ROLE OF CT IN THE STUDY OF PREVALENCE OF NEUROCYSTICERCOSIS IN A RURAL AND SUBURBAN TEACHING HOSPITAL

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ABSTRACT

AIMS AND OBJECTIVES

The main aim of the study was to identify the prevalence, lobar distribution of neurocysticercosis in a suburban and rural centre of southern Karnataka. Its distribution in male and female population, distribution among various age groups, distribution in lobes, describe its stages, and presence of perilesional oedema.

MATERIAL AND METHODS

The study was conducted in the Department of Radiodiagnosis, MVJ Medical College and Research Hospital, Bangalore, for a period of 6 yrs. from January 2009 to December 2015. Data collection was prospective. A computer-assisted search of all the reports of CT brain with the diagnosis of NCC was conducted within the departmental database. A total of 198 patients with NCC were identified and the study was conducted.

RESULTS

Total 4582 CT studies of brain were reviewed. 198 cases of neurocysticercosis (4.3%) were detected. 115 were males and 83 were females. The age range was 8 to 70 years with a mean age of 30 years. The number of patients under age 20 years (n=46) was more compared to the adult patients. Maximum numbers of patients were seen in 41 to 50 years age group followed by 51 to 60 years.

Stage 2 of the disease was noted to be predominant in our study with 61 cases (30%) followed by stage 4. However, individual cases showing multiple stages of NCC were observed in 56 cases. Parietal lobe was more commonly involved followed by temporal, frontal, and occipital lobe. Multiple stages of the NCC in individual cases were seen in 56 cases. The presence of an eccentric enhancing scolex was noted in 57 cases.

CONCLUSION

The prevalence of neurocysticercosis in our study over a period of six years is 4.3% and it is more commonly observed in the paediatric age group (23.2%) and in male sex (58%).

KEYWORDS

Neurocysticercosis, Seizures, Paediatric, Computed tomography.

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INTRODUCTION: Neurocysticercosis is the most common parasitic disease of the nervous system and is the main cause of acquired epilepsy in developing countries. It has also been a problem in industrialised countries because of the immigration of tapeworm carriers from areas of endemic disease. This form of cysticercosis is a relevant cause of seizures in endemic areas. Cysticerci may be located in brain parenchyma, subarachnoid space, ventricular system, or spinal cord causing pathological changes that are responsible for the pleomorphism of neurocysticercosis.

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AIMS AND OBJECTIVES: The main aim of the study was to identify the prevalence, lobar distribution of neurocysticercosis in a suburban and rural centre of southern Karnataka. Its distribution in male and female population, distribution among various age groups, distribution in lobes, describe its stages, and presence of perilesional oedema.

MATERIAL AND METHODS: The study was conducted in the Department of Radiodiagnosis, MVJ Medical College and Research Hospital, Bangalore for a period of 6 yrs. from January 2009 to December 2015. Data collection was prospective. A computer-assisted search of all the reports of CT brain with the diagnosis of NCC was conducted within the departmental database. Patients with incidental findings of NCC were also included in the study. A total of 198 patients with NCC were identified. Approval was obtained by

institutional review board. Patient informed consent was waived.

Only those patients with diagnosis of NCC on CT were included. A total of 198 patients were included in the study each of whom had NCC in different stages and calcified granulomas. In all the cases, the indication for CT scan was to rule out intracranial lesions for headache, seizure. Patients with repeat CT head for followup, motion artifacts, and postop cases were excluded. The study design did not involve patient contact, hence approval by the local ethics committee was not required.

All CT examinations were performed with single slice CT scanner (Toshiba Asteion, Japan) and 16-slice MDCT scanner (GE Brivo 385, Milwaukee, USA). The imaging parameters for the scout film and the scan are as follows in the normal as well as helical mode. In non-helical mode, the parameters are: Scout kVp 120, mA 10 ww/wl: 500/50. Scan kVp 120, mA 120, TET 12S, Interval 10 mm, thickness: 2.5 mm, Rotation Time 2s, FOV: ISO: 1.3 s. ww/wl: 50/150. In helical mode, the parameters are: Scout kVp 120, mA 10 ww/wl: 500/50. Scan kVp 120, mA 120, TET 10.3S, interval 5 mm, thickness: 2.5 mm, rotation time 1s, FOV, pitch, and speed 1.375:1/13.75, ISO: 1.3 s. ww/wl:50/150. Images were acquired and analysed on the GE advantage workstation. The images were archived using magnetic optic discs and external hard drives.

