Open Access
CODEN: IJBNHY
ISSN: 2278-778X
International Journal of Bioassays

CASE REPORT

Neurocysticercosis presenting as Meningoencephalitis

Sourajit Routray, Rajib Ray, Radha Tripathy and Raj Kumar Paul*
Department of Pediatrics, Hi-Tech Medical College and hospital, Bhubaneshwar, Odisha, India

Received for publication: November 29, 2014; **Revised:** December 11, 2014; **Accepted:** January 28, 2015

Abstract: Human neurocysticercosis, the infection of the nervous system by the larvae of *Taenia solium*, is a cause of epileptic seizures and other neurologic morbidity worldwide. The disease occurs when humans become intermediate hosts of *Taenia solium* by ingesting its eggs from contaminated food or, most often, directly from a taenia carrier by the fecal-to-oral route. 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. The most common clinical manifestation being the seizures (70-90%), but many patients present with focal deficits, intracranial hypertension, or cognitive decline. The accurate diagnosis of neurocysticercosis is possible after interpretation of clinical data together with findings of neuroimaging studies and/ or results of immunological tests. Encephalitis is the inflammation of brain parenchyma presenting as acute febrile illness with altered level of consciousness, confused behavioral abnormality and depressed level of consciousness ranging from mild lethargy to coma and evidence of either focal or diffused neurological sign and symptoms. Parenchymal brain cysticerci in the acute encephalitic phase have been recognized since the first reports of CT in patients with neurocysticercosis. These lesions were described as focal low densities surrounded by oedema and ring-like enhancement after giving contrast medium. The abnormal enhancement of these lesions were related to the breakdown in the blood-brain barrier caused by the inflammatory reaction around dying cysticerci. We report a case of 10-year-old female child presenting with fever, headache and altered sensorium. This case report may help the practitioners to identify this disease with different presentations, some with fatal presentation, so that needful imaging and management would be instituted at the earliest keeping in mind that Anticysticercal drugs are contraindicated in patients with cysticercotic encephalitis because they may exacerbate the inflammatory response within the brain parenchyma.

Key words: Fever; seizures; altered sensorium; Antiepileptic drugs; steroid; Anticysticercal drug

INTRODUCTION

Neurocysticercosis (NCC) is a pleomorphic parasitic disease that shows different patterns of clinical manifestations in almost every patient. There are different presentations in NCC, ranging from mild headaches or sporadic seizures which may progress to intractable epilepsy, intracranial hypertension, coma and death. The severity and the variation of disease expression depends on the individual differences in the number and location of the lesions within the Central Nervous system as well as the differences in the degree of the host's immune reaction against cysticerci (1). Several studies have been reported focusing on the severe forms of the disease. Our case is reported to light up towards the use of AED for NCC induced encephalitic seizure and the improvement of a child without the use of anti-cysticercal drug as a treatment modality.

CASE REPORT

A 10-year-old female child weighing 30kg, belonging to a Hindu Banjara family, born after a full term normal vaginal delivery admitted to our department with the history of fever, headache and altered sensorium of 5-day duration without convulsion and vomiting. The fever was continuous in nature (magnitude of 100°-102°C) not associated with chills and rigor. The child had no previous history of convulsion and there was no family history of convulsion. There was no history of contact with TB and ear discharge. The child was treated with antipyretics and oral antibiotics before admission. On examination, she was febrile (102°F) with Respiratory rate of 28/min, Pulse rate of 120/min regular, and Blood pressure of 110/70mm Hg, with no lymphadenopathy. Neurological examination revealed altered sensorium with GCS of 12/15, without cranial nerve involvement. Motor system examination revealed hypotonia of all four limbs with normal DTR with plantar extensor on both sides. Sensory system normal. Meningeal signs were positive. All other systems revealed no clinical abnormality.

Hospital Course

Routine hematological and biochemical parameters were within normal limits. Urine routine examination and Stool examination revealed normal findings. Liver function tests and Renal Function Test were normal. RBS-

***Corresponding Author:**
Dr. Raj Kumar Paul,
Department of Pediatrics,
Hi-Tech Medical College and hospital,
Bhubaneshwar, Odisha, India.

