

Original Article

Neurocysticercosis in Kuwait: Computerized Tomography and Magnetic Resonance Imaging Findings

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ABSTRACT

Objective: To study the computerized tomographic (CT) and Magnetic Resonance Imaging (MRI) findings in seven Kuwaiti patients with neurocysticercosis.

Material & Methods: The study population composed of seven Kuwaiti patients aged 3-44 years presented with seizures. The CT and MR imaging findings suggested neurocysticercosis. Serology (n = 2), histology (n = 2) and presence of scolex on neuroimaging studies (n = 3), in addition, close contact with a carrier of neurocysticercosis further confirmed the diagnosis.

Results: CT and MRI depicted all lesions successfully. The lesions involved the brain parenchyma. There was

no involvement of the cerebral ventricles, cisterns or brain stem. All patients, except one, had only one lesion and all were in the colloid vesicular stage. No calcification was noted.

Conclusion: Neurocysticercosis is increasingly diagnosed in Kuwait. CT and MRI play an important role in the diagnosis of neurocysticercosis. Ring enhancing lesion with adjacent white matter edema was the dominant neuroimaging finding in our study. Neurocysticercosis should be an important differential diagnosis among Kuwaiti patients who have CT or MRI brain study showing ring-enhancing lesions.

KEY WORDS: brain, central nervous system, cysticercosis, magnetic resonance imaging, parasite

INTRODUCTION

Cysticercosis is a parasitic disease caused by infection with the larval stage of the pork tape worm *Taenia solium*. In humans, the parasite commonly infects the human CNS producing a clinical disorder known as neurocysticercosis^[1]. It is the most common parasitic disease of the human nervous system affecting thousands of individuals in developing countries and in industrialized areas with a high index in immigrants from endemic areas^[1].

Neurocysticercosis has a pleomorphic clinical picture. Seizure is by far the most common clinical manifestation; less common manifestations include headache, nausea, vomiting and altered mental status^[2].

The correct diagnosis of neurocysticercosis is based on imaging findings and laboratory tests^[2]. Neuroimaging studies are the main methods for diagnosing neurocysticercosis^[3]. Computerized tomography (CT) is the best method for detecting calcification associated with prior infection. However, magnetic resonance imaging (MRI) is more sensitive than CT for depicting cysts in the brain parenchyma, for identifying inflammation

associated with the cysts and for identifying cysts in the ventricles and basilar cisterns^[4,5,6,7].

In Islamic states, the consumption of pork is prohibited and cysticercosis is expected to be absent. However, the employment of individuals from endemic areas can be a source of infection.

A review of the literature suggests that there had been no previous study that had discussed neurocysticercosis among Kuwaitis. In this study, we review the CT and MRI features of neurocysticercosis in seven Kuwaiti patients.

PATIENTS AND METHODS

We studied prospectively, seven patients aged 3-44 years, who presented with seizures and suspected of having neuro-cysticercosis on neuroimaging studies. CT and MRI studies of the brain were obtained for all patients included in the study. Serum enzyme-linked immunosorbent assay (ELISA) antibodies was used in all patients. However, positive results were not considered for confirming the diagnosis of neurocysticercosis in our study, because recent studies have demonstrated a large number of false-positive and false-negative results^[8]. Two patients

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underwent brain surgery and tissue diagnosis of neurocysticercosis was obtained. In three patients, neuroimaging studies showed a small cyst containing an eccentric nodule representing the scolex of the worm and was considered a definitive confirmation of neurocysticercosis¹⁸. In two patients diagnosis of neurocysticercosis was based on the combined results of the neuroimaging findings and a positive serum enzyme-linked immunoelectrotransfer blot [EITB] test. In non-surgically proven cases, three patients were treated with antiepileptic and anticysticercal drugs. Two patients were treated symptomatically with only antiepileptic drugs.