CT examination results were interpreted by four experienced radiologists each with more than five years of experience in interpreting brain CT examinations. Each scan was evaluated for location, number, perilesional oedema, calcification, contrast enhancement, and other associated lesions. All scans were interpreted in brain parenchymal window settings. The number, locations of the NCC were assessed.

Statistical analysis was done using SPSS software 17.0. Quantitative variables were expressed as percentages.

RESULTS: Among the 4582 CT studies of brain reviewed, the study yielded a total of 198 cases of neurocysticercosis (4.3%). 115 were males and 83 were females. The age range was 8 to 70 years with a mean age of 30 years. The number of patients under age 20 years (n=46) was more compared to the adult patients. Maximum numbers of patients were seen in 41 to 50 year age group followed by 51 to 60 years (Table 1 and 2).

Stage 2 of the disease was noted to be predominant in our study with 61 cases followed by stage 4 (Table 3). However, individual cases showing multiple stages of NCC were observed in 56 cases. Parietal lobe was more commonly involved followed by temporal, occipital, frontal and occipital lobe. Table 4. Multiple stages of the NCC in individual cases were seen in 56 cases. (Fig. 1, 2a-d, 3a-d). The presence of an eccentric enhancing scolex was noted in 57 cases.

Bilateral symmetrical extensive involvement of cerebral hemispheres, basal ganglia, thalami, cerebellar hemispheres, facial muscles, temporalis, occipital muscles, and tongue involvement was noted in two cases. (Fig. 4).

Racemose type of cysticercosis involving basal cisterns and spinal subarachnoid spaces noted in one case.

Age Group	No. of Patients	Percentage
1-10	19	9.6
11-20	27	13.6
21-30	32	16.1
31-40	21	10.6
41-50	36	18.2
51-60	34	17.2
61-70	31	15.6

Table 1: Age Distribution of Patients

Sex	No. of Patients
Males	117
Female	83

Table 2: Sex Distribution of Patients

Stage of NCC	No. of cases	Percentage
Stage I	12	6
Stage II	61	30.1
Stage III	23	11.6
Stage IV	48	24.2
Multistage	56	28.2

Table 3: Stages of NCC

Lobar Involvement	No. of cases
Frontal	16
Parietal	45
Temporal	20
Occipital	15
Multiple lobes	104

Table 4: Lobar Distribution of the Lesions

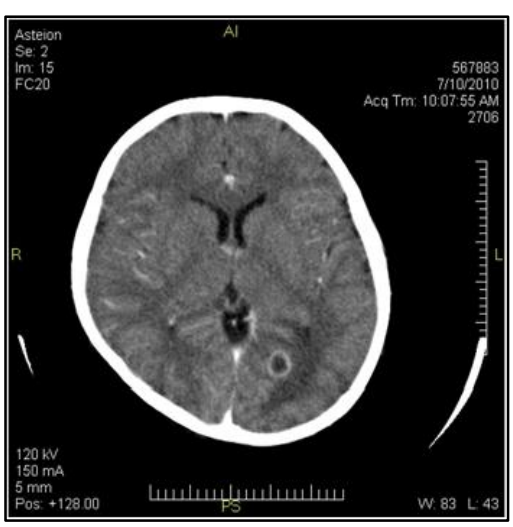

Fig. 1: Axial CECT in a 10-Year-Old Boy Showing Stage II NCC in Left Occipital Lobe

