Review

Imaging spectrum of neurocysticercosis

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Abstract

Neurocysticercosis is the most common parasitic disease of the central nervous system, and also one of the most common causes of seizures in endemic areas. Globalization has caused the disease to spread around the world beyond the endemic regions. With no specific clinical symptoms of the disease, medical imaging plays an important role in the diagnosis of neurocysticercosis. Familiarity with these imaging findings may help greatly in early diagnosis, appropriate treatment decision, and follow-up of patients with neurocysticercosis.
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1. Introduction

Adult Taenia solium tapeworms infect only human beings (the definitive host). Humans become infected with these tapeworms by eating raw or poorly cooked pork products containing viable T. solium cysts — the encysted larval stage of the T. solium. Cysticercosis is a tissue infection by the encysted larval stage of the T. solium [1]. Although pigs are the main intermediate hosts of the T. solium, humans can also be infested by the larval stage of this tapeworm through the so called fecal-oral transmission or autoinfection pathways [2]. When the central nervous system (CNS) of humans (the most common place) is involved by the encysted larva, it is known as neurocysticercosis.

Neurocysticercosis is the most common parasitic disease of the CNS, and also one of the most common causes of seizures

in endemic areas (Latin America, parts of Oceania, Asia, Eastern Europe, and Africa). Recently, this disease has a tendency to spread all over the world (including the USA and Australia) as the population migrating around the world [2–4].

Cysticercosis has been known for centuries. The name "cysticercosis" derived from the Greek word "Kystic" meaning bladder and "Kercos" signifying tail. The second name "cellulose" was given by Rudolphi in 1809, due to its great affinity for connective tissue. It was not until 1855 that the relationship between Cysticercus cellulosae and T. solium was first demonstrated by Kuichenmeister. He made one of his prisoners eat cysticerci from a pig and recovered a T. solium worm from the prisoner's intestine after his death. Cobbold was thought to be the first one that described a case of neurocysticercosis in 1864 [5–7].

In the initial decades of the Twentieth Century, with the help of radiographs in detecting calcifications, hydrocephalus, mass effect etc., many clinical reports of neroucysticercosis had been published mainly focusing on the complications of such infections [6]. At that time, neurocysticercosis could only be recognized when patients developed symptoms. The

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symptoms were non specific, including seizures, headache, nausea or vomiting, focal neurological deficits and fever, stiff neck, etc. The knowledge of the disease was improved mainly by pathological studies at autopsy. Escobar and Nieto described four essential forms of neurocysticecosis: a) meningeal, b) ventricular, c) parenchymatous, and d) mixed forms. The meningeal and ventricular forms were found to be the predominant forms, and the racemose form was first established as a special type of neurocysticecosis [8,9].

Since the end of the Twentieth Century, with the clinical use of CT and MRI, our knowledge about neurocysticercosis infection and disease process has been revolutionized [1]. Multiple calcifications in cerebral parenchyma (Fig. 1) were the most common findings of neurocysticercosis in many initial CT studies [10−12]. There were also some unusual neuroradiological features of intracranial cysticersosis that had been reported [13,14]. As MRI became available, the comparison between CT and MRI in diagnostic ability of neurocysticercosis had been well documented. MRI had been proved to be superior to CT in detection and depiction of neurocysticercosis in both parenchymal and ventricular forms. But CT was better in showing the calcifications representing the end stage of parenchymal neurocystisercosis [15−19]. And for the first time, the imaging demonstration of the various stages of parenchymal neurocysticercosis was well correlated with pathology [17].

Nowadays, the advanced imaging of neurocysticercosis gives us the good opportunity to learn and understand this disease better. It helps greatly in the development of clinical classifications of neurocysticercosis, and is of paramount importance for determining the rational therapeutic approach for the different forms of the disease [20,21].

In this essay, we reviewed the typical and unusual imaging spectrum of neurocysticercosis.

