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Prevalence, clinical characteristics, and seizure outcomes of epilepsy due to calcific clinical stage of neurocysticercosis: Study in a rural community in south India

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ABSTRACT

Objective: The objective of this research was to study the prevalence, clinical characteristics, and seizure remission rates of epilepsy due to calcific stage of neurocysticercosis (cNCC) in a rural community in south India.
Material and methods: Comprehensive Rural Epilepsy Study South India (CRESSI) is a prospective longitudinal study of epilepsy care in a rural community in south India. As part of this study, prevalence of epilepsy was studied in a population of 74,086 in 22 villages. The prevalence study identified 451 people with epilepsy including 62 (13.7%) with epilepsy due to cNCC. Diagnosis of cNCC was based on computed tomography (CT) findings. The clinical characteristics and seizure outcomes were studied in this cohort of 62 patients. The data collected included demographics, seizure type, antiepileptic drugs (AEDs), seizure remission rates, and predictors of long-term seizure remissions.
Results: The crude prevalence of epilepsy due to cNCC in this rural community was 0.84 per 1000 (95% confidence interval [CI]: 0.65–1.07). This lesion accounted for 41% of the established etiology among 451 prevalence cases of epilepsy. Mean age at presentation was 28.87 ± 14.45 (range: 8–65 years) with equal gender distribution. The common location of the lesion was in the perirolandic region. Focal onset motor seizures were the common seizure type. Seizure remission (≥2 years) rate was 80.3%. The independent predictor of drug resistance was failure to respond to monotherapy (odds ratio: 63.9; 95% CI: 8.4–485.4; $p < 0.0001$). Focal impaired awareness behavioral arrest/automatisms with lesion located in the temporal lobe in all the three patients were drug-resistant.
Conclusions: In this rural community in south India, epilepsy due to cNCC was the commonest acquired epilepsy in people aged ≥20 years. Long-term seizure remission rates were high, and failure to respond to monotherapy was the predictor of drug resistance. Drug-resistant epilepsy was extremely rare with this lesion.

1. Introduction

Single or multiple calcific lesions measuring less than 20 mm are a common computed tomography (CT) finding in patients with epilepsy in countries endemic to neurocysticercosis (NCC) [1–5]. This lesion represents the residual parenchymal lesion in the life cycle of the pork tapeworm, *Taenia solium* [2,6–8]. Community-based prevalence studies in the countries endemic to NCC reported high prevalence of epilepsy due to calcific stage of NCC (cNCC) [9–11]. The debating issue is whether this CT brain lesion in people with epilepsy (PWE) is an innocent bystander or the cause of epilepsy. A recent large community CT-based study in rural Ecuador provided robust epidemiological evidence favoring the relationship between cNCC and epilepsy. This study demonstrated that PWE in the study population had three times the odds of having cNCC than those without epilepsy [12]. The epilepsy due to cNCC lesion is not well characterized [4]. This study characterizes the epilepsy due to cNCC lesion in a rural community in south India.

2. Material and methods

Byrraju Foundation is a nongovernmental organization in Hyderabad, India. As part of its community service, it adopted 68 villages as part of its rural uplift program around Bhimavram town in West Godavari district in Andhra Pradesh, a province in south India. The Foundation initiated skill development programs, public health programs, and many health initiatives as part of its rural uplift program.

The organizational structure of the Foundation is given in Fig. 1. Village health center (VHC) in the adopted villages is the nodal operational unit in the organizational structure of the Foundation. It is manned by a medical graduate, who is assisted by multipurpose health

