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Neurocysticercosis. A frequent cause of seizures, epilepsy, and other neurological morbidity in most of the world.

J Bustos[1,2], **AE Gonzales**[1], **H Saavedra**[1], **S Handali**[3], **HH Garcia**[1,2] **CWGP**

[1] -Cysticercosis Unit, Instituto Nacional de Ciencias Neurológicas, Lima, Peru

[2] -Center for Global Health, Universidad Peruana Cayetano Heredia, Lima, Peru

[3] -Division of Parasitic Diseases and Malaria, Parasitic Diseases Branch, Center for Global Health, Centers for Disease Control and Prevention, Atlanta, Georgia

INTRODUCTION

Taenia solium taeniasis/cysticercosis is endemic in Central and South America, Sub-Saharan Africa, Indian Ocean (Madagascar), and large regions of Asia including India, China, and Southeast Asia (1–4). In these regions the infection of the human brain by *T. solium* larvae (neurocysticercosis, NCC) is a frequent cause of neurological morbidity and it is claimed to be the most frequent cause of acquired seizures and epilepsy, accounting for 30% of seizure disorders (1, 5). The degree of endemicity varies, and seems to be higher in Sub-Saharan Africa (6). In addition, immigration and travel result in many cases of NCC diagnosed in non-endemic countries, where they cause significant costs to the health system (7–9). In the US, there are more than 1800 hospitalizations per year for NCC. Annual hospitalizations and charges to the health system are more than for all of the other NTD's combined (10). The clinical manifestations, diagnostic and therapeutic approaches, and prognosis of NCC very enormously depending on the type, stage, location, number and size of parasites in the nervous system, as well as the immune response of the host.

In the usual life cycle of *T. solium*, humans acquire intestinal taeniasis by ingesting poorly cooked pork containing the parasitic cystic larvae or cysticerci. Once the adult tapeworm develops in the human small intestine, its microscopic eggs are shed with the stools of the tapeworm carrier. In places with poor sanitation and domestic pig raising, pigs ingest human stools and get infected with cysticercosis. Humans can also acquire cysticercosis by fecal oral contamination from a tapeworm carrier in their surroundings (11). While cysts in most tissues may pass unnoticed or do not cause symptoms, a proportion of people with cysts in the nervous system will develop symptomatic NCC. The most frequent clinical manifestations of NCC are seizures, headaches, and intracranial hypertension. This review attempts to provide neurologists and clinicians worldwide with a comprehensive and detailed picture of the infection and subsequent disease, as well as its diagnostic and management aspects.

TYPES OF NEUROCYSTICERCOSIS

Appropriate diagnosis and management of NCC require to understand its diverse presentations, since both neuroimaging and serology need to be interpreted in the context

of each particular type and stage of the disease. A very simplified categorization would discriminate NCC lesions in the brain parenchyma (parenchymal NCC) from those in the ventricles or subarachnoid spaces (extraparenchymal NCC) (1).

Parenchymal NCC.

NCC lesions establish in the brain parenchyma as rounded cysts filled with clear liquid contents of a density similar to that of cerebrospinal fluid (CSF). Larvae initially establish as viable cysts and then as they are attacked by the host immunity, go through an involution process that ends in their resolution, complete or leaving a residual calcified scar (12).

Viable parenchymal cysts are usually between 0.5 and 2 cm in diameter, containing the scolex (the invaginated head of the tapeworm). Macroscopically the scolex can be seen as a white nodule, and it is also visible on CT or MRI images (Figure 1). Viable cysts are assumed to actively evade the host immune system and cause minimal perilesional alterations, although mild inflammation and gliosis have been shown on pathologic examination (13). On neuroimaging, viable, non-inflamed cysts do not show alterations in the surrounding brain tissue (14).

At some point in their evolution, the host immune system detects the cyst and launches an inflammatory response with disruption of the blood brain barrier, influx of immune effector cells and release of proinflammatory cytokines. This process becomes evident in neuroimaging as pericystic contrast enhancement (reflecting the BBB disruption) and edema. As a product of this attack the cyst losses its homeostasis, its contents gradually increase in density and become colloidal to then disappear, and the lesion shrinks to become an enhancing nodule (Figures 2 and 3). This process was described long ago by Escobar in pathology specimens (12).

As cyst degeneration progresses, the lesion is no longer visible on CT or MRI. Months later, in around 30 to 40% of resolved cysts, a calcified scar appears (15, 16). Calcifications represent dead larvae, however parasite tissue remnants can be found in the calcium matrix in some cases (17, 18). Calcifications are easily (and likely exaggeratedly) seen on CT as well-defined hyperdense rounded, nodular or punctuate solid lesions (Figure 4), but are difficult to find on MRI images. Although new MRI protocols may improve calcification detection, CT is much more sensitive for this type of lesions (14).

Single Enhancing Lesion (Solitary Cysticercal Granuloma).—In the Indian subcontinent, the vast majority of cases of symptomatic neurocysticercosis correspond to young patients with one or two small degenerating cysts, presenting with seizures. In general these patients have a benign clinical evolution, with lesion resolution and seizure remission in most cases. This presentation is also seen elsewhere but in a much lower proportion (19, 20).

Massive infections.—In rare cases, a patient may harbor a large number of larvae in the brain that may reach hundreds of cysts (Figure 5). Some of these patients have few symptoms (seizures usually), but in a subset of cases, a marked inflammatory reaction about the parasites may behave as a diffuse encephalitic syndrome (cysticercotic encephalitis)

(21). This presentation is more frequent in young females, and it is life-threatening. Control of inflammation is key and urgent, and the use of antiparasitic agents is contraindicated because damage to the cysts would enhance the existing inflammatory reaction, worsen the intracranial hypertension and lead to herniation and death. Individuals with many viable brain cysts are frequently found to also harbor the intestinal tapeworm by the time of the diagnosis of NCC (22).

Extraparenchymal NCC.

When the parasitic larvae establish outside the brain parenchyma they tend to grow and infiltrate the neighboring spaces, and the clinical manifestations are related to mass effects and intracranial hypertension rather than seizures (23). Extraparenchymal lesions may locate in the ventricles or in the subarachnoid space.

Ventricular NCC.—Cysts in the cerebral ventricles act as space occupying lesions that frequently block the CSF circulation and result in obstructive hydrocephalus (Figure 6). Cysts are more frequently located in the fourth ventricle but this may resulted from more symptomatic expression in this cavity (18).

Subarachnoid NCC.—Since cysticerci enter the CNS through the terminal vessels, many of them have contact with the bottom of a sulci or have some of their surface exposed to the subarachnoid space. However, in a majority of them most of their surface is covered and pressed by brain parenchyma (Figure 7), they maintain a spherical shape, do not grow considerably (with some exceptions), and follow the involutive process characteristic of parenchymal cysts. When a significant proportion of the cyst surface is open to the subarachnoid space, however, a different process occurs. The membrane grows, the cyst expands to the surrounding spaces, the membrane grows and infiltrate neighboring gaps, and at some point, the scolex is not evident anymore, likely “dissolved” in the expanding membrane. (24) This type of progressive subarachnoid NCC has long been known and described in the literature as “racemose” cysticercosis in relation to its appearance of a bunch of grapes, caused by multiple pseudocystic vesicular liquid entrapments in a membranous magma (25) (Figures 8 and 9).

