Case Report

Neurocysticercosis-uncommon presentation

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Abstract

12 Years old female child presented with jerky abnormal moments of neck lasting for few seconds, occurring 3-4 times in a day since last three months. Her CT Brain with contrast revealed granulomatous lesion, most likely suggestive of Neurocysticercosis. She was treated with oral Albendazole and Prednisolone with Carbazepine in standard doses. She was discharged after two days of uneventful hospitalization. Repeat CT Brain scan after two months shown resolution of cystic lesion. She was clinically absolutely normal and symptoms free.

Key Words: Neurocysticercosis, CT Brain Scan, Tapeworm.

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Received Date: 04/10/2015 Accepted Date: 12/10/2014

Access this article online

Quick Response Code:

Website: www.medpulse.in

DOI: 15 October 2014

INTRODUCTION

Neurocysticercosis is a preventable parasitic infection caused by larval cysts (enclosed sacs containing the immature stage of a parasite) of the pork tapeworm (Taenia solium). The larval cysts can infect various parts of the body causing a condition known as cysticercosis. Larval cysts in the brain cause a form of Neurocysticercosis is a brain infection that can cause seizures and sometimes can be fatal. Neurocysticercosis is considered a neglected Parasitic Infection, one of a group of diseases that results in significant illness among those who are infected and is often poorly understood by health care providers.

CASE REPORT

12 year old female Neha Patil R/O N-8 CIDCO, Aurangabad, born of non consanguineous marriage, 1st by order of birth brought by parents with complaints of abnormal jerky movements of neck**,** lasted for few seconds, occurs three to four times in a day since last three months. There was no history of fever, seizures, loss of consciousness or focal neurodeficit. She attended school regularly. She was non vegetarian. There was no significant family or past history. On examination she was afebrile, vitals stable, fully conscious, oriented in time, place and person. Systemic examination revealed no significant abnormality. No evidence of any neurodeficit. Only finding noted in child was abnormal jerky movements of neck lasting for few seconds. Child was hospitalized and investigated for the same. CBC showed HB – 10gm%, WBC- 6900/cmm, platelet count – normal, no evidence of eosinophilia. Stool examination – normal. Her CT scan report showed:

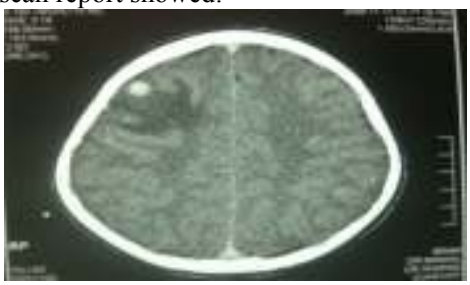

Figure 1:

- CT brain with contrast
- Serial axial section of brain were studied, before and after non-ionic IV contrast.
- There is a 8x7 mm nodular enhancing lesion in the right frontal lobe with perilesional oedema.
- Rest of the cerebral parenchyma does not reveal any area of altered tissue density or loss of tissue.
- There is no evidence of midline shift.
- Cerebellum and brainstem appear normal.
- The ventricular system and the CSF spaces show normal configuration.
- Skull bones are normal
- Impression
- CT brain with contrast reveals a 8x7 mm nodular enhancing lesion in the right frontal lobe with perilesional oedema, suggestive of

granulomatous lesion, most likely neurocysticercosis.

In view of CT scan report she was diagnosed to have neurocysticercosis and started with oral Albendazole 15 mg /kg day in BD doses for 28 days. She also received oral Prednisolone 2mg/kg/day and oral anticonvulsant Carbazepine in standard doses. During hospitalization, her course was uneventful. She was discharged after 2 days. Repeat CT scan was done after 2 months shown resolution of cystic lesion. Clinically she was absolutely normal and symptoms free.

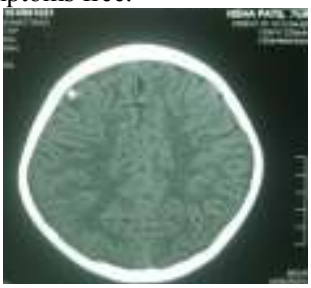

Figure 2

- Diagnosed case of neurocysticercosis post treatment follow up
- CT brain plain
- Serial axial sections of brain were studied
- There is a 3x3 mm calcified lesion in the right frontal lobe with minimal perilesional hypodensity.
- Reset of the cerebral parenchyma does not reveal any area of altered tissue density or loss of tissue.
- There is so evidence of midline shift
- Ce-rebellum and brainstem appear normal.
- The ventricular system and the CSF spaces show normal configuration.
- Skull bones are normal.
- Impression
- CT brain plain reveals 3x3 mm calcified lesion in the right frontal lobe with minimal perlisonal hypodensity suggestive of calcified/healed neurocysticercosis.

