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Original article

Is antiparasitic treatment beneficial in chronic subarachnoid neurocysticercosis? A comparative case series

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ABSTRACT

Background: Neurocysticercosis is particularly severe when affecting the subarachnoid space. While antiparasitic therapy effectively controls the infection, it can lead to significant complications, especially in subarachnoid neurocysticercosis (SUBNCC). This study aims to characterize a cohort of patients with SUBNCC, with a focus on their clinical course depending on therapeutic interventions.
Methods: We conducted an observational, retrospective study involving patients diagnosed with SUBNCC at a tertiary hospital between November 1985 and July 2022. The primary endpoint was to delineate the clinical progression and demographic features of the cohort. A secondary objective was to compare relapse rates between patients receiving antiparasitic treatment and those who did not.
Results: Fifteen patients were included, with a median age of 31 years (range 24–54), and 53% were female. The most common countries of origin were Bolivia and Ecuador, with a median duration from immigration of 8.1 years (range 3–16). Approximately 46.7% of patients experienced at least one relapse, with rates of 46% in patients initially treated with antiparasitic medication and 50% in those treated with steroids alone. Complication rates were similar between both groups. Comparison of time to relapse between episodes treated with antiparasitic medication versus corticosteroids alone revealed no statistically significant difference (27 episodes in total; 17 treated with antiparasitic medication versus 10 with corticosteroids only; $p = 0.376$).
Conclusions: In patients with SUBNCC, clinical relapses managed with corticosteroids alone do not appear to result in worse outcomes in terms of complications and relapse rates compared to those managed with antiparasitic medication.

¿Es el tratamiento antiparasitario beneficioso en la neurocisticercosis subaracnoidea crónica? Una serie de casos comparativa

Palabras clave:
Neurocisticercosis
Espacio subaracnoideo
Complicaciones
Fármacos antiparasitarios
Recurrencia

RESUMEN

Introducción: La neurocisticercosis es especialmente severa cuando afecta el espacio subaracnoideo (SUBNCC). A pesar de que el tratamiento antiparasitario controla eficazmente la infección, puede causar complicaciones, especialmente en la SUBNCC. El objetivo del estudio es describir una cohorte de pacientes con SUBNCC, con especial atención en la evolución clínica según la aproximación terapéutica.

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Métodos: Realizamos un estudio observacional, retrospectivo, con pacientes diagnosticados de SUBNCC en un hospital terciario entre noviembre de 1985 y julio de 2022. El objetivo primario era describir sus características demográficas y progresión clínica. El objetivo secundario era comparar las tasas de recurrencia entre pacientes que recibieron tratamiento antiparasitario con aquellos que no lo recibieron.
Resultados: Quince pacientes fueron incluidos, con una edad media de 31 años (rango 24-54), el 53% eran mujeres, la mayoría de Bolivia o Ecuador, con una media de años desde la inmigración de 8,1 (rango 3-16). Aproximadamente el 46,7% de pacientes sufrió al menos una recidiva; entre aquellos tratados con antiparasitarios la proporción fue del 46% y en los que solo recibieron corticoides fue del 50%. La proporción de complicaciones fue similar entre ambos grupos. El tiempo entre recaídas entre episodios no mostró diferencias estadísticamente significativas entre los dos grupos (27 episodios en total; 17 tratados con antiparasitarios, 10 tratados con corticoides; p = 0,376).
Conclusión: En pacientes con SUBNCC, las recaídas clínicas tratadas solo con corticoides no parece que conlleven un peor resultado en cuanto a complicaciones y tasas de recaída en comparación con aquellas tratadas con tratamiento antiparasitario.

