A comprehensive review of imaging findings in human cysticercosis

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Abstract Cysticercosis is a zoonotic disease most commonly affecting brain, eye and muscles, causing significant morbidity. Transmitted by faeco-oral route, this disease was endemic in many developing countries; it is now seen worldwide due to globalisation. Manifestations are produced by the mass effect of the parasite and by the immune response mounted by the host on the parasite. There are myriad clinical features and imaging findings. Radiological features depend on the number, stage and location of the parasite and associated complications. Knowledge of radiological findings is necessary to make an accurate diagnosis of this pleomorphic disease.

Keywords Neurocysticercosis · Computed tomography · Magnetic resonance imaging · Ocular cysticercosis · Intramuscular

Introduction

Cysticercosis is a common parasitic disease affecting many tissues in the body. Humans carrying the adult tapeworm in the intestines are the definitive hosts in the natural

life cycle of *Taenia solium*. Pigs are the intermediate hosts and are infected with the larval form (cysticercus) most commonly in the muscles. If humans accidentally ingest *T. solium* eggs, the larval stage develops in various tissues like brain, muscles, subcutaneous tissues and results in human cysticercosis [1, 2].

Cysticercosis is endemic in many developing countries. Owing to increased migration of people to developed countries, cysticercosis has gained global significance and has become a major health problem worldwide [1–3]. A thorough knowledge of the imaging findings of this pleomorphic disease is necessary to make an accurate diagnosis.

Cysticercosis most commonly affects the central nervous system causing neurocysticercosis (NCC). The signs and symptoms depend on the location of the parasite and the immunological response of the host on the parasite. It is the major cause of acquired epilepsy worldwide. Though CT may help in depicting the calcified stage and complications like hydrocephalus and stroke, MRI is the workhorse in the diagnosis of NCC and its various complications. Advanced MRI sequences like susceptibility-weighted imaging (SWI) and constructive interference in steady state (CISS) have aided in easier diagnosis. Orbital and muscular cysticercosis occurs less frequently than NCC, but causes significant morbidity. Disseminated cysticercosis occurs with heavy infection of the parasite and has characteristic plain radiograph findings as well.

In this article, we discuss and illustrate neurocysticercosis, orbital, muscular and disseminated cysticercosis, the life cycle of *Taenia solium*, pathogenesis, classification, the myriad clinical manifestations and radiological characteristics with the role of advanced MRI sequences.

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Epidemiology

Cysticercosis is endemic in countries in Africa, Latin America and Asia. With growing industrialisation and globalisation, the disease has spread to non-endemic regions owing to migration of people from endemic countries. Significant variation in the prevalence of disease is observed within each continent. Highest prevalence of active infection is seen in African countries. Occurrence of infection is determined by age and immunity of the human host, while exposure is mostly related to environmental factors such as poor hygiene, improper sanitary conditions and open defecation which varies from community to community [4].

Parasitology

Cysticercosis and taeniasis are zoonotic diseases caused by infection with larval and adult forms of the tapeworm, *Taenia solium*. Taeniasis occurs after the ingestion of undercooked pork infected with cysticercus. *Taenia solium* has a scolex or head with four suckers and a double crown of hooks, a neck and a large strobila measuring 2–4 m consisting of several proglottids or segments. The worm attaches strongly to the mucosa of the upper small intestine by means of its suckers and hooks. After about 2 months, gravid proglottids begin to detach and are excreted in the faeces. Each proglottid contains about 50×10^3 fertile eggs. After ingestion by the intermediate host (pig), the eggs are digested by gastric juices and develop into larvae (oncospheres) which penetrate the small bowel and are deposited throughout the body of the pig, especially in the skeletal muscles. The oncosphere then develops into a cysticercal or encysted larva. When humans consume raw or undercooked pork, the cysticercal larvae develop into adult tapeworms, resulting in taeniasis, and completing the life cycle [5].

Humans get cysticercosis when they swallow these eggs that are excreted in the stool of people with the adult tapeworm. This happens when people drink water or eat food contaminated with tapeworm eggs or by external autoinfection when they put their contaminated fingers in their mouth. Thus, vegetarians and other people who do not eat pork can acquire cysticercosis. Water, wind, flies, and other indirect means of infection play little part in transmission. Internal autoinfection by regurgitation of proglottids into the stomach in taeniasis has also been proposed [6].

Pathogenesis

The embryos present in the eggs, released by the action of gastric acid and intestinal fluids, enter the bloodstream and

disseminate haematogenously to various tissues, encysting as cysticerci. The cyst wall has numerous vesicles. The outer layer, called the tegmentum, is composed of fine hair-like projections, and the inner layer has a loose fibrillated matrix and network of canaliculi representing the excretory system of the parasite, fascicles of muscles, calcareous corpuscles and few foci of calcification. The cyst fluid is clear as long the parasite is alive, with invagination in its wall corresponding to the head of the parasite or scolex. As long as the cyst wall is intact, there is minimal or no inflammatory reaction. The cysts may be located in the brain, subcutaneous tissue, muscles, eyes and rarely spinal cord and other tissues, in decreasing order of frequency. The signs and symptoms are produced by the presence of the parasite in the form of mass effect, inflammatory reaction mounted by the host and residual scarring due to long-standing inflammation [7].

