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Disseminated Neurocysticercosis With Intraventricular and Cisternal Extension Without Hydrocephalus: A Case Report

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ABSTRACT

Neurocysticercosis, a leading cause of acquired epilepsy, can present as disseminated disease with widespread cystic lesions. A 33-year-old male with seizures and weakness showed radiologically confirmed neurocysticercosis. Treatment with steroids, antiparasitics, and antiseizure medication led to improvement. Early diagnosis and public health measures are crucial to reducing the burden.

1 | 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 in severe form. 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.

2 | Case History/Examination

The patient, a 33-year-old male, presented with new-onset seizures characterized by focal motor activity progressing to generalized convulsions, associated with loss of consciousness, tongue biting, and urinary incontinence. His postictal state lasted approximately an hour, followed by persistent headache and left-sided weakness. There was no preceding history of trauma, fever, or signs of raised intracranial pressure. The

patient's medical history is significant for hypertension diagnosed 2 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 examinations, the patient was conscious, 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 Babinski was positive. Sensory examination showed intact pain and touch sensations. The examination of the respiratory and cardiovascular systems was unremarkable.

3 | Differential Diagnosis

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 at the time of admission, 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,

TABLE 1 | Laboratory report.

Category	Test	Value	Normal range
Hematology	Hemoglobin	13.5 g/dL	13.0–17.0 g/dL
	Total Leukocyte Count	6200 cells/μL	4000–10,000 cells/μL
	Platelets	218,000 cells/μL	150,000–450,000 cells/μL
Differential leukocyte count	Neutrophils	60%	40%–75%
	Lymphocytes	32%	20%–45%
	Monocytes	6%	2%–1%0%
	Eosinophils	2%	1%–6%
	Basophils	0%	0%–1%
Biochemistry	Sodium	140 mmol/L	135–145 mmol/L
	Potassium	4.1 mmol/L	3.5–5.0 mmol/L
	Urea	14 mg/dL	7–20 mg/dL
	Creatinine	0.9 mg/dL	0.6–1.2 mg/dL
	SGOT/AST	21 U/L	0–40 U/L
	SGPT/ALT	16 U/L	0–40 U/L
	ALP (Alkaline Phosphatase)	70 U/L	44–147 U/L
	Total Bilirubin	0.5 mg/dL	0.1–1.2 mg/dL
	Direct Bilirubin	0.2 mg/dL	0–0.3 mg/dL
	Indirect Bilirubin	0.3 mg/dL	Derived value
	Total Protein	6.8 g/dL	6.4–8.3 g/dL
	Albumin	4.3 g/dL	3.5–5.0 g/dL
	Globulin	2.5 g/dL	2.0–3.5 g/dL
	A/G Ratio	1.72	1.2–2.0
Coagulation assay	Prothrombin Time (PT)	13.5 s	11.0–15.0 s
	INR	1.02	0.8–1.2
Serological exam	HBsAg Spot/Quick	Non-Reactive	Negative
	HCV Ab Spot/Quick	Non-Reactive	Negative
	HIV Ab Spot/Quick	Non-Reactive	Negative

mucus and blood. Parasitology investigation done twice (at the time of admission and 3 days after the admission) revealed no parasites. Given the patient's acute onset of seizures and left-sided weakness, an urgent neuroimaging study was warranted to rule out emergent causes such as stroke, intracranial hemorrhage, or space-occupying lesions. While a Computed Tomography (CT) scan is often the initial imaging modality in emergency settings to rapidly detect hemorrhage or significant structural abnormalities, Magnetic Resonance Imaging (MRI) was prioritized in this case due to its superior sensitivity in detecting parenchymal and extra-parenchymal neurocysticercosis lesions which was the suspected pathology given the patient's raw and uncooked meat, particularly those involving the ventricular and cisternal spaces [8]. 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. (Figures 1–4) There was no suppression of the T2/FLAIR sequence whereas T2 FLAIR showed vasogenic white matter edema and had no obvious dilatation of the ventricle.

4 | Conclusion and Results (Outcome and Follow-Up)

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 g) and intravenous mannitol 20% (100 mL) for 1 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 1 week along with albendazole 400 mg twice daily in combination with oral praziquantel 1200 mg three times daily for a total of 14 days. Upon follow-up (at 1st week, 4th week, and 2 months after discharge), the patient demonstrated clinical improvement, with resolution of seizure and body weakness. He regained his ability to ambulate as he did prior to the illness, and no further episodes of seizures or unconsciousness were reported.