Introduction

Neurocysticercosis (NCC) stands as the predominant parasitic affliction of the Central Nervous System (CNS), attributable to the larval stage of the pork tapeworm *Taenia solium*. Its endemicity is notably concentrated in low-income countries characterized by inadequate excreta disposal and free-ranging pig husbandry, posing a substantial public health challenge across Latin America, Asia and sub-Saharan Africa.[1] The escalating immigration from these regions has notably increased in the incidence of NCC cases within countries with low local transmission rates.[2]

Clinical presentations of NCC vary depending on number, inflammation and anatomical location of the cystic lesions. Cysts within the brain parenchyma predominantly precipitate seizures, whereas extraparenchymal manifestations, such as ventricular or subarachnoid cysts (SUBNCC), may impede cerebrospinal fluid circulation, culminating in hydrocephalus and intracranial hypertension.[1,3] SUBNCC confers a heightened risk of morbidity and mortality.[4]

Differing from parenchymal NCC, extraparenchymal NCC comprises structurally aberrant, slowly proliferating *T. solium* cysts devoid of scolex, typically organizing into conglomerates with a racemose appearance.[5] The prolonged incubation period is characteristic owing to the slow growth. Eventually, cystic expansion may elicit mass effects; however, characteristic symptoms predominantly arise from an inflammatory arachnoiditis provoked by the host's response to deteriorating cysts. Subsequent stages may involve inactive disease, which may occur spontaneously or as a result of effective treatment.[6,7]

Paradoxically, treatment may incite host inflammatory reactions directed toward injured parasites, causing clinical manifestations similar to the infection itself.[8,9] Although it is rare, instances of cerebral infarction ensuing from antiparasitic therapy-induced angiitis have been documented.[10–12] In one series of 65 patients with cerebrovascular complications due to NCC, there were 10 patients (15%) who suffered a stroke while receiving anticysticeral drugs.[13] High-dose corticosteroids are widely advocated to forestall treatment-induced inflammation, especially in cases of vasculitis or giant subarachnoid cysts, which pose heightened risks of stroke.[14–16] Notably, the 2017 clinical practice guidelines for NCC management accentuates that even though antiparasitic drugs are strongly recommended, it is imperative to initially temper inflammation with high-dose corticosteroids before commencing antiparasitic therapy.[17]

In routine clinical practice, patients with SUBNCC often present with clinical manifestations linked to persistent inflammation caused by cyst locations susceptible to dreadful consequences upon degeneration. In such scenarios, antiparasitic therapy can precipitate adverse outcomes, leading clinicians to opt for corticosteroid treatment exclusively.

In light of these considerations, this study endeavors to delineate the clinical trajectory and therapeutic management of a cohort of SUBNCC patients, discerning whether those subjected to antiparasitic therapy manifest disparate clinical outcomes relative to those administered corticosteroid monotherapy.

Methods

In this single-center, observational, retrospective study, we enrolled consecutive patients diagnosed with SUBNCC at a tertiary hospital between November 1985 and July 2022. Inclusion criteria encompassed patients meeting published NCC diagnostic criteria[18] and seeking medical attention related to the infection. Cases lacking subarachnoid involvement or pertinent information were excluded.

All data were extracted by two different physicians through individual review of each patient's medical records. Variables gathered for each patient included: age, gender, birthplace, immigration date, date of clinical onset and diagnosis, signs and symptoms, cysts location, serology results, treatment, complications, and relapses. Relapses were defined as any new neurological clinical expression attributable to SUBNCC or acute changes in neuroimaging that motivated a treatment choice.

All neuroimaging was obtained by magnetic resonance imaging (MRI), most of them in a 1'5T or 3T scanner, with a variable frequency depending on the medical needs of each case. All the scans included at least T1, T2, fluid-attenuated inversion recovery (FLAIR), diffusion-weighted, and enhanced T1-weighted sequences. Cestode antigen analyses were predominantly conducted utilizing an enzyme-linked immunosorbent assay (ELISA), facilitated by the laboratory facilities within our institution. The sensitivity of these assays was established to be $\geq$2. Lumbar punctures were performed selectively guided by clinical necessity, primarily for differential diagnostic purposes, rather than as routine procedures before and after treatment initiation. Treatment strategies were individualized, with decisions made collaboratively by neurologists and infectologists, taking into consideration various factors such as the clinical status of the patient, cyst location, and the potential risk of vascular involvement during relapse. Antiparasitic therapy typically consisted of albendazole administered at a dosage of 15 mg/kg/day either as monotherapy or in combination with praziquantel (albendazole 15 mg/kg/day plus praziquantel 50 mg/kg/day).[17] Concurrent administration of high-dose corticosteroids was employed, with subsequent tapering regimens aligned

