Received: 22 August 2023 | Revised: 23 October 2023 | Accepted: 26 October 2023

DOI: 10.1002/ccr3.8166

CASE REPORT

Clinical Case Reports Open Access WILEY

Neurocysticercosis mimicking craniopharyngioma: A case report

Aliya F. Rehman[1] | **Alex F. Lazo-Vasquez**[2] | **Parjanya K. Bhatt**[3] | **Tanya Quiroz**[1] | **Joelle-Ann Joseph**[4] | **Sibel Gultekin**[1] | **Nadine Montreuil**[4] | **Candice A. Sternberg**[4] | **Folusakin Ayoade**[4]

[1]Jackson Health System, Miami, Florida, USA

[2]Atlantic Health System, Morristown, New Jersey, USA

[3]B.J. Medical College, Ahmedabad, India

[4]University of Miami Miller School of Medicine, Miami, Florida, USA

Correspondence
Folusakin Ayoade, University of Miami Miller School of Medicine, 1120, NW 14th Street, Miami, FL 33136, USA.
Email: fxa375@med.miami.edu

Key Clinical Message

In patients with appropriate epidemiological risk factors, neurocysticecosis should be considered as part of the differential diagnosis of suprasellar or parasellar mass lesions. As neuroimaging findings can be nonspecific, serology may be helpful, but when still in doubt, brain biopsy, and histopathology may be necessary to make the correct diagnosis.

Abstract

Neurocysticercosis (NCC) is a well-documented central nervous system helminth infection that is, frequently observed in developing countries. Known sites of NCC infection include the highly vascular gray-white matter junction, basal cistern, brain parenchyma, subarachnoid space, ventricular system, and spinal cord. This case highlights an uncommon yet intriguing site of NCC infection within the suprasellar area, which presented with similar clinical and imaging characteristics as suprasellar masses or lesions. The 44-year-old female initially complained of headaches and nausea that persisted for 5 years and progressed to vision problems and short-term memory loss. A craniopharyngioma was initially suspected, based on imaging findings of a partially calcified suprasellar tumor. However, cysticercosis was confirmed by histopathology and serological testing positive for Cysticercus IgG antibodies. The patient was successfully treated with albendazole and tapering doses of steroids, which improved her presenting symptoms and resolved prior imaging findings. This case serves as a reminder to consider NCC in the differential diagnosis of sellar and suprasellar masses or lesions, particularly when an epidemiologic risk factor is present.

KEYWORDS

craniopharyngioma, Cysticercus or *Taenia solium*, neurocysticercosis, suprasellar mass

1 | INTRODUCTION

Neurocysticercosis (NCC) is a helminthic infection of the central nervous system that is mostly observed in developing countries in the setting of poor sanitary conditions. Anatomically, NCC can be parenchymal, extra-parenchymal, or both. NCC typically affects the highly vascular gray-white matter junction, basal cistern, brain parenchyma, subarachnoid space, ventricular system, or spinal cord. Rarely, NCC can also affect the suprasellar regions and mimic clinical and radiological manifestations of noninfectious suprasellar lesions, space-occupying masses, and malignancies.[1,2] This unique presentation of NCC is easily missed, misdiagnosed, and often not considered by clinical providers. NCC is a neglected tropical disease and results in significant comorbidity when undiagnosed.[3] Here, we explore the diagnostic challenges of a patient who presented with a suprasellar mass lesion initially thought to be craniopharyngioma but later confirmed to be NCC on histopathology.

2 | CASE REPORT

This was a 44-year-old female who was referred to our hospital due to persistent headaches and nausea for 5 years. She reported progressively worsening vision in both eyes, as well as short-term memory loss over this time period. She had an MRI of the brain at an outside facility which showed a suprasellar mass with calcifications. This was thought to represent a craniopharyngioma requiring neurosurgical evaluation and intervention, so the patient was referred to our institution. The patient was originally from Honduras and moved to the United States about 20 years prior. Since then, she frequently traveled back and forth to Honduras. She had no fevers, night sweats, rash, oral, or genital ulcers, weight loss, or sick contacts. She had no other significant medical history, had no pets, and denied tobacco smoking.

