



RESEARCH ARTICLE

A STUDY OF NEUROCYSTICERCOSIS PUBLIC HEALTH PROBLEM IN THE PEDIATRICS WARD OF TERTIARY CARE CENTRE IN KUMAUN REGION, INDIANutan Singh^{*1}, Amit Kumar Singh², Sandeep Gaur³, Rajnish Kumar⁴¹Associate Professor, Department of Pediatrics, Government Medical College, Haldwani, Distt-Nainital (Uttarakhand), India.²Assistant Professor, Department of Pediatrics, Government Medical College, Haldwani, Distt-Nainital (Uttarakhand), India.³Lecturer, Department of Pharmacology, Government Medical College, Haldwani, Distt-Nainital (Uttarakhand), India.⁴PG JR-3, Department of Pediatrics, Government Medical College, Haldwani, Distt-Nainital (Uttarakhand), India.

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ABSTRACT

Neurocysticercosis (NCC) is a common cause of seizures and neurologic disease. Although there may be variable presentations depending on the stage and location of cysts in the nervous system. Most children (> 80%) present with seizures particularly partial seizures. Neurocysticercosis has been increasingly recognized as a major cause of neurologic disease worldwide and an important problem among immigrant populations in India. Neurocysticercosis is the most common parasitic infection of the CNS. Tissue-invading larval forms of the pork tapeworm *Taenia solium* cause the disease. The study based on to determined incidence of Neurocysticercosis (NCC) in patients presented with seizures. and to define the age group of children which was more prone for neurocysticercosis. The study was based on 1223 total number of patients admitted with neurocysticercosis (NCC) over a period of six years. These all patients were admitted in department of pediatrics, UFHT medical college & hospital, Haldwani (presently known as Government Medical College & Hospital, Haldwani). All Neuro imaging investigations were investigated in deptt of radiodiagnosis, government medical college and hospital, Haldwani. Pathological and microbiological investigation also done in centre lab of government medical college and Hospital, Haldwani. All the patients were examined clinically and were investigated with neuroimaging (CT Scan and MRI). In addition to above X Ray chest, Mantoux test, haematological investigations including ESR and CSF was done to rule out Tuberculoma. Routine stool examination was also done.

A strikingly high incidence of Neurocysticercosis (NCC) was found among the pediatric patients of our tertiary care hospital. A high incidence of NCC in kumaun region reflects the endemic presence of *taenia solium*.

Keywords: Neurocysticercosis, Pediatric patients, Seizures, *Taenia solium*, Neurologic disease.

INTRODUCTION:

Cysticercosis refers to tissue infection after exposure to eggs of *Taenia solium*, the pork tapeworm. The disease is spread via the fecal-oral route through contaminated food and water, and is primarily a food borne disease^[1]. After ingestion the eggs pass through the lumen of the intestine into the tissues and migrate preferentially to the brain and muscles. There they form cysts that can persist for years. In some cases the cysts will eventually cause an inflammatory reaction presenting as painful nodules in the muscles and seizures when the cysts are located in the brain^[2]. Symptomatic disease from *Taenia solium* cysts in the brain is referred to as neurocysticercosis and is the most common tapeworm infection of the brain worldwide. Cysticercosis should be differentiated from taeniasis:

carriage of the adult tapeworm in the intestine^[3] (which is through ingestion of cysts in an intermediate host, not the ingestion of the eggs as in cysticercosis). These represent two different stages of the parasite's life cycle. Though both forms of infection can potentially occur in the same individual at the same time, they are distinct disease entities and have different treatments and potential outcomes^[4].

Historically, neurocysticercosis was endemic to only Latin America, Asia, and Africa, although it has become increasingly frequent in the United States since the 1980s. Because of this epidemiologic change, all general pediatricians should become familiar with this disease process. The disease has been the subject of several recent reviews^[5].

The term neurocysticercosis is generally accepted to refer to cysts in the parenchyma of the brain. It presents with seizures and, less commonly, headaches [6]. *Cysticercus* in brain parenchyma are usually 5–20 mm in diameter. In subarachnoid space and fissures, lesions may be as large as 6 cm in diameter and lobulated. Cysts located within the ventricles of the brain can block the outflow of cerebrospinal fluid and present with symptoms of increased intracranial pressure [7].

Racemose neurocysticercosis refers to cysts in the subarachnoid space. These can occasionally grow into large lobulated masses causing pressure on surrounding structures. Neurocysticercosis involving the spinal cord, most commonly presenting as back pain and radiculopathy [8].

The variable manifestations include seizures, hydrocephalus, and other neurologic dysfunctions.