Neurocysticercosis

The parasite commonly affects the central nervous system, causing neurocysticercosis, a heterogeneous clinical disorder. Cysticerci are located in the brain parenchyma, subarachnoid space, ventricles and rarely in the spinal cord. Cysticerci may remain dormant for a long time in this stage, protected by the blood–brain barrier and active immune-evasion mechanisms by the cysticerci [5]. After a variable period of time, the parasite undergoes degeneration with associated immune-mediated inflammation. Cysticerci cause symptoms because of mass effect or by hampering the circulation of cerebrospinal fluid, but most symptoms in neurocysticercosis are the direct result of the inflammatory process that accompanies cyst degeneration [8, 9].

Clinical presentation

Signs and symptoms are related to the number, size, stage, location of lesions and severity of the host's immune response to the parasites [10, 11]. Seizures are the most common presentation in parenchymal neurocysticercosis (NCC) followed by headache [7]. In endemic regions, new onset of seizures in an otherwise healthy young adult or middle-aged person is highly suggestive of NCC [12]. Features of raised intracranial hypertension are seen in subarachnoid and intraventricular NCC. It may be caused by the parasite itself in the ventricles or cisternal spaces causing obstruction of flow of cerebrospinal fluid, or ependymal reaction or postinflammatory fibrosis [13, 14].

Imaging findings in neurocysticercosis

Parenchymal neurocysticercosis

Parenchymal neurocysticercosis is the most common type of neurocysticercosis. Cerebral hemispheres are commonly affected. As with other haematogenously transmitted diseases, it typically involves the grey-white matter junction due to the smaller calibre of the blood vessels. It is also seen in the basal ganglia, brainstem and cerebellum [15]. Few authors have suggested that parenchymal forms are actually subarachnoid forms located along the deep sulci in cerebral hemispheres and along the perivascular spaces in the basal ganglia [16]. However, the parenchymal forms behave distinctly with respect to clinical features and respond effectively to medications as opposed to the subarachnoid forms [17, 18].

Parenchymal NCC is classified into 5 types by Escobar—non-cystic, vesicular, colloidal vesicular, granular nodular and calcified nodular [19].

Non-cystic

This is asymptomatic and cannot be detected with imaging studies. It can be detected by laboratory tests only.

Vesicular

In this stage, the parasite has a transparent fluid-filled ‘vesicle’ surrounded by a thin transparent membrane which is friable. The invaginated larva is seen within, adopting a curled-up position measuring about 4–5 mm in length.

On imaging, the cyst fluid has similar characteristics to cerebrospinal fluid (CSF) in both CT and MRI. The cyst wall is thin, smooth and measures 2–4 mm. The lesion measures 10–20 mm. Scolex is seen eccentrically within the lesion which is pathognomonic of this stage. It is hyperdense on CT, iso- to hyperintense on T1-weighted and T2-weighted sequences. Scolex is better seen in proton density weighted images. Fluid-attenuated inversion recovery (FLAIR) and DWI also aid in identifying the scolex. As long as the larva is alive, it evades the host immune mechanisms by the development of immune tolerance, partly aided by the intact blood–brain barrier. There is no enhancement on postcontrast sequences. There is no perilesional oedema or calcification (Fig. 1). The parasite remains in this stage for several years until it undergoes degeneration either by itself or due to treatment.

Colloidal vesicular

The parasite begins to show degenerative changes either due to aging or due to antihelminthic treatment. There is loss of immune tolerance of the parasite with the alteration in vitality resulting in osmotic imbalance of the cyst

membrane. The cyst shows hyaline degeneration and early mineralisation. The cyst fluid is extruded out, inciting an inflammatory reaction in the surrounding tissues, and eventually gliosis of the adjacent parenchyma. The cyst wall becomes thicker and the fluid becomes turbid with increased protein content. There is breakdown of blood–brain barrier by the host’s inflammatory response which causes postcontrast enhancement. The breakdown of the blood–brain barrier has been demonstrated by dynamic contrast-enhanced MRI wherein Kep values correlated best with serum MMP-9 expression which represents closely the degree of blood–brain barrier breakdown, and it is highest in the colloidal vesicular stage [20].

On CT, the cyst becomes more hyperattenuating than CSF, with perilesional hypodensity due to oedema. The cyst wall becomes thick and irregular in later stages. The wall shows enhancement on postcontrast sequences. Scolex may or may not be seen. On MRI, the cyst fluid is hyperintense on T1-weighted and T2-weighted images due to its increased protein content. Similar to CT, there is perilesional oedema and postcontrast enhancement (Fig. 2). It is at this stage that it comes under the category of single small enhancing lesions (SSEL) [18]. In the absence of scolex, it is difficult to diagnose confidently as NCC. Hence, it needs comprehensive analysis including proper history, serum and CSF examination for proper diagnosis.