2. Parenchymal neurocysticercosis

Neurocysticercosis most commonly manifests in the parenchyma of the brain and typically involves the cerebral hemispheres. Basal ganglia, brainstem, and cerebellum can

Fig. 1. Multiple calcified neurocysticercosis lesions. (Case diagnosed by clinical and lab data) Axial CT scan.

also be involved. The lesions are commonly found at the gray−white matter junction, presumably resulting from deposition of the larvae in terminal small vessels of these regions [22]. However, some authors mentioned that the "parenchymal" location of parasites as described at cross-sectional imaging actually represented subarachnoid cysticercosis located in deep sulci or in perforating branches of perivascular spaces [23]. But, in most of the current clinical and radiologic studies, parenchymal disease is still considered to be a separate and distinct form of neurocysticercosis. Because patients with parenchymal neurocysticercosis most commonly present with seizures, which is relatively easy to treat, and (except in individuals with heavy infections) has a fairly good prognosis [1].

After they have been ingested by humans, T. solium oncospheres (embryos form of larvae) pass through the stomach wall and reach the small blood vessels in the brain, carried by the bloodstream. In the brain parenchyma, they may develop into viable cysts after 2−3 months [1]. The earliest form of larval invasion is noncystic and is usually not detectable on imaging, because of silent clinical symptoms. However, occasionally lesions may develop associated edema and focal enhancement before cystic transformation. The reason is not quite clear, it may represent the inflammation of surrounding tissues [24]. Escobar's four pathological stages, which depicted the natural evolution of neurocysticercosis, had been well correlated with advanced imaging findings (CT and MRI) [25]. This classification is still most commonly used in current imaging studies in neurocysticercosis [20,22,23,26].

2.1. Vesicular stage (active)

After lodged in brain parenchyma for 2−3 months, the larva can be seen as a small marginal nodule (the scolex) projecting into a small cyst containing clear fluid (the cyst). The parasites are viable, escaping the host immune surveillance, and elicit little or no inflammatory responses in surrounding tissue. Usually there is no symptom in this stage as the cysticercal cysts mostly are small in size with an approximate diameter of 5−20 mm and no or little mass effect.

CT and MRI can both demonstrate the clearly marginated cyst with thin wall (2−4 mm), which usually does not show contrast enhancement. The cyst fluid is similar to the cerebrospinal fluid (CSF) in density or signal intensity (Fig. 2A). A discrete, eccentrically located scolex within the cyst is the pathognomonic imaging hallmark of this stage. The scolex can be iso-intensity or hyper-intensity on both T1-weighted and T2-weighted MR images, and may exhibit contrast enhancement. Fluid-attenuated inversion-recovery (FLAIR) MR images and diffusion-weighted (DW) images can frequently improve the visualization of the scolex (Fig. 2B) [27,28]. As mentioned above, there is no immune response by the host at this stage, and no surrounding edema is seen. Sometimes, these parasites are so numerous that the brain resembles a 'swiss cheese' appearance. The cyst may remain in this stage for months to years [25]. For reasons that are unclear, perhaps as a result of the natural degeneration of the parasite or as a
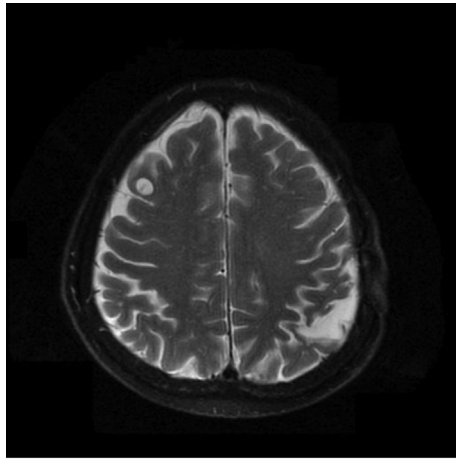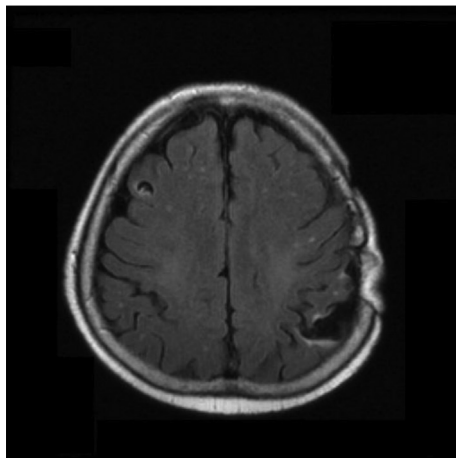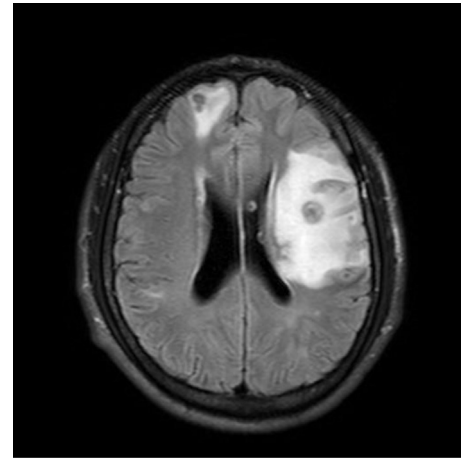
Fig. 2. Vesicular stage neurocysticercosis. (Case diagnosed by clinical and lab data, with medicine treatment follow up) A, Axial T2WI. B, Axial FLAIR. Note the CSF like cyst with an eccentric scolex in the right frontal region, obviously seen on FLAIR.

