Neurocysticercosis: A Clinical and Radiological Appraisal from Kerala State, South India

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ABSTRACT

Objectives: Little has been published on Neurocysticercosis (NCC) in the State of Kerala, South India. This study was conducted to describe the clinical and radiological features of NCC in Kerala and also to study the frequency of NCC as seen in a tertiary referral setting.

Materials and methods: We evaluated retrospectively 11 patients who were admitted with a diagnosis of NCC from 1986 to 1998. A pre - abstracted proforma containing detailed demographic data, dietary habits, clinical features and history of travel outside Kerala and neuroimaging findings were obtained from patient records and the data was tabulated and analysed. Follow up assessment was made three months after treatment.

Results: There were a total of 11 patients, including nine males and two females in the age range of 24 to 62 years and a mean age of 35.2 years. All were nonvegans, only 36% were pork eaters while 18% claimed consumption of salads and uncooked vegetables. 55% of patients were migrants. Migrants were defined as those who lived outside the state of Kerala for more than six months. Seizure was the most common presenting complaint and occurred in all patients (100%). Multiple ring enhancing lesions were seen on computerised tomography (CT) and magnetic resonance imaging (MRI) scans in 60%. Calcified lesions were noted in two patients. An isolated instance of miliary or disseminated cysticercosis with subcutaneous nodules and multiple brain lesions in MRI scan was observed. All patients received anticysticercal therapy besides anticonvulsants.

Conclusions: It appears that NCC is rather uncommon in Kerala. Better socioeconomic status, high literacy rate, improved sanitation and health care in the state of Kerala could be reasons for this observation. A prospective case-control study of NCC in Kerala is needed to study these factors.

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INTRODUCTION

The disease Taeniasis or pork tapeworm infection results from infestation of the small intestine by *Taenia solium*, the adult tapeworm. Humans are both the definitive and intermediate hosts for *Taenia solium*. Cysticercosis is a disease caused by the presence of *Cysticercus cellulosae* and *Cysticercus racemose*, the larval forms of *Taenia solium* in tissues. Humans can acquire the disease with either or sometimes with both. Cysticerci have a predilection for migrating to the central nervous system(CNS), eyes and striated muscle. The high glycogen or glucose content of these tissues may be responsible for such a tropism exhibited by cysticerci. When the CNS or eye is involved by cysticercosis, the patient has Neurocysticercosis (NCC). NCC is the most common parasitic disease of the human nervous system. Its prevalence varies greatly according to the geographical region and is not yet precisely known[1]. The increased ease of international travel, increasing number of migrants from developing countries and improved diagnostic techniques have led to widespread recognition of NCC as a common infection not only in developing countries, but also in the United States and other areas with large immigrant populations from Latin America[2,3]. The geographic distribution of cysticercosis is wide, with high prevalence reported from Mexico, Central and South America, India and Sub-Saharan Africa[4].

An excellent account of the pathology, pathogenesis and clinical features of cysticercosis had been described occurring in the British troops stationed in India[5,6]. In the pre-computerised tomography (CT) scan era, NCC as a cause of epilepsy in India was reported to vary from 2.2 to 9.6%[7,8]. After the advent of CT and magnetic resonance imaging (MRI), NCC has been identified as the cause in 9 to 18.6% of patients with epilepsy[9]. In a study from Delhi, as high as 24% of 361 epileptic

Fig. 1 Map of Kerala State with the districts to which the patients belonged (black diamonds).

patients had unequivocal NCC on MRI[10]. In India, the commonest form of NCC is the solitary parenchymal cyst. It is seen as a single, small (< 2 cm) enhancing lesion on the CT scans. This lesion commonly presents as simple partial or secondarily generalised seizures[11]. Brain biopsy specimens from patients with such lesions in India showed that nearly all of these resolving lesions were due to NCC[12]. In South India, single CT enhancing lesions (SCTEL) and NCC together accounted for 67% of provoking factors for acute symptomatic seizures[13]. Of the 215 patients initially diagnosed to have SCTEL (< 2cm) , 98.9% had solitary cerebral cysticercal granuloma (SCCG) on follow-up evaluation[14].