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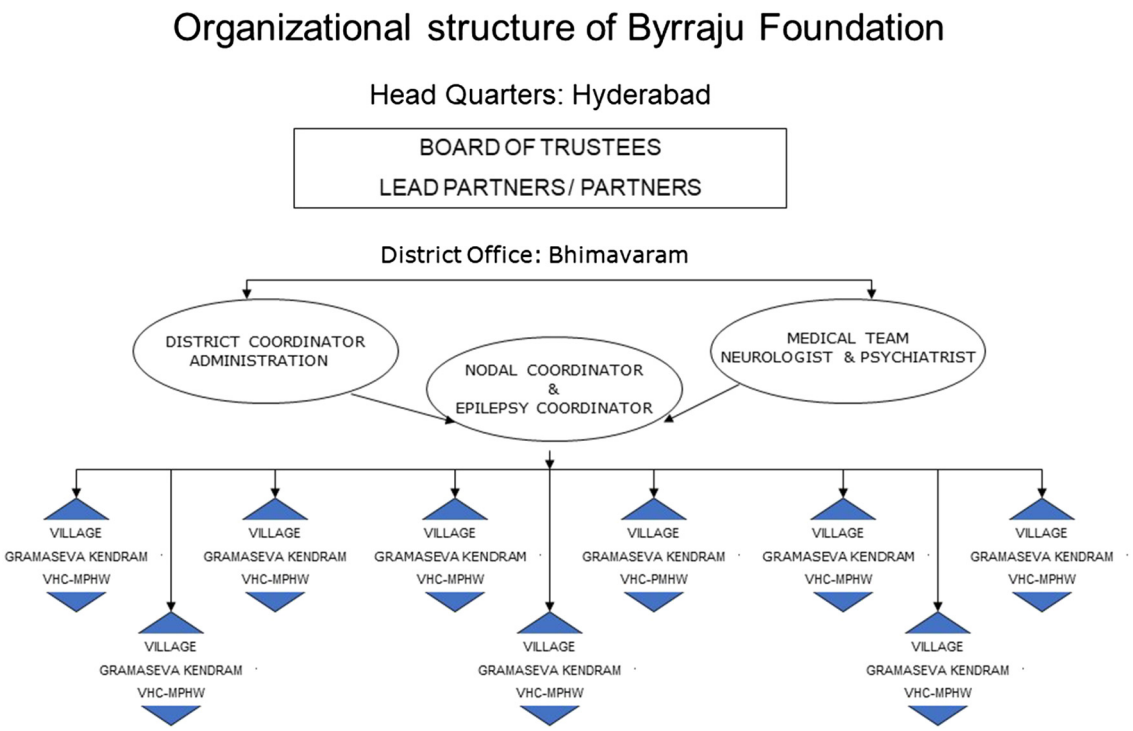

Fig. 1. The organizational structure of the Byrraju Foundation (EC: epilepsy coordinator; MPHW: multipurpose health worker).

worker (MPHW), and administrative staff. Doctor runs outpatient clinic six days in a week and attends to the common health ailments and also helps in the other health initiatives. Multipurpose health worker helps the doctor to run the outpatient clinic, and dispenses the drugs and counsels the patients regarding the importance of drug adherence. Multipurpose health worker also acts as village coordinator and coordinates activities of all the health initiatives run by the Foundation. The Foundation provides drugs free of cost for chronic diseases like hypertension, diabetes, and epilepsy once a month. Nodal coordinator coordinates the administrative activities of all the programs including health initiatives of the Foundation in the villages under his jurisdiction. Gramaseva Kendram is attached to the VHS and includes village elders and leaders. They act as liaison between the Foundation and the community. They help in improving the awareness about various diseases among the community and also encourage the patients to attend VHC at regular intervals.

We are involved in the Comprehensive Rural Epilepsy Study South India (CRESSI), a model of care for PWE initiated by the Foundation. The objectives of CRESSI model of care were to identifying PWE in the adopted villages by the Foundation and provide antiepileptic drugs (AEDs) free or at an affordable cost and also to improve knowledge about epilepsy and create a positive attitude among the community towards PWE. This model of care was integrated with the organizational structure of the Foundation (Fig. 1). To the existing organizational structure, four epilepsy coordinators (EC) were recruited. They had basic training in identifying PWE, treatment of epilepsy, monitoring drug adherence, drug adverse events, and management of breakthrough seizures. The medical team includes a neurologist (JMKM) and a psychiatrist (VS), both with interest in epilepsy.