Fig. 3. Colloidal vesicular stage neurocysticercosis. (Cases diagnosed by clinical and lab data, with medicine treatment follow up) A, Axial FLAIR. The cysts show turbid fluid inside with thick wall, obscured scolex, and obvious surrounding brain edema. B, Axial T1WI with contrast in a different patient. Note the enhancement of the walls of multiple cysts.

consequence of antihelminthic treatment, these viable cysts are eventually recognized by the host. An intense inflammatory response is initiated then, which causes a stepwise series of degenerative changes.
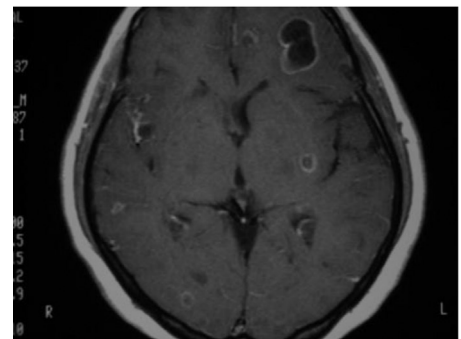
2.2. Colloidal vesicular stage (active)

In this stage, the larva begins to degenerate from the scolex, with signs of hyaline degeneration and shows gradual shrinkage of its size. The cyst fluid becomes turbid with proteinaceous content and its wall becomes thicker secondary to surrounding inflammatory response in the brain parenchyma.

On imaging, the cyst wall becomes thicker and irregular (late stage) with obvious contrast enhancement, which represents the break-down of the blood−brain barrier (BBB) by inflammatory process (Fig. 3). A recent study with T1-weighted dynamic contrast enhanced MRI indicated that K_{ep} may be used as a non-invasive image biomarker of BBB

break-down in different stages of neurocysticercosis [29]. The fluid within the cyst becomes hyperdense than that of the CSF on CT, and is slightly hyperintense on T1-weighted images, markedly hyperintense on T2-weighted or FLAIR images. With degeneration, the scolex decreases in size, and eventually disappears on imaging study.

When there is a single cyst at this stage with ring-like contrast enhancement, the differential diagnosis of single enhanced lesion (SEL) should be considered [1]. The clinical history of being from endemic area and the positive test result of serum or CSF immune assay (e g. Lentil lectin glycoprotein enzyme-linked immunoelectrotransfer blot, LLGP-EITB) may be helpful in the diagnosis of neurocysticercosis.