Its clinical effects vary depending upon site of larval lodging, larval burden and host reaction. These effects include seizures, headache, focal neurological symptoms, visual disturbances and localised skeletal muscle nodules and pain [9] (Kraft R).

NCC is not only a public health problem in developing countries but its incidence is increasing currently in developed countries too due to migration movements from endemic countries [10].

Patho-physiology of *T.solium*:

The larvae of *T solium* (*Cysticercus cellulosae*) cause neurocysticercosis. This pork tapeworm can vary in size but is notable for a scolex (head) with approximately 25 hooklets, 4 suckers, and a body with 700-1000 proglottids [11]. The ova of the tapeworm are spread via the fecal-oral route and are approximately 40 microns in diameter with a radially striated shell [12]. The intermediate host is the pig, which harbors the larvae after eating ova, and the definitive host is the human being.

If pig products infected with larvae are ingested, a tapeworm infection in the intestines ensues; however, if ova are ingested, neurocysticercosis may occur in this normally intermediate host. The ingested ova develop into larvae (cysticerci) and lodge in soft tissues, especially skin, muscle, and brain. Cysticerci are fluid-filled oval cysts, approximately 1-2 cm in diameter, with an internal scolex [13].

The eggs are found in human feces because humans are the only definitive hosts. Greatest risk for infection occurs in regions where plants in gardens or farms are fertilized

with human feces and humans are exposed to contaminated soil [14].

In the CNS, *T solium* is deposited in the cerebral parenchyma, meninges, spinal cord, and eyes. Unless large numbers of cysts are present, the body's immune system does not act to destroy the organism, and cysts can live for many years undetected [15]. A live cyst can go undetected for as long as 5 years before dying or causing symptoms in the host.

Neurologic symptoms arise when the encysted worm dies and the human mounts an associated inflammatory response. If the cyst lodges in the ventricular system (especially the fourth ventricle), hydrocephalus can occur [16].

MATERIAL AND METHODS:

The retrospective study was conducted on 1223 patients admitted in IPD, Deptt of Pediatrics government medical college hospital, Haldwani (STM hospital) from January 2005 to December 2010. All admitted patients have complained seizure disorder.

Patients were classified according to their age groups, residence rural or Urban and the type of seizures. Through history regarding seizures type, presence of fever, number of episodes of unconsciousness was taken. Other important history i.e H/O contact with tuberculosis, family H/O seizures, socio-economic status environmental and sanitation facilities, water supply, sewage and drainage system, type of toilets. Proper clinical, general and systemic examination was done.

INVESTIGATIONS:

Haematological investigations, RBS, S. electrolytes (Na, K, Ca), SGPT, Serum Creatinine, Blood Urea, X-Ray chest, Mantoux test, neuroimaging was done. In most cases plain CT Scan was done. In doubtful cases contrast CT Scan and MRI was done as needed.

RESULTS:

The total number of 1223 children patients were studied over a period of 6 years. In 1223 numbers of children 438 patients are suffering with Neurocysticercosis, and the average percentage is 33.92% (Table -1). 263 Patients were suffering from generalized tonic clonic seizures, 153 patients suffering from Complex partial seizure and 22 patients were suffering from simple partial seizure (Table-2).

Table 1: Neurocysticercosis (NCC) with seizures.

S.NO	YEARS	CHILDREN (0-15yrs)		
		Seizure	Neurocysticercosis	Percentage (%)
1.	2005	68	13	19.11
2.	2006	215	62	28.83
3.	2007	151	43	28.47
4.	2008	293	105	35.83
5.	2009	301	105	34.88
6.	2010	195	110	56.40
TOTAL		1223	438	33.92

Table 2: Types of Seizures

Type of seizure	Number of patients	Percentage
Generalized Tonic – clonic seizures	263	60.04
Complex partial seizure	153	34.94
Simple partial seizure	22	5.02
Total	438	100

