Original Article

Demographics and Clinical Characteristics of Patients with Neurocysticercosis: A Retrospective Study from Dali, China

Han-Xiao Zhu†, Yang-Chao Li†, Xue-Ping Yang[1], Yu-Hao Chu[1], Wang Guo[1], Ruo-Xia Chen[1], Dan-Dan Guo, Li-Jing Cheng, Yun Li

Department of Neurology, The First Affiliated Hospital of Dali University, [1]Clinical Medical College, Dali University, Dali, Yunnan, China
†These authors contributed equally to this work and share the first authorship.

Abstract

Background: Neurocysticercosis (NCC), a predominant parasitic disease that affects the central nervous system and presents with diverse clinical manifestations, is a major contributor to acquired epilepsy worldwide, particularly in low-, middle-, and upper middle-income nations, such as China. In China, the Yunnan Province bears a significant burden of this disease.

Objective: To describe the demographic, clinical, and radiological features as well as serum and cerebrospinal fluid antibodies to cysticercus in patients with NCC from Dali, Yunnan Province, China.

Materials and Methods: This retrospective study included patients who were diagnosed with NCC at The First Affiliated Hospital of Dali University between January 2018 and May 2023 and were residing in Dali, Yunnan Province, China.

Results: A total of 552 patients with NCC were included, of which 33.3% belonged to Bai ethnicity. The clinical presentation of NCC exhibited variability that was influenced by factors such as the number, location, and stage of the parasites. Epilepsy/seizure (49.9%) was the most prevalent symptom, with higher occurrence in the degenerative stage of cysts ($P < 0.001$). Compared with other locations, cysticerci located in the brain parenchyma are more likely to lead to seizures/epilepsy (OR = 17.45, 95% CI: 7.96–38.25) and headaches (OR = 3.02, 95% CI: 1.23–7.41). Seizures/epilepsy are more likely in patients with cysts in the vesicular (OR = 2.71, 95% CI: 1.12–6.61) and degenerative (OR = 102.38, 95% CI: 28.36–369.60) stages than those in the calcified stage. Seizures was not dependent on the number of lesions. All NCC patients underwent anthelminthic therapy, with the majority receiving albendazole (79.7%).

Conclusion: This study provides valuable clinical insights into NCC patients in Dali and underscores the significance of NCC as a leading preventable cause of epilepsy.

Keywords: Acquired epilepsy, China, cysticercus, demographics, epidemiology, epilepsy, neurocysticercosis, seizure, Taenia

Address for correspondence: Prof. Yun Li, Department of Neurology, The First Affiliated Hospital of Dali University, Dali 671000, Yunnan, China.
E-mail: yy_neuron@163.com

Submitted: 29-Jun-2023 **Revised:** 19-Aug-2023 **Accepted:** 28-Aug-2023 **Published:** 06-Oct-2023

Access this article online	
Quick Response Code:	Website: https://journals.lww.com/sjmm
	DOI: 10.4103/sjmms.sjmms_298_23

How to cite this article: Zhu HX, Li YC, Yang XP, Chu YH, Guo W, Chen RX, *et al*. Demographics and clinical characteristics of patients with neurocysticercosis: A retrospective study from Dali, China. Saudi J Med Med Sci 2023;11:283-91.

INTRODUCTION

Neurocysticercosis (NCC), caused by the cystic larvae of *Taenia solium*, represents a parasitic infection that affects the human nervous system. This condition is endemic in numerous developing and certain developed countries, including sub-Saharan Africa, Eastern Europe, Latin America, and specific regions of Asia, such as parts of China.[1] The estimated number of NCC infections exceeds 20 million people worldwide, based on a conservative approximation.[2,3]

The lifecycle of *T. solium* involves the pig, an intermediate host that houses the parasitic larvae, and the human, a sole definitive host that harbors the adult tapeworm and the larval stage. Tapeworm eggs or proglottids are expelled from the fecal matter of infected humans into the environment. In regions with poor sanitation, pigs may ingest these contaminated feces, consuming the tapeworm eggs.[4] The activated eggs hatch, attach, and penetrate the pig's intestinal epithelium, spread throughout its tissues, and develop into fluid-filled larvae or cysticerci.[5] Consumption of improperly cooked or raw pork contaminated with these eggs by humans leads to their transformation into oncospheres in the intestinal wall. They then penetrate the intestinal mucosa, enter the bloodstream, and spread to various tissues, including the central nervous system (CNS).[6,7] Human can serve as accidental intermediate hosts by consuming uncooked pork containing cysticerci or have direct contact with *Taenia* carriers via the fecal–oral route.[8]

A retrospective survey of 16,508 cases of cysticercosis in China revealed that the majority of cases were concentrated in the north-eastern, central, and south-western regions.[9] Although China's social and economic development in recent years has contributed to improvements in the environment and hygiene, cysticercosis is still prevalent in some remote areas, such as Sichuan and Yunnan Provinces in western China, due to complex socio-ecological factors.[10-12]

