Parenchymal Neurocysticercosis: A Case Study on Diagnostic and Treatment Approach

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ABSTRACT

Neurocysticercosis (NCC) is the one of the endemic diseases mostly occurring in Africa, Asia and Latin America making it a major cause of acquired epilepsy in these regions. Transmitted through *Taenia solium* (the pork tapeworm), contaminated food and water are some of the major sources of infection. Clinical manifestations mostly include seizures, headache and loss of vision and in some cases weakness of extremities. Although a precise incidence of NCC in India is not documented, number of cases has been reported especially in rural areas of the country and among economically backward persons due to unsanitary living conditions. In this case report, we summarize regarding a patient who developed parenchymal Neurocysticercosis after a history of travel that was managed based on guidelines and symptomatic treatment concomitant with supportive care.

Keywords: Neurocysticercosis, T. solium, Epilepsy, Anti-helminthic, Albendazole.

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Received: 21-01-2025; Revised: 13-03-2025; Accepted: 15-05-2025.

INTRODUCTION

Neurocysticercosis (NCC) is the most common type of helminthic infection affecting the central nervous system and the meninges by the larval stage of *Taenia solium*, the pork tapeworm. In NCC, two hosts are involved-humans and pigs in which humans act as both definitive and intermediate host whereas pigs act as only intermediate host. In most case, human cysticercosis occurs when human ingest the eggs of *T. solium* from contaminated food or water. On reaching the human intestine, these eggs become oncospores which are carried through bloodstream into the CNS and other tissues, where they develop into larval forms called cysterci.^{1,2}

The cysticerci may be located in the brain parenchyma, subarachnoid space, ventricular system or spinal cord. The area of location together with number of lesions and the intensity of immune reaction against parasites determines patient's clinical manifestations. Seizures are the most common clinical manifestations, whereas people may also experience headache, intracranial hypertension, focal deficits and cognitive decline. NCC can be diagnosed based on neuroimaging studies along with immunological test like ELISA for detecting the anti-cysticercal antibodies.



DOI: 10.5530/ijopp.20250341

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Publishing Partner : Manuscript Technomedia. [www.mstechnomedia.com]

The first line treatment should always be in treating the clinical symptoms of patients such as seizures using anti-epileptics, anti-inflammatory drugs and corticosteroids. Surgical procedure such as cystextraction, ventricular shunt placement may also need in some cases. The use of cysticidal agents such as praziquantel (recommended dose is 50 mg/kg/day for 10 to 15 days) and albendazole (recommended doses varies between 15 and 30 mg/ kg/day, to be taken in 2 doses per day, for 8 to 10 days) helps to change the prognosis of NCC and reduce the overall burden of disease.³

CASE DESCRIPTION

A 49-year-old male patient was presented to the emergency department of Lourdes Hospital, Kochi with complaints of multiple episodes of seizure with tongue bite and frothing from mouth. On examination, he was stuporous and febrile with Glasgow coma score of 7/15 ($E_1V_1M_5$) and temperature of 38.1°C. The accompanying informant reported that the patient was a contract worker from Jharkhand who had recently travelled via train to Kerala for 4 days.

On initial examination, patient's vital signs showed tachycardia with a pulse rate of 170 bpm, blood pressure of 160/80 and also tachypnea with a respiratory rate of 26 breaths/min along with low oxygen saturation (SpO₂) of 69%. In view of poor GCS and low SPO₂ patient was ventilated and intubated, and admitted to ICU for further evaluation.

His CT report showed multiple scattered tiny CSF density cystic space with eccentric nodular calcific foci in supra and infratentorial brain parenchyma suggesting the possibilities of neurocysticercosis and cerebral Koch's disease.

MRI was done on day 2, showed multiple sub-centimetric cystic lesions with eccentric enhancing nodules in bilateral cerebral and cerebellar hemisphere, left thalamus and right hemi pons. Few of the lesions showed minimal perilesional edema with one lesion measuring 9.2 mm. Thus, imaging features were suggestive of parenchymal neurocysticercosis in vesicular and colloid vesicular stages (Figure 1).

His EEG was also done on day 2, showed epileptiform abnormalities arising from right fronto temporal region.

