

Neurocysticercosis of 4th Ventricle – A Rare Site Encountered with Rare Presentation

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Abstract

Neurocysticercosis (NCC) is the commonest parasitosis of the Central Nervous System (CNS), endemic in developing countries and also seen in developed nations. The parasite usually invades the brain parenchyma and the patients present with a variety of neurological manifestations, mostly seizures and neural deficits. But we present a case of a 16 years old boy who presented with occipital headache, vomiting and difficulty in walking. CT scan and MRI showed a cystic mass lesion in the 4th ventricle causing obstructive hydrocephalus. Histopathological examination confirmed it to be neurocysticercosis. We present this case due to its rare site and unusual presentation. This case highlights the importance of careful interpretation of head CT scans in context of presentations of headache and other neurological deficit in a susceptible population.

Keywords: Endemic; Hydrocephalus; Neurocysticercosis

Introduction

Cysticercosis is the commonest parasitic infection of the central nervous system worldwide. It is caused by the ingestion of the eggs or larva of the tapeworm taenia solium, found in fecally contaminated water and undercooked pork, affecting the gut initially and spreading haematogenously [1]. Patients often have a long asymptomatic period and can present with a variety of neurological manifestation like focal neurological deficits, migraine, visual disturbances and seizures [2].

Neurocysticercosis is a serious public health problem in several developing countries, including India [3]. Most infected individuals are aged between 20-50 years and debilitation from cysticercosis causes considerable human suffering. Humans are definite hosts and pigs are the intermediate hosts for Taenia solium [4]. In cysticercosis, humans become the intermediate hosts by ingestion of the eggs of T.solium from contaminated food and water. After entering through the intestinal wall, the embryo invades the blood stream and can lodge in various organs such as CNS, eyes, skeletal muscle and subcutaneous tissue [5]. In the brain it usually affects the brain parenchyma predominantly; rarely ventricles or meninges are affected where it forms a cyst [6]. The depth of the parasitic infestation causes a noticeable release of toxic yield, leading to intense inflammatory reactions and tissue damage. Initial medical treatment with cysticercosis is with antihelminthic drugs like albendazole or praziquantel. However, early surgical removal of the parasite is the treatment of choice especially in neurocysticercosis before it causes parenchymal damage or ventricular obstruction as in our case.

Case Report

A 16 year old male presented with occipital headache for 1 year duration that was aggravated since last 1 month. He also complained of irregular bouts of vomiting in last 3 months and swaying to both sides on walking for 1 month. There was no history of seizures, fever, paresis, vision disturbances or swallowing difficulties. He was not a known case of tuberculosis or had any contact history with it. Clinical examinations revealed no abnormality, vision, fundus and corneal reflexes were normal. There was no cranial nerve deficit, motor and sensory functions were normal, plantar B/L upgoing, wide based gait, tandem walking not possible, no other cerebellar signs, bowel and bladder were normal, skull and spine-normal. CT scan showed midline rounded intra 4th ventricular cystic mass lesion not enhancing with contrast with a small isodense nodule posteriorly within the lesion with gross hydrocephalus (Figure 1).

MRI of 4th ventricle revealed fairly marginated rounded cystic non-enhancing mass lesion of size 60mm in diameter, T1W hypointense, T2W hyperintense with a small enhancing isodense nodule in its posterior part causing obstructive hydrocephalus.

Operative findings were –Midline incision was given in prone position from external occipital protuberance to Cz. Midline posterior fossa craniectomy was done. Midline intercerebellar approach was taken. A cystic mass with yellow colored walled bulged out with 2 daughter cysts. The mass was removed and hemostasis achieved, dura was closed. Post operative CT scan after 1 month showed no residual lesion. The gross specimen received was whitish flap like and polypoid cystic structures measuring 3×2×.5 cm (Figure 2). Histopathological

