



Impact of Infection with *Toxocara Canis* on Human Health Status

KEYWORDS

human health status, ocular larva migrans, toxocariasis, toxocara canis

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ABSTRACT *Toxocara canis* is an ascarid nematod which causes human toxocariasis when human host is infected by ascarid larvae, by ingesting embryonated eggs excreted in dog feces, from contaminated sources, larvae from undercooked giblets, dirty hands or raw vegetables. Nowadays, human toxocariasis challenge several different syndromes: capsulated toxocariasis, ocular larva migrans, visceral larva migrans, neurological toxocariasis and also covert toxocariasis. We present a clinical case which presented in our department, of whom imunological and inflamatory analysis were inconclusive, but clinical symptoms and computer tomography scan imaging have led us to suspect an human toxocariasis with pulmonary and ocular involvement. This case report highlights the impact of a zoonosis on human health and force the clinician to suspect this diagnosis and the public health policies to protect humans by toxocara canis infection.

Toxocara canis is an ascarid nematod from order Ascaridida, superfamily Ascaridiodea, family Toxocaridae which causes human toxocariasis (helminthozoonosis disease) when human host is infected by ascarid larvae, by ingesting embryonated eggs excreted in dog feces. (Dickson 2003; Qualizza, Incorvaia, Grande & Makri 2011)

Humans are paratenic hosts and the final host is lumen of the small intestine of domestic dogs where ascarids live as adults. Humans become infected by ingesting either embryonated eggs from contaminated sources (pica, geophagia), larvae from undercooked giblets, dirty hands or raw vegetables (Dickson 2003; Magnaval, Glickman, Dorchie & Morassin 2001). Even if, the larvae do not develop into adult worms in the human body, they may migrate to various tissues and organs where they can survive for several years, giving clinical symptoms according to the organs affected. (Magnaval, Glickman, Dorchie & Morassin 2001)

Wilder was the first who described in 1950 the human infection with *Toxocara spp.* by identified a nematode larva of unknown species within a retinal granuloma of a child (Wilder 1950). Since then, many years this zoonotic disease was seen as a unusual pediatric disease; fortunately nowadays due to availability of sensitive and specific immunodiagnostic tests the knowledge of human toxocariasis was improved.

Due to the fact that the disease induces nonspecific symptoms, the real incidence and prevalence of this infestation in humans has not yet been assessed (Magnaval, Glickman, Dorchie & Morassin 2001). The seroprevalence of toxocara is high in tropical areas and developing countries, but low in developed countries. There were conducted several epidemiological studies in Europe, USA and Africa which shown different prevalence of seropositivity for *Toxocara canis*, in different regions, for example in Sweden the prevalence of seropositivity was 7% in children and adult population (Ljungstrom & van Knapen F 1989) while in an Italian adult study *T. canis* antibody seropositivity was 3.9% (Genchi, Di Sacco, Sangalli & Scaglia 1990), also the seroprevalence among

adult populations is 2.4% in the USA and 4.0% in Switzerland, but 20.5% in Brazil and 30.4% in Nigeria. (Fan, Lan, Hung, Chung, Liao et al., 2004).

Pathogenesis of toxocariasis in human follow the route of migration as for paratenic hosts (Magnaval, Glickman, Dorchie & Morassin 2001). The larvae could be found in the eye, heart, liver, lung and central nerve system developing hemorrhage, necrosis and inflammation with eosinophils especially (Young-Soon, Chang-Hoon, Young-Ae, Sung-Youn, Ho et al., 2009). Visceral larva migrans to this organs are largely dependent upon the death of larvae. The immediate hypersensitivity response to dying and dead larvae in the viscera: lungs, liver and brain develop symptoms characteristic to visceral larva migrans (VLM). In the lung, their death occurs due to both delayed and immediate hypersensitivity responses and results in an eosinophilic granulomatous reaction and pulmonary infiltrate (Dickson 2003). In the eye, even a single larva (ocular larva migrans) can develop inflammatory response which can lead to partial or total retinal detachment with visual loss. Migrating third stage larvae can damage the retina developing granulomatous reactions leading to impaired visual. (Magnaval, Glickman, Dorchie & Morassin 2001).

Nowadays, there are several different syndromes of human toxocariasis:

Capsulated toxocariasis recognised by mild symptoms like nonspecific illness with fever, recurrent cough, abdominal pain, headache and behavioral disorders. (Qualizza, Incorvaia, Grande & Makri 2011).

Ocular larva migrans is limited to the eye which may cause visual loss, conjunctival hyperemia, recurrent conjunctivitis. Extremely rare, after long-term infection with *Toxocara*, a choroidal neovascular membrane could develop after presenting earlier as chorioretinitis (Monshizadeh, Ashrafzadeh & Rumelt, 2000; Verallo, Fragiotta, Verboschi & Vingolo 2012).

Visceral larva migrans (VLM) was first described in 1952 by Beaver et al. in children with an enlarged liver and hypereo-

sinophilia, being mainly a disease of children under 5 years old (Magnaval, Glickman, Dorchie & Morassin 2001). This syndrome include fever, asthenia, behavioral depression, respiratory symptoms like dyspnea, persistent cough, recurrent asthmatic and bronchitic manifestations, pleuritis and bronchopneumonia (Dickson 2003, Rubinsky-Elefant G, Hirata CE, Yamamoto JH, Ferreira MU (2010)).

Neurological toxocariasis is extremely rare described in the literature and the clinical symptoms of neurological toxocariasis are nonspecific leading to possible misdiagnostic of this medical condition (Magnaval, Glickman, Dorchie & Morassin 2001).

