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Case Report

Concurrent Extraparenchymal and Spinal Neurocysticercosis in an Immunocompetent Patient Treated for Obstructive Hydrocephalus and Spinal Cord Compression

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Abstract:

Introduction: Neurocysticercosis (NCC) is a condition caused by infection of the central nervous system (CNS) by Taenia solium, or pork tapeworm, larvae. Despite commonly infecting the CNS, it may be easily misdiagnosed for more common pathologies, such as arachnoid cysts or tumors. Though most frequently seen in endemic areas, immigration patterns are altering its traditional distribution. Extraparenchymal neurocysticercosis (including ventricular, spinal, and subarachnoid types) carries a poorer prognosis

Objective: We present a case of NCC presenting in a young immunocompetent patient which was initially thought to be an arachnoid cyst due to non-pathognomonic imaging results and symptoms. This case illustrates a challenging NCC diagnosis.

Case Report: A 24-year-old male without known past medical history presented with 2 years of chronic headache, 2 months of blurry vision in the left eye, and 1 week of progressive nausea and vomiting found to have aqueductal stenosis with obstructive hydrocephalus and bilateral 1.5 cm, peripherally-enhancing cystic lesions of the cerebellopontine angle with associated mass effect, initially described as arachnoid cysts, as well as a left sided foramen magnum cyst. He had an external ventricular drain placed and later underwent ventriculoperitoneal shunt (VPS) insertion. He presented 10 days later with sepsis symptoms and the initial workup was concerning for tuberculosis-associated meningitis and he underwent shunt externalization and re-internalization after clinical improvement, though he was lost to follow up. Two months later he presented with acute urinary retention and right sided weakness. He had spinal cord compression of the cervicothoracic and thoracolumbar spine due to cystic lesion and underwent right C7-T1 and T10-T11 hemilaminotomies for cyst resection, with pathology consistent with NCC.

Conclusion: Neurocysticercosis (NCC) is a complex disease which is largely overlooked and is increasing in prevalence in non-endemic areas such as the United States. NCC has various symptoms and presentations depending on location and progression of larval development, which may mimic more common pathologies. The clinical manifestations depend on the parasite load and the host's immune response regarding their location in CNS and their evolutionary stage. There are two types of NCC, the parenchymal and the extraparenchymal form. The extraparenchymal racemose NCC has the worst prognosis among all forms of NCC with mortality ranging between 30% and 80%. This reinforces the need for a broad differential diagnosis in these scenarios, as well as the importance of close Infectious Disease follow up for patients diagnosed with concurrent cranial and spinal NCC..

Keywords: neurocysticercosis; clinical trials; case report; central nervous system infection; helminthic infection; taenia solium

Abbreviations

T-Spot TB: Tuberculosis-specific enzyme-linked immunospot assay

TB: Tuberculosis

EVD: External ventricular drain

CSF: Cerebrospinal fluid

WBC: White blood cell

PCR: Polymerase chain reaction

MRI: Magnetic Resonance Imaging

CT: Computed tomography

VP: Ventriculoperitoneal

CNS: Central nervous system

RLE: Right lower extremity

RUE: Right upper extremity

NCC: Neurocysticercosis

DALYS: Disability Adjusted for Life Years

KPS: Karnofsky Performance Scale

Introduction

Neurocysticercosis (NCC) is characterized by infection of the central nervous system (CNS) by the larva of Taenia solium, or pork tapeworm. Once infected with Taenia solium, CNS involvement is very common, reaching upwards of 60-90% of cases. [1,2] Infection occurs via ingestion of tapeworm eggs, with subsequent release of embryos from the egg that are able to cross the bowel wall and enter the bloodstream, resulting in systemic distribution. Humans are the definitive host for Taenia solium**,** though both humans and pigs can act as intermediate hosts and can shelter and shed larvae for transmission and infection of others. [1-8]

NCC has been cited as the most common helminthic disease of the CNS in humans and accounts for upwards of 30% of epilepsy cases within endemic areas (Latin America, sub-Saharan Africa, India, and Southern Asia) [8,11,13-15,36,40,44]. However, immigration is associated with increases in non-endemic areas, including the United States and Europe. [7-9,13,30,35,37-39,41-42] The Global Burden of Disease Atlas in 2019 estimated that NCC caused 1.37 million Disability Adjusted for Life Years (DALYS) globally, which places a large burden on not only those patients, but also on the health systems providing care to these patients. [30]

