

Imaging and Serological-Evidence of Neurocysticercosis Among Patients with Seizures in Odisha, an Unexplored Eastern Coastal Province in India

PRIYADARSHI SOUMYARANJAN SAHU¹, SHUBHRANSU PATRO², PAYOD KUMAR JENA³, SANTOSH KUMAR SWAIN⁴, BIDYUT KUMAR DAS⁵

ABSTRACT

Introduction: Neurocysticercosis being a potential to human transmitted disease, is the major cause of seizures and a public health problem in tropical countries. Though India is known to be highly endemic, there are many provinces where reports are still unavailable thereby underestimating its actual burden.

Materials and Methods: Anti-Cysticercus IgG antibodies in sera from cases presenting with seizures were screened by ELISA in a preliminary study in Odisha state which is a province in Eastern coastal India that was never explored before. Patients presenting with recent onset of seizures within age group 5 to 50 years, either local residents of Odisha or inhabitants from other parts of the country living for at least one year period in the study area were included.

Results: The present study showed 28.12% cases with seizures to be confirmed neurocysticercosis (NCC) based on serology and brain imaging. However, statistically no association was established between anti-Cysticercus antibody detection and radio imaging characteristics (location, number of lesions, and stage).

Conclusion: This is the first study in Odisha presenting a series of cases with serological evidence of exposure to the parasite along with imaging characteristics which was consistent with NCC. It is recommended that NCC must be considered for a differential diagnosis in each active epilepsy case irrespective of prior prevalence information in all unexplored provinces in India and other endemic regions; also a compulsory reporting is warranted in order to aid in quantifying its actual burden.

Keywords: *Cysticercus cellulosae*, ELISA, Epilepsy, *Taenia solium*

INTRODUCTION

Cysticercosis of the central nervous system (CNS) called as neurocysticercosis (NCC) is known to be a debilitating infectious cause of neurological disorders in human since many decades in the past particularly in the tropical developing countries [1]. However, only recently its importance is being recognized as a potential human to human transmitted parasitic disease, and the major cause of epilepsy; thus causing a significant neurological health problem and disability particularly in the tropics [2-6]. Acute epileptic seizure with altered sensorium and raised intracranial pressure may require ventilatory support in an intensive care unit that remains as the mainstay of management of NCC during acute presentations [7]. Human gets infected either by accidental ingestion of *Taenia solium* eggs through contaminated food or vegetables or due to endogenous autoinfection because of the carriers of the adult worm in their intestine; by either means eggs containing the hexacanth larvae disseminate hematogenously to the brain developing into metacestode larvae or cysts.

A recent meta-analysis summarizes the proportion of NCC among patients with epilepsy (PWE), and suggests that in endemic communities nearly one-third of PWE are living with *T. solium* cystic lesions in their brain [8]. This disease is known to be highly endemic in Indian subcontinent and the whole of South East Asia [9]. In a recent Indian study, 89.66% cases having neurological manifestations with inflammatory granulomas were found consistent with diagnosis of NCC [10]. However, the serodiagnostic confirmation was not performed in those cases. NCC was found to be associated with approximately one-third of all cases presenting with active epilepsy in either urban or rural regions as per a study reported from Vellore district of Tamil Nadu (India) [11]. The pleomorphic and unpredictable course of NCC can be related to the biological stage of the parasites, their numbers as well as the immunogenetic factors in man [12].

Though India is known to be highly endemic, there are many provinces where reports are still unavailable or scanty thereby underestimating its actual burden in this country [13]. The present study is done in Odisha that is considered to be a Gate way to the eastern India, located on the Bay of Bengal coast. With the rapidly changing economy as well demography, the underlying prevalence of *T. solium* cysticercosis was never explored in this area. From this region, the first published case of NCC (as per MEDLINE database) was identified in a psychiatric patient with a manic episode of seizure which was confirmed by CT scan of brain in one of the major medical center in Cuttack. In that case, psychiatric manifestation showed a gradual decline following treatment with medication [14]. After a gap of four years (as per MEDLINE database), there was another case reported from a tertiary care corporate medical center in the western part of Odisha; a young woman presented with the features of pseudotumor cerebri, where medical treatment failed and finally the cysticercous cyst was detected in the spinal canal only during a lumbo-peritoneal shunt to save her vision [15]. In this case, she did not show any evidence of spinal involvement directly, thereby eluding its correct diagnosis; however, the diagnosis was confirmed later by histopathological examination. Since then there have been a lot of changes that have occurred in the demographics of this developing state affecting the local communities due to rapid increase in both inflow and outflow of populations into and out of this province for various purposes including but not limited to trading, education, research, healthcare, tourism, as well as pilgrimage.

