Toxocariasis of the central nervous system: with report of two cases

Toxocaríase do sistema nervoso central: com descrição de dois casos

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ABSTRACT

Clinical involvement of the nervous system in visceral larva migrans due to Toxocara *is rare, although in experimental animals the larvae frequently migrate to the brain. A review of the literature from the early 50's to date found 29 cases of brain involvement in toxocariasis. In 20 cases, various clinical and laboratory manifestations of eosinophilic meningitis, encephalitis, myelitis or radiculopathy were reported. We report two children with neurological manifestations, in which there was cerebrospinal fluid pleocytosis with marked eosinophilia and a positive serology for* Toxocara *both in serum and CSF. Serology* for Schistosoma mansoni, Cysticercus cellulosae, Toxoplasma *and cytomegalovirus were negative in CSF, that was sterile in both cases. Improvement of signs and symptoms after specific treatment (albendazole or thiabendazole) was observed in the two cases. A summary of data described in the 25 cases previously reported is presented and we conclude that in cases of encephalitis and myelitis with cerebrospinal fluid pleocytosis and eosinophilia, parasitic infection of the central nervous system should be suspected and serology should be performed to establish the correct diagnosis and treatment.*

Key-words: *Toxocariasis.* Toxocara canis. Eosinophilic meningitis. Eosinophilic encephalitis.

RESUMO

Envolvimento do sistema nervoso, com manifestações clínicas, na infecção pelo Toxocara *é raro, embora, nos modelos experimentais a larva freqüentemente se localize no sistema nervoso central. Uma revisão da literatura a partir de 1956, quando a síndrome foi descrita, até 2002, mostrou a publicação de 29 casos de neurotoxocaríase, dos quais em 20 havia relato de alterações clínicas e laboratoriais indicativas de meningite, ou encefalite, ou mielite ou radiculite eosinofílicas. Nessa comunicação estamos relatando observações em duas crianças que apresentaram sinais e sintomas neurológicos, com pleocitose e eosinofilia acentuada no líquor e com sorologia positiva para* Toxocara *no soro e no liquor. Sorologia para* Schistosoma mansoni, Cysticercus cellulosae, Toxoplasma *e citomegalovirus foram negativas no liquor, que era estéril nos dois casos. Houve melhora dos sinais e sintomas após o tratamento específico (albendazol e tiabendazol) nos dois casos. É apresentado um sumário dos principais achados nos casos relatados na literatura e se conclue que em casos de meningite, encefalite ou mielite com líquor apresentando pleocitose com eosinofilia acentuada, a suspeita de infecção parasitária deve ser levantada, sendo necessário sorologia especifica para diagnóstico e tratamento adequados.*

Palavras-chaves: Toxocaríase. *Toxocara canis. Meningite eosinofílica. Encefalite eosinofílica.*

The expression visceral larva migrans was first used by Beaver[5] to describe the syndrome associated with any infection caused by paratenic nematode larvae that migrate through organs. Although no consensus has been reached, some authors include the unusual migration of any nematode larvae, including those that naturally infect humans, as visceral larva migrans[44]. The syndrome is typically expressed by fever, persistent eosinophilia, hepatomegaly and pulmonary symptoms and usually results in a benign self-limited course[5].

Central nervous system involvement in visceral larva migrans syndrome is usually infrequent, but frequency can vary with different species of migrating larvae. Clinical involvement of the nervous system in visceral larva migrans due to *Toxocara* is rare, although in experimental animals the larvae frequently migrate to the brain [6].

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Recebido para publicação em 4/4/2003
Aceito em 16/2/2004

The frequency and localization of *Toxocara* larvae in the central nervous system in humans is unknown. Autopsy studies of isolated cases have revealed *Toxocara* larvae in leptomeninges [7], gray and white matter of cerebrum and cerebellum[15 26 27 40], thalamus[4] and spinal cord[7]. Most of these cases did not present clinical neurological signs. For this reason the clinical significance and true frequency of cerebral localization of *Toxocara* larvae in non-fatal cases remain unclear. At the Children's Hospital Nossa Senhora da Glória, in Vitoria, where the frequency of positive serology for *Toxocara* is around 30% of admissions[28], the frequency of granulomas due to larva migrans in the central nervous system was 0.68% in a random sample of 308 autopsies of children 1 to 15 years old (Musso et al: unpublished data)

Lewis et al[20] reviewed 58 cases of visceral larva migrans syndrome and found mention of convulsions in only three patients. In a case control study Magnaval et al[22] demonstrated that *Toxocara* infection is not associated with a recognizable neurological syndrome, although several cases had a positive Western blot for both cerebrospinal fluid and serum. However a significant association between seizures and positive serology for *Toxocara* has been reported in Italy[1] and in Bolivia[29].

