Diagnostic Imaging in Neurocysticercosis

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Abstract

Neurocysticercosis is a major parasitic disease of the central nervous system, affecting endemic areas worldwide. It spreads through intermediary hosts, pigs and eventually men to his definitive host: humans. It causes lesions in the CNS which have different evolutive stages and can be asymptomatic or clinically evident. Imaging studies play an important role in the diagnosis of the disease and in its appropriate control.

Key Words: Neurocysticercosis – Computed Tomography – Magnetic Resonance Imaging

Introduction

Neurocysticercosis (NCC) is the more frequent parasitic disease of the CNS in immunocompetent patients associated with infection due to its larval form of *Taenia solium*.

It has a broad distribution worldwide and generates morbidity in infected populations as a result of the neurologic compromise.

In this article we will explain in details the epidemiology, the etiopathogenesis, the development of the disease and, particularly, how it is seen in CT and MRI.

Epidemiology

It is believed that approximately 50 million people are infected with the taeniasis-cysticercosis complex around the world. In America, 350,000 patients suffer from this disease and a high percentage of them are affected neurologically.

These data reflect that the cysticercosis is a serious health problem which affects Latin America, Asia and Africa (1). It is seen mainly in developing countries where there are worse hygiene conditions and a lower socioeconomic and cultural level, factors which contribute to its transmission.

Although NCC constitutes a serious health problem in developing countries, migrations cause the dissemination of the disease to non-endemic areas. (Diagram 1)

The correlation between the socioeconomic level and the development of the complex taeniasis-cysticercosis is given by hygiene habits and the traditional breeding of pigs, both related to the dissemination of the causal agent of this disease.
**Endemic areas in the world.** Distribution of global prevalence areas: low, medium and high according to WHO data.

**Etiopathogenesis**

The infection is caused by *Taenia solium*, which presents three developmental stages: egg, larva and adult.

*Taenia solium* produces intestinal infection in its adult stage (taeniasis) or an invasive infection in its larva stage (cysticercosis) (**Diagram 2**).

**Cycle of the parasite.** Meat contaminated with larvae of *T. Solium* (2) is consumed by humans causing the development of intestinal tapeworms (3). Eggs released through the stool (4) food contaminate both pigs (1) as that of humans (5), any intermediate hosts or cysticercosis causing invasive infection with brain involvement (6).

The intestinal infection is developed in humans (the only definite host) when there is an intake of larvae or cysticercus in infected pork (2). Larvae enter into the intestine, they evert and their scolex with double crown of hooks and vacuums adheres to the intestinal wall, producing a slight inflammation. The body of the parasite grows until it reaches its adult stage which can measure up to 2 to 4 meters divided in multiple segments known as proglottids, bearer of 1,000 to 2,000 eggs each, that are set free with intestinal evacuation.
Invasive infection or cysticercosis occurs when there is an intake of embryonated eggs disseminated in contaminated food or water. The major form of contamination is directly, i.e. through manipulation or consumption of contaminated food or through direct contact with an infected person.

Once they are ingested, the eggs release an embryo which breaks through the intestinal wall and it is transported via the bloodstream to the different tissues, mainly to the muscles and to the central nervous system.

The interaction between the parasite and the intermediary host (pigs and eventually men) is determined by the evasive mechanisms of the cestode immunity which allows it to survive for many years, without producing symptoms in the host.

Clinical Presentation of NCC

According to its presentation, laboratory test and imaging studies, NCC can be divided in active and inactive forms.

The active forms are subject to the interaction host-parasite, depending on the attenuation of the generated immune response which generally reflects an asymptomatic infection for many years.

The most common symptoms are not specific, like headaches and convulsions due to perilesional and edema\(^{(3)}\), and less frequently brain infarcts and vasculitis.

If the cysts are located near eloquent areas there will be focal manifestations, like motor deficit, ataxia, etc.

Diagnosis

The clinical suspicion in patients of endemic areas associated with diagnostic imaging studies, and laboratory and cerebrospinal fluid (CSF) tests searching for specific Immunoglobulin G (IgG) through Enzyme Linked ImmunoSorbent Assay (ELISA), contributes to reaching the NCC diagnosis.

Presentation in CT and MRI

Traditionally, there have been different forms of NCC presentation in CT and MRI: parenchymatous, subarachnoid, intraventricular and spinal forms. Nowadays, it is known that the parenchymatous presentations are produced by localized cysts in the subarachnoid space which has deep lines and perivascular spaces\(^{(4)}\).

The MRI is the method with more information regarding CNS lesions, parasitosis stage and associated findings.

It allows for the detection of cysts in the cistern or ventricular system due to a subtle difference between liquid cysts\(^{(5)}\) and CSF signal.
However, the CT has better performance in Neurocysticercosis cases in their calcified stage due to its greater sensibility to detect calcium in the lesions than MRI.

NCC is classified according to imaging studies in five stages $^{(6)}$:

- Non Cystic
- Vesicular
- Colloidal Vesicular
- Granular Nodular
- Calcified Nodular

In spite of this division, it is common to find an overlap of stages (50%) in studies and mixed stages in the same patient $^{(7)}$.

**Non Cystic Stage**

It is asymptomatic and cannot be identified in imaging studies. It can only be detected by lab tests.