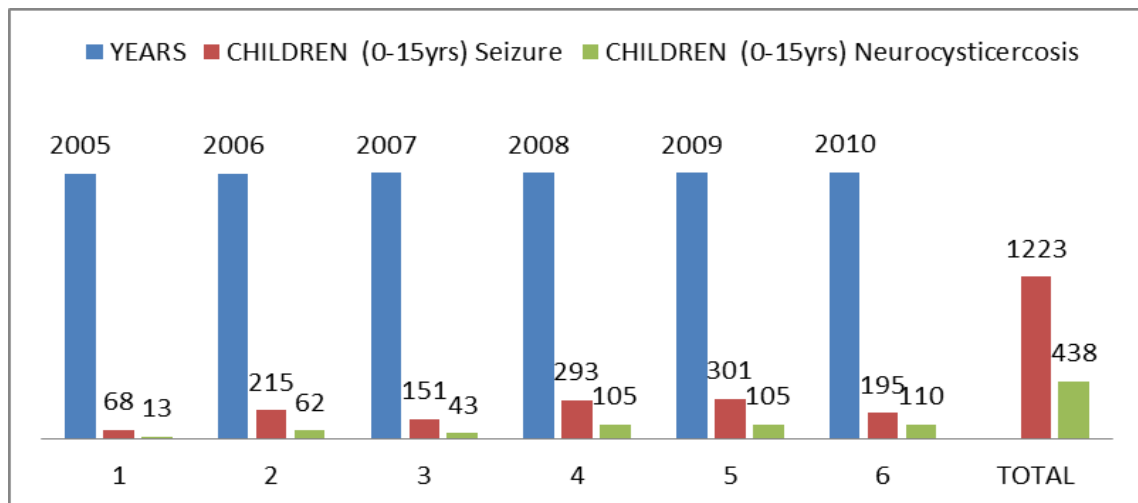


Figure 1: Graphical representation of seizures and Neurocysticercosis.

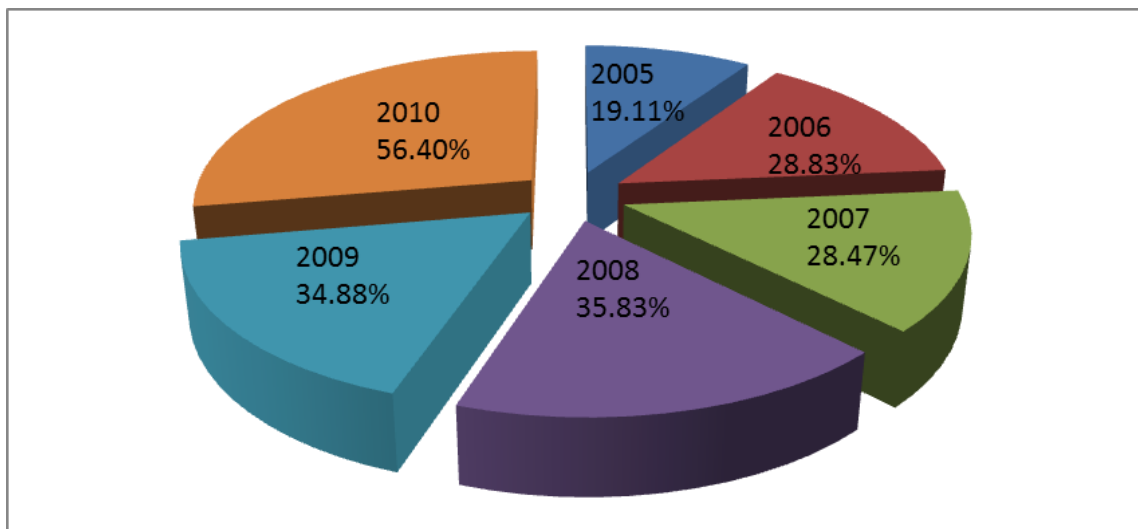


Figure 2: Percentage of neurocysticercosis in different years.

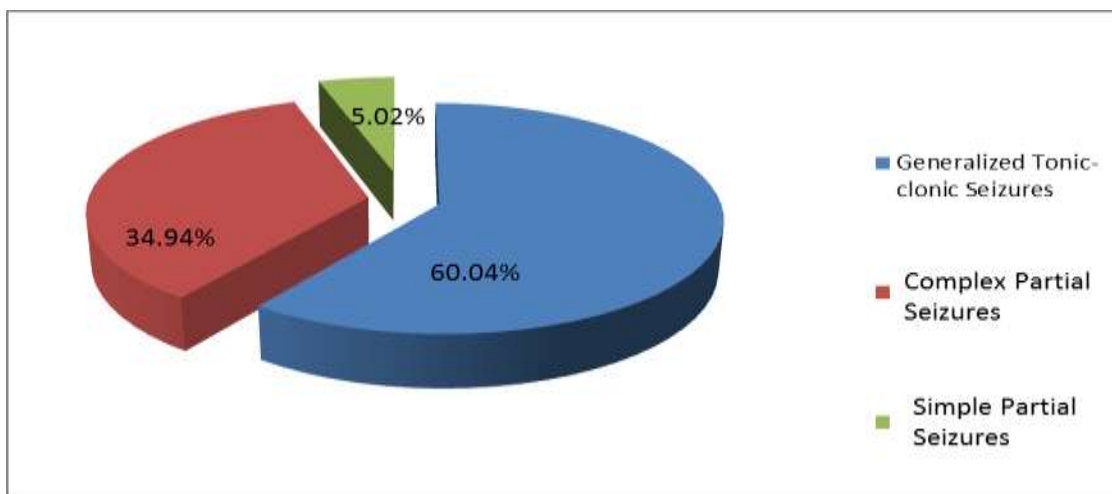


Figure 3: Percentage type of seizures

DISCUSSION:

Many standard texts mention that 1st episode of single unprovoked seizure need not be investigated and treated. But the rising trend in the incidence of NCC was seen during our study period of 6 years which could be attributed to neuroimaging being an essential investigation in every case of seizures due to high index of suspicion .A significantly high incidence of NCC was seen in pediatric age group as compared to adults.

During our 6 years of study period NCC has emerged as an alarming public health problem of our region and its incidence has risen from 19.11% in 2005 to 56.4% in 2010 in pediatric patients .The rising incidence could be due to high index of suspicion in every case presenting with seizures. Earlier thought to be a disease of developing nation, the incidence of NCC is rising currently in developed countries as well; due to migration movement from endemic countries . Various studies have shown that NCC is the most common cause of acquired seizures worldwide. A study in vellore district showed overall incidence of NCC to be about 28.4% among total patients of active epilepsy .which is comparable to the results of our study which shows an overall incidence of NCC to be 33.92 % in all age groups pediatrics.

Clinical presentation of NCC in patients depends on the site of focus of the cysticerci .Different studies mention seizures as the commonest presentation. In our study 1223 patients presenting with seizures 33.92% was due to NCC.

CONCLUSION:

During the course of our study we concluded that NCC should be suspected in every case of seizures irrespective of its type in endemic regions, and subjected to neuroimaging even in cases of first episode of unprovoked seizures though some standard texts suggest that neuroimaging is generally not recommended after a

first episode of unprovoked seizures (Nelson textbook of paediatrics 17th ed.pg 1994).

More over we conclude that a high incidence of NCC in our region reflects the endemic presence of taenia solium which is a cause of high morbidity and financial burden to the individual and society. Keeping in view the preventable nature of the disease more emphasis should be laid on providing better sanitation facilities and safe drinking water. The masses should be educated to practice better hygiene, use filtered water, avoid eating anything raw.

The education programme should start from schools, panchayats where audio-video programmes can be shown to leave a better and long lasting impact. The government agencies should provide facilities for safe drinking water by installing water purifier and filtration units at common places like hospitals ,schools , working places .The vegetable markets should be on a raised platform with water points where the vendors can wash the vegetables. The drainage facilities should be adequate to avoid water logging.

RECOMMENDATION:

All cases of seizures whether first or recurrent should be thoroughly investigated including neuroimaging as the most common type of seizure in pediatric patients is generalized tonic clonic seizure which is potentially life threatening.

REFERENCES:

1. Prasad KN, Prasad A, Verma A, Singh AK. Human cysticercosis and Indian scenario: a review. *J Biosci.* Nov 2008;33(4):571-82.
2. Serpa JA, Yancey LS, White AC Jr. Advances in the diagnosis and management of neurocysticercosis. *Expert Rev Anti Infect Ther.* Dec 2006;4(6):1051-61.

