

# Clinical Profile of Childhood Neuro Cysticercosis

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**Abstract:** *Neurocysticercosis (NCC) is a biological marker of poverty and underdevelopment<sup>1</sup>. The frequency of this brain parasitic disease is probably high worldwide, as well as epilepsy; however, little is known about the natural history of the infection in humans. Both, NCC and epilepsy, are an increasing burden on the welfare and economy of developing countries<sup>2</sup>. This is why knowledge of these diseases should be improved, in order to prevent them. The anticysticercal drugs albendazole and praziquantel have been extensively used, and found to be effective for all types of neurocysticercosis. However, recently controversy has been raised about their safety, and long-term clinical usefulness. Preventive health measures, such as provision of safe drinking water and excretion disposal, still offer the best ways to manage this disease.*

**Keywords:** neurocysticercosis ,seizures ,CT brain, ring enhancing lesions , Albendazole

## 1. Introduction

Neurocysticercosis (NCC) is the most common cause of acquired epilepsy in developing countries. It can present variably depending on the location and stage of cysts in the nervous system, and the host immune response. The most common presentation of parenchymal NCC is with seizures that are usually focal and brief; status epilepticus occurs in some cases. About a third of cases have headache and vomiting. Diagnosis is made by either CT or MRI. Single, small, contrast enhancing lesions are the most common; visualization of a scolex is diagnostic. Some cases have multiple cysts with a characteristic starry-sky appearance. Although treatment with cysticidal therapy continues to be debated, there is increasing evidence that it helps through increased and faster resolution of CT lesions; whether there is any improvement in long-term seizure control needs further study. It should not be used in cysticercus encephalitis or in ophthalmic NCC and used with caution in extraparenchymal NCC. It is of no use in calcified lesions. Corticosteroids are used simultaneously to reduce cerebral oedema. Seizures respond well to a single antiepileptic, and the seizure recurrence rate is low in cases with single lesions; those with multiple, persistent or calcified lesions usually have recurrent seizures. Extraparenchymal NCC is often associated with intracranial hypertension, hydrocephalous and chronic meningitis; it has a guarded prognosis; surgical intervention is required in many cases. Management of NCC needs to be individualized. NCC is potentially eradicable; proper sanitation, hygiene and animal husbandry are warranted.

## 2. Clinical Manifestations

Epilepsy is the most common presentation of neurocysticercosis. Seizures are the presenting manifestation in 92% of patients with intra-parenchymal lesions and in 74% of patients with mixed intra- and extra-parenchymal neurocysticercosis. Usually patients have partial seizures 'With or without secondary generalisation, although a few patients may have primary generalised seizures. Partial status epilepticus and post-ictal transient focal neurological

deficits can also occur. Simple partial seizures are much more common than complex partial seizures.

An acute encephalitic form of neurocysticercosis occurs mainly in childhood and adolescents and is the result of widely disseminated small intraparenchymal cysts. These patients have frequent seizures and acutely evolving intracranial hypertension. Systemic disseminated neurocysticercosis is seen in India, with frequent seizures, dementia, muscular pseudohypertrophy few localising signs and often intracranial hypertension<sup>3</sup>.

Focal neurological deficit of vascular origin is another common manifestation of neurocysticercosis. It is usually caused by inflammatory occlusion of the arteries at the base of brain secondary to cysticercotic arachnoiditis. Involvement of spinal cord has variably been reported at 1-5%. Leptomeningeal cysticercosis is frequently seen in association with cysticerci of the posterior fossa due to downward migration of the cysts from basal cisterns of the brain into spinal cord through CSF pathway. Haematogenous spread is responsible for intramedullary (spinal) cysticercosis. The clinical manifestations are directly related to the location of the parasite.

