

NEUROCYSTICERCOSIS: A CONTEMPORARY REVIEW OF EPIDEMIOLOGY, PATHOGENESIS, DIAGNOSIS, AND MANAGEMENT

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Abstract

Neurocysticercosis (NCC), infection of the central nervous system by the larval stage of the pork tapeworm *Taenia solium*, is the most common parasitic disease of the human brain and the leading preventable cause of acquired epilepsy worldwide. Endemic across Latin America, sub-Saharan Africa, and much of Asia, it has become an increasingly frequent imported disease in high-income countries through migration and travel. Clinical expression ranges from silent calcified granulomas to life-threatening cysticercotic encephalitis, ventricular obstruction, and stroke, depending on parasite burden, evolutionary stage, anatomic compartment, and host immune response. Diagnosis rests on neuroimaging interpreted in epidemiological and clinical context, supported by antibody and antigen assays and, increasingly, molecular methods. Management is compartment-specific: antiparasitic drugs (albendazole, praziquantel) with corticosteroid cover for viable disease, antiepileptic therapy for seizures, and neurosurgical or endoscopic intervention for hydrocephalus and extraparenchymal cysts. Because NCC is fundamentally a product of human–pig–environment transmission, control programs combining treatment of human tapeworm carriers, porcine vaccination, and sanitation improvement offer a realistic path toward elimination. This review synthesizes current evidence on the epidemiology, life cycle, pathogenesis, clinical spectrum, diagnosis, treatment, and prevention of NCC, highlighting both the progress of the last four decades and the persistent gaps in evidence and implementation.

1. Introduction: The Most Common Brain Parasite You Have Never Seen

Few diseases so neatly expose the unequal geography of human health as neurocysticercosis. It is at once a leading cause of seizures among billions of people in the developing world and a diagnostic surprise in the emergency departments of New York, London, or Sydney, where it appears as an imported illness in patients from endemic regions. The World Health Organization classifies cysticercosis among the neglected tropical diseases and estimates that *T. solium* causes a substantial share — often quoted at 30% or more — of the burden of acquired epilepsy in endemic settings, with some African studies attributing up to half of late-onset seizures to this single, fully preventable infection.

The clinical arc of NCC is inextricably tied to its biology. Humans develop neurocysticercosis not by eating pork, as folk wisdom sometimes holds, but by ingesting microscopic tapeworm eggs — usually through food or water contaminated with human feces from a tapeworm carrier, occasionally through autoinfection. Once swallowed, the eggs release oncospheres that cross the intestinal wall, enter the bloodstream, and lodge preferentially in the brain, where each develops into a fluid-filled bladder worm (*Cysticercus cellulosae*) roughly a centimeter across. A single asymptomatic person with intestinal taeniasis, shedding eggs in an environment of poor sanitation and free-roaming pigs, can seed an entire community. The disease, in other words, is a mirror of sanitation, poverty, and food systems — which is also why it is, in principle, eradicable.

2. The Parasite and Its Life Cycle

Taenia solium is a cestode with a two-host life cycle in which humans are the definitive (intestinal) host and pigs the usual intermediate host. The adult tapeworm, which can live for years in the human small intestine, releases gravid proglottids containing thousands of eggs; these are passed in feces and contaminate soil, water, and food. Pigs scavenging in such environments ingest the eggs, and larvae encyst in porcine muscle. Humans who eat undercooked 'measly pork' acquire the adult worm — taeniasis, usually a mild or unnoticed intestinal infection. Critically, when humans ingest eggs directly, they act as dead-end intermediate hosts: oncospheres disseminate hematogenously and encyst in human tissue, most dangerously in brain, but also in skeletal muscle, subcutaneous tissue, and eye.

This dual-path transmission explains two of NCC's most characteristic epidemiological features. First, the infection is maintained by human fecal contamination, not by pork consumption — a fact of central importance for prevention. Second, cases cluster in settings where pigs have access to human feces and where open defecation or

inadequate sewage treatment is common; gender and occupation then shape risk, with agricultural workers and women who raise pigs in many endemic communities bearing disproportionate exposure.