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

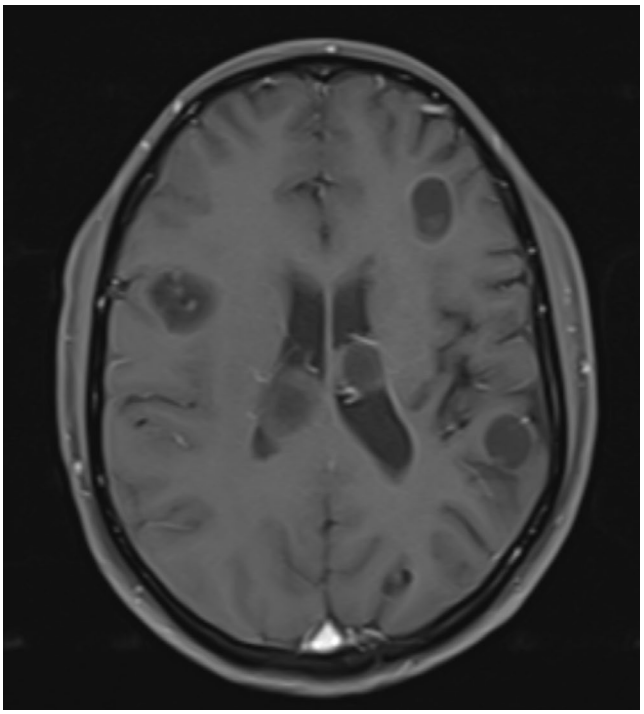

FIGURE 1 | Axial T1 weighted MRI showing ring-enhancing lesion in parenchyma and intraventricular area.

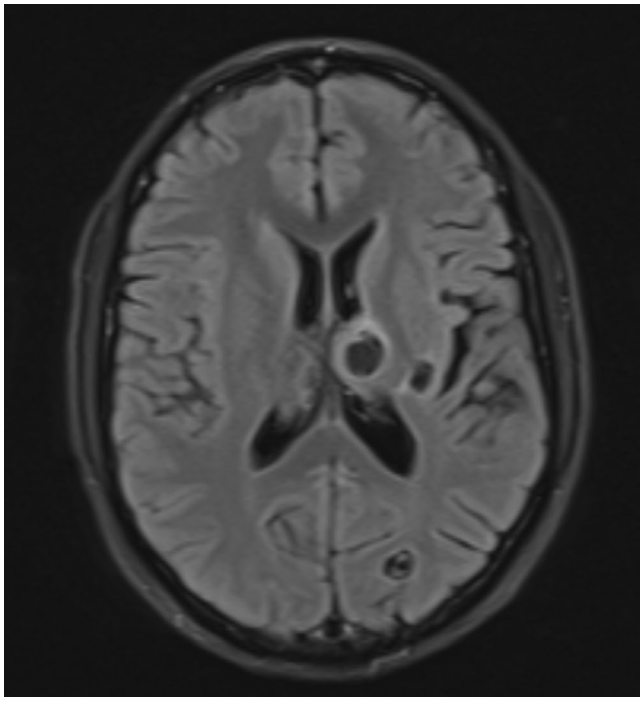

FIGURE 2 | Axial FLAIR MRI showing a hyperintense lesion surrounded by edema.

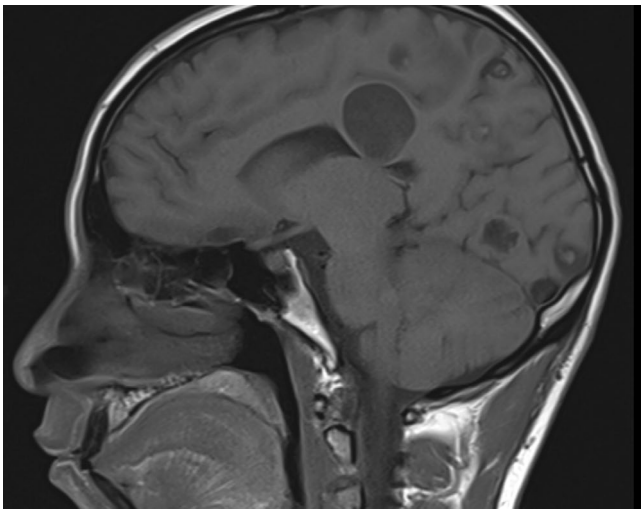

FIGURE 3 | MRI sagittal view with a well-defined fluid-filled ring-enhancing lesion.

