MED-PHOENIX: JOURNAL OF NATIONAL MEDICAL COLLEGE

CASE REPORT

NEUROCYSTICERCOSIS AS A FIRST SEIZURE IN A 5 YEARS OLD CHILD: A CASE REPORT

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Date of Submission : April 02, 2024
Date of Acceptance : April 15, 2024
Date of Publication : June 25, 2024

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Citation:
Chaudhary J, Niroula S.Neurocysticercosis as a first seizure in a 5 years old child: A case report. Medphoenix. 2024;9(1):33-36.
DOI:https://doi.org/10.3126/medphoenix.v9i1.67242
Conflict of interest: None, ***Funding:*** None

Publisher: *National Medical College Pvt. Ltd.*
MedPhoenix - Journal of National Medical College (JNMC); 2024,9(1), available at www.jnmc.com.np
ISSN:2631-1992 (Online); ISSN:2392-425X (Print)

ABSTRACT

Introduction: *Neurocysticercosis (NCC) is a parasitic infection of the central nervous system caused by Taenia solium, a pig tapeworm. Humans acquired the infection via contaminated food, water, and poor hygiene. NCC has different clinical manifestations based on their location, like focal deficits, headaches, seizures, hydrocephalus, etc. Diagnosis can be made based on radio-imaging findings, supported by laboratory assays. The treatment plan includes the control of symptoms with the initiation of anti-parasitic medications, corticosteroids, and surgery as indicated based on the location of the NCC. NCC is a preventable disease, proper hygiene and sanitation, intake of properly cooked meals are helpful. Early diagnosis and early initiation of treatment play a crucial role in the management of NCC with good outcomes.*

Keywords: *Neurocysticercosis, Seizure, Taenia solium*

INTRODUCTION

Neurocysticercosis (NCC) is an acquired, preventable parasitic infection of the central nervous system caused by larval cysts of the tapeworm (Taenia solium), which usually presents with seizures.[1, 2] T. solium (pork tapeworm) completes its life cycle in two hosts, pigs and humans, where humans acquired it by taking contaminated food/water and practicing poor hygiene. [1] NCC has four stages: the first vesicular stage with cysts and scolex; the second colloidal stage with ring enhancement lesion and edema; the third nodular-granular stage with decreased enhancement and edema along with the initiation of calcification; and the and the fourth stage is the calcified stage with calcification on a magnetic resonance imaging (MRI) brain or computed tomography (CT) scan.[3] Based on anatomic location, clinical presentation, and radiologic appearance, NCC can be parenchymal, intra-ventricular, subarachnoid, spinal, or ophthalmic, with significant differences in the prognosis and management. [3, 4] Parenchymal NCC is the most frequent location of NCC; it accounts for ~70% of cases, typically presents with seizures. [1]

The diagnosis of NCC can be made based on the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH) recommendations:

- Clinical presentations of seizures and raised intracranial pressure.
- History, examination, and neuroimaging studies [MRI brain and non-contrast CT scan].
- Enzyme-linked immunotransfer blot test in suspected patients.[4]

Management of NCC includes symptoms control, the initiation of antiparasitic therapy along with corticosteroids therapy for perilesional edema, suppression of parasitic inflammation, and surgery as indicated for hydrocephalus as in intra-ventricular NCC. [2]

In this case report, we present a case of a 5-year-old child without a significant birth history or neonatal infections who presented to us with the first episode of a seizure, diagnosed as a case of NCC.

CASE SUMMARY

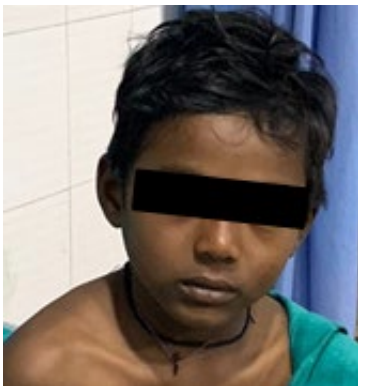


Figure 1: Patient with Neurocysticercosis

5 years/male, without any co-morbidities from Parsa, Nepal, presented to the National Medical College & Teaching Hospital (NMCTH), Birgunj; emergency with complaints of:

- Abnormal body movements for 7 hours prior to presentation
- Vomiting for 7 hours prior to presentation

According to the informant, the patient was apparently well 7 hours prior to presentation, when he developed the first episode of abnormal body movements of <30 seconds, associated with tightening of bilateral upper and lower limbs, up rolling of the eyes, frothing from the mouth with a decrease in level of consciousness that occurred without any stimulation. There were a total four episodes, where the first three episodes were tonic, and the last episode was generalized tonic and clonic, and the duration of abnormal body movements was increasing in each episode. History of one episode of vomiting after 10 minutes following the first episode of abnormal body movements, non-projectile, non-bilious, amount equal to a size of half a cup, i.e., ~100–125 ml, containing ingested food particles without blood stain.

