# **Title Page**

**Manuscript type: Case Report**

# **Disseminated Neurocysticercosis with Intraventricular and Cisternal Extension** **without Hydrocephalus: A Case Report**

# **Authorship**

1. Bibek Shrestha, Maharajgunj Medical Campus, Tribhuvan University, Institute of Medicine, Kathmandu Nepal, Email: [shresthabibek85iom@gmail.com](mailto:shresthabibek85iom@gmail.com)
2. Priyesh Shrestha, Department of Radiology, Tribhuvan University Teaching Hospital, Kathmandu Nepal, Email: priyeshshrestha7@gmail.com
3. Bikram Gajurel, Department of Internal Medicine, Tribhuvan University Teaching Hospital, Kathmandu Nepal, Email: [Bikramgajurel@hotmail.com](mailto:Bikramgajurel@hotmail.com)
4. Grishma Kandel, Maharajgunj Medical Campus, Tribhuvan University, Institute of Medicine, Kathmandu Nepal, Email: [kandelgrishma10@gmail.com](mailto:kandelgrishma10@gmail.com)
5. Laxmi Shah, Maharajgunj Medical Campus, Tribhuvan University, Institute of Medicine, Kathmandu Nepal, Email: [Shahlaxmi203@gmail.com](mailto:Shahlaxmi203@gmail.com)

# **Statement of Contribution**

Bibek Shrestha played a central role in the research, contributing to conceptualization, data curation, formal analysis, and methodology. He was responsible for project administration and took the lead in writing the original draft, as well as in the review, editing, and visualization processes. Priyesh Shrestha, Bikram Gajurel, Grishma Kandel and Laxmi Shah were involved in the supervision, validation, and investigation phases of the project, ensuring the study's accuracy and credibility.

# **Corresponding author**

Bibek Shrestha, Maharajgunj Medical Campus, Tribhuvan University, Institute of Medicine, Kathmandu Nepal, Email: [shresthabibek85iom@gmail.com](mailto:shresthabibek85iom@gmail.com)

# **Disclosure**

None

# **Data availability statement**

None

# **Funding statement**

None

# **Conflict of interest**

None

# **Patient consent statement**

Written informed consent was obtained from the patient for publication of this case report and accompanying images, complying with the requirements as mentioned in Wiley’s CCR Consent Form.

# **Ethical Approval**

The institutional review board (IRB) of Institute of Medicine, Maharajgunj Medical Campus does not mandate ethical approval for Case Report.

# **Disseminated Neurocysticercosis with Intraventricular and Cisternal Extension without Hydrocephalus: A Case Report**

# **Abstract**

Neurocysticercosis, caused by the larval stage of Taenia solium, is a significant cause of acquired epilepsy in endemic regions. Disseminated Neurocysticercosis, a rare and severe form, involves multiple cystic lesions in the central nervous system and other body sites, leading to complications such as hydrocephalus, increased intracranial pressure, and neurological deficits. We report a 33-year-old male presenting with recurrent seizures, progressive left-sided weakness, and radiological findings of disseminated NCC with intraventricular and cisternal extension. Diagnosis was confirmed through MRI, revealing cystic lesions in various developmental stages with the characteristic “cyst-with-dot” sign. Treatment included intravenous dexamethasone, mannitol, and levetiracetam, with significant clinical improvement observed. This case underscores the importance of early diagnosis and multimodal management using antiparasitic therapy, corticosteroids, and seizure control to mitigate morbidity and mortality. Public health measures, including improved sanitation and education, are critical to reducing the burden of NCC in endemic areas.