NCC, the most common parasitic infection affecting the CNS, manifests through a diverse array of clinical presentations contingent upon the location, number, and stage of the parasites, as well as the inflammatory reaction elicited in the surrounding area.[13] Cysticercus within the CNS can assume various stages, encompassing the viable (vesicular) stage, transitional (colloidal) stage, and inactive (calcified) stage.[14,15] Inactive (calcified) cysts manifest as small, high-density nodular lesions on CT, sometimes accompanied by perilesional inflammation. MRI can reveal perilesional gliosis surrounding the cysts. Common clinical manifestations of NCC include seizures, chronic headaches, cognitive impairment, focal neurological deficits, and symptoms of intracranial hypertension. Notably, cysts can exist for a sustained period, provoking a few neurological symptoms.[16] Even in the calcified stage, the lesions can cause recurrent episodes of perilesional edema, which result in recurrent neurological symptoms such as seizures.[3]

Treatment modalities for NCC comprise antiparasitic therapy, medical management utilizing symptomatic medications, and surgical interventions.[17,18] However, for the long term, the destruction of viable parenchymal NCC lesions with antiparasitic drugs reduces the recurrence of seizures.[3] Antiparasitic drugs, such as praziquantel and/or albendazole, are recommended for patients with intraparenchymal or extraparenchymal cysts; however, these drugs can elicit local perilesional inflammation in the initial period, which may lead to the emergence/exacerbation of neurological symptoms. Nonetheless, the use of adjuvant corticosteroids prior to antiparasitic drug administration can help mitigate this effect.[15,19] Further, NCC patients with epileptic seizures should receive appropriate anti-epileptic drugs. When the cyst is located in the intraventricular region, the cysticerci can be removed by minimally invasive neuroendoscopy. Cerebrospinal fluid (CSF) diversion with medical therapy is the recommended approach when surgical removal is hazardous.[15]

The incidence of cysticercosis remains alarmingly high in western China, especially Yunnan province, which has a diverse population and certain areas where unhealthy lifestyle is practiced. In fact, the practice of consuming raw/undercooked meat is common among the Bai ethnicity residing in Dali.[12,20] Besides, in some low-income areas where toilet facilities are poor or non-existent, people may defecate in the open. The situation is further exacerbated by a lack of pig management, or in some cases, pigpens being connected to human latrines.[12] The objective of this study was to provide a comprehensive description of the demographic, clinical, and radiological characteristics as well as serum and CSF antibodies to cysticercus in NCC patients from Dali city, Yunnan Province.

MATERIALS AND METHODS

Study design, setting, and participants

This retrospective study included patients who were diagnosed with NCC at The First Affiliated Hospital of Dali University between January 2018 and May 2023

and were residing in Dali, Yunnan Province, China. The research protocol was approved by the Ethics Committee of The First Affiliated Hospital of Dali University, Dali, China. Data were retrieved from the hospital's database using the keywords "cerebral cysticercosis" or "intracranial parasitic infection."

Diagnostic criteria for neurocysticercosis

Each patient's case was analyzed by two investigators to confirm the diagnosis of NCC, following the diagnostic criteria for NCC outlined by Del Brutto *et al*.[21] NCC cases were classified as definitive or probable based on established criteria. In addition to the exclusion of other pathologies producing similar neuroimaging findings, definitive NCC was defined as the existence of one absolute criterion; two major neuroimaging criteria plus any clinical/exposure criteria; one major and one confirmative neuroimaging criteria plus any clinical/exposure criteria; or one major neuroimaging criterion plus two clinical/exposure criteria (including at least one major clinical/exposure criterion). Probable NCC was defined as the presence of one major neuroimaging criterion plus any two clinical/exposure criteria, or one minor neuroimaging criterion plus at least one major clinical/exposure criterion.

Cystic stages within the CNS were classified into three stages: vesicular, degenerative, and calcified.[22] Vesicular cysts are characterized by rounded, low-density cystic structures containing fluid, with a diameter ranging from 0.5 cm to 2 cm. These cysts may exhibit minimal or no signs of inflammation and can often display an eccentrically located tapeworm scolex, which appears in equal intensity to CSF on CT scans. In the degenerative stage, cysts demonstrate indistinct borders and are associated with obvious peripheral edema. They appear as isointense fluid on CT and are frequently accompanied by contrast enhancement in the surrounding region. Calcified cysts, on the other hand, manifest as small, high-density nodular lesions on the CT and are sometimes accompanied by perilesional inflammation. MRI can effectively delineate perilesional gliosis surrounding these calcified cysts.

The location of parasites in the CNS is the main determining factor of clinical manifestations in NCC patients. We defined lesions located in the cerebral cortex as parenchymal NCC, which contains some small cysts that grow into the subarachnoid space but are embedded in the parenchyma. Subarachnoid NCC refers to lesions located in Sylvian fissures or basal cisterns, which are generally small in size and lack a scolex in many cases. Those lesions located in the ventricles or basal cisterna were known as ventricular NCC, which may be associated with ventricular distention and hydrocephalus on CT. When the lesion was located in multiple intracranial regions, it was defined as multiple-site cysts.

Classification of epileptic seizures

In accordance with the ILAE 2017 classification,[23] our study classified epileptic seizures into two main categories: focal seizures and generalized seizures. Focal seizures were further subcategorized into focal impaired awareness seizures, focal aware seizures, and focal to bilateral tonic–clonic seizures. On the other hand, generalized seizures encompassed non-motor seizures, motor seizures, and tonic–clonic seizures.