As part of CRESSI model of care, point prevalence of epilepsy (prevalence day: January 1, 2001) was studied by door-to-door survey in the 22 randomly selected villages from among the 68 adopted villages by the Foundation. The 22 adopted villages had a population of 74,086 and 15,355 households. The study population included subjects of both genders and all age groups except infants. The instrument used was the questionnaire developed by Institute of Tropical Medicine, Limos, France. The questionnaire was validated in the Neurology Outpatient Clinic, CARE Hospitals, Hyderabad. The study methodology in brief was as follows: Step I: Identifying the probable subjects with epilepsy visiting every household (door-to-door survey) in all the 22 adopted villages by the EC and MPHW attached to the VHC. They were trained in the administration of the questionnaire; Step II: Probable subjects with epilepsy were brought to the VHC, where the diagnosis of epilepsy (≥2 unprovoked seizures) was ascertained by the medical team; Step III: The data collected included demographics, age of onset and duration of seizures, detailed eyewitness account of seizure semiology, neurologic findings, drug history, and present medication; Step IV: Diagnostic workup included plain and contrast brain CT and electroencephalography (EEG) in a selected group of patients. Classification of seizures and epilepsies was done by the medical team. In the event of disagreement, the final results of classification were based on discussion of the eye witness account of the seizure semiology and EEG and contrast CT findings; Step V: Patients with epilepsy were prescribed appropriate AEDs based on the seizure type, epilepsy, or epilepsy syndromes. Only standard AEDS, phenytoin, carbamazepine, valproate, and phenobarbital, were the drugs prescribed. Free drugs were given by the Foundation for a month during the monthly visits of patients to the VHC or delivered at home by the MPHW; and Step VI: Patients were followed up at 3-month interval by the medical team. During the follow-up visits, the data collected included the following: drug adherence, seizure remission, breakthrough seizures, and adverse drug events. The EC visits each village once a month and collects the information regarding the drug adherence, adverse drug events, and seizure recurrence and sends the information to the neurologist.

The study identified 462 persons with ≤2 epileptic seizures. For this prevalence analysis, patients with epilepsy due to cNCC on the prevalence date (January 1, 2001) were only included. None of the screen-failed subjects for epilepsy developed unprovoked seizures during the follow-up period of 10 years. It is very unlikely that we could have missed any cases as the CRESSI model of care brings healthcare services to the proximity of people's homes.

2.1. Operational definitions

2.1.1. Neurocysticercosis

Diagnosis of NCC was based on the diagnostic criteria proposed by Del Brutto et al. [13].

2.1.2. Epilepsy

Epilepsy was defined as recurrence of two or more unprovoked epileptic seizures 24 h apart [14].

2.1.3. Seizure categorization

Seizures due to degenerative phase of NCC were categorized under acute symptomatic seizures, and seizures due to cNCC were categorized under unprovoked seizures [2].

2.1.4. Seizure type

Seizures were classified based on the mode of onset into focal, generalized, and unknown onset seizure using the recent seizure classification proposed by the International League Against Epilepsy 2017 [15].

2.1.5. Seizure remission

Patient was considered to be in seizure remission when the patient was seizure-free for two or more years on AEDs.

2.2. Statistical methods

Point prevalence of active epilepsy as on January 1, 2001 was estimated and expressed as number of PWE per 1000 population. Categorical variables were represented as frequencies and percentages. The statistical significance in the difference of the outcome variables was assessed by chi-square test, and p value <0.05 was considered as statistically significant. Multivariate logistic regression was used for predicting the outcome variable. R-Studio was used for analysis. This project is a prospective ongoing study and has been approved by the Institutional Ethics Committee, CARE Hospitals, Hyderabad, India.

3. Results

3.1. Prevalence of epilepsy due to cNCC

The prevalence study identified 462 people with ≥2 epileptic seizures on the prevalence day, January 1, 2001. Of the 462 patients with ≥2 seizures, in 83 persons, a putative etiology could be established from history, physical and neurologic examinations, and EEG findings. Of the remaining, 379 (82%) persons had plain and contrast CT of the brain. Computed tomography showed single or multiple cNCC lesions in 62 (16.35%) patients and cysticercus lesions in degenerative phase in 11. Seizures due to degenerative phase of NCC were categorized under acute symptomatic seizures and were excluded from further analysis. The details of the seizure disorder due to degenerative phase have been published earlier in another journal [16]. Seizures due to cNCC were categorized under unprovoked seizures, and patients with ≥2 seizures were considered to have epilepsy [2]. The crude prevalence of epilepsy due to cNCC was 0.84 per 1000 (95% confidence interval [CI]: 0.65–1.07) (Table 1).