102mg/dl; with normal calcium and electrolyte levels. Antinuclear antibody, viral markers for human immunodeficiency virus (HIV), and dengue, rapid malarial test, Mantoux test, gastric lavage for AFB were negative. Blood and urine culture were sterile. Chest X-ray revealed no abnormality. CSF analysis showed clear fluid, and pleocytosis with predominant Lymphocyte and elevated protein and normal glucose. CSF-PCR for Herpes DNA was negative.

The child developed seizure after 1 day of admission, generalized tonic-clonic convulsion, first episode lasted for over 10mins. The child was managed with injectable anticonvulsants starting with Lorazepam and Fosphenytoin, but seizures continued. The child deteriorated with Neurological examination revealing GCS of 10/15. So, another anticonvulsant was added (Phenobarbitone). CECT-revealed multiple dot calcified foci seen in both cerebral hemispheres without any significant contrast enhancement. Few hypo dense areas seen in gray white interface [Fig.1] CEMR revealed multiple sub centimeter ring lesion with eccentric dots seen in both cerebral hemispheres which were not evident on non-contrast T1W images. Significant T2W hyper intense edema seen around few ring enhancing lesions. No meningeal enhancement and no basal exudates [Fig.2,3]. Finally, it was diagnosed as Encephalitic stage of neurocysticercosis.

MRI: T1Weighted demonstrating parenchymal cyst with protoscolices.

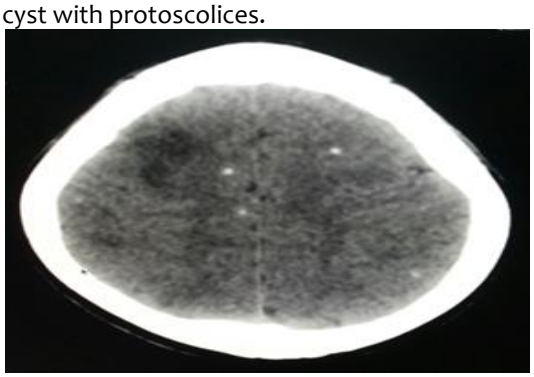

Figure 1: CECT images showing multiple dot calcified foci seen in both cerebral hemispheres without any significant contrast enhancement

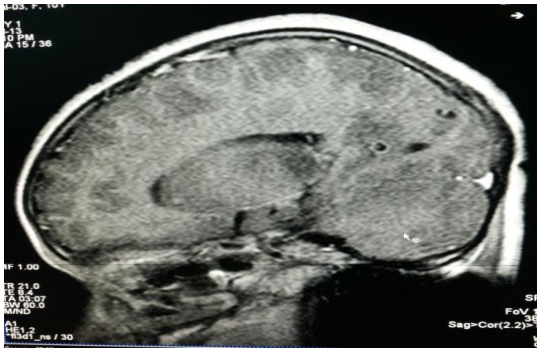

Figure 2: Saggital section

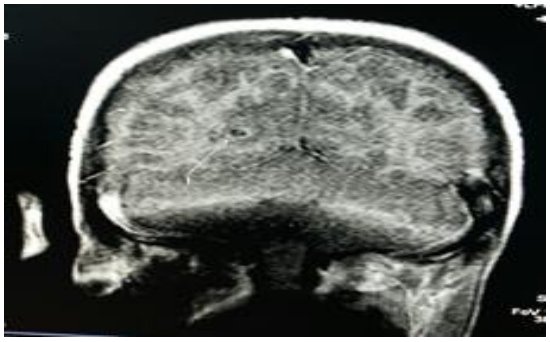

Figure 3: Coronal section

The child was also started on Dexamethasone and Injection Mannitol. The child improved after 3 days of injection with deaxmethasone and was continued on same medications and she gradually improved after 11 days of admission. The child is presently doing well and continuing with oral phenytoin.