Data collection

Demographic information, including age, gender, place of residence, and dietary habits, was recorded. Clinical data were collected for each patient, including details of clinical symptoms such as headaches, seizures, and other neurological manifestations, as well as information on serum and CSF cysticerci antibodies. Enzyme-linked immunosorbent assay (ELISA) tests were performed to detect IgG antibodies to cysticerci in patients' serum and CSF. Moreover, the results of MRI/CT scans and the administered parasitic treatment were documented. The prescribed MRI scans were conducted using either a 3.0-T or 1.5-T imaging system. The majority of patients in the study underwent neuroimaging examinations as part of routine diagnostic assessments.

Statistical analysis

Descriptive statistics were used to analyze the distribution of age, gender, dietary habits, ethnic group, place of residence, serum and CSF antibodies to cysticercus, MRI imaging results, main clinical symptoms, and treatment modalities. Categorical variables were presented as percentages, while continuous data were expressed as mean ± standard deviation (SD). The Chi-square test or Fisher's exact test was applied, as appropriate, to compare categorical variables. In addition, a multiple logistic regression model was used to analyze the influencing factors (i.e., variables with $P < 0.05$ in the univariate analysis) among different groups. $P < 0.05$ was considered as statistically significant.

RESULTS

Demographic characteristics

A total of 552 patients with NCC were included, of which 364 (66%) were male and 188 (34.1%) were female (male to female ratio: 1.9:1). The mean age was 44.8 ± 19.3 years (range: 3–80 years). Further, 460 (83.3%) patients lived in rural areas and 92 (16.7%) in urban areas.

Table 1: Demographic characteristics of the patients (*N*=552)

Variable	*n* (%)
Gender	
Female	188 (34.1)
Male	364 (65.9)
Age (years)	
<14	44 (8)
14–44	192 (34.8)
45–59	160 (29.0)
≥60	156 (28.3)
Ethnic group	
Bai	184 (33.3)
Tibetan	44 (8)
Han	152 (27.5)
Lagu	12 (2.2)
Lisu	52 (9.4)
Wa	8 (1.5)
Yi	96 (17.4)
Zhuang	4 (0.7)
Place of residence	
Urban	92 (16.7)
Rural	460 (83.3)
Habits of eating raw meat or vegetables	
Yes	288 (52.2)
No	264 (47.8)

In terms of ethnicity, patients were primarily from the Bai (33.3%), Han (27.5%), Yi (17.4%) ethnic groups; other minorities accounted for 21.7% of the population. In addition, most patients had a history of eating raw meat or lettuce before being diagnosed with NCC (52.2%) [Table 1].

Clinical features

A total of 451 NCC patients from our study had MRI and clinical examination results available for analysis. The most common clinical feature was seizures/epilepsy (*n* = 225; 49.9%), followed by headaches (142; 31.5%), and other neurological signs/symptoms (84; 18.6%). Other neurological signs/symptoms include cognitive dysfunctions (*n* = 16), psychiatric disorders (7), focal neurological deficits (33), and increased intracranial pressure (28). Further, 61.8% of the patients with epilepsy presented with generalized tonic–clonic seizures, and 38.2% presented with focal seizures [Table 2].

Compared with other locations, cysticerci located in the brain parenchyma are more likely to lead to seizures/epilepsy (OR = 17.45, 95% CI: 7.96–38.25) and headaches (OR = 3.02, 95% CI: 1.23–7.41), which may be attributed to perilesional edema caused by inflammation. On the other hand, subarachnoid cysts are highly unlikely to cause epilepsy seizures (OR = 0.07, 95% CI: 0.01–0.29). Headaches are more common in NCC patients with intraventricular cysts than seizures (OR = 3.11, 95% CI: 1.53–6.32) and other neurological symptoms (OR = 6.71, 95% CI: 2.66–16.90). Similarly, the individuals with subarachnoid cysts are more likely to experience headaches than those with seizures/epilepsy (OR = 11.77, 95% CI: 2.54–54.50) [Table 3]. Patients with cysts in the vesicular stage (OR = 2.71, 95% CI: 1.12–6.61) and degenerative stage (OR = 102.38, 95% CI: 28.36–369.60) are more likely to experience seizures/epilepsy compared with those in the calcified cyst stage [Table 3].

NCC patients with three (OR = 5.53, 95% CI: 1.683–18.14) or more than three lesions (OR = 2.96, 95% CI: 1.617–5.41) are more likely to experience headaches than those with a single lesion; there was no difference in seizures or other neurological symptoms [Table 3].

Neuroimaging and electroencephalography

All patients (*n* = 552) in the study underwent neuroimaging examinations: 451 (82%) underwent MRI, and 30 (5%) underwent only CT; the remaining 71 (13%) patients had undergone neuroimaging examinations at other hospitals a few days before admission and diagnosis at our hospital. A total of 438 and 13 cases met the criteria for definitive and probable cases, respectively.[21] EEG was documented for 220 (40%) patients, of which around 51% had a normal EEG [Table 2].

Among patients with MRI evidence (*n* = 451), about 51% and 40% had degenerative and viable cysts, respectively. The remaining patients only showed calcifications. Parenchymal cysts were found in 218 (48.3%) patients, intraventricular cysts in 63 (14%), subarachnoid cysts in 44 (9.8%), and multiple-site cysts in 126 (27.9%) patients. Further, most patients had more than three cysticerci (54.3%), followed by one cysticerci (34.8%). Ring enhancement was observed in 143 (31.7%) cases, cerebral edema around lesions was present in 237 (52.6%) cases, and hydrocephalus was detected in 71 (15.7%) cases [Table 2].