On admission, her vital signs were within normal limits. A physical exam was significant for decreased peripheral vision in both eye fields, but no other neurological focal deficits were identified. Formal eye exam by ophthalmology did not show any signs of ocular cysticercosis. Initial laboratory revealed a white blood cell count of 7000 cells/mcL, hemoglobin of 12 g/dL, platelets of 333,000 cells/mcL, creatinine of 1 mg/dL, glucose of 109 mg/dL, aspartate aminotransferase of 111 units/L, and alanine aminotransferase of 149 units/L. The only hormonal deficiencies identified were low cortisol of 1.1 mcg/dL and low adrenocorticotropic hormone (ACTH) of 5.8 pg/mL. MRI of the brain showed a partially calcified suprasellar/parasellar mass with both solid and cystic components, the largest segment measuring approximately 1.0 × 1.3 cm in the right parasellar region (Figure 1A,B).

The patient underwent a right-sided pterional craniotomy for resection of the mass, which was found to be significantly adherent to all surrounding vascular and neural structures, including the optic chiasm, which was considered to be quite unusual. Intraoperative frozen sections were obtained which showed fibrotic capsule and inflammation, and the possibility of a craniopharyngioma capsule was entertained. The surgery was later aborted due to the unusual characteristics of the mass, but the optic chiasm was decompressed and additional tissue samples were sent for histopathology. The permanent tissue sections showed no squamous or basaloid epithelium, wet keratin, or stellate reticulum. Instead, they showed a nonviable, partially calcified lesion. The Periodic acid–Schiff (PAS) stain highlighted the layers of a cyst wall, which was compatible with Cysticercus, leading to the final diagnosis of NCC (Figure 2A,B). Cytokeratin cocktail, B-catenin, and BRAF V600E immunohistochemical studies were all negative.

Based on these findings, additional serological testing was requested. Cysticercus IgG antibody testing via western Blot (Quest Diagnostic-Infectious disease, Inc) was positive with 50, 42–39, and 24 kilodaltons (Kd) specific bands present. According to the test instrument, the detection of antibodies to any six specific *Taenia solium* glycoprotein bands of molecular weights 50, 42–39, 24, 21, 18, and 14 Kd is interpreted as a positive result. Our patient had antibodies to three out of six specific bands. Enzyme-linked immunoelectrotransfer blot was not sent, given pathology did confirm the diagnosis. Echinococcus IgG antibody testing was positive but the Western Blot was negative. This was felt to represent cross-reactivity.[4]

The patient was started on treatment for NCC with albendazole 400 mg twice daily by mouth for a total of 10 days, as well as a dexamethasone taper course (starting with 6 mg daily by mouth for 5 days, then decreasing the dose by 2 mg every 3 days until completion). The patient had significant improvement with complete resolution of her headaches, nausea, and visual symptoms upon discharge from the rehabilitation center. However, she had continued difficulty with immediate memory, short-term memory, and working memory for a few years before returning to her baseline. Six months after antiparasitic treatment, a repeat MRI of the brain showed resolution of the previously seen suprasellar cystic mass (Figure 3A,B).

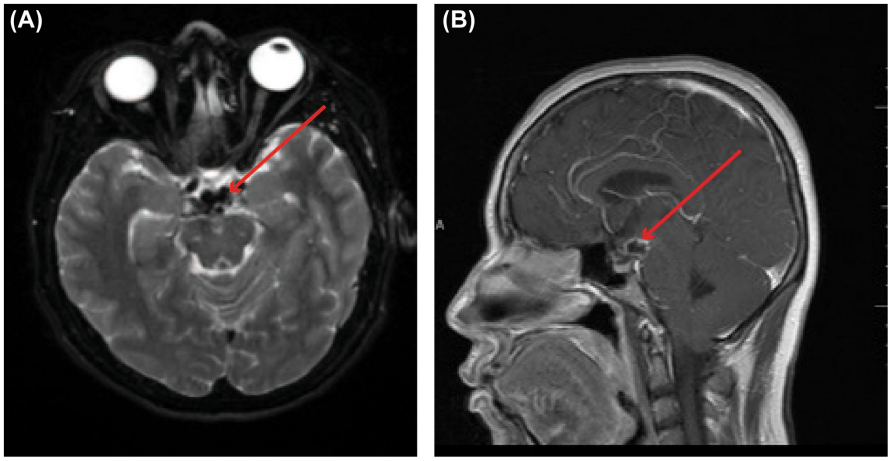

FIGURE 1 (A) T2 axial brain MRI shows the parasellar mass lesion with arrows pointing at areas of hypo intensity suggestive of calcifications. (B) T1 Sagittal brain MRI view shows a partially calcified, ring-enhancing 1.0 × 1.3 cm suprasellar mass with both solid and cystic components.