Average time for evolution of cysticercosis types and stages.—While there is an enormous variability between different individuals, fragmentary information can be extracted from the literature about the duration of each stage and type of NCC. There are reports of individuals with viable cysts associated with neurological symptoms for many years, , and a blinded clinical trial demonstrated that 87% of cysts were viable after six months of observation in the comparison, placebo group (26). Clinical series demonstrate an average age for individuals with calcified NCC between 5 and 10 years more than those with viable NCC. All together this ecological sets of data suggest that the viable cyst stage lasts for a few years and in some cases cysts will survive long periods, although since community surveys also find many individuals with calcifications but no viable cysts, it is also possible that a large proportion calcifies in a shorter period with little or no symptoms. A very similar scenario occurs for subarachnoid NCC, where the pre-patent period may be

much longer (27). Individuals presenting with a single enhancing cyst, however, present a different picture. In many cases these lesions will resolve in a few months (19, 20).

Other localizations.

Two other localizations of cysticercosis in the central nervous system include the spine and the retina.

Spinal NCC.—In the spine, NCC is most frequently found in the extramedullary subarachnoid spaces, less frequently in the extradural spaces, and rarely as intramedullary cysts. Clinical manifestations of spinal NCC are related with compression and mass effects, or chronic arachnoiditis (28).

Retinal NCC.—NCC cysts can affect the retina causing visual disturbances, and much more rarely the optic nerve. Retinal cysts are evident on fundoscopy, and subretinal cysts are better diagnosed by ocular ultrasound and optical coherence tomography (29).

CLINICAL MANIFESTATIONS

The variable number, stage, and localization of the infecting parasites, added to the inflammatory response of the host, configure a wide array of possible symptoms that include most neurological manifestations. Among these, the most frequent are seizures in parenchymal NCC and headache and intracranial hypertension in extraparenchymal NCC.

Seizures.

Seizures are the hallmark of parenchymal NCC and in most cases are localization-related to at least one parasitic lesion (30), although in recent years a substantial body of evidence relates NCC with the development of hippocampal sclerosis and, apparently less frequently, temporal epilepsies (31). Seizures in NCC result from a focal lesion associated with inflammation and tissue alterations including gliosis and neuronal damage (32–34).

Does NCC cause epilepsy?—The presence of inflammation has long confused clinicians on whether seizures in NCC could be purely acute provoked seizures and not really the result of a defined epileptogenic process. Seizures in NCC, however, persist in many cases for years after the parasite has calcified, (35) are usually of the same type and focality, and can occur with no evidence of inflammation on MRI. Seizure relapse in individuals with calcified NCC rounds 50% or more, and relapses occur after long periods of months or years. Multiple studies in diverse endemic regions have consistently estimated odds ratios of approximately three times for the association between cysticercosis and epilepsy. All together there is no doubt that NCC is an important cause of epilepsy worldwide. Acute provoked seizures may occur in the early stages of cyst degeneration without leading to epileptogenesis, particularly in the Indian scenario where most cases are young individuals with only one or two small degenerating cysts. In this particular subgroup, the prognosis for those individuals who clear the lesion without residual calcification is very good, not so for those whose lesions calcify.

Headache.

Headache is a very frequent complaint in NCC, and it includes headaches arising from intracranial hypertension, post ictal headaches, or even primary headaches including tensional headaches or migraine (36–38). Chronic headache should raise the suspicion of extraparenchymal NCC. With the exception of patients with established intracranial hypertension where papilledema can be found and neck stiffness in chronic meningitis, physical examination in patients with NCC and headache is usually normal.

Intracranial hypertension.

As seizures are the hallmark for intraparenchymal NCC, intracranial hypertension is the major clinical manifestation in extraparenchymal NCC and should prompt rapid neuroimaging evaluation and subsequent intervention. Intracranial hypertension can result from direct mass effects in large cysts or cyst conglomerates, perilesional edema, or from ventricular obstruction and hydrocephalus. Hydrocephalus can be caused by intraventricular lesions or chronic ependymitis or arachnoiditis.

Focal deficits.

Mass effect and subsequent focal deficits may result from large cysts or cyst clusters in the subarachnoid spaces, from perilesional edema, or after repeated partial seizures (like Todd's paralysis). The later may take a few days to resolve.

Cognitive and psychiatric manifestations.

While cognitive and psychiatric alterations have always been mentioned in the NCC literature, (39, 40) systematic assessments of their frequency and characteristics were lacking. Recent works demonstrate a high frequency of depression and cognitive alterations in patients with NCC (41, 42).

DIAGNOSIS

The diagnosis of NCC rests in neuroimaging and can be confirmed by appropriate serological testing. Ideally, both neuroimaging and serology should be made available and their correlations or discrepancies should be carefully interpreted in the context of a given case.

Neuroimaging.

Computed tomography (CT) and magnetic resonance imaging (MRI) are the usual tools for the diagnosis of NCC. CT is cheaper and more available than MRI in poor regions, and has the advantage of being highly sensitive for the detection of calcified lesions. On the other hand, the definition of CT images is less precise than MRI, and it would underperform to detect small lesions or lesions close to the skull or in the posterior fossa. Similarly, intraventricular or cisternal lesions are less defined on CT than in MRI, where the use of different signal protocols allow better distinction of lesion characteristics, edema, and membranes or cysts in ventricles or cisterns. In addition, the capacity of MRI of

multiple planar reconstructions without losing imaging definition makes it a much better tool, although much less available and affordable.

Serology.

While neuroimaging is the pillar of the diagnosis of NCC, proper use of serological tests is extremely useful not only to confirm the diagnosis but also to characterize the viability of the infection, to monitor its evolution and response to therapy, and to rule out other differential diagnoses. The test of choice for antibody detection is the enzyme-linked immunoelectrotransfer blot assay (EITB) using lentil-lectin purified glycoprotein parasite antigens (LLGP) (43). The immunoblot strip consists of seven specific antigen bands belonging to three protein families (GP50, T24/42, and the 8kDa families), and has a sensitivity of 94 – 98% and a specificity of 85 – 100% for more than one viable cyst but lower sensitivity and specificity in cases with a single viable cyst (44, 45). Analyzing the presence and appearance of antibodies to the three protein families can orient clinicians on the likelihood of cyst viability or more severe infection. The sequential appearance of specific anti-*T. solium* antibodies to these proteins could be seen in Figure 10. The GP50 appears early, followed by the T24/42 and the 8 kDa family proteins. Analysis of band patterns reveals more information on the diagnosis and staging of NCC. GP50 alone is rarely associated with established infections with multiple cysts or extraparenchymal NCC. Recognition of T24/42 antibody bands suggests non-viable NCC and viable intraparenchymal NCC. The appearance of all families was associated with high-burden intraparenchymal and extra parenchymal infections (46). The LLGP-EITB is a complicate assay requiring access to purified antigens and laboratory capacities frequently not available in rural endemic regions. When available, however, it outperforms all other antibody diagnostic tests for NCC by far. Recombinant antigens of GP50 and T24 and synthetic peptides of the 8 kDa protein family have been used to make the test simpler and more reproducible. The performance of these antigens in combination was comparable to the LLGP-EITB with sensitivities of 98-99% and specificities of 98% for more than one viable cyst. The sensitivities are 56% - 65% for solitary viable cyst, while the sensitivities in LLGP-EITB are 52% - 79% (47). As these LLGP-EITB and recombinant antigens- and synthetic peptide immunoblot are not widely available, several companies have produced commercial antibody detecting tests. Two commercially available ELISA kits have very low sensitivities to detect antibodies in patients with viable NCC (22.2%-44.4%) with high cross-reactivity to sera from patients with hydatid cysts (48). This study supported previous publication on evaluating the performance of five commercialized assays that all have sensitivities below 72% and cross-reactivities to E. granulosus positive samples (49).