Cysticercosis antibody

A total of 200 NCC patients had detailed serology test results, of which 129 patients were positive (65%). Detection of antibodies in CSF was performed in 64 patients, of which 44 (68.8%) were positive [Table 2].

Of the 129 NCC patients with positive serological antibodies, 44 patients (34%) were in the vesicular stage, 57 (44%) in the degenerative stage, and 28 (22%) in the calcified stage. Furthermore, 49 (38%) patients had intraparenchymal cysts, 27 (21%) had intraventricular cysts, 19 (15%) had subarachnoid cysts, and 34 (26%) had multiple-site cysts [Table 2].

Treatment

All NCC patients were treated with anthelminthic therapy: 440 with albendazole (15 mg/kg/d in two daily doses)

Table 2: Clinical information (*N*=451)

Variable	*n* (%)
Clinical signs/symptoms	
Generalized	139 (30.8)
Focal	86 (19.1)
Headache	142 (31.5)
Other neurological symptoms	84 (18.6)
Cyst(s) location	
Intraparenchymal cyst(s)	218 (48.3)
Intraventricular cyst(s)	63 (14)
Subarachnoid cyst(s)	44 (9.8)
Multiple-site cyst(s)	126 (27.9)
Number of cyst(s)	
1	157 (34.8)
2	19 (4.2)
3	30 (6.7)
>3	245 (54.3)
Stage	
Vesicular stage	180 (39.9)
Degenerative stage	228 (50.6)
Calcified stage	43 (9.5)
Features of MRI	
Ring strengthening	143 (31.7)
Peripheral edema	237 (52.6)
Hydrocephalus	71 (15.7)
EEG	
Normal	112 (50.9)
Abnormal	108 (49.1)
Cysticercosis antibody from serum	
Positive	129 (64.5)
Negative	71 (35.5)
Cysticercosis antibody from CSF	
Positive	44 (68.8)
Negative	20 (31.3)
Surgical treatment	
Yes	68 (12.3)
No	484 (87.7)
Antiparasitic drugs	
Albendazole	440 (79.7)
Praziquantel	80 (14.5)
Albendazole and praziquantel	32 (5.8)

MRI – Magnetic resonance imaging; CSF – Cerebrospinal fluid; EEG – Electroencephalography

for 10 days, 80 with praziquantel (20 mg/kg/d in three daily doses) for 10 days, and 32 with both praziquantel (20 mg/kg/d in three daily doses) and albendazole (15 mg/kg/d in two daily doses) for 10 days. In addition to the antiparasitic treatment, low-dose corticosteroids (5–10 mg/d) was used as an adjunctive anti-inflammatory therapy in all patients. Surgical treatment was applied to 68 patients who had intraventricular or subarachnoid NCC. Minimally invasive neuroendoscopy was used to remove the cysticerci when they were situated in the intraventricular region. In cases of evident hydrocephalus, ventriculoperitoneal shunting was used. Antiepileptic drugs, such as valproic acid and levetiracetam, was given to 86% of NCC patients with seizures [Table 2].

Most patients needed to undergo at least three courses of antiparasitic therapy to achieve a favorable clinical outcome. Each treatment lasted for 10 days, and a second deworming was done after 3 months. After receiving treatment, the clinical outcome was favorable in 458 (83%) cases, unfavorable in 29 cases, and unknown in 65 cases (due to lost to follow-up).

DISCUSSION

In this retrospective study, we present the clinical profiles of 552 NCC patients over a 5-year period. The age group 14–44 years accounted for about 35% of the patients, which aligns with the findings of a previous study.[22] In addition, a significantly higher proportion of the patients in the present study were male, which is consistent with the findings of a comprehensive study from Shandong Province, another region in China with a high prevalence of cysticercosis.[24]

In the current study, one-third of the patients belonged to the Bai ethnicity, indicating that their dietary practice of consuming raw/undercooked pork is the most important contributing factor of NCC, an observation consistent with prior research.[20] There was a concentrated spatial distribution of NCC patients in rural areas, where people are primarily engaged in raising free-range livestock and domestic poultry, suggesting that inadequate environmental sanitation practices may be contributing to the public health issue posed by NCC in Dali City. These findings align with previous studies in other epidemic areas.[25,26]

NCC patients often harbor multiple cysts at different locations and stages within the brain, potentially mimicking a wide range of neurological disorders.[3] Consequently, the clinical manifestations of NCC are diverse and not well understood. Notably, 49.9% of NCC patients with clinical symptoms presented with epileptic seizures, which is lower than the 70%–90% range reported in the literature.[27] One possible reason for this may be a lack of understanding of epilepsy, leading to many atypical seizures being ignored.

