# An Unusual Cause of Paediatric Epilepsy in Europe: a Case of Neurocysticercosis

Simon Segers [a], Charles Etobou [b], Françoise Delmelle [b], Marie-Cécile Nassogne [c], Emmanuel Bottieau [d], Nadia Amini [e], Dimitri Van der Linden [f]

[a] Centre Hospitalier de Wallonie Picarde, Emergency Department, Tournai, Belgium
[b] Centre Hospitalier de Wallonie Picarde, Department of Paediatrics, Tournai, Belgium
[c] UCLouvain, Cliniques Universitaires Saint-Luc, Department of Paediatric Neurology, Brussels, Belgium
[d] Institute of Tropical Medicine, Department of Clinical Sciences, Antwerp, Belgium
[e] Centre Hospitalier de Wallonie Picarde, Department of Radiology, Tournai, Belgium
[f] UCLouvain, Cliniques Universitaires Saint-Luc, Department of Paediatric Infectious Diseases and Institut de Recherche Expérimentale et Clinique, Brussels, Belgium

simon.segers@chwapi.be

## Keywords

Neurocysticercosis ; seizures ; Taenia solium ; child : case report.

## Abstract

We describe a case of symptomatic epilepsy in an 8-year-old child of Burundian origin who was admitted to an emergency department in Belgium. Neurocysticercosis is a rare cause of epilepsy outside areas endemic for *Taenia solium*. The 'pig tapeworm' is responsible for neurocysticercosis by larval invasion of the central nervous system. The clinical presentation and therapeutic options are reviewed, with a focus on the severity, stage, and location of the infection.

## Introduction

We describe a case of symptomatic epilepsy in an 8-year-old child of Burundian origin who was admitted to an emergency department in Belgium. Neurocysticercosis is a rare cause of epilepsy outside areas endemic for *Taenia solium*. The 'pig tapeworm' is responsible for neurocysticercosis by larval invasion of the central nervous system. The clinical presentation and therapeutic options are reviewed, with a focus on the severity, stage, and location of the infection.

An 8-year-old boy of Burundian origin was admitted to the paediatric emergency department of Wallonie Picarde Hospital Centre (CHWAPI) in January 2023. While at home, the child exhibited oral automatisms and decreased responsiveness, followed by loss of consciousness and a tonic seizure with urinary incontinence. The emergency team administered benzodiazepines rectally at home, after which the child gradually regained consciousness during his transfer to the hospital.

On admission, the boy was in good general condition, alert, and breathing normally. He had amnesia of the events. General examination was normal except for nasal congestion and a tongue bite. The neurological examination was normal.

The boy had a similar episode one year earlier at school, where he experienced non-specific malaise, loss of responsiveness, and confusion. The local medical team found him to be conscious, oriented, and with normal neurological status. The boy complained of transient occipital headache prior to the loss of responsiveness. He did not exhibit abnormal movements or involuntary urinary incontinence. After a few hours of observation, he was discharged with follow-up in general paediatrics.

There was no contributing medical, surgical or family history. The patient was born and raised in Burundi until the age of 6 years, when he arrived in Belgium in November 2020. He received the recommended vaccination schedule in Burundi, including the BCG vaccine, and then continued with the standard vaccination schedule in Belgium. There was no ongoing chronic treatment.

Blood tests on admission were normal. The electroencephalogram (EEG) performed on the same day revealed slow brain activity for his age, but no ongoing epileptic activity. A 24-hour EEG performed 2 days later was normal.

The diagnostic workup included cerebral imaging with computed tomography scan (CT) and magnetic resonance imaging (MRI), which

**Figure 1**: Cerebral MRI (A, B and C) and CT-scan (D and E). The solid arrow indicates the calcified cysticercus. The hollow arrow indicates a sequela ring of demyelination.

**A**: Axial T2WI shows an hypointense granulomatous punctiform lesion in the left parietal lobe surrounded by a slight vasogenic oedema. **B**: Axial T1 post-contrast reveals peripheral enhancement. **C**: Axial DWI image shows the absence of restricted diffusion, demonstrating the absence of a viable scolex. **D** et **E**: Axial CT slices in bone (D) and parenchymal (E) reconstruction confirming the calcified nature of the lesion.

