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Neurotoxocariasis: A Literature Review

Deshayes S[1], **Bonhomme J**[2] **and de La Blanchardiere A**[3*]

[1]*Service de Médecine Interne, CHU Cote de Nacre, avenue Cote de Nacre, 14033 Caen Cedex 9, France*

[2]*Service de Microbiologie, CHU Cote de Nacre, avenue Cote de Nacre, 14033 Caen Cedex 9, France*

[3]*Service des Maladies Infectieuses et Tropicales, CHU Cote de Nacre, avenue Cote de Nacre, 14033 Caen Cedex 9, France*

[*]**Corresponding author:** Arnaud de La Blanchardiere, Service des Maladies Infectieuses et Tropicales, CHU Cote de Nacre, avenue Cote de Nacre, 14033 Caen Cedex 9, France, Tel: +33633189289; Fax: +33231065439; E-mail: delablanchardiere-a@chu-caen.fr

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Abstract

Introduction: Toxocariasis is a widespread zoonosis, which may result in central nervous system injury.

Objectives: To discuss the epidemiology, the clinical manifestations and the drug therapy of neurotoxocariasis described in literature through a systematic review.

Methods: We conducted a systematic literature review of MEDLINE, SciELO, ScienceDirect and Google Scholar up to April 2015 using a combination of the following search terms: “neurotoxocariasis” or “neurotoxocarosis”, “toxocariasis” or “toxocarosis”, and “cerebral” or “neurologic”.

Results: One hundred cases of neurotoxocariasis were identified in literature. The majority of patients were male (58%), with a median age of 42 years. The predominant clinical pictures were myelitis (60%), encephalitis (47%) and/or meningitis (29%). Fever was inconstant (23%). The suspected mode of transmission, mentioned in only 49% of cases, was mainly contact with dogs and/or cats (67%) and ingestion of contaminated food (31%). Diagnostic imaging examinations were often abnormal, with hypodense lesions in cerebral scanner sequences and hyperintense lesions in cerebral MRI T2-weighted sequences in 65% and 57% of encephalitis cases respectively, and in 92% of myelitis cases in medullary MRI T2-weighted sequences. The detection of antibodies against Toxocara spp. was almost constant in blood and cerebrospinal fluid (CSF), 99% and 93% respectively. The two most commonly used drugs were corticosteroids (72%) and/or albendazole (68%) for a period of at least 3 weeks, which often needed to be repeated. Despite a low mortality rate (6%), complete remission was observed in only 40% of cases.

Conclusion: Neurotoxocariasis, a completely preventable zoonosis, could lead to severe sequelae failing prompt diagnosis. A compatible clinical picture, presence of risk factors, blood eosinophilia and high titers of antibodies against Toxocara spp. in CSF should alert physicians to this poorly understood disease.

Keywords: Neurotoxocariasis; Neurotoxocarosis

Introduction

The genus Toxocara includes *T. canis* and *T. cati*, whose definitive host is the upper digestive tract of dogs, foxes and cats [1]. The prevalence of infection in dogs with adult Toxocara worms is about 25% in Western countries, while the rate in cats in France is 30 to 60% [2]. This high prevalence together with the high fecundity of Toxocara, as well as the increasing number of pets in Western countries, explain the high level of soil contamination with Toxocara eggs in parks, playgrounds, and other public places [2]. The adult female sheds up to 200,000 unembryonated eggs a day, which can become infectious in soil under appropriate conditions after an incubation period of 1-2 weeks [3,4].

Human infection is a result of accidental ingestion of embryonated eggs from soil (via geophagy which is a specific type of pica, contaminated hands or onychophagy) or ingestion of infected raw fruits and vegetables [5,6,9,10]. Humans are less frequently infected by ingesting larvae via raw or undercooked meat or giblets from paratenic hosts, such as chickens, cows, ducks, deer, pigs, sheep, rabbits, or ostriches [2,3,6–8].

These embryonated eggs then hatch in the small intestine and release immature larvae [3], which penetrate the small intestine mucosa, migrate to the liver via the portal circulation, then lungs and left heart, from where they disseminate via the systemic circulation, especially to muscles, optic nerves and, in rare cases, the central nervous system. They migrate through the bloodstream, are arrested in small caliber vessels and can migrate into surrounding tissues, giving rise to the name visceral larva migrans. Toxocara larvae cannot develop to adult worms in humans [1,5,6,11,12].