There was no history of fever, cough, contact with tuberculosis patients, family history of similar illnesses, or routine medication use. There is no significant birth history or history of neonatal infections. There is no history of previous hospital admissions. Growth and development history were appropriate to age. The vaccination status was up-to-date. He was known to occasionally consume pork. There was a history of poor sanitation, personal hygiene, and oral hygiene.

I have no history of such episodes/similar illnesses in the past.

On general examination, the patient was well-built, ill looking. GCS: E4V3M6, Pallor: present.

Vitals: Pulse rate: 121 beats/minute, regular, normal in volume and character, no radio-radial or radio-femoral delay. All peripheral pulses are palpable.

Blood pressure: 100/60 mm of Hg, right arm, supine position.

Temperature: 97.6° F, right axillary

Respiratory rate: 21 breaths per minute, regular

Spo2: 99% in room air; GRBS: 175 mg/dl

Body weight: 20 kg.

On systemic examination: respiratory, cardiovascular, and CNS, per abdominal examination were within normal limits.

Lab investigations in the emergency department are as follows:

Table 1: Complete blood count report

Blood Count	Values	Normal Range	Remarks
Hemoglobin	10.8 gm%	13-18	Low
TLC	16,400 cells/Cumm	4,000-11,000	High
DLC	N60 (%) L32 (%) M02 (%) E06 (%)	40-70 20-45 02-10 01-06	
PCV	33 %	37-47	Low
MCV	81 fl	76-98	
MCH	27 pg	26-36	
MCHC	33 gm/dl	31-37	
Platelet count	470,000 cells/Cumm	150,000-400,000	High
C-Reactive Protein	2.1 mg/L	<6	

Blood count showed: Normochromic, normocytic anemia (10.8 gm%) with leukocytosis

ECG: Sinus Rhythm, Chest X-ray: Normal findings.

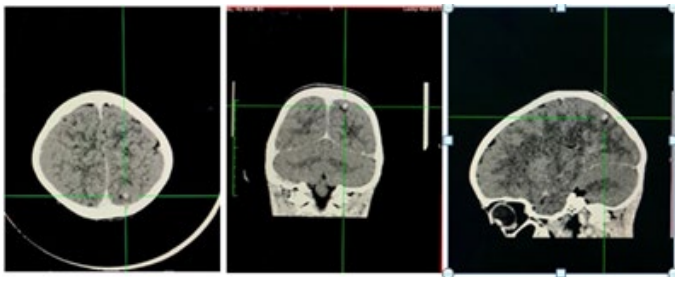
Table 2: Electrolytes report

Tests	Values	Normal Range	Remarks
Sodium (Na^+)	134 mEq/L	135-150	Low
Potassium (K^+)	3.54 mEq/L	3.5-5.5	
Blood Urea	29mg/dl	10-45	

Serum Creatinine	0.40 mg/dl	0.6-1.3	
Ionized calcium	1.11 mmol/L	1.05-1.38 (2-18 years)	

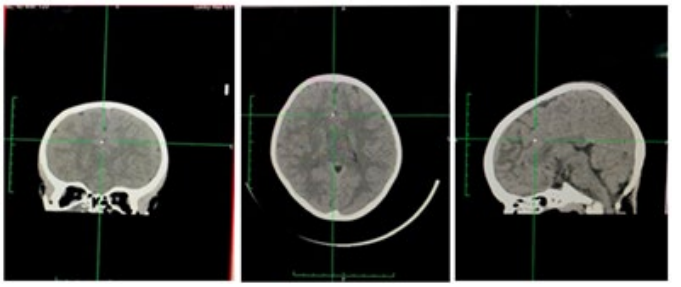
Blood test showed: Mild Hyponatremia (134 mEq/L).

Non-contrast Computed tomography (NCCT) scan was done to find out the cause of seizure.