ECG report showed sinus tachycardia for which cardiology consultation was sought (Figure 2). Echocardiogram was done for further evaluation which revealed normal RV function and an ejection fraction of 60%.

His lab investigations were done upon admission and it showed signs of infection along with hypokalemia and elevated liver enzymes level. Additionally, his blood glucose level was significantly elevated to a value of 427 mg/dL.

His respiratory system examination showed bilateral crepitations in both lungs and cardiac examination was unremarkable. The CPK value was found to be elevated on day 4 of hospital stay indicative of muscle injury due to seizure (Table 1).

By re-evaluating the patient's history through family members, it was revealed that he had a history of alcohol use and followed a vegetarian diet.

MANAGEMENT AND OUTCOME

Due to poor GCS and desaturation, he was immediately intubated to secure airway. Inj.Etomidate 20 mg and Inj. Vecuronium 4 mg was administered prior to intubation. Seizures were managed with Inj. Loraz (lorazepam) 4 mg and Inj. Levipil (levetiracetam) 1 g given as stat. Inj. Dexamethasone 8 mg was given as stat dose to manage inflammation which was continued as 4mg thrice daily after CT and MRI investigations. Antipyretics (Inj. paracetamol 1 g thrice daily) were given for fever management.

During ICU stay, patient was restless and experienced multiple episodes of seizures, so sedation and neuromuscular blockade was achieved using Inj. Midazolam 2 mg and Inj.Atracurium 10 mg to manage agitation and facilitate mechanical ventilation. Inj. Fentanyl 50 mcg was also administered to prevent discomfort associated with endotracheal tube. Inj. Neostigmine 2.5 mL was later on administered for reversal of neuromuscular blockade



Figure 1: MRI scans showing multiple sub-centimetric cystic lesions with eccentric enhancing nodules in multiple areas of brain.

along with Inj. Glycopyrrolate 0.4 mg to prevent side effects associated with use of cholinesterase inhibitors.

Seizure management was initially addressed with Inj. Levetiracetam 500 mg twice daily which was later on increased to thrice daily. Inj. Lacosamide 100 mg twice daily was subsequently added for further symptom control. Inj. Cefoperazone/sulbactam 1.5 g was initiated to prevent risk of any bacterial co-infection. Inj. Metronidazole was also added on this therapy and discontinued after two doses. Prophylaxis for bacterial infection was then continued using single antibiotic therapy. Inj. Pantoprazole 40 mg was given to prevent gastric irritation. Hypokalemia was managed by using intravenous fluids (500 mL) containing 20 mEq of Inj. KCl which was later on given as Syp.KCl 15 mL thrice daily during his hospital stay. Due to elevated liver enzymes, Tab.Ursocol (ursodeoxycholic acid) 300 mg was administered via Ryle's tube. Inj. Stelvit B1 100 mg (thiamine) was also added given the patient's history of alcohol use. Respiratory function was maintained using budesonide nebulization given 8th hourly.

On day 2, the frequency of seizure episodes was decreased; however, patient had two episodes of seizures in the afternoon prompting immediate administration of Midazolam injection. By day 3 in ICU, patient started showing improvement in his condition. The seizure episodes started to reduce in intensity. By day 4, sedatives and neuromuscular blockers were stopped. Patient had started responding to call and GCS score also improved to 9/15 ($E_4V_TM_5$). Under the advice of the critical care team, the patient was maintained on NPO and shifted to CPAP with a plan to extubate patient considering the improvement in his condition. IV fluid were continued and on the 4th day of ICU stay, patient was extubated after administration of Inj. Xylocard

Lab Parameter	Normal range	Value on day of admission	Day 2	Day 3	Day 4
Hemoglobin	13-18 g/dL	12.0	-	10.5	9.9
Total count	5000-11000/mm	16900	-	12200	10500
RBS	60-140 mg/dL	427	185	135	134
Creatinine	0.6-1.3 mg/dL	0.9	0.8	0.7	0.7
Sodium	135-145 MEq/L	141	138	141	144
Potassium	3.5-5.5 MEq/L	3.1	3.1	4.0	3.3
Calcium	8.5-11 mg/dL	9.0	-	8.2	8.1
Magnesium	1.8-2.8 mg/dL	2.0	-	1.7	2.0
СРК	25-200 mU/mL				10013
SGOT	Up to 40U/L	101	-	228	297
SGPT	Up to 40U/L	116	-	124	137

Table 1: Lab	o investigations	during	admission	and hospital stay.	•
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Figure 2: The ECG showing sinus tachycardia (rate ~150), 75% sinus rhythm, PR interval 200 ms, and LVH by voltage criteria.

