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CASE REPORT

Neurocysticercosis

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ABSTRACT

Neurocysticercosis (NCC), the most common parasitic brain disease worldwide, is endemic in countries with poor sanitation, and is increasingly being reported in developed countries due to globalization and immigration. One of the most intriguing aspects of NCC is that presumably a high percentage of the individuals harboring NCC remains asymptomatic. It has also been stated that almost any neurological symptomatology may be found, ranging from mild headache or treatable acute seizures to very severe neurological manifestations, such as intracranial hypertension (ICH), dementia or even death.

We are reporting a case of NCC who presented with various clinical manifestations of brain parenchymal lesions, which got cured with treatment. It was confirmed with repeat computed tomography (CT).

Keywords: Extraparenchymal neurocysticercosis, Leptomeninges, Neurocysticercosis.

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INTRODUCTION

Neurocysticercosis (NCC) predominately affects the adults in their third and fourth decades of life and is relatively uncommon in children and elderly.[5,6] Clinical manifestations of NCC are determined mainly by the locations of the parasite within the central nervous system (CNS), evaluative phase of the parasite, immunological response of the patients expressed as severity of disease activity.[1,2]

Clinical manifestations of parenchymal NCC are different from those of extraparenchymal NCC. Main symptoms of parenchymal NCC are epileptic seizures, focal neurological deficits. Extraparenchymal NCC results in intracranial hypertension, cranial nerve abnormalities and hydrocephalus.

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There is also clinical heterogeneity across geographical area. Most cases from Indian subcontinent present with single degenerative disorder, whereas from Latin America present with few viable cysts.[7] These differences are probably due to complex interaction between host, parasites and environmental factors.[8] Genetic susceptibility to NCC has been suggested by reported positive association of HLA-DRBII 13 with single contrast enhancing CT lesions.[9] A myriad of papers have reported wide range of diverse symptoms and signs related to NCC, such as manifestations of brain stem dysfunction cerebellar ataxia, sensory deficits, involuntary movements, stroke like symptoms, extrapyramidal signs, dementia and cortical blindness. However in some cases, it is not possible to establish a clear cause-effect relationship between these pathologies and NCC and fortuitous relationship among them may occur.

EXTRAPARENCHYMAL NEUROCYSTICERCOSIS

The extraparenchymal location (around 15–30% of cases) develops different clinical manifestations. Headache and signs of intracranial pressure are more frequent in the extraparenchymal location (88%), in comparison with the parenchymal location (10%).

When cysticerci are located inside the ventricular system, life-threatening acute intracranial hypertension as a result of hydrocephalus may occur; as a consequence, severe headache, dizziness and consciousness alteration are the predominant clinical manifestations.

Cysts in the subarachnoid space may lodge in the Sylvian fissure or basal cisterns and grow to a big size (racemose form), causing intracranial hypertension. This is associated with an intense inflammatory reaction, and fibrosis and progressive thickening of the leptomeninges at the base of the brain. Spinal cord cysticercosis is rare. Patients, experience nonspecific clinical manifestations, such as nerve root pain or spinal cord compression syndromes, depending on the level of the lesion.[12]

CASE REPORT

A 25-year-old female presented with history of one episode of partial generalized seizures on with h/o tongue bite and loss of consciousness which lasted for 2 hours. She had two more seizures. Next day patient complained of weakness, numbness, muscle cramps and

jerky movements in the lower limbs. It was more in the left than right. Patient complained of diminution of vision in both the eyes and double vision. Patient also complained of headache since 1 month with increasing intensity since 1 week for which she was given some treatment.

On examination, patient was conscious, oriented to time, place, person, all vital parameters within normal limits. General examination and systemic examination were within normal limits. Fundoscopy showed papilledema, pupils were normal and reactive to light. Sixth nerve examination of both eyes showed lateral rectus palsy. Motor system, left upper limb and lower limb power was grade 4, deep tendon reflexes were normal and left plantar reflex was extensor. Sensory system examination was normal. Power in the left upper and lower limbs was normal. Blood investigations were within normal limit was normal. Computed tomography (CT) scan of brain showed ring enhancing lesion with a nidus and perilesional edema in the right high parietal region (Fig. 1). Possibility of neurocysticercosis was considered. Magnetic resonance imaging (MRI) was suggestive of granuloma in the right parasagittal high parietal lobe-neurocysticercosis and a small hyperintense area within the lesion suggestive of scolex.

Patient was treated with intravenous mannitol, oral glycerol, intravenous levetiracetam, for 1 week, tab albendazole 400 mg bis in a day (BID), tab wysolone 40 mg once daily (OD) with tapering dose for 21 days. Patient clinically improved. Repeat MRI after 30 days showed resolution of the cyst.

DISCUSSION

Cysticercosis consists of infection with the small bladder-like larvae of the pork tapeworm *Taenia solium*. The life cycle of parasite is maintained between man and pig infected with cysticerci. The disease is usually spread by eating foods that contains the tapeworm's eggs. The foods most commonly believed to be the cause are uncooked vegetables. The tapeworm eggs are usually from feces of a person infected with the adult worms. This infection is known as taeniasis when adult pork tapeworms live within the intestines. Taeniasis may also be due to eating cysts in poorly cooked pork. Both forms of infection can occur in the same person at the same time. The term NCC is generally accepted to refer to cysts in the parenchyma of the brain. It presents with seizures and, less commonly, headaches. Cysticerci in brain parenchyma is usually 5 to 20 mm in diameter. In subarachnoid space and fissures, lesions may be as large as 6 cm in diameter and lobulated.