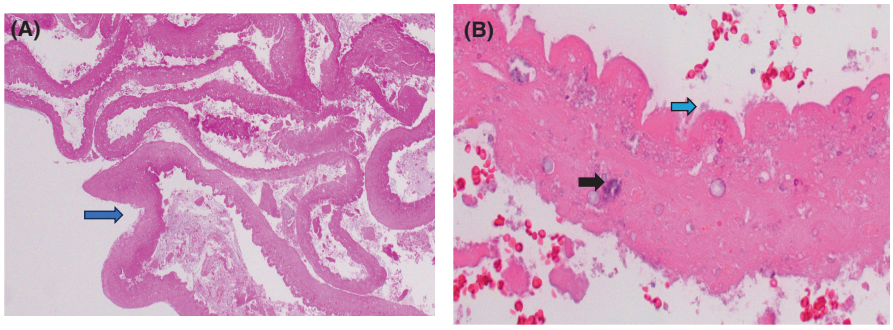

FIGURE 2 (A) PAS stain showing the dead Cysticercus cyst wall, (blue arrow). Magnification ×10. (B) Hematoxylin and eosin stain showing the layers of the Cysticercus cyst wall from top to bottom: outer cuticular layer with hair-like protrusions (blue arrow), middle syncytial layer which is not obvious here due to degradation and the inner reticular layer with mineral deposits (black arrow). Magnification ×400.

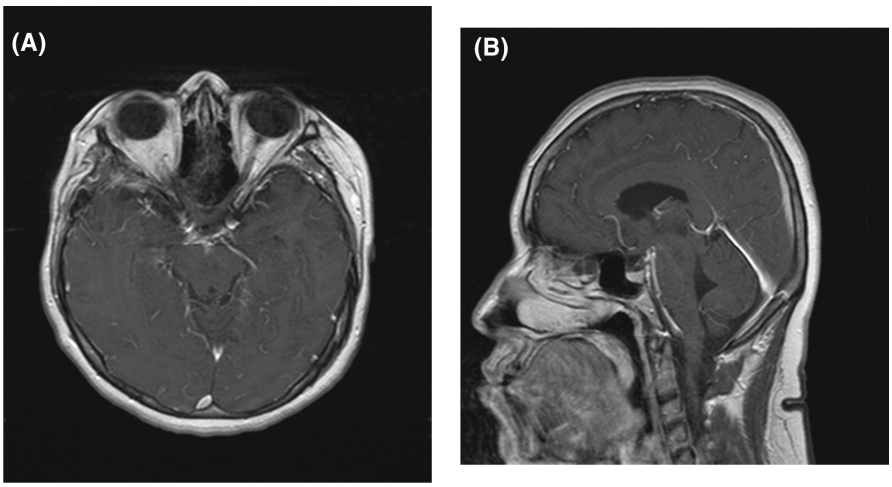

FIGURE 3 (A)T1 axial post-contrast brain MRI 6 months after antiparasitic treatment shows complete resolution of the previously seen suprasellar cystic mass. (B) T1 Sagittal post-contrast brain MRI view 6 months after antiparasitic treatment shows complete resolution of the previously seen suprasellar cystic mass.

3 | DISCUSSION

NCC is the most common parasitic infection of the central nervous system and is caused by the larvae of *Taenia solium*, a pork tapeworm parasite endemic in many developing countries.[5] NCC can cause nonspecific symptoms such as seizures, headaches, and focal neurological deficits,[6] and this vague clinical presentation can make it challenging to successfully diagnose. As a result, many cases of NCC have been misdiagnosed as hydrocephalus, arachnoid cysts, or craniopharyngioma.[2,5,7–9] Moreover, the relatively obscure suprasellar location of a neurocysticercosis lesion can render neuroimaging imprecise, with the potential risk of missing or mischaracterizing these lesions.[2] Our case is one of the very few reported cases of NCC presenting as a suprasellar mass. A literature review

TABLE 1 Literature review of all extra-parenchymal NCC presenting as suprasellar masses.