A complementary immunodiagnostic approach is the detection for circulating parasite antigen (50, 51). The assay platform is an ELISA using a pair of monoclonal antibodies to capture and detect the target antigens. As these monoclonal antibodies were developed against *T. saginata*, the assays are not species but genus-specific. This strategy provides the assay with increased specificity at the genus level and limits cross-reactions as other cystic larval *Taenia* infections very rarely infect humans (*coenurosis, T. serialis*) or do not infect humans at all (*T. saginata* or *T. hydatigena* cysticercosis). Ag-ELISA is less sensitive than the LLGP-EITB, but its advantages relate to demonstrating live parasite infections (test

results are negative in cases with resolved cysticercosis) and its rapid decay after effective treatment, allowing its use to monitor therapy results.

While serology has a complementary role in NCC, a positive antibody or antigen test without diagnostic imaging should not be interpreted as NCC as myocysticercosis or ocular cysticercosis could be positive without NCC (52, 53).

Molecular tests.

So far, PCR tests for NCC are restricted to exploratory reports in CSF of extraparenchymal NCC without convincing data on their performance for intraparenchymal NCC where most diagnostic doubts arise (54–57). Metagenomic analysis in CSF is increasingly reported to find unsuspected NCC cases and can become a useful tool in the near future.

Diagnostic criteria.

Diagnostic criteria for cysticercosis were first published by Del Brutto et al in 1996, (58) then refined and focused on neurocysticercosis rather than all cysticercoses in 2001 (59). The 2001 criteria was validated by an external group and found to be 93.6% sensitive and 81.1% specific. The most recent update, published in this same journal in 2017 (60, 61) (Table 1), includes significant improvements. It now focuses on neuroimaging as the primary support for diagnosis, and modified clinical/exposure criteria to make them more functional. Modifications to the Del Brutto criteria to make it more suited to specific settings such as India and Africa have been suggested (62, 63).

TREATMENT

Symptomatic drugs.

The first approach to a patient with NCC is appropriate symptom management. Analgesic, antiepileptic, anti-inflammatory and anti-edema drugs should be as indicated, and surgical management should be contemplated in cases with hydrocephalus or mass lesions. Carbamazepine is the most used AED drug (mostly because of availability in endemic regions), although levetiracetam is being increasingly prescribed (64). Some authors recommend the use of clobazam (65). Steroids are of use when patients present with perilesional inflammation or intracranial hypertension (66).

Antiparasitic drugs.

There is an extensive literature on the use of antiparasitic drugs to treat NCC. Interestingly, there was no specific agent until 1978, when Mexican researchers observed an effect of praziquantel in cysticercotic pigs, and then translated it to treat a child with inflamed parenchymal cysts, with good results (67). Albendazole was introduced a few years later as a more efficacious alternative (68). More than 40 years after the introduction of PZQ, most experts agree that antiparasitic treatment is of radiological and clinical benefit in most cases of NCC, particularly in individuals with multiple viable cysts or those with subarachnoid NCC (69). The usual regime includes one to two weeks of albendazole at 15 mg/k/day divided in two doses, a regime that may extend to one month or more for subarachnoid disease. In multiple intraparenchymal cysts praziquantel can be added at 50 mg/k/day.

Antiinflammatory treatment during antiparasitic therapy. Risks of long term steroids.—As noted, antiparasitic treatment affects parasite homeostasis, release antigen, and cause perilesional inflammation and blood brain barrier disruption, with focal edema and increased symptoms. Steroids are very efficacious in controlling this undesirable effect. However, long term steroid therapy carries significant side effects itself and steroid-sparing agents such as methotrexate and anti-TNF agents seem promising alternatives (70, 71).

Effect of parasite destruction in the evolution of seizures.—After praziquantel was used for first time for NCC in a child in Mexico in 1979 (71), it was well received in most endemic regions, where clinicians deal frequently with multicystic viable NCC cases, or extraparenchymal NCC, that frequently course very poorly if left untreated (67, 68, 72–74). The enthusiasm was lower in India, in centers treating pediatric NCC, and in non-endemic countries (all scenarios where most cases of NCC correspond to a single enhancing brain lesion, with a relatively benign course with symptomatic therapy alone) (75–77). While the difference between single or multiple lesion parenchymal, subarachnoid and ventricular NCC was long known, there was little awareness that they could respond differently to a pharmacological treatment. Once the use of PZQ and ABZ became more common, side effects including exacerbation of neurological symptoms, seizures, intracranial hypertension and even death were noted days after therapy onset. These manifestations were assumed to result from local inflammation against antigens released by the damaged parasite, and responded well to the addition of concomitant steroid therapy (78, 79). Adding to confusion, some authors claimed that antiparasitic therapy did not destroy the cysts, do not reflect in better seizure evolution, and may even result in a poorer prognosis (80, 81). Several randomized trials of antiparasitic treatment in multicystic viable parenchymal NCC are available now and consistently demonstrate that antiparasitic treatment resolves viable cysts (and associated mass effect), reduces the likelihood of disease progression, and results in fewer seizure relapses, making antiparasitic treatment the preferred approach in most NCC cases (26, 82–86). Complete resolution of all cysts however, does not warrant permanent seizure remission and a subgroup of patients will continue presenting seizure relapses.

Surgery.

Surgical options in the management of NCC include placement of ventricle-peritoneal shunts to control hydrocephalus, as well as excision of large intraparenchymal cysts, subarachnoid cyst clusters, or intraventricular cysts. Neuroendoscopical exploration and cyst exeresis is currently the approach of choice for intraventricular NCC. Neuroendoscopy is minimally invasive and safe although in some cases cysts adhered to the ventricular may lead to intraventricular bleeding (87, 88).

PROGNOSIS

Parenchymal NCC is associated with a reasonable good evolution. However, up to 50% of individuals with calcified parenchymal NCC will experience seizure relapses and require long term AED therapy (89–91). Cognitive and psychiatric alterations are common but usually not severe enough to alter daily performance.

Extraparenchymal (subarachnoid or ventricular) NCC does not respond to antiparasitic treatment as readily as parenchymal NCC and prolonged or multiple courses of therapy are required. Also in these cases the likelihood of complications such as vasculitis or shunt blockage is higher and should be taken into account when deciding on concomitant anti-inflammatory therapy.

Most factors associated with breakthrough seizures or further seizure relapses in NCC are similar to those in other epilepsies (number of prior seizures, length of seizure-free period, the length of AED therapy before withdrawal). From disease specific factors, the presence of a residual calcification or a gliotic scar are consistently associated with a much higher likelihood of seizure relapses, and recent work by our group also suggests that the size of the original cyst, the baseline inflammatory response (92) and the characteristics of the anti-inflammatory and antiparasitic treatments used (93) may influence the likelihood of seizure relapse.

CONTROL AND ELIMINATION

Endemicity and sustained transmission of *Taenia solium* require the coexistence of domestic pig raising and poor sanitary conditions (1, 79). Development and urbanization result in the disappearance of these conditions and consequently transmission gradually disappears (94–96). Since development may take decades or even never occur, active intervention programs to eliminate *Taenia solium* transmission have been planned since the early 1980s. The potential for elimination and posterior eradication (97) rests on effective diagnostics and medical treatments for taeniasis and cysticercosis, having pig as the only usual (and handy) intermediate host, and no invertebrate vector. Praziquantel was used for mass deworming in Ecuador in the mass human deworming in 1985 (98) and then in other countries in Latin America (99–101) and Africa (102). A decrease in transmission and morbidity was reported in Salama, Honduras following a sustained health education and control program (103). More recently, a large elimination program covering a population of approximately 80,000 people demonstrated elimination of transmission in 105 out of 107 villages by using a combination of human and porcine mass chemotherapy, pig vaccines, and stool coproantigen case confirmation (104). Sustained poor living conditions and domestic pig raising are the norm in most developing countries. However, cysticercosis continues to be a neglected disease and no systematic efforts towards its elimination through active intervention have been enacted anywhere.