Toxocariasis is a cosmopolitan disease, probably the most common zoonotic helminthiasis in temperate climates, which mainly affects children under 10 years of age because of their play habits and their tendency to put their fingers in their mouths [1,13]. The exact prevalence of toxocariasis is difficult to assess as most infections remain asymptomatic [4]. Seroprevalence is 2-5% in urban areas and 14-37% in rural areas of the United States and Europe, but may range from to 39 to 93% in tropical regions [2,4,6,11,14–16]. Such variations

can be explained by poor hygiene, the rate of infected dogs and cats, and their access to public places, rare administration to dogs and cats of antihelminthics, children's playing habits which facilitate transmission, and the humid climate which favors the survival of eggs [4,16,17].

Toxocariasis can present itself as visceral larva migrans, ocular larva migrans, neurotoxocariasis, and most frequently as "covert toxocariasis" in children (fever, headache, behavioral and sleep disturbances, cough, anorexia, abdominal pain, hepatomegaly, nausea and vomiting) and as "common toxocariasis" in adults (weakness, pruritus, rash, breathing difficulties and abdominal pain) with the latter two defined solely by serum conversion and unspecific symptoms [15,17]. The typical clinical presentation of visceral larva migrans combines general signs (hyperthermia, anorexia, body weight loss, malaise) with plurivisceral involvement in children, mainly pulmonary (cough, sputum, dyspnea with bronchospasm, recurrent bronchitis and/or pneumonia, pleural effusion), hepatic (hepatomegaly), ocular, gastrointestinal (diarrhea, nausea, vomiting), cutaneous (rash, hives, angioedema), cardiac, rheumatologic and/or lymphatic [8,18–22]. Symptoms depend on the number of ingested larvae, host sensitivity and immune status, affected tissues, previous exposure to larvae, host age, and persistent sources of contamination [8,9,23]. Mortality is very low and mainly the result of pulmonary or cardiac involvement [12].

This disease is responsible for non-specific biological abnormalities, including hyperleukocytosis, polyclonal hypergammaglobulinemia predominant on immunoglobulin E, increased isohemagglutinin A and B titers, elevated erythrocyte sedimentation rate, hepatic cytolysis or cholestasis, and eosinophilia [13,24]. Growth arrest of larvae in humans renders useless stool examination for eggs and larvae [24]. Diagnostic confirmation is based on serology mainly by ELISA using Toxocara excretory-secretory antigens. Its sensibility ranges from 73 to 90%, and its specificity is about 93% (due to cross-reactivity with Fasciola hepatica and Ascaris suum), requiring confirmation by Western blot, which does not distinguish between *T. canis* and *T. cati* [6,15,25]. The latter is considered to have an absolute specificity when considering lower molecular weight bands [6]. The relevance of nucleic acid detection methods by polymerase chain reaction is still under consideration, but would allow interspecies distinctions [15,26,27]. Direct diagnosis by excision of larvae is exceptional [4,12,28–36].

Although Toxocara larvae are frequently found in brain of experimental animal models, central nervous system injury in humans is rare, unpredictable and poorly known [6,11,16,17,37–39]. Peripheral nervous system involvement has been reported, such as Bell's palsy, with a doubtful relationship, and thus was not included in this review [40,41]. Likewise, epilepsy was not included, because its high prevalence, about 5 to 10 per 1,000 population, associated with the high seroprevalence of toxocariasis, precludes any definitive association [42]. A meta-analysis of retrospectives studies showed a weak positive association between Toxocara spp. seropositivity and epilepsy, with a common odds ratio of 1.92, without possibility to demonstrate a temporal relationship [17]. Furthermore, it is known that some psychiatric comorbidities are both risk factors for development of epilepsy and for geophagy, and that children suffering from epilepsy and/or mental retardation are responsible for a high number of falls to the ground, two conditions that predispose them to Toxocara spp. exposure [17,42].