The number, location, and viability/stage of cysts play a significant role in determining the clinical manifestations of NCC patients. The diversity of cyst sites and stages may partially account for the wide range of clinical presentations observed in NCC cases.[28] Previous literature indicates that most NCC patients with parenchymal cysts experience epileptic seizures, whereas patients with extraparenchymal cysts tend to exhibit nonspecific symptoms such as dizziness and headaches. Cysts located in the ventricles or subarachnoid space have a tendency to grow and invade the surrounding areas, leading to clinical symptoms associated with hydrocephalus, mass effects, vasculitis, and chronic arachnoiditis, which generally carry a poorer prognosis.[28,29]

Table 3: Clinical symptoms and related findings on neuroimaging

Variable	OR	*P*	95% CI	
			Lower bound	Upper bound
Epileptic seizures/other neurological symptoms				
Number of cyst(s)				
>3	1.289	0.340	0.765	2.171
3	1.345	0.626	0.408	4.434
2	4.931	0.133	0.615	39.536
1	1			
Cyst(s) location				
Intraparenchymal	17.453	<0.001	7.964	38.251
Intraventricular	2.160	0.118	0.823	5.667
Subarachnoid	0.065	<0.001	0.014	0.288
Multiple	1			
Stage				
Vesicular	2.714	0.028	1.115	6.606
Degenerative	102.375	<0.001	28.357	369.601
Calcified	1			
Headache/other neurological symptoms				
Number of cyst(s)				
>3	2.957	<0.001	1.617	5.410
3	5.525	0.005	1.683	18.137
2	5.200	0.150	0.552	48.963
1	1			
Cyst(s) location				
Intraparenchymal	3.020	0.016	1.230	7.410
Intraventricular	6.706	<0.001	2.661	16.899
Subarachnoid	0.760	0.485	0.352	1.641
Multiple	1			
Stage				
Vesicular	1.400	0.382	0.658	2.977
Degenerative	23.800	<0.001	7.121	79.542
Calcified	1			
Headache/epileptic seizures				
Number of cyst(s)				
>3	2.295	0.001	1.395	3.775
3	4.108	0.001	1.760	9.588
2	1.055	0.932	0.312	3.562
1				
Cyst(s) location				
Intraparenchymal	0.173	<0.001	0.093	0.322
Intraventricular	3.105	0.002	1.526	6.316
Subarachnoid	11.765	0.002	2.539	54.504
Multiple	1			
Stage				
Vesicular	0.516	0.163	0.204	1.307
Degenerative	0.232	0.002	0.094	0.574
Calcified	1			

CI – Confidence interval; OR – Odds ratio

Consistent with these findings, our study also revealed that the majority of NCC patients with parenchymal cysts presented with epilepsy/seizures, whereas subarachnoid cysts were less likely to cause such seizures. In addition, headaches were more commonly reported in patients with intraventricular cysts compared with seizures and other neurological symptoms.

In terms of the stages of cerebral cysticercosis, patients with cysts in the vesicular and degenerative stages were found to be more prone to seizures/epilepsy. These findings are consistent with previous literature indicating that patients with NCC in the degenerative stage commonly exhibit various clinical characteristics.[3] While some theories propose that the epileptogenesis in NCC may be attributed to local inflammation and/or the formation of reactive gliotic scars, the underlying molecular mechanisms and regulatory processes involved remain unknown.

Our findings indicate that NCC patients with multiple lesions are more likely to present with headaches compared with patients with single lesions. However, a report by Stelzle *et al*.[30] contradicts this observation and suggests no difference in headache occurrence between single and multiple lesions.

Calcified granulomas are the most common radiological finding in NCC. The presence of calcifications suggests

a previous infection with NCC, which is significant for diagnosing parasitic diseases. In the presence of intracranial calcifications, NCC should be strongly considered.[31] Previous studies have shown that a portion of the calcification is a focus of intermittent seizure activity and is usually associated with peripheral transient perifocal edema, which may be caused by host inflammation. Perifocal edema was defined as MRI-detected episodic edema and enhancement around calcifications closest to the calcified lesion, which is a characteristic pattern of edema caused by disruption of the blood–brain barrier.[32] Patients with calcified NCC may have a higher rate of seizure recurrence compared with other stages of NCC. We hypothesize that exploring the potential advantages of inhibiting calcification formation or suppressing peripheral inflammation to prevent seizures would be a promising direction for future research.

ELISA-based detection of parasitic antigens and antibodies in both serum and CSF is currently used in clinical studies. It is worth noting that circulating parasitic antigens can only be found in the serum of patients with viable parasitic tissue, and their levels decrease rapidly following successful antiparasitic treatment or surgery.[33,34] The detection of cysticercal antibodies in the CSF using ELISA shows higher sensitivity (89%) and specificity (93%) in patients with viable NCC infections.[35] Whereas the sensitivity of serum testing for parenchymal NCC is relatively poor, ranging from 72% to 86%, and it is often negative in patients with only a few live cysts.[36,37]

In our study, serological testing for *T. solium* in serum and/or CSF was reported in only 200 cases with NCC by ELISA test. The probable reason for this is that the diagnosis of NCC primarily relies on neuroimaging (CT or MRI), with the examination of cysticercus antibodies serving as a supplementary tool. About 65% of the patients tested positive for serum cysticercus IgG antibodies and 69% tested positive for IgG antibodies against CSF cysticercus. Notably, the most specific serological test currently available for diagnosing NCC is the enzyme-linked immunoelectrotransfer blot (EITB) assay, which is considered the serological reference standard for NCC diagnosis.[38] However, the EITB assay has a significant limitation in that it exhibits low sensitivity (50%–60%) in patients with only one intracranial cysticercus, particularly when the cysticerci are calcified. Interestingly, antibodies can persist in the body long after the death of parasites. Thus, a positive result in patients with calcified cysticercosis does not necessarily indicate the presence of live parasites.[38] It also implies that our understanding of the biological and immunological characteristics of the tapeworm's various life cycles is still lacking. Sahu *et al*. also found a strong association between the live stage of the parasite and the detection of antibodies in sera and CSF of NCC patients using ELISA with ES antigens.[39] However, we were neither able to determine the circumstances under which patients were tested for cysticercus antibodies nor determine the time point at which it was performed.