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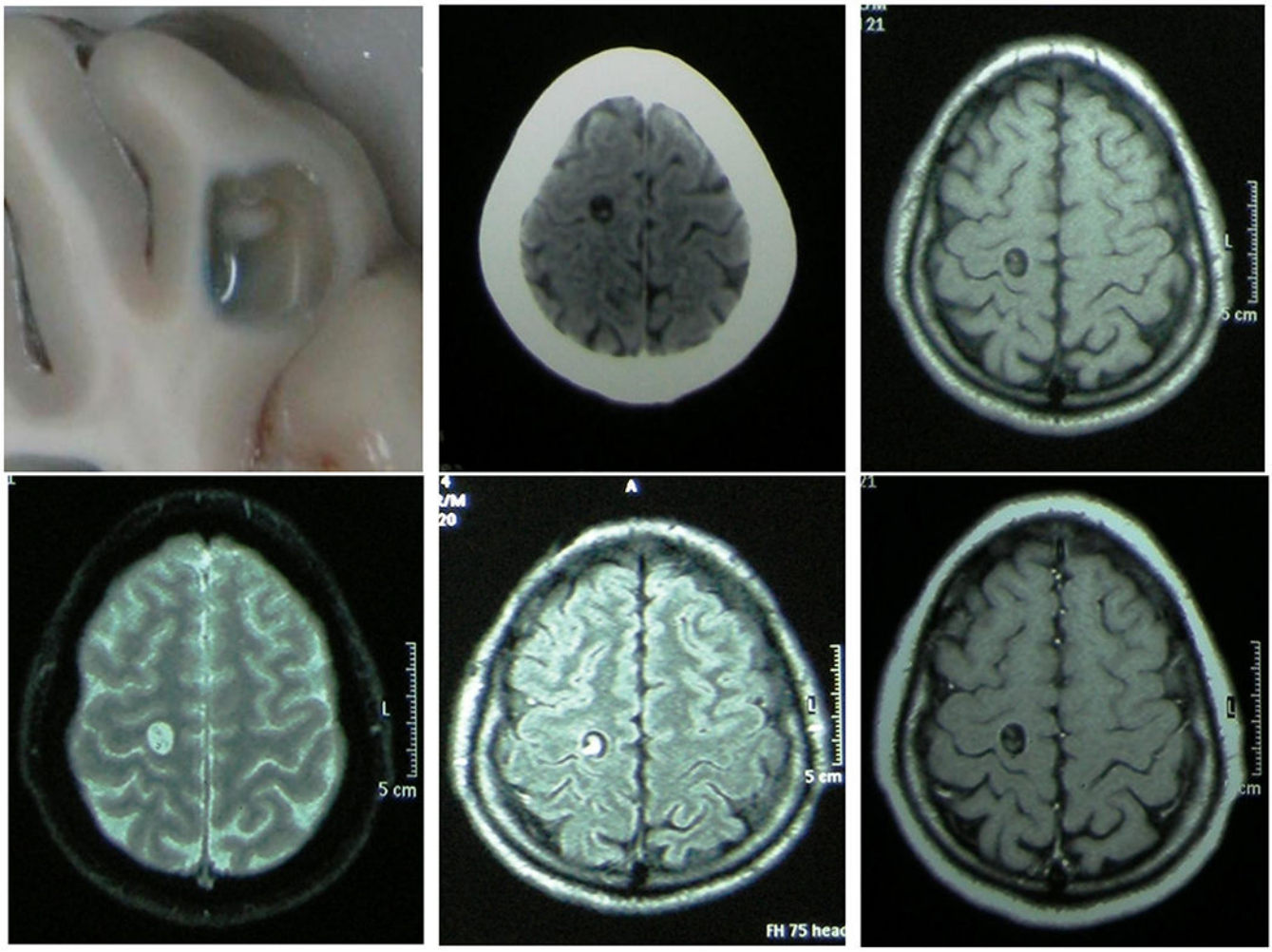

Figure 1, Viable cyst without inflammation.

From left to right, upper row: macroscopic view of a cysticercus in the brain of a pig showing the cystic cavity and the scolex; contrast-enhanced CT scan, and non-contrasted T1 MRI. Lower row: non-contrasted T2 and FLAIR, and contrast-enhanced T1 MRI images. Note the absence of contrast enhancement or perilesional edema.

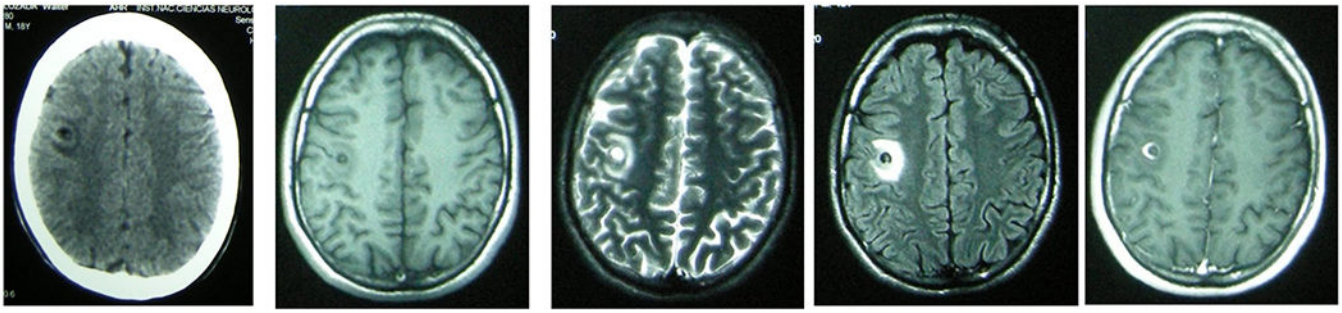

Figure 2, Viable cyst with inflammation.

From left to right: contrast-enhanced CT scan, non-contrasted T1, T2 and FLAIR, and contrast-enhanced T1 MRI images. Note the presence of perilesional edema and contrast enhancement.

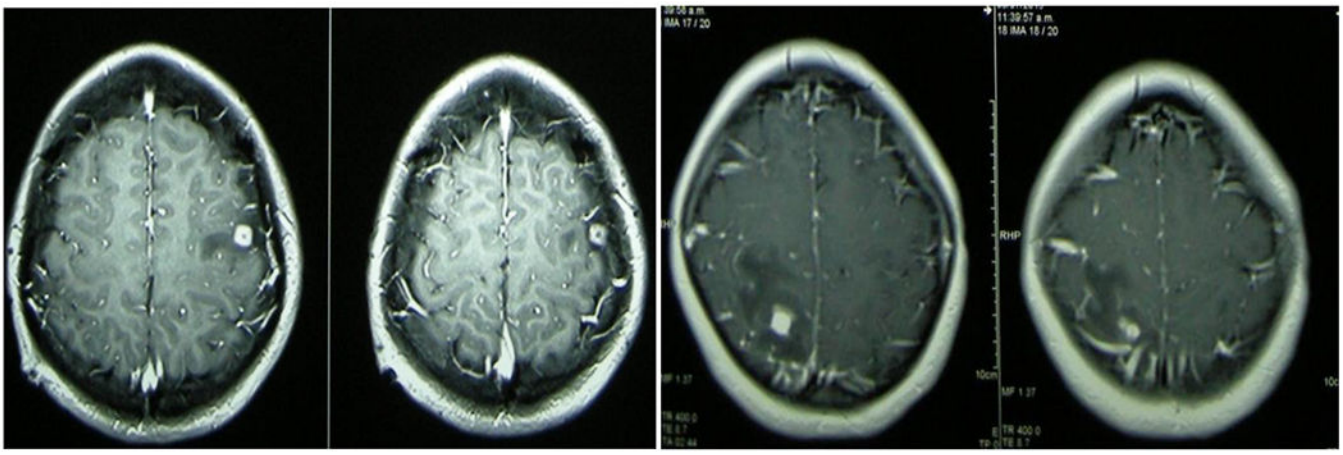

Figure 3.