Either due to lack of a confirmatory diagnosis or unawareness about the necessity of its documentation, there was a lack of reported cases or notifications in this region which probably has given the false impression about its low prevalence or rareness in this region. The latest published information on diagnosis of NCC from Odisha state was 2 cases of disseminated cysticercosis with multiple cystic

lesions in brain [7]. Assuming the real scenario to be worse, a screening study was done to find out the possible burden of NCC among cases of epilepsy or recent onset of seizures.

Hence, the objective of the present study was to address the issue of NCC prevalence among cases in Odisha, those who present with recent onset of seizures. In order to supplement the clinical suspicion of NCC, usefulness of a serological test detecting antibody by ELISA in addition to brain imaging are discussed.

MATERIALS AND METHODS

This study was conducted in a University teaching hospital in Odisha State where patients were recruited from Neurology clinic as well as Medicine Out Patient Department between September 2012 to April 2013. Cases were also referred from another hospital located in the Western part of Odisha, India. All the samples were analyzed in one laboratory employing same protocol. After due approval of the protocol and permission from the institutional ethics committee, a total of 64 cases with a recent onset seizures were recruited against their written consent. A detailed history was collected from each patient and a routine clinical examination was conducted before to recruit them for this study. The age range of patients was between 5 to 50 years with male predominance (male:female \approx 2.5:1).

Inclusion criteria

Patients presenting with recent onset of seizures within age group 5 to 50 years, either local residents of Odisha or inhabitants from other parts of the country living for at least one year period in the study area were included.

Exclusion criteria

Subjects above the age of 50, pregnant woman, and children below the age of 5 years were excluded; individuals having past history of accident and trauma in brain, current history of any other chronic diseases, symptoms of febrile seizures (particularly children) were excluded from the study. Short term visitors or tourists, and out of state individuals living in the study area for less than 1 year period were also excluded.

Brain imaging by computed tomography (CT)

CT imaging was performed in all cases. The study subjects were initially hypothesized as possible cases of NCC based on two minor plus one epidemiological criterion as per the revised diagnostic criteria suggested by Del Brutto [16].

Sample collection

Peripheral whole blood samples (3ml from each) from the recruited cases were collected aseptically upon written consent. Serum was separated from each sample aseptically and then preserved at -80°C till use.

Taenia solium IgG ELISA in serum

A commercially procured ELISA Kit (NovaTec Diagnostics, Germany) was employed for detection of anti-Cysticercus IgG antibodies in all the collected sera following manufacturer's instruction. Briefly, antigen coated wells (as supplied by the kit manufacturer) were incubated with 1:10 diluted patient serum (diluted with the serum diluent fluid provided in the kit). A negative control serum, a low positive control serum, and a high positive control serum (all supplied by the manufacturer) were also used for validity of the test. Absorbance at 450nm (OD_{450}) was measured. The sensitivity and specificity of this anti-Cysticercus antibody ELISA in serum was 85% and 94% respectively based on results of the test using sera from cases with a confirmed diagnosis of NCC and healthy normal group as described in our past study [17].