## 3. Diagnosis

The most useful procedure for the diagnosis of neurocysticercosis is computed tomographic (CT) imaging. The CT picture depends on the number, location and stage of the lesions. In the vesicular stage (viable cysts) the cysts appear circumscribed and hypodense. These lesions do not enhance after contrast administration. Surrounding oedema may be mild or absent. It is not uncommon to find an eccentric scolex, present in about 44% of cases. In the colloidal stage, which represents a dying cyst, there is a ring-enhancing lesion surrounded by white matter oedema. In the next nodular-granular stage, the lesions are homogeneously enhancing, and ultimately calcify. The calcified stage; representing a dead parasite, appears as a hyperdense lesion on non-contrast CT scans. Where neurocysticercosis is endemic, patients with epilepsy often have single intracranial calcifications<sup>4,5</sup>.

Magnetic resonance imaging (MRI) shows living parenchymatous cysts, 5-20 mm in diameter, as round lesions of CSF-equivalent density on both T1 and T2-weighted images. An isodense to hyperdense scolex can be identified within most cysts producing a 'pea in the pod' appearance. In dying cysticerci the difference between scolex and cysts becomes unclear. The fluid shows greater and increasing signal intensity than CSF, in both T1- and T2-weighted images. MRI has been shown to have some superiority over CT as it may show cysticerci missed by CT. But calcified lesions are better seen with CT<sup>4,5</sup>

Several immunological tests in the serum and CSF have been developed over the years. The latest, enzyme-linked immunoelectrotransfer blot assay in serum<sup>6</sup> is highly sensitive and specific. more widely available enzyme-linked immunosorbent assay (ELISA) showed sensitivity and 65% specificity in CSF, while older tests (cg, complement fixation) are insufficiently sensitive and specific. Patients presenting only with calcified CT lesions are generally serologically negative. When CSF show inflammatory changes, serological tests for the detection of anticysticercal antibodies in CSF by ELISA is almost always positive.

Stool examination for ova and parasites can occasionally be used to diagnose intestinal infection with *T. solium*. However, most people diagnosed with cysticercosis do not have viable *T. solium* tapeworm in their intestine, so eggs are not typically found.

#### 4. Treatment

Apart from symptomatic treatment other options for the treatment of neurocysticercosis include anticysticercal drugs, corticosteroids, CSF shunting, surgical removal or decompression of the cyst. Albendazole was initially administered at doses of 15 mg/kg/d during 1 month.<sup>7</sup> However, further studies showed that similar results may be obtained when the length of therapy is shortened to 1 week.<sup>8</sup>

#### 5. Materials And Methods

All Children aged between 1-12 years who were admitted in the department of pediatrics, government general hospital over a period of one year with the complaint of seizures (unprovoked) were studied. It's a prospective clinical study. Detailed history was taken, clinical examination and systemic examination was done. All the neurological deficits if present were noted. Routine investigations, complete hemogram, EEG, CT scan brain (plain and contrast) and MRI were done.

#### 6. Observations

Total number of outpatient cases over the study period was 54066 out of which 3093 cases were admitted as inpatients. Among 3093 Patients the total number of cases with seizures (unprovoked) was 148. Among the above said cases 31 cases were proved out to be neurocysticercosis.

#### 7. Results

##### Age distribution

Age in Yrs	1	2	3	4	5	6	7	8	9	10	11	12
No. of Cases	0	0	4	3	2	0	1	2	3	5	4	7

No. of cases less than 5 yrs were 9, more than 5 years were 22, maximum no. of cases were seen around 12 yrs age group.

##### Sex distribution

Male	17	54.80%
Female	14	45.20%

##### Rural versus urban incidence:

rural	29	93.50%
urban	2	6.50%

##### Socio economic groups:

Low	29	93.60%
Middle	1	3.20%
high	1	3.20%

##### Food habits

Veg	16	51.60%
Non veg	15	48.40%

##### Clinical Presentations

###### 1. Seizures

Cps	14	45.20%
Sps	10	32.30%
GS	7	22.60%

2. headache : 11 (35.5%)
3. vomittings: 8 (25.8%)
4. fever: 4 (12.9%)
5. pyramidal tract signs :2 (6.5%)
6. cranial nerve involvement: UMN type of facial weakness :1 (3.2%)
7. papilloedema: early papilloedema : 1 (3.2%)
8. Cerebellar signs-not seen
9. Hydrocephalus-not seen.