Degenerating cyst (“enhancing lesion”). Contrast-enhanced CT demonstrating a ring enhancing lesion (left) and a nodular enhancing lesion (right), both with perilesional edema. Liquid cyst contents are not noticeable anymore.

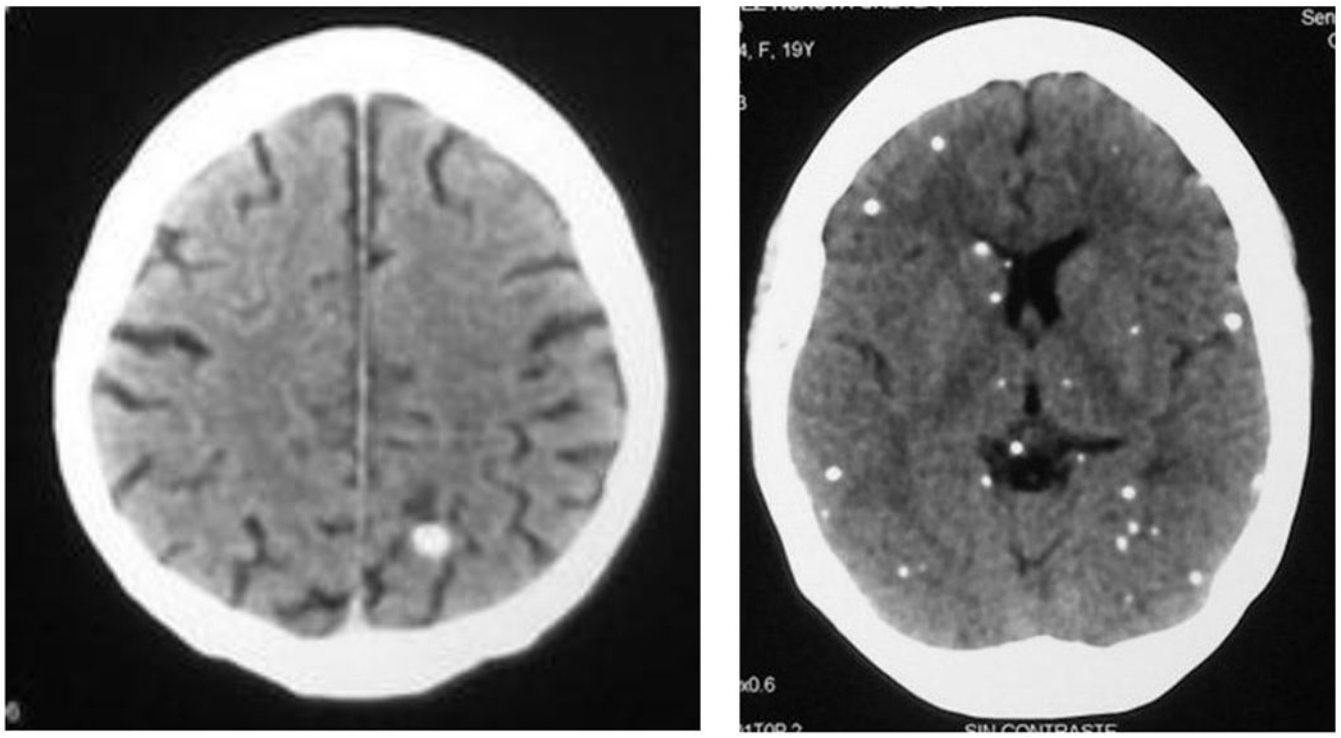

Figure 4.
Single and multiple calcified cysts on non-contrasted CT scan

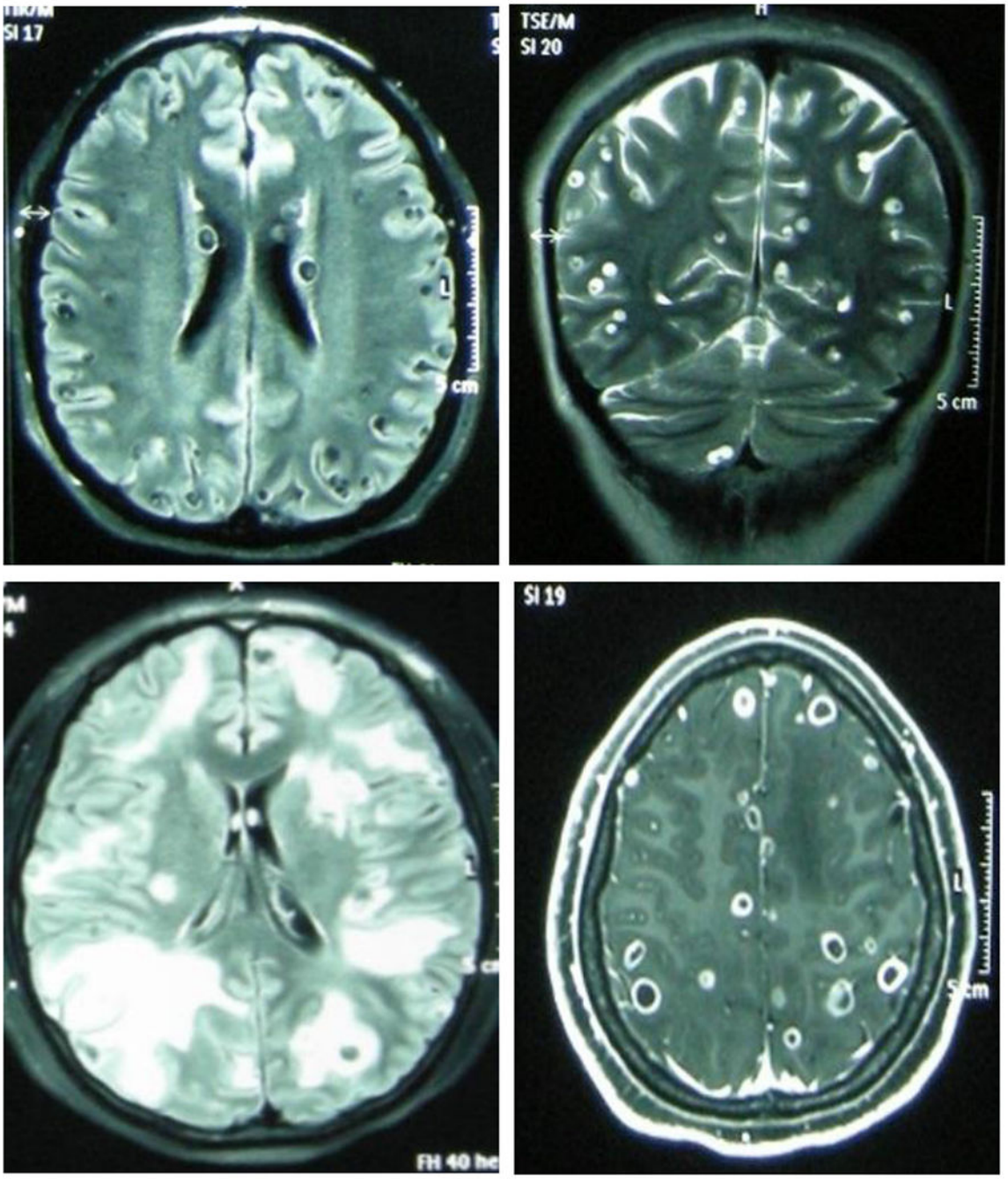

Figure 5.
Massive parenchymal NCC, non-encephalitic (above, FLAIR and T2 MRI protocols) and encephalitic (below, FLAIR and post-contrast T1)