Table 1
Prevalence of seizure disorder due to various evolutive stages of NCC.

Seizure disorder due to all evolutive stages of NCC	0.98 per 1000[a] (95% CI: 0.78–1.23)
Acute symptomatic seizures due to degenerative phase of NCC	0.14 per 1000 (95% CI: 0.08–0.26)
Epilepsy (unprovoked seizures) due to cNCC	0.84 per 1000 (95% CI: 0.65–1.07)

[a] Patients in all groups had ;≥2 seizures.

Table 2
Epilepsy due to cNCC: clinical characteristics.

Age distribution	
Age (yrs)	n (%)
1–10	6 (10)
11–20	14 (23)
21–30	13 (21)
31–40	15 (24)
;≥40	14 (22)

Mean age: 28.87 + 14.45 (range: 8–65 years).
Male:female: 32:30.
Seizure clusters: 23% of patients.
Long interval between first seizure and second seizure in 18% of patients.

3.2. Clinical characteristics of epilepsy due to cNCC

The mean age of patients with epilepsy due to cNCC at presentation was 28.87 ± 14.45 years (range: 6–65 years). Epilepsy due to cNCC was more common in people aged ≥20 years. The gender distribution was equal (male:female was 32:30). Seizure clusters occurred at the time of presentation or during the early course of the disease in 14 (22.6%) patients. The interval between the first unprovoked and the second seizure was more than 5 years in 11 (17.7%) patients. Of the 62 patients with epilepsy due to cNCC, 51 (82%) had single lesion, and 11 (18%) had multiple lesions (2–3 lesions) (Table 2). None of these patients had any other cause to attribute for the seizure disorder. This lesion accounted for 41% of the established etiology among the 451 prevalence cases of epilepsy.

The CT location of the lesion(s) was frontal in 15 (24%);, perirolandic in 25 (40.3%);, posterior parietal in 12 (19.4%);, temporal in 3 (5%), and occipital in 7 (11.3%). Focal onset seizure was the common seizure type in 74% of patients (Table 3). None of the patients had EEG. Seizure type was determined by the detailed analysis of seizure semiology. The clinical seizure pattern was clearly distinctive to allow it to be localized in to the location of the CT lesion or nearer to the lesion in 46 (74%) (temporal in 3;, occipital in 3;, frontal in 15;, and perirolandic in 25) patients. There was discordance between seizure semiology and CT location of the lesion in 16 (26%) (posterior parietal in 12 and occipital in 4) (Table 3). Of the 62 patients with cNCC, perilesional edema was observed around a single lesion in 21 (34%) of the scans, a proven association with seizures in these patients [17].

Of the 62 patients, 51 (83.8%) patients were on monotherapy (carbamazepine or phenytoin), and 10 patients were on dual therapy (carbamazepine + valproate or phenytoin + phenobarbital). Of the 61 (one patient migrated out of the study area), 49 (80.3%) patients achieved seizure freedom for ≥2 years. Three patients with lesion in the temporal lobe and focal impaired awareness behavioral arrest/automatisms seizures had no seizure remission. The remaining 9 patients had infrequent seizures. The determinants of seizure remission on univariate analysis were absence of seizure clusters at presentation or

Table 3
Seizure classification using ILAE 2017 classification and location of the lesion in brackets.

Seizure type		n 62 (%)
Focal onset seizures		46 (74)
Focal aware motor-onset seizures (perirolandic)	3	
Focal impaired awareness motor-onset seizures (frontal or perirolandic)	37 (60)	
Focal impaired awareness sensory (visual) (occipital)	3	
Focal impaired awareness behavioral arrest/automatisms seizure (temporal)	3	
Generalized onset		10 (16)
Generalized-onset motor tonic–clonic (posterior parietal)	10	
Unknown onset motor seizure (Occipital)		6 (10)

Table 4
Epilepsy due to cNCC: seizure outcome by seizure type.