###### Lateralization

RT	11/21	52.40%
LT	10/21	47.60%
CT CHARACTERS		RAISED ICT
Solitary	21/31	67.70%
multiple	10/31	32.30%
	7/21	33.30%
	6/10	60%

Hypodense	2/21	19.50%
Ring enhancing	12/21	57.10%
Disc enhancing	7/21	33.30%

#### 8. Discussion

NCC is one of the most serious problems of public health in the developing nations. It has varied clinical presentations. CT scan has proved helpful not only in suspecting the etiology but in finding the number, localization and extent of lesion. In our institute 1% cases among total admissions in 1

year were neurocysticercosis. 22 patients (70.97%) among 31 belongs to the age group more than 5 years. There is no sex predilection. Mostly Neurocysticercosis is commonly seen in rural population (93.5%). It is also common in low socioeconomic families (93.6%). The incidence of neurocysticercosis is almost equal among non vegetarian and vegetarians. Seizures are the most common complaint followed by headache and vomiting. Kuruvilla et al (2001) reported 100% incidence of seizure in their study<sup>9</sup>. Among seizures complex partial seizures was the common manifestation (45.2%) followed by simple partial seizures, CPSw with secondary generalization and generalized seizures. Headache was reported in 35.5% in our study which was correlated with the study of varma et al who reported it to be 37.5%.<sup>10</sup> focal neurological deficits were rarely reported.

In CT scan brain parenchymal lesions are common among which single lesions were found in 21 (67.7%) patients and majority of them were ring enhancing lesions. Common site is the parietal lobe seen in 14 patients (45.2%) followed by frontoparietal lobe with 4 patients (12.9%).

Most of the parietal lobe lesions presents with simple partial seizures. Frontoparietal and frontal lesions presented with complex partial seizures. Multiple lesions presented with generalized seizures. Raised intra cranial pressure is seen with multiple lesions with extensive perifocaledema.

## 9. Summary

Epilepsy and NCC are common diseases in poor countries, and NCC is increasingly diagnosed in developing countries. Though serological tests & Histopathological examination are important in the diagnosis of neurocysticercosis, because of their non-availability and cost, CT scan plays an important role in the diagnosis of neurocysticercosis. The outlook is very good because of improved neuroimaging and therapeutic modalities. No sequelae were left in all neurocysticercosis cases after treating with steroids, albendazole for 2-3 weeks and anti epileptic drugs for 6 months.

## 10. Recommendations

Further research should be undertaken in order to clarify the natural history of T. Solium / cysticercosis disease, the variability of antihelminthic treatment and its efficacy, the factors that contribute to clinical heterogeneity of NC, immunological response of the host. Health authorities should focus on prevention and eradication of taeniasis / cysticercosis in order to decrease the number of individuals with seizures / epilepsy and other consequences.

## References

- [1] Carpio A. Neurocysticercosis: an update. *Lancet Infectious Diseases*. 2002;2:751-762.
- [2] Pal DK, Carpio A, Sander JWAS. Neurocysticercosis and Epilepsy. *J NeurolNeurosurgPsychiatry* 2000;68:137-143.
- [3] Brown WJ, Voge M. Cysticercosis: modern day plague. *Pediatr Clin North Am* 1985;32:953-69
- [4] Garcia HH, Del Brutto OH. Cysticercosis Working Group in Peru. Neurocysticercosis: updated concepts about an old disease. *Lancet Neurol* 2005;4(10):653-61.
- [5] Del Brutto OH. Neurocysticercosis. *Semin Neurol* 2005;25(3):243-51.
- [6] Tsang VC, Brand JA, Boyer AE *J Infect Dis*. 1989 Jan; 159(1):50-9.
- [7] Escobedo F, Penagos P, Rodriguez J, Sotelo J. Albendazole therapy for neurocysticercosis. *Arch Intern Med*. 1987;147(4):738-741
- [8] Garcia HH, Gilman RH, Horton J, et al. Albendazole therapy for neurocysticercosis: a prospective double-blind trial comparing 7 versus 14 days of treatment. *Neurology*. 1997;48(5):1421-1427.
- [9] Kuruvilla A, Pandian JD, Nair M, Radhakrishnan VV, Joseph S. Neurocysticercosis: a clinical and radiological appraisal from Kerala State, South India. *Singapore Med J* 2001;42: 297-303
- [10] Verma A, Gaur KJ. The clinical spectrum of neurocysticercosis in Uttaranchal. *J Assoc Physicians India* 2002;50:1398-400