No literature review on neurotoxocariasis has been published since 2007 [11]. Through a systematic review of 100 published cases of neurotoxocariasis, we discuss the epidemiology, the clinical manifestations and the drug therapy of neurotoxocariasis.

Methods

We have performed a systematic review of literature in English, Spanish, Portuguese, and French, from the first described case in 1951 to April 2015, via an electronic search of Pubmed, Scielo, ScienceDirect and Google Scholar using the following key words: "neurotoxocariasis" or "neurotoxocarosis", "toxocariasis" or "toxocarosis", and "cerebral" or "neurologic". We also retrieved articles from the reference lists of papers found via our searches. Meeting abstracts published in supplements of medical journal were included. Categorical variables were reported as percentages with their 95% confidence interval and continuous variables were expressed as medians.

Sex ratio (Male/Female)	1.49 / 97	
Median age (years) [range]	42 / 100 [7 months - 79 years]	
Suspected mode of transmission §	Contact with dogs or cats (%) [95% CI]	33 / 49 (67.3) [54.2-80.5]
	Contaminated food (%) [95% CI]	15 / 49 (30.6) [17.7-43.5]
	Geophagy (%) [95% CI]	2 / 49 (4.0) [0-9.6]
	Not found or not mentioned (%) [95% CI]	51 / 100 (51) [41.2-60.8]
Myelitis signs § (%) [95% CI]		60 / 100 (60) [50.4-69.6]
	Sensation disorders (%) [95% CI]	45 / 100 (45) [35.2-54.8]
	Motor disorders (%) [95% CI]	35 / 100 (35) [25.7-44.3]
	Autonomic disturbances (%) [95% CI]	32 / 100 (32) [22.9-41.1]
Encephalitis signs § (%) [95% CI]		47 / 100 (47) [37.2-56.8]
	Focal deficits (%) [95% CI]	33 / 100 (33) [23.8-42.2]
	Confused state (%) [95% CI]	23 / 100 (23) [14.8-31.2]
	Seizure (%) [95% CI]	14 / 100 (14) [7.2-20.8]
	Cognitive disorders (%) [95% CI]	11 / 100 (11) [4.9-17.1]
Meningeal signs § (%) [95% CI]		29 / 100 (29) [20.1-37.9]
	Headaches (%) [95% CI]	22 / 100 (22) [13.9-30.1]
	Stiff neck / Neck pain (%) [95% CI]	15 / 100 (15) [8.0-22.0]

	Nausea or vomiting (%) [95% CI]	8 / 100 (8) [2.7-13.3]
	Kernig's / Brudzinski's sign (%) [95% CI]	3 / 100 (3) [0-6.3]
Hyperthermia (%) [95% CI]		23 / 100 (23) [14.8-31.2]
Other signs of visceral larva migrans (prior/ contemporaneous) (%) [95% CI]		22 / 100 (22) [13.9-30.1]

Table 1: Epidemiologic and clinical parameters of 100 cases of Neurotoxocariasis.

§ Total count may exceed 100% (one patient may have multiple occurrences).

Results and Discussion

Since the first publication of neurotoxocariasis by Beautyman and Woolf in 1951 [29], recognized as such in 1966 [43], one hundred cases of neurotoxocariasis have been published in sixty-six articles [3–9,13,14,16,20,23,26,28,33–39,44–88] and eight meeting abstracts [89–96] (Table 1).

Six other articles were not included because Toxocara larvae were detected accidentally at autopsy [12,30–32,43,97]. The majority were case reports, with the largest series including 17 cases of Toxocara canis myelitis in Lebanon [6].

Diagnosis of neurotoxocariasis is based on a body of arguments, such as a compatible clinical picture, the presence of risk factors, eosinophilia in blood and/or cerebrospinal fluid (CSF), high titers of antibodies against Toxocara spp. in blood and/or CSF, clinical and/or radiological improvement after antihelminthic therapy, and absence of any alternative diagnoses [5,6,38].

Although visceral larva migrans is mainly a childhood disease, neurotoxocariasis predominantly affected middle-aged male subjects (sex-ratio 1.49, median age 42 years old) and less frequently children <18 years (19%) [39] (Table 1 and Figure 1). The main suspected mode of transmission was contact with dogs and cats (67.3%), followed by ingestion of contaminated food (30.6%) and geophagy or pica (4%). Interestingly, no mode of transmission could be found or was mentioned in 51% of cases (Table 1). Only one patient was immunocompromised [85].