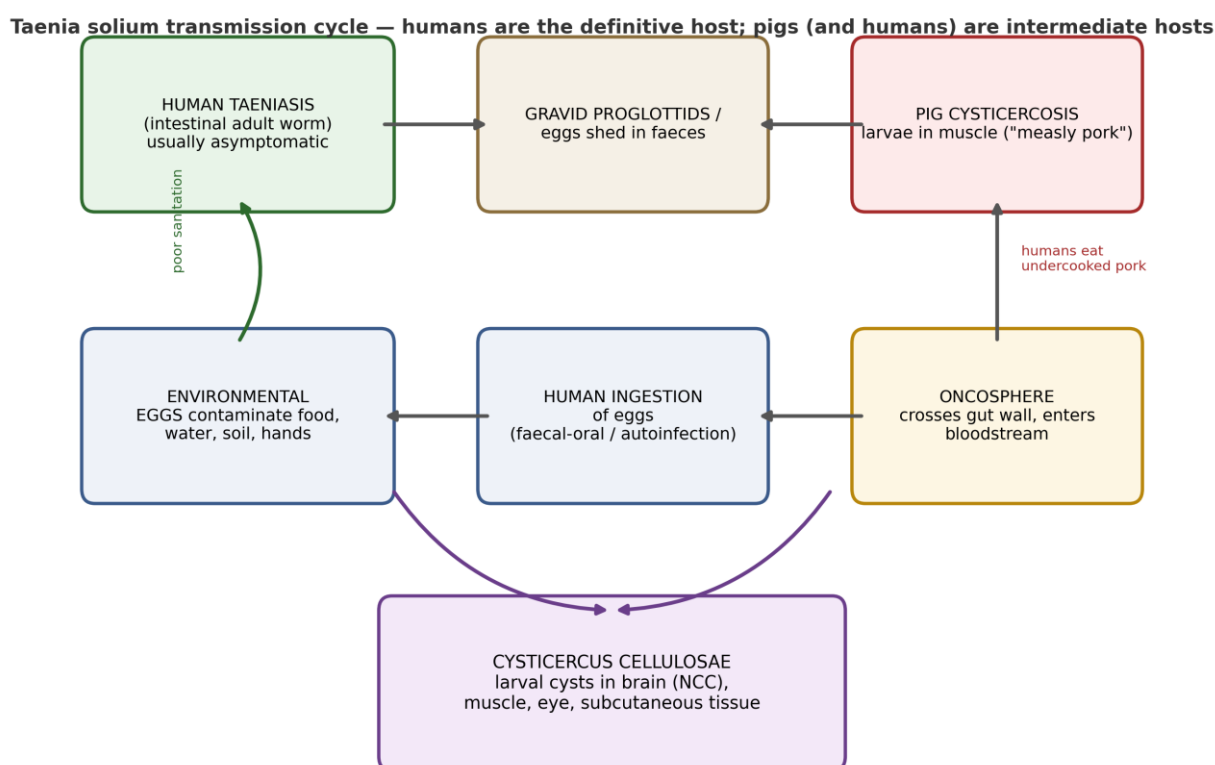


Figure 1. Transmission cycle of *Taenia solium*. Humans acquire neurocysticercosis by ingesting eggs (faecal–oral route), not by eating pork.

3. Pathogenesis and the Four Evolutionary Stages

The living cysticercus is a remarkably successful immunological evader. Its bladder wall secretes antigens that modulate the host response, and viable cysts provoke little inflammation — many people carry silent lesions for life. Disease begins when the parasite dies or degenerates, releasing antigenic material that ignites a vigorous inflammatory cascade involving $\text{TNF-}\alpha$, IL-6, $\text{IFN-}\gamma$, and complement. In the brain parenchyma this perilesional inflammation lowers the seizure threshold and produces the clinical picture most clinicians recognize: new-onset focal seizures, sometimes with Todd's paresis, in a young or middle-aged adult.