Granular nodular

The cyst continues to shrink in size and walls become thicker and finally it transforms into a granulomatous lesion. Cyst contents are transformed to coarse granules. Perilesional oedema decreases and there is gliosis in the surrounding parenchyma of variable severity. The imaging findings are similar to the colloidal vesicular stage with thicker rim or nodular enhancement with decreasing perilesional oedema.

Calcified nodular

The cyst contents are further transformed to coarse granules with mineralisation with calcium salt. Calcification starts in the cephalic portion and progresses to the vesicular wall, to leave a round, whitish, calcified nodule. CT is better than MRI in depicting the calcified nodule (Fig. 3). Gradient-echo sequence and SWI are useful in identifying the calcified lesion.

Along with SSELs, single small calcific lesions are common findings in patients with seizures. Usually, the calcified lesion of NCC is not destructive to the surrounding brain parenchyma and there is no inflammation, necrosis or oedema. There is only gliosis in the surrounding parenchyma which is presumed to be epileptogenic. The parasite

Fig. 1 Vesicular stage of NCC—axial MR images show well-defined round lesion in right temporal lobe with CSF intensity (a T2WI, b FLAIR, c T1WI) and eccentric scolex within (arrows). No perilesional oedema or enhancement on postcontrast sequences (d)

may not undergo sequential and complete degradation and may retain the antigenic properties. This explains the presence of perilesional oedema and enhancement on postcontrast sequences in some lesions [21]. A variety of appearances are seen in SWI which is explained in detail later.

Subarachnoid neurocysticercosis

Here, the cysts are located within the subarachnoid space in the cortical sulci, Sylvian fissures and basal cisterns. Those in the sulci are small owing to pressure from

adjacent brain parenchyma and they show various stages of evolution similar to the parenchymal type (Fig. 4). Those in the Sylvian fissures and basal cisterns may grow uninterrupted to larger sizes as there is no limiting parenchymal tissue in these sites. This type differs from the parenchymal type by the absence of usual temporal development of degenerative changes and non-visualisation of scolex [22]. There is segmentation of the cysticercus with sprouting of new cysts. There is expansion of these new cysts with degeneration of the scolex. These cysts are non-viable, forming degenerated interconnected cysts

Fig. 2 Colloidal vesicular stage of NCC—axial MR images (**a** T2WI, **b** FLAIR, **c** T1WI) depict two lesions. There is an irregular altered signal intensity lesion hyperintense on T2WI and FLAIR and hypointense on T1WI with peripheral enhancement on postcontrast sequence (**d**) in frontal lobe. There is perilesional oedema. Another lesion is seen in right parietal lobe which is well defined and rounded. The signal intensity is similar to CSF. There is mild perilesional oedema and thin peripheral postcontrast enhancement. The scolex is not seen within the lesion. These two lesions represent the late and early colloidal vesicular stages, respectively

of various sizes, giving a cluster of grapes appearance (Fig. 5). Hence, this type is also known as racemose NCC. It is seen as abnormal growth of cystic membranes without the scolex in the suprasellar, Sylvian, perimesencephalic and other basal cisterns [23]. Racemose NCC is thought to be caused by an aberrant form of *T. solium* or a coenurus cerebralis, which is an intermediary stage of the adult tapeworm, *Taenia multiceps* [24].

CT is not of much use in the diagnosis of racemose NCC. On MRI, cysts are seen within the basal cisterns with CSF intensity causing distortion and mass effect on the adjacent brain structures. Degenerated scolex may also be seen. Cyst walls and scolex are better seen with FLAIR and CISS sequences (Fig. 8a, b). A degenerating cyst can

incite an inflammatory reaction on the surrounding meninges which is seen as contrast enhancement. Meningitis and arachnoiditis may cause vasculitis, resulting in cerebral infarctions and cranial nerve palsies [25].

Intraventricular neurocysticercosis

Intraventricular NCC constitutes about 7–25 % of NCC. The fourth ventricle is the commonest site due to either gravity and CSF flow patterns or direct entry into the choroid plexus. This location is associated with higher risk of brain herniation and basal meningitis. Patients present with features of raised intracranial pressure—headache, vomiting and visual disturbances. The lesions are usually silent

Fig. 3 Calcified stage of NCC—axial sections reveal multiple calcified lesions in both frontal lobes. No perilesional oedema seen

Fig. 4 Subarachnoid NCC—axial MR images (**a** T2WI, **b** FLAIR, **c** post-Gd) show well-defined round lesion with CSF intensity in the extra-axial location along anterior right Sylvian fissure with eccentric scolex within (*arrows*). No enhancement on postcontrast sequence (**c**)

until obstruction occurs, either due to the mass effect of the parasite itself or following ependymitis by granulomatous inflammatory response. The cysts are known to migrate within the ventricular cavity in relation to head movement, known as the ventricular migration sign. Free-floating cysts in ventricles can cause Brun's syndrome, characterised by intermittent foramen obstruction causing episodic recurrent headaches, vertigo, ataxia and drop attacks [26, 27].