NCC has multiple forms, the most common being intraparenchymal followed by extraparenchymal, each having different presentations, management, and treatment.[32] The intraparenchymal presentation is characterized by embryos forming cysts within the brain parenchyma, which may lead to seizures, chronic headaches, or in some cases focal neurologic deficits.[29,43] In extraparenchymal cases, cysts are located within the cerebral ventricles, subarachnoid space, or within the spine.[32,43] Extraparenchymal NCC involving the subarachnoid space primarily present with symptoms of intracranial hypertension, with some reported cases of hydrocephalus, as well as stroke. [23-29] Spinal NCC can occur when cysts are located within the subarachnoid space or within the parenchyma of the spinal cord proper. The most common clinical presentations of spinal NCC are myelopathy and progressive weakness caused by cord compression, though this may differ depending on relevant spinal levels. [2,3,5,6] The subject of our case report is an interesting presentation of NCC where the initial radiologic findings were more suggestive of other non-parasitic cystic pathology, but was later found to have concurrent subarachnoid and spinal NCC. Ultimately this case presents an excellent teaching opportunity and addition to the discussion surrounding NCC

Case

Our patient is a 24-year-old male without known past medical history who presented with 2 years of chronic headache and 2 months of blurry vision in the left eye. In particular, one week before presentation he developed persistent nausea and vomiting associated with constant left sided headaches. He was seen by an ophthalmologist, who noted papilledema and sent him to the emergency department. On exam, he was awake, alert, and fully oriented, following commands full strength without any focal neurologic deficit or sensory changes.

He was born in Guatemala and moved to the United States approximately one year before presentation and he now works as a dishwasher. He denied any known infectious history, such as known tuberculosis exposure. He stated that when he was 10 years old he had severe head trauma, though did undergo any medical evaluation at that time. He previously lived with his family on a farm with many animals, including dogs, cats, pigs, and cows. A tuberculosis-specific enzyme-linked immunospot assay (T-SPOT.TB) was positive for tuberculosis (TB) in the Emergency Department.

MRI brain with and without contrast was obtained, which revealed aqueductal stenosis with associated enlargement of the bilateral lateral and third ventricles, as well as mild transependymal edema consistent with obstructive hydrocephalus. He was also found to have bilateral 1.5 cm, peripherally-enhancing cystic lesions of the cerebellopontine angle with associated mass effect, initially described as arachnoid cysts, as well as a left sided foramen magnum cyst (**Figure 1**). A right frontal external ventricular drain (EVD) was placed and cerebrospinal fluid (CSF) was sent for analysis, with mildly elevated white blood cell (WBC) count (60/mm3), normal glucose (56 mg/dL) and elevated protein (126 mg/dL). Infectious disease was consulted and recommended a CSF meningitis/encephalitis panel, in addition to broad serum antigen testing, with the highest concern for TB meningitis. CSF TB polymerase chain reaction (PCR) testing was negative. Computed tomography (CT) scan of the chest revealed a left upper lobe lesion consistent with calcified granuloma. He remained clinically well during this time and underwent right ventriculoperitoneal (VP) shunt placement before discharge home with planned clinic follow up.