MR images were obtained with 1.5 Tesla MRI system (GE Medical System). T1-weighted and T2-weighted images in the axial, sagittal and coronal planes were obtained. After intravenous injection of (0.1-0.2 ml/kg) of gadopentate dimeglumine (GD-DTPA), T1-weighted axial, sagittal and coronal images were obtained. CT images were obtained with Pro Speed (GE Medical System). Axial images before and after intravenous injection of nonionic contrast were obtained.

RESULTS

As shown in table 1, half of the study group were children under the age of 10 years. Six patients (85%) had only one brain lesion. Six lesions (75%) were noted in parietal lobe. CT studies showed a small cyst that measured 5-15 mm. The cyst showed a low-density cavity and a smooth wall that enhanced with intravenous contrast (Fig. 1). There was substantial white matter edema adjacent to the

cyst in all patients.

T1-weighted MR images showed a small cyst with a low signal intensity cavity and a smooth ring enhancing wall (Fig. 2 A & B). On T2 weighted images the cyst showed high signal intensity cavity, low signal intensity wall and adjacent white matter edema (Fig. 2 C). There was no involvement of the cerebral cisterns or cerebral ventricles. CT and MR images were equally successful in depicting the cysts. MRI, however showed better delineation of brain edema.

DISCUSSION

Cysticercosis is an infection caused by the encysted larval stage of tapeworm *Tenia solium*. The parasite has a complex life cycle in which pigs



Fig. 1: Post-contrast axial CT scan showing a thin, regular ring enhancing cyst in the right parietal lobe (long arrow). Reduced attenuation of the adjacent white matter represents edema (short arrow).

Table 1

CT & MR imaging features and immunological tests of the study group suspected of having neurocysticercosis

Patient			CT & MRI characteristics of brain lesions							ELISA	EITB	Surgical
n	Age (years)	Sex	CT MRI	n	Size	Scolex	Calcification	Location	Edema	Serum	Serum	Biopsy
1	3	m	CT	1	15 mm	Not detected	No	Left parietal lobe	Detected	Negative	Positive	-
			MRI	1	15 mm	Not detected	No					
2	5	f	CT	1	10 mm	Detected	No	Right Parietal lobe	Detected	Positive	-	-
			MRI	1	10mm	Detected	No					
3	7	f	CT	2	5-10mm	Detected	No	Right frontal and Left parietal lobe	Detected	Negative	-	-
			MRI	2	5-10 mm	Detected	No					
4	36	f	CT	1	10 mm	Not detected	No	Left temporal lobe	Detected	Negative	-	Positive
			MRI	1	10 mm	Not detected	No					
5	44	f	CT	1	15 mm	Not detected	No	Right Parietal lobe	Detected	Negative	-	Positive
			MRI	1	15 mm	Not detected	No					
6	36	m	CT	1	6 mm	Not detected	No	Left parietal lobe	Detected	Positive	-	-
			MRI	1	6 mm	Detected	No					
7	5	f	CT	1	10 mm	Not detected	No	Left parietal lobe	Detected	Negative	Positive	-
			MRI	1	10 mm	Not detected	No					



Fig. 2a: Axial T1-weighted MR image showing a small cyst of low (but higher than CSF) intensity in the right parietal lobe (long arrow) with surrounding edema of low signal intensity (short arrow).

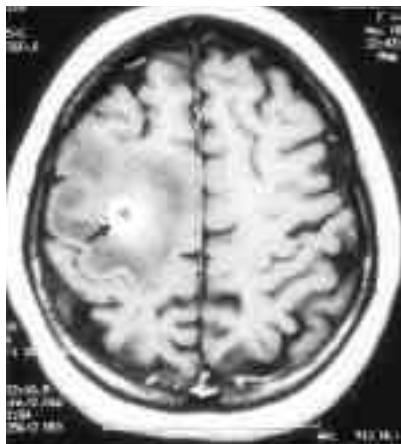


Fig. 2b: Post-contrast axial T1-weighted MR image showing a ring-enhancing cyst in the right parietal lobe.