RESULTS

In this study, a positive anti-Cysticercus IgG-ELISA result was observed in 18 of the total 64 cases indicating an overall positivity of 28.12% [Table/Fig-1]. There were 43.75% (28 of 64) cases presenting with generalized seizure and 56.25% (36 of 64) cases with partial seizure (Complex partial= 16 cases; simple partial= 20 cases). The mean OD_{450} values of the ELISA on sera identified to be positive or negative for anti-Cysticercus-IgG antibodies ($n_1=18$ and $n_2=46$ respectively) were found to be extremely statistically significant (two-tailed p-value < 0.001) using GraphPad QuickCalcs free statistical calculators available on web (<http://www.graphpad.com/quickcalcs>). The age range of the cases was found to be 12 to 50 y (median age = 25 y) and there were 15 male and 3 female patients who tested positive by ELISA. The age range of male cases was 5-39 y and that in female cases was from 10 to 45 y. There was history of migration of 6 cases from other neighboring states in India. The ELISA results demonstrating anti-Cysticercus-IgG antibodies in sera from the recruited cases presenting with different patterns of seizure are stated in [Table/Fig-1]. Among the ELISA positive cases, majority presented with generalized seizure (35.71%), followed by simple partial seizure (25%), and complex partial seizure (18.75%). Headache was the most common problem being presented in each of our cases. Other clinical presentations in an decreasing order of occurrences are vomiting (50%), pallor (27.78%), altered sensorium (22.23%), and muscle weakness (11.11%).

[Table/Fig-2,3] summarize the anti-Cysticercus-IgG test results with respect to the CT findings showing number of lesions and their distribution in brain. In the present study, majority of the cases presented with a single space occupying lesion ($n=41$) whereas only 14 cases had ≥ 2 lesions based on CT. Normal CT was reported in 9 cases of which one case only appeared positive for anti-Cysticercus-IgG. The distribution of lesions in various parts of the brain is presented in [Table/Fig-3]. In this series the most common location was the parietal lobe followed by frontal lobe; however the percentage of cases positive for anti-Cysticercus antibodies

Type of seizure or seizure pattern	Number of cases recruited	Anti-Cysticercus IgG-ELISA		
		No. (%) of sera tested positive	No. (%) of sera tested negative	Statistical analysis
Generalized seizure	28	10 (35.71)	18 (64.29)	$\chi^2=1.59$ df=2 *p=0.452
Simple partial seizures	20	5 (25)	15(75)	
Complex partial seizures	16	3 (18.75)	13 (81.25)	
	Total=64	18 (28.12)	46 (71.88)	

[Table/Fig-1]: Results of ELISA showing anti-Cysticercus antibodies in sera from patients with respect to different seizure patterns

Percentage of sera tested positive under each location in brain was calculated based on total cases under each category.

* p value was calculated based on non-parametric chi square analysis using Epi Info2001. No significance was estimated between the seizure patterns vs. antibody positivity ($p=0.452$)

	Total cases (n)	Anti-Cysticercus IgG-ELISA		
		No. (%) of sera tested positive	No. (%) of sera tested negative	Statistical analysis
Number of lesions in brain (n=64)**				
No lesion found (Normal Scan)	9	1 (11.11)	8 (88.89)	$\chi^2=2.83$ df=2 *p=0.243
Multiple lesions	14	6 (42.85)	8 (57.15)	
Single lesion	41	11 (26.82)	30 (73.18)	
	Total=64	18 (28.12)	46 (71.88)	

[Table/Fig-2]: ELISA demonstrating anti-Cysticercus antibodies in sera from patients with epilepsy with respect to number of lesions in brain

Percentage of sera tested positive under each location in brain was calculated based on total cases under each category

* p value was calculated based on non-parametric chi-square analysis using Epi Info2001; there was no statistical difference between the cases with single lesion vs those with multiple lesions in brain and the ELISA-positive results ($p=0.243$)

** Number of lesions, and location was based on the CT features and of 64 total number of cases fifty five cases presented with either one or more number of lesions in the brain whereas 9 cases did not show any lesion (normal scan); n= total number of cases

was comparable. Occipital lobe was the least common site. In this screening study CT scan revealed presence of lesion(s) in the brain in 17 of 18 serologically positive cases. Presence of either single or multiple lesions, the lesion characteristics are shown in [Table/ Fig-4]. In this series, majority had a calcified cyst (8 cases single and 4 cases multiple= 12 cases). Active perilesional inflammation was detected in 4 cases. A single case with a positive ELISA test had the parasite at the vesicular stage.