# **Keywords**

hemiplegia; neurocysticercosis; seizure; taenia solium

# **Introduction**

Neurocysticercosis (NCC) is a common parasitic infection of the central nervous system caused by the larval stage of the pork tapeworm Taenia solium 1. Humans acquire NCC through the accidental ingestion of T. solium eggs, which have been shed in the stools of a person harboring the adult tapeworm 2. NCC is a major cause of acquired epilepsy worldwide, especially in endemic areas 3. The clinical manifestations of NCC can vary widely, but seizures are the most common presentation, occurring in 70-90% of patients 4. Other common symptoms include headaches, intracranial hypertension, and focal neurological deficits 5. Rarer presentations, such as intraventricular or cisternal involvement, can also occur. Disseminated NCC with intraventricular and cisternal involvement is a rare and severe manifestation of the disease. This form of NCC can lead to complications such as hydrocephalus, increased intracranial pressure, and significant morbidity and mortality. The cysts in the ventricular system and subarachnoid spaces can cause mass effect, obstructive hydrocephalus, and inflammatory reactions, leading to a more severe clinical presentation 6,7. This case is notable for its unique clinical presentation, which included recurrent seizures, progressive left-sided weakness, and radiological findings indicative of disseminated NCC in various stages of development. The patient’s history of consuming raw meat and unfiltered water further underscores the public health significance of addressing modifiable risk factors associated with NCC in endemic regions.

This report seeks to highlight the diagnostic process, management strategies, and outcomes for a rare and severe case of disseminated NCC with intraventricular and cisternal extension. By contributing to the existing body of literature, this case offers valuable insights into the complexities of diagnosing and treating advanced manifestations of NCC, while reinforcing the need for timely identification and intervention to improve patient outcomes.

**Case History/ Examination**

A 33-year-old married male, employed as a hydropower operator and identifying as Hindu, presented to the Emergency Department with chief complaints of loss of consciousness, left-sided weakness, and abnormal body movements persisting for two days. The patient was reportedly in his usual state of health until two days prior to the presentation. At approximately 10:00 AM, while lying in bed, he experienced an abnormal movement localized to the third to fifth fingers of his left hand, which gradually progressed to involve the entire left upper limb. After approximately one minute, the patient attempted to rise from bed but suddenly fell and lost consciousness. According to an eyewitness account provided by his friend, the patient exhibited frothy oral discharge, tongue biting, and urinary incontinence during the episode. His body initially became stiff, followed by a period of muscle relaxation, and then a recurrence of stiffness. Consciousness was regained after approximately one hour, at which point the patient reported a persistent headache. The patient also reported a history of left-sided weakness, which began acutely 10 days prior and has been gradually progressive. This weakness was associated with slurred speech, although there were no difficulties with swallowing. There was no history of head trauma, vomiting, fever, cough, or shortness of breath preceding or following the event. The patient's medical history is significant for hypertension diagnosed two years ago, for which he has been on regular antihypertensive medication. His personal history includes smoking, with a cumulative exposure of 10 pack-years, and no reported history of alcohol consumption. The patient reported a habitual consumption of raw and undercooked meat and reliance on unfiltered water as his primary source of drinking water.

On examination, the patient was alert, cooperative, and oriented to time, place, and person, with intact higher mental functions. Vital signs were stable, and there were no signs of meningeal irritation. Neurological examination revealed normal muscle bulk and tone in all four limbs, with power graded as 4/5 in the left upper and lower limbs and 5/5 in the right limbs. Deep tendon reflexes, including biceps, triceps, supinator, knee, and ankle reflexes, were 2+ bilaterally, and plantar reflexes were down going. Sensory examination showed intact pain and touch sensations. The examination of the respiratory and cardiovascular systems was unremarkable.

