UPDTE IN RADIOLOGY

Imaging findings in neurocysticercosis

S. Sarria Estrada*, L. Frascheri Verzelli, S. Siurana Montilva, C. Auger Acosta, A. Rovira Cañellas

Unitat de Ressonància Magnètica (IDI), Servei de Radiologia, Hospital Universitari Vall d’Hebron, Barcelona, Spain

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KEYWORDS
Taenia solium; Cysticercosis; Neurocysticercosis; Computed tomography; Magnetic resonance imaging

Abstract Neurocysticercosis, caused by the larvae of Taenia solium, is the parasitic infection that most commonly involves the central nervous system in humans. Neurocysticercosis is endemic in practically all developing countries, and owing to globalization and immigration it is becoming more common in developed countries like those in western Europe.

The most common clinical manifestations are epilepsy, focal neurologic signs, and intracranial hypertension.

The imaging findings depend on the larval stage of Taenia solium, on the number and location of the parasites (parenchymal, subarachnoid, or intraventricular), as well as on the host’s immune response (edema, gliosis, and arachnoiditis) and on the development of secondary lesions (arteritis, infarcts, or hydrocephalus).

The diagnosis of this parasitosis must be established on the basis of the clinical and radiological findings, especially in the appropriate epidemiological context, with the help of serological tests.

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PALABRAS CLAVE
Taenia solium; Cisticercosis; Neurocisticercosis; Tomografía computarizada; Imagen por resonancia magnética

Neurocisticercosis. Hallazgos radiológicos

Resumen La neurocisticercosis es una parasitosis humana causada por las larvas de la Taenia solium, que es la que con mayor frecuencia afecta el sistema nervioso central. Esta infección es endémica en prácticamente todos los países en vías de desarrollo, pero debido a la globalización y a las migraciones humanas su frecuencia ha aumentado en países desarrollados como los de Europa Occidental.

Las manifestaciones clínicas más frecuentes son la epilepsia, signos neurológicos focales e hipertensión intracraneal.


* Corresponding author.

E-mail address: ssarria@idi.cat.org (S. Sarria Estrada).
Introduction

Cysticercosis is a parasitic disease caused by the larva of *Taenia solium*. Human cysticercosis occurs after a person ingests eggs that are passed in the feces of a *T. solium* carrier (fecal-oral contamination).\(^1\)\(^-\)\(^3\) Although infection may develop in any organ, the central nervous system (parenchyma, subarachnoid spaces, ventricles and spinal cord), eyes and muscles are the most commonly involved.\(^3\)

Cysticercosis is endemic in virtually all developing countries (Latin America, South-East Asia and Africa), with the exception of Muslim countries, where pork is not consumed (Fig. 1). In recent years, the incidence of cysticercosis has risen in developed countries due to immigration from endemic areas.\(^2\)\(^,\)\(^3\)

Neurocysticercosis is the most common parasitic disease of the human central nervous system, being the main cause of acquired epilepsy in endemic areas and a major public health problem worldwide.\(^1\)\(^-\)\(^4\) In 1993, the International Task Force for Disease Eradication declared that cysticercosis is potentially eradicable, and proposed to declare neurocysticercosis a reportable disease.\(^5\)\(^,\)\(^6\)

The objective of this paper is to update the most important radiologic features of neurocysticercosis and describe the etiopathogenesis of the disease, clinical presentation, and differential diagnosis. A brief description of the recommended treatment is also provided.

Etiopathogenesis

*Taenia solium* is one of the eight species of cestodes that infect humans. *T. solium* has a scolex and a body with several hundreds of proglottids. Its life cycle includes the egg and larval stage and the adult stage.\(^1\) The hexacanth embryo (thin-walled larval cysts) measures 10–20 mm in length and contains an invaginated scolex (larval head). The cyst, whose wall is rich in glycoproteins, is filled with a clear fluid during this phase, but it becomes cloudy after the parasite dies.\(^1\)

The adult *Taenia* is 2–4 m long and inhabits the small intestine of humans, attached by the scolex.\(^1\)\(^-\)\(^3\) Proglottids containing thousands of eggs detach daily and are passed in the feces, contaminating water and soil.\(^2\) The eggs are eaten by pigs, and once in the pig’s intestine, they transform into oncosphere that enter the bloodstream and disseminate to target organs where the larvae develop. In humans, the larvae reaches the small intestine after the ingestion of contaminated pork, raw or undercooked, the scolex attaches to the intestinal wall and begins to form proglottids. Lastly, cysticercosis occurs when humans become intermediate hosts by ingesting *Taenia solium* eggs\(^1\)\(^,\)\(^2\) (Fig. 2).