A review of the literature from the early 50's to the present date found 29 cases of brain involvement in toxocariasis[2 3 4 7 8 11 12 13 14 15 17 19 26 27 30 31 34 35 37 38 39 40 43 45 47 48 49]. Of these 28 cases, 20 reported different clinical and laboratory manifestations of eosinophilic meningitis, encephalitis, myelitis or radiculopathy (the main data of each reported case are summarized in Table 1). Here we report two cases of *Toxocara* infection in which the most prominent manifestations were neurological, with cerebrospinal fluid eosinophilia and positive serology for *Toxocara* in both serum and cerebrospinal fluid.

CASE REPORTS

Case 1- A five-year-old girl was admitted with a four day flu-like, febrile illness, which was treated with aspirin. Three days later the child complained of abdominal pain and was vomiting with bloody streaks. After admission, endoscopy revealed acute hemorrhagic gastritis and the child received ranitidine. Five days after admission the child was lethargic, with slurred speech, nystagmus, right convergent squint and left deviation of labial commissure. There was paresis of both inferior and superior members, nuchal rigidity and bilateral Kernig and Lasègue responses. There were right paralyses of the VI, VII and XII cranial nerves, urinary retention and fecal incontinence. The cerebrospinal fluid contained: 54mg/dL glucose, 31mg/dL protein and 187 leukocytes/ul (2% monocytes, 41% lymphocytes and 57% eosinophils). The white blood cell count was 4900 leukocytes/ul (band neutrophils 79/ul, neutrophils 4582/ul, eosinophils 711/ul, lymphocytes 2370/ul, monocytes 158/ul). The blood and cerebrospinal fluid were sterile and because of the high level of eosinophils in the cerebrospinal fluid, a hypothesis of a parasitic meningoencephalitis was proposed. Stool examinations revealed larvae of *Strongyloides stercoralis*, but were negative for other helminths in the five samples examined. Treatment with thiabendazole was initiated. A second lumbar puncture was performed three days after the first puncture, and the results were similar, with a marked eosinophilic pleocytosis. Serology for *Toxocara* (ELISA IgG with secretory-excretory antigen) was positive in both the serum and cerebrospinal fluid. Serology for *Schistosoma mansoni*, *Cysticercus cellulosae* and toxoplasmosis were negative in the cerebrospinal fluid. Cranial MRI (performed 25 days after admission) showed small irregular lesions situated in the posterior portion of the spine-bulbar transition and pedunculus cerebellaris, with hyperintense signal in T2 and DP, but intermediary signal in T1. There was no enhancement after intravenous contrast, nor were there signs of tissue compression. An inflammatory lesion was suggested, most likely produced by *Toxocara* larvae. After 14 days of thiabendazole the child received albendazole for 10 days. Improvement was evident after the use of albendazole. The child was discharged 36 days after admission with discrete dysarthria and dysmetria and a mild paresis of VI and VII cranial nerves. Six months later the child presented without neurological manifestations.

Case 2 - A five-year-old boy was admitted with palsy of the inferior limbs and urinary retention. Cerebrospinal fluid was clear with 63 mg/dL glucose, 17.5mg/dL proteins and 23 leukocytes/ul (3% neutrophils, 57% eosinophils, 25% lymphocytes and 15% monocytes). The total leukocyte count was 22100/ul(442 myelocytes, 221 metamyelocytes, 1547 band neutrophils, 15028 neutrophils, 221 eosinophils, 4199 lymphocytes, 442 monocytes). Fecal examination was negative (five samples). Cerebrospinal fluid was sterile and presented negative serology for toxoplasmosis, *Schistosoma mansoni* and *Cysticercus cellulosae*. Corticotherapy was started until a positive serology for *Toxocara* was detected in both the serum and cerebrospinal fluid. Corticotherapy was replaced with thiabendazole for 15 days. Diagnosis of possible transverse myelitis produced by *Toxocara larvae* was noted. The child was discharged 34 days after admission with partial improvement of palsy. One month later the palsy had disappeared, and the child presented no signs of sequelae.

DISCUSSION

In both cases reported here there were signs of neurological lesions and pleocytosis with eosinophilia in sterile cerebrospinal fluid. In Case 1 MRI showed lesions in the spine-bulbar border and in the pedunculus cerebellaris. The localization of these lesions was compatible with the clinical manifestations. In Case 2 image examination was not performed. Sterile cerebrospinal fluid, with pleocytosis and marked eosinophilia, was an indication of the possible parasitic origin of the neural and meningeal lesions, reinforced by the positive serology for *Toxocara*.