	Total number of cases	Anti-Cysticercus IgG-ELISA		
		No. (%) of sera tested positive	No. (%) of sera tested negative	Statistical analysis
Location of lesions in brain (n=55) no lesion found in 9 cases**				
Occipital	8	1 (12.5)	7 (87.5)	$\chi^2=1.49$ df=2 *p=0.4745
Parietal	32	11 (34.38)	21 (65.62)	
Frontal and fronto-parietal	15	5 (33.34)	10 (66.66)	
Total=55		17 (30.9)	38(69.1)	

[Table/Fig-3]: Results of ELISA showing anti-Cysticercus antibodies in sera from patients with epilepsy with respect to location of lesions in brain. Percentage of sera tested positive under each location in brain was calculated based on total cases under each category.

** The lesion location was based on the CT features and of 55 cases presented with either one or more number of lesions in the brain; n= total number of cases.

* p-value was calculated based on non-parametric chi square analysis using Epi Info2001. No statistical difference was found when the relative distribution was compared among different parts of the brain (p=0.4745)

Number of lesions	Lesion type	No. (%) of sera tested positive by anti-Cysticercus IgG-ELISA
Single lesion	Vesicular	1 (5.88)
	Granular-nodular with inflammation	2 (11.76)
	Calcified cyst	8 (47.06)
>1 lesions	Calcified cysts	4 (23.45)
	Calcified + granular nodular stage (with inflammation)	2 (11.76)
	Total number positive cases with brain lesion	17 (100)

[Table/Fig-4]: Categories of the cystic lesion in brain based on CT scanning in cases those tested positive for anti-Cysticercus IgG antibodies by ELISA

** The type of lesion was based on the CT features following the criteria as described elsewhere [18]

DISCUSSION

NCC has been suspected as a major definable risk of epilepsy in our country [19]. Our study attempted for the first time reporting a series of cases from Odisha state in India where more than one fourth of the total number of epilepsy cases were found to be possibly due to the *T. solium* cysticercosis as evidenced based on the serum antibody reactivity in a validated commercial assay. Therefore, by detection of anti-Cysticercus IgG antibodies in serum the possible potentiality of NCC can be identified as an underlying cause of the recent onset of seizures as studied in our region. There were few earlier studies from other Indian provinces which showed similar levels of occurrence of cysticercosis as a cause of acquired seizure [20-22]. Among the series of epileptic cases NCC has been reported in 34.6% of patients with seizure of either single or recurrent types taken together as stated in a review; 59.2% of those presented with a single seizure, whereas 23.7% of those with recurrent seizure disorder [23]. We also noted majority of our patients experiencing at single episode of seizure before admission. Whereas only a few provided history of recurrence. The regional variation in the prevalence rates of epilepsy in India may be attributed to the actual prevalence of NCC in those communities as recommended elsewhere [24].

In the current screening study we found 'partial seizure' to be the major pattern of seizure as clinically evaluated. A predominance of generalized seizure was reported in earlier studies, and also significantly higher titre of antibodies was reported in confirmed

patients of NCC when compared with cases with other neurological disorders as well as normal controls subjects [25]. However, the trend of generalized seizure to be the major presentation in our NCC patients was contradictory to another Indian study [26]. This may be related to the patient population and the age group since it has been noticed that children with underlying NCC present more with focal seizure. Our patients showed headache as the major common presentation. Other presentations were vomiting, pallor, altered sensorium, and muscle weakness. There was no hemiparesis case in this series. No case of hydrocephalous was also recorded in the studied cases. Similar incidence was also reported in earlier Indian study [27].

There were predominantly adult than paediatric NCC cases in our case series; and predominantly more male patients than female ones. In other Indian study also maximum incidence of NCC was found in the age group between 21 and 30 y [28]. However, the scenario of seizure occurrences in Mexico has been reported to be more frequent in children compared to adult subjects [29]. The younger children might also acquire this infection as reported from a study conducted in Ecuador [30]. However this observation may not be universalized among paediatric population worldwide. An increase in the number of live vesicular cysts and a decrease in the number of dead and or degenerating cysticerci might correlate with aging process which was observed in earlier study; the immuno-endocrinological factors are thought to be playing a role in susceptibility and pathogenesis with respect to the age of the infected individual [31]. However, in our case series, no such trend has been observed. This may be clarified better if we compare the incidences among children and adults separately in future surveys. Hence, there is a clear message that NCC should be suspected in every case with seizures irrespective of the seizure pattern, or gender, or age group especially in endemic areas.