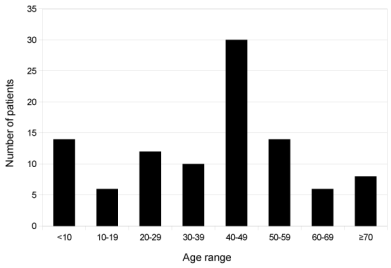

Figure 1: Distribution by age at diagnosis of neurotoxocariasis.

The main clinical pictures of neurotoxocariasis were myelitis (60/100), encephalitis (47/100), and meningitis (29/100) with frequent associations (Table 1). In detail, we found clinical pictures of 45 isolated myelitis (45%), 18 isolated encephalitis (18%), 16 meningoencephalitis (16%), 8 encephalomyelitis (8%), 6 isolated meningitis (6%), 5 meningoencephalomyelitis (5%), and 2 meningomyelitis (2%). Fever was very inconstant (23/100). Diagnosis could be delayed from 2 weeks to 9 years [6,35–37,55,83,91].

Blood eosinophilia in neurotoxocariasis was present in only 65.6% of cases. CSF abnormalities were frequent: pleocytosis (62.5%), with a predominance of eosinophils, an elevated protein level (54.5%) and in rare cases a low glucose level (6.9%) [27,50] (Table 2).

Median eosinophil count (mm^3) [range]		820 / 65 [40-17750]
Eosinophilia¶ (%) [95% CI]		59 / 90 (65.6) [55.7-75.4]
Median WBC count* (mm^3) [range]		9200 / 44 [4000-25000]
Hyperleukocytosis*¤ (%) [95% CI]		21 / 48 (43.7) [29.7-57.8]
Elevated CRP# (%) [95% CI]		8 / 13 (61.5) [35.1-88.0]
HyperIgEemia# (%) [95% CI]		10 / 12 (83.3) [62.2-100]
Lumbar puncture	Median cell count (mm^3) [range]	13 / 61 [0-1420]
	Pleocytosis@ (%) [95% CI]	45 / 72 (62.5) [51.3-73.7]
	Median protein level (g/l) [range]	0.47 / 54 [0.175-3.2]
	Hyperproteinorachia+ (%) [95% CI]	36 / 66 (54.5) [42.5-66.6]
	Median glucose level (g/l) [range]	0.6 / 43 [0.14-0.88]
	Hypoglycorachia° (%) [95% CI]	4 / 58 (6.9) [0.4-13.4]
Positive serum antibodies to Toxocara# (%) [95% CI]		90 / 91 (98.9) [96.8-100]
Positive antibodies to Toxocara in cerebrospinal fluid# (%) [95% CI]		57 / 61 (93.4) [87.2-99.7]
Cerebral tomodensitometry abnormalities (%) [95% CI]		15 / 23 (65.2) [45.8-84.7]
Cerebral Magnetic Resonance Imaging abnormalities (%) [95% CI]		35 / 61 (57.4) [45.0-69.8]
Medullary Magnetic Resonance Imaging abnormalities (%) [95% CI]		44 / 48 (91.7) [83.8-99.5]
Electroencephalogram abnormalities (%) [95% CI]		16 / 18 (88.9) [74.4-100]

Table 2: Biological, radiological and electrophysiological parameters of 100 cases of neurotoxocariasis.

* Under 12 years excluded

¶ defined by eosinophil count ≥ 500 mm^3 or considered as such by the authors in the absence of eosinophil count mentioned in the article

¤ defined by white blood cell count ≥ 10000 mm^3 or considered as such by the authors in the absence of white blood cell count mentioned in the article

defined by a value above the upper normal limit, mentioned in the article

@ defined by white blood cell count in cerebrospinal fluid ≥ 5 mm^3 or considered as such by the authors in the absence of white blood cell count in cerebrospinal fluid mentioned in the article

+ defined by protein level in cerebrospinal fluid ≥ 0.4 g/l or considered as such by the authors in the absence of protein level in cerebrospinal fluid mentioned in the article

° defined by glucose level in cerebrospinal fluid ≤ 0.4 g/l or less than 60% of glucose blood level or considered as such by the authors in the absence of glucose level in cerebrospinal fluid mentioned in the article.