The lesions are occult in CT, as they are isoattenuating to CSF. Depending on their location, intraventricular cysts produce dilatation of the ventricles. There is ring enhancement of the wall of the cyst, reflecting an inflammatory reaction. Alternately, ventricular

or periventricular enhancement suggests ependymitis. Before the advent of MRI, intraventricular NCC was diagnosed by CT ventriculography. An intraventricular cyst presents as a regular, rounded filling defect similar to an inverted cup, whereas a cul-de-sac suggests inflammatory ventricular obstruction. MRI is more sensitive and superior to CT. It is challenging to diagnose intraventricular NCC with conventional MR sequences alone as the cyst is isointense to CSF. A T1-weighted sequence and proton density sequence are better than a T2-weighted sequence [27–29] (Fig. 6). Advanced sequences like CISS are reported to be useful; this is discussed in detail later [30].

Fig. 5 Racemose NCC—axial FLAIR MR images **a–c** show multi-loculated cystic CSF intensity lesions in anterior interhemispheric fissure (*thick arrow*), suprasellar cistern (*thin arrow*), ambient cistern (*dotted arrow*) and prepontine cistern with hydrocephalus. The

walls of the lesion are better seen in FLAIR images than in T2WI (not shown). Post Gd MR images (**d–f**) of the same patient shows no demonstrable enhancement. On CISS images (**g–i**), the walls and extent of the lesion are better made out

Spinal neurocysticercosis

Spinal NCC is rare and constitutes about 1.2–5.8 % of all cases of NCC. Mostly it is found in the spinal

subarachnoid space in the intradural extramedullary location. The spinal cord itself is affected in fewer cases. Even rarer is an extradural location [31].

Fig. 6 Intraventricular NCC—axial MR images (**a** T2WI, **b** precontrast and **c** postcontrast 3D-MPRAGE) depict rounded CSF intensity lesion in left lateral ventricle with smooth well-defined wall and subtle enhancement of the wall in the postcontrast image

Subarachnoid NCC in spinal canal

This is the commonest form of spinal NCC. Clinical features of cysts in an intradural extramedullary location vary depending on the mass effect on spinal nerves, spinal cord and other structures, and on the inflammatory reaction. The occurrence has been thought to be due to direct dissemination of the larvae in CSF throughout the craniospinal subarachnoid space. Manifestations are of the cyst itself and of diffuse arachnoiditis. The imaging features of the cyst are similar to that seen in the subarachnoid space in the brain. While the cyst may show rim enhancement, arachnoiditis has homogenous sheet-like enhancement [32]. Diffuse severe arachnoiditis may also cause infective syringomyelia due to subarachnoid adhesions, which in turn causes parenchymal vascular insufficiency and spinal cord atrophy [33].

Intramedullary NCC

This is 6–8 times less frequent than subarachnoid spinal NCC. Clinical features include pain, spasticity, paraparesis, bowel and bladder incontinence and sexual dysfunction. In the spinal cord, the thoracic cord is most commonly affected due to higher blood flow. The cyst undergoes various stages of degradation and the signal intensity and patterns of enhancement are very similar to brain NCC. The scolex is not usually visualised. The scolex is isointense to spinal cord parenchyma on T1-weighted sequences and is isointense to cyst fluid on T2-weighted sequences. Syringomyelia may also occur [32].

Role of advanced MRI sequences

DWI/DTI

Diffusion-weighted imaging (DWI) is the simplest form of diffusion imaging based on measuring the random Brownian movement of water molecules within each voxel of tissue [34]. Diffusion-weighted imaging has a limited role in demonstrating the scolex in selected stages during the evolution of the NCC. An eccentric dot-like or curvilinear area of diffusion restriction is seen within these lesions (Fig. 7). This is presumed to represent the degenerating scolex. This finding is most commonly seen in parenchymal forms, in the colloidal vesicular and granular nodular stages. It is also seen in subarachnoid forms. It is presumed to occur in the ‘late colloidal vesicular’ stage, when the contents of the cyst becomes viscous and appears hyperintense on FLAIR and no longer has the signal intensity matching the CSF. And it disappears in the ‘early’ granular nodular stage when the contents changes from viscous fluid to solid material. The overall role of DWI comes in these stages by helping in the identification of the scolex [35]. Diffusion tensor imaging (DTI) is not used routinely in the diagnosis of NCC. However, studies indicate that mean diffusivity values decreased and fractional anisotropy increased progressively from vesicular to granular nodular stages [36].