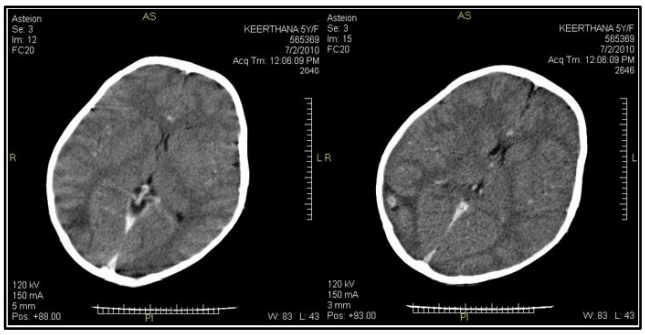

Fig. 2a and 2b: NCCT and CECT brain in a 5-year-old Girl Showing Ring Enhancing Lesion in Right Parietotemporal Lobe with Perilesional Oedema

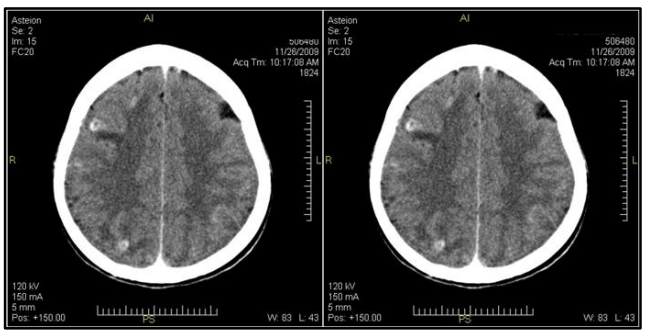

Fig. 3a and 3b

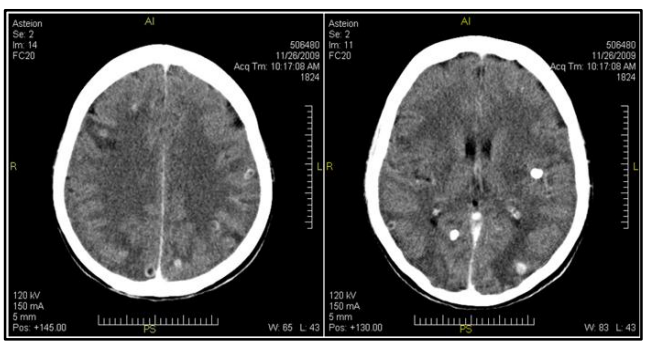

Fig. 3c and 3d: Axial NCCT and CECT Brain in a 45-year-old Patient Showing Different Stages of NCC

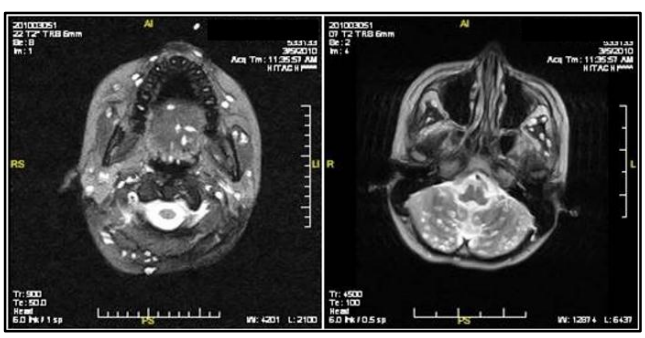

Fig. 4a and 4b

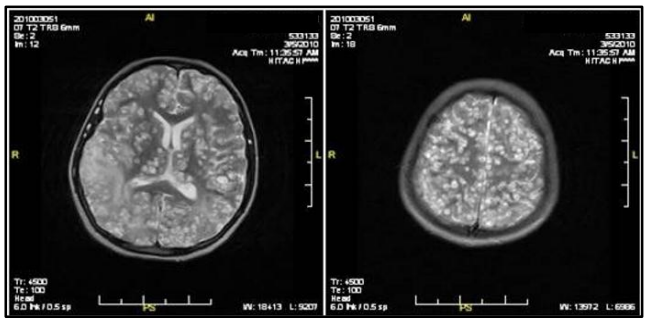

Fig. 4c and 4d: Axial MRI T2W Sequences in a 16-year-old Girl Showing Bilateral Symmetrical Extensive NCC in Cerebral Hemispheres, Basal Ganglia, Thalami, Cerebellum, Facial Muscles, Occipital Muscles, and Tongue. Patient's CT Showed Diffuse Cerebral Oedema with Multiple Calcifications