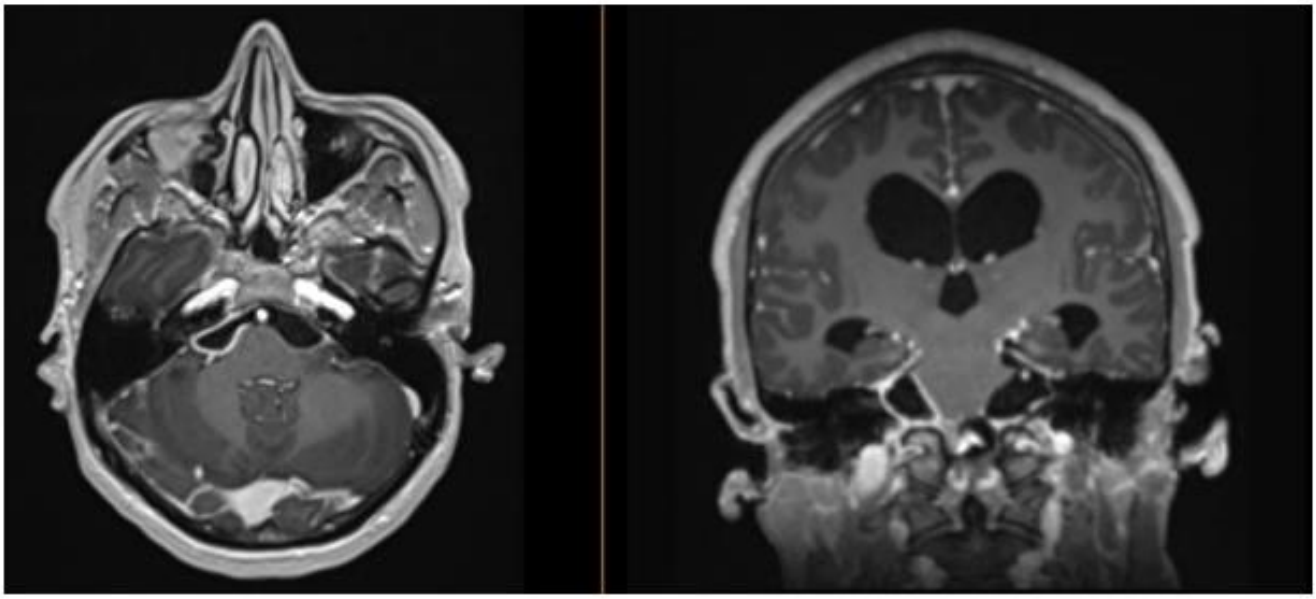
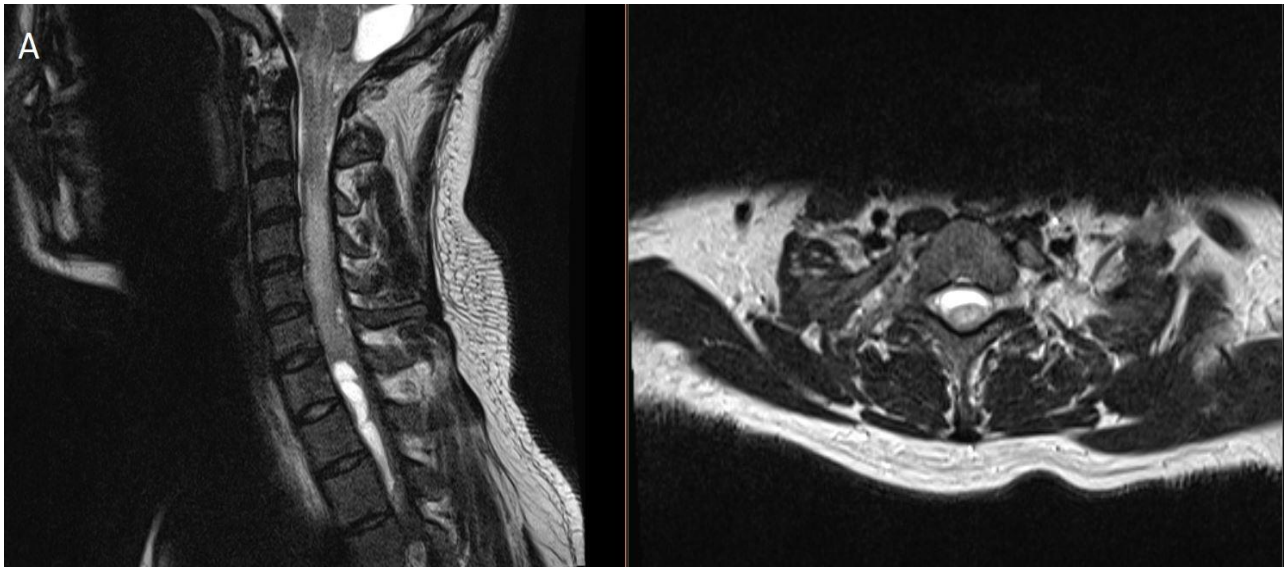


Figure 1: Post-contrast T1-weighted MRI of the brain, with axial and coronal sections revealing bilateral peripherally enhancing cystic lesions in the cerebellopontine angles and associated obstructive hydrocephalus.

Approximately 10 days post-insertion, he returned with fever, chills, nausea, and headaches concerning for sepsis. He was started on empiric antibiotics and underwent VP shunt tapping, lumbar puncture, and VP shunt externalization. CSF revealed very elevated WBC (961/mm3), low glucose (<4 mg/dL), and protein (>2500 mg/dL). He was also started on a central nervous system (CNS) TB regimen, including isoniazid and pyridoxine, rifampin, pyrazinamide, and levofloxacin. Repeat CSF testing revealed improvement in values and the patient underwent VP shunt internalization and revision. He was discharged on long term anti-mycobacterial treatment, though he was lost to follow up.

Approximately two months after VP shunt revision, he presented to the ED with 3 days of urinary retention and right sided weakness (R hang grip strength ⅗, otherwise ⅘ strength diffusely) and diminished right upper and lower extremity sensation. An MRI of the spine revealed multiple cystic lesions, with associated mass effect on the cervical and thoracic cord. The two largest lesions were eccentric to the right near the cervicothoracic and thoracolumbar junctions, for which the patient underwent urgent right C7-T1 and T10-T11 hemilaminotomies for cyst resection (**Figure 2**). Pathology was consistent with neurocysticercosis and serum cysticercosis IgG was positive. Postoperatively, his strength improved with right lower extremity (RLE) full strength and his right upper extremity (RUE) with handgrip 4+/5 and otherwise full strength, with mild persistent paresthesias. He was started on long term albendazole and praziquantel, as well as latent TB treatment, with plan for outpatient follow up and possible enrollment in neurocysticercosis clinical trials.