SWI

Susceptibility-weighted imaging (SWI) is a gradient-echo sequence that enhances the visibility of susceptibility differences. Original SWI data consist of magnitude and raw source phase images. High-pass filtered images are

Fig. 7 Role of DWI—**a** DWI image and **b** ADC map show small eccentric dot-like area of restriction corresponding to scolex within the lesion in left frontal lobe

produced by using high-pass filtering and phase masking of raw source phase images. Automated processing of the data results in four images: magnitude image, phase image, SWI data (combination of magnitude and phase information) and minimum-intensity projection image. Phase images are sensitive to changes in the magnetic field caused by different components in tissues, such as deoxyhaemoglobin, haematoma, or calcification, and thus can be used for differentiating the susceptibility differences among tissues. In a left-handed system, paramagnetic substances, such as deoxyhaemoglobin, haemosiderin, and ferritin, increase the magnetic field, resulting in a positive phase relative to the surrounding parenchyma, while diamagnetic substances, such as calcium, cause a negative phase shift [37].

SWI in addition to the conventional MRI protocol improves both lesion detection and scolex visibility. It has aided in understanding the pathogenesis behind continued epileptogenesis in the calcified stage [21]. Any lesion in CT with Hounsfield value >80 is considered calcified. What appears to be a calcified lesion in CT actually contains a high concentration of iron and other minerals in addition to calcium [38, 39]. A variety of appearances are demonstrated on MRI depending on the stage of degradation. Sequential degradation and calcification may not occur in all cases as expected. Cysts can bypass the stage of degradation. Incomplete degradation may occur and the cyst may begin to calcify from an early stage. In a CT calcified cyst, scolex or a degraded parasite can be identified in MRI. If the parasite has undergone sequential degradation and calcification, it will contain only diamagnetic substance which will merge with the calcification of cyst wall. Presence of a scolex with paramagnetic substance (positive phase in left-handed system) within a calcified cyst wall represents a

completely calcified vesicular cyst without undergoing degradation. Presence of a scolex with diamagnetic substance (negative phase in left-handed system) within a calcified cyst represents a partly degenerated cyst with preservation of scolex. If the scolex is identified, the parasite did not undergo complete degradation and has its antigenic components, which explains the presence of rim enhancement and perilesional oedema in the calcified nodular stage of NCC [40] (Fig. 8). Significant correlation has also been demonstrated between the Hounsfield values measured in CT and $R2^*$ values measured in MRI. Presence of a scolex within the calcified lesion is considered pathognomonic of NCC and helps us to differentiate it from other granulomatous lesions [38].

CISS

Three-dimensional (3D) constructive interference in steady state (CISS) is a fully refocused steady-state gradient-echo MRI sequence in which both pre- and post-excitation signals are sampled. Thin sections, short Echo time with limited signal loss due to magnetic susceptibility effect and low flip angles with limited T1-weighting available in this sequence allow excellent spatial resolution and signal-to-noise ratio. This sequence is frequently used in MRI to investigate a wide range of pathologies when conventional MRI sequences do not provide the desired anatomic information. CISS has different names according to different manufacturers—Fast Imaging Employing Steady-state Acquisition (FIESTA) by General Electric, true Fast Imaging with Steady-state Precession (FISP) by Siemens, balanced Fast Field Echo (FFE) by Philips, and true Steady-State Free Precession (SSFP) by Toshiba [41]. The CISS

Fig. 8 Role of SWI—there is a lobulated calcified lesion on CT (a) in right caudate nucleus. This lesion is hypointense on T2WI (b) and 3D-MPRAGE (c) sequences. This lesion shows increased susceptibility on SWI (d). There is an eccentric area of positive phase shift suggesting presence of paramagnetic substance like iron within it, corresponding to the scolex. There is subtle postcontrast rim enhancement

(f). Presence of scolex within the calcified lesion in CT is pathognomonic of calcified nodular stage of NCC. Also, presence of non-degraded or partly degraded scolex within the lesion is hypothesised to be the cause of persisting postcontrast enhancement in calcified lesion of NCC

sequence plays an important role in evaluating structures surrounded by CSF. The cyst wall and scolex are well visualised on CISS. The increased sensitivity of this sequence in identifying the cyst wall, cyst fluid and scolex is due to its increased resolution and due to accentuation of the T2 value between the cyst fluid and CSF [42] (Fig. 9).

Magnetic resonance spectroscopy (MRS)

MRS gives an in vivo assessment of chemical metabolites in a tissue. MRS adds specificity and helps in cases of NCC where the conventional MR sequences fail to make a confident diagnosis. ^1H MRS shows elevated levels of lactate, amino acids, pyruvate, acetate, choline and decreased levels of *N*-acetylaspartate (NAA) and creatine. Amino acids such as alanine and threonine are seen. Alanine peaks at 1.3 and

1.4 ppm and inverts at 144 ms. Threonine is demonstrated at the same location as lactate at 1.33 ppm and another peak is seen at 3.6 ppm which inverts at 135 ms. Pyruvate and succinate resonate close to each other at 2.4 ppm with a narrow difference in chemical shift which is difficult to differentiate. However, it has been supported by in vitro analysis of cyst fluid which showed the presence of pyruvate. Pyruvate, the end product of glycolysis may undergo aerobic oxidation to form acetate or anaerobic degradation to lactate in these organisms [43]. Pyruvate is also the predominant metabolite in the racemose type [44]. In cases of single enhancing lesions (SEL), the diagnosis is most often narrowed down to either tuberculoma or NCC. MRS may help to differentiate between these two conditions. Tuberculomas have elevated levels of lipids and choline and decreased levels of NAA and creatine. The choline/creatine

