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CASE REPORT

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Repair capacity of *Taenia solium* extraparenchymal cysts: radiological and *in vitro* evidence

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ABSTRACT

Extraparenchymal neurocysticercosis (EP-NC) responds poorly to anthelmintic treatment. Several factors are involved in this low responsiveness, including the host's heterogeneous immune response and the ability of the parasite to evade it. In this study, we present radiological and *in vitro* findings that demonstrate that *Taenia solium* cysts have the capacity to repair from injuries. Six patients (three with cases of subarachnoid, two with cases of intraventricular, and one with a case of mixed subarachnoid and intraventricular cysts) presented with neurological complaints and underwent either medical or surgical treatment. Follow-up magnetic resonance imaging (MRI) showed apparent resolution of the cysts. However, months later (10–56) new MRI scans revealed cysts at the same sites observed before treatment. Cysts surgically removed were maintained in RPMI-1640 medium supplemented with 10% fetal bovine serum. Monthly assessments demonstrated the growth of the parasites and the release of HP-10. Our findings demonstrate the ability of *T. solium* extraparenchymal cysts to grow and repair themselves. This capacity is likely another factor involved in the disease's poor treatment response.

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Neurocysticercosis; *Taenia solium*; extraparenchymal neurocysticercosis; magnetic resonance imaging; *in vitro* tests

1. Background

Neurocysticercosis (NC) is an infection of the central nervous system (CNS) caused by the presence of the larval stage of *Taenia solium*. Despite recent advances in reducing the endemicity of this disease [1], it continues to affect developing countries with inadequate sanitary conditions. In addition, owing to migratory flux, patients with this disease are diagnosed at considerable frequency in the United States and Europe [2,3].

NC is a pleomorphic disease whose symptoms depend on the number of parasites, their location, and the heterogeneous host-parasite interaction [4]. Cysts lodged in the brain parenchyma commonly cause seizures and headaches. This form of the disease has a relatively benign course (except for massive parasitic loads or giant cysts) and can be effectively managed using anthelmintics and antiseizure medications [5].

Conversely, in extraparenchymal NC, cysts are located within the cerebrospinal fluid (CSF) compartments (i.e., the subarachnoid space and ventricles). Extraparenchymal cysts may be asymptomatic for several years; however, when inflammation is triggered, they can lead to vasculitis, CSF imbalance with hydrocephalus, and increased intracranial pressure [6]. Mortality and morbidity rates in patients with extraparenchymal NC are higher than those with parenchymal NC [7].

In addition, extraparenchymal NC poses additional challenges for diagnosis and treatment; computed tomography and conventional magnetic resonance imaging (MRI) have low accuracy for the detection of cysts. New volumetric MR acquisition techniques are helpful for cyst detection [8]. However, MRI is not widely available, particularly in countries where this disease is more prevalent. Extraparenchymal NC is less responsive to medical treatment with anthelmintics [9]. Besides, anthelmintic treatment may trigger symptoms related to inflammatory responses in the subarachnoid space, making it necessary to use corticosteroids to control inflammation; however, this has the side effect of reducing the effectiveness of the anthelmintic treatment [10]. Therefore, many patients require several repetitions of anthelmintic cycles to eliminate the cysts.

Poor responsiveness of extraparenchymal cysts has been shown to be related to low concentrations of systemic albendazole (ABZ) due to the heterogeneity of absorption,

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Article highlights

- The extraparenchymal location of *Taenia solium* larvae in patients with neurocysticercosis represents the most severe form of the disease, primarily due to its poor response to treatment.
- Factors previously associated with this poor response include low concentrations of anthelmintic drugs in cerebrospinal fluid, a high parasite burden, and a weak immunoinflammatory response.
- To investigate parasite-related factors contributing to this poor responsiveness, we present six patients who experienced recurrence of parasitic infection after apparent successful surgical or medical treatment.
- We also provide images demonstrating the larvae's ability to grow *in vitro* following surgical removal.
- These observations suggest that the parasites' repair capacity plays a key role in their resistance to treatment.
- Therapeutic strategies aimed at reducing this capacity should be considered as potential treatment options.

high volume of parasite cysts, and modest immunoinflammatory responses against them [11,12]. As albendazole sulfoxide (ASOX), the active metabolite of ABZ, diffuses passively from the plasma in the CSF [13,14], low concentrations of ASOX in the CSF are likely involved in the low responsiveness. Notably, CSF is a low-cellular compartment that allows the parasite to be hidden from host immune surveillance.