# **Methods**

Based on the patient's history and clinical examination, a range of differential diagnoses were considered, including neurocysticercosis, cerebrovascular events such as stroke, brain tumor, intracranial tuberculosis, epilepsy, and metabolic or toxic encephalopathy. To further refine the diagnosis, appropriate laboratory investigations and advanced imaging modalities were undertaken. Laboratory investigations, including hematological parameters, differential leukocyte count, biochemical analysis, coagulation profile, and serological tests, were conducted and reported to be within normal limits (Table 1). His stool examinations were unremarkable with absence of pus cells, RBC, mucus and blood. Parasitology investigation revealed no parasites. Additionally, advanced radiological imaging, specifically magnetic resonance imaging (MRI) of the brain and spine, was performed to further find out the etiology. There were variable sized cystic lesions on T1 and T2 MRI with eccentric hyperintense nodules giving “Cyst with dot” sign involving bilateral cerebral, cerebellar hemisphere along with right sylvian and right cingulate gyrus. (Figure 1, Figure 2, Figure 3 and Figure 4) There was no suppression of the T2/FLAIR sequence whereas T2 FLAIR showed vasogenic white matter edema. There was no obvious dilatation of the ventricle.

**Conclusion and Results**

The history, examination and radiological investigation confirmed the diagnosis of disseminated neurocysticercosis (at different development stages) with intraventricular and cisternal extension. The patient was managed with intravenous levetiracetam (1 gram) and intravenous mannitol 20% (100 mL) for one day to address the seizure episode. This was followed by oral levetiracetam at a dose of 500 mg twice daily. For hypertension, oral amlodipine 5 mg once daily was initiated. Regarding the treatment of neurocysticercosis, dexamethasone 8 mg was administered intravenously once daily for one week. Upon follow-up, the patient demonstrated significant clinical improvement, with resolution of symptoms, including weakness. He regained his ability to ambulate as he did prior to the illness, and no further episodes of seizures or unconsciousness were reported.

**Discussion**

Disseminated cysticercosis is a systemic parasitic infection caused by the larval stage of Taenia solium, the pork tapeworm 8. It is characterized by multiple cystic lesions in the brain and at least two other body parts, including muscles, skin, and eyes 9. Neurocysticercosis, the central nervous system involvement, is a common cause of adult-onset seizures in tropical regions 8. Symptoms may include headaches, vomiting, seizures, and altered sensorium and intraventricular involvement can even lead to hydrocephalus, ventriculitis and periventricular edema in which fourth ventricular is the most affected 10,11. The clinical manifestations of NCC can vary widely, but seizures are the most common presentation occurring in 70-90% of patients 4. The clinical presentation of ventricular NCC is relatively specific because symptoms are primarily due to acute and/or chronic obstruction of cerebrospinal fluid (CSF) flow and associated inflammation, usually centered in and around the fourth ventricle 11. Disseminated neurocysticercosis, particularly involving cisternal and ventricular regions, is primarily caused by the larval form of the tapeworm Taenia solium, acquired through fecal-oral contamination 12. In our case, the patient presented with complaints of seizure, unconsciousness, hemiplegia and headache for 2 days however there was no history of increased intracranial pressure such as projectile vomiting and neck rigidity. Accurate diagnosis of NCC relies on the integration of clinical data, neuroimaging findings, and immunological tests 3. Neuroimaging techniques, such as computed tomography (CT) and MRI, can detect the different stages of NCC, including the viable, degenerative, and calcified phases 13. Magnetic resonance imaging (MRI) plays a crucial role in diagnosing neurocysticercosis with ventricular involvement and cisternal extension. T1-weighted images can detect intraventricular cysts, showing cyst walls, mural nodules, and increased signal intensity of cyst fluid. Cisternal cysts appear like intraventricular cysts but rarely display mural nodule 15. Three-dimensional MRI sequences, such as enhanced SPGR and FIESTA, have shown promise in detecting intraventricular cysts and scolices 16. Serological tests, such as the enzyme-linked immunosorbent assay (ELISA) and the enzyme-linked immunoelectrotransfer blot (EITB) assay, can also aid in diagnosis 14. In this case, T1 and T2 MRI with hyperintense nodules giving “Cyst with dot” sign involving both cerebral, cerebellar hemisphere along with right sylvian and right cingulate gyrus without hydrocephalus.