3. Shandera WX, Kass JS. Neurocysticercosis: current knowledge and advances. *Curr Neurol Neurosci Rep.* Nov 2006;6(6):453-9.
4. Singhi P, Singhi S. Neurocysticercosis in children. *J Child Neurol.* Jul 2004;19(7):482-92.
5. Sinha S, Sharma BS. Neurocysticercosis: A review of current status and management. *J Clin Neurosci.* Apr 24 2009.
6. Singhi P. Neurocysticercosis. *Ther Adv Neurol Disord.* Mar 2011;4(2):67-81.
7. Serpa JA, Graviss EA, Kass JS, White AC Jr. Neurocysticercosis in Houston, Texas: an update. *Medicine (Baltimore).* Jan 2011;90(1):81-6.
8. Sorvillo F, Wilkins P, Shafir S, Eberhard M. Public health implications of cysticercosis acquired in the United States. *Emerg Infect Dis.* Jan 2011;17(1):1-6.
9. Zee CS, Go JL, Kim PE, DiGiorgio CM. Imaging of neurocysticercosis. *Neuroimaging Clin N Am.* May 2000;10(2):391-407.
10. Wallin MT, Kurtzke JF. Neurocysticercosis in the United States: review of an important emerging infection. *Neurology.* Nov 9 2004;63(9):1559-64.
11. Garcia HH, Del Brutto OH, . Neurocysticercosis: updated concepts about an old disease. *Lancet Neurol.* Oct 2005;4(10):653-61.
12. Chaoshuang L, Zhixin Z, Xiaohong W, Zhanlian H, Zhiliang G. Clinical analysis of 52 cases of neurocysticercosis. *Trop Doct.* Jul 2008;38(3):192-4.
13. Jung H, Cardenas G, Sciutto E, Fleury A. Medical treatment for neurocysticercosis: drugs, indications and perspectives. *Curr Top Med Chem.* 2008;8(5):424-33.
14. Ruiz-Garcia M, Gonzalez-Astiazaran A, Rueda-Franco F. Neurocysticercosis in children. Clinical experience in 122 patients. *Childs Nerv Syst.* Nov-Dec 1997;13(11-12):608-12.
15. Talukdar B, Saxena A, Popli VK. Neurocysticercosis in children: clinical characteristics and outcome. *Ann Trop Paediatr.* Dec 2002;22(4):333-9.
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.* May 2011;5(5):e1152.
17. Rosenfeld EA, Byrd SE, Shulman ST. Neurocysticercosis among children in Chicago. *Clin Infect Dis.* Aug 1996;23(2):262-8.
18. Michelet L, Fleury A, Sciutto E, Kendjo E, Fragoso G, Paris L, et al. Human neurocysticercosis: comparison of different diagnostic tests using cerebrospinal fluid. *J Clin Microbiol.* Jan 2011;49(1):195-200.
19. Garg RK, Sinha MK. Multiple ring-enhancing lesions of the brain. *J Postgrad Med.* Oct-Dec 2010;56(4):307-16.
20. Fleury A, Hernandez M, Avila M, et al. Detection of HP10 antigen in serum for diagnosis and follow-up of subarachnoidal and intraventricular human neurocysticercosis. *J Neurol Neurosurg Psychiatry.* Sep 2007; 78(9):970-4.
21. Almeida CR, Ojopi EP, Nunes CM, et al. Taenia solium DNA is present in the cerebrospinal fluid of neurocysticercosis patients and can be used for diagnosis. *Eur Arch Psychiatry Clin Neurosci.* Aug 2006; 256(5):307-10.
22. Rodriguez S, Dorny P, Tsang VC, Pretell EJ, Brandt J, Lescano AG. Detection of Taenia solium antigens and anti-T. solium antibodies in paired serum and cerebrospinal fluid samples from patients with intraparenchymal or extraparenchymal neurocysticercosis. *J Infect Dis.* May 1 2009; 199(9):1345-52.
23. Balaji J, D M. Clinical and Radiological Profile of Neurocysticercosis in South Indian Children. *Indian J Pediatr.* Mar 12 2011.
24. Figueroa JJ, Davis LE, Magalhaes A. Extraparenchymal neurocysticercosis in albuquerque, New Mexico. *J Neuroimaging.* Jan 2011;21(1):38-43.
25. Odermatt P, Preux PM, Druet-Cabanac M. Treatment of neurocysticercosis: a randomised controlled trial. *J Neurol Neurosurg Psychiatry.* Sep 2008;79(9):978.
26. Thussu A, Chattopadhyay A, Sawhney IM, Khandelwal N. Albendazole therapy for single small enhancing CT lesions (SSECTL) in the brain in epilepsy. *J Neurol Neurosurg Psychiatry.* Mar 2008;79(3):272-5.
27. Carpio A, Kelvin EA, Bagiella E, Leslie D, Leon P, Andrews H. Effects of albendazole treatment on neurocysticercosis: a randomised controlled trial. *J Neurol Neurosurg Psychiatry.* Sep 2008;79(9):1050-5.
28. Ramirez-Zamora A, Alarcon T. Management of neurocysticercosis. *Neurol Res.* Apr 2010;32(3):229-37.
29. Abba K, Ramaratnam S, Ranganathan LN. Anthelmintics for people with neurocysticercosis. *Cochrane Database Syst Rev.* Jan 20 2010;CD000215.
30. [Best Evidence] Del Brutto OH, Roos KL, Coffey CS, Garcia HH. Meta-analysis: Cysticidal drugs for neurocysticercosis: albendazole and praziquantel. *Ann Intern Med.* Jul 4 2006;145(1):43-51.