Age/gender	Main clinical presentation	Diagnosis (CT/MRI/serology/biopsy with Histopathology	Management (antiparasitic/steroids)	References
44/	Progressive worsening vision and short-term memory loss	MRI showed a partially calcified, suprasellar/parasellar mass with both solid and cystic components, Cysticercus IgG antibodies were positive, path showed cyst wall compatible with Cysticercus	Albendazole & dexamethasone	Index case
28/M	Headaches, worsening visual acuity	MRI showed cystic lesion in mid to left side of the suprasellar region extending into the sellar region, histopathology report showed findings compatible with cysticercosis	NA	Shakya et al.[1]
45/F	Headaches, worsening visual acuity	MRI with cystic image in sellar and suprasellar region; pathology with parasitic wall & positive lentil lectin glycoprotein enzyme-linked immunoelectrotransfer blot	NA	Cuellar-Hernandez et al.[15]
39/M	Headaches, decreased visual acuity	MRI showed complex, predominantly cystic suprasellar lesion. CT showed partial calcification of the lesion. Pathology showed necrotic and partially calcified tissue along with cysticerci remnants	Deemed not a candidate for medical treatment due to calcification of the cysts	Snyder et al.[8]
61/F	Headache	CT and MRI showed multiple intracranial cysts in the suprasellar area, several cysts were removed, and path compatible with cysticercosis	Albendazole	Ramirez et al.[16]
31/F	Headaches, deteriorated vision	Imaging revealed a suprasellar localization, autopsy revealed Cysticercus cysts in the cisterns of the posterior cranial fossa and severe lesions of the pia matter	NA	Hullay et al.[17]

Abbreviations: F, female; IgG, immunoglobulin G; M, male; NA, not available.

of previously published extra-parenchymal neurocysticercosis presenting as suprasellar mass is shown in Table 1.

The diagnosis of NCC is usually made with a combination of neuroimaging, clinical findings, and serology. The serologic test of choice is enzyme-linked immunoelectrotransfer blot (EITB) with 95% sensitivity for NCC, but monoclonal antibody-based antigen-detection assays are an alternative option.[10,11] In certain situations, antibody testing can be falsely negative and even EITB can have cross-reactivity with other parasitic infections, especially echinococcosis,[12] as seen in our case. In addition, in the Quest instrument used for our patient, a positive result without reactivity to the 50 and 42–39 glycoprotein bands may reflect cross-reactive antibodies induced by echinococcosis.[4] Our patient had reactivity to both of these glycoprotein bands which further reinforced our NCC diagnosis. There is a potential role of Next-Generation Sequencing as a tool for the diagnosis of NCC, especially in atypical cases as reported by Liu et al., although more studies are needed in this area for utility verification and validation.[13] In our patient, because pathology showed a nonviable, partially calcified lesion with a cystic component and PAS-positive cyst wall, the decision was made to treat for NCC. Antiparasitic therapy is typically warranted for patients with viable and/or degenerating cysts on neuroimaging, regardless of location. In cases where neuroimaging is suggestive of <2 viable or degenerating cystic lesions, treatment consists of albendazole (15 mg/kg/day) in twice-daily doses for 1–2 weeks. Praziquantel (50 mg/kg/day) is added to albendazole for patients with >2 viable/degenerating parenchymal cysts.[14]

In conclusion, NCC can masquerade as a craniopharyngioma when affecting the suprasellar area. Therefore, in patients with epidemiological risk factors, NCC should be considered as part of the differential diagnosis of suprasellar or parasellar mass lesions. Radiographic manifestations of NCC are varied and can lack specificity. In such cases, where neuroimaging findings are consistent but not diagnostic of NCC, serology (EITB) can be useful as part of the confirmatory evaluation. Brain biopsy remains an important element of diagnosis for difficult or unclear cases, and pathologists should specifically look for histopathologic evidence inclusive of NCC on tissue sections, especially in the context of epidemiological risk.

AUTHOR CONTRIBUTIONS

Aliya Rehman: Writing – original draft. **Alex F. Lazo-Vasquez:** Writing – original draft. **Parjanya Bhatt:** Writing – review and editing. **Tanya Quiroz:** Investigation; resources. **Joelle-Ann Joseph:** Writing – review and editing. **Sibel Gultekin:** Investigation; writing – review and editing. **Nadine Montreuil:** Writing – review and editing. **Candice A. Sternberg:** Supervision; writing – review and editing. **Folusakin Ayoade:** Conceptualization; supervision; writing – review and editing.