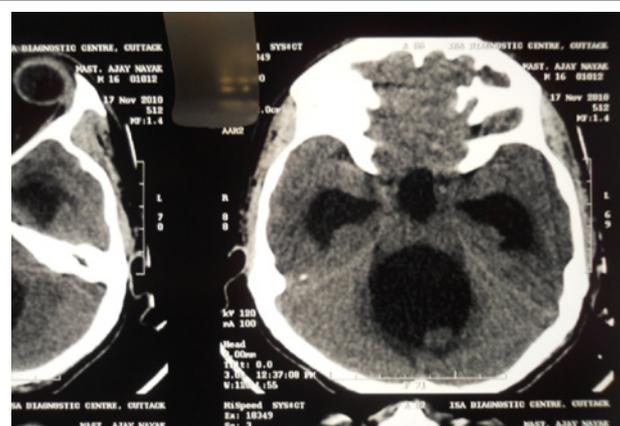


Figure 1: CT scan showing 4th ventricular cystic swelling with a small isodense solid nodule.

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Figure 2: Gross picture of translucent cystic structures in cysticercosis.

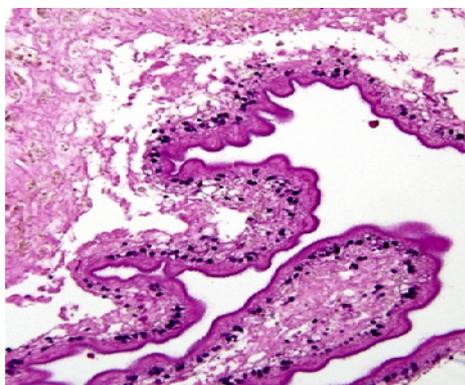


Figure 3: The outer cuticular with middle & inner layer in cysticercosis. H&E stain $\times 400$.

examination showed (Figure 2) the cyst wall mainly consisting of an outer corrugated cuticular layer with the hair like protrusions (microtrichia), a thin middle cellular layer and a thick inner layer containing a loosely packed network of small canaliculi (Figure 3). Few hooklets of the larva were also seen. So, a diagnosis of cysticercosis was given.

Discussion

Human cysticercosis is ubiquitous worldwide [7], including north-eastern India where consumption of improperly cooked pork, contaminated salad and inadequate hygiene are all possible causes of taeniasis and thereby can cause autoinfection. Food habits, poor hygiene, autoinfection or travelling patterns may be responsible for their distribution. This patient was from low economic status and poor hygiene must have played a vital role in infection. Clinical presentations of neurocysticercosis are variable. If the larva settles in the subarachnoid space or ventricles, the cysticercus becomes multilocular and spreads over a wide area. These racemose cyst often causes hydrocephalus and cranial nerve palsies. Small cerebral and meningeal vessels adjacent to the cysts often display peculiar intimal thickening and sclerosis, cysticercus endarteritis. In our case the parasite had lodged in the 4th ventricle which is a rare site and caused hydrocephalus which also is not much frequent. Headache and vomiting along with disturbances in the gait must have been due to hydrocephalus.

In the developing countries, seizures may be caused due to cerebral edema. Often symptoms like meningitis, hemorrhage or ischemic

stroke may occur, although these symptoms were not a feature in our case. The differential diagnosis of a cystic cerebral lesion on CT or MRI includes abscess, tubercle, metastasis or glioblastoma. Parasitic CNS infections should always be considered. The clinical picture is variable depending upon the localization of the cyst. According to post mortem studies, 80% of neurocysticercal infections remain asymptomatic [8]. Vaidya et al. had reported a rare case of disseminated neurocysticercosis which was asymptomatic who presented with dysphagia and achalasia cardia [9]. Diagnosis of neurocysticercosis is always based upon clinical presentation, neuroimaging abnormalities and histopathological examination. In our case histopathological examination helped in confirming the diagnosis. Surgical intervention is necessary in the setting of intracranial hypertension caused by hydrocephalus or giant cysts. In the present case, the patient was completely cured after surgical intervention along with antihelminthic drugs. Even though surgical evacuation of intraventricular cysticercosis is usually done recently an endoscopic approach is practised with fiberoptic endoscope [10].

NCC poses a complex diagnostic and treatment dilemma because of its varied presentation. Factors determining treatment include whether symptoms are present, the location of the cysts, and whether there is a host immune response. This case highlights that, in developing countries, the diagnosis of neurocysticercosis should be considered in patients presenting with neurological deficits and space occupying lesions in central nervous system even in the 4th ventricle.

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