Another possible mechanism related to the lower response to medical treatment that has not been previously documented may be the repair capacity of *T. solium* after injury. In this study, we provide in vitro and radiological evidence to support this hypothesis.

2. Clinical cases

2.1. Collection of clinical and radiological information of patients with extraparenchymal NC

All subjects provided informed consent for the use of their anonymized clinical-radiological information. Institutional Review Board approval was not necessary as case information is retrospective.

We revised the database of patients that visited the Neurocysticercosis Clinic of the Instituto Nacional de Neurología y Neurocirugía (INNN) in Mexico City, Mexico, and the Neurosurgical Division of Hospital das Clínicas da Faculdade de Medicina de Botucatu, Brazil. We present six cases of extraparenchymal NC that were successfully treated medically or surgically, but with parasite recurrence in the same location several months later. In all these cases, the patients were treated with ABZ (30 mg/kg/d for 10 days, combined with corticosteroids) or with surgical removal of the cysts through endoscopic procedures. A definitive diagnosis was made based on the current diagnostic criteria [15].

2.2. Presentation of cases - radiological findings

Patient #1 was a 31-year-old male who presented with a headache and diplopia. An MRI scan revealed cysts in the left cerebellobulbar cistern (Figure 1 (1a)). The patient underwent one cycle of albendazole (30 mg/kg/d for 10 days). A six-month follow-up MRI scan showed a clear reduction in cyst volume (Figure 1 (1b)). However, 10 months later, when the

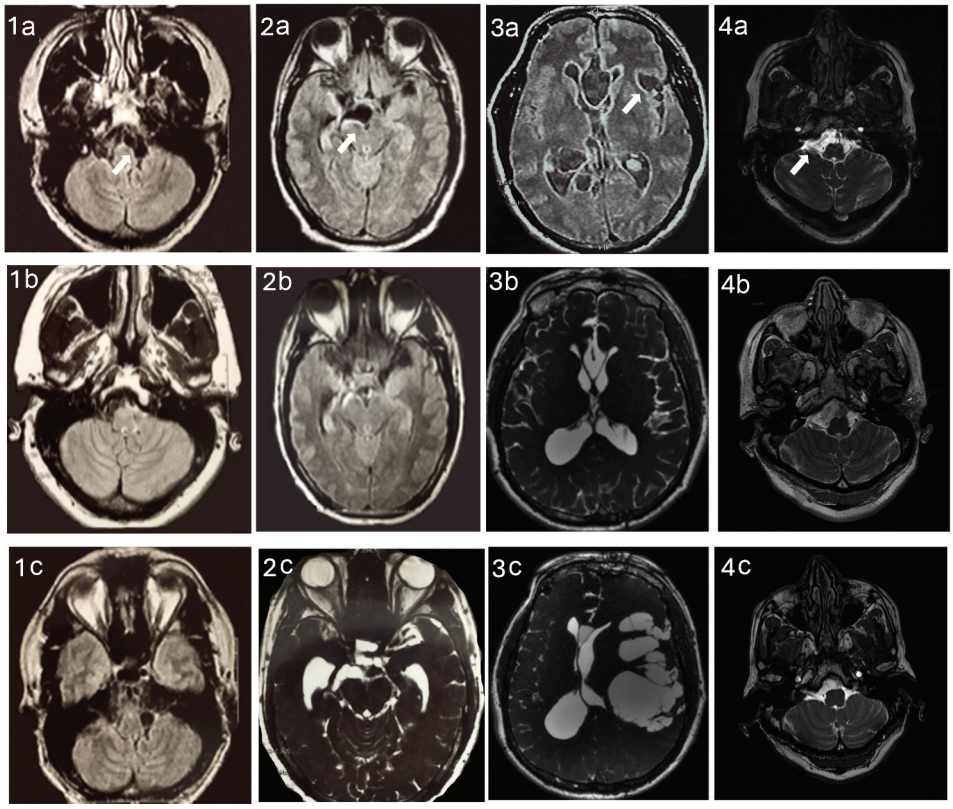

Figure 1. MRI images from patients (#1 to #4) with subarachnoid neurocysticercosis (NC) at initial presentation (a) Arrows point the cysts), after treatment (b) and at relapse (c).

