breed dogs. T. canis infections are not influenced by the floor type of the kennels (i.e. concrete or soil floor) [44]

**EGGS OF TOXOCARA CANIS**

**Figure 9**

Graphic 3: Prevalence of animals kept at home in Baziya (ECP) South Africa

Toxocara canis eggs are passed in dog feces, especially puppies’ feces. Humans do not produce or excrete eggs; therefore, the presence of these eggs is not a diagnostic finding in human toxocariasis. The egg to the left is fertilized but not yet embryonated, whereas the egg to the right contains a well-developed larva. The latter egg is infectious if it is ingested by a human (frequently, a child).

We found four studies (Medline) reporting the prevalence of T. canis in dogs and of T. cati in cats in South Africa. In a study conducted in 1998-99 on 63 stray dogs destined to be euthanized in the Bloemfontein area of the Free State Province (SA), the prevalence of T. canis was estimated at 21% based on examination of blood, feces and organ samples [45]. Another cross-sectional survey was conducted by the same authors in 1997-98 among 164 domestic dogs and dogs destined to be euthanized. In this study, the prevalence of T. canis infection based on stool examination was not reported, but 36% of 69 necropsied dogs were positive and again the prevalence of infection was significantly higher in puppies [46]. All these studies were conducted in resource-poor communities. In a survey conducted on 1,502 cats in the area of Pretoria in 1980-81, the authors estimated a prevalence of 11% of infection with T. cati. As expected, juvenile cats (defined as the presence of deciduous teeth) had a higher prevalence of T. cati infection.
(14%) as compared to adult cats (8%) [47] Only one study with data collected between 1960 and 1977 has been conducted in our province. Out of 12 dogs treated with arecoline with hydrobromide, three expelled T. canis [48], these results suggest that T. canis was found in our province forty years ago but the current prevalence remains unknown despite the prevalence of dog is high. We found a prevalence of 23% for dogs among other animals kept at home in Baziya location.

Figure 10
Table II: Parasitic zoonoses. (NISA means: not in South Africa) [65]

<table>
<thead>
<tr>
<th>Agent</th>
<th>Definitive host(s)</th>
<th>Intermediate host(s)</th>
<th>Accidental host(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taenia solium</td>
<td>Human, cigar</td>
<td>NA</td>
<td>Human</td>
</tr>
<tr>
<td>Toxocara canis</td>
<td>Cottontail, rats</td>
<td>NA</td>
<td>Human</td>
</tr>
<tr>
<td>Toxoplasma gondii</td>
<td>Cats, dogs, sheep</td>
<td>Lambs</td>
<td>Humans</td>
</tr>
<tr>
<td>Cryptosporidium spp</td>
<td>Cats, dogs, deer</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Taenia (multiceps)</td>
<td>Cattle, goats</td>
<td>Sheeps, goats</td>
<td>Humans</td>
</tr>
<tr>
<td>Paragonimus spp</td>
<td>Mammals, including humans</td>
<td>Snails (16), crabs (20),</td>
<td>NA</td>
</tr>
<tr>
<td>Selassius sericeus</td>
<td>Mammals</td>
<td>Snails</td>
<td>NA</td>
</tr>
<tr>
<td>Ascaridoides canis</td>
<td>Rats, mice, birds</td>
<td>Mollusks (snails),</td>
<td>Humans</td>
</tr>
<tr>
<td>Enterobius vermicularis</td>
<td>Dogs, cats</td>
<td>Mice</td>
<td>Humans</td>
</tr>
<tr>
<td>Baylisascaris procyonis</td>
<td>Raccoons, Small mammals and birds</td>
<td>Humans</td>
<td></td>
</tr>
</tbody>
</table>

The prevalence of gastrointestinal parasites in dogs was studied in the province of Córdoba (Spain), with special attention to those parasites that can be transmitted to man. The prevalence of any intestinal parasitic infection was 71.33%. Authors concluded that it is important to point out the presence of T. canis only in puppies younger than one year and of Uncinaria, more frequent in adult dogs. Soil samples of parks revealed the presence of eggs of Toxocara spp, and it suggests the existence of real risk for human infection. [49]