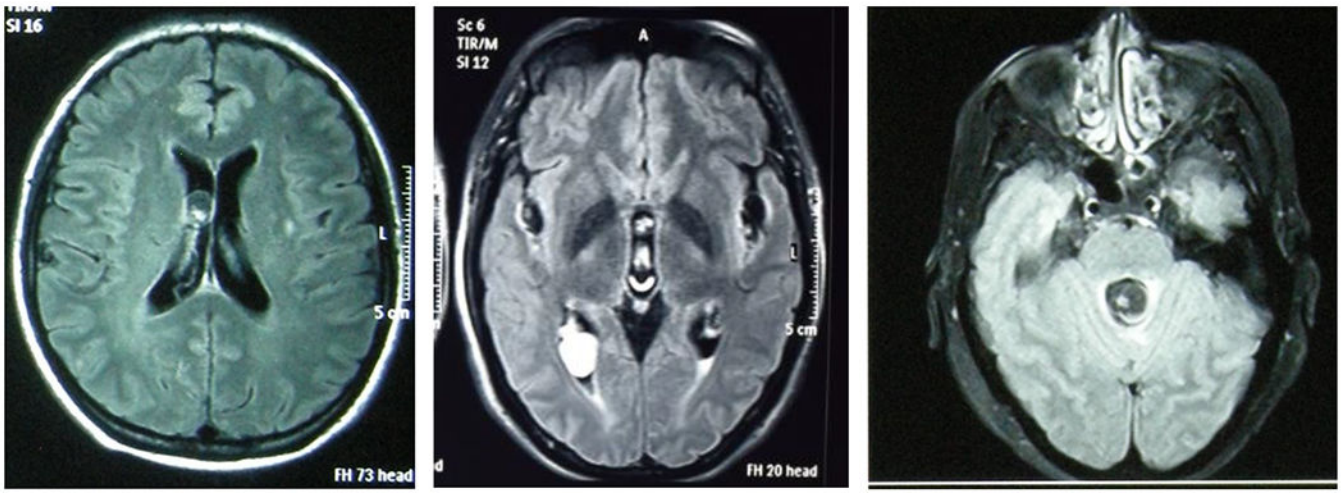

Figure 6.

Ventricular NCC. Left-viable cyst in a lateral ventricle; Center - degenerating cyst in the occipital horn of the right lateral ventricle – note the change in the intensity of signal of the cyst contents; Right - IV ventricle cyst growing towards the aqueduct. All images are non-contrasted MRI, FLAIR protocol.

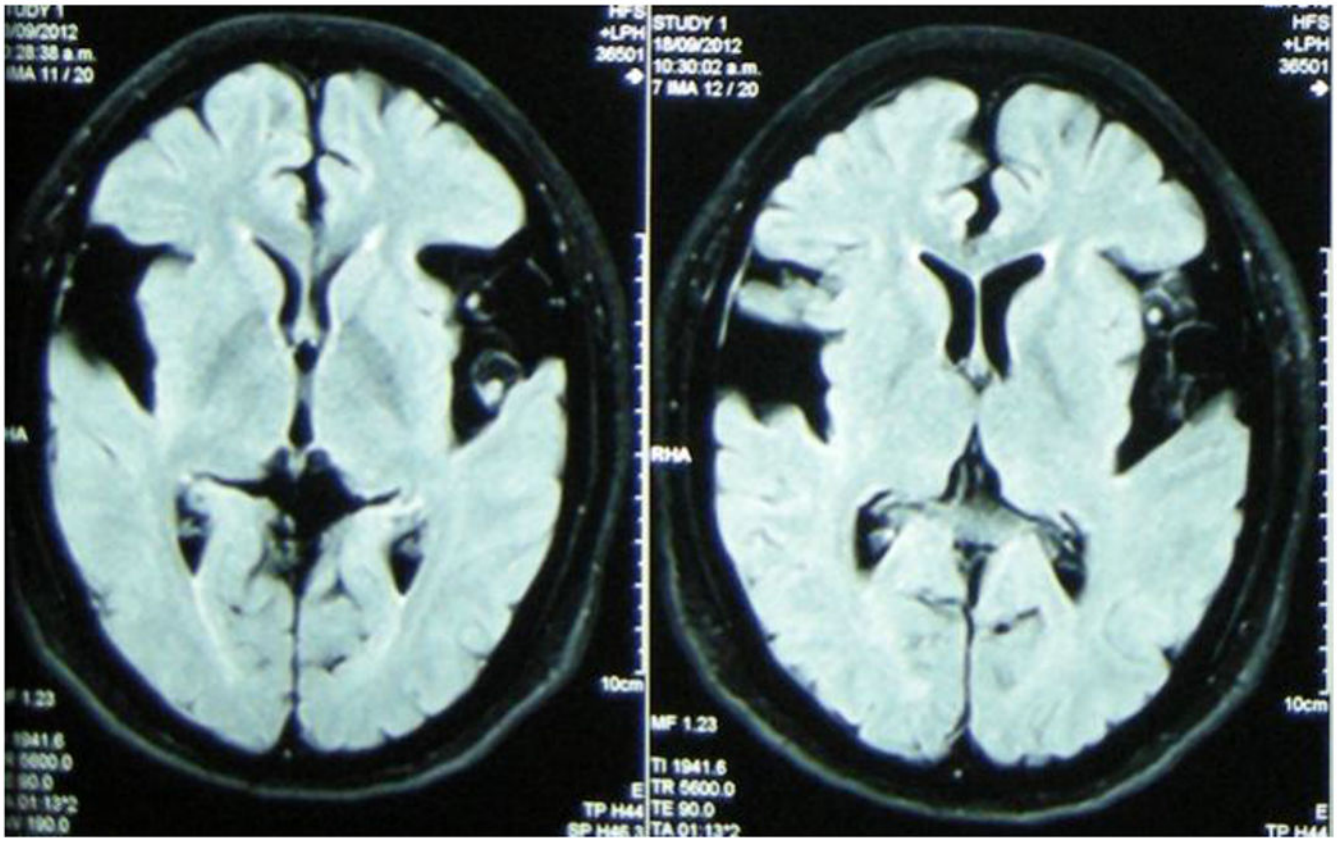

Figure 7.

NCC cysts in contact with the subarachnoid space at the Sylvian cisterns, with visible scolices (non-contrasted MRI, FLAIR protocol).