In the present series, the distribution of lesions in various parts of the brain showed parietal lobe being the major site of infection followed by frontal lobe; however the percentage of cases positive for anti-Cysticercus antibodies was comparable in the above two brain parts. In an earlier Indian study it was reported that a high probability of harboring either a single CT enhancing lesion, or single small cerebral calcific CT lesion, or multiple small cerebral calcific CT lesions in in a child with partial seizures with no obvious causation [32]. The same group also reported in another retrospective studies that NCC, small single cerebral calcific CT lesion and single CT enhancing lesion and together accounted for 40% of etiological factors of seizure [33]. No such assumption is made for adult NCC. However, it may be the similar situation in adult cases as seen in our study, because majority of the cases diagnosed to be positive by serology had either of the above three types of brain lesions on CT. Occurrence of disseminated cysticercosis with multiple cystic lesions in brain and other body locations have been reported from this province before [7]. In our patient series, cysts were not found in eye, subcutaneous tissue and other parts.

A sudden onset of the clinical signs in NCC is reported to coincide with intense inflammatory reactions to the dying or degenerating cyst within the brain [34]. The inflammatory reactions surrounding cystic lesions usually indicate the disease to be resulted primarily from host inflammatory response to the dying parasites [35]. Usually provoked seizures disappear following death of all the parasites, leaving a smaller percent of patients with residual epilepsy mostly due to formation of scars [36]. In the present article there was no scope of including the patient follow up data, however, at the first visit to clinics and in those who were advised for CT, calcified lesion in brain was observed in majority of cases which included either single lesion or multiple ones, and also either with or without perilesional inflammation.

The prevalence picture of an infection may be different in different studies even from different provinces within one country. For

instance, a recent report indicates that cysticercosis is not a major cause of epilepsy among the residents from Kerala state in India [37]. One factor affecting the above differences may be the variable sensitivity and specificity of each of the diagnostic tests used in different studies. Sensitivity of a diagnostic test while detecting anti-parasite antibodies may also be related to the number of lesions present in the brain. In the present case series, no significant association was observed between number of lesions in brain and the ELISA-positivity. However, there was more intense color development that may be due to release of more antigens from multiple larvae that results in mounting a stronger antibody response in the infected host as suggested in another study [38].

It is clear from various studies that NCC is one of the major differential diagnoses in every case with seizures particularly in the tropics where seroepidemiological studies may be very useful in control programs of this disease [25]. Thus NCC must be suspected in each case of seizures and a wide variety of neurologic disorders [39]. Most of the cases with NCC do not show the typical features on CT or MRI; many a times diagnosis of single brain lesion might also be a diagnostic challenge [40]. Hence, it is essential to choose an appropriate diagnostic modality for a proper diagnosis of the actual etiology in patients with seizures. Though, CT scan is a potent diagnostic tool compared to antibody detection however, the diagnostic decision is known to be better taken when both these modalities are considered together [41].

In our study, presence of antibodies was assessed among all the cases either having intracranial lesion or no lesion. Though presently employed commercial ELISA kit detects antibody against antigen of the parasite which are not defined, however it was found useful in confirming the presumptive cases as diagnosed based on clinical symptoms as well as imaging characteristics. Further study should be conducted to screen antibodies specific to more defined antigens of the larval cestode in order to better understand the clinical course or infection along with the information on viability of the parasite in human brain. In the present screening study there was one interesting case of a child having no abnormality on CT scanning of brain but was found to be positive by the ELISA. This patient might have *T. solium* larval infection elsewhere in the body that showed a detectable antibody level in serum when tested by ELISA, however the seizure occurrence may also be due to any other reason, or possibly the location of the parasite cyst in brain might not have been located by CT in this case.

In this series 4 of 14 patients with multiple lesions in brain disclosed prior history of anti-tubercular medication but there was confirmatory diagnosis of tuberculosis in none. Of these 1 tested positive for cysticercosis. Since intracranial tuberculoma and NCC are the most frequent granulomatous infections in the CNS, it may be true that they will appear similar on imaging [42]. Also, it may be necessary to rule out both these conditions because the lesions may also appear like brain tumor. So there is recommendation to consider tuberculoma and NCC in the differential diagnosis of brain tumour in tropical countries [43].