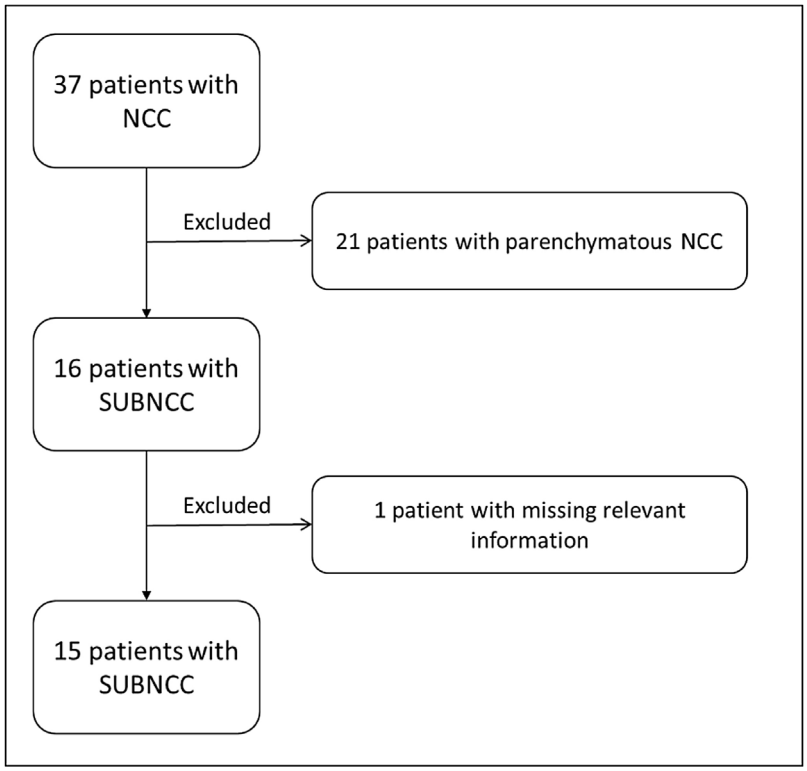

Fig. 1. Flowchart for patient inclusion. NCC: Neurocysticercosis; SUBNCC: subarachnoid neurocysticercosis.

with established treatment guidelines. Patients were closely followed up during and post-treatment, and patients with the highest risks due to cyst locations were admitted at the hospital for strict treatment follow-up.

The primary endpoint was to describe the clinical evolution and demographic characteristics of patients with SUBNCC. A secondary endpoint compared relapses based on whether patients received antiparasitic treatment or not. For this secondary analysis, comparisons were not restricted to patient groups assigned based on treatment at the initial presentation. Instead, each episode—whether a first presentation or a relapse—was treated as an independent event.

Statistical methods

Primary endpoint was assessed using a descriptive analysis. Categorical variables were presented as absolute frequencies. Demographic and clinical variables were presented as median and ranges or mean and standard deviation according to the distribution. For the secondary comparative analysis, Kaplan–Meier survival analysis and Cox regression test were performed to assess the time to relapse in each group. All tests were studied with confidence intervals of 95% and a significance level of 5%. Statistical analyses were performed in SPSS v.22 (SPSS Inc, Chicago, USA).

Ethical standards

This study was approved by the local Research Ethics Committee, with reference PR080/24. Data were collected anonymously. Patient information confidentiality was handled in accordance with Spanish regulations.

Results

An initial registry of 38 patients with NCC was obtained after individual case review. Twenty-one were excluded for lacking subarachnoid involvement, and one patient was excluded for lacking relevant information. A total of 15 patients with SUBNCC were finally included (see Fig. 1).

The median age of patients at diagnosis was 30.1 years (range 24–54 years), with 8 of them being women (53%). The most frequent birthplaces were Bolivia, Ecuador, and Honduras, with a median time since immigration of 8.1 years (range 3–16 years).

Demographic and clinical variables are summarized in Table 1. Headache and seizures were the most common initial clinical presentations. MRI revealed concomitant parenchymal involvement in most cases (13/15, 87%). Antiparasitic treatment at the initial episode was mainly albendazole (13/15, 87%), except for two patients.