Humans acquire the infection through fecal-oral contamination by infected individuals hosting the parasite. Humans are the only definitive hosts of *Taenia solium*, while both humans and pigs are intermediate hosts.\(^1\)\(^-\)\(^3\)

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Los hallazgos radiológicos dependen del estadio larvario de la *Taenia solium*, número y localización de los parásitos (parenquimatosa, subaracnoidea e intraventricular), así como de la respuesta inmune del huésped (edema, gliosis, aracnoiditis) y del desarrollo de lesiones secundarias (arteritis, infartos o hidrocefalia).

El diagnóstico de esta parasitosis debe establecerse en función de los hallazgos clínicos y radiológicos, especialmente en un contexto epidemiológico adecuado, con apoyo de la serología. © 2011 SERAM. Publicado por Elsevier España, S.L. Todos los derechos reservados.

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**Figure 1** Epidemiology. Cysticercosis is endemic to Africa, Asia and South America and particularly favored by poor socio-economic conditions. Source: modified from Román et al.\(^6\)
Figure 2 Life cycle of *Taenia solium*. The cycle starts with the ingestion of raw or undercooked meat containing larvae. The larvae reach the small intestine and attach to the intestinal wall. The proglottids containing thousands of eggs detach daily, are passed with the feces, contaminating water and soil. Animals like pigs become infected by ingesting food contaminated with eggs that develop in their intestine. They enter the bloodstream and spread to muscle where larvae develop. When humans ingest this undercooked or raw meat containing the cysticerci, the life cycle starts over again. Lastly, cysticercosis occurs when humans become intermediate hosts by ingesting the eggs of *Taenia solium*.

**Forms of infection with the cysticercus**

Humans may develop two types of disease, taeniasis and cysticercosis. Taeniasis is acquired by eating pork meat infected with cysts, while cysticercosis occurs by ingestion of *T. solium* eggs present in the feces of a human tapeworm carrier.

In taeniasis, the ingestion of tissues infected with larvae allows the development of the adult form of the parasite in the intestinal tract of the definitive host (i.e., humans), where eggs are produced and discharged, thus, perpetuating the cycle.

In contrast, cysticercosis develops by means of fecal-oral contamination. The embryos, hatched from the ingested eggs, reach the systemic circulation after actively crossing the intestinal mucosa, although some are cleared by the liver.

Cysts lodge in capillaries, mostly in muscle and brain tissue, where they develop into immature cysts and, up to three months later, into larval cysts. These cysts are protected from the host's immune response by the blood-brain barrier and, as long as the cyst wall remains intact, there is no inflammatory response.

When the parasite dies, from natural causes or therapy, an inflammatory response with edema ensues, followed by calcification.

Thereby, *Taenia solium* can cause the disease by the following mechanisms:

1. Presence of the parasite itself (mass effect or obstruction).
2. Inflammatory response (edema).
3. Presence of sequelae (fibrosis, granuloma, and calcifications).

**Clinical presentation of neurocysticercosis**

Neurocysticercosis is a pleomorphic disease whose manifestations depend on individual variation in the number, location, size, stage of parasites, and degree of the host inflammatory response.

Most symptomatic patients are 15–40 year old, and the disease shows no predilection for sex or race.

The most common clinical findings include epilepsy, intracranial hypertension, encephalitis and meningitis.

Epilepsy is the most common clinical manifestation, seen in more than 70% of patients. In endemic areas, neurocysticercosis is the leading cause of late-onset epilepsy. Seizures are secondary to perilesional inflammation in degenerating cysts, although infarction and vasculitis may also act as predisposing factors. Calcified granulomas may also cause epilepsy.

Clinical manifestations usually have a slowly progressive onset; however, an acute presentation such as cerebral infarction secondary to vasculitis, may also occur.