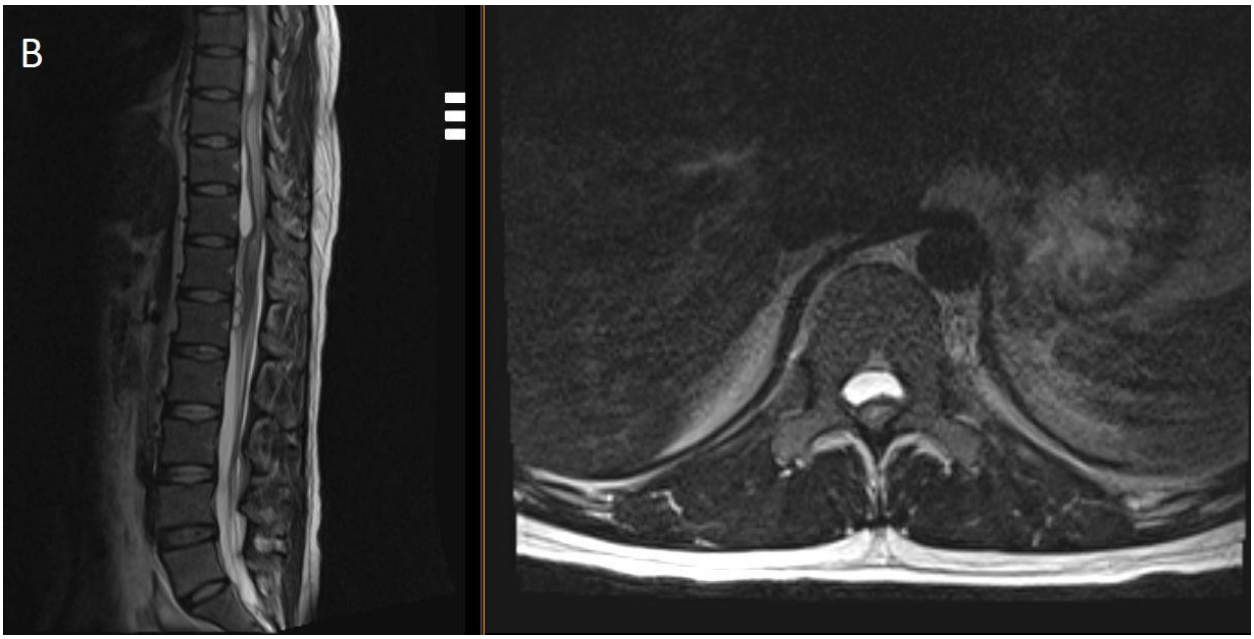


Figure 2: T2-weighted sagittal and axial MRI of the spine at the A.) C7-T1 and B.) T11 levels revealing right-sided hyperintense, cystic lesions with notable mass effect on the spinal cord with associated signal change.

Discussion

NCC may be easily mistaken for other pathologies, such as arachnoid cysts, tumors, or in this case manifestations of TB meningitis. In our patient, the initial imaging was read as arachnoid cyst and the Infectious Disease work up did not point to NCC. Park et al. documented a case of spinal NCC in a 72-year-old man which was initially believed to be either a herniated disk or a subarachnoid tumor, given a history that was not initially suggestive of NCC, such as not hailing from a traditionally endemic country (Republic of Korea), as well as non-pathognomonic imaging studies. [6] Pal et al. reported a similar case in a 44-year-old man who was planned for an arachnoid cyst excision, though during surgery it was discovered to be multiple cystic lesions, later confirmed to be spinal NCC. The preoperative MRI showed non-enhancing elongated cystic lesions which were more characteristic of an arachnoid cyst rather than NCC. [5] Though quite rare, NCC should be included in the differential diagnosis for patients that are presenting with arachnoid cysts, even when not presenting from naturally endemic areas or if there is non-specific imaging studies.