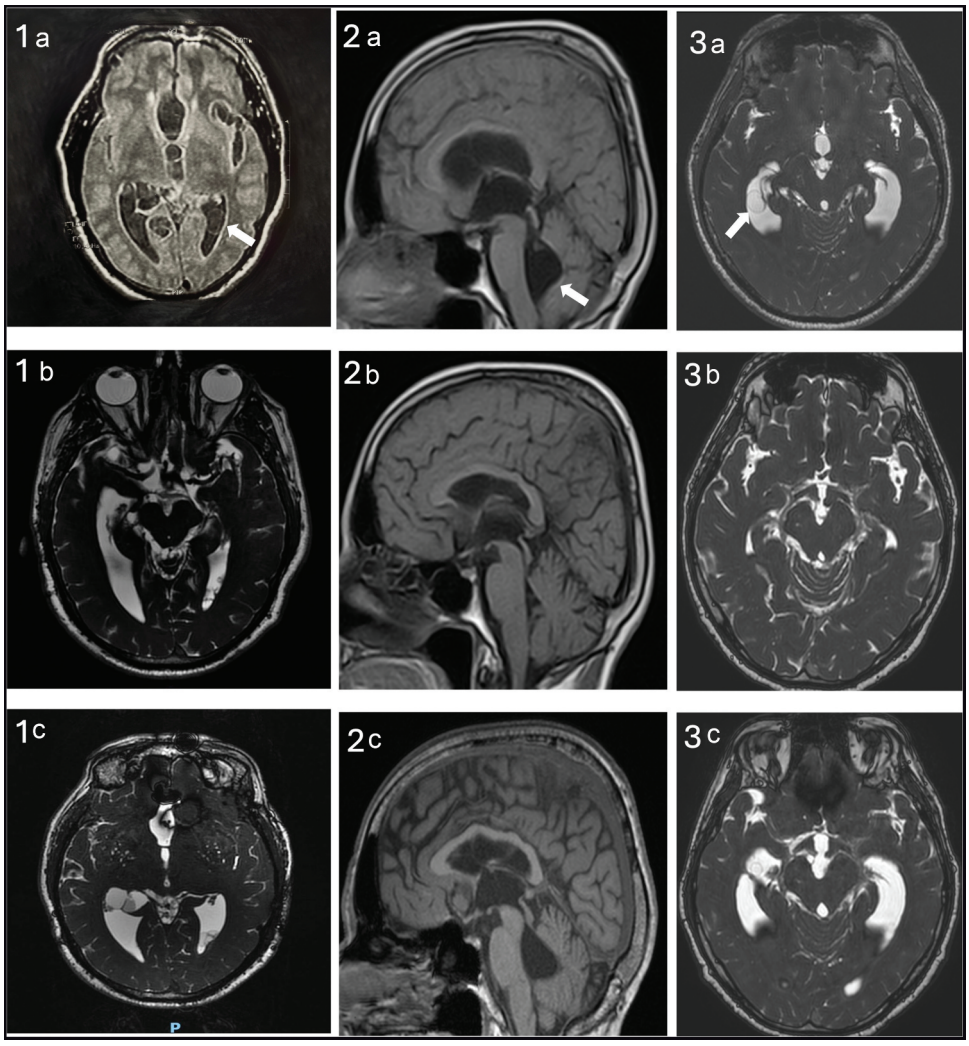

Figure 2. Magnetic resonance imaging (MRI) scans of patients (#1 to #4) with ventricular NC at initial presentation (a) Arrows point the cysts), after treatment (b) and at relapse (c).

patient was feeling well, another follow-up MRI scan revealed the presence of a cyst in the previously affected area (Figure 1 (1c)).

Patient #2 was a 29-year-old male who presented with headaches and dizziness. An MRI scan revealed a cyst in the right crural cistern (Figure 1 (2a)). The patient underwent three cycles of albendazole (30 mg/kg/d for 10 days) over the next three years (one cycle/year), and by the end of this period, a follow-up MRI scan showed cystic lesions (Figure 1 (2b)). Four years after the initial diagnosis, the patient developed headaches again, and another follow-up MRI scan revealed a cyst at the same site (Figure 1 (2c)).