The state of Kerala is located in the South-West coast of Indian peninsula, at latitude, between 8º 18' - 12º 48' N and 74º 52' - 77º 22' E. It is a narrow strip of land extending from Kasarkod in the north to Trivandrum in the south and from the Western Ghats in the east to the Arabian Sea in the west . It covers a geographic area of 38863 km^2, (Fig. 1). Kerala has a population of 29 million and a growth rate of 14%. It made rapid strides of achievement in the field of education and health in the last decade[15]. Neurocysticercosis has been uncommon in Kerala and only anecdotal reports occur. This is possibly due to the impact of high literacy rate on health awareness and personal hygiene of the people. This study was carried out to (a) describe the clinical and radiological features of NCC and (b) its frequency in a tertiary referral center.

MATERIALS AND METHODS

Sree Chitra Tirunal Institute for Medical Sciences and Technology (SCTIMST) is a tertiary referral center situated in Trivandrum, the capital of Kerala state. This Institute offers super-specialty services in neurological and cardiovascular diseases, and is designated as an Institute of National Importance under the Government of India.

We evaluated retrospectively a cohort of patients who were admitted to SCTIMST with a diagnosis of NCC during the years 1986 - 1998. A pre-abstracted proforma was designed and details about demographic data, dietary habits and travel outside Kerala were collected from patient records. The assessment of socioeconomic status (SES) was based on total monthly household income in Indian Rupees (INR). As per SCTIMST guidelines, the patients were divided into two major groups based on whether their monthly household income was less than (category A) or greater than (category B) the median income of all patients in our study. Literacy criteria were based on the ability to read, write and understand in any of the languages spoken in India[16]. A description of the presenting symptoms and neurological signs were documented after a review of the charts (by A.K.). All patients underwent either CT scan or MRI of the brain at the time of admission and follow up. A diagnosis of SCTEL was based on clinical and radiological criteria[14]. The clinical criteria were: (a) seizure should be the presenting complaint, (b) there should be no evidence for persistent raised intracranial pressure, progressive neurological deficit or an active systemic disease. The CT diagnostic criteria were: (a) evidence for a solitary contrast enhancing lesion measuring 2 cm or less in its maximal dimension, (b) edema may or may not be present; but if present, it should not be severe enough to produce a midline shift. The diagnosis of Neurocysticercosis was based on clinical, radiological, immunological and epidemiologic data of patients[17]. All patients had to fulfill at least one of the absolute criteria with additional major, minor and epidemiologic criteria. Clinical and radiological responses were assessed over a period with a mean duration of 2.7 months (range: 1 - 30 months).

RESULTS

There were 11 patients observed in the 12 years under study. They comprised nine males and two females in the age range of 24 to 62 years (mean 35.2 years). The median monthly household

Table I. Symptoms & Signs of NCC.

Symptoms & Signs	n = (%)
1. Seizure	11 (100%)
a. Simple Partial	-
b. Complex Partial	1 (9%)
c. Simple Partial with secondary generalisation	5 (46%)
d. Complex Partial with secondary generalisation	1 (9%)
e. Generalised seizure	4 (36%)
2. Headache/Vomiting	7 (64%)
3. Papilledema	3 (27%)
4. Hemiparesis	1 (9%)
5. Cerebellar Ataxia	1 (9%)
6. Subcutaneous nodules	1 (9%)

Table II. Imaging Findings.

	Single CT enhancing lesions (CTEL)	Multiple ring enhancing lesions	Calcified lesions
1. CT Scan (n=10)	4 (40%)	6 (60%)	2 (20%)
2. MRI (n = 8)	3 (40%)	5(60%)	--

income of all patients included in this study was INR 1500. Eight patients (73%) belonged to lower SES (category A) , while three (27%) belonged to the higher (category B). All patients except one (82%) were literate. All patients were nonvegans by dietetic habits and 36% reported intake of pork, while salad and uncooked vegetables were consumed by 18%. Five patients (45%) had no history of travel outside Kerala, while 6(55%) were migrants. Among the native Keralites, two out of five patients were pork eaters. The most common initial clinical presentation was seizure, which occurred in all patients (Table I). Complex partial and simple partial seizures with secondary generalisation occurred in 1(9%)and 5(46%) patient/s respectively. Generalised seizure was the presenting symptom in 4(36%) patients. Headache and vomiting occurred in seven patients, of these bilateral papilledema was seen in 3(27%). Hemiparesis, cerebellar ataxia and subcutaneous nodule were found in one patient each.