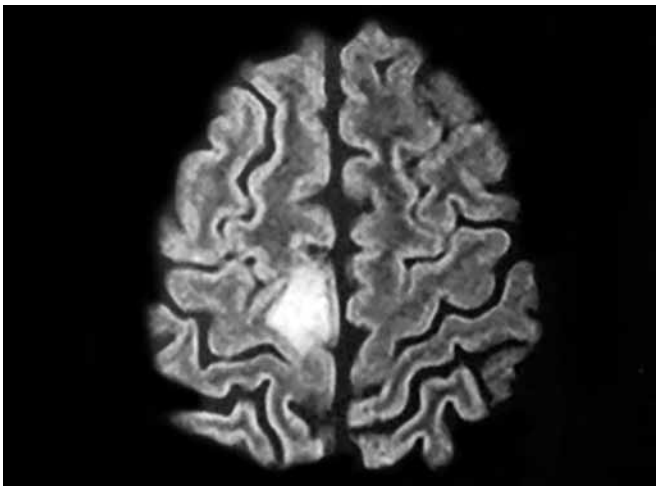

Fig. 1: Computed tomography of brain showing ring enhancing lesion with right high parietal region

Cysts located within the ventricles of the brain can block the outflow of cerebrospinal fluid and present with symptoms of increased intracranial pressure.

Epilepsy is the most common presentation and occurs in 50 to 80% of patients. Generalized tonic-clonic seizures are associated with multiple lesions, whereas single lesion can present as simple or complex partial seizures. Myoclonic seizures have also been described with this disease. Headache, chronic migraine-like or those associated with intracranial hypertension, both can be present. Clinical manifestations in order of decreasing frequency are seizures (80%), headache (40%), visual changes (20%), confusion (15%), ataxia (6%), psychosis (5%) and, in minority, cranial nerve palsies or other focal neurological manifestations. Neurocysticercosis involving the spinal cord, most commonly presenting as back pain and radiculopathy.

Computed tomography scan and MRI will provide objective evidence about topography of the lesions and the degree of the host inflammatory response to the parasite.[10]

Immunological and Imaging Diagnosis

Two main techniques, the enzyme-linked immunosorbent assay (Ab-ELISA) and enzyme-linked immunoelectrotransfer blot (EITB) assay, are used for immunological diagnosis of NCC. Enzyme-linked immunosorbent assay test for antibodies or antigen detection have showed higher sensitivities and specificities in cerebral spinal fluid (CSF) than in sera. Computed tomography and MRI have been useful in the study of the parasite evolution within the brain parenchyma. Once the oncosphere has passed into the parenchyma, it grows and evolves through vesicular, colloidal, granular-nodular and calcified phases. In the vesicular phase, the larva lives inside a translucent liquid filled cystic structure surrounded by a thin membrane. The CT scan depicts circumscribed, rounded, hypodense areas, varying in size and number,

without enhancement by contrast media. With the MRI, the vesicular larva appears with a CSF-like intensity signal on all sequences. Both MRI and CT may show a high intensity, 2 to 3 mm. Mural nodule, depicting the scolex, in the interior of some vesicular cysts. It is better seen on fluid attenuated inversion recovery (FLAIR) sequence.

Neurocysticercosis most often presents as hydrocephalus and acute onset seizures, thus the immediate mainstay of therapy is emergent reduction of intracranial pressure and anticonvulsant medications. Once the seizures have been brought under control, antihelmintic treatments may be undertaken. The decision to treat with antiparasitic therapy is complex and based on the stage and number of cysts present, their location, and the patient's specific clinical presentation. Antiparasitic treatment should be given in combination with corticosteroids and anticonvulsants to reduce inflammation surrounding the cysts and lower the risk of seizures. Surgical intervention is much more likely to be needed in cases of intraventricular, racemose, or spinal NCC. The treatment includes direct excision of ventricular cysts, shunt procedures and endoscopic removal of cysts.

Guidelines issued in April 2013 by the American Academy of Neurology, recommends use of albendazole along with a corticosteroid for the treatment of parenchymal NCC. The guideline, which is also endorsed by the American Epilepsy Society, recommends treatment with albendazole and corticosteroids to decrease the number of active lesions on brain imaging studies and reduce long-term seizure frequency.[11]

Our patient presented with a history of headache which is common in 40% of the cases because of raised ICT, three episodes of seizure which is common in 60 to 90% of the patients, weakness in left upper and lower limb suggestive of Todd's paresis, dimness of vision, double vision suggestive of 6th nerve palsy, papiledema, myoclonic jerks in both lower limbs. Patient responded to the above treatment.

CONCLUSION

So far, diagnosis of NCC is mainly done by neuroimaging. New imaging techniques have improved detection of the scolex inside the cysts, which can be considered pathognomonic of NCC. There is no atypical clinical manifestation of NCC. Location of the parasite in the CNS, age, sex and immunological response of the patient, all seem to play an important role in occurrence of symptoms and signs; however, the relative contribution of these factors, alone or in combination, is still unknown.

In the parenchymal location, seizures are the most frequent clinical manifestation, followed by headache, motor focal deficits, and psychiatric and cognitive symptomatology. Diagnosis of extraparenchymal NCC is even more difficult, because of nonspecific symptomology of intracranial hypertension and meningitis with or without signs of CSF inflammation. The clinical manifestations due to parenchymal location are usually benign and are sometimes transitory in time; on the contrary, clinical presentation of the extraparenchymal location is life-threatening and may develop permanent sequels.

Properly designed studies are required to learn more about the natural history of the disease and the true distribution of clinical manifestations. There is also an urgent need to develop new consensus diagnostic criteria for NCC.

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