Fig. 2c: Corresponding axial T2-weighted MR image shows a hypo-intense capsule (long arrow) with anterior thickening, representing a scolex (short arrow). The center of the lesion is hyper-intense. The adjacent brain edema is clearly depicted as a hyper-intense area (thick arrow).

are the usual intermediate host and humans serve as either definitive or intermediate host. When human ingests undercooked pork meat infected with cysticerci, the larva invaginates into the intestine, its scolex attaches to the intestinal mucosa and it begins forming segments (proglottides) that arise from the caudal end of the scolex.

Humans act as intermediate host for *Taenia solium*, by accidental ingestion of food contaminated with *Taenia solium* eggs and oro-fecal contamination in tapeworm carriers. Once ingested, the embryos are liberated by the action of gastric acid and intestinal fluid, and they actively cross the bowel wall, enter blood stream and are carried to the brain, muscle, skin and eyes. In humans the parasite commonly infects the human CNS where it produces a clinical disorder known as neurocysticercosis^[2,9].

Clinical manifestations of neurocysticercosis are varied due to individual differences in the number, size of lesions and the severity of the host immune response. Epilepsy is the most common form of presentation. It occurs in 50%-80% of patients with parenchymal brain cysts or calcifications^[10]. Other neurological manifestations include pyramidal tract signs, sensory deficits, involuntary movements and cerebellar ataxia.

The diagnosis of neurocysticercosis has become easier in the last 20 years with the advent of CT and MRI and the development of more sensitive and specific immunologic assays^[2]. A wide range of serological tests have been used in diagnostic studies of neurocysticercosis, however, most of these are of limited value because of poor sensitivity and specificity. The current assay of choice is EITB which has documented sensitivity and specificity of 98% and 100% respectively for patients who have two or more cystic lesions. The major weakness of this test is

the frequent false negative results in patients with a single lesion^[8].

CT has proved to be valuable in detecting lesions typical of neurocysticercosis. However, MRI appears to be superior to CT in almost all patients, particularly in patients with lesions in the base of the brain, brain stem cysts, intraventricular cysts and spinal lesions, except those with only small calcification^[11].

Cysticerci in the CNS may be located in the brain parenchyma, subarachnoid space, ventricles or spinal canal. CT and MR imaging findings in parenchymal neurocysticercosis depend on the stage of evolution of cysticerci. Four stages of cyst formation have been described and imaging findings in each stage reflect underlying changes in the disease process and host response^[12,13].

In the initial stage, the embryo is non-cystic and is often invisible on CT & MR imaging. During the development of the embryo into larva, focal non-enhancing area of edema may be seen that progresses to small homogeneously enhancing lesions^[12]. In the vesicular stage, the larva develops into a cyst containing the scolex. Mature cysts, measures 5-20 mm, has a thin smooth wall and a 2-4 mm scolex identified within the cyst^[2]. These cysts are common at the grey-white matter junction and readily apparent on CT and MR images. They show a cystic structure with a thin walled capsule that rarely enhances. Internally the cyst fluid parallels the cerebrospinal fluid intensity and the scolex appear as a mural nodule, which is iso-intense with brain parenchyma^[4].

In the colloid vesicular stage, the larva begins to degenerate, the scolex disintegrates, and the host inflammatory response causes a fibrous capsule to form with surrounding parenchymal edema. CT shows ring enhancing cystic lesion with

hyperdense cystic fluid and surrounding edema. T1 weighted MR images show a cyst hyper-intense to CSF because of proteinaceous fluid within the cyst cavity. T2 weighted MR images show a hyper-intense cyst surrounded by hyper-intense parenchymal edema^[13]. The cyst capsule shows low signal intensity which may reflect a paramagnetic substance such as free radicals generated during active phagocytosis by the abundant macrophages in the capsule wall^[14].