Antiparasitic treatment was excluded in two patients due to elevated cerebrovascular risk. The first patient was diagnosed after undergoing an MRI for headache, which revealed a racemose cyst in contact with the origin of the right middle cerebral artery. She was subsequently admitted and diagnosed with probable meningitis secondary to SUBNCC. The second patient presented with headache and blurred vision associated with papilledema. An MRI revealed a cystic, multinucleated lesion along the trajectory of the left middle cerebral artery within the Sylvian fissure. Neither patient showed signs of systemic cysticercosis.

The mean follow-up time was 11.3 years (range 1–30 years). A total of 7 patients (46.7%) experienced at least one relapse. The recurrence rate was 46% (6/13) for patients receiving antiparasitic treatment at onset, versus 50% (1/2) for patients treated with

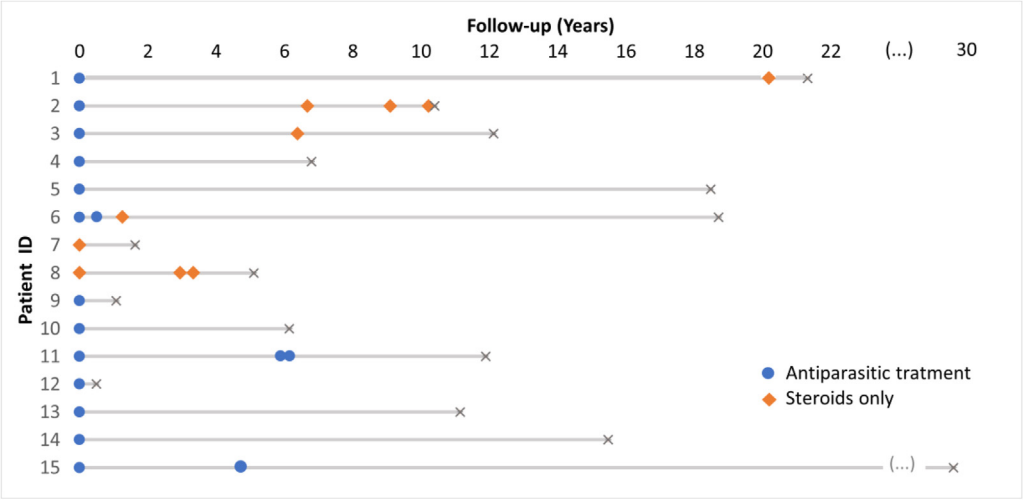

Fig. 2. Swimmer plot showing each patient's follow-up, representing a timeline with a figure for each clinical episode and an × for the end of follow-up.

Table 1
Demographic and clinical variables.

	n = 15
Age at diagnosis in years [median (range)]	30.1 (24–54)
Gender [n (%)]	
Male	7 (47)
Female	8 (53)
Birthplace [n (%)]	
Bolivia	5 (33)
Ecuador	4 (27)
Honduras	3 (20)
Perú	2 (13)
Spain	1 (7)
Years from immigration [median (range)]	8.1 (16–3)
Symptoms [n (%)]	
Headache	11 (73)
Seizures	7 (47)
Neurological deficit	4 (26)
Other	3 (20)
Cyst location in MRI[a] [n (%)]	
Subarachnoid + parenchymal	13 (87)
Subarachnoid	2 (13)
Serum serologies [n (%)]	11 (73)
Positive	7/11 (64)
Negative	4/11 (36)
Cerebrospinal fluid serologies [n (%)]	4 (27)
Positive	3/4 (75)
Negative	1/4 (15)
Antiparasitic treatment, debut episode [n (%)]	
Albendazole	12 (80)
Albendazole + praziquantel	1 (7)
None	2 (13)

[a] MRI: magnetic resonance imaging.

steroids alone. Complications and management details, including relapses, are described in Table 2. A swimmer plot of patients' follow-up and events is depicted in Fig. 2.

Regarding complications, two patients required ventriculoperitoneal shunts (VPS) at disease onset, and one patient suffered a stroke after receiving antiparasitic treatment. During follow-up, two other cases required a VPS, and one case was diagnosed with vasculitis secondary to SUBNCC.