Pathologically and radiologically, parenchymal cysts pass through four stages. The vesicular (viable) cyst is a thin-walled, CSF-isodense bladder often containing the invaginated scolex — the pathognomonic 'hole-with-dot' on imaging. In the colloidal stage the parasite degenerates: the cyst fluid becomes turbid, the wall thickens and

irregularly enhances, and perilesional edema appears, clinically announcing itself with seizures. The granular-nodular stage shows a shrinking, solidifying nodule with waning inflammation, and finally the calcified (inactive) stage leaves a dense granuloma — visible on CT but often MRI-invisible — that can nonetheless periodically inflame and provoke recurrent seizures years later. Extraparenchymal disease follows a different logic: cysts in the ventricles and basal subarachnoid cisterns cause mechanical obstruction and chronic inflammation rather than seizures, and the multilobulated 'racemose' form in the basal cisterns behaves like an expanding, poorly treatable mass.

Natural evolution of a parenchymal cysticercus over months to years

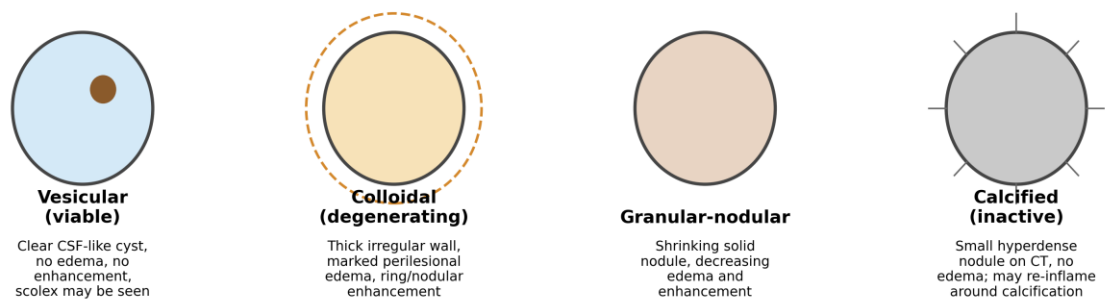


Figure 2. The four evolutionary stages of a parenchymal cysticercus, from viable vesicular cyst to calcified nodule, with corresponding imaging behavior.

Table 1. Imaging characteristics of the four evolutionary stages of parenchymal neurocysticercosis.

Stage	CT appearance	MRI appearance	Clinical correlate
Vesicular (viable)	Round hypodense cyst, no enhancement, $\pm$ scolex	CSF signal on all sequences; scolex bright on T1	Usually asymptomatic; incidental finding
Colloidal (degenerating)	Ill-defined lesion with ring/nodular enhancement, edema	Thick hypointense wall, perilesional FLAIR edema, dense enhancement	Seizures; focal deficits; headaches

Granular-nodular	Shrinking enhancing nodule, decreasing edema	Solid nodule, decreasing enhancement	Resolving inflammation
Calcified (inactive)	Small hyperdense nodule; CT-sensitive	Often invisible; blooming on GRE/SWI	Silent; may re-inflame → late recurrent seizures

4. Clinical Manifestations

No single symptom defines NCC; the illness is a chameleon whose presentation depends on the number and stage of cysts, their compartment, and the vigor of the host response. Seizures dominate — reported in roughly 70% or more of symptomatic patients and as the presenting feature in most series — and constitute the disease's principal global burden through epilepsy. Headaches, focal neurological deficits, and signs of intracranial hypertension follow. In the ventricles, a freely mobile cyst can intermittently obstruct CSF flow, producing episodic positional headache and drop attacks ('Bruns syndrome') before culminating in obstructive hydrocephalus. In the subarachnoid space, chronic inflammation causes arachnoiditis and vasculitis, which can infarct vessels in the circle of Willis and present as stroke — a presentation that has repeatedly masqueraded as atheroembolic disease in non-endemic emergency departments.