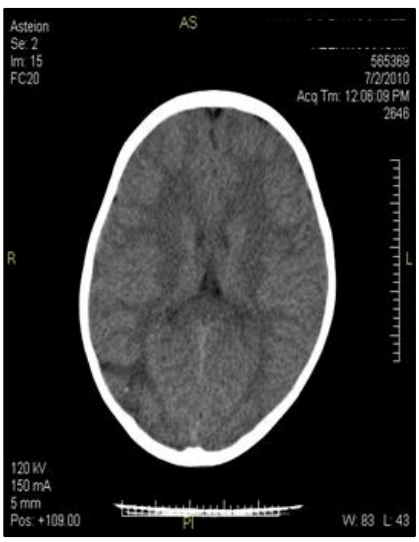

Fig. 5: NCCT Brain Axial Section Showing Significant Reduction in Perilesional Oedema in a Stage III NCC Patient After Appropriate Treatment

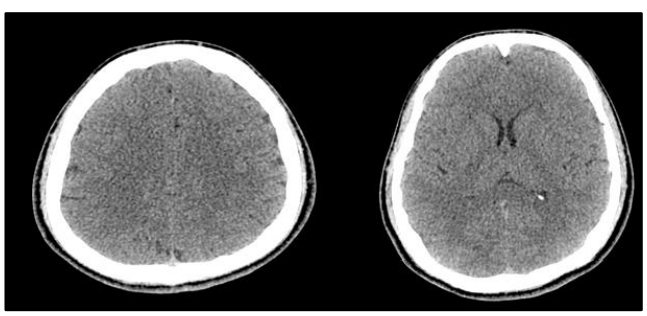

Fig. 6a and 6b: NCCT Brain Axial Sections of Another Patient with Stage II and III of NCC Showing complete Resolution After Treatment

DISCUSSION: Globally, neurocysticercosis is endemic in Central and South America, sub-Saharan Africa, and in some regions of the Far East including the Indian Subcontinent, Indonesia, and China, reaching an incidence of 3.6% in some regions. In a community survey of 50,617 individuals from South India, the prevalence of active epilepsy was 3.83 per 1000 and NCC was detected in 28.4% of them by CT[(1)] single cyst infection (range 47.7-53.4%) is the most common in the Indian subcontinent.[(2,3)] The relationship between NCC and epilepsy has been explored for long time and debates are still going on about their relationship.

Because of the high prevalence of epilepsy and NCC, there exists a cause and incidental relationship between the two.[4] The prevalence of active epilepsy related to NCC varied from 1.3 to 4.5 per 1,000 population in Indian studies.[5,6,7,8] In a farming community, prevalence of 6.6% was reported for epilepsy and 18.6% for Taenia solium infection.[9] In our study, the prevalence is 4.3%.

At present, neurocysticercosis represents the most common parasitic disease of the human central nervous system, is the most common cause of acquired epilepsy, and is a major public health problem worldwide.[10,11,12]

The disease is also a health problem in urban centres of developing countries where neurocysticercosis is a major cause of admissions to neurological hospitals.[13] However, neurocysticercosis has also been recognised in persons with no history of travel to endemic areas most of who get infected through a household contact harbouring the adult Taenia solium in the intestine.[14] Although, neurocysticercosis appears to affect men and women equally. There is some evidence to suggest that inflammation around the parasites maybe more severe in women than in men.[15] In our study, the prevalence is more in the males.