Patient #3 was a 40-year-old male who presented with headaches and memory impairment. A MRI scan revealed cysts in the left Sylvian fissure (Figure 1 (3a)) and lateral ventricle (Figure 2 (1a)). He was treated with two cycles of albendazole (30 mg/kg/d for 10 days) in the year of diagnosis, and a 27-month follow-up MRI scan showed no visible cysts (Figures 1 (3b) and 2 (1b)). Another follow-up MRI scan was performed four years after the initial diagnosis and showed large cysts in the left Sylvian fissure (Figure 1 (3c)), which were surgically treated. Ten months later, cysts developed in the lateral ventricle (Figure 2 (1c)).

Patient #4 was a 52-year-old female experiencing convulsions and confusion. Before visiting the INNN, the patient had been diagnosed with NC in the cerebellobulbar cistern region. Two cycles of albendazole (10 mg/kg/days for one month each, separated by 1 month without treatment) were administered. An MRI scan was performed at INNN one month after the last treatment and it showed only membranes in the cerebellobulbar cistern, which were remnants of the parasite (Figure 1 (4a)). Six months later, small vesicles appeared (Figure 1 (4b)), which developed into two parasites 51 months after the first MRI scan (Figure 1 (4c)).

Patient #5 was a 46-year-old male who presented with headaches, gait disturbance, and vomiting. An MRI scan showed a cystic lesion in the fourth ventricle (Figure 2 (2a)). The patient underwent endoscopic removal of the cyst. To avoid complete insertion of the endoscope through the aqueduct, the cyst was removed by suction combined with screw movement; thus, it is likely that a remnant of the cyst wall remained after the procedure.

A six-month postoperative MRI scan revealed a fourth ventricle with normal dimensions (Figure 2 (2b)). Four years later, the patient again presented with the initial symptoms, and a new MRI showed a large cyst in the fourth ventricle (Figure 2 (2c)). The patient underwent a new surgery with suboccipital craniotomy, and the entire cyst was removed.

Patient #6 was a 55-year-old male who presented with headaches and mental confusion due to intraventricular cysts (Figure 2 (3a)). The patient had their cysts endoscopically removed; however, a cyst in the temporal horn of the right lateral ventricle could not be accessed, and the patient then underwent treatment with three cycles of albendazole (30 mg/kg/d for 14 days). Eight months later, a control MRI scan showed no significant lesions in the temporal horn (Figure 2 (3b)). One year later, the patient was admitted to the emergency room with new-onset headache, nausea, and vomiting. A subsequent MRI scan revealed a cyst in the right temporal horn (Figure 2 (3c)).

A summary of the main characteristics of the six patients included in this study is presented in Table 1.

3. In vitro findings

3.1. Collection and storage of parasites

At the INNN, two subarachnoid cysts were surgically removed from a patient and transferred immediately after extraction to RPMI-1640 medium. The patient presented to the emergency department with focal, secondarily generalized seizures and hemiparesis. A CT scan revealed multiple cysts in the Sylvian fissure, causing a mass effect on the brain. The patient had not previously received anthelmintic treatment, and extraction surgery was decided. The methodology used to culture the parasites was similar to previously described methods [16], with minor modifications.

Parasites were washed with sterile phosphate buffer solution (PBS; pH 7.4) with 1% antibiotics (100 U/mL penicillin and 100 mg/mL streptomycin) for 5 min and subsequently placed in 25 cm^3 culture flasks with 10 mL of RPMI-1640 medium supplemented with 10% fetal bovine serum (FBS), 2 mm L-glutamine, 1% antibiotics, 1% non-essential amino acids, and 1% pyruvate (all reagents were obtained from Gibco, Brazil). The parasites were maintained for six months at 37°C in a 5% CO_2 humidified atmosphere and medium was changed every 6 ± 3 d. Photographs of parasite growth were taken weekly using a Leica MZ6 stereoscopic microscope. The Leica IM500 image manager program was used to capture the photographs (copyright 1992–2005; Imagic Bildverarbeitung, AG).

After each change in the medium, a sample was preserved and immediately frozen. The presence of the parasite-specific antigen HP10 was determined by ELISA as previously described [17]. Specific antigen determination is a sensitive tool for assessing the viability of cysticerci [18,19].

3.2. Evolution of parasites

Gradual changes in the two parasites were documented photographically (Figure 3(a,b)). Morphological changes are

Table 1. Summary of patients' features, radiological findings, treatment modality, and time elapsed from an initial magnetic resonance imaging (MRI) scan showing a patient as disease-free to recurrence.