Two special syndromes deserve emphasis. Cysticercotic encephalitis, seen mostly in children and young women with massive infections, is a diffuse inflammatory cerebral edema with altered consciousness and papilledema that is a genuine neurological emergency; paradoxically, antiparasitic drugs are contraindicated in the acute phase because killing the parasites would worsen the swelling. Ocular and spinal cysticercosis are rare but sight- or cord-threatening; an intraocular cyst must be removed surgically before any antiparasitic therapy, since drug-induced death of the parasite can destroy the eye.

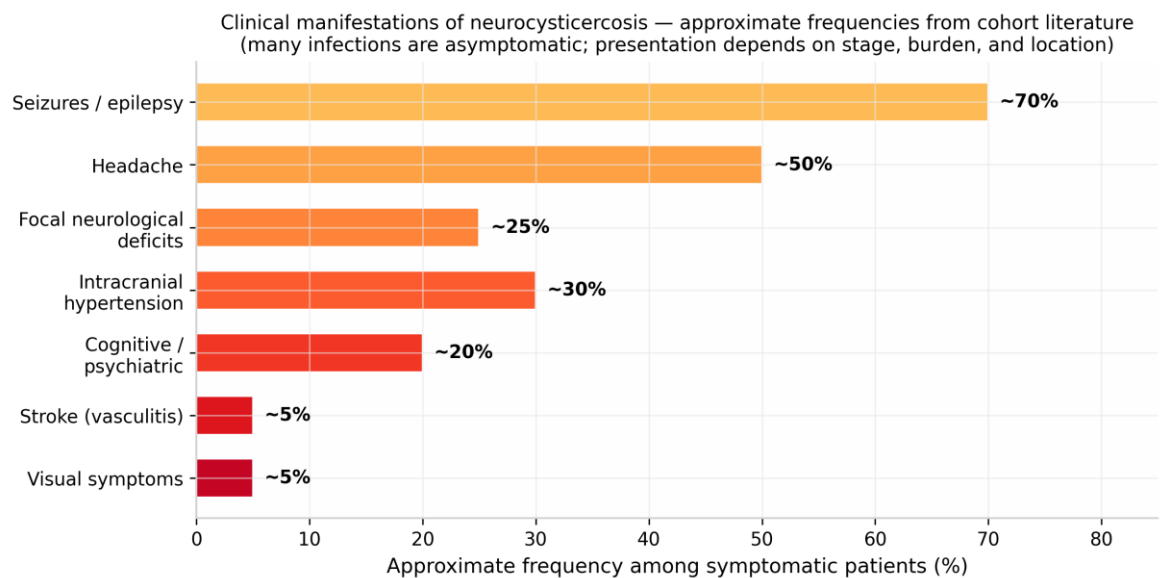


Figure 3. Approximate frequency of clinical manifestations among symptomatic neurocysticercosis patients, compiled from published cohorts.

Table 2. Clinical phenotypes of neurocysticercosis by anatomic compartment.

Compartment	Typical presentation	Imaging hallmark	Prognosis
Parenchymal	Seizures, focal deficits, headache	Cyst(s) at gray–white junction; stages as in Table 1	Good; epilepsy may persist after cure
Intraventricular	Obstructive hydrocephalus, Bruns syndrome	Mobile CSF-isointense cyst; best seen on CISS/FIESTA MRI	Variable; may require shunt ± endoscopy
Subarachnoid (racemose)	Chronic meningitis, cranial neuropathies, stroke (vasculitis), hydrocephalus	Multilobulated basal cistern cysts, leptomeningeal enhancement	Poor; high relapse and mortality
Cysticercotic encephalitis	Diffuse edema, coma, raised ICP	Hundreds of small cysts, diffuse edema	Severe; steroids first, avoid antiparasitics acutely