We present a clinical case which presented in our department, of whom immunological and inflammatory analysis were inconclusive, but clinical symptoms and computer tomography scan imaging have led us to suspected an human toxocariasis with pulmonary and ocular involvement.

A 57-year-old man presented to our Department of Pneumology of University of Medicine and Pharmacy "Iuliu-Hațieganu", Cluj-Napoca in October 2012 complaining for 1 month for dyspnea, persistent cough, asthenia and fever. He also charge recurrent conjunctivitis. There was no previous pulmonary or ophthalmic history, and his general health was good. He was not taking any medications. Computed tomography scan have led in the detection of unexplained multiple pulmonary patchy infiltrate, especially subpleural with an aspect of with „honeycomb” aspect. (Figure 1 (A) and 1 (B)).



Figure 1 A

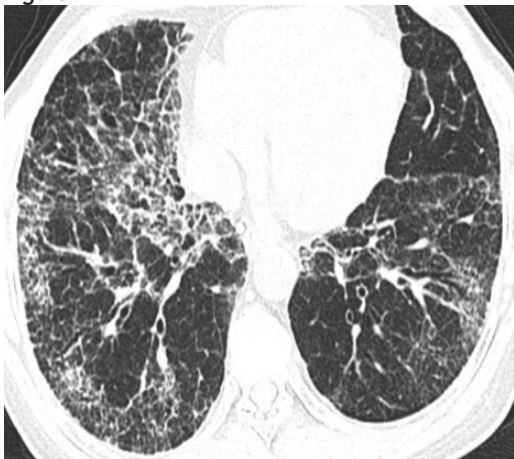


Figure 1 B
Figure 1 (A and B): Multiple pulmonary patchy infiltrate on computed tomography (CT) especially subpleural with an

aspect of with "honeycomb" aspect.

We made bronchoscopy, where we found local eosinophilia 11,4% (n.v <0.5%) at broncho-alveolar lavage. Tests for Mycobacterium Tuberculosis and for Aspergillus Fumigatus were negative. Pulmonary tests were in normal limits, but transfer factor of carbon monoxide detect a slight reduction of diffusion with transfer preserving. The results of laboratory tests showed eosinophilis high according to normal values, 21.3% (n.v 0-5%) and absolute eosinophilis count was $2.35 \times 10^3/\text{mL}$ (n.v.: $0-0.45 \times 10^3/\text{mL}$). Results of serum electrolyte, blood urea nitrogen, glucose and liver function tests were normal. Immunological, inflammatory and allergical markers were testing which were negative. We made Toxocara enzyme-linked immunosorbent assay (ELISA) with Toxocara excretory-secretory antigen (TES-Ag) with Ac IgG Anti-Toxocara canis positive with value of 23.2 NTU/ml. After ophthalmological evaluation, due to recurrent conjunctivitis, fundus examination did not revealed any pathological lesions. The pharmacological treatment consisted of a 5-day course of oral albendazole 400 mg (10 mg/kg of body weight/day in two divided doses) two times a day and oral cortico-therapy with Prednisone for 2 months with gradual dose reduction. After treatment period patient's condition is improved with symptoms remission and stationary aspect of computed tomography pulmonary patchy infiltrate.

The clinical case presented above confirm the possibility of eating such aliments which can which can initiate the development of this pathology. We must actively discouraged population for eating raw meat or unwashed raw vegetables.

Even if visceral larva migrans occurs in children, our patient was an adult one, with pulmonary involvement, that's why, every time when we faced with unexplained pulmonary patchy infiltrate and values eosinophil higher than normal in peripheral blood and bronchoscopic lavage we should consider for Toxocara ELISA test be performed in all patients. Toxocara larva migrans in lung is usually asymptomatic or have only mild symptoms, like our case, and the definitive diagnosis of visceral larva migrans in the lung can be made by pathology examination by tissue biopsy, but is difficult to perform because larva is mobile and also this investigation is invasive (Monshizadeh, Ashrafzadeh & Rumelt, 2000). The patient was proposed to surgery, but it was refused.

Because patient related episodes of recurrent conjunctivitis, even if fundus examination was normal, we consider that toxocara canis larva affect also patient eye, and in the future we should have careful attention with ophthalmological examinations, even optical coherence tomography (OCT). There are described in the literature cases of ocular toxocara canis lesions with important involvement of the eye, even visual loss or retinal detachment (Magnaval, Glickman, Dorchie & Morassin 2001). In our case extension of lesions was limited only at the conjunctiva.

This case report highlights the impact of a zoonosis on human health and force the clinician to suspect this diagnosis in his medical practice in order to treat appropriate this pathology, and also force the public health policies to protect humans by toxocara canis infection.

The seroprevalence of Toxocara is high is developing countries, as is Romania, but the reality is that is very difficult to prevent infection with Toxocara canis, because dogs defecate in soil and human ingest soil though pica behaviors, especially during childhood, or when soil gets on their hands during work or play. A lot of studies shown that soil in parks, private gardens are often contaminated with Toxocara canis eggs. There are guidelines for prevention of Toxocara canis published by CDC, the American Association of Veterinary Practitioners and Companion Animal Parasite Counsel, and according to this for prevent Toxocara canis contamination, dogs should be de-wormed, kept on a leash and also dog feces cleaned up. In other words, dogs should be excluded

from parks, personal gardens, housing in order to prevent toxocara canis infection. Our case report confirm possession of a personal dog live in his garden. (Jones, Kruszon-Moran, Won, Wilson & Schantz , 2008).

The understandings of public health organizations to develop appropriate prevention and control strategies to prevent toxocara canis transmission, will facilitating an health improvement status.

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