(lignocaine 2%) 2 mL stat. Nebulization using budesonide respules were also given as stat to maintain respiratory functions. Ryle's tube was maintained with feeding rate of 100 mL 2 hourly.

After stabilizing general conditions of patient and control of inflammation as well as seizure episodes using corticosteroids and antiepileptics. Tab. Albendazole 400 mg was started twice daily via Ryle's tube.

By the day 6 patients showed significant improvement. He was able to take oral feeds; hence Ryles's tube was removed. Antiepileptics and other IV medications were changedto oral medications. Inj.Deriphylline, Tab.Clearnac (N-acetylcysteine) along with Budesonide and Duolin (ipratropium and levosalbutamol) nebulization were used for bronchodilation and relief from mild respiratory distress. Chest physiotherapy of grade 2 was also administered to reduce chest secretions.

On day 7, patient was shifted to ward and treatments continued.

Clinical condition of patient further improved and he was able to perform daily activities without support. There were no further seizure episodes. Tab.Quetiapine 25 mg, HS was added after patient expressed lack of sleep and occasional delirium. On 9th day of hospital stay, patient was discharged following his request to return to hometown. Since patient's condition was stabilized and no longer posed any risks, he was discharged with the following medications: Tab.Albendazole 400 mg as antiparasitic treatment, Tab.Levesam (levetiracetam) 500 mg and Tab.Lacosam (lacosamide) 100 mg for seizure management, Tab.Omnacortil (prednisolone) 40 mg as steroid therapy for inflammation associated with neurocysticercosis and Tab.Quetiapine 25 mg along with tablet pantoprazole and vitamin supplements.

Patient was further educated about the severity of his disease condition and was advised to follow stringent hygiene habits to prevent re-infection. Abstinence from alcohol was also advised. A review after 2 weeks was also recommended to patient and his bystanders to ensure complete clinical cure.

DISCUSSION

Clinical manifestations of Neurocysticercosis often depend on the location and stage of cysts. Essentially four distinct forms of NCC have been described by Escobar and Nieto according to primary location of cysts. Of these four forms, parenchymatous is the commonly reported which is characterized by parenchymal cysts of small and rarely larger than 10 mm in diameter.⁴ Similar to the case we discussed, seizures are reported in most cases of parenchymal NCC especially in granular or calcified lesions. Variety of cases on Neurocysticercosis have been reported with most cases arising from Africa, Asia and cases of immigrants presented with NCC symptoms in countries like USA and Australia.⁴⁻⁶ A case from Ukraine presents a 22-year old male patient with sudden episodes of seizures with his MRI showing a few small conglomerating peripherally enhancing thick-walled infective granulomas in left frontal lobe with extensive surrounding edema in the left fronto-parietal lobe.7 Similar to our case, this patient was also vegetarian and had travelled to Ukraine for study purposes. The patient was treated with albendazole, levetiracetam, methylprednisolone and pantoprazole. The clinical condition and subsequent MRI results taken after 3 months of treatment showed improvement. A similar case has also been reported in Himachal Pradesh, India where 38-year-old man was admitted with a history of 6-7 episodes of Generalized Tonic-Clonic (GTC) seizures.8 His Computed Tomography (CT) of the head showed multiple calcified lesions in bilateral cerebral hemispheres scattered diffusely with a few showing peripheral edema, being suggestive of NCC. The patient was also diagnosed as having GTC seizures with status epilepticus induced rhabdomyolysis and AKI. He was treated with AEDs, steroids for cerebral edema, and other supportive measures. The first line approach to treatment in NCC diagnosed patients includes managing seizures and hydrocephalus. Neurocysticercois-induced epilepsy can be managed by a variety of Anti-Epileptic Drugs (AEDs) such oxarbamazepine, valproic acid and levetiracetam. AEDs such as phenytoin and carbamazepine are often avoided when co-administered with anti-helminthic treatment like albendazole as they may reduce the plasma concentrations of albendazole. Studies have shown the benefit of levetiracetam compared to other AEDs in NCC induced epilepsy due to its fewer incidences of side effects and also decreased recurrence of seizures. Similar efficacy has been shown by lacosamide especially in treating children.³ Corticosteroids are used to control inflammatory response of immune system towards colloidal stage and granular stage cysts. Though there are chances of inflammatory complications, the use of corticosteroids is currently necessary as its advantages outnumber its disadvantages.9

