

TREATMENT OF CEREBRAL CYSTICERCOSIS WITH ALBENDAZOLE IN ELEVATED DOSAGES

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Neurocysticercosis is the most common parasitic infection of the central nervous system (CNS)¹⁻³. It has been estimated that 50 million people in the underdeveloped countries are infected and the disease is endemic to Latin America (Equatorial Zone and Brazil), Mexico, Central America, Asia and Africa^{1,4}. Because it is a transmissible disease its high incidence reflects the bad conditions of the local basic sanitation². Neurocysticercosis treatment is still surrounded by doubts. The decision about whether to have a clinical or a surgical treatment is, most of the times, very difficult, especially when the location is ventricular or subarachnoidal⁵.

Whenever the clinical treatment is chosen the doubts still remain, regarding the cysticide dosage and the time of the treatment.

The case we report is illustrative in this way.

CASE

A 38 years old woman was carrier of racemose cysticercosis with cysticercus in different stages, located in the cisterna and in the parenchyma, since 1996 (Fig 1A). History of seizures, which have been controlled by antiepileptic drugs.

In 1999, the patient developed cysts in the quadrigeminal cisterna, with supratentorial dilatation of the ventricular system, without symptoms of intracranial hypertension (Fig 1B). The patient evolved in an asymptomatic way, despite the growing cysts inside the quadrigeminal cisterna, which were evident in a cranial computed tomography (CT) in 2001 (Fig 1C). In September 2004, the lesion in the quadrigeminal cisterna disappeared (Figs 1D and 1E). There are no known documents or reliable reports about the use of cysticides during this time.

In October 2005, the patient developed right eyelid ptosis. The magnetic resonance imaging (MRI) revealed cystic lesions, which suggested cisternal cysticercosis in the suprasellar and perimesencephalic region on the right side. The third cra-

nial nerve was compromised, in association with communicant hydrocephaly. In December 2005, the patient was admitted at SEMPER hospital prostrated, with headache and vomiting. CT revealed an increase in the cystic lesion of the prepontine cisterna and a discrete increase of the hydrocephaly regarding previous studies (Figs 2A, 2B). The patient was submitted to ventriculoperitoneal shunt and the symptoms were solved (Fig 2C). However, the oculomotor paralysis persisted on the right side. During this hospitalization, the patient was also submitted to a treatment with albendazole 15 mg/Kg for 8 days.

In January 30th 2006, the patient was readmitted with paralysis of the right third nerve and left hemiparesis. MRI revealed a resolution of the hydrocephaly and an increase on the size of the lesion in the prepontine cisterna, with a significant compression of the mesencephalous, which justified the alternate syndrome (Fig 2D and 2E). Also, there is still presence of bilateral chronic subdural hematoma, bigger at right, which was probably a consequence of the shunt (Fig 2F). The patient went through surgical drainage of the hematoma but there was no improvement of the hemiparesis and ptosis. The ventricular catheter was not removed.

A new therapeutic treatment was accomplished using albendazole 30 mg/Kg dose, for 30 days, along with dexamethasone IV during the first week of treatment. The patient was discharged and completed the treatment at home, with oral corticoids in a decreasing way.

Control CT after 15 days of treatment showed a resolution of the subdural hematoma, but also the persistence of the lesion in the cistern (Figs 3-A and 3-B). The patient's neurological deficits did not change.

After the therapeutic treatment was finished, the patient returned with significant improvement of the hemiparesis and ptosis. No side effects were noticed during this time. A CT scan, 60 days after the beginning of the treatment, revealed a decrease in the size of the cyst of the basal cistern, and totally recovery of the focal deficits (Fig 3C).