It must be noted that one case could be documented by the presence of Toxocara DNA in CSF by polymerase chain reaction [78]. Antibodies directed against Toxocara spp. were positive in serum (98.9%) in all but one case [94], and in most cases in the CSF (93.4%) (Table 2). But serologic interpretation in blood (as well as blood eosinophilia) was difficult for patients who had lived in tropical areas, due to common antigenic determinants between different tropical nematodes and Toxocara spp. [1].

Diagnostic imaging examinations often identified abnormalities with an abnormal brain computed tomography (CT) in 65.2% of cases, an abnormal brain magnetic resonance imaging (MRI) in 57.4% of cases, and an abnormal medullary MRI in 91.7% of cases (Table 2). Brain CTs found multiple subcortical, cortical, or white matter circumscribed hypodense lesions [9]. These lesions were hypointense on T1-weighted magnetic resonance images and hyperintense on T2-weighted magnetic resonance images, with a homogenous enhancement after the administration of contrast agents, and are thought to correspond to granuloma and/or to ischemic lesions [9,54,76]. When specified, these lesions were mainly multiple (23/31, 74.2%) and supratentorial (19/28, 67.9%), then both infra and supratentorial (7/28, 25%). A focal meningeal contrast enhancement next to an active inflammatory lesion could be observed [26]. Brain MRIs could also identify areas of vasculitis and obstructive hydrocephalus [38,74]. Spine MRIs revealed a swelling and enlargement of a spinal segment, with a single iso- or hypointense lesion in T1 sequences and hyperintense lesion in T2 and FLAIR sequences, predominantly in the posterior and posterolateral segment of the spinal cord. This lesion was enhanced in a focal nodular pattern after injection of gadolinium, secondary to a break in the blood-brain barrier, which was probably associated with reactive inflammatory process or secondary demyelination [6,7,65]. During a follow-up MRI, this lesion could migrate [7,98].

Biopsies of the CNS were performed in 11 cases with 10 cerebral biopsies and 1 medullar biopsy which revealed granulomas with epithelioid cells, giant cells and eosinophils, predominantly in cerebral and cerebellar white matter around vessels, but also in the spinal cord, grey matter and meninges in 10 cases (91%) [4,16,33-34,36,64,79,87,88,90]. Larvae were found inside in only 4 cases (37%) [4,34-36].

From a physiopathological point of view, damage was assumed to be mainly due to the inflammatory response, rather than to the larva itself [16]. Several mechanisms have been discussed:

- A T helper type 2-driven immune response, activated by larval glycosylated proteins and leading to the secretion of interleukin-5 and immunoglobulin E [3,16];

- An immuno-allergic mechanism, mediated by type III hypersensitivity IgE-dependent [14];

- A vasculitis, secondary to endothelial injury caused by cationic proteins released by eosinophils and interleukin-6 and/or formation of immune complexes [72,74,84,99,100];

- Release of larval toxins, with a direct neuronal toxicity [74,76];

- Ischaemic lesions, linked to chronic inflammation with production of anticardiolipin antibodies, increase of factor VIII and release of Major Basic Protein and Eosinophilic Cationic Protein [62,101].

Treatment is still a matter of debate, due to a lack of controlled studies on neurotoxocariasis therapy [26]. The most frequently used drugs were albendazole (67.8%), thiabendazole (14.4%), diethylcarbamazine (13.3%), and mebendazole (10%) [54,73] (Table 3).