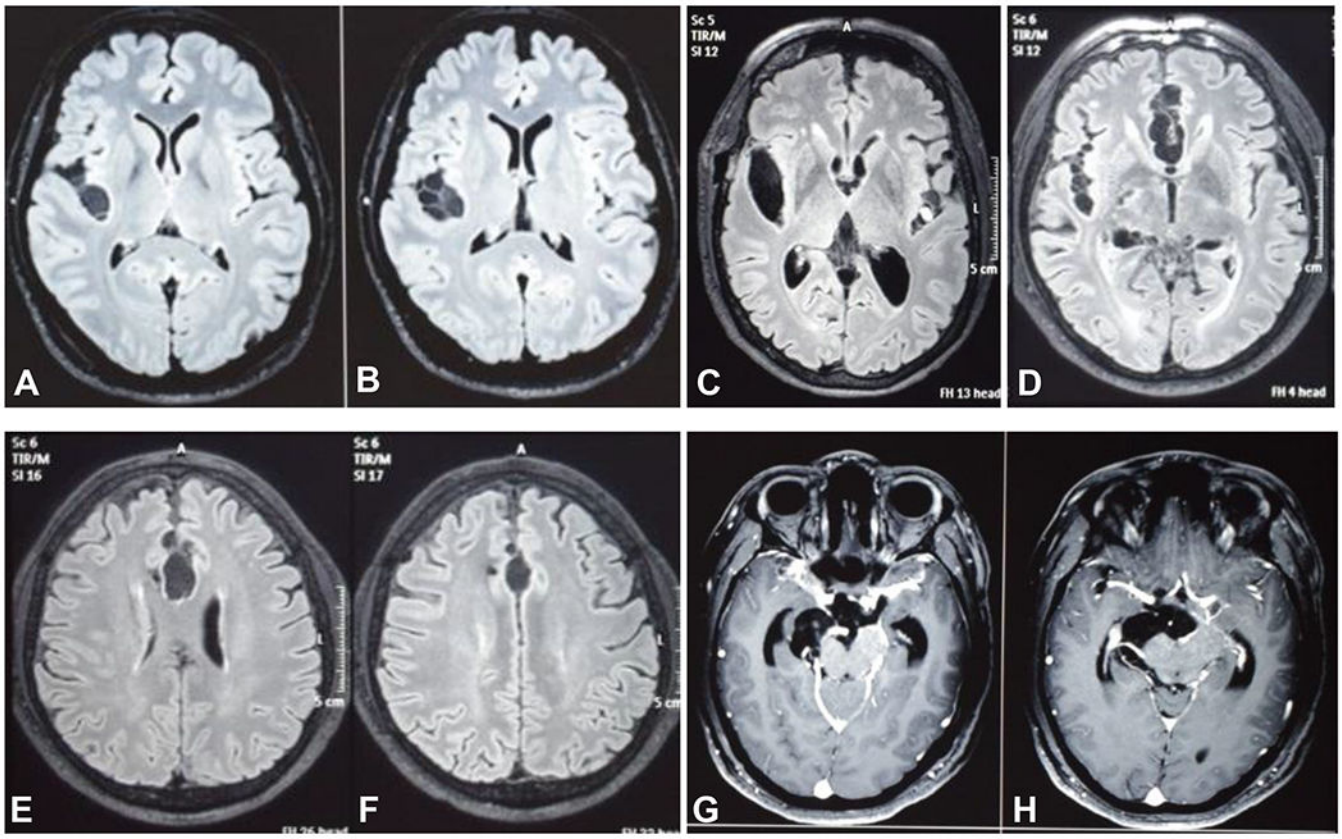

Figure 8.

Subarachnoid NCC. A - C) Sylvian; D) lower interhemispheric; E,F) upper interhemispheric; G,H) surrounding the brainstem (A to F, non-contrasted MRI FLAIR; G and H, post contrast T1-weighted images).

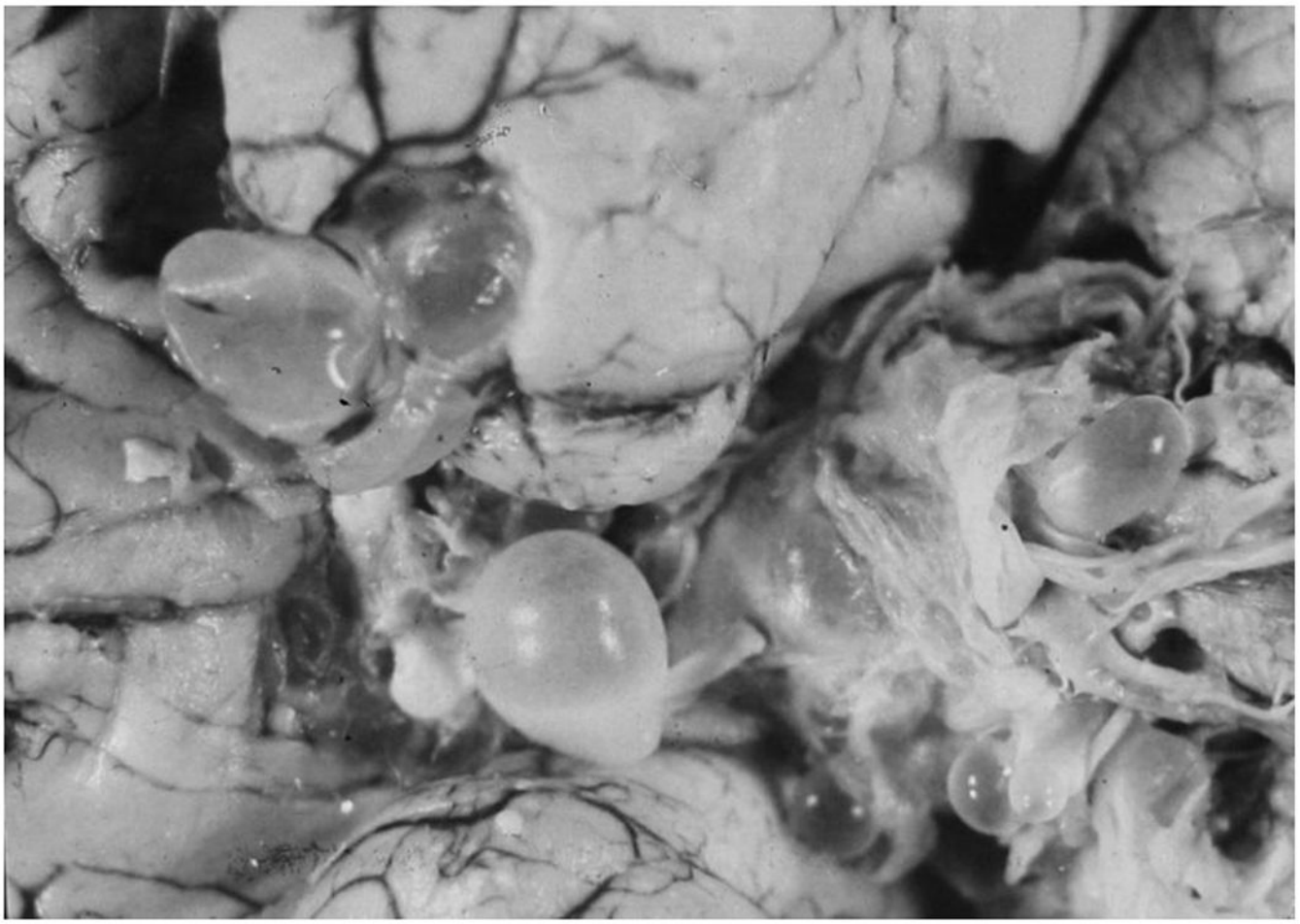

Figure 9.
Macroscopic pathology of basal subarachnoid NCC (Reproduced with permission from Garcia HH, Martinez SM (ed). 1996. Taeniasis/cisticercosis por Taenia solium. Editorial Universo, Lima, Peru).