A secondary analysis compared time to relapse between episodes treated with antiparasitic treatment versus corticosteroids only. For this purpose, we did not focus only on the treatment administered during the initial presentation of the disease, but instead, each relapse was analyzed as an independent episode. A total of 27 episodes were registered, with 17 treated with antiparasitic treatment and 10 with corticosteroids only. The median time to relapse in the antiparasitic treated group was 6.7 years versus 2.9 years for the corticosteroid treated group, with no statistical difference between groups (LL = −30.77, $Chi^2(df = 1) = 0.73$, $p = 0.39$) (Fig. 3). Regarding complications, in the antiparasitic treated group, three patients needed a VPS and one experienced a stroke due to treatment; whereas two patients needed a VPS in the corticosteroid treated group. One patient in the corticosteroid group suffered bilateral osteonecrosis of the knee.

Discussion

We present a retrospective series of 15 patients with SUBNCC treated at a Spanish hospital over a period of more than 30 years. Given the chronic nature and high recurrence rate of the disease, long-term follow-up of patients is crucial. Our results regarding patients' demographics and clinical presentation are consistent with published data,[4,7] reinforcing the importance of considering this diagnosis in young immigrant patients from endemic areas who present with headache, seizures, or other neurological manifestations of unknown origin.

The proportion of relapses in our cohort was similar to previously reported case series.[4,19] Nearly half of the patients experienced at least one relapse, typically manifesting as lymphocytic meningitis, with some cases necessitating VPS to control secondary hydrocephalus. Most clinical episodes were managed with antiparasitic treatment both at the onset and during relapse.

No difference was observed in relapse rates between patients initially treated with antiparasitic medication and those who were not, although the distribution of cases in each group was notably heterogeneous (14 cases vs. 2, respectively). Moreover, the follow-up times until recurrence or loss to follow-up were uneven and notably shorter in the two patients initially treated with corticosteroid therapy. Consequently, a survival analysis method was used considering each clinical episode independently, comparing the time to new relapses between episodes treated with antiparasitic agents (17 cases) and those treated with steroids alone (10 cases). Although the results should be interpreted with caution due to the small sample size and its heterogeneity, no statistically significant differences were observed between the two therapeutic approaches.

Table 2
Description of all the episodes and its treatment.