Coronal view Transverse view Sagital view

Figure 2: CT Head with well-defined, intra-axial, hypodense lesions with surrounding hyperdense rim and eccentric hyperdense foci (representing scolex) noted in the grey and white mater junction of left parietal lobe associated with mild perilesional edema. On post contrast study a fore, mentioned lesion shows wall enhancement-likely colloidal vesicular stage of NCC.



Coronal view Transverse view Sagittal view

Figure 3: CT Head with well-defined intra-axial hyperdense focus (HU 160) measuring approximately 2.6mm noted in right frontal lobe without surrounding edema-likely; nodular calcified stage of NCC.

Based on history, examination, lab tests, and radiological findings, the diagnosis of "Parenchymal Neurocysticercosis (colloidal vesicular stage; nodular calcified stage)" was established. The patient and patient party were counseled about patient status and a further plan of management. Further, the patient was screened for latent tuberculosis (tuberculin skin test), which was negative. Enzyme-linked immunosorbent assay (ELISA) IgG for NCC was positive. Fundoscopic examination revealed no significant findings. The patient was treated with an anti-convulsant, anti-inflammatory, and anti-parasitic medication regimen. The child received 5 mg/kg/day of phenytoin as the anti-convulsant for the control of seizures. As an anti-inflammatory, prednisone was given intravenously at a starting dose of 2 mg/kg/day before the initiation of antiparasitic medication. The anti-parasitic medication Albendazole was started at a dose of 15 mg/kg/day, divided into two daily doses for 14 days, along with supportive measures.

The patient improved, and was hemodynamically stable. The patient was discharged on an oral anti-convulsant, oral corticosteroid, and oral anti-parasitic regime of the same dose after 5 days of hospital stay. Two weeks follow up reports of liver function tests and a complete blood count showed no abnormalities. One month follow up showed no seizures or any symptoms. The patient was scheduled for a brain MRI in six months. The patient party was counseled for the screening of NCC in the family members in follow up.

DISCUSSION

NCC is a clinical variant of cysticercosis affecting the central nervous system.[2] NCC primarily affects the poor and rural areas, occurring where humans are in close proximity to pigs and where proper fecal disposal is poor.[3]At the time of the diagnosis, the patient was 5 years old, indicating recent contamination. In our case, the patient presented with a complaint of seizure that is explained by the presence of the parenchymal NCC, which is the most common presentation.[1] Seizure is precipitated by the presence of inflammation in the brain parenchyma. The presence of calcified cysts and peri cystic inflammation, where cyst degeneration induces an inflammatory response to kill intracellular parasites, releasing cytokines leading to blood brain barrier leakage to facilitate seizure activity,.[5] Parenchymal NCC is relatively easy to manage with a better prognosis, except in multiple lesions/heavy infections. Viable cysts degenerate and then disappear or remain as a calcified scar. The diagnosis of NCC is supported by the epidemiological distribution of the disease, clinical manifestations, radio-imaging, and other supportive laboratory investigations.[4, 6] In our case, radio-imaging findings were suggestive of NCC with two different stages of NCC. A CT scan of the brain is economical, more accessible today. The treatment of choice for this patient was a combination of anti-convulsant, anti-inflammatory, and anti-parasitic drugs. . Phenytoin was given at 5 mg/kg/day as an anti-convulsant. Prednisolone, as an anti-inflammatory at 2 mg/kg/day, started before the first dose of anti-parasitic and was given for 2 weeks. Albendazole, an anti-parasitic with a dosage of 15 mg/kg/day per orally divided twice daily for 14 days, was given in our case with a good response and improvements in symptoms.[4] In the general population, proper hygiene and sanitation, safe drinking water, and well-cooked meals can prevent the disease. Mass chemotherapy for tapeworm carriers, mass treatment of pigs, and improved community health and sanitation can decrease or even eliminate the disease

transmission of NCC.[3]

CONCLUSION

NCC is a preventable disease. History, examination, and radio-imaging findings, along with supportive laboratory investigations, help in diagnosis. NCC must be one of the differential diagnoses of seizures, especially in endemic areas. Proper personal hygiene, sanitation, and the practice of healthy food intake and safe drinking water can help prevent the disease as well as decrease its burden. Screening of other family members is vital to detecting NCC in its early stages. Early initiation of medications with other supportive measures improves the patient's symptoms with good outcomes.

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