CT scan was done in 10 and MRI brain in eight patients (Table II). Multiple ring enhancing lesions constituted the major abnormality in CT and MRI scans (60%). SCTEL formed 40% of the findings in CT scan. Calcified lesions were observed in two patients. The single contrast-enhancing lesions were located on the corticomedullary (gray-white) junction in majority (80%) of cases , while the remainder had a lesion on the deep nucleus in addition to a calcified lesion on the cerebral cortex. Multiple ring enhancing lesions were predominantly noted again on the corticomedullary junctions (70%). In five patients

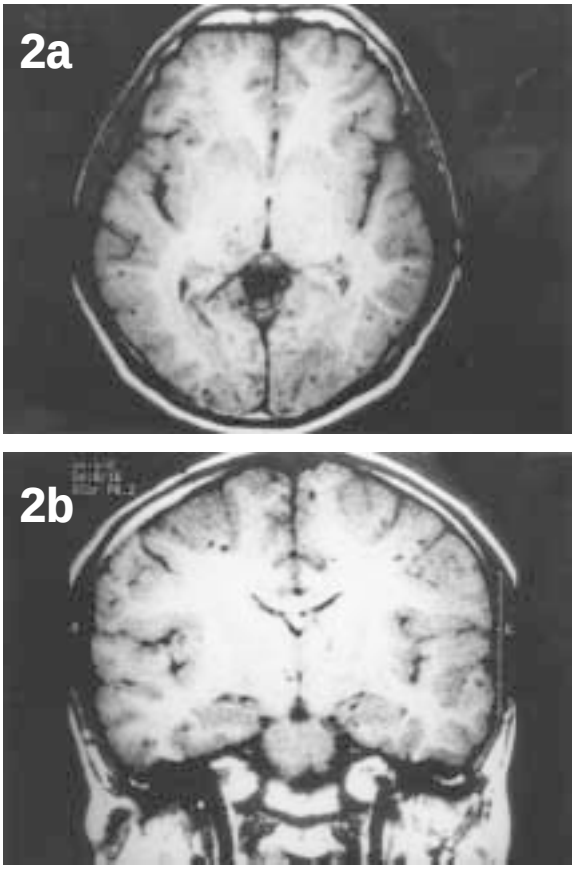

Fig. 2a, b T_1 weighted MR Images (SE,TR 800/30/1.5 nex/256x192) in axial and coronal plane reveal nodular hypointense lesions of varying sizes predominantly distributed in the gray-white junctions of cerebral, cerebellar hemispheres and brain stem.

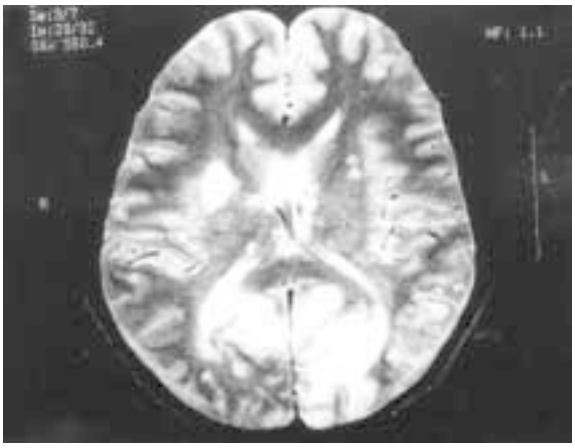

Fig. 3 T_2 weighted MR image (SE TR 2200/90/1 nex/ 256x192) in axial plane reveals hyperintense lesions with profound perilesional oedema.

with multiple lesions in MRI, different stages of cysticerci were observed. Vasogenic edema was noted perilesionally in many cases (70%). However, mass effect or midline shift was not a feature in any. One patient with miliary or disseminated cysticercosis showed multiple small lesions of varying sizes predominantly distributed in the gray-white junctions of cerebral, cerebellar hemispheres, and brain stem (Fig. 2-4). Biopsy of the