Although EITB has 100% specificity and an overall sensitivity of 98%, approximately 30% of patients with a single brain parasite may test negative [3]. DW imaging and magnetic resonance spectroscopy (MRS) may help in differential diagnosis, but overlaps of these imaging characteristics make things complex. Absence of diffusion restriction on DW imaging may help in differentiating neurocysticercosis from abscess, but cannot help to differentiate it from metastatic disease. ^{1}H nuclear MRS of the cysticercal fluid may demonstrate elevated choline, lactate, lipid, succinate, alanine,
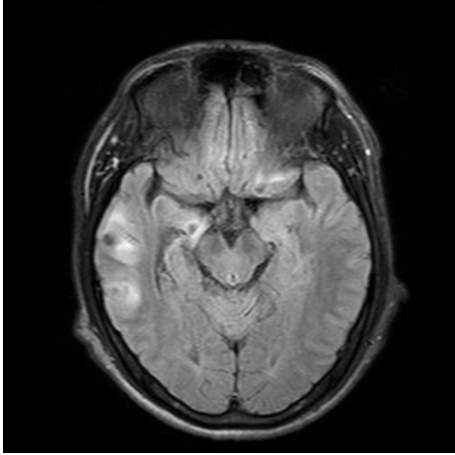
Fig. 4. Nodular-granular stage neurocysticercosis. (Case diagnosed by clinical and lab data, with medicine treatment follow up) Axial FLAIR. Note the shrinking cysts with minimum mass effect and surrounding gliosis.
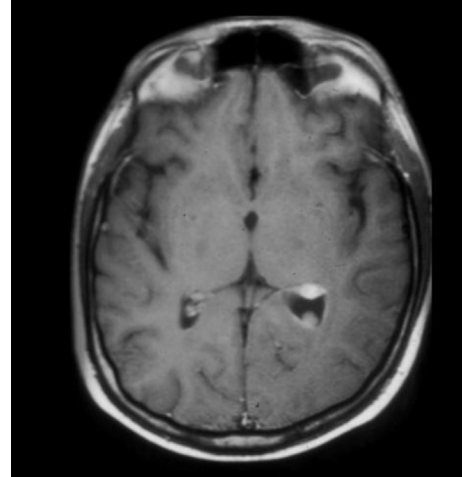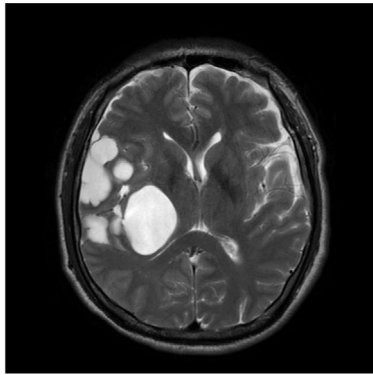

Fig. 6. Intraventricular neurocysticercosis. (Case diagnosed by clinical and lab data, with medicine treatment follow up) Axial T1WI with contrast. Note the obviously enhanced scolex within the cyst in the trigone of left lateral ventricle helps to make the correct diagnosis.

and acetate, and decreased N-acetylaspartate and creatine. Other infectious diseases may also have the similar MRS findings. However, MRS may help to differentiate neuro-cysticercosis from necrotic parts of neoplasm or cystic metastasis which demonstrates only lactate and lipid peaks [22]. When there are multiple lesions in this stage, the host immune response to the degenerated cysts may cause diffuse brain edema and collapse of the ventricular system without midline shift. This is called acute cysticercosis encephalitis, commonly seen in children [30].

2.3. Granular nodular stage (active)

As degeneration of the cysticercus progresses, the cyst decreases in size and transforms into a smaller granulomatous nodular lesion. Pericystic gliosis of variable severity is the most common pathologic finding [27]. The cyst may be seen as a nodular or a thick, small, ring-like enhancement. Sur-rounding edema is not as extensive as the late colloidal ve-sicular stage and decreases gradually (Fig. 4) [25].