A recent study in Nigeria showed the prevalence of helminth parasites was significantly higher (p= 0.05) in free-ranging than in kenneled dogs. The prevalence of helminth parasites was also significantly higher (p= 0.05) in African shepherds than in Alsatians and other exotic breeds. Each helminthic parasite had similar prevalence and intensities among both genders (p= 0.05) except for T. vulpis [50]

Humans are paratenic hosts for T canis. Paratenic hosts are transport hosts in which the larvae never develop into adult worms. The infection is acquired by ingesting T canis embryonated eggs. Sources of these eggs include areas where dogs defecate, such as parks. As much as 20-30% of soil samples from public parks and children's sandboxes are contaminated with Toxocara eggs. Infections acquired by ingestion of raw snails and raw lamb have also been reported [43]

The cat roundworm, T catis, has a life cycle similar to that of T canis except that vertical transmission is due to lactation more than transplacental transmission. T catis causes fewer cases of human infection than T canis, probably because of the defecation patterns of cats, which make environmental infestation less frequent.

Tissue damage is due to the host inflammatory reaction more than the infection itself. The larvae produce glycosylated proteins, usually referred to as Toxocara excretory secretory antigens. These antigens induce a Th2-type CD4+ cellular immune response characterized by the production of interleukin 4 that promotes the switching of B-cell isotype to the production of immunoglobulin E (IgE) and interleukin 5. These, in turn, promote eosinophil differentiation and vascular adhesion.

Although Toxocara organisms are the most common causes of VLM, case reports have noted other zoonotic nematodes that cause VLM, including Ascaris suum, Baylisascaris procyonis (raccoon ascariid), and Lagochilascaris minor (opossum ascariid). [51]

The prevalence of seropositivity varies not only from country to country but also in different regions within a country. The real prevalence of TC is difficult to estimate because tests are performed only when the diagnosis is suspected, and most infections are asymptomatic. The seroprevalence of children, as measured with enzyme-linked immunosorbent assay (ELISA), varies from 4-8%. Seroprevalence is higher in the southeastern United States and Puerto Rico. Minorities, such as black and Hispanic groups, have rates as high as 16-30%. [43]

The prevalence of human TC in tropical regions is higher than in the United States. The highest seroprevalence ever recorded was in a village of Santa Lucia, West Indies, where the prevalence was 86% in children aged 6 months to 6 years. This community had an extraordinarily high rate of canine T canis infection combined with peridomestic areas contaminated with canine waste and pica habits among the
children. Serologic surveys in different countries reveal seropositivity rates of 19% in the Netherlands, 2.5% in Germany, 39% in Brazil, 5.8-36% in the Czech Republic, 0-37% in Spain, 5.2% in Cuba, 10.9% in Jordan, 47.5% in Colombia, 81% in Nepal, and 13% in the Slovak Republic.

In 1988 Lynch et al [52] confirmed high prevalence in disadvantaged sector from Venezuela like urban-slum dwellers (20%), rural farmers (25%), and Amazon Indians (35%) compared with patients from advantage areas such as middle-class urban peoples (1.8%).

Apart from the three classic clinical presentations of TC, it can be associated with other clinical conditions and simulating another such as: Well syndrome (eosinophilic cellulites) of unknown origin, chronic urticaria, lymphoma likes, lymphedema, eosinophilic ascites, Henoch-Schönlein purpura, Loeffler endomyocarditis, and pericardial tamponade have been reported [53-60]

After an initial study demonstrating T canis antibodies in 65% of patients with chronic urticaria (n=51), compared with 21% in controls (n=81), others found a seroprevalence of 20% in chronic urticaria (n=128) versus 13% in controls (n=236), a seroprevalence of 8% in chronic urticaria (n=110), a seroprevalence of 13% in children with the condition, and a seroprevalence of 30% in controls. [61]

Wheezing is a common sign of VLM. Progression to eosinophilic pneumonia and respiratory failure has been reported. Isolated reports describe diffuse noncavitating pulmonary nodules and pleural effusions. A case report that described a 65-year-old previously healthy male with a 2-week history of fever and night sweats; weight loss; eosinophilia; high erythrocyte sedimentation rate; and abnormal chest radiograph findings that revealed bilateral hilar and mediastinal lymphadenopathy and discrete bilateral pleurisy demonstrates that, in its acute presentation, VLM can be confused with lymphoma.[62]