**Vesicular Stage**

It is characterized by the presence of multiple cysts of different sizes, smaller than 20 mm, distributed in the subarachnoid and perivascular spaces near the basal ganglia, the cortico-subcortical interface, in cisterns and ventricular system $^{(8)}$.

Cysts present similar characteristics to CSF in CT and MRI and evidences neither calcifications nor perilesional edema ($\textbf{Figures 1 and 2}$). Scolexes can be demonstrated in up to a 50% of the cases, located eccentrically at the walls of the cystic lesions. They appear in images as small structures, measuring less than 5 mm, and they are slightly hyperintense in Fluid Attenuated Inversion Recovery (FLAIR) MRI sequences ($\textbf{Figure 3}$) and in CT as small hyperdense small nodules ($\textbf{Figure 4}$).

The cluster shape consists in multiple confluent cysts located typically in the cisterns of the skull base $^{(9)}$ ($\textbf{Figure 5}$).
**Colloidal Vesicular Stage**

It is determined by the alteration of the larva vitality related to the loss of the developed immune tolerance, triggering an osmotic unbalance through the cyst membrane and inflammatory response which is generated in the adjacent encephalic parenchyma, associated with edema and gliosis.

Cystic lesions at this stage can present signal intensity different from CSF in MRI studies, being slightly more hyperintense in all sequences (Figure 6). There is evidence of a fibrous capsule which is enhanced after contrast injection (Figure 7) and there is a perilesional edema, more evident on T2 and FLAIR sequences (Figure 8).

**Granular Nodular Stage**

At this stage, there is a cystic retraction and collapse that leads to the appearance of nodular structures. Annular lesions and nodules can be identified in MRI and they show enhancement after contrast injection (Figure 9) and a perilesional edema due to the inflammatory response from the host to the parasite. In T2 and FLAIR sequences there is perilesional hyperintensity in relation to vasogenic edema (Figure 10). Diffusion sequences show variable intensity and hyperintensity in Apparent Diffusion Coefficient (ADC) (Figures 11 and 12).

**Calcified Nodular Stage**

It is the inactive stage of parasitosis where there is a total involution of the non-vital cyst with calcium deposits. There is neither perilesional edema nor enhancement; it is best observed in CT studies (Figure 13). In MRI, the Gradient Weighted sequences in T2 help improve the sensibility for the detection of calcium lesions at this stage.
Fig 1: CT images showing cystic lesions adjacent to the cortical surface of the temporal and occipital lobes. Larger lesions have a small nodule inside corresponding to scolex.

Fig 2: MRI T2 sequence of the same patient: cysts with similar signal to the LCR.

Fig 3: MRI on FLAIR sequence showing a small image associated to a hyperintense cyst wall: the parasitic scolex.

Fig 4: Multiples small nodular images corresponding to scolex (arrows) associated with NCC cysts in CT imaging.
**Fig 5:** Increased subarachnoid basal spaces at the level of the cisterns due to the presence of confluent cysts in a patient with racemose NCC.

**Fig 6:** Cystic images at different stages in axial FLAIR MRI sequence. Hyperintense lesions are observed at the colloidal vesicular stage.

**Fig 7:** Axial T1 MRI Sequence with Gadolinium showing a cystic lesion at colloidal vesicular stage exhibits peripheral enhancement corresponding to the thin capsule.
**Fig 8:** Axial FLAIR sequence of the same patient shows the same lesion associated with perilesional edema.

**Fig 9:** Axial T1 Sequence showing nodular lesion with intense enhancement after Gadolinium injection in a patient with NCC granular nodular stage.

**Fig 10:** FLAIR Sequence in the same patient than Fig. 9: perilesional edema is best demonstrated.
Fig 11: Vasogenic edema appears slightly hyperintense on DW Sequence and hyperintense on ADC Sequence.

Fig 12: CT scan showing multiple calcifications in patient with nodular calcified stage of NCC.

Complications and Associated Findings

During the colloidal vesicular stage, the inflammatory reaction around the lesion can cause a meningeal granulomatous response. Arachnoiditis can be focal or diffuse, generating leptomeningeal fibrosis and neuropathy because of cranial nerves compression. It can be identified in imaging studies by the enhancement of the basal cisterns after contrast injection.

Vasculitis (cysticercus angiitis) is another radiological manifestation related to NCC. There is arteries focal narrowing which can compromise the bloodstream and develop brain infarcts generally in relation to the subarachnoid forms of the disease.

Intraventricular location of cysts can cause hydrocephalus. FLAIR sequences improve the detection in MRI showing a slight cysts hyperintensity in relation to ventricular CSF (Figure 14).
**Fig 13:** Axial FLAIR MRI Sequence showing a cystic lesion in the fourth ventricle that appears slightly hyperintense relative to CSF.

**Differential Diagnosis**

NCC lesions in its different stages should be differentiated from other cystic lesions such as abscesses, arachnoid cysts, cystic neoplasms, including metastasis and other parasitosis.

In order to make a differential diagnosis, it is important to take into account the presence of calcifications, scolexes, cysts location and form, and the coexistence of lesions in different stages.

**Conclusion**

CT and MRI are essential tools in NCC diagnosis, given the often asymptomatic course of the disease.

The clinical suspicion in patients from endemic areas leads to a diagnosis of parasitosis. Diagnosis methods are very important for the diagnosis of this disease, for the staging and for the dismissal of complications.

**Bibliography**