Currently, there is no clear consensus on the optimal treatment for NCC. Since the 1980s, albendazole and praziquantel have been widely used in the management of NCC.[40] In our study, albendazole was the most commonly prescribed treatment, which is consistent with the recommendations in the guidelines.[15] However, 80 patients (15%) were treated with praziquantel alone. This may have been due to the findings in previous studies showing that both albendazole and praziquantel are significantly effective in treating cerebral cysticercosis and have similar medium- and long-term efficacy.[41]

The dosage and duration of treatment varied depending on factors such as the location, stage, and number of cysts, as well as the severity of clinical symptoms. Albendazole monotherapy was administered to all cases with a single intraparenchymal NCC. Adjunctive corticosteroid therapy was used in all patients who received antiparasitic therapy, which is consistent with the recommendations in the guidelines. Further, 68 NCC patients underwent surgery, and minimally invasive neuroendoscopy was used to remove the cysticerci when they were situated in the intraventricular region. In cases of evident hydrocephalus, ventriculoperitoneal shunting was used. However, it should be noted that not all cysticerci can be removed during surgery, and thus anti-parasitic drugs are still required after the procedure.

In this study, multiple sites of cysts were present in 126 patients, but only 32 received combined antiparasitic treatment. According to the Guidelines for the Clinical Management of Patients with NCC,[15] patients with more than two viable parenchymal cysticerci should be treated with a combination of albendazole and praziquantel, rather than albendazole monotherapy. The findings of this study should be an indicator for clinicians to standardize treatment according to the accepted guidelines.

Schistosomiasis, a parasitic infection caused by *Schistosoma japonicum*, has similar neurological symptoms to NCC such as focal or generalized seizures. Other clinical manifestations are associated with increased intracranial pressure.[42] However, both these diseases differ in terms of their source, route of infection, and transmission.

Nonetheless, Dali, Yunnan Province, previously had a high prevalence of schistosomiasis, and thus it is important to differentiate between the two.

In schistosomiasis, the infection occurs when the skin comes into contact with contaminated freshwater, typically while swimming. In rare cases, they can affect the CNS.[42,43] On the other hand, NCC is the most common parasitic infection affecting the CNS.[9] Typically, single or multiple mass-like lesions in NCC are observed in MRI, primarily affecting the cortex and subcortical areas of the cerebral hemispheres. Less commonly, the lesions may involve the cerebellum and brainstem. On contrast-enhanced MRI, these lesions exhibit a unique enhancement pattern characterized by clusters of central linear enhancement surrounded by multiple enhancing punctate nodules, which is often referred to as an "arborized" appearance.[44] The tendency for these foci to fuse together is a distinctive characteristic of cerebral schistosomiasis, allowing differentiation from NCC and brain tumors.[42,44,45] To confirm the diagnosis, immunological tests can be conducted on serum or CSF. Therefore, based on the patient's history of schistosome exposure, positive results from serological screening tests (or CSF/feces analysis), and the characteristic imaging features observed on CT/MRI, it was possible to differentiate NCC from neuroschistosomiasis in our study. Nonetheless, it should be noted that due to sustained efforts through the implementation of a new integrated control strategy in 2004 in China,[46-48] the morbidity and prevalence of schistosomiasis in Dali City is currently at record low levels.

CONCLUSION

This study contributes to a better understanding of the features of cerebral cysticercosis and offers a comprehensive depiction of the demographic and clinical profiles of NCC patients in Dali, Yunnan Province, China. Our findings unequivocally affirm that the neurological symptoms associated with NCC exhibit substantial diversity contingent upon the number, location, and developmental stage of the parasites. Epilepsy/seizures and headaches were found to be predominant manifestations of NCC, and thus NCC can be considered as a key contributor to preventable epilepsy within Dali city, China.

Ethical considerations

The study was approved by the Ethics Committee of The First Affiliated Hospital of Dali University, Dali, Yunnan, China (Project No.: DFY20220301001; date: March 1, 2022). Owing to the study design, written informed consent for participation was not required in accordance with the national legislation and institutional requirements. The study adhered to the principles of the Declaration of Helsinki, 2013.

Peer review

This article was peer-reviewed by three independent and anonymous reviewers.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author contributions

Conceptualization: Y.L. and H-X.Z.; Methodology: Y-C.L. and L-J.C.; Data analysis: Y-C.L., Y-H.C., W.G. and X-P.Y.; Writing–original draft preparation: H-X.Z.; Writing – review and editing: D-D.G. and R-X.C; Supervision: Y.L.

All authors have read and agreed to the published version of the manuscript.