Radiographic Diagnostics

Magnetic resonance Imaging (MRI) and CT allow for greater detection of NCC and current guidelines advocate for the use of both studies when available.[14] As stated, NCC has varying radiographic presentations depending on the stage of larval development.[33] Raibagkar *et al* documents and discusses the intricacies and nuances of the radiographic impacts of NCC larval staging. The initial stage is the vesicular stage which is described as one or more spherical cystic lesions with a central hyperintense/hyperdense focus (scolex); this stage should present with no surrounding edema or contrast enhancement. Upon entering the subarachnoid space these NCC cysts are referred to as racemose cysts. Once within the brain parenchyma this becomes concerning for neurocysticercosis encephalitis and non-encephalitic NCC, with the distinction being edema surrounding the cystic lesions which is characteristic of NCC encephalitis. Colloidal and granular (also known as granular nodular) stages display thick-walled contrast-enhancing lesions that have noted surrounding edema; the thicker the wall and the greater contrast enhancement is the distinction between the two stages with granular being the greater of the two. There is also a calcified stage (also known as calcified nodular) that is identified as punctate hyperdense lesions on CT. CT has greater sensitivity for NCC once in this calcified stage, while MRI possesses greater utility for staging and identification of cysts, in particular those that are within the subarachnoid space and the ventricular system.[15] *Higuera-Calleja et al*. performed a study testing the diagnostic merit of intrathecal gadolinium MRI as a possible radiographic technique for NCC detection and visualization. Their study concludes that it can be a particularly useful tool for the diagnosis of intraventricular and subarachnoid NCC, though admittedly this is not a standard imaging modality.[4] The initial read on the imaging studies described arachnoid cysts, which when combined with somewhat atypical features (i.e. mild peripheral contrast enhancement) may raise suspicion for NCC or another diagnosis.

Medical Management: Cysticidals

This patient was ultimately placed on a regimen of albendazole and praziquantel which are both antihelminthic agents used in the treatment of NCC. [10,12,17-20,34] Albendazole is the first line treatment for NCC, which inhibits glucose channels in the helminthic cell membrane, reducing glucose uptake and leading to death of the parasite. [17,45] Albendazole has also been shown to penetrate certain areas, such as the eyes and cerebral ventricles, that other antiparasitic agents do not. Multiple studies have found that albendazole is considered the superior agent when compared to praziquantel alone. [12,16,20,24,26,27,31,34] Praziquantel has also been shown to be effective in the treatment of NCC and has particular utility when treating the intraparenchymal variant of the disease. Praziquantel's mechanism of action is not well understood in the treatment of larvae as seen in NCC. An important consideration for prescribers is that praziquantel may have its plasma levels affected by interactions with common therapeutic agents used in NCC treatment, such as antiepileptics (phenytoin, carbamazepine, phenobarbital, and primidone) or corticosteroids, such as dexamethasone.[45] A study conducted by *Mahanty et al.* theorizes a potential synergistic and beneficial therapeutic effect with combined praziquantel and albendazole therapy, which our patient is taking as they pursue clinical trials. They also proposed that alkaline phosphatase microassay could be as a quantitative marker of therapeutic effects of anthelmintic agents such as Albendazole and Praziquantel.[10] This can be useful in the treatment of NCC because it will give clinicians a quantitative measure in order to

ensure therapeutic levels of these agents are reached inorder to treat this difficult disease.

Surgical management

NCC, specifically the extraparenchymal presentation of the disease, may be associated with increased intracranial pressure and hydrocephalus, as documented in this case. Multiple studies have described hydrocephalus occurring at onset of disease in 16-51% of patients with NCC, though this increases to closer to 64-72% in patients with the extraparenchymal form. [21-24,46] This patient was found to have developed obstructive hydrocephalus and was treated initially with EVD placement and eventual VP shunting. *Cruz. et al.* conducted a study in which 108 patients with definitive extraparenchymal NCC underwent VP shunt placement, of which nearly 80% presented with hydrocephalus. Their study found that 44.4% of VP shunts experienced some form of dysfunction and the vast majority of these occurred within the first year of placement (66.7%). Clinical impact was determined via the use of The Karnofsky Performance Scale (KPS) which is a clinician reported a score between 0 and 100 (0 is death, while 100 is normal) which assess performance and functionality. Patients 2 years after VP shunt placement with regular follow up showed a KPS of 84.6 with a standard deviation of ±15.2. The author's compared the outcomes of their 108 patients compared to recorded outcomes of other published works covering VP shunting in NCC patients which had historical showed poorer outcomes some which showed upwards of 50% mortality within the first two years of VP shunt placement.[27] Their study ultimately found that VP shunts showed a statistically and clinically significant improvement in patients with extraparenchymal NCC that developed hydrocephalus and only documented one patient dying due to factors relevant to VP shunt placement within that two year period. Endoscopic tools have also allowed for additional management of hydrocephalus via endoscopic third ventriculostomy.[46] This procedure has been used in the treatment of NCC related hydrocephalus and is an additional option for treatment in this regard, though it does have risk of failure in some cases due to pronounced inflammatory reactions which can lead to the close of the ostomy site.[46] *Hamamoto Filho et al.* provided additional considerations surrounding the surgical treatment of NCC with regard to the surgical removal of cysts [46]As discussed earlier, endoscopic tools have allowed for additional means of treating conditions such as NCC, so when surgeons are performing the endoscopic third ventriculostomy they are able to extract intraventricular cysts during the operation.[46] Depending on the locations of cysts there are several options which can be used for cyst extraction. Cysts which are located within the lateral and third ventricles can be extracted using rigid endoscopes, while cysts within the fourth ventricle can be managed with flexible endoscopes.[46] Though fourth ventricle cysts can also be extracted via telovelar approach posterior fossa craniotomy.[46] *Jang et al.* documented a case of recurrent primary spinal NCC in which the patient had previously only received surgical therapy, concluding that subarachnoid NCC is more optimally treated with the combination of surgery and medication, such as albendazole.[3] Patients that are found to have spinal NCC commonly are found to have concurrent intracranial disease and it is believed that it is due to larval migration through the ventricular system. When spinal NCC is present it has been found that approximately 80% of the time cysts are located intradural within the subarachnoid space.[3] In these patients it is important to screen for both intracranial as well as spinal NCC due to their high likelihood of occurring concurrently.