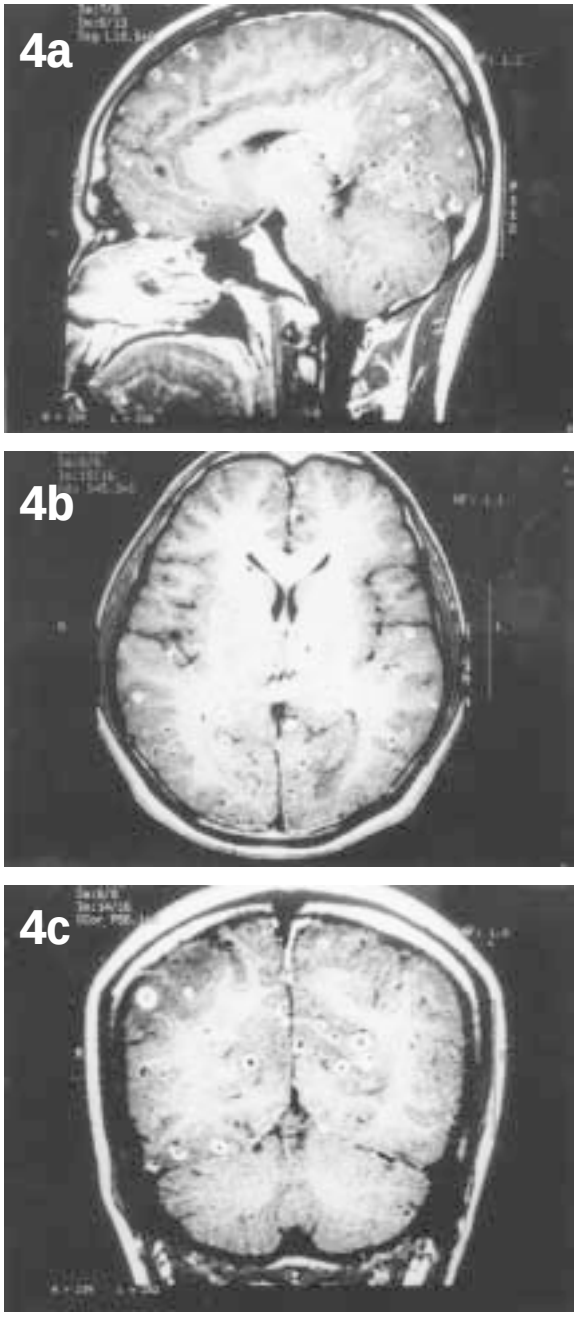

Fig. 4a, b, c T_1 weighted, post contrast sagittal, axial and coronal MR images reveal nodular hyperintense ring enhancing lesions around the hypointense areas.

subcutaneous nodule from this patient revealed typical cysticercus granuloma (Fig. 5).

CSF examination was done in four patients and showed elevated proteins with normal glucose without pleocytosis. Anticysticercal antibody could be done in serum and cerebrospinal fluid (CSF) in two patients and both were positive. EEG was abnormal in 3 (27%), showing mild to moderate focal slowing in two and mild generalised slowing in another patient.

Seven patients (64%) were treated with Albendazole in a dose of 15 mgm/kg for four weeks, while 2 (18%) received Praziquantel in a dose of

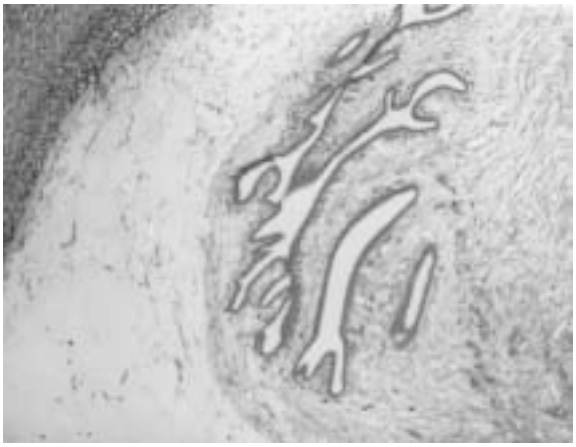

Fig. 5 Photomicrograph showing characteristic cysticercus cellulosae, bordered by dense fibrous capsule (Hematoxylin & Eosin x 150).

50 mgm/kg for 14 days, 2 (18%) patients were given a combination of Albendazole and Praziquantel. All patients (100%) received anticonvulsants besides anticysticercal therapy.

Follow up ranged between 1 month to 30 months with a mean of 2.7 months. 82% of patients showed remarkable improvement in symptoms with resolution of lesions on repeat brain imaging done three months after the completion of cysticidal treatment. One patient had aggravation of intracranial pressure following Albendazole therapy which was promptly controlled with parenteral steroids. Two patients (18%) had recurrence of symptoms with non-resolution of intracranial lesions on neuroimaging. All patients showed good seizure control at last follow up.