DISCUSSION

Cysticercosis is a parasitic tissue infection caused by larval cysts of the tapeworm *Taenia solium*. These larval cysts infect brain, muscle, or other tissue, and are a major cause of adult onset seizures in most developing countries. A person gets cysticercosis by swallowing eggs found in the feces of a person who has an intestinal tapeworm. People do **not** get cysticercosis by eating undercooked pork. Eating undercooked pork can result in intestinal **tapeworm** if the pork contains larval cysts. Pigs become infected by eating tapeworm eggs in the feces of a human infected with a tapeworm.

Figure 3: CDC APRIL 16 2014

PATHOGENESIS

The cystic stage usually do not provoke immunologic response when they live alive and intact. however, when initial infection is massive or if cysts obstructs CSF flow then patient may show symptoms. Most cyst remain viable for 5 to 10 years and then begin to degenerate followed by vigorous host response. They resolved either by complete resorption or calcification.[1] Symptomatic disease results almost exclusively from the invasion of the nervous system (neurocysticercosis) and the eye and is clearly different in parenchymal neurocysticercosis and extraparenchymal neurocysticercosis. The usual presentation of parenchymal neurocysticercosis is with seizures or focal neurologic deficit.[1] Occasionally, the cysts may grow and produce a mass effect. Extraparenchymal infection may cause hydrocephalus by mechanical obstruction of the ventricles or the basal cisterns, either by the cysts themselves or by an inflammatory reaction (ependymitis and/or arachnoiditis). The so-called racemose variety occurs in the ventricles or basal cisterns and is characterized by abnormal growth of cystic membranes with degeneration of the parasite's head (scolex)[3,4]. These cases follow a progressive course, and even after ventricular shunting, the membranes or inflammatory cells and proteins frequently block the shunt. In most patients, neurocysticercosis seems to produce symptoms years after the initial invasion of the nervous system by the parasite[5], by either inflammation around the parasite, mass effect, or residual scarring[6]. There is a clear association between inflammation around one or more cysts and development of symptoms, especially with regard to seizures[7]. Seizures are generalized in 80 % of cases but they begin with simple or complex partial seizures. Other rare presentation of the disease are llike, cerebral infarction resulting from obstruction of small terminal arteries or vasculitis, intellectual deterioration or dementia due to involvement of frontal lobe.[1] The most useful diagnostic study for parenchymal disease is MRI brain which gives information about cyst viability, associated inflammation, number and location of cyst. Some times protoscolex is visible within cyst which is pathognomic for neurocysticercosis.[1]

Table 1: Diagnostic criteria for Neurocysticercosis[8]

Criterion
Absolute:
Histologic demonstration of the parasite from biopsy of a brain or spinal cord lesion.
Cystic lesions showing the scolex on CT or MRI
Direct visualization of subretinal parasites by fundoscopic examination
Major
Lesions highly suggestive of neurocysticercosis on neuroimaging studies
Positive serum immunoblot for the detection of anticysticercal antibodies
Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel.
Spontaneous resolution of small single enhancing lesions.
Minor
Lesions compatible with neurocysticercosis on neuroimaging studies
Clinical manifestations suggestive of Neurocysticercosis
Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens
Cysticercosis outside the central nervous system
Epidemiologic
Evidence of a household contact with *T. solium* infection
Individuals coming from or living in an area where cysticercosis is endemic
History of frequent travel to disease-endemic areas

CSF eosinophilia is not very specific while serum antibody testing has > 90% sensitivity and specificity. Mainstay of treatment of neurocysticercosis includes treatment of seizures with first line antiepilectic drugs which should be continued for 2-3 years if seizures are recurrent or associated with calcification. Use of antiparasitic drugs is controversial but if it is to be used the Albendazole (15mg/kg/day bid) per oral for 28 days is drug of choice. Other alternative is praziquantel (50- 100 mg/kg/day tds) per oral for 28 days. Worsening of symptoms can be follow with use of antiparasitic drugs which seen on second or third day of starting treatment due to host response to dying parasite with increased inflammation. This can be controlled with use of corticosteroid prednisolone (1-2mg/kg/day bid) for 2-3 days prior to treatment and during therapy. Injection Mannitol can be given to treat increased intracranial pressure. All family members of index cases of cysticercosis should be examined for signs of disease or evidence of adult worms. Close attention to personal hygiene, proper handwashing and use of clean fruits and vegetables can avoid ingestion of eggs. All pork should be cooked thoroughly.

CONCLUSION

Though neurcysticercosis is common in developing countries like India, presentation of this condition is not always with typical sign and symptoms. High degree of suspicion is needed to diagnose this condition as there can be atypical presentation are seen in some patients as in this case. Early diagnosis followed by early treatment can cure this condition, also avoids life threatening complications at times. This is treatable and curable condition with 100% recovery if given timely treatment.

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Source of Support: None Declared
Conflict of Interest: None Declared