In the granular nodular stage, the cyst retracts, and forms a granulomatous nodule that will latter calcify. CT depicts enhancing nodule with mild surrounding edema. MR images shows a thick enhancing ring or nodule with or without surrounding edema. In the nodular calcified stage, the granulomatous lesion shrinks and calcifies. CT shows calcified nodule and MR images show a hypo-intense nodule in all pulse sequences^[12].

Cysticerci may occasionally involve the subarachnoid space and cause hydrocephalus due to the space-occupying lesion effect or due to basal arachnoiditis^[15]. The CT and MR imaging findings include hydrocephalus and abnormal enhancement of the leptomeninges. The cysts in the basal cistern may undergo disproportionate growth, with extension processes attached to each other, which tend to group in clusters resembling a bunch of grape (racemose cysticercosis). In these cases the scolex is frequently absent^[4].

Cysticercosis may rarely involve the spinal cord. It may involve the subarachnoid spaces and manifests as an intradural extramedullary cyst or arachnoiditis. It may involve the spinal cord and manifests as ring enhancing lesions expanding the spinal cord^[16,17].

In our study, children less than 10 years of age constituted 56% of the study group. Baby-sitter was the source of infection among the children. Housekeeper was the source of infection among older patients. These workers came from endemic areas like India and Indonesia, and they were infected with cysticercosis. The Kuwaiti patients were infected either by ingesting ova in contaminated food or after direct contact and transfer of ova from hand to mouth.

Parenchymal neurocysticercosis are common at the grey-white matter junction but are also seen in the basal ganglia, cerebellum and brain stem^[12]. In our study, all lesions were seen at the grey-white matter junction and there was no involvement of the cerebellum basal ganglia and brain stem. The parietal lobe was involved in all patients except one. A single lesion was seen in 85% of the study group. In one patient, CT and MR images showed two cysts at the same stage of evolution.

The dominant CT and MRI finding was that of a small, spherical, ring-enhancing cystic lesion with adjacent white matter edema, which was considered as the vesicular-colloid stage when the cyst started to involute. Nearly all patients with symptomatic parenchymal disease have evidence of inflammation (edema, enhancement) on neuroimaging studies in contrast to asymptomatic patients who are imaged for conditions other than neurocysticercosis, where there is no edema or enhancement^[19]. In our study, all patients presented with seizures and CT and MR images showed also enhancement and edema.

CT and MR imaging were equally successful in depicting all lesions in our study. This can be explained by the lack of cerebral calcification that may not be detected by MR imaging and lack of involvement of cerebral cisterns, ventricles and brain stem that may not be detected by CT. However, MR imaging was superior in delineating brain edema. None of our patients had symptoms related to spinal cord disease, therefore, MR imaging of the spinal cord was not done.

The diagnostic consideration of a small, spherical ring-enhancing lesion with a regular margin located at the corticomedullary junction and displaying substantial white matter edema include the following: (1) metastasis which characteristically lodge at corticomedullary junction and can enhance in a ring like pattern, (2) parasitic infection such as neurocysticercosis, echinococcus, paragonimiasis, especially in endemic areas, (3) cerebral abscess caused by bacterial, fungal, or granulomatous agents, (4) primary brain tumor which more likely to be heterogenous with irregular borders, (5) less commonly, subacute infarct, and resolving haematoma may enhance in a ring pattern. The clinical history, however would suggest the diagnosis.

CONCLUSION

Neuroimaging studies that include CT and MRI, in addition to serological test, have resulted in the increasing recognition of the high prevalence of neurocysticercosis worldwide. Cysticercosis is expected to be almost an unknown condition in Islamic states, like Kuwait. However, employment of people from endemic areas can be a source of infection. The dominant CT and MRI finding among the Kuwaiti patients was a ring enhancing cystic lesion in the brain parenchyma with substantial adjacent white matter edema. We think that neurocysticercosis is underestimated in Kuwait and it should be an important differential diagnosis of a ring enhancing lesion in the brain among Kuwaiti patients, both adults and children.

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