Two mechanisms can cause intracranial hypertension: (1) obstructive hydrocephalus secondary to intraventricular cysts, arachnoiditis or granular ependymitis; and (2) mass effect in cases of very large cysts. Patients with neurocysticercosis of the fourth ventricle may have transient obstruction of the Sylvian aqueduct, whose symptoms and signs are known as Bruns' syndrome. This syndrome is caused by an intraventricular mobile lesion, which leads to episodic obstructive hydrocephalus. This syndrome is characterized by headache, papilledema and loss of consciousness with rapid recovery induced by rotational movements of the head.

Cysticercotic encephalitis results from intense inflammatory response of the host to massive cysticercal infection of the brain parenchyma. Encephalitis is most common in young women and children, who present with altered consciousness, seizures, visual anomalies, headache, vomiting, and papilledema.

Invasion of subarachnoid spaces may result in thickening of the leptomeninges, but without signs of meningeal irritation. Thickening along the base of the skull and the ventral aspect of the brainstem may entrap the optic chiasma and the cranial nerves, leading to nerve palsy or dysfunction.

Up to 3–4% of cases of neurocysticercosis manifest as brain strokes, with a prevalence of 3.4%. They include lacunar or cortical infarcts, transient ischemic attacks and brain hemorrhage. Cerebrovascular complications are the result of different mechanisms, including luminal narrowing due to subintimal thickening, vasospasm due to arteritis in perforating and midsized vessels, inflammatory pseudoaneurysms and thrombi.

The progressive midbrain syndrome is the result of multiple infarctions in the midbrain and thalamus secondary to occlusion of the perforating arteries that arise from the basilar artery. These patients typically have a history of shunted hydrocephalus secondary to diffuse leptomeningi-tis, with neurological impairment, somnolence, paraparesis,
impaired vertical gaze, fixed and dilated pupils, and urinary incontinence.\textsuperscript{1,12}

Spinal involvement is seen in only 1.5% of cases. Less frequently, spinal leptomeningitis can manifest as radicular pain associated with muscle weakness. The most common form is intradural-extramedullary spinal cysticercosis, which usually manifests as spinal cord section syndrome.\textsuperscript{17,18}

Classification

The cysticercus enters the central nervous system through the bloodstream, initially invading the subarachnoid space, and then the cortex and the cortical–juxtacortical junction. The macroscopic appearance of cysticerci varies according to their location within the nervous system and stage of the disease. Different stages of the disease and locations may coexist in the same patient.\textsuperscript{19}

Neurocysticercosis may be classified according to the location and stage of the disease. According to the location, cysticerci are classified as subarachnoid, parenchymal, ventricular and spinal. Based on the stage, they are classified as non-cystic, vesicular, colloidal-vesicular, granular-nodular, and calcified nodular.

Classification according to the location

Subarachnoid cysticerci

The subarachnoid location is the most common. The parasite reaches the basal cisterns, subarachnoid spaces and meninges by hematogenous spread, eliciting an intense perilesional inflammatory reaction. This reaction involves the leptomeninges at the base of the skull and may extend to the foramen magnum, resulting in basilar leptomeningitis that can lead to entrapment of cranial nerves and arteries. Involvement of the foramina of Luschka and Magendie may result in hydrocephalus. While subarachnoid cysticerci in the depth of cortical sulci are small, lesions located in the Sylvian fissure or in the basal cisterns may reach 5 cm in size (Fig. 3). This location facilitates hydropic degeneration caused by continuous adsorption of cerebrospinal fluid.

Figure 3  Subarachnoid cysticerci. FLAIR MRI in the transverse plane (A) shows small cysts in the depth of the sulci of both parietal lobes. Cystic lesions in the basal cisterns. Contrast-enhanced CT (B) and T2-weighted MRI (C), both in transverse planes, show the basal cisterns occupied by cystic lesions with content similar to the cerebrospinal fluid.