ACKNOWLEDGMENTS

Special thanks to April Mann, and the Writing Center at the University of Miami, for providing additional editing and proofreading of the manuscript.

FUNDING INFORMATION

This work received no external funding.

CONFLICT OF INTEREST STATEMENT

The authors do not have any conflicts of interest to disclose in relation to this work.

CONSENT STATEMENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

ORCID

Folusakin Ayoade https://orcid.org/0000-0002-3680-1278

REFERENCES

1. Shakya S, Gurung P, Shrestha D, Rajbhandhari P, Pant B. A case of sellar/suprasellar neurocysticercosis mimicking a craniopharyngioma. *Asian J Neurosurg*. 2021;16(1):204-207.
2. Del Brutto OH, Del Brutto VJ. Intrasellar cysticercosis: a systematic review. *Acta Neurol Belg*. 2013;113(3):225-227.
3. Coyle CM, Mahanty S, Zunt JR, et al. Neurocysticercosis: neglected but not forgotten. *PLoS Negl Trop Dis*. 2012;6(5):e1500. doi:10.1371/journal.pntd.0001500
4. Diwan AR, Coker-Vann M, Brown P, et al. Enzyme-linked immunosorbent assay (ELISA) for the detection of antibody to cysticerci of *Taenia solium*. *Am J Trop Med Hyg*. 1982;31(2):364-369. doi:10.4269/ajtmh.1982.31.364
5. Zada G, Lopes MBS, Mukundan S, Laws E. Neurocysticercosis of the Sellar region. In: Zada G, Lopes MBS, Mukundan S Jr, Laws ER Jr, eds. *Atlas of Sellar and Parasellar Lesions: Clinical, Radiologic, and Pathologic Correlations*. Springer International Publishing; 2016:419-422.
6. Rajshekhar V. Neurocysticercosis: diagnostic problems & current therapeutic strategies. *Indian J Med Res*. 2016;144(3):319-326.
7. Musara A, Soko N, Shamu S. Suprasellar cysticercosis cyst with optic nerve compression masquerading as an arachnoid cyst. *Middle East Afr J Ophthalmol*. 2019;26(2):114-116.
8. Snyder MH, Marino AC, Shepard MJ, et al. Neurocysticercosis presenting as an isolated suprasellar lesion. *World Neurosurg*. 2020;141:352-356.
9. Rajpal S, Tomberlin C, Bauer A, Forsythe RC, Burneikiene S. Chiari malformation and hydrocephalus masking neurocysticercosis. *World Neurosurg*. 2018;114:68-71.
10. Del Brutto OH, Nash TE, White AC Jr, et al. Revised diagnostic criteria for neurocysticercosis. *J Neurol Sci*. 2017;372:202-210.
11. Rodriguez S, Wilkins P, Dorny P. Immunological and molecular diagnosis of cysticercosis. *Pathog Glob Health*. 2012;106(5):286-298.
12. Kong Y, Kang SY, Cho SY, Min DY. Cross-reacting and specific antigenic components in cystic fluid from metacestodes of Echinococcus granulosus and *Taenia solium*. *Kisaengchunghak Chapchi*. 1989;27(2):131-139.
13. Liu P, Weng X, Zhou J, et al. Next generation sequencing based pathogen analysis in a patient with neurocysticercosis: a case report. *BMC Infect Dis*. 2018;18(1):113.
14. White AC, 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)1. *Clin Infect Dis*. 2018;66(8):1159-1163.
15. Cuellar-Hernandez JJ, Valadez-Rodriguez A, Olivas-Campos R, et al. Intrasellar cysticercosis cyst treated with a transciliary supraorbital keyhole approach—a case report. *Surg Neurol Int*. 2020;11:436.
16. Ramirez Mdel P, Restrepo JE, Syro LV, et al. Neurocysticercosis, meningioma, and silent corticotroph pituitary adenoma in a 61-year-old woman. *Case Rep Pathol*. 2012;2012:340840.
17. Hullay J, Gombi R, Velok G, Boczán G, Borus F. Intra- and suprasellar tumours: surgical results. *Acta Chir Acad Sci Hung*. 1980;21(1):87-97.

How to cite this article: Rehman AF, Lazo-Vasquez AF, Bhatt PK, et al. Neurocysticercosis mimicking craniopharyngioma: A case report. *Clin Case Rep*. 2023;11:e8166. doi:10.1002/ccr3.8166