Patient	Sex, age	Symptoms	Location	Treatment	Time (month) between MRI "free" and relapse
1	M, 31	Headache, diplopia	SA, cerebellobulbar cistern	AH	10
2	M, 29	Headache, dizziness	SA, crural cistern	AH	10
3	M, 40	Headache, memory impairment	SA, Sylvian fissure, lateral ventricle	AH	27 (Sylvian)/ 37 (ventricle)
4	F, 52	Convulsions, confusion	SA, cerebellobulbar cistern	AH	51
5	M, 46	Headache, gait abnormality	4^{th} ventricle	Surgical removal	48
6	M, 55	Headache, mental confusion	Lateral ventricle	AH	10

AH, anthelmintic; F, female; M, male; SA, subarachnoid.

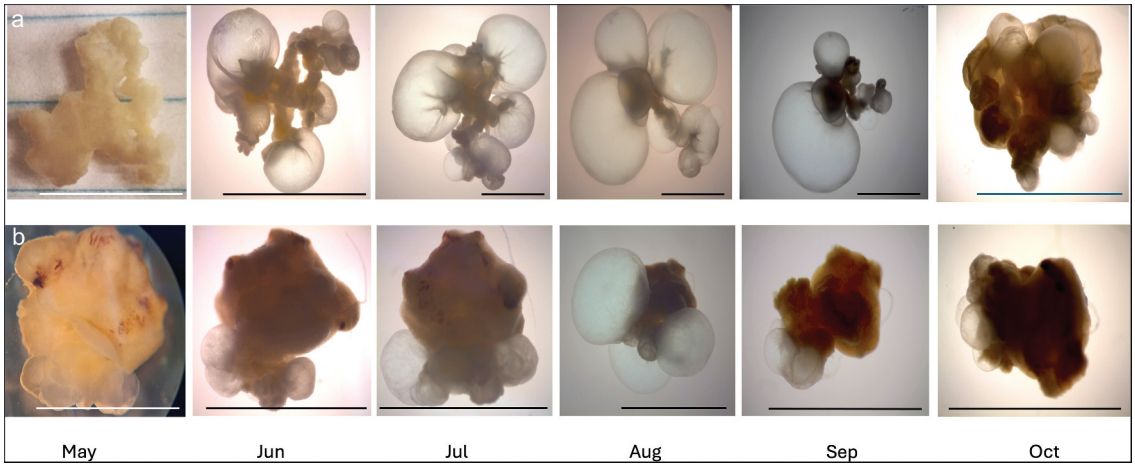

Figure 3. Photographic records of the two parasites (a and b) throughout the observational period. Bar scale: 10 mm.

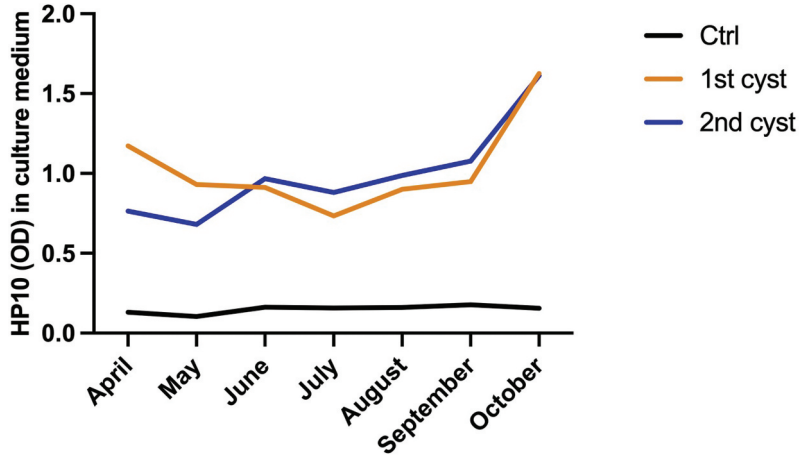

Figure 4. Parasite-specific antigen (HP10) concentrations in the medium during each month of observation for each parasite. Ctrl, control.

evident in these parasites. At the time of surgical extraction, membranes with a few small vesicles were observed. As the months progressed, the vesicles enlarged, and new ones appeared. A decrease in the volume of vesicles was observed between September and October for parasite A, and between August and September for parasite B, with no clear explanation for the cause.

The change in HP10 specific antigen concentration in the culture medium was assessed monthly. As shown in Figure 4, HP10 Ag was present in each evaluation and remained constant throughout the parasite culture. Interestingly, when the volume of parasite vesicles decreased (between September and October), an increase in the concentration of HP10 Ag in the culture medium was observed.