Figure 4  Parenchymal cysticerci. Contrast-enhanced CT (A), T2-weighted (B) and T1-weighted gadolinium-enhanced MRI sequences in the transverse plane (C) show small cysts in the right frontal cortex, with minimal perilesional edema, ring enhancement and scolex visible in the interior (arrow).
fluid to the vesicle. In this situation, cysticerci do not develop a protoscolex or the scolex degenerates (racemose cysticercosis).\textsuperscript{4,19}

**Parenchymal cysticerci**

Parenchymal neurocysticercosis is the second most common form of neurocysticercosis after the arachnoid form. Cysts are usually located in the cerebral cortex and basal ganglia, areas that get more blood supply (Fig. 4). Parenchymal cysts are usually small, and are rarely larger than 10 mm in diameter, because their growth is limited by the pressure exerted by the brain parenchyma.\textsuperscript{4} Parenchymal forms include different stages, from viable cysts (cysts with scolex) to degeneration that ends in calcifications.

**Ventricular cysticerci**

Intraventricular cysticerci account for less than 33% of all cases of neurocysticercosis. Cysts vary in size and are usually solitary. The fourth ventricle is the most common site (50%), followed by the lateral ventricles (35%) and, less

![Figure 5](image_url)  
**Figure 5** Intraventricular cysticercus. Unenhanced CT (A) image shows a cyst in the fourth ventricle. T1-weighted gadolinium-enhanced (B) and T2-weighted (C) MRI sequences in the axial plane, and high-resolution T2-weighted (D) and T1-weighted gadolinium-enhanced (E) sequences in the sagittal planes show one intraventricular cysts obstructing the fourth ventricle, with a lower mural nodule that enhances with gadolinium (arrow). Follow-up MRI after antecestodal drugs (F and G) shows a decrease in size of intraventricular lesion, with persistence of minimal gadolinium enhancement in the lower margin of the fourth ventricle (arrow). Courtesy of Dr. Antoni Rovira, Corporació Sanitària Parc Taulí, Sabadell.
frequently, the third ventricle (10%) and the Sylvian aqueduct (5%)\textsuperscript{120-22} (Fig. 5). Parasites may adhere to the ependymal layer, leading to ventriculitis (granular ependymitis and subependymal gliosis), or may float freely within the ventricular cavities, leading to obstruction of cerebrospinal fluid. Persistent obstruction leads to hydrocephalus and intermittent obstruction causes Bruns’ syndrome.

**Spinal cysticerci**

Spinal cysticerci are very rare (1–3% of cases). The cysts are located in the subarachnoid space that surrounds the spine as a result of the spread of the larva throughout the cerebrospinal fluid (Fig. 6). Intramedullary cysticerci are even rarer (<1%), most cases occurring in the dorsal spine.\textsuperscript{23} Macroscopic appearance of spinal cysticerci is similar to that of brain cysts.\textsuperscript{20} Leptomeningeal cysts may move freely within the subarachnoid space and change their position according to the movements of the patient during examination.

**Stages of neurocysticercosis**

Based on the stage and radiologic findings, neurocysticercosis is divided into five stages: non-cystic, vesicular, colloid-vesicular, granular-nodular, and calcified-nodular\textsuperscript{1} (Table 1).

**Non-cystic stage**

The lesions correspond to tissue invasion by the cysticerci. This stage is usually asymptomatic and therefore no imaging studies are normally performed, but if performed, images show focal areas of edema that may be associated with nodular enhancement after contrast administration, on both CT and MRI.\textsuperscript{21}

**Vesicular stage**

The host shows immune tolerance, as a result, there is only minimal inflammatory reaction. The cysticercus appears as a rounded cyst, a thin capsule surrounds the viable larva and the fluid-filled vesicle. A cyst cavity that is isointense relative to the cerebrospinal fluid is seen on MR images. The cyst is 5–20 mm in diameter and has a 2–4 mm mural nodule that corresponds to the scolex. The scolex is isointense relative to the brain parenchyma on all sequences, enhancing after IV contrast administration.\textsuperscript{24} The cyst is commonly located in the cortical–juxtacortical junction or in the basal ganglia, cerebellum, midbrain, cisterns or ventricular system (Fig. 7).