DISCUSSION

Encephalitis is the inflammation of brain parenchyma presenting as acute febrile illness with altered level of consciousness, confused behavioral abnormality and depressed level of consciousness ranging from mild lethargy to coma and evidence of either focal or diffused neurological sign or symptoms. Focal or generalized seizure occur in many patients with encephalitis. ([2]) Commonest cause is Viral. Other causes are: Parasitic- Neurocysticercosis, Protozoal- *T. gondii*, *malaria*, Fungal-*Cryptococcus*, Bacterial-*Tubercular, meningitis*, Fungi- *Cryptococcus*. ([3])

Neurological manifestations of cysticercosis vary and are related to the size, number and location of the parasites. Epilepsy is the most common clinical manifestation occurring in almost 70-90% of patients with NCC ([4]) and is most commonly partial siezure. [84-87% cases]([4,5]).

Acute encephalitic stage is a parenchymal neurocysticercosis belonging to colloidal stage. It occurs due to release of metabolite from died scolex in the cyst and breakdown of blood brain barrier, resulting in intense host immune reaction. Radiologically, it is described as low density, surrounded by edema and ring like enhancement after giving contrast ([7,8]). In recent years, it became apparent that the prognosis of patients with multiple parenchymal brain cysticerci in the acute encephalitic phase may be ominous. This form of the disease, called cysticercotic encephalitis, represents a severe form of neurocysticercosis in which the nervous system is harmed by the intense immune reaction mounted by the host against a massive cysticerci infestation of the brain parenchyma.([6]) [Study on Fifty four patients with a single parenchymal brain cysticercus in the acute encephalitic phase by Oscar H Del Brutto]

Table 1: Diagnostic criteria and degrees of diagnostic certainty for cysticercosis (Modified from: [9]).

Diagnostic Criteria
Absolute
i. Histologic demonstration of the parasite from biopsy of a brain or spinal cord lesion.
ii. Evidence of cystic lesions showing the scolex on neuroimaging studies.
iii. Direct visualization of subretinal parasites by fundoscopic examination.
iv. Spontaneous resolution of small single enhancing lesions.
Major
i. Evidence of lesions highly suggestive of neurocysticercosis on neuroimaging studies.
ii. Positive serum immunoblot for the detection of anticysticercal Antibodies.
iii. Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel.
Minor
i. Evidence of lesions suggestive of neurocysticercosis on neuroimaging studies.
ii. Presence of clinical manifestations suggestive of neurocysticercosis.
iii. Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens.
iv. Evidence of cysticercosis outside the central nervous system.
Epidemiologic
i. Individuals coming from or living in an area where cysticercosis is endemic.
ii. History of frequent travel to disease-endemic areas.
iii. Evidence of household a contact with *T. solium* infection.
Degrees of diagnostic certainty
Definitive
i. Presence of one absolute criterion.
ii. Presence of two major plus one minor or one epidemiologic criteria.
Probable
Presence of one major plus two minor criteria
Presence of one major plus one minor and one epidemiologic criteria
Presence of three minor plus one epidemiologic criteria.

In our case, the child presented with fever, headache, malaise and seizure with positive findings of multiple neurocystic lesions in the brain parenchyma, evidence of cystic lesions showing the scolex on neuroimaging study, demonstrating the features of Encephalitic Neurocysticercosis.

There is no universally agreed single protocol for the treatment of NCC. Consensus guidelines for the treatment of NCC recommend an individualized approach [Nash *et al.*, 2006]. Characterization of the disease in terms of viability of cysts, degree of the host's immune response to the parasite, and location and number of lesions is important for rational therapy ([10]). The natural history of parenchymal lesions is to resolve spontaneously with or without antiparasitic drugs. Most children present with solitary parenchymal cyst that resolve with or without therapy whereas other forms are less common. The contrast multiple lesion and complex presentation are typically seen in adult. ([11]). Therapy usually include a combination of symptomatic and cysticidal drugs. Surgery also plays a role in the management of some patients ([12]). Anticysticercal drugs are contraindicated in patients with cysticercotic encephalitis because they may exacerbate the inflammatory response within the brain parenchyma. These drugs may exacerbate the syndrome of intracranial hypertension observed in patients with cysticercotic encephalitis. The administration of steroid is mandatory to avoid the hazard of a cerebral infarct (Dexamethasone 4.5 to 12mg/day, can increase upto 30mg/day)([1°]). If the requirement of steroid is prolonged, replace Dexamethasone with Prednisolone (1mg/kg/day). In cases with disseminated lesions and extensive cerebral oedema, steroids may be required for a prolonged period. In patients with ventricular cysts, the use of cysticidal drugs should be individualized. While albendazole successfully destroys many ventricular cysts, the inflammatory reaction