Seizure remission for ;≥2 yrs	49/61[a] (80.3%)
Seizure type – seizure remission	
Focal impaired awareness behavioral arrest/automatisms seizure	0/3 (0)[b]
Focal onset seizure	35/43 (81.6%)
Generalized or unknown onset motor seizure	15/15 (100%)

Seizure remission (univariate analysis)

	Seizure clusters (n %)	Monotherapy (n %)
Yes	9/14 (64)	48/51 (94)
No	41/47 (87)	2/10 (20)
p value (95% CI)	<0.05 (75.3–94.7)	<0.001(84.8–98.5)

Seizure remission (multivariate analysis)

	Monotherapy (n %)	Duotherapy (n %)
	48/51 (94)	2/10 (20)
Odds ratio	63.9 (95% CI: 8.4–485.4) p value < 0.0001	

[a] One patient migrated to other village after marriage.
[b] The location of the calcific lesion was in temporal lobe.

during early course (9/14 (64%) vs. 41/47 (87%); $p < 0.05$; 95% CI: 75.3–94.7) and response to monotherapy (48/51 (94%) vs. 2/10 (20%); $p < 0.001$; 95% CI: 84.8–98.5). Multivariate analysis showed failure to respond to monotherapy as an independent predictor of drug resistance [48/51 (94%) vs. 2/10 (20%); odds ratio [OR]: 63.9 (95% CI: 8.4–485) [4]] (Table 4). There were no differences in the remission rates between those with single calcific lesion and multiple calcific lesions. None of the three patients with drug-resistant epilepsy with temporal lobe location of the lesion had magnetic resonance imaging (MRI) to exclude secondary mesial temporal sclerosis [18]. None of the patients had serious adverse drug events. In our earlier study, it was shown that the risk of relapse was high with the withdrawal of AEDs, and hence, the patients were advised to have good drug adherence [4].

4. Discussion

In this rural community in south India, the crude prevalence of epilepsy due to cNCC was 0.84 per 1000 population. This etiology accounted for 41% of the established etiology among 451 prevalence cases of epilepsy. The clinical characteristics of epilepsy due to cNCC included the following: more frequency among people aged ≥20 years with equal gender distribution; common seizure type was focal onset motor; high seizure density (seizure cluster) in the initial course of the disease; there can be discordance between seizure semiology and the location of the lesion on neuroimaging; good long-term seizure remission, most commonly with monotherapy; and the determinants of drug resistance were failure to respond to monotherapy. All the three patients with temporal lesion location and complex partial seizures developed drug-resistant epilepsy.

Discordance between clinical localization based on seizure semiology and location of the lesion on neuroimaging is not an uncommon feature in patients with focal epilepsies [19–21]. Clinical characteristics and seizure spread patterns are well studied in occipital lobe epilepsy [22]. Of the 62 patients, in 46 (74%) patients, the seizure semiology allowed localization to the CT location of the lesion or nearer to the CT lesion. There was discordance between seizure semiology and location of CT lesion in 16 (26%) patients. Similar were the findings in our earlier hospital-based study in patients with epilepsy due to cNCC [4]. Remi et al. [23] compared the occurrence and localization of epileptic seizure patterns (ESPs) with the localization of MRI lesion. This study showed that the occurrence and localization of ictal findings differ vastly for lesions in different brain regions. The discrepancies of ESPs with MRI lesions are likely to be more in frontal, parieto-occipital, and central brain regions when compared to temporal lobe [23].

In the cross-sectional observational studies, the reported frequency of perilesional edema in patients with epilepsy due to cNCC ranged from 23% to about 35% [1,24,25]. In this study of the 62 patients, CT brain showed perilesional edema in 21 (34%) patients. A cross-sectional case-controlled showed a significant association between perilesional edema and seizures ($p = 0.002$) [17]. A plausible hypothesis is that perilesional edema represents an inflammatory response to calcified granulomas. The speculation is that an antigen is sporadically released and recognized by the host leading to episodic response [26].