TRATAMENTO DE CISTICERCOSE CEREBRAL COM ALBENDAZOL EM DOSES ELEVADAS

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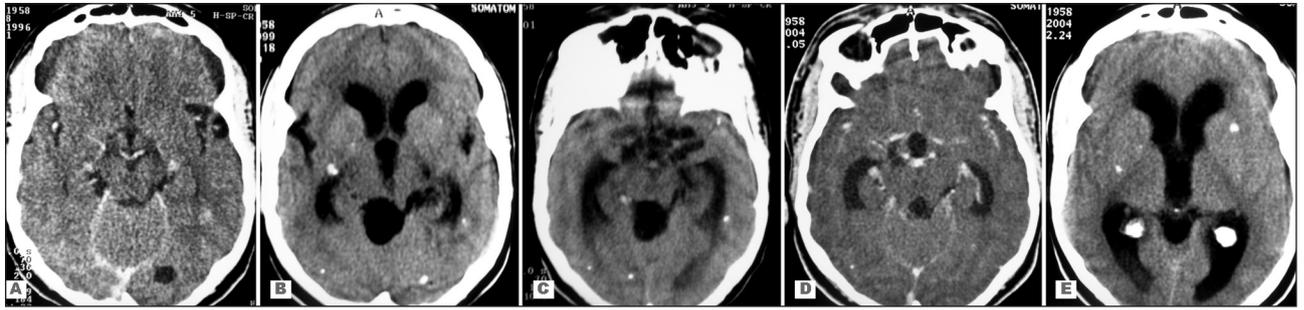


Fig 1. (A) Cranial CT showing lesions of cerebral cysticercosis; (B and C) Cranial CT showing cyst in the quadrigeminal cistern and dilatation of the ventricular system; (D and E) Cranial CT showing the disappearing of the cyst in the quadrigeminal cistern.

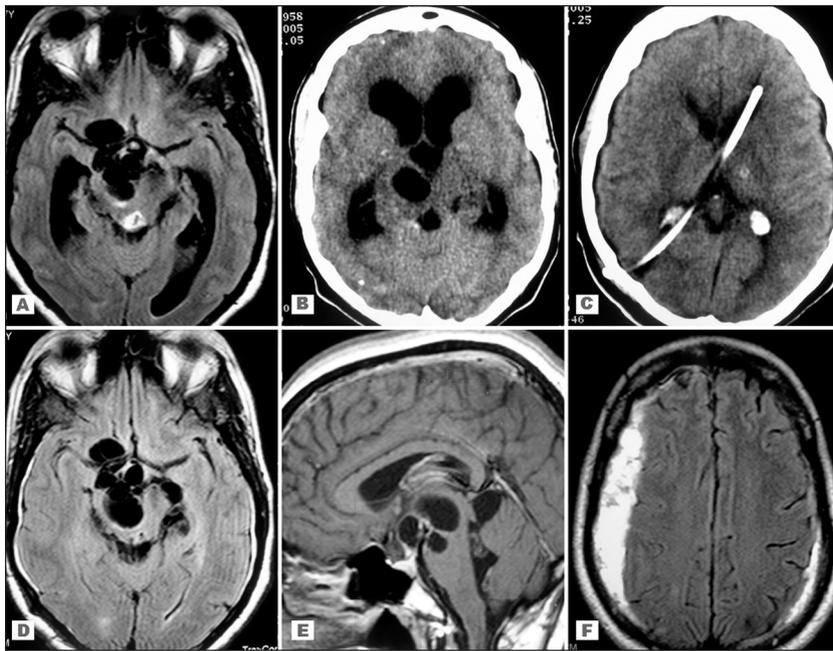


Fig 2. (A) MR of the encephalous revealing cystic lesions in the suprasellar and perimesencephalic region on the right side; (B) Cranial CT revealed an increase in the cystic lesion of the prepontine cistern; (C) Cranial CT with intraventricular catheter; (D and E) MR of the encephalous revealing a resolution of the hydrocephaly and an increase on the size of the lesion in the prepontine cistern; (F) MR of the encephalous revealing bilateral chronic subdural hematoma.

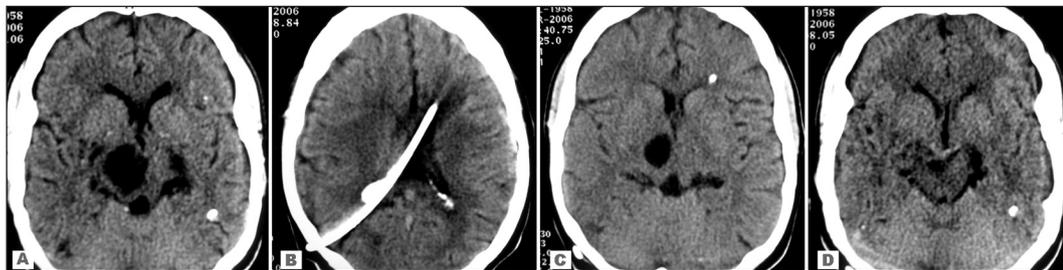


Fig 3. (A and B) Cranial CT after 15 days of treatment with Albendazole (30 mg/Kg) shows a resolution of the subdural hematoma, but also the persistence of the lesion in the cistern; (C) CT scan, 60 days after the beginning of the treatment with Albendazole, revealing a decrease in the size of the cyst of the basal cistern; (D) Within 6 months of evolution the CT shows a almost completely resolution of the lesion caused by neurocysticercosis.

