Persistence of viable cysts in Neurocysticercosis: a serial imaging study

Persistência de cistos viáveis na neurocisticercose: um estudo seriado de imagem

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Abstract

Introduction. Cysticercosis is an endemic disease in developing countries and is the most common parasitic infection of the central nervous system. The diagnosis is difficult and imaging may contribute to the confirmation. Objective. To report the evolution of brain lesions and the clinical response of a patient with a definitive diagnosis of neurocysticercosis (NCC). Methods. We analyzed six computed tomography (CT) and three magnetic resonance imaging (MRI) exams, performed in a period of six years. Results. The serial imaging study revealed the involution of nine viable cysts and two degenerating cysts out of 39 lesions. It occurred after six years of disease and four courses of treatment with Albendazole. The other 28 lesions were calcified. Clinically, there was reduction in frequency of seizures after treatment with Albendazole and the onset of regular use of anticonvulsants (six per year to 1.8 per year). Conclusion. This case illustrates an instance of partial NCC efficacy to antiparasitic therapy, and demonstrates the role of serial imaging studies in the monitoring the evolution of NCC lesions and in characterizing the diversity of lesion appearance over time.

Keywords: neurocysticercosis, magnetic resonance imaging, computed tomography, cerebrospinal fluid, ELISA

Resumo

Introdução. A cisticercose é uma doença endêmica nos países em desenvolvimento. Representa a infecção parasitária mais comum do sistema nervoso central. O diagnóstico é difícil e o exame de imagem pode contribuir para a confirmação. Objetivo. Relatar a evolução das lesões encefálicas, assim como avaliar a resposta clínica de um paciente com um diagnóstico definitivo do NCC. Métodos. Foram analisadas seis tomografias computadorizadas (TC) e três ressonâncias magnéticas (RM) realizadas durante o período de seis anos. Resultados. O estudo de imagem seriada revelou a involução de nove cistos viáveis e dois cistos em degeneração de 39 lesões. Isso ocorreu após seis anos de evolução da doença e quatro cursos de tratamento com albendazol. As outras 28 lesões encontravam-se calcificadas e aumentaram para 36 em número. Clinicamente, houve redução na frequência das crises após o tratamento com albendazol e do início do uso regular de anticonvulsivantes (seis por ano para 1,8 por ano). Conclusão. O caso demonstra a importância dos estudos da imagem seriada no acompanhamento das lesões da neurocisticercose (NCC), considerando a possibilidade de resistência medicamentosa e a necessidade da repetição do tratamento.

Palavras-chave: neurocisticercose, ressonância magnética, tomografia computadorizada, líquido cefalorraquidiano, ELISA

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Neurocysticercosis (NCC) is a growing disease endemic to Latin America, Africa and Asia, and is caused by the larvae of the tapeworm *Taenia solium*. It is the most common parasitic infection of the central nervous system. The mortality concentrates in patients with subarachnoid NCC.

The disease is difficult to diagnose because clinical manifestations are varied and not specific, including seizures, intracranial hypertension syndrome, meningitis, and psychological disorders.

Complementary exams confirm the diagnosis and include neuroimaging study and anticycsticercosis antibodies detection using Western blot tests in serum, and immunoenzymatic tests (ELISA) in the cerebrospinal fluid (CSF). The neuroimaging study confers a definitive diagnosis for the disease if one observes the presence of cystic lesions with scolex (pathognomonic for NCC and an absolute criterion).

The MRI classification of the lesions is based on the cysticercus’ evolutionary stages: 1) Vesicular Stage (viable cyst with no contrast enhancement); 2) Colloidal Stage (degenerating cyst with peripheral contrast enhancement); 3) Nodular Granular Stage (degenerating cyst with nodular contrast enhancement); 4) Calcified Stage (dead cyst).

We report a case of NCC with multiple viable cysts that persisted for many years in spite of treatment. We will also discuss the need of serial imaging in clinical follow-up.

**Case Report**

A 34-year-old man born in Minas Gerais presented an 18-years history of tonic-clonic generalized seizures followed by loss of consciousness. He was diagnosed with epilepsy and treated irregularly with anticonvulsant medication (Phenobarbital), without improvement (averaging six episodes a year). In January 2003, at the age of 29, following another seizure, he was hospitalized and, after performing a cranial CT scan, diagnosed as NCC with the presence of nine viable cysts. He was treated with an antiparasitic drug (Albendazole, for 10 days) and an anticonvulsant (Phenytoin and, later, Carbamazepine), with good clinical control. The electroencephalogram (EEG) was normal.