VLM is usually associated with hepatomegaly. When histological results are available, they usually reveal granulomatous hepatitis. The spleen is enlarged less often than is the liver. Generalized lymphadenopathy is an infrequent manifestation of toxocariasis. A 24-month-old boy in whom lymphedema was the main clinical manifestation of toxocariasis has been reported. [63]

Although infrequently involved, the heart can be affected. The most common problem is myocarditis and Loeffler endomyocarditis has been reported [64]

Manifestations such as polyarthralgias, monoarthritis, migratory cutaneous lesions, and small-vessel vasculitis may coincide with VLM. One case report describes Henoch-Schönlein purpura in a 17-year-old male in association with anti-Toxocara immunoglobulin G (IgG) and IgE that spontaneously resolved.[63]. Two cases of isolated eosinophilic ascites due to Toxocara have been reported.[64]

NTOX is one of the causes of encephalitis, larval invasion of the brain parenchyma, solitary mass lesions that cause seizures, static encephalopathy, arachnoiditis, spinal cord lesions, optic neuritis, and eosinophilic meningitis, a form of aseptic meningitis in which the WBCs in the cerebrospinal fluid mainly consist of eosinophils.[65]

Covert toxocariasis (CoTox) is the medical term used to identify a less specific syndrome that was recognized with the wider use of serodiagnostic assays for Toxocara infection where most patients are asymptomatic and eosinophilia is less frequent. Less pronounced with this form than with VLM and Toxocara antibody titers are lower, and usual symptoms are: cough, wheezing, chronic or recurrent abdominal pain, hepatomegaly, sleep disturbances, headache, malaise, and anorexia among other problems. This situation usually depends of those factors that cause clinical manifestations such as:

- Number of infective eggs
- Duration of infection
- Anatomic location of the larvae
- Host immune response

A definitive test for CoTOX does not exist. Positive anti-Toxocara titers in the presence of malaise, chronic weakness, abdominal pain, or allergic signs accompanied by eosinophilia and absence of response to allergens support the diagnosis of CoTOX [65]

The list of parasitic zoonoses is increasing gradually as emergent or re-emergent infections and some of problems caused are also increased as can be see in Table II

INVESTIGATIONS

Most common laboratory investigations findings are: leukocytosis and marked eosinophilia on FBC (≥80%),
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hypermaglobulinemia (particularly IgM) and elevated isoheamaglutin titer for antigens in blood groups A and B, specific titers for Toxocara for ELISA with a cutoff dilution of 1:32 (sensitivity 75% and specificity > 90%) but is important to keep in mind that specificity can be lower in tropical populations due to cross-reactivity with other parasitic disorders like cysticercosis. Western blot is more specific despite it cannot differentiate between new and old infections. For this situation antigen-capture assays with monoclonal antibodies may be useful despite their sensitivity and specificity is not good enough. Polymerase chain reaction (PCR) has good results in identification of Toxocara species in tissues in animal models.

From our review of the medical literature, the best test for accurate confirmation of NTOX is the histological examination of the affected tissue (VLM) which reveals granulomatoses lesions containing large numbers of eosinophils and neutrophils and, rarely, the remnants of dead larvae, but unfortunately it is not always possible and biopsies at rarely performed because obvious reasons. Stool test is not indicated because in humans being the intestinal form of toxocariasis doesn’t exist.[65]

OTHER INVESTIGATIONS

Imaging studies in toxocariasis depend on the location of the disease.

CT scanning and MRI can be useful but there is not a patognomonic sign as can be found in cases with NCC

TREATMENT

Although most patients with toxocariasis recover without therapy for those patients with NTOX or lung or cardiac complications, anthelmintic treatment is mandatory. There are large experience on patients presenting inflammatory reaction due to higher doses of praziquantel or albendazole (400 mg PO bid for 5-10 days) whom responded very well to steroids medication therefore its not an excuse for not treat this problems with anthelmintic drugs.

After review the available medical literature we keep a very strong suspicion about TC among our patients that going to be investigated for confirmation in the near future

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References


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