**Colloidal-vesicular stage**

In this stage, the larva dies and an inflammatory response starts due to the release of metabolites. This causes intense
Table 1  Stages of neurocysticercosis.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Pathophysiology</th>
<th>MRI features</th>
</tr>
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<tbody>
<tr>
<td>Non-cystic</td>
<td>Tissue invasion by the cysticercus</td>
<td>- Local focus of edema</td>
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<td></td>
<td></td>
<td>- There might be nodular contrast enhancement</td>
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<tr>
<td></td>
<td></td>
<td>- Usually no imaging studies are performed</td>
</tr>
<tr>
<td>Vesicular</td>
<td>Minimal inflammatory reaction</td>
<td>- Cyst: hypointense T1/hyperintense T2.</td>
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<td></td>
<td>Cyst with scolex</td>
<td>- CSF-like signal.</td>
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<tr>
<td></td>
<td></td>
<td>- Scolex: isoointense to parenchyma on T1 and T2,</td>
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<td></td>
<td></td>
<td>- hypointense onT2* sequences,</td>
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<tr>
<td></td>
<td></td>
<td>- hyperintense on FLAIR</td>
</tr>
<tr>
<td>Colloidal-vesicular</td>
<td>Death of the parasite</td>
<td>- Vasogenic edema surrounding the cyst.</td>
</tr>
<tr>
<td></td>
<td>Intense inflammatory response</td>
<td>- Cyst: formation of capsule, hypointense on T2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Ring-like contrast enhancement</td>
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<td></td>
<td></td>
<td>- Formation of fluid-fluid level</td>
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<tr>
<td>Granular-nodular</td>
<td>Absorption and cyst retraction</td>
<td>- Residual cyst is smaller in size, thickening of the</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- parenchyma T1-/iso-hypointense on T2-weighted</td>
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<tr>
<td></td>
<td></td>
<td>- sequences.</td>
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<tr>
<td></td>
<td></td>
<td>- Calcified scolex (target appearance)</td>
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<tr>
<td></td>
<td></td>
<td>- Minimal vasogenic edema might persist</td>
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<tr>
<td></td>
<td></td>
<td>- Nodular or micronodular</td>
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<tr>
<td></td>
<td></td>
<td>- contrast-enhancement</td>
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<tr>
<td>Calcified-nodular</td>
<td>Complete cyst involution</td>
<td>- Calcified nodule without</td>
</tr>
<tr>
<td></td>
<td>Mineralization</td>
<td>- contrast-enhancement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Hypointense nodule on T2* sequences</td>
</tr>
</tbody>
</table>

CSF: cerebrospinal fluid.

perilesional edema and formation of a capsule. This capsule is hypointense on T2-weighted images. Ring-like enhancement of the wall is seen in two-thirds of cases after contrast administration. As a result of the parasite’s death, there is hyperattenuation on CT images, and increased signal on MR images. A fluid-fluid level can also be observed. In the stage, the cyst begins to contract (Fig. 8).

Granular-nodular stage
In this stage, absorption of the cyst fluid results in cyst contraction, with capsule thickening and scolex calcification. CT reveals an isointensifying cyst with a hyperattenuating calcified scolex. Peripheric edema and enhancement after contrast administration persist. The residual cyst is isointense on T1-weighted sequences relative to the parenchyma and iso- to hypointense on T2 sequences. Nodular or micronodular enhancement is commonly observed in this stage, which suggests granuloma. A target or bull’s-eye appearance is seen with the calcified scolex in the center of the lesion (Fig. 9).

Calcified-nodular stage
In this stage, the cyst involutes completely. The granulomatous lesion has shrunk to a fraction of its initial size and it is completely calcified. Typically, CT shows a calcified nodule without mass effect or contrast enhancement. However, persistent enhancement can be identified in some calcified nodules. It has been suggested that the presence of these lesions represents a risk factor for seizures after therapy. These lesions appear as small hypointense nodules on both T2- and T2*-weighted sequences (Fig. 10). Cavernomas should be included in the differential diagnosis in this stage. Patients with neurocysticercosis will frequently present with cysts in varying stages and in multiple locations.

Diagnosis

Diagnosis of neurocysticercosis may be difficult because in many cases the infection with *Taenia solium* cannot be demonstrated. Therefore, the diagnosis is based on a combination of epidemiological, clinical, radiological and immunological (serological tests for the detection of anticysticercal antibodies in blood and cerebrospinal fluid) criteria.

In 2000, a group of experts proposed a series of diagnostic criteria based on four categories:

1. Absolute criteria allow unequivocal diagnosis of neurocysticercosis:
   - Histologic demonstration of the parasite in brain or spinal biopsy.
   - Scolex within a cystic lesion on CT or MRI.
   - Direct visualization of subretinal parasites by fundoscopic examination.