	Sex	Age at diagnosis	Cyst location	Date	Debut/relapse	Clinical presentation	Treatment	Complications of treatment
Patient 1	F	39	Subarachnoid + parenchymal	15/01/2001	Debut	Seizure	Albendazole	Vasculitis and stroke. HC
				17/03/2021	Relapse	Headache, cyst progression in MRI	Corticosteroids (10 days)	
Patient 2	M	31	Subarachnoid	19/10/2012	Debut	Lymphocytic meningitis	Albendazole (400 mg/12 h, 10 days)	
				20/06/2019	Relapse	Lymphocytic meningitis and HC	Corticosteroids (64 days)	
				23/11/2021	Relapse	Lymphocytic meningitis and seizures	Corticosteroids (Chronic treatment)	
				02/01/2023	Relapse	Seizures, HC	Corticosteroids + VPS (Chronic treatment)	Osteonecrosis of the knee
Patient 3	F	25	Subarachnoid + parenchymal	10/09/2010	Debut	Vasculitis and lymphocytic meningitis	Albendazole (400 mg/12 h, 15 days)	
				24/01/2017	Relapse	Seizures	Corticosteroids (50 days)	
Patient 4	M	32	Subarachnoid + parenchymal	15/09/2009	Debut	Aphasia	Albendazole (400 mg/12 h, 10 days)	
Patient 5	M	45	Subarachnoid + parenchymal	15/12/2003	Debut	Seizure	Albendazole (400 mg/12 h, 20 days)	
Patient 6	F	41	Subarachnoid + parenchymal	15/03/2004	Debut	Probable meningitis	Albendazole	Hepatic toxicity
				10/09/2004	Relapse	Lymphocytic meningitis	Praziquantel	
				17/06/2005	Relapse	Vasculitis and stroke	Corticosteroids	
Patient 7	F	37	Subarachnoid + parenchymal	04/06/2021	Debut	Probable meningitis	Corticosteroids (327 days)	
Patient 8	F	34	Subarachnoid	13/03/2018	Debut	HC	Corticosteroids	
				18/02/2021	Relapse	Lymphocytic meningitis and HC	Corticosteroids + VPS (25 days)	
				13/03/2021	Relapse	Headache and cyst progression in MRI	Corticosteroids (60 days)	
Patient 9	M	40	Subarachnoid + parenchymal	23/03/2022	Debut	Seizure	Albendazole + praziquantel (400 mg/12 h + 50 mg/kg/d, 30 days)	
Patient 10	F	30	Subarachnoid + parenchymal	30/01/2017	Debut	Asymptomatic (cyst found in an MRI)	Albendazole (400 mg/12 h, 20 days)	
Patient 11	M	29	Subarachnoid + parenchymal	15/07/2010	Debut	Lymphocytic meningitis	Albendazole	
				01/06/2016	Relapse	Lymphocytic meningitis and HC	Albendazole + VPS (400 mg/12 h, 15 days)	
				09/07/2016	Relapse	Lymphocytic meningitis	Albendazole + praziquantel (400 mg/12 h + 50 mg/kg/d, 90 days)	
Patient 12	M	24	Subarachnoid + parenchymal	15/03/2023	Debut	Seizure	Albendazole (400 mg/12 h, 15 days)	
Patient 13	F	36	Subarachnoid + parenchymal	30/08/2012	Debut	Seizure	Albendazole (400 mg/12 h, 20 days)	
Patient 14	M	54	Subarachnoid + parenchymal	15/02/2007	Debut	Aphasia	Albendazole	
Patient 15	F	44	Subarachnoid + parenchymal	15/11/1993	Debut	Seizure and HC	Albendazole + VPS	
				02/08/1997	Relapse	Arachnoiditis and HC	Praziquantel	

M: male, F: female. MRI: magnetic resonance imaging. HC: hydrocephalus. VPS: ventriculoperitoneal shunt.

In a randomized controlled trial conducted in 2008 to evaluate the efficacy of albendazole treatment in patients with NCC,[20] a group of 57 patients receiving albendazole was compared with a placebo group of 90 patients, both of which received corticosteroids. While a clear advantage was noted in the disappearance of active cysts by 12 months in the albendazole group when analyzing the entire sample, this difference was not significant when considering only cases of SUBNCC. Subsequently, another study was conducted with the same sample,[21] assessing the effects of albendazole treatment on non-seizure outcomes in symptomatic NCC patients. Although patients treated with albendazole exhibited significantly lower odds of memory loss and confusion, and increased odds of affective disorders, no differences were observed in the incidence of new headaches or neurological deficits between the two groups, suggesting that the clinical benefits of anthelmintic treatment remain uncertain.

Complications observed in our cohort were consistent with those reported in prior studies,[7] indicating no significant differences in the incidence of complications between patients treated with antiparasitic agents and those who were not. In our cohort, four patients required VPS placement during follow-up, and one patient experienced central nervous system vasculitis. Antiparasitic drugs for the treatment of SUBNCC are highly recommended in the current NCC guidelines,[17] with prior initiation of high-dose corticosteroids to prevent treatment-related complications. However, such complications were documented in our series, as one patient