In the period comprising 2004 and 2005 the patient continued to suffer convulsive episodes (four in total), and conducted control CT scans. Three cranial CT scans in 2004 showed no evolutionary changes. The first cyst to degenerate was observed in a CT scan in August 2005. The persistence of eight viable cysts was an indication for a second round of antiparasitic treatment with Albendazole. The treatment was repeated once again in April 2006. In May 2006 the patient began to be treated at the Neuroinfection Section at HUGG/UNIRIO. The authors did not agree with the excessive number of CT scans performed on the patient in a short period of time. In November 2006 the patient had a tonic-clonic seizure episode. A brain MRI performed in January 2007 showed five viable cysts. He was given a new antiparasitic treatment with Albendazol (1g per day for 8 days) and corticotherapy (dexametasone - 6 mg per day for 14 days).

Throughout 2007 the patient had three tonic-clonic general seizures. The dose of Phenytoin was increased (200mg to 500mg per day). In January 2008, a brain MRI was performed as part of the post-treatment follow-up and it revealed two degenerating lesions, which were also present in the MRI exam a year later (January 2009). Between 2003 and 2009 the nine viable cysts regressed. There were no new convulsive episodes in 2008 (Figure).

The patient’s epidemiological history showed that he spent time in a pig farm. The neurological exam was normal.

**Complementary Exams**

Routine laboratory exams (full blood, biochemistry, urine and stool analysis) were normal. Patient serum sample was positive in the Western Blot test using lentil-lectin affinity-purified glycoprotein antigens obtained from *T. solium* cysticercus. The CSF was normal, but for an IgG index of 0.96 (RRd”0.7) and the ELISA test for cysticercosis was reactive (ENZITESTECISTICERCOSE IgG, RNA laboratórios, Cascavel-PR, Brazil). The direct exam and culture were negative for common germs, *Mycobacterium tuberculosis* and fungi.

Radiographic studies showed calcifications in the soft parts of both thighs, compatible with calcified cysticerci. In the first cranial CT scan (January 2003), a total of 39 lesions were identified, nine of which were viable. Of this total, 28 were calcified and the other 11 cysts were composed of nine cysts in the vesicular stage and two lesions with nodular contrast enhancement. After the total period of the evaluation of these CT exams, spanning six years, we could observe in the final CT scan the presence of 36 calcified, five degenerating, and three nodular lesions, and two colloidal cysts. (Figure)

During this same period of six years the patient underwent a total of three brain MRIs (Figure). The
last MRI, performed in a 3T scanner demonstrated the presence of lesions of various evolutionary stages (vesicular, vesicular colloidal, granular nodular and calcified nodular). Two cystic lesions showed enhancement after gadolinium injection and presented images that suggested a scolex in the interior (Figure).

**Discussion**

The diagnosis of NCC was established according Del Brutto et al. criteria. The interpretation of these criteria allows for a definitive or probable diagnosis of NCC. This case presented a definitive diagnosis of the disease based on the presence of one absolute criterion (an image exam revealing a cystic lesion with scolex), four major criteria (cystic lesions without scolex, lesions with linear or nodular post-contrast enhancement and calcifications, resolution of the lesions after the use of Albendazole, and serum Western Blot positivity for anticysticercus antibodies), three minor criteria (clinical history suggestive of NCC, positive ELISA test of the CSF, calcifications in the soft parts), and one epidemiological criteria (endemic area).

MRI has more sensitivity for evaluation of viable and degenerating lesions in NCC, whereas computed tomography performs better in the visualization of dead cysts (calcifications). Although, in this case report, the patient's CT scans and MRI were not performed on the same day, we can confirm the literature, since the calcified lesions were clearly visible in the CT scans and practically not viewed in the MRIs.

The serial imaging study indicated and followed-up the antiparasitic treatment of NCC. The presence of more than five cysts without contrast enhancement in CT or MR implies treating for parasites with Albendazole and corticotherapy. In this particular case, the first head CT scan taken on January 2003 showed a total of nine viable cysts. It took six years to see the regression of all of these viable cysts. Comparing the involution of these lesions after the antiparasitic treatment, the outcome was below expectations in terms of the time. Reduction of 82% in the number of cystic lesions after 10 months, and of 85 and 97% after three months treatment has been reported. A recent study demonstrated the...
disappearance of viable cystic lesions in brain parenchyma in 31% of patients one month after being treated with Albendazole, compared to 11% in the control group. It is not clear the resistance of some cystic lesions despite repeated treatment. It may be associated with inadequate Albendazole levels, resistance or no adherence to the treatment.

The clinical evolution showed a reduction in the frequency of convulsive episodes since the first treatment with antiparasitic medication (Albendazole) and at the onset of regular treatment with anticonvulsants (given that the seizures averaged six episodes a year). In the five following years the average went down to 1.8 a year (a decrease of 70%). Thus, the patient in this study was within the expected average (67% in 30-months).

The clinical improvement shown by the patient was most likely related to his adhesion to the anticonvulsant therapy. Based on the findings in the literature, we concluded that the evolution time of the lesions was greater than expected after treating with antiparasitic drugs and greater than the natural evolution span of the disease itself. This case represents a resistant form of NCC with the presence of lesions in all evolutionary stages as observed in a series of images spanning a considerable length of time.

Conflict of interest: No conflict of interest to declare.

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