High prevalence of NCC has been estimated in countries endemic to cysticercosis. In the meta-analysis of 12 studies conducted in Latin America, sub-Saharan Africa, and Southeast Asia, the proportion of NCC among persons with epilepsy was estimated at 29.62% (95% CI: 23.5%–36.1%) [27]. The association between NCC and epilepsy was significant, OR of 2.8 (95% CI: 1.9–4.0) [28]. A recent large CT-based study in Ecuador demonstrated that PWE had three times the odds of having cNCC than those without epilepsy, providing robust epidemiological evidence favoring the relationship between cNCC and epilepsy [12].

In this rural community-based study, the crude prevalence of epilepsy due to cNCC was 0.84 (95% CI: 0.65–1.07) per 1000 population. The other prevalence studies in India suggested similar prevalence rates. The reported crude prevalence of epilepsy due to cNCC in another study from south India was 0.91 per 1000 population [9]. In a rural community-based study in Uttarakhand, a province in north India, the reported crude prevalence was 0.99 per 1000 population [10].

Neurocysticercosis is the most common acquired infectious cause of epilepsy worldwide [29]. In countries endemic to NCC, cNCC is a common cause of epilepsy in people ≥20 years of age [12]. In this prevalent cohort, the age distribution was the same. Similar was the age distribution in the two hospital-based studies from India [4,5]. The seizure and epilepsy type were focal onset and motor, similar to the observations in the other studies [4,5].

In this prevalent cohort, ≥2 years seizure remission was 80.3%. The independent predictor of drug resistance in this study was failure to respond to monotherapy. In our earlier study, 71.5% (95% CI: 7–85.4) of patients achieved 3-year remission, and 66% (95% CI: 32.4–88.2) achieved 5-year remission [4]. In the study by Leon and colleagues, seizure freedom for ≥3 months in patients with cNCC was 46.03%, and there was no difference when compared to epilepsies due to other structural brain lesions (47.62%) [30].

Drug resistance is very rare with this type of lesion in epilepsy. In a study of 512 patients with drug-resistant epilepsy from Brazil, only 8 patients had isolated calcific lesion, and in only two patients, drug-resistant epilepsy was considered to be due to calcification lesion [31]. In a large volume epilepsy surgery center in India, 35 (0.9%) of 3895 patients with drug-resistant epilepsy had epilepsy due to cNCC [32].

In a review assessing the role of the cysticercosis in medically-refractory epilepsy, three different clinical presentations were identified as follows: (i) the lesion was epileptogenic; (ii) there was dual pathology (cysticercosis, with other lesion usually being hippocampal sclerosis); and (iii) the lesion was not related to the epileptogenic focus [33]. There is growing evidence to suggest the possibility of NCC playing a role in the pathophysiology of mesial temporal lobe epilepsy hippocampal sclerosis [34–36]. In this prevalent cohort, all the 3 (4.8%) patients with mesial temporal seizure semiology were drug-resistant. Similarly, in our earlier hospital-based study of 97 patients, 4 (4.1%) with mesial temporal seizure semiology had drug-resistant epilepsy [4]. In all the 7 patients, the calcific lesion was in the temporal lobe. None of them had MRI. In addition to temporal location of the lesion, perilesional gliosis is another important substrate for drug-resistant epilepsy in patients harboring a cNCC lesion [33,37,38].

Epilepsy due to cNCC is a common cause of acquired epilepsy in countries endemic to NCC in people aged ≥20 years. Seizure type is often focal onset motor seizures. In the absence of neuroimaging, there is a risk of categorizing these patients under epilepsy due to unknown cause. Long-term seizure remission can be achieved, most

often with monotherapy in 4 out of 5 patients. Drug resistance is extremely uncommon.

Certificate

'We confirm that we have read the journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.'