The ingestion of eggs permits development of larva in the soft tissues of the intermediate host. The subsequent ingestion of larva-infested tissues permits formation of the adult in the intestinal tract of the definitive host where additional eggs are produced and discharged, thereby perpetuating the cycle. Infective embryos reach the systemic circulation after actively crossing the intestinal mucosa. Some cysts are cleared by the liver.[16] Cysts lodge in capillaries mostly in muscle and brain tissue where they develop into immature cysts and later into larval cysts taking up to 3 months to reach this stage.[17] Cysts are protected from the host's immune response by the blood-brain barrier; thus, as mentioned earlier, no inflammatory response is noted as long as the cyst wall (rich in glycoproteins) remains intact.[18] When the parasite dies by natural processes or as a result of therapy, an inflammatory response with perilesional oedema ensues followed by calcification.[10,19]

After entering the central nervous system, cysticerci are in a vesicular viable stage in which the parasites have a transparent membrane, a clear vesicular fluid, and a normal invaginated scolex. The first stage of involution of cysticerci is the colloidal stage in which the vesicular fluid becomes turbid and the scolex shows signs of hyaline degeneration. Thereafter, the wall of the cyst thickens and the scolex is transformed into mineralised granules; this stage, in which the cysticercus is no longer viable, is called the granular stage. Vesicular cysticerci elicit little inflammatory reaction in the surrounding tissue. In contrast, colloidal cysticerci are often surrounded by a collagen capsule and by a mononuclear inflammatory reaction that includes the parasite itself. When parasites enter into the granular and calcified stages, the oedema subsides, but the astrocytic changes in the vicinity of the lesions may become more intense and epithelioid cells appear and coalesce to form multinucleated giant cells.[19]

The clinical presentation of patients with neurocysticercosis is highly nonspecific and depends on the number, location, size, and stage of the parasites, as well as the degree of the host's inflammatory response.[20,21] Thus, almost any neurologic symptom has been reported and a highly pleomorphic clinical presentation may occur with epilepsy[22,23] and headache being the most common symptoms followed by those caused by CSF obstruction.[20] In contrast, signs of meningeal irritation are uncommon.[24] Symptoms may be delayed for several years or may remain subclinical, but some patients can present with life-threatening conditions.[21] Most symptomatic patients are 15-40 years old and the disease has no gender or race predilection.[10] The most common clinical presentations are as follows. Seizures as a result of perilesional inflammation in degenerating cysts, although infarction and vasculitis may also act as predisposing factors and even calcified granulomas have been implicated in this clinical presentation.[25] Approximately, 50%-70% of patients experience recurrent seizures.[20,21,26]

Focal neurological signs have been described in up to 20% patients with neurocysticercosis. Pyramidal tract signs predominate, but sensory deficits, language disturbances, involuntary movements, parkinsonian rigidity, and signs of brainstem dysfunction may occur in some patients.[27] Some patients with neurocysticercosis develop intracranial hypertension associated or not with seizures or focal neurological signs. The most common cause of this syndrome is hydrocephalus, which maybe either related to cysticercotic arachnoiditis, granular ependymitis, or ventricular cysts.[27]

Intracranial hypertension and encephalitis is secondary to flow obstruction by intraventricular cysts, arachnoiditis, or ependymitis secondary to massive inflammatory response around a heavy load of parasites[19,28,29] and mass effect in cases of very large cysts. This is common in young females, children, and after anthelminthic therapy.[19] Patients with neurocysticercosis of the fourth ventricle may develop so-called Bruns syndrome, which is characterised by headache, papilledema, and even unconsciousness with rapid recovery triggered by rotatory movements of the head.[30]

Cerebrovascular complications are the result of multiple mechanisms. The spectrum of vascular complications includes lacunars infarction or large vessel disease, progressive midbrain syndrome, transient ischaemic attacks, and brain haemorrhage.[30,31] Lacunar infarction is the most common cerebrovascular manifestation of neurocysticercosis.[32]

Some patients with neurocysticercosis may present psychiatric manifestations ranging from poor performance to a severe dementia.[25] Patients with cysticerci located in the sellar region present with ophthalmologic and endocrinologic disturbances.[33] Spinal arachnoiditis usually present with motor and sensory deficits that vary according to the level of the lesion.[34]

Neurocysticercosis is commonly diagnosed with the routine use of diagnostic methods such as Computed Tomography (CT) and Magnetic Resonance Imaging (MRI)

of the brain. Peripheral leucocytosis, eosinophilia, and elevated erythrocyte sedimentation rate may be found on routine blood work. Reports have shown a decrease in N-acetylaspartate (NAA) and creatine levels and elevated lactate and metabolites such as alanine and succinate on magnetic resonance spectroscopy. Analysis of the Cerebrospinal Fluid (CSF) is indicated in every patient presenting with new-onset seizures or neurologic deficit in which neuroimaging shows a solitary lesion, but does not offer a definitive diagnosis. The results are usually abnormal (50-80%) when parasites are present in the basal cisterns or in the ventricles. CSF findings include mononuclear pleocytosis, normal or low glucose levels, elevated protein levels, high Immunoglobulin G (IgG) index, and in some cases, the presence of oligoclonal bands.