2. Major criteria are suggestive of neurocysticercosis, but do not confirm the disease:
   - Neuroimaging studies highly suggestive of neurocysticercosis.
Figure 7  Vesicular stage. Unenhanced CT (A) shows a non-calcified scolex (white arrow) and a small parenchymal calcification adjacent to the cyst (nodular-calcified stage) (black arrow). Axial T1-weighted gadolinium-enhanced (B), T2-weighted (C) and proton density (D) MRI sequences. The cysts have similar signal and intensity to those of the cerebrospinal fluid. On the proton-density sequence, the scolex is hyperintense (white arrow) and shows no enhancement after IV contrast administration. No inflammatory reaction is observed.

- Identification of anticystercal antibodies by EITB (Enzyme-linked immunoelectrotransfer blot assay) using purified antigens of *Taenia solium*.
- Resolution of cysts after therapy with albendazole or praziquantel.
- Spontaneous resolution of small ring-like enhancing lesions (<20 mm) in patients with seizures, without other symptoms.

3. Minor criteria are common but not specific of the disease:
   - Neuroimaging studies demonstrating lesions compatible with neurocysticercosis.
   - Clinical features suggestive of neurocysticercosis, such as epileptic seizures, focal neurological signs, intracranial hypertension and dementia.
   - Positive CSF ELISA (enzyme-linked immunosorbent assay) for the detection of anticystercal antibodies or antigens.
   - Cysticercosis outside the CNS.

4. Epidemiologic criteria refer to circumstantial evidence supporting the diagnosis of neurocysticercosis:
   - Evidence of household contact with *Taenia solium*.
   - Individuals living or coming from endemic countries.
   - History of frequent visits to endemic areas.

The interpretation of these criteria permits two levels of diagnostic certainty:

1. Definitive diagnosis, in patients with one absolute criterion or in those with two major criteria, one minor and one epidemiological criteria.
2. Probable diagnosis, in patients with one major and two minor criteria, in those who have one major, one minor and one epidemiological criteria, and in those who have three minor and one epidemiological criteria.

**Neuroimaging studies**

CT findings are based on the identification of a well-defined cystic lesion in the cortical–juxtacortical junction, basal ganglia, cerebellum, midbrain, cisterns or ventricular system. The lesion may show minimal contrast enhancement, and an enhancing mural nodule, corresponding to the scolex. Chronic lesions of neurocysticercosis appear as calcifications, readily visible on CT images.

Generally, MRI is more sensitive than CT for the diagnosis of the disease, since it allows us to identify a larger
number of lesions, classify the stage of the disease, and determine the degree of inflammatory response (perilesional edema and blood-brain barrier disruption). T1- and T2-weighted MRI sequences depict the cyst, which shows similar signal to that of the cerebrospinal fluid in the vesicular stage. Diffusion MRI sequences demonstrate the fluid behavior within the lesions and help differentiate them from abscess. At the colloidal-vesicular stage (death of the parasite), the protein content of the cyst increases and so does the signal intensity of basic MRI sequences. In addition, during this stage there is formation of a capsule that is hypointense on T2-weighted sequences. This hypointensity is due to the presence of free radicals produced by macrophages; however, this finding is not specific since it can also be found during the early encapsulation of parenchymal abscesses.

Among the multiple imaging findings of neurocysticercosis, the presence of the scolex within the cyst is considered patognomonic. The identification of the scolex on the different sequences becomes therefore a primary goal. The scolex is depicted on T1- and T2-weighted sequences as an isointense nodule relative to brain parenchyma, and as mildly to moderately hypointense on T2* sequences. Proton-density and FLAIR sequences are more sensitive for the detection of the scolex, which appears as a hyperintense eccentric nodule within the cyst.