Disseminated NCC have a high rate of morbidity and mortality in which multimodal treatment modality is used which includes antiparasitic therapy, steroid and seizure management. Intravenous or oral corticosteroids, such as dexamethasone or methylprednisolone, are often used to reduce the acute symptomatic edema and inflammation associated with disseminated NCC. Prolonged steroid treatment may be required due to the recurrent and prolonged inflammatory status in disseminated NCC 17. Steroid therapy is crucial to prevent adverse inflammatory reactions and transient clinical deterioration caused by antiparasitic therapy 18. Antiparasitic drugs, such as albendazole and praziquantel, are the mainstay of treatment for NCC. These drugs are effective in treating both parenchymal and extra parenchymal (intraventricular and cisternal) forms of NCC. Antiepileptic drugs (AEDs), such as carbamazepine, oxcarbazepine, levetiracetam, and phenytoin, are used to control seizures associated with NCC. Levetiracetam is often preferred as it has a favorable safety profile and minimal drug interactions 19. Seizures secondary to NCC usually respond well to first-line single AED therapy, but some cases may require multi-drug therapy 20. In this case, the patient was treated with dexamethasone and mannitol as primary therapies, levetiracetam to manage seizures, and amlodipine to control hypertension. Key finding of this study is the successful diagnosis and management of disseminated NCC with intraventricular and cisternal extension, a condition presenting with severe neurological symptoms, including recurrent seizures and hemiparesis. The case demonstrates the effectiveness of a comprehensive treatment regimen comprising corticosteroid and anticonvulsants in improving clinical outcomes.

**Conclusion**

Disseminated NCC with intraventricular and cisternal extension represents a rare and severe form of the disease, often leading to significant morbidity due to complications such as seizures, intracranial hypertension, and hydrocephalus. Effective management requires a multimodal approach, integrating antiparasitic therapy, corticosteroids, and antiepileptic drugs to address the parasitic burden, reduce inflammation, and control seizures. Imaging modalities, particularly MRI, play a critical role in diagnosis, allowing for the identification of cystic lesions in various stages. The case highlights the importance of timely and accurate diagnosis, supported by neuroimaging findings and clinical correlation, in guiding therapy and improving outcomes. Additionally, addressing modifiable risk factors, such as the consumption of undercooked meat and unfiltered water, is essential in endemic regions to prevent the disease.