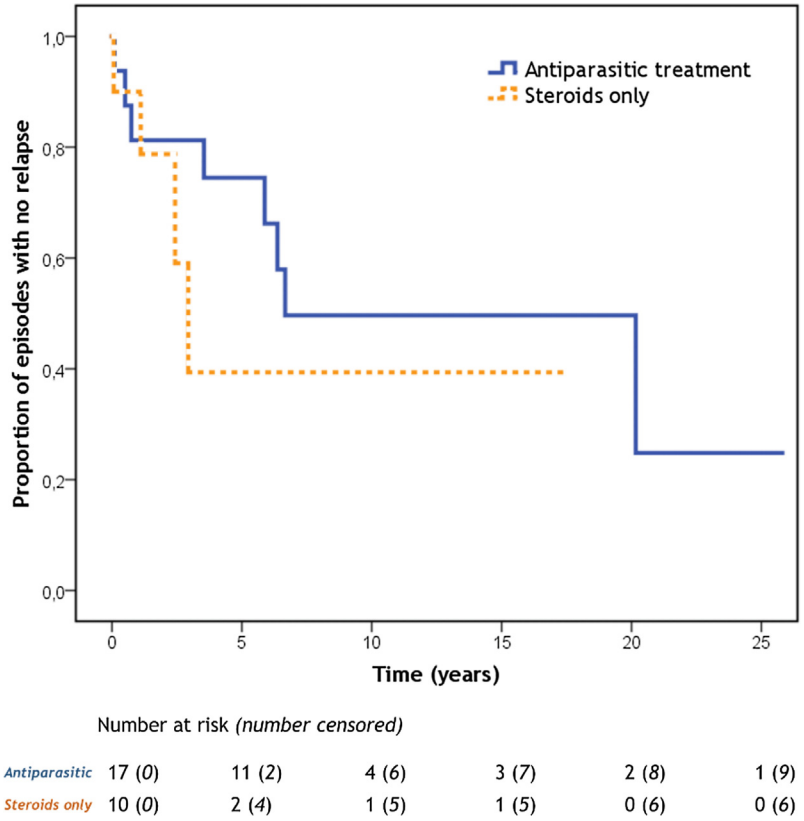

Fig. 3. Kaplan–Meier surveillance showing time to next relapse after each clinical episode for both arms of treatment.

experienced a stroke following the initiation of antiparasitic treatment. Such complications are particularly concerning given their potential severity in young, productive patients; therefore, it is crucial to prioritize the prevention of iatrogenic harm, to minimize the risk of these adverse outcomes.

Several limitations must be acknowledged that may affect the generalizability and the statistical power of the study results. Firstly, the retrospective design of the study may result in missing important clinical details or unrecorded episodes. Additionally, there was no documentation of corticosteroid dosages, timing of treatment initiation, or tapering schedules. Furthermore, the small sample size, heterogeneity of cyst locations and clinical characteristics such as initial disease severity or immune response, as well as an unequal distribution of treated and untreated patients, may also influence the study outcomes.

Another potential limitation of this study is the predominant use of monotherapy with albendazole for 10–20 days as the antiparasitic regimen in our cohort. This preference reflects that most patients were treated before randomized controlled trials demonstrated the superior efficacy of combination antiparasitic therapy and before guidelines recommended extended dual therapy. This limitation may have influenced the outcomes observed in our study.

This study does not provide definitive conclusions regarding the prioritization of inflammation control over infection treatment in SUBNCC. However, it initiates a debate regarding whether all cases of SUBNCC truly benefit from antiparasitic treatment, as there are no discernible differences supporting one therapeutic strategy over another, while the potential iatrogenic effects of antiparasitic therapy in this patient subgroup can be significant. We would suggest treating the patients with antiparasitic drugs at the debut, but maybe to treat relapses only with steroids in selected patients. To establish more robust conclusions, further prospective studies with larger sample sizes are warranted, ideally using randomized clinical trial designs to compare treatment strategies in selected patients, specifically addressing the management of relapses with cysticidal treatment versus corticosteroids alone.

Conclusion

In patients with SUBNCC, clinical relapses treated with steroids alone do not appear to result in worse outcomes in terms of complications and relapses compared to those treated with antiparasitic medication.

Data sharing and data accessibility

The datasets used and/or analyzed during this study are available from the corresponding author on reasonable request from any qualified investigator.

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Conflict of interest

Pablo Arroyo-Pereiro, Antonio Martínez-Yélamos, Sergio Martínez-Yélamos and Albert Muñoz-Vendrell have received honoraria from Teva, Lilly, Lundbeck, Roche, UCB, Bial, Chiesi, Allergan, Esai, Zambon, Kern Pharma, Pfizer, Biogen Idec, Bristol Myers Squibb, Novartis, TEVA, Merck, Janssen, Neuraxpharm, Genzyme, Sanofi, Bayer, Almirall and/or Celgene.

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