Fig. 9 Role of CISS—intraventricular NCC—there is dilatation of the fourth ventricle. The lesion is not visible in axial T2WI MR image (a). The lesion is subtly visible in the 3D MP-RAGE image

(b). Cystic lesion almost similar to the CSF signal intensity is seen within the fourth ventricle (arrows). The lesion is better made out in CISS image (c)

ratio is greater than 1 in tuberculoma. But MRS findings of NCC become indistinguishable from tuberculoma in the colloidal vesicular stage [45]. MRS done following treatment demonstrates decreased levels of lactate, alanine and pyruvate: the peaks decrease and later disappear.

Perfusion MR imaging

Perfusion MR imaging gives a non-invasive assessment of haemodynamic parameters and demonstrates variations in the microenvironment. Techniques of perfusion MR imaging include dynamic susceptibility contrast-enhanced imaging (which is either T2 or T2*-weighted), arterial spin labelling and T1-weighted dynamic contrast-enhanced MR imaging (DCE-MRI). Typically hyperperfusion is absent in NCC which helps us to differentiate it from tumours [46–48]. Ktrans, Kep, Ve and Vp values are found to be significantly higher in lesions with postcontrast enhancement and perilesional oedema. These values are also higher in lesions with postcontrast enhancement and with no perilesional oedema but to a lesser extent than those lesions with both these findings. This indicates opening of the blood–brain barrier (BBB) in these lesions. These values are nil in lesions with no postcontrast enhancement and perilesional oedema, indicating an intact BBB. Matrix metalloproteinases-9 (MMP-9) plays a key role in the disruption of BBB. Kep values are found to correlate best with serum MMP-9 and are highest in the colloidal vesicular stage and lowest in the calcified stage. Perilesional inflammation is present in symptomatic patients at the calcified stage of NCC which can be quantified using DCE-MRI, where Kep values are higher in symptomatic patients than in asymptomatic patients [20, 49].

Complications

Chronic meningitis occurs due to long-standing inflammation resulting in the abnormal thickening of the leptomeninges along the base of the skull, ventral aspect of brainstem entrapping the optic chiasm and cranial nerves, leading to various cranial nerve palsies. The patient does not have typical signs of meningeal irritation. Subarachnoid forms cause severe *arachnoiditis* with development of adhesions resulting in hydrocephalus. *Hydrocephalus* may also occur in the intraventricular type, either as a result of the presence of the parasite or due to ependymitis following death of the larva. Occasionally, ependymitis causes ventricular entrapment resulting in double-compartment syndrome [17].

Dementia is a rare complication and sometimes the presenting feature in racemose NCC. The mechanisms proposed for dementia are raised intracranial pressure, location of the parasite and the host's immune response. The prognosis is better with cognitive decline caused by raised intracranial pressure than by other causes [50].

Isolated spinal NCC has also been reported to cause *lymphocytic meningitis* in the absence of an intracranial lesion. Hence, in a suspected case, spinal imaging is needed to rule out NCC [51].

Cerebrovascular disease is a common but under-recognised complication of NCC. About 3 % of patients with NCC develop a cerebral infarct while 2.5 % of patients with cerebral infarcts have NCC. Infarcts caused by small- and large-vessel occlusions, progressive midbrain syndrome and transient ischaemic attacks are the commonly reported vascular complications.

Lacunar infarction occurs due to occlusion of lenticulostriate branches of anterior or middle cerebral arteries resulting from occlusive endarteritis secondary to arachnoiditis. This occurs due to intense inflammatory reaction resulting in abnormal thickening of the leptomeninges in the base of skull with consequent entrapment and occlusion of blood vessels in the circle of Willis. Cerebral angiography may be completely normal (as the affected vessels are too small to visualise) or may demonstrate segmental narrowing of major arteries due to severe vasculitis.

Large-vessel disease due to cysticercotic vasculitis causing infarctions in the territory of major intracranial arteries has also been reported. It is caused by occlusive endarteritis and disruption of endothelium secondary to invasion of vessel wall by inflammatory reaction induced by cysticerci. There is thickening of the adventitia, fibrosis of the media and hyperplasia of the endothelium in the involved vessels.

Progressive midbrain syndrome is a serious complication of NCC. It is usually seen in patients following shunting for hydrocephalus secondary to diffuse cysticercotic arachnoiditis. The patient undergoes neurological deterioration characterised by somnolence, urinary incontinence, dilated pupils and impaired vertical gaze in a few months following shunting. Imaging reveals multiple areas of ischaemia in the midbrain and thalamus due to occlusion of paramedian thalamopeduncular branches of the mesencephalic artery. These branches are particularly liable to get occluded in the interpeduncular and prepontine cisterns by diffuse arachnoiditis. The prognosis is usually bad.