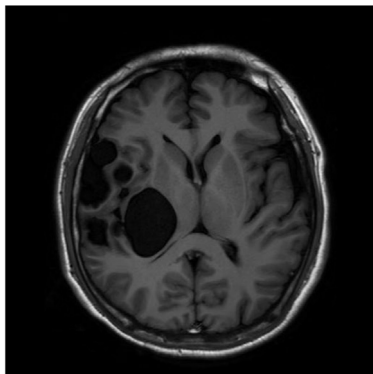### 2.4. Nodular calcified stage (nonactive)

In this final stage, the lesion has shrunk to one half or one quarter of its original size, and almost completely mineralized, with no surrounding edema. CT, better than MRI, can clearly depict the calcified nodule (Fig. 5). MRI may show signal void lesion on some sequences, particularly gradient echo sequence or susceptibility weighted imaging. Although this stage is a nonactive form of neurocysticercosis, recurrent seizure activity may be associated with the calcified nodule. Presumably, this is caused by an inflammatory response to an antigenic substance released from the cysticercal remnants in the apparently dead parasite. Mild contrast enhancement on MRI may be seen surrounding the calcification, sometimes with
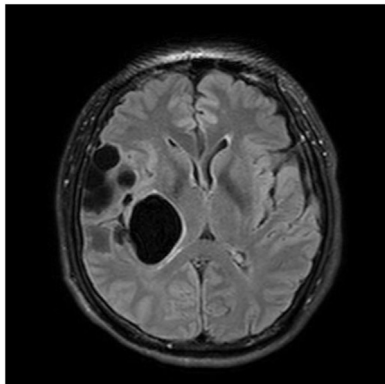

Fig. 7. Intraventricular neurocysticercosis. (Case diagnosed by clinical and lab data, with medicine treatment follow up) Axial CT scan shows the shunt placed for a cyst in the anterior horn of left lateral ventricle with obstructive hydrocephalus.
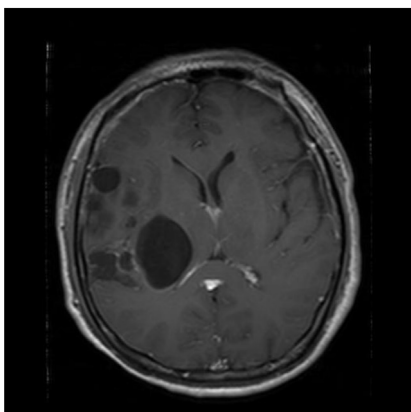

Fig. 5. Nodular calcified stage neurocysticercosis. (Case diagnosed by clinical and lab data) Axial CT scan. Note multiple calcified lesions.

A

B

C

D

Fig. 8. Subarachnoid neurocysticercosis. (Case diagnosed by pathology and lab data) A, Axial T2WI. B, Axial T1WI. C, Axial FLAIR. D, Axial T1WI
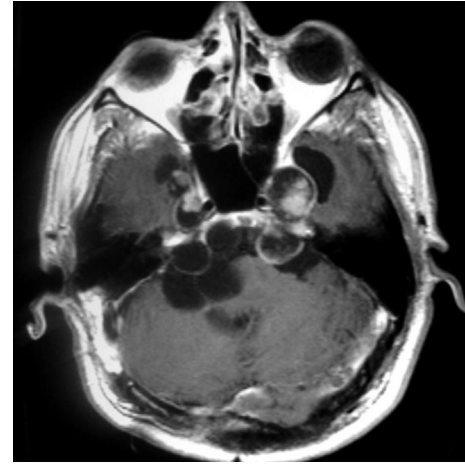

Fig. 9. Subarachnoid neurocysticercosis. (Case diagnosed by pathology and lab data) Axial T1WI with contrast shows multiple subarachnoid cysts agglomerated at the skull base with some showing enhancement and degenerated scolex.

minimal edema [31−33]. In cases that the calcified nodule is located within the hippocampus, the medically intractable epilepsy can be well controlled by hippocampal resection [34].

2.5. Imaging pearls of parenchymal neurocysticercosis

2.5.1. Vesicular stage
Small CSF-like cyst with thin wall and an eccentrically located scolex, no contrast enhancement of the cyst's wall, no surrounding tissue edema.

2.5.2. Colloidal vesicular stage
The density and signal intensity of the cystic fluid change from that of CSF. The cystic wall is thicker. The scolex becomes ill defined and finally shrinks in its size. Ring-like enhancement is seen. The surrounding tissue edema is obvious.

