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CLINICAL OBSERVATION

Human toxocariasis: a report of nine cases

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Abstract

Aim: Human toxocariasis is caused by infection with the larval stage of nematode parasites of dogs and cats, *Toxocara canis* or *Toxocara cati*. These helminths are not able to complete their life cycle in undefinitive hosts and so undergo aberrant migrations in the tissues causing a wide spectrum of signs and symptoms. Eosinophilia is often severe and sometimes represents the only sign of infection, except in ocular and neurological forms.

Methods: We describe the clinical features of nine children affected by toxocariasis admitted to our Infectious Diseases department from 2004 to 2006.

Results: Fever and hepatomegaly were the most common clinical findings. In two cases eosinophilia was not present. Diagnosis was performed by enzyme-linked immunosorbent assay employing excretory–secretory antigens of *Toxocara. canis* larvae. All patients were successfully treated with oral albendazole with no side effects.

Conclusion: Toxocariasis should be considered in differential diagnosis of eosinophilia and in patients with seizures of uncertain origin, isolated hepatomegaly and splenomegaly, bronchospasms or skin rash.

INTRODUCTION

Human toxocariasis is caused by infection with the larval stage of *Toxocara canis* or *Toxocara cati,* which are nematode parasites of dogs and foxes (*T. canis*), and cats (*T. cati*), respectively. Immature eggs are expelled in the faeces mostly by puppies and become infectious developing in the surrounding environment within 2 to 4 weeks. Infective larvae can be found in the faeces of those puppies infected transplacentally (1).

Human infection is more frequent in children less than 5 years of age and is due mainly to contact with contaminated soil or infected puppies (2). Consumption of raw meat from infected chicken, cattle and swine has also been associated with toxocariasis especially in adults (3).

Even if there are known cases of complete maturation (4), generally these helminths are not able to complete their life cycle in humans and so undergo prolonged, aberrant migrations or locate abnormally in the tissues as underdeveloped larvae, stimulating an eosinophilic inflammation (visceral larva migrans syndrome). Clinical manifestations range from no symptoms to eosinophilia or lung, hepatic, ocular or neurological involvement (2). Eosinophilia is often severe and sometimes represents the only sign of infection, while in ocular and neurological forms it may be modest or absent.

Usually parasites are not able to mature to the adult stage, thus stool examination for the parasite and its eggs is unsupportive. Direct parasitologic diagnosis by biopsy is extremely difficult to achieve, thus serological methods are the mainstay for the diagnosis. Toxocariasis is prevalent wherever dogs are found. According to recent studies, in Italy 63.5% of soil samples examined contained *Toxocara spp.* eggs (5). About 80% of 4 months puppies and 22% of adult dogs were found infected (6). Prevalence in humans varies widely depending on the population tested. However, the disease is probably underestimated.

We describe the clinical features of nine children affected by toxocariasis admitted to our Infectious Diseases department from 2004 to 2006.

CASE REPORT

Our patients were six boys and three girls aged from 2 to 11 years, coming from different areas of western Sicily. Patients' parents gave informed consent to the work. Signs and symptoms of affected children are reported in Table S1 (in Supplementary Material online).

In seven patients clinical suspicion was based on: (a) risk factors for toxocariasis like exposure to potentially contaminated soil and pica; (b) clinical findings suggestive for toxocara infection; (c) absolute eosinophil count $\geq 300/mm^3$ with no history of atopy or intestinal helminthiasis. Patients 4 to 9 were affected by covert toxocariasis with a lower eosinophil count than patients 1 to 3. Particularly patient 7 and patient 8 showed neither the typical syndrome nor eosinophil count $>300/mm^3$. Toxocariasis was hypothesized because of the recurrence of skin rash (patient 7) or bronchospasms (patient 8) episodes without any recognized allergic cause.

Diagnosis was performed by the detection of specific antibodies by enzyme-linked immunosorbent assay (ELISA) employing extracts of larval excretory-secretory (LES) antigens of *Toxocara canis* (LMD Toxocara serology Alexon-Trend Inc). In all positive cases, a cross-reaction caused by other Ascarididae was excluded by stool examination. Chest X ray examination performed on all seropositive patients

showed a pulmonary infiltrate in a child with respiratory symptoms (patient 9).

All patients were treated with oral albendazole 15 mg/kg once daily for 8 days. Prednisone at an oral dose of 0.5 mg/kg daily was coadministered for the first 5 days of therapy to prevent allergic reactions due to accelerated larval lysis. A rapid improvement of both symptoms and laboratory findings was obtained and no side effects were complained. Specific antibodies titre became negative within 1 year after treatment.

DISCUSSION

Toxocariasis is believed to be the second most common helminth infection in developed countries after oxyuriasis. In industrialized countries the even more common spreading of pets and consequently of their parasites could cause the increasing of some zoonoses characterized by low human pathogenicity (toxocariasis, but also ocular dirofilariosis, toxoplasmosis, etc.) (7).

In our case series the typical clinical presentation characterized by fever, hepatomegaly and eosinophilia was observed only in two cases (22.2%). The high percentage of patients without fever (44.4%) reminds that toxocariasis has to be taken into consideration even in the differential diagnosis of isolated hepatomegaly and splenomegaly, bronchospasms or skin rash.

Severe complications are rare, nevertheless central nervous system invasion (8), serosal effusions (9) and liver abscess (10) have been described in untreated patients.

Diagnosis is suggested by clinical manifestations and laboratory findings (eosinophilia or leukocytosis). Direct diagnosis obtained by finding larvae in the affected tissues by histological examination is fortuitous due to the parasite's very small size, and not recommended. The ELISA employing LES product has a reasonably high sensitivity (approximately 78%) and specificity (approximately 92%), even in *T. cati* infection and is considered the best indirect test for diagnosis (2).

Antibodies to LES antigen can also be detected in different fluids, such as bronchoalveolar lavage fluid and vitreous and aqueous fluid (3).

In conclusion, we suggest that toxocariasis should be considered in differential diagnosis of eosinophilia together with other more frequent causes (idiopathic eosinophilia, atopy, intestinal parasitic infection). Patients with isolated hepatomegaly and splenomegaly, bronchospasms or skin rash should also be investigated for *Toxocara* infection. Toxocariasis should be suspected also in patients with unilateral loss of vision and suspicious ophthalmic lesions, or in presence of seizures of uncertain origin. In fact eosinophilia is inconstant in ocular and neurological forms, as mentioned above.

Moreover an active surveillance of *Toxocara* infection in pets could be useful to prevent children disease.

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Supplementary material

The following supplementary material is available for this article:

Table S1 Clinical and laboratory signs of 9 children with toxocariasis

This material is available as part of the online article from: http://www.blackwell-synergy.com/doi/abs/10.1111/j.1651-2227.2008.00902.x
(This link will take you to the article abstract).

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