There was one case with a single cyst in the brain found to be negative for anti-Cysticercus antibodies by ELISA, but clinically suspected to be NCC strongly based on epidemiological information albendazole therapy was given with steroidal anti-inflammatory regimen. On a follow up visit this patient was later found to be positive on a repeat ELISA (OD₄₅₀ not shown). This indicates either the single lesion in brain was at very early stage of development hence no antibodies detected or there was undetectable limit of antibodies in the patient serum. Whereas the other case with a vesicular stage was found positive by antibody-ELISA. This may be possibly a transitional stage between vesicular to nodular granular stages of the parasite with a detectable limit of anti-parasite antibodies in serum. Otherwise there may be cysts elsewhere in the body that showed a higher antibody response. A rapid and appropriate diagnosis, revealing the

biological stage of this parasite would probably be of a great value in initiating an appropriate treatment modality. Also in the present study no detailed analysis of the social customs and related factors was done; which might be unique to this region and thereby those can be indirectly responsible for a high incidence rate of NCC in this province that need to be verified.

CONCLUSION

This is the first study from Odisha province where clinically suspected cases of NCC were confirmed serologically. Because this is only a preliminary attempt based on screening in hospital, however, this iceberg tip is tempting to explore the way up to its base provided a larger screening program is set with a scope to screen high risk community. It is warranted to consider a serological evidence of exposure to the parasite along with imaging characteristics for a confirmatory diagnosis of NCC in each and every case of active epilepsy irrespective of prior prevalence information in all yet to explore provinces in India, and must be reported in order to aid in quantifying its actual countrywide burden.

ETHICAL CONSIDERATIONS

This study was carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki).

ACKNOWLEDGEMENT

The authors acknowledge the Kalinga Institute of Medical Sciences Hospital authority for administrative assistance to conduct this study. This work was supported by the Indian council of Medical Research (ICMR) under Ad-hoc Research Grant (IRIS ID:2010-10530).

REFERENCES

- Bern C, Garcia HH, Evans C, Gonzalez AE, Verastegui M, Tsang VC, et al. Magnitude of the disease burden from neurocysticercosis in a developing country. *Clin Infect Dis*. 1999;29:1203-09.
- Garcia HH, Del Brutto OH, Nash TE, White AC Jr, Tsang VC, Gilman RH. New concepts in the diagnosis and management of neurocysticercosis (*Taenia solium*). *Am J Trop Med Hyg*. 2005;72:3-9.
- Montano SM, Villaran MV, Ylquimiche L, Figueroa JJ, Rodriguez S, Bautista CT, et al. (2005) Neurocysticercosis: association between seizures, serology, and brain CT in rural Peru. *Neurology*. 2005;65:229-33.
- Medina MT, Durón RM, Martínez L, Osorio JR, Estrada AL, Zúñiga C, et al. Prevalence, incidence, and etiology of epilepsies in rural Honduras: the Salamá Study. *Epilepsia*. 2005;46:124-31.
- Asnis D, Kazakov J, Toronjadze T, Bern C, Garcia HH, McAuliffe I, et al. Neurocysticercosis in the infant of a pregnant mother with a tapeworm. *Am J Trop Med Hyg*. 2009;81:449-51.
- Singhi P. Infectious causes of seizures and epilepsy in the developing world. *Dev Med Child Neurol*. 2011;53:600-09.
- Sharma A, Mahajan C, Rath GP, Mohapatra S, Padhy UP, Kumar L. Neurocysticercosis: Acute presentation and intensive care management of two cases. *Indian J Crit Care Med*. 2011;15(3):185-87.
- Ndimubanzi PC, Carabin H, Budke CM, Nguyen H, Qian YJ, Rainwater E, et al. A systematic review of the frequency of neurocysticercosis with a focus on people with epilepsy. *PLoS Negl Trop Dis*. 2010;4:e870.
- Rajshekhar V, Joshi DD, Doanh NQ, van De N, Xiaonong Z. *Taenia solium* taeniosis/cysticercosis in Asia: epidemiology, impact and issues. *Acta Trop*. 2003;87:53-60.
- Balaji JDM. Clinical and radiological profile of neurocysticercosis in South Indian children. *Indian J Paediatr*. 2011;78:1019-20.
- Rajshekhar V, Raghava MV, Prabhakaran V, Oommen A, Muliylil J. Active epilepsy as an index of burden of neurocysticercosis in Vellore district, India. *Neurology*. 2006;67(12):2135-39.
- Kashyap B, Das S, Jain S, Agarwal A, Kaushik JS, Kaur IR. Correlation between the clinico radiological heterogeneity and the immune-inflammatory profiles in paediatric patients with neurocysticercosis from a tertiary referral centre. *J Trop Paediatr*. 2012;58(4):320-23.
- Prasad KN, Prasad A, Verma A, Singh AK. Human cysticercosis and Indian scenario: a review. *J Biosci*. 2008;33:571-82.
- Mishra BN, Swain SP. Psychiatric morbidity following neurocysticercosis. *Indian J Psychiatry*. 2004;46(3):267-68.
- Mohapatra RN, Pattanaik JK, Satpathy SK, Joshi S. Isolated and silent spinal neurocysticercosis associated with pseudotumor cerebri. *Indian J Ophthalmol*. 2008;56(3):249-51.
- Del Brutto OH. Diagnostic criteria for neurocysticercosis, revisited. *Pathog Glob Health*. 2012;106(5):299-304.
- Sahu PS, Parija SC, Narayan SK, Kumar D. Evaluation of an IgG-ELISA strategy using *Taenia solium* metacestode somatic and excretory-secretory antigens for