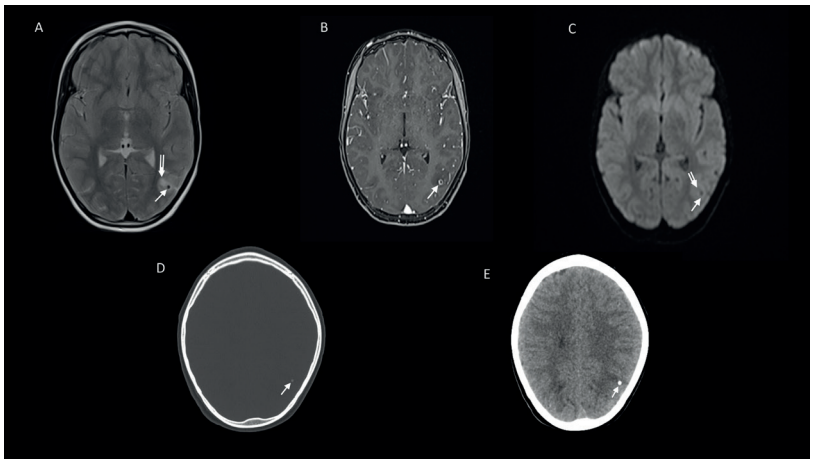

**Table 2:** Laboratory data on admission.

| CEREBROSPINAL FLUID ANALYSIS | | | | |
|---|---|---|---|---|
| **Cytology, chemistry** | | | | |
| **Analysis** | **Unit** | **Index** | **Normal values** | **RESULT** |
| Red blood cells | /µL | | < 10 | 0 |
| White blood cells | /µL | ++ | < 10 | **13** |
| Lymphocytes | % | | 40 - 80 | few |
| Monocytes/macroph. | % | | 15 - 45 | rare |
| Chloride | mEq/L | + | 120 - 130 | **132** |
| Lactate | mg/dL | | 9 - 26 | 12.9 |
| Proteins | mg/L | | 150 - 450 | 279.1 |
| Glucose | mg/dL | - | 50 - 80 | *46.5* |
| **Parasite serology** | | | | |
| **Analysis** | **Unit** | **Index** | **Normal values** | **RESULT** |
| Taenia solium IgG (ELISA) | Ratio | | <1.00 | **3.58** |
| Taenia solum antigen | | | Negative | **26** |
| **Molecular diagnosis** | | | | |
| **Analysis** | **Unit** | **Index** | **Normal values** | **RESULT** |
| PCR multi | | | Negative | Negative |
| E. coli K1, H. influenzae, L. monocytogenes, M. pneumoniae, N. meningitidis, S. agalactiae, S. pneumoniae. | | | | |
| Cytomegalovirus, Enterovirus, H. simplex 1, H. simplex 2, Human herpesvirus 4 (EBV), Human herpesvirus 6, Human parechovirus, Varicella zoster virus, Cryptococcus neoformans/gattii | | | | |
| **Bacteriology** | | | | |
| **Analysis** | **Unit** | **Index** | **Normal values** | **RESULT** |
| Direct examination | | | | Negative |
| Culture | | | | Negative |

revealed small calcifications in the left parietal region, right frontal region and lentiform nucleus bilaterally, and in the quadrigeminal cistern (Figure 1). A subsequent MRI of the spinal cord showed no lesions and an ophthalmological examination was normal. Cerebrospinal fluid (CSF) analysis was positive for intrathecal *Taenia solium* antibodies (ELISA) and antigen (Table 1).

These findings suggested epilepsy secondary to neurocysticercosis (NCC). The diagnosis of NCC was made definitively based on the Del Brutto criteria: parenchymal brain calcifications on neuroimaging (major neuroimaging criterion), detection of specific anticysticercal antibodies or cysticercal antigens by immunodiagnostic tests, clinical manifestations suggestive of NCC and an individual coming from an area with endemic cysticercosis (three major clinical/exposure criteria) (1). The standard therapeutic management of neurocysticercosis with positive antigen testing includes antiparasitic treatment. However, in this case, since the calcifications found by cerebral imaging indicate old cysts without viable parasites and due to the long seizure-free interval, no antiparasitic treatment nor long-term anti-epileptic treatment was initiated.

No stool sample was analysed as the work-up indicated a calcified, late form of NCC, probably acquired before the patient left Burundi. Other family members never presented with symptoms suggestive of cysticercosis or NCC, and no work-up was initiated for them at this time.

## Taenia solium: two clinical presentations

*Taenia solium* is a segmented flatworm that infects pigs and humans. In humans, the adult tapeworm resides in the small intestine and attaches itself to the intestinal wall. The tapeworm's body is made up of proglottids, i.e. segments containing eggs, which are shed in the faeces. Pigs become intermediate hosts when they ingest the eggs found in human faeces. These eggs release larvae that penetrate pig's the intestinal wall and form cysticerci in different tissues, particularly muscles. When humans consume undercooked pork meat, they ingest cysticerci, which will mature and develop into adult worms in the intestine, leading to taeniasis (2). Infected individuals may have mild or no symptoms, but they excrete *Taenia solium* eggs in their faeces. Diagnosis is made by identifying eggs or proglottids in the stool, although the sensitivity of the test is low (around 40%) (3).