The low sensitivity and specificity of MRI in comparison to CT for the determination of the presence of calcium has been traditionally considered a limitation. The detection of small parenchymal calcifications is a finding suggestive of neurocysticercosis (calcified-nodular stage). Currently, the use of higher magnetic fields and new sequences like magnetic susceptibility MRI help to overcome this limitation. Signal recording in magnetic susceptibility sequences allows reconstruction of phase and magnitude images from the raw data. Magnitude images clearly depict lesions with magnetic susceptibility, but cannot differentiate microhemorrhage from calcification, unlike phase images, which can be used for their differentiation. The processed phase images show a negative phase (hypointense) for paramagnetic substances and a positive phase (hyperintense) for diamagnetic substances. Calcifications are considered diamagnetic substances and thus they appear with the opposite signal intensity in filtered phase images (hyperintense). Comparison of filtered phase images with magnitude images helps identify calcifications and differentiate them from chronic microhemorrhages. This sequence may demonstrate small, markedly hypointense nodules.
Figure 9 Granular-nodular stage. Axial T1-weighted gadolinium-enhanced MRI sequences (A and B) and FLAIR sequences (C and D) show small lesions with nodular and ring-like hyperenhancement located in left basal ganglia, left posterior temporal lobe and both occipital lobes (black arrows) with associated vasogenic edema (white arrows).

in the brain parenchyma, often indistinguishable from cavernomas.

The use of a contrast agent permits identification of lesions with nodular enhancement in non-cystic and granular stages, and ring-like enhancement of the cystic wall in the colloidal-vesicular stage. Acquisition of T1-weighted sequences in delayed phases after contrast administration increases the sensitivity for identification of lesions. Atypical presentations of neurocysticercosis may simulate other neurological diseases. Basal arachnoiditis should be differentiated from carcinomatous meningitis, granulomatous diseases like sarcoidosis, tuberculosis and fungal meningitis. In spinal neurocysticercosis, the differential diagnosis should include ependymomas, cystic astrocytomas, syringomyelic cavities, hydatid cysts and congenital cysts (arachnoid and dermoid).

Differential diagnosis

Differential diagnosis of neurocysticercosis in endemic areas may be extremely difficult due to the coexistence of tuberculosis and other parasitic infections. Ring-like lesions (solitary or multiple) in the parenchyma of the central nervous system are not specific for neurocysticercosis and represent a diagnostic problem, since serological tests are often negative. In colloidal-vesicular neurocysticercosis, the differential diagnosis should include tuberculomas, pyogenic abscesses, toxoplasmosis, neurosyphilis, hydatidosis, as well as primary and secondary neoplasms, while in the calcified-nodular stage the differential diagnosis should be made with cavernomas and amyloid microangiopathy.

Atypical presentations of neurocysticercosis may simulate other neurological diseases. Basal arachnoiditis should be differentiated from carcinomatous meningitis, granulomatous diseases like sarcoidosis, tuberculosis and fungal meningitis. In spinal neurocysticercosis, the differential diagnosis should include ependymomas, cystic astrocytomas, syringomyelic cavities, hydatid cysts and congenital cysts (arachnoid and dermoid).

Treatment

Treatment of neurocysticercosis is controversial and depends on the number, location and viability of cysts, and the presence of complications. Medical therapy is considered the first choice, except in cases of intracranial hypertension requiring surgery. The use of cysticidal drugs is based more on the geographic area and the clinician experience than on consensus protocols. Cysticidal therapy itself initiates a host inflammatory response that increases brain edema and may result in intracranial hypertension.
In general, treatment includes antiepileptic and cysticidal drugs (e.g., albendazole and praziquantel), corticosteroids and other anti-inflammatory agents used to control the severe host inflammatory response.1−4

Conclusion

Due to the migration phenomenon that has taken place over the last decade, neurocysticercosis is no longer a rare infection in Spain. As neurocysticercosis is the most common parasitic disease of the central nervous system and the most common cause of acquired epilepsy in developing countries, radiologists should be familiar with the radiologic findings of the disease and include it in the differential diagnosis of intracranial lesions, especially in patients from endemic areas.

Authorship

1. Responsible for the integrity of the study: SSE, LFV and ARC.
2. Conception of the study: SSE, LFV and ARC.
3. Design of the study: SSE and ARC.
4. Acquisition of data: SSE, SSM and CAA.
5. Analysis and interpretation of data: SSE.
6. Bibliographic search: SSE, LFV and ARC.
7. Writing of the paper: SSE, LFV and ARC.
8. Critical review with intellectually relevant contributions: SSM, CAA and LFV.
9. Approval of the final version: SSE, ARC, LFV, CAA and SSM.
10. All the authors have read and approved the final version of the manuscript.

Conflict of interest

The authors declare that they do not have any conflict of interests.

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