may cause acute hydrocephalus if the cysts are located within the fourth ventricle or near the Foramen of Monro. Finally, patients with calcifications alone should not receive cysticidal drugs since these lesions represent already dead parasites (Garcia *et al.,*[10]). The administration of a single first-line antiepileptic drug usually results in control of seizures in patients with neurocysticercosis-related epilepsy.

Recurrence of seizures after AED withdrawal is correlated with the presence of multiple lesions prior to starting cysticidal therapy, and persistence or calcification of lesions after therapy [Goel *et al.,* 2010; Singhi *et al.,* 2003b; Del Brutto, 1994]. The optimal duration of AED therapy has been a matter of debate. Although the usual practice has been to use AED for 2 years seizure-free interval, shorter durations of AED have been proposed. In a randomized study of 106 children with SSECTL, seizure recurrence after AED therapy for 1 year versus 2 years seizure-free interval was not significantly different and correlated significantly with persistence or calcification of lesions and an abnormal EEG at the time of withdrawal [*Singhi et al., 2003b*]. It seems prudent to withdraw anticonvulsants after a 1-year seizure-free interval in cases where the lesion has disappeared and the EEG has normalized; longer durations are needed for those with persistent or calcified lesions.

The motto of presenting this case is to enlighten the pediatricians and general practitioners regarding the clinical entity of encephalitic state of neurocysticercosis and their outcome without the use of anticysticercal drug.

REFERENCE

1. Del Brutto OH, Sotelo J. Neurocysticercosis: an update. Rev Infect Dis 1988; 10:1075-87.

2. Karel L Roos, Kenneth L Tyler Ch: 381 Meningitis, Encephalitis, Brain abscess and Empyema. Pg: 3421: Harrison principles of Internal Medicine. 18th edition.

3. OP Ghai, Vinod K Paul, Arvind Bagga 7th ed: Neurocysticercosis; 546-47.

4. Del Brutto OH, Santibañez R, Noboa CA, *et al.,* Epilepsy due to neurocysticercosis: analysis of 203 patients. Neurology 42:389–392, 1992.

5. Medina MT, Rosas E, Rubio-Donnadieu F, *et al.,* Neurocysticercosis as the main cause of late-onset epilepsy in Mexico. Arch Intern Med 150:325– 327, 1990.

6. Del Brutto OH, Sotelo J. Neurocysticercosis: an update. Rev Infect Dis 1988; 10: 1075-87.

7. Mervis B, Lotz JW. Computed tomography (CT) in parenchymatous cerebral cysticercosis. Clin Radiol 1980; 31:521-8.

8. Rajshekhar V. Albendazole therapy for persistent, solitary cysticercus granulomas in patients with seizures. Neurology 1993; 43: 1238-40.

9. OH Del Brutto, V Rajshekhar, AC White *et al.,* Proposed diagnostic criteria for neurocysticercosis, Neurology, vol. 57, no. 2, pp. 177–183, 2001.

10. HH Garc´ıa, CAW Evans, TE Nash *et al.,* Current consensus guidelines for treatment of neurocysticercosis, Clinical Microbiology Reviews, vol. 15, no. 4, pp. 747–756, 2002.

11. Ronald Blanton Ch 295: Cysticercosis: pg: 1235; Nelson Textbook of Pediatrics 19th edition.

12. TE Nash, G Singh, AC White *et al.,* Treatment of neurocysticercosis: current status and future research needs, Neurology, vol. 67, no. 7, pp. 1120–1127, 2006.

Cite this article as:
Sourajit Routray, Rajib Ray, Radha Tripathy and Raj Kumar Paul. Neurocysticercosis Presenting As Meningoencephalitis - A Case Report, International Journal of Bioassays, 2015, 4 (03), 3697-3700.

Source of support: Nil
Conflict of interest: None Declared