Humans are the final hosts of *Taenia solium* and thus the only transmitters of the eggs: human cysticercosis occurs when humans ingest eggs, in areas with suboptimal faecal hygiene, either in the environment or via tapeworm carrier, sometimes themselves (autoinoculation). Food or water is contaminated by the ingested eggs or faecal-oral transmission occurs, and the larvae hatch and migrate to the tissues. Symptoms vary depending on the location of the cysts. Cysts of the brain and eye can cause severe disorders (NCC), while cysts in muscles or skin usually cause mild symptoms (cysticercosis). The diagnosis of cysticercosis involves antibody- or antigen-based serological blood tests, and radiological identification of cysticerci.

Cysticercosis is a common disease in several regions of the world, including Latin America, sub-Saharan Africa, South-East Asia, and parts of India. Assessing taeniasis prevalence is challenging due to the mild symptoms of taeniasis and limited the access to diagnostics in endemic areas. Areas endemic for cysticercosis are defined by the WHO as areas with porcine cysticercosis (4).

## Neurocysticercosis

The term neurocysticercosis refers to the infection of the central nervous system by the larvae of *Taenia solium*, which encyst themselves and form cysticerci. The clinical manifestations vary depending on the host's immune response, as well as the location, number, stage and size of the cysticerci (5).

Cysticerci undergo distinct stages of evolution, which are associated with specific symptoms and imaging characteristics. During the vesicular stage, the cysts are initially small and filled with fluid, in which a scolex can sometimes be identified. These cysts are viable and often asymptomatic, although they can cause headaches or visual disturbances. On neuroimaging, they appear as rounded or oval cysts with a thin wall and an eccentric scolex. During the colloidal stage, cysts become gelatinous and undergo inflammatory transformation. This results in a thickening of the cyst wall and surrounding vasogenic edema. The scolex is often no longer visible. This stage is associated with the onset of more pronounced neurological symptoms, including seizures, and severe attacks of headaches. Finally, the cysts reach the granular-nodular and then calcified stages, during which time the perilesional vasogenic edema resolves and calcium deposits develop. Fully calcified cysts are generally asymptomatic, although they may also cause epileptic seizures. They ca be identified on cerebral CT as hyperdense lesions.

Parenchymal cysts primarily manifest as epilepsy and motor disorders, sometimes accompanied by headaches. The clinical intensity correlates with the stage of cysticerci evolution and their quantity.

Ventricular or subarachnoid cysts often asymptomatic at the initial stage, but may subsequently present with sudden clinical manifestations, including headaches, dizziness, and vomiting when mobile ventricular cysts obstruct cerebrospinal fluid circulation. Racemose NCC is a form of extraparenchymal NCC, that is characterised by the presence of numerous neurocysticerci, which are clustered in appearance. This form of NCC s associated with a poorer prognosis.

Overall, epilepsy is the most common manifestation of NCC, which is the leading cause of late-onset epilepsy in endemic areas.

## Diagnosis

The diagnosis of NCC is complex due to the varying diagnostic criteria according to the disease stage (6). The del Brutto criteria have become nowadays the reference for establishing a diagnosis of NCC (1). A definitive diagnosis is made when the parasite is demonstrated in histological samples or when brain imaging reveals a cystic lesion with a scolex, or on a set of epidemiological, clinical, serological, and imaging criteria, as all elements have variable sensitivity and specificity.

Antibody-based serological tests are poorly sensitive when the parasitic load is low and cannot distinguish active infection from past exposure. Antigen-based assays lack sensitivity but usually reflect the presence of viable cysts for which anthelminthic treatment may be beneficial. These tests performed on the serum sample are unable to differentiate between cysticercosis and NCC. The same tests can be performed on CSF but the correlation between serum and CSF test results is partially dependent on the localisation of the cysticerci and the severity of NCC (7).

Although our patient displayed positive antigen testing on CSF, the low value and the radiological findings being more suggestive of a calcified form of NCC meant that no anthelminthic treatment was considered. Furthermore, as CSF analysis results were obtained early during the work-up, no blood serology was performed.

The sensitivity of brain CT is not optimal for vesicular lesions, which are better identified by MRI. However, CT is superior in the detection of calcifications, which are not common in young patients. The MRI can better predict the stage of the NCC based on the presence of the scolex or surrounding vasogenic edema. The imaging workup will specify the form of the infection based on the location of the lesions.

## Treatment options

A multidisciplinary management approach must be tailored for each patient with NCC, involving neurologists, neurosurgeons and infectious disease specialists (8). Therapeutic options include anthelminthic drugs (albendazole and/or praziquantel), systematically combined with corticosteroids to prevent or treat cerebral inflammation in symptomatic patients. Surgery may be considered for unique, large, critical, or intracranial pressure-inducing cysts. Symptomatic treatment addresses specific symptoms with antiepileptic drugs although no specific epilepsy treatment modality exists for NCC.