Within 6 months of follow-up, the patient presented no neurological deficits, and the CT showed almost completely resolution of the lesion caused by neurocysticercosis (Fig 3D).

DISCUSSION

Most people with cyticercosis are assymptomatic.

Their clinical manifestation depends on the location, size and number of cysts and on the host's immune response to the parasite¹. Seizures appear in 70% to 90% of the patients¹. Intracranial hypertension, headache, meningeal syndrome and psychiatric disturbs are other recurrent manifestations⁶.

The hexacanth embryo reaches the CNS, via the vascular system. It passes through the capillar wall in the encephalic parenchyma, develops into the cystic form and then turns into *Cisticercus celulosae*, a benign form of the disease^{2,5}. In this period, it shows a thin membrane with a colorless liquid in the inside associated with the scolex.

The cysticercus which develop inside the ventricle and subarachnoidal space assumes the *Cisticercus racemosus* form, a malign form of the disease, wich is characterized by an irregular enhance membrane, absence of scolex and grouped in multiples vesicles². Hydrocephaly is the most common manifestation. The prognosis is bad, and the therapeutic response is low, poor and the life span is short.

In the CNS, the cysticercus shows a natural evolutive process that culminates in its degeneration in an approximated time of 2 to 5 years². During this period, they go through 4 stages⁷: (1) vesicular: active form, with an albescent membrane, translucent, with colorless liquid and the scolex in the inside; (2) colloidal: there is an enhancing membrane e substitution from the lipid liquid by an albescent gel; (3) granular: calcium is deposited in the gel; (4) nodular: there is a completely calcification of the cyst.

Treatment of neurocysticercosis may be clinical or surgical. Surgical treatment is traditionally recommended for the intracranial forms which improve with local compression of the encephalous and cranial nerves or intracranial hypertension.

The effectiveness of the surgical procedure to remove the cistycercus from the cisterna of the base is doubtful². Ordinarily, cistycercus are multiple and show a partial degeneration as well as adherence to the cranial nerves, vessels and encephalic parenchyma because of arachnoiditis, meaning that an attempt to completed lysis can be disastrous². When a choice must be made between a surgical or a pharmacological conduct, there is still much controversy, especially if the location of the cyst is at the ventricular or subarachnoidal space. The conduct must be individualized for each case⁵.

Because these considerations, we choose to try the clinical treatment of the reported patient. Albendazole has been used in a 15 mg/Kg dose for 15 days and more recently for 8 days².

Singhi³, in a prospective, randomized, double blind study with 122 children with neurocysticercosis, treated half of the patients with albendazole (15 mg/Kg) for 7 days and the other group was treated with the same drug and the same dosage for 4 weeks. He came to the conclusion that the 1 week treatment was as effective as the treatment for 28 days. The exactly time of the therapy with

albendazole still remains unclear³. Other authors recommended the treatment with albendazole for 28 to 30 days and that can reach even 60 days of treatment^{1,4,5}. Controversy arises also over the dose of treatment. Recently studies have been made regarding the treatment of racemose neurocysticercosis with elevated dosages of albendazole.

Comparative studies between the treatment of subarachnoidal and intraventricular forms of the parasite with a 15 and a 30 mg dose of albendazole, had shown an involution of the cysticercus much more significant in the groups treated with higher doses of the cysticid^{5,8,9}. In our patient we used 30 mg/Kg dose, for 30 days.

It seems to be consensual as well as in our reported case, the treatment among with corticoid therapy (dexamethasone)^{1,2,4,8}. Predisone may be employed when a long-term treatment is needed, especially in cases of chronic arachnoiditis. For the intracranial hypertension, manitol would be indicated as well. Some authors recommended antihistaminics instead of dexametasona, and this is also useful in diabetes and blood hypertension^{5,9}.

Elevated dosage of albendazole does not seem to be associated to the increase of side effects when compared with the usual doses^{5,8,9}.

In conclusion, administration of elevated dosages of albendazole has been shown to be more effective and free from the side effects when compared to the treatment with conventional dosages. Clinical follow-up for longer periods seems to be necessary. The need of repeated cycles of the treatment is still questioned by some authors.

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