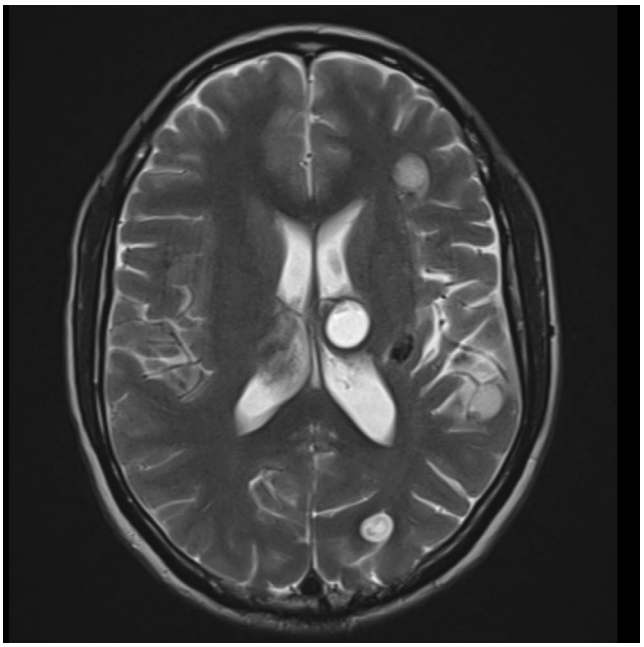

FIGURE 4 | Axial T2 weighted MRI showing hyperintense ring-enhancing lesion with surrounding vasogenic edema.

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.

5 | Discussion

Disseminated cysticercosis is a systemic parasitic infection caused by the larval stage of Taenia solium, the pork tapeworm [9]. It is characterized by multiple cystic lesions in the brain and at least two other body parts, including muscles, skin, and eyes [10–14]. (Table 2) Neurocysticercosis, the central nervous system involvement, is a common cause of adult-onset seizures in tropical regions [9]. Symptoms may include headaches, vomiting, seizures, and altered sensorium and intraventricular involvement can even lead to hydrocephalus, ventriculitis, and periventricular edema in which the fourth ventricular is the most affected [15, 16]. 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 [16]. 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 [17]. 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 CT and MRI, can detect the different stages of NCC, including the viable, degenerative, and calcified phases [18]. 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 [19]. Three-dimensional MRI sequences, such as enhanced SPGR and FIESTA, have shown promise in detecting intraventricular cysts and scolices [20]. Serological tests, such as the enzyme-linked immunosorbent assay (ELISA) and the enzyme-linked immunoelectrotransfer blot (EITB) assay, can also aid in diagnosis [21]. In this case, T1 and T2 MRI with hyperintense nodules gave "Cyst with dot" sign involving both cerebral, cerebellar hemisphere along with the right sylvian and right cingulate gyrus without hydrocephalus.

Disseminated NCC has a high rate of morbidity and mortality in which a multimodal treatment modality is used which includes antiparasitic therapy, steroids, 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 [22]. Steroid therapy is crucial to prevent adverse inflammatory reactions and transient clinical deterioration caused by antiparasitic therapy [23]. 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 [24]. Seizures secondary to NCC usually respond well to first-line single AED therapy, but some cases may require multi-drug therapy [25]. The Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH) guidelines recommend individualized management for extraparenchymal NCC, advocating surgery primarily for obstructive hydrocephalus while recognizing that medical therapy alone may be sufficient in select cases [26]. A recent review by De Marco et al. supports this, demonstrating successful non-surgical management of intraventricular and subarachnoid NCC with antiparasitic therapy and corticosteroids. Given the absence of hydrocephalus in our case, we opted for a conservative approach, achieving favorable clinical outcomes [27]. Evidence-based guidelines recommend albendazole with either dexamethasone or prednisolone for both adults and children to decrease active lesions and long-term seizure frequency [28]. In this case, the patient was treated with dexamethasone and mannitol as primary therapies, levetiracetam to manage seizures,

TABLE 2 | Literature review table including clinical findings and summary.