It has been suggested in the literature that NCC produces transient neurological dysfunction resembling *transient ischaemic attacks*. The pathogenesis is not clearly known and is not uniform. It may be of vascular origin or may be related to parenchymal lesions. Positional cerebral ischaemia has also been reported due to a large cysticercus located near the internal carotid artery, causing its occlusion [52].

Cysticercotic encephalitis is a severe and often fatal neurological disorder. It is seen in young females. It is caused by severe inflammatory response rather than by mere mass effect of multiple cysts. There is diffuse brain oedema and invariably all patients develop subacute severe intracranial hypertension and visual compromise [53] (Fig. 10).

Treatment

The principles of management of neurocysticercosis used for the parenchymal type differ from the other types. Antihelminthic agents such as albendazole and praziquantel are the primary treatment strategy in the parenchymal type. Patients with subarachnoid NCC require several months

Fig. 10 Cysticercotic encephalitis—axial T2WI MR images show multiple lesions of NCC in vesicular and colloidal vesicular stages diffusely scattered in both cerebral hemispheres, corpus callosum, basal ganglia and thalami with diffuse oedema

of treatment with both antihelminthic agents and steroids to avoid complications such as hydrocephalus and vascular events during treatment. Radiological evolution and serum and CSF antigen levels determine the success of treatment in the subarachnoid type. Surgery in the form of minimally invasive endoscopic removal of cysts is the mainstay of treatment in intraventricular type.

Ocular cysticercosis

Ocular cysticercosis is endemic in Sub-Saharan Africa, India, East Asia, Mexico and Latin America. Reported incidence varies from 10 to 30 % in endemic countries. Ocular cysticercosis may be extraocular (in the subconjunctival or orbital tissues) or intraocular (in the vitreous, subretinal space, or anterior chamber). The most common location of ocular cysticercosis is the subretinal region (35 %), followed by the vitreous (22 %), conjunctiva (22 %), and anterior segment (5 %) while involvement of other regions (e.g., extraocular muscles, optic nerve) are relatively less common. Among the extraocular muscles, no one particular muscle has increased incidence. Either eye can be involved. Bilateral involvement is rare. Multiple cysts may occur in the same eye. The most common location of ocular cysticercosis varies from one region to another [54].

Fig. 11 Orbital cysticercosis—coronal (a) and sagittal (b) T1 post Gd images show lesion in left lateral rectus with enhancing wall and surrounding tissues. Multiple nodular and ring enhancing lesions are seen in both cerebral hemispheres

Pathogenesis and clinical features

Choroidal vasculature is the site of entry, from where it reaches the vitreous through the subretinal space, resulting in a chorioretinal scar. Ciliary and retinal vessels are other sites of entry. The cyst appears as a well-defined translucent mass with a dense white spot at the region of the scolex. When the larva is alive, the cyst is mobile and induces inflammation. When the larva dies, motility is lost and the cyst becomes less translucent with surrounding inflammatory membrane due to release of cyst fluid. It causes vitritis, retinal haemorrhages, vitreoretinopathy, retinal detachment, oedema of the disc and finally phthisis bulbi. Cysticercosis in the anterior chamber causes iridocyclitis, secondary glaucoma and iris atrophy. Strabismus, painful proptosis, diplopia and recurrent redness are seen in extraocular cysticercosis. Compression of the optic nerve causes decreased vision and disc oedema [55].

Imaging findings

B-scan demonstrates a well-defined cystic lesion with an echogenic scolex within it. Associated findings such as retinal detachment and vitreous haemorrhage can also be seen [56]. CT scans reveal a hypodense lesion with central hyperdensity suggestive of a scolex. Peripheral rim enhancement may be observed. Adjacent soft tissue inflammation may also be seen. The scolex may not be visible in some cases, such as following the death of the larva or rupture of the cyst following trauma or treatment. MRI offers excellent soft tissue resolution and aids in the accurate diagnosis of ocular cysticercosis. It shows a well-defined lesion of fluid intensity with eccentric scolex within. Peripheral rim enhancement is seen in some cases (Fig. 11). Associated complications are also demonstrated.

If the conventional MR sequences fail to demonstrate the lesion, then a 3D CISS sequence will help in making the diagnosis [57].

Muscular cysticercosis

In the muscles, the oncospheres encyst and form cysticercus cellulosae. These remain dormant but viable, even up to 10 years in humans, evading immune recognition as long as the larva is alive. When the larva dies, it incites a granulomatous inflammatory reaction and produces symptoms and signs.

Clinical features

Four clinically distinct manifestations are seen in the muscular form. They are myalgic or myopathic type, nodular or mass-like type, abscess-like and pseudo-hypertrophic type. When the larva dies, it incites intense inflammatory reaction and causes pain in myalgic type. In the other types, the cyst degenerates and leaks the cyst fluid intermittently into the surrounding tissues causing a chronic inflammatory reaction [58].