Treatment §	Albendazole (%) [95% CI]	61 / 90 (67.8) [58.1-77.4]
	Thiabendazole (%) [95% CI]	13 / 90 (14.4) [7.2-21.7]
	Diethylcarbamazine (%) [95% CI]	12 / 90 (13.3) [6.3-20.4]
	Mebendazole (%) [95% CI]	9 / 90 (10) [3.8-16.2]
	No antihelminthic	7 / 90 (7.8) [2.2-13.3]
	Median duration of antihelminthic therapy (days) [range]	21 / 53 [3-180]
	Corticosteroids (%) [95% CI]	69 / 96 (71.9) [62.9-80.9]
	Median duration of corticosteroids therapy (days) [range]	21 / 24 (71.9) [3-56]
Clinical outcome	Recovery without sequelae (%) [95% CI]	29 / 72 (40.3) [28.9-51.6]
	Partial recovery (%) [95% CI]	33 / 72 (45.8) [34.3-57.3]
	Stable clinical situation (%) [95% CI]	5 / 72 (6.9) [1.1-12.8]
	Deterioration of the clinical situation (%) [95% CI]	1 / 72 (1.4) [0-4.1]
	Death (%) [95% CI]	4 / 72 (5.6) [0.3-10.8]

Table 3: Treatment and clinical outcomes of 100 cases of neurotoxocariasis.

§ Total count may exceed 100% (one patient may have multiple occurrences)

Albendazole must be administered three times per day, due to a better pharmacological profile than other antihelminthic drugs (good penetration in CSF, low toxicity, high serum concentration of its active metabolite) [11,39]. It resulted in an improvement in 47% of patients with toxocariasis in the literature [2,26]. Thiabendazole use is hampered by its side effects (50%) but may have anti-inflammatory and immunomodulatory properties [2,54,61]. Improvement occurred in 50% of patients with toxocariasis in the literature [2,26]. Finally, diethylcarbamazine resulted in an improvement in 70% of patients with toxocariasis in the literature, but with a Mazzoti-like reaction in 10% of subjects [2,54].

The therapy duration must generally be between than 21 to 28 days and until complete resolution of the clinical symptoms and normalization of the MRI [26,98], although complete remission was observed without any antihelminthic treatment [45,66,75,88,93]. The therapy sometimes needed to be repeated [39,47,64,69,71,81,86].

Corticosteroids were often used simultaneously to decrease acute inflammatory and immunologic manifestations (71.9%) despite the lack of results confirming the superiority of this combined treatment to a single therapy [11,13,53,54] (Table 3). They also increased albendazole levels by approximately 50% [39]. In addition to antiepileptic drugs and surgery, other treatments have been tried, including plasmapheresis, intravenous immunoglobulins and azathioprine [47,92]. For post-treatment follow-up of toxocariosis, only the eosinophil count appeared helpful, with a significant decrease within a month of treatment, whereas the serum total IgE concentration and specific anti-Toxocara IgG by ELISA remained unchanged [2,9].

Neurotoxocariasis could lead to complete remission (40.3%) or neurological sequelae with residual sensory complaints, cognitive deficits or paresis [6,26,27]. Experimental neurotoxocariasis in mice suggested a progression toward chronic neurodegenerative disorders [27]. Four deaths (5.6%) could be attributed to central nervous system involvement [33,34,36,63] (Table 3).

Toxocariasis doesn't result in lifelong immunity and re-infestations may occur [25,50]. Thus, prevention remains paramount for this disease, based on individual measures (deworming pets, hand hygiene, washing of vegetables, avoiding raw or undercooked meat) and collective actions (deworming campaign for stray cats and dogs at 2 to 3 weeks of age and two times a year for adults, municipal orders to prevent pet dogs from entering parks and playgrounds and the need for owners to remove their pet's feces from public areas) [2,13,22].

Conclusion

Toxocariasis is a worldwide zoonosis responsible for variable clinical pictures. Central nervous system involvement could present predominantly as myelitis, sometimes as encephalitis or meningitis. Diagnosis of neurotoxocariasis remains difficult because it generally requires the following four findings: blood and/or CSF eosinophilia, hypodense lesions on brain CTs and/or hyperintense lesions on T2-weighted brain or spine MRIs, high titer of anti-Toxocara antibodies in blood and CSF, clinical and/or radiological improvement after antihelminthic therapy. Treatment should probably include a high dose albendazole, sometimes with corticosteroids, for an extended period. Despite a low mortality rate, neurotoxocariasis, whose therapy is not standardized, frequently leads to sequelae, probably due to late diagnosis. Prevention, based on individual measures and collective actions, remains the cornerstone of its management.

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