Spinal	Radiculopathy, myelopathy	Intramedullary or leptomeningeal cysts	Variable; surgical decompression if compressive
Ocular	Visual loss, floaters	Intraocular cyst on slit-lamp/US	Remove before antiparasitics; vision loss risk

5. Diagnosis

Definitive diagnosis integrates epidemiological exposure, clinical picture, neuroimaging, and laboratory confirmation. MRI is the most sensitive modality — superior for small, ventricular, brainstem, and racemose cysts and for demonstrating perilesional edema — while non-contrast CT remains unmatched for detecting calcifications and remains the pragmatic mainstay where MRI is unavailable. A thin-walled cyst with an eccentric scolex ('hole-with-dot') is diagnostic in context, and the coexistence of lesions in different evolutionary stages strongly supports NCC. For extraparenchymal disease, heavily T2-weighted CSF-sensitive sequences (CISS/FIESTA) are essential because these cysts are otherwise nearly invisible.

Serology supports but rarely clinches the diagnosis. The enzyme-linked immunoelectrotransfer blot (EITB) on serum approaches 100% sensitivity in multiple-active-cyst and extraparenchymal disease but falls substantially with single parenchymal lesions or calcifications alone; CSF antibody testing is more sensitive but invasive. Antigen-detection assays (monoclonal antibody ELISA) detect viable parasite burden and are increasingly used for monitoring treatment response, particularly in subarachnoid disease, and PCR methods offer near-perfect specificity where validated. Stool examination identifies the adult tapeworm in only a minority of NCC patients and cannot exclude the diagnosis. Formal diagnostic criteria — integrating absolute, major, minor, and epidemiological criteria, as proposed by Del Brutto and colleagues — remain useful for research and for standardizing care in resource-limited settings.

Table 3. Diagnostic tests in neurocysticercosis — performance and practical roles.

Test	Sensitivity	Specificity	Role / limitation
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MRI (with CISS/FIESTA)	High (best for ventricular/subarachnoid)	High	Modality of choice; limited access/cost in endemic areas
Non-contrast CT	High for calcifications; moderate for viable cysts	Moderate–high	Best for calcified stage; poor for basal cisterns
Serum EITB (antibody)	~100% with multiple active cysts; low with single/calcified lesions	~100%	Excellent confirmatory test; insensitive in light disease
CSF antibody ELISA/EITB	Higher than serum in single lesions	Moderate–high	Requires lumbar puncture; avoid with mass effect
Antigen ELISA (serum/CSF)	Correlates with viable burden	High	Useful for diagnosis and treatment monitoring
PCR (CSF/tissue)	Variable; near-100% specificity	~100%	Reference standard where available; not routine
Stool microscopy	10–15% positive	High if positive	Detects tapeworm carriage; negative result excludes nothing

6. Treatment: Matching Therapy to Compartment and Stage

Modern management of NCC rests on three pillars used in combination: antiepileptic drugs for seizure control, antiparasitic therapy with anti-inflammatory cover, and surgery for mechanical complications. Albendazole (15 mg/kg/day in divided doses, typically for

8–15 days) is the first-line cysticidal drug; it achieves better CSF penetration than praziquantel (50 mg/kg/day) and, per the 2017 IDSA/ASTMH guidelines, is used as monotherapy for one or two viable parenchymal cysts and combined with praziquantel when more than two viable cysts are present. Killing parasites provokes inflammation, so corticosteroids — dexamethasone or prednisolone — are given concomitantly and tapered carefully, a precaution that is not optional in subarachnoid disease. Cure rates with single courses are humbling (on the order of 60% parasite reduction and 40% complete clearance), so retreatment is common, and drug interactions matter: antiepileptics lower praziquantel levels, and corticosteroids lower them further, while cimetidine can partially compensate.