Figure 1: Neurocysticercosis (Parieto-Occipital Lesion)

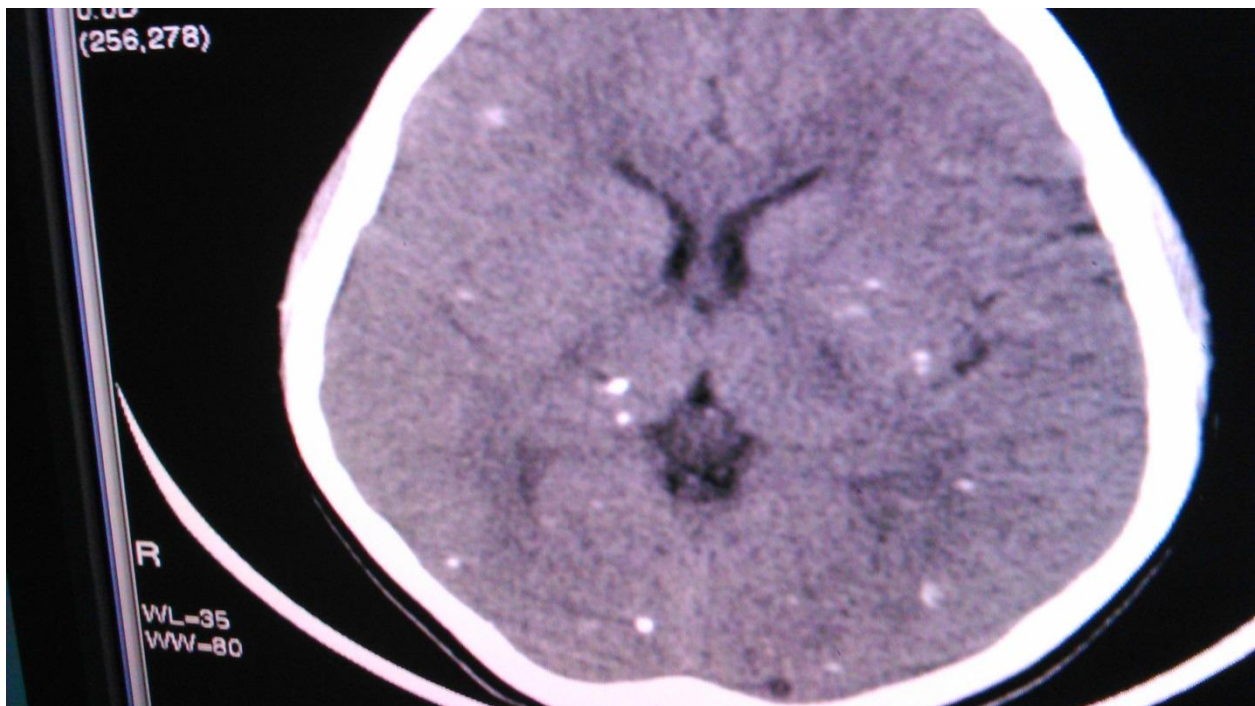


Figure 2: Neurocysticercosis –Multiple Calcifications (Healed Lesions)