Tapeworm carriers maybe identified by examining the stool of the relatives of a patient with cysticercosis encephalitis. Enzyme-Linked Immunosorbent Assay (ELISA) is the most widely used test of Cerebrospinal Fluid (CSF). It has a sensitivity of 50% and a specificity of 65% for neurocysticercosis. In patients with more than two lesions, 90% sensitivity has been reported. Enzyme-Linked Immunoelectrotransfer Blot (EITB) assay in serum using lentil lectin glycoprotein antigens of T. solium cysts is also highly sensitive and specific initially described as 98% and 100% respectively for detection of antibodies in serum and cerebrospinal fluid.

CT has a high sensitivity and specificity in most forms of neurocysticercosis and is superior to MR imaging in identifying calcified granulomas. However, intraventricular cysts might be difficult to identify with CT due to the similar attenuation of CSF and cyst fluid.[29] The main advantage of MR imaging over CT is its higher contrast resolution, which makes for better lesion conspicuity.

In general, intraventricular cysts are suspected on the basis of mass effect, ventricular obstruction, the presence of a cyst rim, or CSF flow void adjacent to the cyst.[29] Vesicular cysticerci appear on CT and MRI as small and rounded cysts that are well demarcated from the surrounding brain parenchyma.[35] There is no oedema and no contrast enhancement. Many of these lesions have in their interior an eccentric hyperdense nodule representing the scolex giving them a pathognomonic scolex appearance. Colloidal and granular cysticerci appear as ill-defined lesions surrounded by oedema; most of them show a ring or a nodular pattern of enhancement after contrast medium administration. This pattern correspond is commonly referred as to cysticercus granuloma.[27] Calcified cysticerci normally appear on CT as small hyperdense nodules without perilesional oedema or abnormal enhancement after contrast medium administration.

Vasculitis must be suspected when segmental narrowing, a beaded appearance, or an abrupt or tapered area of vascular obstruction is noted at angiography.[21] On the basis of these criteria, arteritis is seen in up to 53% of patients with subarachnoid neurocysticercosis including asymptomatic patients with the middle and posterior cerebral arteries being most commonly affected.[21] Multivessel involvement is noted in nearly 50% of cases, and infarction associated with arteritis is seen in 2%-12%.[28] Cystic lesions located within CSF cisterns usually have a multilobulated appearance, displaced neighbouring structures, and behave as mass occupying lesions.[27]

Treatment of neurocysticercosis is controversial and depends on the form and type of disease as well as the location and number of cysts, the symptoms, and associated complications.[29] In general, antiepileptic drugs, cysticidal agents, albendazole and praziquantel, and corticosteroids and other immunosuppressive or anti-inflammatory agents are used to control the potentially harmful host inflammatory response.[26,36] Although, surgical intervention may eventually become necessary. It is rarely used nowadays because the diagnosis is being made at earlier stages and pharmacologic therapy is usually sufficient.[33,34]

In most patients with neurocysticercosis, the prognosis is good. Associated seizures seem to improve after treatment with anticysticercal drugs and once treated the seizures are controlled by a first line antiepileptic agent. However, the racemose[21] form of this disease is associated with poor prognosis and elevated mortality rate.

The main drawback of our study was less number of patients, inability to identify scolex in all the cases, which further required MRI examination.

CONCLUSION: The prevalence of neurocysticercosis at our study over the period of six years is 4.3%. More commonly observed in paediatric age group (23.2%) and in male sex (58%). CT plays a crucial role in identifying various stages of NCC especially in a rural and semi-urban setup where patients don't have access to state of the art MRI centres.

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