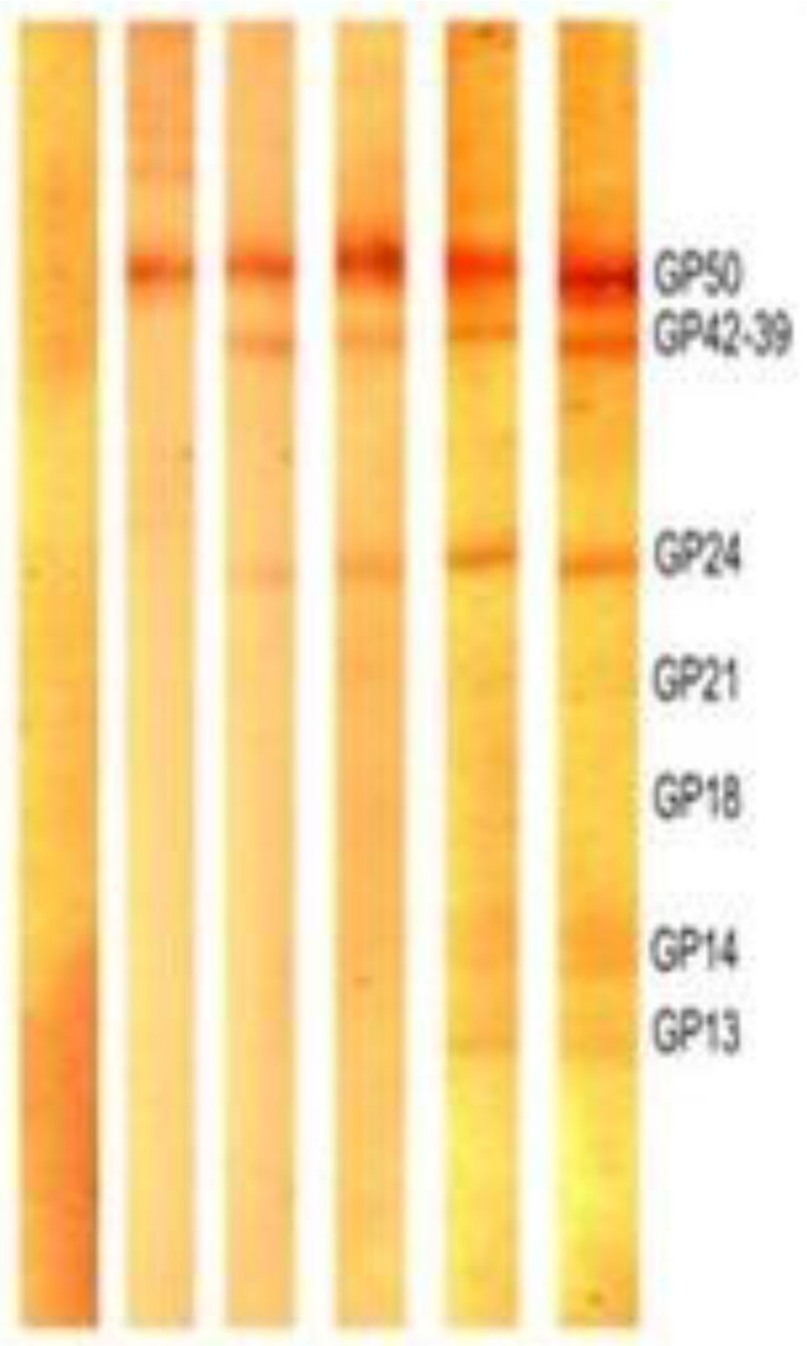

Figure 10.
Sequential appearance of specific anti-*Taenia solium* antibodies on enzyme-linked immunoelectrotransfer blot assay using lentil.lectin purified parasite glycoprotein antigens (LLGP-EITB) after experimental pig infection.

Table 1.

Del Brutto's diagnostic criteria for neurocysticercosis (from Del Brutto OH, et al. J Neurol Sci, 2017; 372: 202, with permission).

DIAGNOSTIC CRITERIA

Absolute Criteria:

· Histological demonstration of the parasite from biopsy of a brain or spinal cord lesion.

· Visualization of subretinal cysticercus.

· Conclusive demonstration of a scolex within a cystic lesion on neuroimaging studies.

Neuroimaging criteria:

Major neuroimaging criteria:

· Cystic lesions without a discernible scolex.

· Enhancing lesions. *

· Multilobulated cystic lesions in the subarachnoid space.

· Typical parenchymal brain calcifications. *

Confirmative neuroimaging criteria:

· Resolution of cystic lesions after cysticidal drug therapy.

· Spontaneous resolution of single small enhancing lesions. †

· Migration of ventricular cysts documented on sequential neuroimaging studies. *

Minor neuroimaging criteria:

· Obstructive hydrocephalus (symmetric or asymmetric) or abnormal enhancement of basal leptomeninges.

Clinical/exposure criteria:

Major clinical/exposure:

· Detection of specific anticysticercal antibodies or cysticercal antigens by well-standardized immunodiagnostic tests. *

· Cysticercosis outside the central nervous system. *

· Evidence of a household contact with *T. solium* infection.

Minor clinical/exposure:

· Clinical manifestations suggestive of neurocysticercosis. *

· Individuals coming from or living in an area where cysticercosis is endemic. *

DEGREES OF DIAGNOSTIC CERTAINTY

Definitive Diagnosis:

· One absolute criterion.

· Two major neuroimaging criteria plus any clinical/exposure criteria.

· One major and one confirmative neuroimaging criteria plus any clinical/exposure criteria.

· One major neuroimaging criteria plus two clinical/exposure criteria (including at least one major clinical/exposure criterion), together with the exclusion of other pathologies producing similar neuroimaging findings.

Probable Diagnosis:

· One major neuroimaging criteria plus any two clinical/exposure criteria

· One minor neuroimaging criteria plus at least one major clinical/exposure criteria.

[*] Operational Definitions.

Cystic lesions: rounded, well defined lesions with liquid contents of signal similar to that of CSF on CT or MRI

Enhancing lesions: single or multiple, ring- or nodular-enhancing lesions of 10–20 mm in diameter, with or without surrounding edema, but not displacing midline structures

Typical parenchymal brain calcifications: single or multiple, solid, and most usually <10mm in diameter

Migration of ventricular cyst: Demonstration of a different location of ventricular cystic lesions on sequential CTs or MRIs

Well-standardized immunodiagnostic tests: so far, antibody detection by enzyme-linked immunoelectrotransfer blot assay using lentil lectin-purified *T. solium* antigens, and detection of cysticercal antigens by monoclonal antibody-based ELISA

Cysticercosis outside the central nervous system: demonstration of cysticerci from biopsy of subcutaneous nodules, X-ray films or CT showing cigar-shape calcifications in soft tissues, or visualization of the parasite in the anterior chamber of the eye

Suggestive clinical manifestations: mainly seizures (often starting in individuals aged 20–49 years; the diagnosis of seizures in this context is not excluded if patients are outside of the typical age range), but other manifestations include chronic headaches, focal neurologic deficits, intracranial hypertension and cognitive decline

Cysticercosis-endemic area: a place where active transmission is documented.

[†] The use of corticosteroids makes this criterion invalid.