S. N	Author	Clinical findings	Summary
1.	Lahiri et al. (2021) [11]	Acute-onset symmetric paraparesis Sensory loss below the umbilicus Double sphincter dysfunction Multiple pea-sized nodules on skin and tongue Diffuse perilesional edema in the spinal cord	62-year-old male from India presenting with acute dorsal myelopathy due to intramedullary cysticercus, an extremely rare initial manifestation of disseminated cysticercosis (DCC). The patient exhibited progressive lower limb weakness, sensory impairment, and sphincter dysfunction. MRI revealed cystic lesions in the spinal cord, brain, and muscles, consistent with disseminated cysticercosis. The diagnosis was confirmed via histopathological examination and ELISA assay. The patient showed improvement with steroid therapy, but antihelminthic treatment was avoided due to extensive CNS involvement. This report highlights a rare but treatable case of DCC, emphasizing the importance of early recognition in tropical medicine.
2.	Singh et al. (2013) [12]	Body swelling (pseudohypertrophy of skeletal muscles) Bilateral proptosis with extraocular muscle involvement and retinal cystic lesion Presence of palpable subcutaneous nodules Multisystem involvement with cystic lesions noted in the brain, orbit, skeletal muscles, and heart	An 8-year-old boy with disseminated cysticercosis presenting with a constellation of findings including marked body swelling due to muscular pseudohypertrophy, bilateral proptosis, and subcutaneous nodules. Imaging studies revealed multiple non-enhancing vesicular cysts in the brain (producing a "starry sky" appearance), extraocular muscles, skeletal muscles, and cardiac tissues. Uniquely, a Doppler study identified thrombosis of the right common femoral vein finding not previously reported in disseminated cysticercosis in children. The patient was managed with a combination of corticosteroids, albendazole, antiepileptics, and anticoagulants, leading to complete recovery and resolution of lesions on follow-up.
3.	Feher et al. (2024) [13]	A young female presenting with chronic headache unresponsive to NSAIDs History of intracranial hypertension managed by neuroendoscopic surgery and ventriculoperitoneal shunting Follow-up imaging revealing multiple new cysts in the basal cisterns and spinal subarachnoid space with compression of the pons Progressive increase in the size and number of cysts despite treatment with albendazole and corticosteroids	A 22-year-old female with a long-standing history of headache and previous surgical treatment for intracranial hypertension, who later developed multiple cystic lesions in both the intracranial and spinal compartments. Initially misdiagnosed as neurocysticercosis due to overlapping radiological features, the patient's lack of response to antiparasitic therapy and negative CSF antigen/antibody assays prompted further evaluation. Surgical intervention followed by histopathological and immunohistochemical analysis ultimately confirmed the diagnosis of neurenteric cysts. The report highlights the diagnostic challenges in differentiating neurenteric cysts from neurocysticercosis, especially in regions where the latter is endemic, and underscores the potential for recurrence and dissemination, which necessitates careful long-term monitoring.
4.	Bothale, K.A., et al. (2012) [14]	Progressive loss of memory and judgment Muscular pseudohypertrophy in the neck and arm region Mild-to-moderate cognitive deficits (attention, orientation, memory) Presence of neurocysticercosis with cystic lesions in the brain and muscles	A 36-year-old male presented with disseminated cysticercosis (DCC), affecting the brain, skeletal muscles, and subcutaneous tissues. Initial treatment with Albendazole was ineffective; subsequently, Praziquantel with steroids led to improvement. The case highlights the challenges in diagnosing and managing DCC due to its widespread organ involvement and atypical presentations. CT and MRI imaging play a crucial role in identifying cystic lesions, and histopathology confirms the diagnosis.

and amlodipine to control hypertension. The 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 corticosteroids and anticonvulsants in improving clinical outcomes.

Author Contributions

Bibek Shrestha: conceptualization, data curation, formal analysis, methodology, project administration, writing – original draft, writing – review and editing. **Laxmi Shah:** investigation, supervision, validation. **Priyesh Shrestha:** investigation, supervision, validation. **Bikram Prasad Gajurel:** investigation, supervision, validation. **Grishma Kandel:** investigation, supervision, validation.

Disclosure

The authors have nothing to report.

Ethics Statement

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

Consent

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.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The authors have nothing to report.

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