- diagnosis of neurocysticercosis revealing biological stage of the larvae. *Acta Trop*. 2009;110:38-45.
- [18] Zee CS, Go JL, Kim PE, DiGiorgio CM. Imaging of neurocysticercosis. *Neuroimaging Clin N Am*. 2000;10:391-407.
- [19] Raina SK, Razdan S, Pandita KK, Sharma R, Gupta VP, Razdan S. Active Epilepsy as Indicator of Neurocysticercosis in Rural Northwest India. *Epilepsy Research and Treatment*. 2012;2012:802747. doi:10.1155/2012/802747.
- [20] Hussain J, Srinivasan S, Serane VT, Mahadevan S, Elangovan S, Bhuvaneshwari V. Cranial computed tomography in partial motor seizures. *Indian J Paediatr*. 2004;71:641-44.
- [21] Thakur LC, Anand KS. Childhood neurocysticercosis in south India. *Indian J Paediatr*. 1991;58:815-19.
- [22] Udani V. Paediatric epilepsy -- an Indian perspective. *Indian J Paediatr*. 2005;72:309-13.
- [23] Singh G, Singh P, Singh I, Rani A, Kaushal S, Avasthi G. Epidemiologic classification of seizures associated with neurocysticercosis: observations from a sample of seizure disorders in neurologic care in India. *Acta Neurol Scand*. 2006;113:233-40.
- [24] 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(1):37-40.
- [25] Sharma P, Ganguly NK, Mahajan RC, Malla N. Clinical and laboratory analysis of neurocysticercosis in children. *Indian J Med Microbiol*. 1995;13:92-94.
- [26] Singhi PD, Baranwal AK. Single small enhancing computed tomographic lesions in indian children--II. Clinical features, pathology, radiology and management. *J Trop Paediatr*. 2001;47:266-70.
- [27] Kalra V, Suri M, Jaikhanani BL. A profile of childhood neurocysticercosis. *Indian J Paediatr*. 1994;61:33-42.
- [28] Kotokey RK, Lynrah KG, De A. A clinico-serological study of neurocysticercosis in patients with ring enhancing lesions in CT scan of brain. *J Assoc Physicians India*. 2006;54:366-70.
- [29] Gaffo AL, Guillén-Pinto D, Campos-Olazábal P, Burneo JG. Cysticercosis as the main cause of partial seizures in children in Peru. *Rev Neurol*. 2004;39:924-26.
- [30] Del Brutto OH. Neurocysticercosis in a 2-year-old boy infected at home. *Pathog Glob Health*. 2012;106:122-23.
- [31] Fleury A, Bouteille B, Garcia E, Marquez C, Preux PM, Escobedo F, et al. Neurocysticercosis: validity of ELISA after storage of whole blood and cerebrospinal fluid on paper. *Trop Med Int Health*. 2001;6:688-93.
- [32] Murthy JM, Yangala R. Etiological spectrum of localization-related epilepsies in childhood and the need for CT scan in children with partial seizures with no obvious causation--a study from south India. *J Trop Paediatr*. 2000;46:202-06.
- [33] Murthy JM, Yangala R. Etiological spectrum of symptomatic localization related epilepsies: a study from South India. *J Neurol Sci*. 1998;158:65-70.
- [34] Marquez-Monter H. Cysticercosis. In: Marcial-Rojas RA, editor. Pathology of Protozoal and Helminthic Diseases. Baltimore: Williams and Wilkins; 1971. Pp. 592-617.
- [35] Pal DK, Carpio A, Sander JW. Neurocysticercosis and epilepsy in developing countries. *J Neurol Neurosurg Psychiatry*. 2000;68:137-43.
- [36] White AC Jr. Neurocysticercosis: a major cause of neurological disease worldwide. *Clin Infect Dis*. 1997;24:101-13.
- [37] Cherian A, Syam UK, Sreevidya D, Jayaraman T, Oommen A, Rajshekhar V, et al. Low seroprevalence of systemic cysticercosis among patients with epilepsy in Kerala--South India. *J Infect Public Health*. 2014;7(4):271-76.
- [38] Zea-Vera A, Cordova EG, Rodriguez S, Gonzales I, Pretell EJ, Castillo Y, et al. Cysticercosis Working Group in Peru. Parasite antigen in serum predicts the presence of viable brain parasites in patients with apparently calcified cysticercosis only. *Clin Infect Dis*. 2013;57(7):e154-59.
- [39] Singhi P, Singhi S. Neurocysticercosis in children. *J Child Neurol*. 2004;19:482-92.
- [40] Giri S, Parija SC. A review on diagnostic and preventive aspects of cystic echinococcosis and human cysticercosis. *Trop Parasitol*. 2012;2:99-108.
- [41] Sahu PS, Seepana J, Padela S, Sahu AK, Subbarayudu S, Barua A. *Rev Inst Med Trop Sao Paulo*. 2014;56(3):253-58.
- [42] Lu Z, Zhang B, Qiu W, Hu X. Disseminated intracranial tuberculoma mimicking neurocysticercosis. *Intern Med*. 2011;50(18):2031-34.
- [43] Gothi R. Consider tuberculoma and cysticercosis in the differential diagnosis of brain tumour in tropical countries. *BMJ*. 2013;347:f6604.