Financial support and sponsorship

This work was funded by a grant (No.: 2022J0703) from the Scientific Research Fund Project of Yunnan Provincial Department of Education, a grant (No.: 2021KBG047) from the Dali Science and Technology Bureau Project, and a grant from the Science and Technology Talent Project (Reserve Talent) of The First Affiliated Hospital of Dali University.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Garcia HH, Gonzalez AE, Gilman RH. Neurocysticercosis as an eradicable cause of epilepsy: A plan and actions are needed. JAMA Neurol 2021;78:1045-6.
2. Del Brutto OH. Current approaches to cysticidal drug therapy for neurocysticercosis. Expert Rev Anti Infect Ther 2020;18:789-98.
3. Garcia HH, Gonzalez AE, Gilman RH. *Taenia solium* cysticercosis and its impact in neurological disease. Clin Microbiol Rev 2020;33:e00085-19.
4. Del Brutto OH. Neurocysticercosis. Continuum (Minneap Minn) 2012;18:1392-416.
5. Flisser A. Taeniasis and cysticercosis due to *Taenia solium*. Prog Clin Parasitol 1994;4:77-116.
6. Lescano AG, Garcia HH, Gilman RH, Gavidia CM, Tsang VC, Rodriguez S, *et al. Taenia solium* cysticercosis hotspots surrounding tapeworm carriers: Clustering on human seroprevalence but not on seizures. PLoS Negl Trop Dis 2009;3:e371.
7. Mendlovic F, Garza-Rodríguez A, Carrillo-Farga J, González-Domínguez F, Maravilla P, Flisser A. From stillness to motion: 80 years after the first description of *Taenia solium* oncosphere hatching. Parasit Vectors 2014;7:12.
8. Garcia HH. Neurocysticercosis. Neurol Clin 2018;36:851-64.
9. Xu LQ, Jiang ZX, Zhou CH, Zhang XQ, Ren ZX, Chang J, *et al.* Distribution of cysticercosis in China. Zhongguo Ji Sheng Chong Bing Fang Zhi Za Zhi 1999;12:30-2. (In Chinese).