DISCUSSION

Cysticercosis occurs worldwide. An estimated 2.5 million people harbour the porcine tapeworm, *taenia solium*, and many more are infected with the larval form, ie. Cysticercosis[18]. In endemic areas, as many as 4% of the population is known to be affected with NCC[19,20]. Cysticercosis is common in Africa, India, China, Eastern Europe and Indonesia, in addition to Mexico, Central and South America. The human disease of cysticercosis results when man becomes the inter-mediate host for the larval stage, known as *cysticercus cellulosae* or *cysticercus racemose.* The infection occurs from contamination of food (usually vegetables) with viable eggs from human excreta, from fecal-oral contact or from auto-infection due to reverse peristalsis of proglottids in to the stomach. The embryo most commonly mature in the brain, skeletal muscle and eye[2]. Once situated in the tissue, cysticerci evolve through an important natural history of four stages. Stage - 1 is mild or inapparent in most patients. As the parasites locate in the CNS, there may be diffuse edema, especially if the number is high.

Stage - 2 develops over several months with a thin-walled cyst containing fluid and a live larva. Stage - 3 is an inflammatory reaction which surrounds and damages the cyst to fill with caseous material while the larva degenerates and dies. This is the stage when the cyst contents and antigens are released, and thus may be easier to detect in serum and cerebrospinal fluid (CSF). Stage - 4 or the final stage is an inactive one when the cyst degenerates itself becoming nonviable and calcified[1]. Thus the manifold and diverse clinical presentation of NCC is determined by (i) location of cysts (ii) size of cysts (iii) cyst load (number of cysts) (iv) and the host-cyst inflammatory reaction[2]. Serial CT studies have revealed that after the inflammatory reaction begins, cysts often resolve without treatment[21].

Regional differences in the clinical manifestations of NCC have been reported. Economic, cultural and religious factors strongly affect the prevalence of cysticercosis in any particular geographic area[1]. While arachnoiditis (48.2%) and hydrocephalus due to meningeal inflammation (25.7%) are the common forms of NCC in Mexico[20], parenchymatous form of NCC is more common in India[22] and this has been our experience also. However, several caveats do bear mention during this discussion. Physical findings are inconstant in NCC. The parenchymatous cysts can be solitary or multiple, and focal or generalised seizures are the most common (up to 92%) symptom[23]. Two types of cysts can develop in the brain. The less virulent form, *Cysticercus cellulosae*, is a small (< 2 cm), thin-walled round cyst that lodges in the parenchyma or subarachnoid space and provokes only a minor inflammation. Consequently, these cysts often remain silent. On the other hand, the larger, more virulent and intense form, *Cysticercus racemose*, grows actively to form grape-like clusters of cysts in the basal cisterns and ventricles. *Cysticercus cellulosae* has a predilection for the dorsolateral subarachnoid space, while the *Cysticercus racemose* has a predilection for the basal subarachnoid cisterns[24]. Cysticercotic arachnoiditis, also called subarachnoid neurocysticercosis, usually presents with signs of meningitis. This variant is caused by racemose form of cysticercosis, which also can cause the obstruction of fourth ventricle. In addition to seizures, this form may cause an associated vasculitis, including occlusion of the internal carotid artery[3]. Intraventricular cysticercosis is frequently found in conjunction with subarachnoid NCC. Because of its location, the racemose form causes an intense inflammatory reaction leading to communicating hydrocephalus. If these intraventricular cysts are strategically located, they can cause unusual complications like isolated fourth ventricle. This type of obstructive hydrocephalus results when both the cerebral aqueduct and the foramina of Luschka and Magendie are suddenly blocked[1]. Free-floating and pedunculated cysts projecting into the ventricles can present with Brun's syndrome: a condition due to positional, intermittent, obstruction of foramen leading to acute episodic headaches, ataxia, vertigo, and at times drop attacks[23]. Perhaps, these distinctively different manifestations of *Cysticercus cellulosae* and *Cysticercus racemose* could also explain the regional differences between NCC in India and Central America. In addition, we suspect that such geographical variations may be related to the differences in host-cyst immune response. Sex-related differences in the extent of brain inflammation have been described[25]. This may possibly be caused by the differences in attachment of human leukocyte antigens on the parasitic surface[26].

In a study comprising 406 cases of NCC described by Singhal and Ladiwala[22], 360 had a solitary cyst, 45 had multiple lesions and only one had disseminated disease. In contrast to the above, although the number of cases of NCC in our study is too small to make any valid comparison, there were more cases (60%) with multiple cysts of NCC, which could probably be related to referral bias. Five of our patients with multiple lesions in MRI showed different stages of cysticerci. This reflects the natural history of the tissue infection. In addition, since some patients ingest ova repeatedly over a long duration, several tissue cysts may be in different stages at the same time , thereby resulting in a mixed picture[1]. Disseminated form of cysticercosis, where brain and muscles are studded with innumerable cysts, has been described rarely from India[27]. The single case of miliary or disseminated cysticercosis seen in the present study is similar to the three cases reported by Wadia et al[27].