Our patient was also initially started on AEDs and corticosteroids along with other supportive measures to control the clinical presentations. Both levetiracetam and lacosamide proved effective in controlling seizures and preventing their recurrence. Use of dexamethasone effectively reduced the inflammatory responses in the patient caused by the calcifying cysts. An initial management of clinical manifestations was required in our patient due to the severity of his condition and the importance of stabilization of patient before managing NCC with anthelminthic therapy. Albendazole 400 mg twice daily was initiated as anthelmintic drug after stabilization.

Use of albendazole in NCC has been discussed in many cases around the world due to its better efficacy compared to other anti-parasitic drugs. A 34-year-old female, presented with complaints of mild seizures and her Magnetic Resonance Imaging (MRI) of the head without contrast reveling a nodular focus of enhancement within the multilobulated cystic mass in the brain with the suspicion of NCC was started on albendazole 400mg twice daily as part of her NCC management.¹⁰ Albendazole have shown to be highly effective in treating parenchymal Neurocysticercosis in a study conducted by Francisco Escobedo *et al.* Meta-analysis of existing data, have shown that albendazole is an effective therapeutic agent in treating NCC and is considered the first line treatment along with praziquantel. Use of cyticidal drug therapy helps in better resolution of colloidal and vesicular cysticerci along with reduction in rate of seizures and lower risk of recurrence.¹¹ The combined use of anti-helminthic, AEDs and corticosteroids in this case have been a major factor in the quick recovery of patient.¹² Continuation of medications and increased hygiene practices are required for the complete eradication of infection and also to lower the risk of re-infection. Healthcare professionals must educate patients on these topics in order to reduce the global impact of NCC especially in endemic regions.

CONCLUSION

Neurocysticercosis is life endangering but preventable central nervous system infection that highlights the importance of proper sanitary measures and food safety practices. Timely diagnosis and proper treatment are crucial factor in reducing the complications of this infection. Patient once infected by NCC must be educated on the importance of medication adherence as well as the importance of basic sanitary habits like washing hands, regularly safe food preparation and drinking clean water. Emphasis on public health awareness can help to reduce the prevalence of NCC and its impact.

ACKNOWLEDGEMENT

The authors would like to sincerely thank Dr. Kevin Reji, Consultant, Department of Neurology, Lourdes Hospital, along with the hospital management, for their valuable support and encouragement. We also extend our heartfelt gratitude to St. Joseph's College of Pharmacy's management for their guidance and support.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

AED: Antiepileptic drugs; AKI: Acute kidney injury; CNS: Central nervous system; CPK: creatine phosphokinase; CSF: Ceberospinal fluid; CT: Computed tomography; ECG: echocardiogram; ELISA: Enzyme-linked immunosorbent assay; GCS: Glasgow coma scale; GTC: Generalised tonic clonic seizure; LVH: left ventricular hypertrophy; MRI: Magnetic resonance imaging; NCC: Neurocysticercosis; NPO: Nothing by mouth; RBS: Random blood sugar; SGOT: Serum glutamic-oxaloacetic transaminase; SGPT: Serum glutamic-pyruvic transaminase.

DECLARATION OF PATIENT CONSENT

The authors would certify that they have obtained all appropriate patient consent.

SUMMARY

In summary, this is a case of patient who developed parenchymal neurocyticercosis due to possible contaminated food and improper hygiene. Treatment was managed based on established guidelines, with a prompt approach by the doctor, along with supportive care, leading to recovery.

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Cite this article: Thomas A, Fathima D, Ramachandran L, Reji K. Parenchymal Neurocysticercosis: A Case Study on Diagnostic and Treatment Approach. Indian J Pharmacy Practice. 2025;18(4):466-70.