Imaging findings

Myriad sonographic appearances have been described in the literature. (1) A soft-tissue mass centred on a well-defined round or elliptical cystic lesion with an eccentric echogenic structure suggestive of scolex within it. The scolex is sometimes calcified. This appearance represents the cysticercus cyst with surrounding inflammatory reaction due to death of the larva. (2) An irregular cyst with or without minimal fluid surrounding it is seen. Eccentric scolex is not seen within it. This appearance is due to

collapse of the cyst with escape of fluid and scolex outside. This occurs following treatment or trauma (Fig. 12). (3) A large irregular collection is seen within the muscle with cysticercus cyst with eccentric scolex. This occurs due to chronic intermittent leakage of fluid causing inflammatory exudative collection within the muscle. This can be distinguished from other collections by the identification of cysticercus cyst with scolex. (4) Multiple small millet

Fig. 12 Muscular cysticercosis—sonogram of the neck shows roughly oval cystic lesion in left strap muscle. The scolex is not seen within

seed-shaped calcifications are seen in the muscles which can be seen on plain radiography, characteristic of calcified cysticercosis [58].

Though CT is useful in neurocysticercosis, it is not of much use in muscular cysticercosis. MRI, on the other hand, is very useful in diagnosing the presence of infection, stage of infection and the plane of lodgement of the cysticercus. The appearance on MRI depends on the growth stage of the parasite and the host's immune response. Presence of a round or oval lesion of fluid intensity with a well-defined wall is characteristically seen within the muscle. The scolex may or may not be seen within it. The cyst fluid is hypointense on T1-weighted sequences and hyperintense on T2-weighted sequences (Fig. 13). Peripheral rim

Fig. 14 Muscular cysticercosis—axial STIR image shows a well-defined cystic lesion in left external oblique muscle with intense inflammation in the surrounding tissues

Fig. 13 Muscular cysticercosis—coronal T2WI MR images show multiple well-defined cystic lesions with eccentric scolex in both masseter muscles, tongue and left mylohyoid muscle. Multiple

lesions in vesicular and colloidal vesicular stages of NCC are seen diffusely scattered in both cerebral hemispheres and basal ganglia

Fig. 15 Disseminated cysticercosis—lateral and anteroposterior view of right (a) thigh shows multiple rice grain or cigar-shaped calcifications in the soft tissue of thigh and pelvic regions. b Frontal radiograph of chest shows similar calcifications in the soft tissue of the chest and neck

enhancement is seen in some cases. Surrounding tissues show inflammatory oedema [59] (Fig. 14).

Treatment options include antihelminthic agents like albendazole and praziquantel in conjunction with steroids. Simple excision may be required in some cases. Surgical excision is losing popularity with the introduction of effective antiparasitic therapies.

Subcutaneous and disseminated cysticercosis

Subcutaneous cysticercosis is common in Asia and Africa. Outside the central nervous system, cysticercosis causes no major symptoms. Subcutaneous cysticercosis presents as small, movable, painless nodules that are most commonly seen in the arms or chest. After a period of few months or sometimes years, these nodules become swollen, tender and inflamed, and then they gradually disappear. High resolution ultrasound may help in the diagnosis. The ultrasound appearances are similar to muscular cysticercosis. Biopsy or fine-needle cytology of a subcutaneous nodule helps to confirm the diagnosis of cysticercosis infection [9].

Disseminated cysticercosis is an uncommon form mostly reported in endemic countries. There is widespread dissemination of the cysticerci in virtually any part of the body. Signs and symptoms depend upon the location and number of the cysticerci and host's immune response. Commonly, patients present with enlargement of the muscles, subcutaneous and lingual nodules. A 'Herculean' appearance or muscular pseudohypertrophy is caused by heavy infection of the muscles. Calcifications may be seen in solid organs, mesentery and retroperitoneum in addition to muscles and subcutaneous tissues.

Plain radiographs show typical 'rice-grain' shaped or cigar shaped calcification in the soft tissues (Fig. 15). CT demonstrates the calcification in various sites. MRI is, however, more sensitive than CT in imaging non-calcified lesions, aiding better identification of the cysts and scolex [60].

Conclusion

Cysticercosis is one of the common parasitic diseases affecting predominantly the brain, eye and muscles. It is caused by the larval form of *Taenia solium*. It is transmitted to humans owing to poor hygiene and improper sanitation. Though endemic in several developing countries, owing to globalisation, several cases have been diagnosed in developed countries as well. Manifestations of the disease vary depending upon the organ affected. Though muscular cysticercosis remains asymptomatic for a long period of time, neurocysticercosis presents with a multitude of signs and symptoms. MRI remains the cornerstone in the diagnosis of cysticercosis in various tissues. Advanced MRI sequences aid in better visualisation of the cysts. A thorough knowledge of the pathogenesis and imaging features of cysticercosis in various tissues will help us in prompt and early diagnosis and appropriate use of effective treatment.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Informed consent No animal experimentation has been conducted. Images were taken from our database in PACS. Patients' identity has not been revealed in this article.

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