Conclusion

For our patient, an initially clinically uncertain case of suspected TB meningitis and obstructive hydrocephalus thought secondary to an atypical arachnoid cyst was treated with CSF diversion and antitubercular therapy. Upon representation with right sided weakness and multiple areas of spinal cord compression by cystic lesions, he underwent urgent decompression with pathology concerning for NCC. This reinforces the need for a broad differential diagnosis in these scenarios, as well as the importance of close Infectious Disease follow up for patients diagnosed with concurrent cranial and spinal NCC.

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References

1. Hawk MW, Shahlaie K, Kim KD, Theis JH. Neurocysticercosis: a review. *Surg Neurol*. 2005;63(2):123-132. doi:10.1016/j.surneu.2004.02.033
2. Leite CC, Jinkins JR, Escobar BE, et al. MR imaging of intramedullary and intradural-extramedullary spinal cysticercosis. *AJR Am J Roentgenol*. 1997;169(6):1713-1717. doi:10.2214/ajr.169.6.9393195
3. Jang JW, Lee JK, Lee JH, Seo BR, Kim SH. Recurrent primary spinal subarachnoid neurocysticercosis. *Spine (Phila Pa 1976)*. 2010;35(5):E172-E175. doi:10.1097/BRS.0b013e3181b9d8b6
4. Higuera-Calleja J, Góngora-Rivera F, Soto-Hernández JL, et al. Intrathecal gadodiamide for identifying subarachnoid and ventricular neurocysticercosis. *Trop Med Int Health*. 2015;20(7):930-933. doi:10.1111/tmi.12495
5. Pal A, Biswas C, Ghosh TR, Deb P. A rare case of recurrence of primary spinal neurocysticercosis mimicking an arachnoid cyst. *Asian J Neurosurg*. 2017;12(2):250-252. doi:10.4103/1793-5482.144176
6. Park YS, Lee JK, Kim JH, Park KC. Cysticercosis of lumbar spine, mimicking spinal subarachnoid tumor. *Spine J*. 2011;11(4):e1-e5. doi:10.1016/j.spinee.2011.02.009
7. García HH, Gonzalez AE, Evans CA, Gilman RH; Cysticercosis Working Group in Peru. Taenia solium cysticercosis. *Lancet*. 2003;362(9383):547-556. doi:10.1016/S0140-6736(03)14117-7
8. Rajshekhar V, Raghava MV, Prabhakaran V, Oommen A, Muliyil J. Active epilepsy as an index of burden of neurocysticercosis in Vellore district, India. *Neurology*. 2006;67(12):2135-2139. doi:10.1212/01.wnl.0000249113.11824.64
9. Garcia HH, Del Brutto OH; Cysticercosis Working Group in Peru. Neurocysticercosis: updated concepts about an old disease. *Lancet Neurol*. 2005;4(10):653-661. doi:10.1016/S1474-4422(05)70194-0
10. Mahanty S, Paredes A, Marzal M, et al. Sensitive in vitro system to assess morphological and biochemical effects of praziquantel and albendazole on Taenia solium cysts. *Antimicrob Agents Chemother*. 2011;55(1):211-217. doi:10.1128/AAC.00761-10
11. Rajshekhar V. Surgical management of neurocysticercosis. *Int J Surg*. 2010;8(2):100-104. doi:10.1016/j.ijsu.2009.12.006
12. Cruz M, Cruz I, Horton J. Albendazole versus praziquantel in the treatment of cerebral cysticercosis: clinical evaluation. *Trans R Soc Trop Med Hyg*. 1991;85(2):244-247. doi:10.1016/0035-9203(91)90041-v
13. Bustos J, Gonzales I, Saavedra H, Handali S, Garcia HH; Cysticercosis Working Group in Peru. Neurocysticercosis. A frequent cause of seizures, epilepsy, and other neurological morbidity in most of the world. *J Neurol Sci*. 2021;427:117527. doi:10.1016/j.jns.2021.117527
14. White AC Jr, Coyle CM, Rajshekhar V, et al. Diagnosis and Treatment of Neurocysticercosis: 2017 Clinical Practice Guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and