10. Li T, Chen X, Wang H, Openshaw JJ, Zhong B, Felt SA, *et al.* High prevalence of taeniasis and *Taenia solium* cysticercosis in children in Western Sichuan, China. Acta Trop 2019;199:105133.
11. Zang XZ, Li HZ, Qian MB, Chen YD, Zhou CH, Liu HK, *et al.* Extensive disseminated cysticercosis: A case report in Yunnan province, China. BMC Infect Dis 2019;19:535.
12. Qian MB, Xiao N, Li SZ, Abela-Ridder B, Carabin H, Fahrion AS, *et al.* Control of taeniasis and cysticercosis in China. Adv Parasitol 2020;110:289-317.
13. Fleury A, Escobar A, Fragoso G, Sciutto E, Larralde C. Clinical heterogeneity of human neurocysticercosis results from complex interactions among parasite, host and environmental factors. Trans R Soc Trop Med Hyg 2010;104:243-50.
14. Nash TE, Mahanty S, Garcia HH, Cysticercosis Group in Peru. Corticosteroid use in neurocysticercosis. Expert Rev Neurother 2011;11:1175-83.
15. White AC Jr., Coyle CM, Rajshekhar V, Singh G, Hauser WA, Mohanty A, *et al.* Diagnosis and treatment of neurocysticercosis: 2017 clinical practice guidelines by the infectious diseases society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH). Clin Infect Dis 2018;66:e49-75.
16. Carabin H, Ndimubanzi PC, Budke CM, Nguyen H, Qian Y, Cowan LD, *et al.* Clinical manifestations associated with neurocysticercosis: A systematic review. PLoS Negl Trop Dis 2011;5:e1152.
17. Bustos JA, García HH, Del Brutto OH. Antiepileptic drug therapy and recommendations for withdrawal in patients with seizures and epilepsy due to neurocysticercosis. Expert Rev Neurother 2016;16:1079-85.
18. Garcia HH, Nash TE, Del Brutto OH. Clinical symptoms, diagnosis, and treatment of neurocysticercosis. Lancet Neurol 2014;13:1202-15.
19. Singh AK, Singh SK, Singh A, Gupta KK, Khatoon J, Prasad A, *et al.* Immune response to *Taenia solium* cysticerci after anti-parasitic therapy. Int J Parasitol 2015;45:749-59.
20. Li H, Zang X, Hu X, Abe EM, Qian M, Xue J, *et al.* Spatio-temporal distribution characteristics of cysticercosis from 2000 to 2014 in Dali, Yunnan province, China. Geospat Health 2020;15 [doi: 10.4081/gh.2020.815].
21. Del Brutto OH, Nash TE, White AC Jr., Rajshekhar V, Wilkins PP, Singh G, *et al.* Revised diagnostic criteria for neurocysticercosis. J Neurol Sci 2017;372:202-10.
22. Herrador Z, Pérez-Molina JA, Henríquez Camacho CA, Rodriguez-Guardado A, Bosch-Nicolau P, Calabuig E, *et al.* Imported cysticercosis in Spain: A retrospective case series from the+REDIVI collaborative network. Travel Med Infect Dis 2020;37:101683.
23. Scheffer IE, Berkovic S, Capovilla G, Connolly MB, French J, Guilhoto L, *et al.* ILAE classification of the epilepsies: Position paper of the ILAE commission for classification and terminology. Epilepsia 2017;58:512-21.
24. Huang X, Wang Z, Kou J, Liu H, Mao D, Yu Z, *et al.* A large cohort of neurocysticercosis in Shandong province, Eastern China, 1997-2015. Vector Borne Zoonotic Dis 2019;19:901-7.
25. Carabin H, Millogo A, Cissé A, Gabriël S, Sahlu I, Dorny P, *et al.* Prevalence of and factors associated with human cysticercosis in 60 villages in three provinces of Burkina Faso. PLoS Negl Trop Dis 2015;9:e0004248.
26. Zammarchi L, Strohmeyer M, Bartalesi F, Bruno E, Muñoz J, Buonfrate D, *et al.* Epidemiology and management of cysticercosis and *Taenia solium* taeniasis in Europe, systematic review 1990-2011. PLoS One 2013;8:e69537.
27. Debacq G, Moyano LM, Garcia HH, Boumediene F, Marin B, Ngoungou EB, *et al.* Systematic review and meta-analysis estimating association of cysticercosis and neurocysticercosis with epilepsy. PLoS Negl Trop Dis 2017;11:e0005153.
28. Burneo JG, Cavazos JE. Neurocysticercosis and epilepsy. Epilepsy Curr 2014;14:23-8.
29. Takayanagui OM, Odashima NS. Clinical aspects of neurocysticercosis. Parasitol Int 2006;55:S111-5.
30. Stelzle D, Abraham A, Kaminski M, Schmidt V, De Meijere R, Bustos JA, *et al.* Clinical characteristics and management of neurocysticercosis patients: A retrospective assessment of case reports from Europe. J Travel Med 2023;30:taac102.
31. Peisen F, Thaiss WM, Krumm P, Horger M. Calcified cysticercosis and differential diagnosis of intracranial, cutaneous and subcutanoeus calcifications. Rofo 2019;191:595-8.
32. Fujita M, Mahanty S, Zoghbi SS, Ferraris Araneta MD, Hong J, Pike VW, *et al.* PET reveals inflammation around calcified *Taenia solium* granulomas with perilesional edema. PLoS One 2013;8:e74052.
33. Zamora H, Castillo Y, Garcia HH, Pretell J, Rodriguez S, Dorny P, *et al.* Drop in antigen levels following successful treatment of subarachnoid neurocysticercosis. Am J Trop Med Hyg 2005;73:s41.
34. Fleury A, Garcia E, Hernández M, Carrillo R, Govezensky T, Fragoso G, *et al.* Neurocysticercosis: HP10 antigen detection is useful for the follow-up of the severe patients. PLoS Negl Trop Dis 2013;7:e2096.
35. Odashima NS, Takayanagui OM, Figueiredo JF. Enzyme linked immunosorbent assay (ELISA) for the detection of IgG, IgM, IgE and IgA against cysticercus cellulosae in cerebrospinal fluid of patients with neurocysticercosis. Arq Neuropsiquiatr 2002;60:400-5.
36. Rodriguez S, Wilkins P, Dorny P. Immunological and molecular diagnosis of cysticercosis. Pathog Glob Health 2012;106:286-98.
37. Garcia HH, Harrison LJ, Parkhouse RM, Montenegro T, Martinez SM, Tsang VC, *et al.* A specific antigen-detection ELISA for the diagnosis of human neurocysticercosis. The cysticercosis Working Group in Peru. Trans R Soc Trop Med Hyg 1998;92:411-4.
38. Garcia HH, O'Neal SE, Noh J, Handali S, Cysticercosis Working Group in Peru. Laboratory diagnosis of neurocysticercosis (*Taenia solium*). J Clin Microbiol 2018;56:e00424-18.
39. Sahu PS, Parija SC, Narayan SK, Kumar D. Evaluation of an IgG-ELISA strategy using *Taenia solium* metacestode somatic and excretory-secretory antigens for diagnosis of neurocysticercosis revealing biological stage of the larvae. Acta Trop 2009;110:38-45.
40. Ma YX. A clinical study on the treatment of cerebral cysticercosis with praziquantel. Zhonghua Yi Xue Za Zhi 1984;64:79-83.
41. Wu W, Jia F, Wang W, Huang Y, Huang Y. Antiparasitic treatment of cerebral cysticercosis: Lessons and experiences from China. Parasitol Res 2013;112:2879-90.
42. Suthiphosuwan S, Lin A, Gao AF, Munoz DG, Spears J, Bharatha A. Delayed presentation of cerebral schistosomiasis presenting as a tumor-like brain lesion. Neuroradiol J 2018;31:395-8.
43. Ferrari TC, Moreira PR. Neuroschistosomiasis: Clinical symptoms and pathogenesis. Lancet Neurol 2011;10:853-64.
44. Wu L, Wu M, Tian D, Chen S, Liu B, Chen Q, *et al.* Clinical and imaging characteristics of cerebral schistosomiasis. Cell Biochem Biophys 2012;62:289-95.
45. Shih RY, Koeller KK. Bacterial, fungal, and parasitic infections of the central nervous system: Radiologic-pathologic correlation and historical perspectives. Radiographics 2015;35:1141-69.
46. Li Y, Dang H, Zhang L, Li G, Shan X, Yuan M, *et al.* Schistosomiasis surveillance – China, 2015-2018. China CDC Wkly 2020;2:39-43.
47. Yang Y, Zhou YB, Song XX, Li SZ, Zhong B, Wang TP, *et al.* Integrated control strategy of schistosomiasis in the people's republic of China: Projects involving agriculture, water conservancy, forestry, sanitation and environmental modification. Adv Parasitol 2016;92:237-68.
48. Chun-Hong D, Yun Z, Ming-Shou W, Xi-Guang F, Mei-Fen S, Chun-Qiong C, *et al.* Construction and operation of schistosomiasis diagnosis reference (network) laboratory in Yunnan province. Zhongguo Xue Xi Chong Bing Fang Zhi Za Zhi 2019;31:329-32.