Declaration of Competing Interest

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References

[1] Del Brutto OH, Santibanez R, Noboa CA, Aguirre R, Diaz E, Alarcin TA. Epilepsy due to neurocysticercosis: analysis of 203 patients. Neurology 1992;42:389–92.
[2] Carpio A, Escobar A, Hauser WA. Cysticercosis and epilepsy: a critical review. Epilepsia 1998;39:1025–40.
[3] Murthy JMK, Yangala R. Etiological spectrum of symptomatic localization related epilepsies: a study from South India. J Neurol Sci 1998;158:65–70.
[4] Murthy JMK, Reddy VS. Clinical characteristics seizure spread and prognosis of epilepsy associated with single small cerebral calcific lesion. Seizure 1998;7:153–7.
[5] Singh G, Sachdev MS, Tirath A, Gupta AK, Avasthi G. Focal cortical-subcortical calcifications (FCSCs) and epilepsy in the Indian subcontinent. Epilepsia 2000;41:718–26.
[6] Carpio A, Hauser AW. Prognosis for seizure recurrence in patients with newly diagnosed neurocysticercosis. Neurology 2002;59:1730–4.
[7] Rjashekhar V, Jeyaseelan L. Seizure outcome in patients with a solitary cerebral cysticercus granuloma. Neurology 2004;62:2236–40.
[8] Singh G, Rajshekhar V, Murthy JMK, Prabhakar S, Modi M, Khandelwal N, et al. A diagnostic and therapeutic scheme for a solitary cysticercus granuloma. Neurology 2010;75:2236–45.
[9] Rajshekhar V, Raghava MV, Prabhakaran V, Oommen A, Muliyil J. Active epilepsy as an index of burden of neurocysticercosis in Vellore district, India. Neurology 2006;67:2135–9.
[10] Goel D, Dhanai JS, Agarwal A, Mehlotra V, Saxena V. Neurocysticercosis and its impact on crude prevalence rate of epilepsy in an Indian community. Neurol India 2011;59:37–40.
[11] Medina MT, Duron RM, Martinez L, Osorio JR, Estrada AL, Zúniga C, et al. Prevalence, incidence, and etiology of epilepsies in rural Honduras: the Salama study. Epilepsia 2005;46:124–31.
[12] Del Brutto OH, Arroyo G, Del Brutto VJ, Zambrano M, Garcia HH. On the relationship between calcified neurocysticercosis and epilepsy in an endemic village: a large-scale, computed tomography-based population in study in rural Ecuador. Epilepsia 2017;58(68):1955–2017.
[13] Del Brutto OH, Rajshekhar V, White Jr AC, Tsang VC, Nash TE, Takayanagui OM, et al. Proposed diagnostic criteria for neurocysticercosis. Neurology 2001;57:177–83.
[14] Fisher RS, Acevedo C, Arzimanoglou A, Bogacz A, Cross JH, Elger CE, et al. ILAE official report: a practical clinical definition of epilepsy. Epilepsia 2014;55:475–82.
[15] Fisher RS, Cross JH, French JA, Higurashi N, Hirsch E, Jansen FE, et al. Operational classification of seizure types by the International League Against Epilepsy: position paper of the ILAE Commission for Classification and Terminology. Epilepsia 2017;58:522–30.
[16] Murthy JMK, Vijay S, Raju CR, Thomas J. Acute symptomatic seizures associated with neurocysticercosis: a community-based prevalence study and comprehensive rural epilepsy study in south India (CRESSI). Neurol Asia 2004;9(Suppl. 1):86.
[17] Nash TE, Pretell EJ, Lescano AG, Bustos JA, Gilman RH, Gonzalez AE, et al. Perilesional brain oedema and seizure activity in patients with calcified neurocysticercosis: a prospective cohort and nested case–control study. Lancet Neurol 2008;7:1099–105.
[18] Rathore C, Thomas B, Kesavadas C, Radhakrishnan K. Calcified neurocysticercosis lesions and hippocampal sclerosis: potential dual pathology? Epilepsia 2012;53:e60–2.
[19] Manford M, Hart YM, Sander JWAS, Shorvon SD. The national general practice study of epilepsy: the syndromic classification of the International League Against Epilepsy applied to epileptics in general population. Arch Neurol 1992;40:803–8.