Several phenotypes require deviating from the standard recipe. Single enhancing (degenerating) lesions usually resolve spontaneously within months and are managed with antiepileptics, often with a short corticosteroid course; the role of antiparasitics here remains debated, though evidence leans toward benefit for seizure control. Calcified lesions need no cysticidal therapy, but transient perilesional edema around calcifications is a recognized cause of breakthrough seizures, responsive to short steroid bursts. Subarachnoid and racemose disease demand prolonged courses — albendazole for a month or more with long, carefully tapered steroids — and historically carried high fatality without this aggressive approach; some centers add methotrexate as a steroid-sparing agent. Ventricular cysts with hydrocephalus are primarily a neurosurgical problem: CSF diversion (shunting) with endoscopic cyst removal where feasible, rather than upfront medication. Massive infection with encephalitis is treated with steroids, mannitol, and sometimes decompressive craniectomy — withholding antiparasitics until the swelling subsides.

Table 4. Compartment- and stage-specific management of neurocysticercosis (synthesized from IDSA/ASTMH 2017 and WHO guidance; doses in text).

Phenotype	Antiparasitic therapy	Anti-inflammatory	Surgery / other	Antiepileptics
1–2 viable parenchymal cysts	Albendazole 15 mg/kg/day × 8–15 days	Dexamethasone/prednisolone during & taper	Not indicated	Yes, duration individualized

>2 viable parenchymal cysts	Albendazole + praziquantel combined	Mandatory	Not indicated	Yes
Single enhancing lesion	Controversial; many treat with albendazole	Short course benefits seizure control	Not indicated	Yes; most can taper after resolution
Calcified lesions	None	Short bursts for perilesional edema	None	Yes; treat breakthrough seizures
Subarachnoid / racemose	Prolonged albendazole (≥ 1 month), repeated courses	Long-course steroids $\pm$ methotrexate	Selective; shunt for hydrocephalus	As needed
Intraventricular	Defer until CSF flow restored	As needed	Endoscopic removal $\pm$ VP shunt	As needed
Encephalitis (massive)	Withhold acutely	High-dose steroids, mannitol	Decompression if malignant edema	Yes
Ocular	Never before removal	None intraocular	Surgical removal first	N/A

7. Prevention and Control: A Genuinely Eradicable Disease

Unlike most causes of epilepsy, neurocysticercosis can be eliminated. Transmission requires only that tapeworm eggs from human feces reach pigs or other humans — an entirely interruptible chain. The logical control package combines treatment of human tapeworm carriers (mass or targeted administration of praziquantel or niclosamide),

prevention of pig infection (vaccination of pigs with the TSOL18 antigen combined with oxfendazole treatment, or total confinement of pigs away from human feces), meat inspection, and investment in sanitation and hygiene education. Field trials in Peru and elsewhere have demonstrated dramatic, sometimes near-elimination-level, reductions in porcine and human transmission within years of combined intervention, and mathematical models suggest that sustained joint human–pig measures could locally break transmission entirely. The One Health framing — recognizing that human, animal, and environmental health are one system — has moved cysticercosis control from aspiration to demonstrated feasibility, even if funding and political attention remain inadequate relative to the burden.

8. Gaps, Controversies, and Future Directions

For all its clinical importance, NCC care still rests on a surprisingly thin evidence base. Few randomized trials exist; most treatment recommendations carry low-certainty evidence, and fundamental questions — optimal drug dosing and duration, whether combination therapy improves hard outcomes, how long antiepileptics should continue after lesion resolution, and how best to modulate inflammation during parasite death — remain incompletely answered. Pharmacokinetic variability in albendazole sulfoxide and praziquantel, compounded by interactions with antiepileptics and steroids, argues for therapeutic drug monitoring as a research priority. On the diagnostic front, validated point-of-care antigen tests and quantitative PCR could transform management in endemic regions where CT and MRI are luxuries. And the epileptogenic scar left by calcified NCC — why some patients remain seizure-free after cure while others develop chronic epilepsy — offers a tractable model for understanding post-inflammatory epileptogenesis more broadly.