Hygiene (ASTMH). *Clin Infect Dis*. 2018;66(8):e49-e75. doi:10.1093/cid/cix1084
15. Raibagkar P, Berkowitz AL. The Many Faces of Neurocysticercosis. *J Neurol Sci*. 2018;390:75-76. doi:10.1016/j.jns.2018.04.018
16. Sotelo J, Escobedo F, Rodriguez-Carbajal J, Torres B, Rubio-Donnadieu F. Therapy of parenchymal brain cysticercosis with praziquantel. *N Engl J Med*. 1984;310(16):1001-1007. doi:10.1056/NEJM198404193101601
17. Houpt ER, Chaudhry O. Chapter 83 - protozoan and helminthic infections. In: Waldman SA, Terzic A, Egan LJ, et al, eds. *Pharmacology and therapeutics.* Philadelphia: W.B. Saunders; 2009:1171-1186. https://www.sciencedirect.com/science/article/pii/B9781416032915500871.10.1016/B978-1-4160-3291-5.50087-1.
18. Monk EJM, Abba K, Ranganathan LN. Anthelmintics for people with neurocysticercosis. *Cochrane Database Syst Rev*. 2021;6(6):CD000215. Published 2021 Jun 1. doi:10.1002/14651858.CD000215.pub5
19. Takayanagui OM, Jardim E. Therapy for neurocysticercosis. Comparison between albendazole and praziquantel. *Arch Neurol*. 1992;49(3):290-294. doi:10.1001/archneur.1992.00530270106026
20. Sotelo J, Escobedo F, Penagos P. Albendazole vs praziquantel for therapy for neurocysticercosis. A controlled trial. *Arch Neurol*. 1988;45(5):532-534. doi:10.1001/archneur.1988.00520290064015
21. Sinha S, Sharma BS. Neurocysticercosis: a review of current status and management. *J Clin Neurosci*. 2009;16(7):867-876. doi:10.1016/j.jocn.2008.10.030
22. Coyle CM, Tanowitz HB. Diagnosis and treatment of neurocysticercosis. *Interdiscip Perspect Infect Dis*. 2009;2009:180742. doi:10.1155/2009/180742
23. Matushita H, Pinto FC, Cardeal DD, Teixeira MJ. Hydrocephalus in neurocysticercosis. *Childs Nerv Syst*. 2011;27(10):1709-1721. doi:10.1007/s00381-011-1500-3
24. Sotelo J. Clinical manifestations, diagnosis, and treatment of neurocysticercosis. *Curr Neurol Neurosci Rep*. 2011;11(6):529-535. doi:10.1007/s11910-011-0226-7
25. Cuetter AC, Garcia-Bobadilla J, Guerra LG, Martinez FM, Kaim B. Neurocysticercosis: focus on intraventricular disease. *Clin Infect Dis*. 1997;24(2):157-164. doi:10.1093/clinids/24.2.157
26. Sotelo J, Marin C. Hydrocephalus secondary to cysticercotic arachnoiditis. A long-term follow-up review of 92 cases. *J Neurosurg*. 1987;66(5):686-689. doi:10.3171/jns.1987.66.5.0686
27. Cruz L, Pacheco E, Soto W, et al. Neurocysticercosis and hydrocephalus: the value of ventriculoperitoneal shunting in its management. *Trans R Soc Trop Med Hyg*. 2023;117(11):773-779. doi:10.1093/trstmh/trad029
28. Hamamoto Filho PT, Rodríguez-Rivas R, Fleury A. Neurocysticercosis: A Review into Treatment Options, Indications, and Their Efficacy. *Res Rep Trop Med*. 2022;13:67-79. Published 2022 Dec 29. doi:10.2147/RRTM.S375650
29. Hasan R, Iaia A, Flores C. Ischaemic stroke induced by neurocysticerosis, presenting as a clinical and radiological dilemma. *BJR Case Rep*. 2016;2(3):20150254. Published 2016 Jul 28. doi:10.1259/bjrcr.20150254
30. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019 [published correction appears in Lancet. 2020 Nov 14;396(10262):1562]. *Lancet*. 2020;396(10258):1204-1222. doi:10.1016/S0140-6736(20)30925-9
31. Hamamoto Filho PT, Rodríguez-Rivas R, Fleury A. Neurocysticercosis: A Review into Treatment Options, Indications, and Their Efficacy. *Res Rep Trop Med*. 2022;13:67-79. Published 2022 Dec 29. doi:10.2147/RRTM.S375650
