

CASE REPORT

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Toxocariasis Resulting in Seeming Allergy

Rosanna Qualizza¹, Raffaella Megali², and Cristoforo Incorvaia³

¹Allergy Service, Istituti Clinici di Perfezionamento, Milan, Italy

²Rehabilitative Medicine, Istituti Clinici di Perfezionamento, Milan, Italy

³Allergy/Pulmonary rehabilitation, Istituti Clinici di Perfezionamento, Milan, Italy

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ABSTRACT

Toxocara canis is an intestinal nematode affecting dogs and cats that causes human infestations by ingestion of embryonated eggs excreted in dogs' faeces. Humans are transport hosts, in whom the larvae do not develop to adult worms, but may migrate to various tissues and organs, and survive for several years, giving rise to several clinical symptoms, which include allergy-like presentations.

We report three cases presenting as dermatitis, rhinitis, asthma, and conjunctivitis which were diagnosed and unsuccessfully treated as allergy. The correct diagnosis was established after detecting anti-*Toxocara* antibodies by Western blotting. All clinical symptoms showed improvement after starting treatment with mebendazole and subsequent courses of the antiparasitic drug resulted in full recovery. This suggests the possible role of *Toxocara canis* in inducing chronic symptoms of allergic type. This is particularly important for asthma, where it has been demonstrated that *Toxocara canis* infection causes allergic inflammation in the lungs associated with bronchial hyperreactivity. On the other hand, in our patients with asthma and with dermatitis the positive results from allergy tests were a confounding factor in delaying the correct diagnosis, which was finally obtained by the detection of antibodies to *Toxocara canis*.

Key words: Asthma; Allergy; Dermatitis; *Toxocara canis*

INTRODUCTION

Toxocara canis is an intestinal nematode affecting dogs and cats, that causes human infestations by ingestion of embryonated eggs excreted by dogs' faeces. This can easily occur in public parks and children sandboxes, where contamination is estimated

to be 20-30%.^{1,2} Humans are transport hosts, in whom the larvae do not develop to adult worms, but may migrate in various tissues and organs and survive for several years, giving rise to a number of clinical symptoms.³ In Europe, the prevalence of Toxocariasis, assessed by serological testing, varies from 2.5% in Germany to 37% in Spain.⁴ Recent epidemiological data from different world areas indicate a prevalence of less than 1% in New Zealand,⁵ about 8% in Iran,⁶ 14% in the U.S.A,⁷ and 27% in Brazil.⁸

Clinical manifestations are variable: *ocular larva migrans* involves only the eyes, while *visceral larva*

Corresponding Author: Rosanna Qualizza, PhD;
Istituti Clinici di Perfezionamento Via Farini, 9 - 20158 Milano,
Milan, Italy. Tel:(026) 4445 644; Fax:(026) 4445 609, E-mail:
rosanna.qualizza@icp.mi.it

migrans affect the major organs, resulting in fever, malaise, asthenia, urticaria, nodules, cough, wheeze. Clinical manifestations may also involve the heart, the liver, and the brain.² Some *Toxocara* antigens induce a Th2-type lymphocyte response with production of IL-4 and consequent switching of B-cells to synthesize IgE antibodies, and of IL-5 with differentiation and activation of eosinophils.⁹ The latter phenomenon should explain cases of hypereosinophilia otherwise undetermined.¹⁰ Concerning clinical manifestations, some studies reported a relationship between *Toxocara* infestation and allergic sensitisation, as assessed by high serum IgE levels and positive skin prick tests,¹¹ asthma,¹² and rhinitis.¹³ For asthma, a recent study demonstrated in an experimental model that *Toxocara canis* infection causes allergic inflammation in the lungs associated with bronchial hyperreactivity.¹⁴ This finding led Cooper to reconsider the role of *Toxocara canis* as a neglected environmental risk factor for asthma not only in developing but also in industrialized countries.¹⁵

In fact, *Toxocara* infestation is commonly overlooked in subjects with symptoms of suspected allergy, who often undergo a large number of diagnostic procedures but not laboratory investigation for anti-*Toxocara* antibodies. In this regard, it is important, that when the commonly used immunoenzymatic assays¹⁶ are negative, the more sensitive Western blotting assay be performed.¹⁷

Here we report three cases of toxocariasis erroneously diagnosed as allergy, which presented as dermatitis, asthma, and conjunctivitis, respectively.

Case 1

The patient was a 46 year-old woman suffering from dermatitis with itching. The primarily skin manifestations began 30 years ago, when the patient was still a teenager. Dermatitis was located around the eyes. She consulted an Allergy unit and patch tests were performed, resulting positive for benzocain, paraphenylendamine, balsam of Peru, and dyaminodiphenilmethane. She consequently avoided contact with products containing these haptens, without any clinical improvement. Only treatment with oral corticosteroids proved effective in clinical manifestations. During the subsequent years, recurrent labial and palpebral oedema and episodes of generalized urticaria, apparently related to NSAIDs and beta-lactam assumptions occurred. She consequently performed further allergy evaluation, with the following results:

positive skin prick tests (SPT) for egg, milk, cereals, potato, carrot, tomato, celery, banana, apple, peach, and prune extracts; positive patch test for balsam of Peru, parabens and fragrance mix. The exclusion of foods and products containing these haptens did not modify clinical symptoms. She also performed blood examinations including auto-antibodies, specific IgE to other foods and to beta-lactam antibiotics, with negative results. Only the measurement of total IgE showed an increase level of 156 kU/L, (normal value <120 kU/L).