[20] Manford M, Hart YM, Sander JWAS, Shorvon SD. National general practice study of epilepsy (NGPSE): partial seizures in a general population. Neurology 1992;42:1911–7.
[21] Russell WR, Whitty CWM. Studies in traumatic epilepsy 3–visual fits. J Neurol Neurosurg Psychiatry 1955;18:79–96.
[22] Williamson PD, Thadani VM, Darcey TM, Spencer DD, Spencer SS, Mattson RH. Occipital lobe epilepsy: clinical characteristics, seizure spread patterns, and results of surgery. Ann Neurol 1992;31:3–13.
[23] Remi J, Vollmar C, de Marinis A, Heinlin J, Peraud A, Noachtar S. Congruence and discrepancy of interictal and ictal EEG with MRI lesions in foal epilepsies. Neurology 2011;77:1383-30.
[24] Nash TE, Pretell J, Garcia HH. Calcified cysticerci provoke perilesional edema and seizures. Clin Infect Dis 2001;33:1649–53.
[25] Garg RK, Karak B, Mohan Kar A. Neuroimaging abnormalities in Indian patients with uncontrolled partial seizures. Seizure 1998;7:497–500.
[26] Nash TE, Bustos JA, Garcia HH. Disease centered around calcified taenia solium granuloma. Trends Parasitol 2017;33:65–73.
[27] Ndimubanzi PC, Carabin H, Budke CM, Nguyen H, Qian YJ, Rainwater E, et al. A systematic review of the frequency of neurocyticercosis with a focus on people with epilepsy. PLoS Negl Trop Dis 2010;4:e870.
[28] Bruno E, Bartoloni A, Zammarchi L, Strohmeyer M, Bartalesi F, Bustos JA, et al. Epilepsy and neurocysticercosis in Latin America: a systematic review and meta-analysis. PLoS Negl Trop Dis 2013;7(10):e2480. https://doi.org/10.1371/journal.pntd.0002480 [eCollection 2013].
[29] Reddy DS, Volkmer II R. Neurocysticercosis as an infectious acquired epilepsy worldwide. Seizure 2017;52:176–81.
[30] Leon A, Saito EK, Mehta B, McMurray. Calcified parenchymal central nervous system cysticercosis and clinical outcomes in epilepsy. Epilepsy Behav 2015;43:77–80.
[31] Velasco TR, Zanello PA, Dalmagro CL, Araújo Jr D, Santos AC, Bianchin MM, et al. Calcified cysticercotic lesions and intractable epilepsy: a cross sectional study of 512 patients. J Neurol Neurosurg Psychiatry 2006;77:485–8.
[32] Rathore C, Thomas B, Keshavadas C, Abraham M, Radhakrishnan K. Calcified neurocysticercosis lesions and antiepileptic drug-resistant epilepsy: a surgically remediable syndrome? Epilepsia 2013;54:1815–22 l.
[33] Esclaya AL, Burneo J. Epilepsy surgery and neurocysticercosis: assessing the role of the cysticercotic lesion in medically-refractory epilepsy. Epilepsy Behav 2017;76:178–81.
[34] Taveira M, Morita ME, Yasuda CL, Coan AC, Secolin R, Luiz Cunha da Costa A, et al. Neurocysticercotic calcifications and hippocampal sclerosis: a case-control study. PLoS ONE 2015;10(7):e0131180. https://doi.org/10.1371/journal.pone.0131180.
[35] Bianchi MM, Velasco TR, Wichert-Ana L, Alexandre Jr V, Araujo Jr D, dos Santos AC, et al. Characteristics of mesial temporal lobe epilepsy associated with hippocampal sclerosis plus neurocysticercosis. Epilepsy Res 2014;103:1889–95.
[36] Bianchin M, Velasco TR, Ana LW, Santos AC, Sakamoto. Understanding the association of neurocysticercosis and mesial temporal lobe epilepsy and its impact on the surgical treatment of patients with drug resistant epilepsy. Epilepsy Behav 2017;76:168–77.
[37] Pradhan S, Kathuria MK, Gupta RK. Perilesional gliosis and seizure outcome: a study based on magnetization transfer magnetic resonance imaging in patients with neurocysticercosis. Ann Neurol 2000;48:181–7.
[38] Gupta RK, Awasthi R, Rathore RK, Verma A, Sahoo P, Paliwal VK, et al. Understanding epileptogenesis in calcified neurocysticercosis with perfusion MRI. Neurology 2012;78:618–25.