In the present study, seizure was the major presenting symptom (100%), similar to other studies[14,20,22]. Since seizures are caused by abnormal neuronal discharges, usually arising from the cerebral cortex, the NCC lesions situated on the cortex or on the gray-white junction will be more epileptogenic when compared to deep lesions. Hence, the parenchymal cysticerci closer to cortical neurons tend to present with seizure as the most common manifestation. Of the 72 patients with NCC who had seizures, Singhal et al[28] noted only three with complex partial seizures (CPS). This is similar to our study in which only one (9%) had CPS out of 11 patients, thus supporting the observation of the former[28] that CPS is infrequent in NCC. Similar to earlier report[28], electroencephalograms (EEGs) in majority of our patients (73%) were normal.

The remaining 27% of patients showed focal or generalised slowing in EEG's.

Although seizure was the initial presenting symptom in all of our patients, as in other series[2], there was no definite correlation between EEG changes, clinical signs and symptoms. In a study comprising interictal EEG of 50 epileptic patients with parenchymal neurocysticercosis, Chayasirisobhon et al[29] found abnormal EEG in 14 patients(28%): three had focal spike or sharp waves, eight had focal slowing and three had both. These authors concluded that the EEG abnormality does not depend on the number of lesions, but rather on location, host response and viability of cysts. The number of abnormal EEGs in the above study (28%) is comparable to ours (27%), although we had a smaller cohort. EEG reflects cortical neuronal excitability, and hence, hemispheric lesions affect EEGs more consistently and posterior fossa or basal lesions produce EEG abnormalities less frequently. The electrical abnormality overlying a small cortical lesion is not often detected on scalp recording because of wider electrode placement and the interposed skull and scalp. NCC lesions being small (< 2 cm), defining their relationship to the surface electrodes placed according to the International 10-20 sytem is very difficult. It is very conceivable that many of the scalp electrodes did not overlie the lesions, thereby making localisation of epileptogenic foci difficult. With deep brain tumors, the EEG may be normal or only mildly abnormal even by the time the patient comes to craniotomy. In addition, a normal EEG does not preclude an infratentorial tumor[30]. A minority of our patients had lesions involving the posterior fossa (infratentorial) structures, and these lesions would have been largely inaccessible to EEG recording.

In an elaborate study comprising 127 cases of one or more supratentorial tumor, Gastaut et al[31], after performing EEG and CT scan on the same day or within 2 - 3 days, concluded that peritumoral edema is only rarely (9.5% of cases) responsible for the EEG abnormalities. Similarly, although vasogenic edema was noted in 70% of our patients, the EEGs were normal in the majority (73%). As opposed to increased intraventricular pressure, intracranial pressure per se does not alter the EEG. This has been eminently supported by the findings of normal EEGs in patients with benign intracranial hypertension (BIH)[32].

Although ours is a tertiary referral center, we could recruit only 11 cases. It is possible that quite a few patients who had mild manifestations were not referred to our center and only severe and complicated cases were referred. On the other hand, the reason for such a low number could be that NCC is indolent in many patients. Tapeworms usually survive for 3-7 years and some may live as long as 25 years[1]. Lack of clinical manifestations in NCC, and the fact that many patients could remain asymptomatic for a long term, even with severe infections[20], makes the detection of NCC a challenge. This can create added confusion when clinical criteria (for example: seizure should be the presenting complaint) alone is used for entry in to a prospective epidemiological study. Alternatively, we believe that NCC is indeed uncommon in this part of the country. Kerala State in this respect is acknowledged for its high literacy rate (> 90%), low infant mortality rate (IMR), improved health status and efficiency of health delivery system as compared to other states in India[16]. This has been made possible by the dual efforts of government and people participation in implementing public policies. We firmly believe that provision of sanitary water supplies, absence of pig dwelling, improved personal hygiene, washing and cooking of vegetables and meat under hygienic conditions have all contributed to this low occurrence of NCC in Kerala. Nevertheless, we suggest that a case-control population study is urgently needed to address these issues in a more comprehensive manner.

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