Neural involvement by *Toxocara larvae* is highly probable in both cases if one takes into account: a) cerebrospinal fluid pleocytosis with marked eosinophilia; b) positive serology, with IgM anti-*Toxocara*, in both serum and cerebrospinal fluid, and negative serology for *Schistosoma mansoni* and *Cysticercus cellulosae* (parasites that reach the nervous system most frequently in Brazil); c) sterile cerebrospinal fluid and negative serology for other common infections in the central nervous system such as toxoplasmosis, syphilis and cytomegalovirus; and d) improvement of signs and symptoms after treatment

Table 1- Summary of cases of neurotoxocariasis reported from 1956 to 2002.

Author	Age/Gender	Summary of main observations
Cases studied at autopsy		
1-Dent et al 1956 [7]	1.5/M	Multiple granulomas with larvae in CNS
2-VanThiel 1960[47]	6.0/M	Granuloma with larvae (cerebellum)*
3-Moore 1962 [27]	2.0/M	Granuloma with larvae (cerebellum and medulla)
4-Schoenfield et al 1964[39]	5.0/M	Multiple granuloma and larvae in CNS*
5-Beautyman et al 1966[4]	6.0/F	Granuloma with larvae (thalamus)
6-Schochet et al 1967[40]	2.0/M	Multiple granulomas with larvae in CNS*
7-Mikhael et al 1974[26]	1.5/M	Multiple granulomas with larvae in CNS*
8-Hill et al 1985[15]	2.5/F	Larvae (cerebrum, cerebellum and pons)
9-Nelson J et al 1990[30]	3.0/M	Larvae and granulomas (cerebrum and liver)
Cases with clinical data		
1-Sumner & Tinsley 1967[46]*	57/F	Mental confusion. Bilateral extensor plantar responses. CSF: normal. Blood: 4606 eosinophils/μl. Liver biopsy: eosinophilic granuloma with nematode larva (604 um length and 54 mm wide). Serology for *Toxocara* was not performed.
2-Kapur et al 1976[17]**	22/M	Behavior changes, decreased consciousness, hyperreflexia. Nematode larva identified in brain (biopsy).
3-Engel et al[11] 1967	25/M	Meningomyelitis. CSF 80 cells/μl17% eosinophils. Precipitating anti-*Toxocara* antibodies in the serum
4-Anderson et al 1975[2]	1.5/F	Progressive weakness of right arm and leg. Blood eosinophils: 5220/μl. CSF: 356 cells/μl, 80% eosinophils. Serology for *Toxocara* positive in blood and CSF.
5-Wang et al 1983[49]	43/F	Acute retention of urine. Neck rigidity, lower limb weakness, brisk tendon reflexes and flexor plantar responses. CSF 8d after admission: 52 cells/μl 95% lymphocytes. Larva compatible with *Toxocara* detected in the CSF. Improvement after treatment with thiabendazole.
6 -Gould et al 1985[14]	11/F	Pronounced meningism: Kernig's sign positive and generalized hyperreflexia. CT scan was normal. CSF: 150 cells/30% eosinophils. Blood: 1300 eosinophils/μl. Serology positive for *Toxocara*. Although spontaneous recovery the patient was treated with diethylcarbamazine.
7-Russeger & Schmutzhard 1989[37]	55/F	Severe paraparesis. Myelography: space occupying lesion T7-T11. CSF: 177 cells/μl. Blood: 291 eosinophils/μl. Positive serology for *Toxocara* in the blood. Epithelioid granuloma with foreign body type giant cell in the biopsy.
8-Ruttinger & Hadidi 1991[38]	26/F	Epileptic seizures. MRI: multiple hyperintense, irregular lesions in CNS. Blood eosinophils: 546 cells/ml. Serology positive in the blood and negative in the CSF.
9-Fortenberry et al 1991[12]	1/M	Recurrent seizures, truncal ataxia and lethargy. Blood eosinophils: 3000-17000/μl. Serology positive in the blood.
10-Sellal et al 1992[41]	24/F	Paresthesis of legs. Positive serology in blood and CSF. Blood eosinophils: 1500/μl. CSF: 50 cells/μl, 10% eosinophils.
11-Villano et al 1992[48]	53/F	Four-year history of progressive spastic tetraparesis and hypoanesthesia in four limbs and trunk. CT scan: complete C4 block like intradural and extra spinal-cord expansive process. Surgical removal of fibrotic tissue in arachnoidea. Histopathology showed chronic granulomatous inflammation with *Toxocara* larvae
12-Sommer et al 1994[43]	48/M	Ataxia, rigor and neuropsychological disturbances. CT scan and MRI: diffuse and circumscribed lesions in white matter. Positive serology for *Toxocara* in the blood.

Continue....

Table 1 - Continue.