9. Conclusion

Neurocysticercosis condenses an entire world of medicine into one disease: a parasite of poverty that is also a disease of migration; a cause of seizures that is also a cause of stroke, hydrocephalus, and blindness; an illness that is simultaneously undertreated in endemic villages and over-feared everywhere else. The modern framework — staging by imaging, compartment-specific therapy combining albendazole, praziquantel, corticosteroids, antiepileptics, and judicious surgery — has transformed outcomes for the individual patient. The larger prize, however, lies beyond the clinic: because NCC persists only where human feces, pigs, and eggs meet, it belongs to the short list of human

neurological diseases that sanitation, veterinary science, and political will can actually abolish. Until that day, every unexplained new-onset seizure in a patient from an endemic region, and every mysterious hydrocephalus or 'cryptogenic' stroke, should prompt a single question: could this be cysticercosis?

References

1. White AC Jr. Neurocysticercosis: updates on epidemiology, pathogenesis, diagnosis, and management. *Annu Rev Med.* 2000;51:187–206.
2. Del Brutto OH, et al. Diagnosis and Treatment of Neurocysticercosis: 2017 Clinical Practice Guidelines by the IDSA and ASTMH. *Clin Infect Dis.* 2018;66(8):e49–e75.
3. Del Brutto OH, Rajshekhar V, White AC Jr, et al. Proposed diagnostic criteria for neurocysticercosis. *Neurology.* 2001;57:177–183.
4. Garcia HH, Gonzalez AE, Evans CAW, Gilman RH; Cysticercosis Working Group in Peru. *Taenia solium* cysticercosis. *Lancet.* 2003;362:547–556.
5. Nash TE, et al. Treatment of neurocysticercosis: current status and future research needs. *Neurology.* 2006;67:1120–1127.
6. Coyle CM. Neurocysticercosis: an update on diagnosis and treatment. *Interdiscip Perspect Infect Dis.* 2009;2009:180742.
7. Song HO, et al. Clinical Manifestations, Diagnosis, and Treatment of Neurocysticercosis. *Korean J Parasitol / review literature,* 2010–2015.
8. Del Brutto OH. Update on the diagnosis and management of neurocysticercosis. *Arq Neuropsiquiatr.* 2015;73(11):955–958.
9. Takayanagui OM, Jardim E. Therapy for neurocysticercosis: comparison between albendazole and praziquantel. *Arch Neurol.* 1992;49:290–294.
10. Garcia HH, et al. A trial of antiparasitic treatment to reduce the rate of seizures due to cerebral cysticercosis. *N Engl J Med.* 2004;350:249–258.
11. Abba K, et al. Anthelmintics for people with neurocysticercosis (Cochrane review). *Cochrane Database Syst Rev.* 2010;(3):CD000215.
12. Mahanty S, Garcia HH. Cysticercosis and neurocysticercosis as pathogens affecting the central nervous system. *Prog Neurobiol.* 2010;91:172–184.
13. Sarti E, Schantz PM, Plancarte A, et al. Prevalence and risk factors for *Taenia solium* taeniasis and cysticercosis in humans and pigs in a Mexican village. *Am J Trop Med Hyg.* 1992;46:677–685.

14. Gabriël S, et al. Control of *Taenia solium* taeniasis/cysticercosis: the best way forward for sub-Saharan Africa? *Acta Trop.* 2017;165:252–260.
15. Neurocysticercosis—Diagnostic Mystery: Current Status for Europe. *Microorganisms/Trop Med Infect Dis.* 2024;4(3):24.
16. Neurocysticercosis in transition: expanding clinical spectrum, evolving diagnostics, and emerging therapies. *Front Pharmacol.* 2026;17:1748002.
17. WHO. Taeniasis/cysticercosis fact sheet. Geneva: World Health Organization (updated).
18. Centers for Disease Control and Prevention. Clinical Care of Cysticercosis (resource for clinicians). Atlanta: CDC; 2024.