2.5.3. Granular nodular stage
Small enhancing cyst or nodule, with mild surrounding edema and little mass effect.

2.5.4. Nodular calcified stage
Small calcified nodule, no surrounding edema, better seen on CT.

3. Intraventricular neurocysticercosis

Intraventricular cysticercosis is the second common form of neurocysticercosis, which may account for 22% of all neurocysticercosis cases [18]. Usually intraventricular cysts occur in isolated fashion, sometimes may be seen in conjunction with

with contrast. Note multiple cysts with various sizes in the right Sylvan fissure. Some show minimal contrast enhancement.

A

B

C

D

parenchymal or cisternal lesions, which may help the diagnosis.

A solitary intraventricular cyst is the usual manifestation of this form of neurocysticercosis, which is most commonly seen in the fourth ventricle, followed by the third ventricle, the lateral ventricle and cerebral aqueduct of Sylvius. It usually has the same density and signal intensity with the CSF on CT and MRI. But MRI is obviously superior to CT in locating the cysts, depicting the wall, the scolex, the fluid in the cysts (Fig. 6), and the accompanying changes (e g. ependymitis), especially on FLAIR sequence with 100% supplemental O2 or Three-dimensional Constructive Interference in Steady State (3D-CISS). FLAIR sequence with 100% supplemental O2 can increase the signal intensity of CSF, while 3D-CISS is a heavily 3D T2WI high-resolution cistern imaging sequence. Both methods can help in differentiating cyst lesion from CSF [26]. The cysts are identified by the high signal intensity mural nodule, cyst wall outlined by cerebrospinal fluid in the ventricle on T1-weighted and FLAIR images. Granular ependymitis in surgically operated cases may be seen as ring-like or nodular enhancement, and require shunt placement even after surgical removal of the cyst [35]. As the cyst may be adherent to the ventricle wall or migrate in/out the ventricle system, it is important to confirm the location of the cyst before surgery. CT ventriculography, once a relatively invasive procedure in localizing the cyst in ventricle, has now been replaced by the non-invasive MR imaging methods [25].

The cyst may evolve like the parenchymal cyst, with thickened wall, ring-like enhancement and surrounding edema, but calcification is seldom seen [36]. As mentioned before, differentiation of such a ring-like enhancing lesion from neoplastic processes or other inflammatory processes may be difficult, correlation with clinical history, laboratory data, and previous imaging findings is necessary.

Unlike the parenchymal form, intraventricular cysticercosis may cause acute obstructive hydrocephalus, and is potentially lethal. Early diagnosis is critical in management of these patients [35]. Surgical treatment of intraventricular cysts may include cysts resection or just shunt placement (Fig. 7). A series of six cases with endoscopic excision of intraventricular cysticercosis had been reported with good prognosis [37].

3.1. Imaging pearls of intraventricular neurocysticercosis

CSF-like cyst with or without scolex in ventricular system, better demonstrated by MRI, may cause acute obstructive hydrocephalus.

Fig. 10. Subarachnoid neurocysticercosis-racemose form. (Case diagnosed by clinical and lab data, with medicine treatment follow up) A, Axial T2WI. B, Axial T1WI with contrast. C, Sagittal T1WI without contrast. D, Sagittal T1WI with contrast. Note the agglomerated cysts in basal cistern with enhancement of the wall and surrounding leptomeningeal, no obvious scolex can be seen. The basal artery is encased by the cysts with signs of vasculitis.
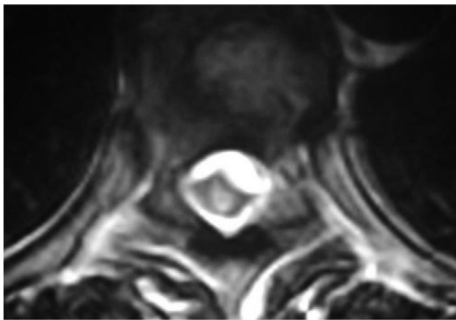
Fig. 11. Spinal neurocysticercosis -Subarachnoid form. (Case diagnosed by pathology and lab data) A, Sagittal T1WI. B, Sagittal T2WI. C, Axial T2WI. Note multiple cysts in the subarachnoid space of the thoracic spine with spinal cord compression.