32. Coyle CM. Neurocysticercosis: an update. *Curr Infect Dis Rep*. 2014;16(11):437. doi:10.1007/s11908-014-0437-6
33. 33.Del Brutto OH. Human Neurocysticercosis: An Overview. *Pathogens*. 2022;11(10):1212. Published 2022 Oct 20. doi:10.3390/pathogens11101212
34. Wu W, Jia F, Wang W, Huang Y, Huang Y. Antiparasitic treatment of cerebral cysticercosis: lessons and experiences from China. *Parasitol Res*. 2013;112(8):2879-2890. doi:10.1007/s00436-013-3459-3
35. Trevisan C, Sotiraki S, Laranjo-González M, et al. Epidemiology of taeniosis/cysticercosis in Europe, a systematic review: eastern Europe [published correction appears in Parasit Vectors. 2019 Feb 18;12(1):84]. *Parasit Vectors*. 2018;11(1):569. Published 2018 Oct 30. doi:10.1186/s13071-018-3153-5
36. Rajshekhar V. Evolution of concepts in the management of cysticercosis of the brain: Then (1970) and now (2018). *Neurol India*. 2018;66(4):919-927. doi:10.4103/0028-3886.236969
37. Owolabi LF, Adamu B, Jibo AM, Owolabi SD, Imam AI, Alhaji ID. Neurocysticercosis in people with epilepsy in sub-saharan africa: A systematic review and meta-analysis of the prevalence and strength of association. *Seizure*. 2020;76. doi: 10.1016/j.seizure.2020.01.005.
38. Mwape KE, Blocher J, Wiefek J, et al. Prevalence of Neurocysticercosis in People with Epilepsy in the Eastern Province of Zambia. *PLoS Negl Trop Dis*. 2015;9(8):e0003972. Published 2015 Aug 18. doi:10.1371/journal.pntd.0003972
39. Melki J, Koffi E, Boka M, Touré A, Soumahoro MK, Jambou R. Taenia solium cysticercosis in West Africa: status update. La cysticercose à Taenia solium en Afrique de l'Ouest : état des lieux. *Parasite*. 2018;25:49. doi:10.1051/parasite/2018048
40. Debacq G, Moyano LM, Garcia HH, et al. Systematic review and meta-analysis estimating association of cysticercosis and neurocysticercosis with epilepsy. *PLoS Negl Trop Dis*. 2017;11(3):e0005153. Published 2017 Mar 7. doi:10.1371/journal.pntd.0005153
41. Brizzi K, Pelden S, Tshokey T, et al. Neurocysticercosis in Bhutan: a cross-sectional study in people with epilepsy. *Trans R Soc Trop Med Hyg*. 2016;110(9):517-526. doi:10.1093/trstmh/trw066
42. Carod JF, Dorny P. Cysticercosis in Madagascar. *J Infect Dev Ctries*. 2020;14(9):931-942. Published 2020 Sep 30. doi:10.3855/jidc.13450
43. Garcia HH, Gonzalez AE, Gilman RH. Taenia solium Cysticercosis and Its Impact in Neurological Disease. *Clin Microbiol Rev*. 2020;33(3):e00085-19. Published 2020 May 27. doi:10.1128/CMR.00085-19
44. Ong S, Talan DA, Moran GJ, Mower W, Newdow M, Tsang VC, Pinner RW; EMERGEncy ID NET Study Group. Neurocysticercosis in radiographically imaged seizure patients in U.S. emergency departments. Emerg Infect Dis. 2002 Jun;8(6):608-13. doi: 10.3201/eid0806.010377. PMID: 12023918; PMCID: PMC2738481.
45. Bittencourt PRM, Gracia CM, Martins R, Fernandas AG, Diekmann HW, Jung W. Phenytoin and carbamazepine decrease oral bioavailability of praziquantel. *Neurology*. 1992;42(3):492. https://doi.org/10.1212/WNL.42.3.492. doi: 10.1212/WNL.42.3.492.

46. Hamamoto Filho PT, Norcia LF, Fleury A, Zanini MA. Current Role of Surgery in the Treatment of Neurocysticercosis. *Pathogens*. 2024;13(3):218. Published 2024 Feb 29. doi:10.3390/pathogens13030218

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