Author	Age/Gender	Summary of main observations
Cases with clinical data		
13-Kumar & Kimm 1994 [19]**	22/F	MRI: cervical cord lesions. Improvement after treatment
14-Ota et al 1994[31*a]	22/F	Meningeal irritation signs and cerebellar ataxia. MRI: cortical and subcortical lesions in cerebrum and cerebellum. CSF: 330 cells/μl 30% eosinophils. Serology positive in the blood and CSF. Treatment with diethylcarbamazine and corticoids but other lesions developed in spinal cord.
15-Duprez TP et al 1996[8]**	58/M	Transverse myelitis
16-Strupp et al 1999[45]	49/M	Subacute weakness of quadriceps muscles. Difficulty with bladder and bowel functions and erectile failure. Paraparesis with discrete hyperesthesia and hypalgesia. CSF: 128 cells/μl 33% eosinophils. No blood eosinophilia. Serology was positive in the blood and CSF. The patient was treated with albendazole and there was partial recovery of signs and symptoms. MRI performed four months after treatment was normal.
17-Goffete et al 2000[13]	40/F	Weakness of right leg and dysesthesia in the right T8-T10 dermatomes. MRI: hypoinsensitivity in the T8-T10 spinal cord area. CSF: pleocytosis with eosinophilia. Serology positive in blood and CSF recovery after treatment with thiabendazole.
18-Ardiles et al 2001[3]	61/M	faciobrachycrural Hemiparesis. CT scan: hypodense areas in the right posterior temporal area. Serology positive in the plasma and negative in CSF. No information on CSF cell counts.
19-Richartz E, Buchkremer G 2002[34]**	65F	Depressive symptoms and cognitive deficits. Normal EEG and CT.Depressive symptoms and cognitive deficits. Normal EEG and CT. CSF eosinophilia and positive serology for *Toxocara*. Improvement of cognitive deficits one year later.
20- Robinson A, Tannier C, Magnaval JC 2002[35]** N.I		Meningoradiculitis. CSF eosinophilia. Positive serology for *Toxocara* both in the serum and CSF.
21- Moreira-Silva et al[#]	5/F	Paresis of both superior and inferior members, nuchal rigidity and bilateral Kornig and Lasègue responses. Urinary retention and fecal incontinence. CSF: 187 leukocytes, 57% eosinophils. Blood eosinophils: 711/ul. Positive serology for *Toxocara* both in blood and CSF. Cranial MRI: small irregular lesions in the posterior portion of spine-bulbar transition and pedunculus cerebellaris. Improvement after treatment with albendazole.
	5/M	Palsy of inferior members and urinary retention. CSF: 23 leukocytes/ml. 57% eosinophils. Blood eosinophils 221/ul. Positive serology for *Toxocara* both in blood and CSF. Improvement after treatment with thiabendazole

*The cause of death was attributed to *T. canis* encephalitis. ** Information collected from abstract. CSF = cerebrospinal fluid. [a] the same case was reported in Journal of Neurology Neurosurgery and Psychiatry 59:197-198,1995. N.I.: age of patient was not informed in the abstract. #cases reported in this publication.

with albendazole and thiabendazole. Furthermore, *Toxocara* infection is frequent in children that are treated at the Children's Hospital Nossa Senhora da Glória in Vitória[28].

These arguments are not irrefutable because eosinophilic meningitis or meningoencephalitis may be idiopathic or produced by larvae of such human helminths (reviewed in reference 19) as *Ascaris lumbricoides* or *Strongyloides stercoralis*[24 25], *Ascaris suum*[16 18 23 32], *Trichinella spiralis*[10] or by larvae of other paratenic nematodes such as the rat nematode *Angiostrongylus cantonensis*[33 42] and raccoon ascarid, *Baylisascaris procyonis*[34]. Since *Ascaris* antigens can cross react with *Toxocara* antigens, one could argue that the positive serology observed in the two cases reported here may be due to this cross reaction. However, the serology was performed after absorption with *Ascaris* antigen. In one case (Case 2) there was *Strongyloides stercoralis* larvae in the feces. Although there are reports of *Strongyloides* larvae entering the central nervous system, this occurrence is extremely rare and is associated with the disseminated form of the infection[9]. The other paratenic nematodes that can cause eosinophilic meningitis or encephalitis have not yet been described in Brazil.

As demonstrated in Table 1, neural involvement in toxocariasis has been reported in all ages without a significant gender prevalence. Eosinophilia occurred in both peripheral blood (7/9 cases) and cerebrospinal fluid (8/11 cases) in those cases in which eosinophil counts were reported. It is noteworthy that some cases occurred without presence of eosinophilia in blood and cerebrospinal fluid. Serology is useful for diagnosis although cross reaction is frequent with larval antigens from other nematode species. For this reason absorption of serum or cerebrospinal fluid with larval antigens from other nematode species would improve the specificity of serology. Additionally, further development of methods to detect IgM anti-*Toxocara* larvae would help to identify recent infection. We conclude that in cases of encephalitis and myelitis with cerebrospinal fluid pleocytosis and eosinophilia, parasitic infection of the central nervous system may be suspicious and serology should be performed to establish the correct diagnosis and treatment.

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