4. Discussion & conclusion

Extraparenchymal NC (EP-NC) is the most severe form of NC, particularly because of its poor response to treatment and the absence of clear management guidelines [20,21]. Indeed, the current guidelines on the treatment of NC provided strong recommendations with high-quality evidence for parenchymal NC, whereas for extraparenchymal NC, the recommendations are only based on low-quality evidence [22]. It is important to decipher the circumstances involved in the poor response to treatment, particularly because understanding this will help improve the strategies employed to cure patients.

In this report, we present clinical and experimental evidence demonstrating the repair capacity of these cysts, which certainly contributes to the difficulties in successfully treating patients with this disease. Radiological images clearly demonstrate this evolution, whereas *in vitro* findings provide evidence that the ruptured cyst walls can repair themselves and grow. In the evolution of the six patients included, the possibility of reinfection cannot be completely ruled out. However, in this case, it would be very difficult to understand why the parasites were located in the same place as at the time of diagnosis.

Different factors may be involved in this process. One factor is the environment in which the cysticerci lie. In EP-NC, parasites are present in the CSF, an acellular medium that is suitable for their growth, owing to the large space and the host's difficulties in mounting a strong immune response. Parasites also downregulate the immune response [12] but the mechanisms involved in this downregulation are not completely understood; however, there have been demonstrations of an increase in T regulator cells [23] and in the immune checkpoint pathway PD-1 and its ligand PD-L1 [10].

These mechanisms explain the difficulties in treatment and are probably involved in the growth and repair capacity of these cysts. Recent studies have demonstrated that growth capacity is linked to proliferation. Indeed, proliferative germinative cells expressing the serine/threonine-protein kinase *plk1* were identified in the walls of subarachnoid cysts, indicating that the growth of these cysts was probably due to abnormal cell proliferation [24]. In addition, transcriptomic analysis of the subarachnoid cysts showed enrichment of signaling pathways related to cell proliferation. The proliferative cells are stimulated by the TGF-b and EGF of hosts, the cyst walls have increased lipid uptake and higher metabolism, and the cysts overexpress detoxification systems [25]. Conversely, these cells and signaling pathways were not found in the vesicular form of *T. solium* in the muscles of naturally infected pigs. The mechanisms involved in the presence or absence of these cells in these two *T. solium* parasites have not yet been elucidated; however, it is probable that mutations or epigenetic factors are involved [25]. Further studies are required to fully elucidate other factors involved.

Interestingly, the proliferative ability of other parasites is particularly well recognized in *Taenia crassiceps*, a cestode parasite closely related to *T. solium*, which is widely used as an animal model of *T. solium* infection [26]. Its asexual proliferation by budding allows it to be maintained in the peritoneal cavity of mice through serial reinoculation [27]. Proliferative cells primarily located in growing buds have recently been visualized in various strains of *T. crassiceps* [28]. Recently, asexual reproduction by *T. crassiceps* was observed in rats with subarachnoid intracranial infection [29]. Interestingly, *T. solium* does not appear to have the ability to reproduce by budding. Accordingly, during the six months in which the cysticerci were maintained in culture, although vesicles developed, no separation was observed. Comparative studies between the two parasites should be

conducted to understand the mechanisms underlying their similarities and differences.

The apparent absence of a parallel relationship between vesicle volume and HP10 antigen levels is interesting. Indeed, at the end of the observation period, HP10 levels increased, whereas vesicle volume decreased. This observation is contrary to the significant positive correlation between parasite load and antigen levels found in a porcine model of cysticercosis [30]. Although the biological function of HP10 is unknown, it was recently demonstrated that *T. solium* excretory/secretory proteins have anti-inflammatory functions that may promote parasite survival [31]. Therefore, it is possible that when signals of "ill health" appear, the secretion of products with anti-inflammatory properties is favored, even if the parasites are in culture.

A limitation of our study is the unavailability of 3D MRI sequences for all included patients, as some scans were performed before the sensitivity of 3D sequences for detecting extraparenchymal cysts was established [8]. We recognize that this limitation should be addressed in future studies.

In conclusion, our findings provide evidence for the ability of *T. solium* extraparenchymal cysts to grow and repair themselves. This observation supports the severity of this form of NC. In addition to the proliferative capacity of these parasites, other mechanisms are likely involved in the repair process. Thus, further studies are required to understand these factors to help optimize medical treatments.

Disclosure statement

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

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