The patient was referred to our Allergy Unit in January 2005, because of worsening of both dermatitis and angioedema. During the consultation she had an important angioedema and erythema around the eyes and at the zygomatic region. SPT with food extracts and fresh foods gave positive results for egg, barley, fennel, potato, tomato, orange, peach, kiwi, and melon, with *in vitro* confirmation by RAST only for barley (0.83 kU/L), potato (0.41 kU/L) and melon (0.37 kU/L). SPT with inhalant allergens were also done to assess possible cross-reactivities between foods and aeroallergens, with positive results to grass, mugwort, ragweed, elder, and *Parietaria* pollen, as well as to cat and dog epithelia, and to *Alternaria*. RAST was positive only to grass pollen (6.97 kU/L) and cat epithelium (1.32 kU/L), and total IgE amounted to 121 kU/L. A second evaluation in June 2005 included complete blood examination with parasitologic testing, ELISA and Western blotting for *Toxocara canis*. This last test resulted positive. Antiparasitic treatment with mebendazole 100 mg one tablet b.i.d. for three days, repeated for three courses at monthly interval resulted in complete remission of symptoms. Repetition of Western blotting showed the absence of anti-*Toxocara* antibodies.

Case 2

The patient was a 37 year-old woman, suffering since 10 years from intermittent rhinitis and, since 7 years, also from persistent asthma. In the first two years asthma was present only in the cold season (November to February) but later asthmatic episodes occurred also in spring and summer.

In 2003 allergy tests performed elsewhere gave positive results to house dust mites (SPT ++, CAP system 0.49 kU/L) A bronchial nonspecific challenge with methacholine showed a moderate reactivity, with a Pd20 of 425,028 mcg (the test is negative when values are above 1200 mcg). A treatment by inhalant route with an association long-acting beta2-agonist plus

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corticosteroid was prescribed. The patient was referred to our Allergy service in October 2004 because of insufficient control of asthma.

Allergy testing gave positive results to SPT with *Dermatophagoides pteronyssinus* (++) , *Dermatophagoides farinae* (++) , cat epithelium (++) , ragweed (+++), and elder (+), and to CAP system with *Dermatophagoides pteronyssinus* (2.05 kU/L), *Dermatophagoides farinae* (1.84 kU/L), and cat epithelium (0.69 kU/L). Also routine blood examination, with no pathologic finding, and parasitologic evaluation by Western blotting were performed. The latter showed the antibody binding to bands specific for *Toxocara canis*. A methacholine challenge was repeated, showing higher bronchial reactivity in respect to the previous challenge (Pd 20: 343,262 mcg). Treatment with mebendazole 100 mg one tablet b.i.d. for three days achieved an initial improvement of asthmatic symptoms, and subsequent 6 courses at monthly intervals led to further recovery, which was allowed to gradually to reduce, starting from 4 months, the drug treatment, until the complete discontinuation in December 2005. At a control visit in November 2006 the patient was free of asthmatic symptoms without using drugs and had normal spirometric values and a negative methacholine challenge. Western blotting showed no antibodies to *Toxocara canis*. This clinical status was maintained also at the latest control visit in November 2007.

Case 3

The patient was a 39 year-old woman suffering from persistent conjunctivitis, for three years with episodes of recurrent blepharitis.. Symptoms started in spring 2004 with tearing, an ocular redness and itching. After spontaneous remission, conjunctivitis reappeared in spring 2005 and led the patient to refer to an allergist, who performed skin tests and *in vitro* IgE tests for

seasonal allergens with negative results. Differently from the first year, ocular symptoms did not recede and was accompanied by episodes of blepharitis around the year. The patient was visited by various ophtalmologists, who prescribed a number of eyedrops with no apparent results. The latest ophtalmologist, following a more detailed clinical history, found that the patient had a cat in her home for 8 years, and thus required a new allergologic evaluation. We performed SPT with the standard panel of environmental allergens, with negative results; also CAP system for cat and house dust mites were negative, total IgE value was 55 kU/L. Routine blood examination detected an eosinophilia (10.3%), suggesting to perform ELISA and Western blotting for *Toxocara canis*, the latter showing positive results.

Treatment with mebendazole 100 mg one tablet b.i.d. for three days repeated after 20 days achieved a significant improvement of conjunctivitis, with complete recovery following two further courses with the same dosage. The patient is thus far asymptomatic and developed a negative Western blotting for *Toxocara canis*. The changes in laboratory data in the three patients are reported (Table 1).

CONCLUSIONS

The cases we presented suggest the possible role of *Toxocara canis* in inducing chronic allergic-like symptoms. This is particularly important for asthma, where it was recently demonstrated that *Toxocara canis* infection causes allergic inflammation in the lungs associated with bronchial hyperreactivity.¹⁴ In our asthmatic patient there was in fact bronchial hyperreactivity, assessed by methacholine challenge, that disappeared only following anti-*Toxocara* treatment. On the other hand, in the patient with asthma as well as with dermatitis, positive results from allergy tests were a confounding factor in delaying the correct diagnosis, which was finally obtained by the detection of antibodies to *Toxocara canis*.

Table 1. Laboratory data in the three patients

Patients	Total IgE (kU/L)		Eosinophils (%)		Antibody to <i>Toxocara</i> by ELISA		Antibody to <i>Toxocara</i> by Western blotting	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
1	121	108	9.5	8.6	Negative	Negative	Positive	Negative
2	732	365	1.3	1.7	Negative	Negative	Positive	Negative
3	55	87	10.3	7.8	Negative	Negative	Positive	Negative

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