With regard to parenchymal forms, the most appropriate treatment varies according to the development stage of the cysticerci (anthelminthic treatment in vesicular stage, combined with corticosteroids) and clinical presentation (with or without antiepileptics). In the case of extra-parenchymal NCC, a more complex case-by-case adaptation is required, frequently involving neurosurgery and a longer course of anthelminthic treatment (9).

Preventive measures include maintaining proper hygiene and treating taeniasis to prevent proglottids release in the environment and ensuring thorough cooking of pork to prevent taeniasis.

The prognosis for NCC is dependent upon a number of factors, including the location, number, severity of symptoms, response to treatment, and the presence of complications. In countries with greater resources, early diagnosis and appropriate treatment have been shown to yield favourable outcomes. Mortality is relatively low, with rates of 0.3% to 3%. However, severe complications such as hydrocephalus and meningitis can result in higher mortality rates.

## Neurocysticercosis in children

NCC occurs mainly in older children because of its mostly asymptomatic latency period. Epilepsy is the most common symptom, raising suspicion especially in endemic areas. Acute headache or atypical neurological symptoms in children may also be suggestive. Occasionally, developmental delay may be the only clinical clue. Due to the reactivity of the child's immune system, inflammatory reactions around cysticerci can be sudden and severe. Ventricular cysticerci are more common in children. Therapeutic approaches are similar to those in adults (10).

Although NCC is a leading cause of acquired epilepsy in endemic areas, the differential diagnoses should not be overlooked. Regardless of the cause, the management of paediatric seizures has the same goals as adult management: crisis management to prevent complications, identification of reversible or immediately life-threatening causes, and prevention of short- and long-term complications.

## Conclusion

The diagnosis of neurocysticercosis is uncommon outside endemic areas for porcine cysticercosis and requires a multidisciplinary evaluation and management. In the emergency room, the occurrence of a first epileptic seizure or the presence of suggestive symptoms in a patient with features consistent with neurocysticercosis should prompt investigation for this diagnosis. Indeed, certain forms or stages of NCC may require specific treatments.

## Conflict of interest

The authors declare that they have no conflict of interest.

REFERENCES


1. Del Brutto OH, Nash TE, White AC, Rajshekhar V, Wilkins PP, Singh G, et al. Revised diagnostic criteria for neurocysticercosis. J Neurol Sci. 2017;372:202–10.
2. Marie C, Petri WA. Infection à Taenia solium et cysticercose (ténia du porc) [Internet]. Le manuel MSD. 2021 [cited 2023 Apr 13]. Available from: https://www.msdmanuals.com/fr/professional/maladies-infectieuses/cestodes-ténias/infection-à-taenia-solium-et-cysticercose-ténia-du-porc.
3. White AC. Cysticercosis: Clinical manifestations and diagnosis [Internet]. UpToDate. 2022 [cited 2023 Apr 13]. Available from: https://www.uptodate.com/contents/cysticercosis-clinical-manifestations-and-diagnosis?search=neurocysticercose&source=search_result&selectedTitle=1~25&usage_type=default&display_rank=1
4. World Health Organization. WHO guidelines on management of taenia solium neurocysticercosis. Geneva; 2021.
5. Takayanagui OM, de Haes TM. Update on the diagnosis and management of neurocysticercosis. Vol. 80, Arquivos de Neuro-Psiquiatria. Associacao Arquivos de Neuro-Psiquiatria; 2022. p. 296–306.
6. Salavracos M. Diagnostic and therapeutic approaches in case of neurocysticercosis in Belgium. Louv Med. 2019 Apr;138(4):239–45.
7. Rodriguez S, Dorny P, Tsang VCW, Pretell EJ, Brandt J, Lescano AG, et al. Detection of Taenia solium Antigens and Anti-T. solium Antibodies in Paired Serum and Cerebrospinal Fluid Samples from Patients with Intraparenchymal or Extraparenchymal Neurocysticercosis. 2009;
8. White AC, Coyle CM, Rajshekhar V, Singh G, Hauser WA, Mohanty A, 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). Vol. 66, Clinical Infectious Diseases. Oxford University Press; 2018. p. e49–75.
9. Hamamoto Filho PT, Rodríguez-Rivas R, Fleury A. Neurocysticercosis: A Review into Treatment Options, Indications, and Their Efficacy. Res Rep Trop Med. 2022 Dec;Volume 13:67–79.
10. de Oliveira RS, Viana DC, Colli BO, Rajshekhar V, Salomão JFM. Pediatric neurocysticercosis. Child's Nerv Syst. 2018 Oct 1;34(10):1957–65.