PARTICULARS OF CONTRIBUTORS:

1. Assistant Professor, Department of Immunology Laboratory, School of Biotechnology, KIIT University, Bhubaneswar, Odisha, India. Division of Pathology, School of Medicine, International Medical University, Kuala Lumpur, Malaysia.
2. Associate Professor, Department of Internal Medicine, Kalinga Institute of Medical Sciences, KIIT University, Bhubaneswar, Odisha, India.
3. Consultant Neurologist, Department of Neurology, Kalinga Institute of Medical Sciences, KIIT University, Bhubaneswar, Odisha, India.
4. Assistant Professor, Department of Internal Medicine, SCB Medical College and Hospital, Cuttack, Odisha, India.
5. Professor, Department of Internal Medicine, SCB Medical College and Hospital, Cuttack, Odisha, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Priyadarshi Soumyaranjan Sahu,
School of Biotechnology, KIIT University, Bhubaneswar, Odisha-751024, India.
E-mail : priyadarshi_sahu@yahoo.com

Date of Submission: **Dec 19, 2014**Date of Peer Review: **Feb 03, 2015**Date of Acceptance: **Mar 14, 2015**Date of Publishing: **May 01, 2015**Date of Last Update: **Feb 01, 2016****FINANCIAL OR OTHER COMPETING INTERESTS:** None.