Fig. 12. Spinal neurocysticercosis-intramedullary form. (Case diagnosed by pathology and lab data) A, T2WI with fat suppression. B, T1WI without contrast. C, T1WI with contrast and fat suppression. The intramedullary nodule shows hypointesity on T2WI, iso-intensity on T1WI precontrast, and intense enhancement postcontrast images.

3.2. Subarachnoid neurocysticercosis

The subarachnoid form of neurocysticercosis is the third common manifestation of this disease, and has been found in 3.5% of neurocysticercosis patients [38].

The cysts located within cortical sulci, like the parenchymal cysts, may be small in size with the pressure effect from surrounding brain parenchyma, and usually follow the same stages of evolution. Sometimes it is difficult to differentiate between the parenchymal form and subarachnoid form of the disease. On the other hand, the cysts located in the Sylvian fissure or within the basal CSF cisterns may reach a large size, with reported cases of lesions 10 cm and larger (Fig. 8) [2].

Cysts within basal cisterns usually manifest as a space-occupying lesion of CSF signal intensity or density. They may cause distortion of the involved cisterns and adjacent brain structures, and tend to agglomerate in a racemose form. It was thought that racemose cysts did not have scolex, but a recent study found that the degenerated scolex could be found in this form of neurocysticercosis [39]. Like the intraventricular form of neurocysticercosis, CT gives little help in depicting this form of lesions. MRI may show the cyst walls, the degenerated scolex, especially on FLAIR sequence or 3D-CISS (Fig. 9).

The contrast enhancement of the surrounding leptomeninges indicates the presence of arachnoiditis (Fig. 10), which is elicited by the degenerating cyst. This inflammation process may involve leptomeninges and corresponding arteries, cause hydrocephalus and vasculitis, even result in cerebral infarction. Segmental narrowing or even occlusion of the major intracranial arteries [40] may be a common angiographic finding in patients with subarachnoid neurocysticercosis, even in patients lacking clinical or neuroimaging evidence of a cerebral infarct [20]. Infectious aneurysms may also be associated with subarachnoid neurocysticercosis [13].

3.3. Imaging pearls of subarachnoid neurocysticercosis

CSF-like cyst (if no scolex present) with non specific imaging findings, small in sulci, large in fissure or cistern, with a tendency to agglomerate, may cause mass effect and arachnoiditis.

4. Spinal neurocysticercosis

Spinal neurocysticercosis is uncommon. The cyst can be found within the spinal cord parenchyma (intramedullary form) or within the subarachnoid space (leptomeningeal form). The lepotmeningeal form is more common and usually accompany with intracranial disease (Fig. 11) [41]. Intramedullary spinal cysticercosis is extremely rare (Fig. 12), with non-specific imaging finding as cyst lesion widening of spinal cord, indistinguishable from other intramedullary pathologic processes (tuberculomas, tumors) [16,42]. In a recent review of 43 patients with intramedullary spinal cysticercosis, most cysts were located at the lower thoracic levels. This segmental distribution is coincidence with the blood supply region of the artery of Adamkiewicz, which most often originates directly from the descending aorta and enters the spinal cord between the T9 and T11 levels. The prognosis of intramedullary cysticercosis may be better for medically-treated patients than for surgically-treated patients [43].

4.1. Imaging pearls of spinal neurocysticercosis

Rare, non specific cyst lesion on imaging, always search intracranial lesions for differential diagnosis.

5. Conclusion

Neurosticercosis is the most common helminthic infestation of the central nervous system in human and a leading cause of acquired epilepsy worldwide. As globalization progresses, this disease has spread to areas previously unknown to be endemic, like the USA and Australia. The neurologic symptoms are always non-specific. The ideal method of diagnosis of this disease is the combination of clinical, imaging, lab and epidemiologic data. . With the clinical use of more advanced imaging equipments, such as 7.0 T MRI scanner, the disease processes of neurocysticercosis may be better understood as anatomical and pathological details are illustrated. Familiarity with these imaging findings may help greatly in early diagnosis, appropriate treatment decision, and follow-up of patients with neurocysticercosis.

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