# **References**

1. Son H., Kim M., Jung K., Choi S., Jung J., Chong Y.et al. Neurocysticercosis: clinical characteristics and changes from 26 years of experience in a university hospital in korea. The Korean Journal of Parasitology 2019;57(3):265-271. <https://doi.org/10.3347/kjp.2019.57.3.265>
2. Serpa J. and White A. Neurocysticercosis in the united states. Pathogens and Global Health 2012;106(5):256-260. <https://doi.org/10.1179/2047773212y.0000000028>
3. Brutto O.. Neurocysticercosis: a review. The Scientific World Journal 2012;2012:1-8. <https://doi.org/10.1100/2012/159821>
4. Batra S., Kumar S., & Shekhawat L.. Neurocysticercosis presenting as bipolar disorder: a case report. General Psychiatry 2021;34(6):e100663. <https://doi.org/10.1136/gpsych-2021-100663>
5. Brutto O.. Neurocysticercosis. The Neurohospitalist 2014;4(4):205-212. <https://doi.org/10.1177/1941874414533351>
6. Singh P. and Singh S. A case of racemose and intraventricular neurocysticercosis in an unusual location. South African Journal of Radiology 2021;25(1). <https://doi.org/10.4102/sajr.v25i1.2171>
7. Pamplona J., Braz A., Conceição C., Rios C., & Reis J. A rare case of racemose neurocysticercosis and its complications. case report. The Neuroradiology Journal 2015;28(4):418-420. <https://doi.org/10.1177/1971400915595305>
8. Ranganathan, L., Karthikeayan, V., Mm, A., R, G., Kj, V., N, T. P., & Mehndiratta, M. (2017). Disseminated cysticercosis and role of spin echo short tau inversion recovery sequence imaging: Case based review. International Journal of Epilepsy, 04(02), 174–180. <https://doi.org/10.1016/j.ijep.2017.10.002>
9. George, J., Goel, V., Agarwal, A., Vishnu, V. Y., Garg, A., & Srivastava, M. P. (2022). Disseminated Cysticercosis-Should we treat? Tropical Doctor, 52(3), 444–445. <https://doi.org/10.1177/00494755221084387>
10. Jawale, R., & Duberkar, D. (2015). Disseminated cysticercosis. Neurology, 84(3), 327. <https://doi.org/10.1212/wnl.0000000000001152>
11. Nash T., Ware J., & Mahanty S. Intraventricular neurocysticercosis: experience and long-term outcome from a tertiary referral center in the United States. American Journal of Tropical Medicine and Hygiene 2018;98(6):1755-1762. https://doi.org/10.4269/ajtmh.18-0085
12. Coyle, C. M., Mahanty, S., Zunt, J. R., Wallin, M. T., Cantey, P. T., White, A. C., O’Neal, S. E., Serpa, J. A., Southern, P. M., Wilkins, P., McCarthy, A. E., Higgs, E. S., & Nash, T. E. (2012). Neurocysticercosis: Neglected but Not Forgotten. PLoS Neglected Tropical Diseases, 6(5), e1500. <https://doi.org/10.1371/journal.pntd.0001500>
13. Sakhuja A., KC S., Wortsman J., Shrestha D. , Aryal B. , Kwatra V. et al.. Severe neurocysticercosis in an immunocompetent male without travel to an endemic region: a case report. Cureus 2023. <https://doi.org/10.7759/cureus.34870>
14. Raffaldi I., Scolfaro C., Mignone F., Aguzzi S. , Denegri F. , & Tovo P.. An uncommon cause of seizures in children living in developed countries: neurocysticercosis -a case report. Italian Journal of Pediatrics 2011;37(1):9. <https://doi.org/10.1186/1824-7288-37-9>
15. Zee, C., Segall, H. D., Boswell, W., Ahmadi, J., Nelson, M., & Colletti, P. (1988). MR Imaging of Neurocysticercosis. Journal of Computer Assisted Tomography, 12(6), 927–934. <https://doi.org/10.1097/00004728-198811000-00004>
16. Filho, F. E. F. M., Machado, L. D. R., Lucato, L. T., & Leite, C. C. (2011). The role of 3D volumetric MR sequences in diagnosing intraventricular neurocysticercosis: preliminar results. Arquivos De Neuro-Psiquiatria, 69(1), 74–78. https://doi.org/10.1590/s0004-282x2011000100015
17. Abraham R.. Steroids in neuroinfection. Arquivos De Neuro-Psiquiatria 2013;71(9B):717-721. <https://doi.org/10.1590/0004-282x20130158>
18. Agaba E. , Modi D. , Gündüz Ö. , & Modi Z.. Subcutaneous nodules of cysticercosis as a sign of asymptomatic neurocysticercosis in an hiv positive patient. Revista Da Sociedade Brasileira De Medicina Tropical 2018;51(6):861-863. <https://doi.org/10.1590/0037-8682-0178-2018>
19. Sankhyan N., Kadwa R., Kamate M. , Kannan L. , Kumar A. , Passi G. et al.. Management of neurocysticercosis in children: association of child neurology consensus guidelines. Indian Pediatrics 2021;58(9):871-880. <https://doi.org/10.1007/s13312-021-2311-6>
20. Sangale D., Adhikari A., & Gajre M. Clinico-demographic profile of children with neurocysticercosis and risk factors for persistent seizures. International Journal of Contemporary Pediatrics 2023;10(3):300-304. https://doi.org/10.18203/2349-3291.ijcp20230423