



A CASE OF NEUROCYSTICERCOSIS PRESENTED WITH SEIZURES: A CASE REPORT

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INTRODUCTION:

Neurocysticercosis (NCC) is defined as the infection of the CNS and the meninges by the larval stage of *Taenia solium*. It is the most common helminthic infection of the CNS worldwide. It is a leading cause of epilepsy in developing countries including India, Africa, Latin America and China. Human cysticercosis is caused by ingestion of *T. solium* eggs from taenia carriers. Parasites may lodge in brain parenchyma, subarachnoid space, ventricular system, or spinal cord, causing pathological changes that account for the pleomorphism of this disease. Staging of cysticerci can be characterized by MRI: Vesicular cysts are stage one and colloidal cysts are stage two. These cysts progress to stage three which has granular nodular degeneration with onset of calcification and finally stage four, or complete calcification on CT and MRI. Seizures/epilepsy are the most common clinical manifestation. The two most common types of cysts are (1) Vesicular cysts which are less epileptogenic and have less mass effect on imaging and (2) Colloid cyst which consists of gelatinous material that exhibits ring enhancement and edema on imaging which is associated with increased epileptogenic potential. Many patients also present with headache, focal deficits, intracranial hypertension, or cognitive decline. Accurate diagnosis of NCC is possible after interpretation of clinical data together with findings of neuroimaging studies and results of immunological tests. However, neuroimaging studies are fundamental for diagnosis because immunological test and clinical manifestations only provide circumstantial evidence of NCC. The introduction of cysticidal drugs changed the prognosis of most NCC patients. These drugs have been shown to reduce the burden of infection and to improve the clinical course of the disease in many patients.

Case Report:

A 22-year-old male Hindu patient with complaining of severe head ache, nausea and vomiting for 3 days followed by involuntary clonic movements of all four limbs, up rolling of eyeball, loss of consciousness and frothing from mouth such 2 episode at home. Then patient brought to GMERS hospital Morbi medical ward on 4th July. History of similar episode 3 years back but not investigated for it. No any other past history of major medical and surgical illness. No any significant family history present. Patient is mixed (vegetarian and non-veg.) by diet.

On examination, patient was semiconscious and responding to painful stimuli. Frothing from mouth present. No pallor, cyanosis, icterus, clubbing, edema, lymphadenopathy seen.

On admission vital, Temp- 98.6 F, pulse rate- 90/min, BP- 128/70 mmHg, RR- 20/min, spo2- 96% on room air, RS- bilateral air entry present and clear, CVS- S1S2+, CNS- unconscious & not responding to deep pain stimulation.

On investigation, HB- 11.2, WBC- 16,400, Platelet- 3.72 lacs, MP- card-negative, PS for MP-negative, dengue card negative for NS-1 and IgM, ESR- 18, creatinine- 0.78, s. urea- 12, s. sodium- 132, s. potassium- 3.5, s. calcium- 9.0, s. bilirubin 1.0 (direct- 0.8/indirect- 0.2), SGPT- 18, SGOT- 20, ALP- 42, S. widal- negative for O and H.

HIV/HBsAg/HCV- Non Reactive. Sputum for AFB was negative. CSF was clear, colourless without cobweb, without bacterial organism and AFB negative. Total count- 4, polymorphs- 00%, neutrophils 100%. CSF-ADA was 8.2 (negative for TB).

CXR and USG-chest— NAD, USG-abdomen— NAD. MRI BRAIN WITH CONTRAST A well-defined, smoothly marginated, altered signal intensity lesion is seen in left frontal lobe. It reveals central hyperintense signal with hypointense rim on T2 images, with corresponding hypointense signal with isointense rim on T1 images. Rim reveals hypointense signal on gradient images. Faint discontinuous area of restricted diffusion with low ADC value of rim is noted. Moderate amount of perilesional white matter vasogenic edema is noted, along with effacement of adjacent cerebral sulci. Lesion measures 11 x 9 x 13 mm. mild blooming on GRE seen which favours calcification. S/o chronic granulomatous lesion p/o neurocysticercosis.

Patient was treated with IV steroids, Albendazole, antibiotics, anti-epileptic drugs for 28 days and discharge with stable vital conditions.

DISCUSSION:

NCC is the most common parasitic infection of the brain, and it is transmitted by the ingestion of *Taenia solium* eggs shed in the stool of a human tapeworm carrier. *Taenia solium* can cause both taeniasis (infection with adult tapeworm) and cysticercosis (infection with cysts). A common misconception is that humans can acquire NCC by consuming undercooked pork. Consumption of undercooked pork will only lead to taeniasis because infected pork contains the larval cysts that develop into adult worms in the human intestine, but does not contain the eggs that cause cysticercosis. The recognition of NCC in the acute setting can be challenging in countries outside of the endemic areas since there is no specific diagnostic finding on routine blood work, including peripheral eosinophilia. Stool examination is also insensitive because several years pass between exposure to *Taenia solium* eggs and the onset of clinical presentation. The approach to diagnosis is based on clinical manifestations, neuroimaging findings, and epidemiologic exposure. The most common clinical manifestation of NCC is a seizure and/or headache. Patients can also present with confusion, vision changes, focal neurologic signs, stroke, and meningitis. Anticonvulsant drug therapy is recommended in patients who present with seizures, even of a single episode, because NCC lesion(s) serve as a

nidus for recurrent seizures. Anti-seizure drugs such as phenytoin, carbamazepine, or levetiracetam can be used. Indications for anti-parasitic therapy are confined to patients with viable and/or degenerating cysts but should be done under close supervision as clinically the patient may get worse initially. Antiparasitic therapy is not recommended in patients with untreated hydrocephalus, high cyst burden, and presence of a calcified lesion(s) only, and hence our patient was not given anti-parasitic therapy. When indicated, albendazole (15 mg/kg/day) is used as the first line. In cases where there are more than two cysts, dual therapy with albendazole and praziquantel (50 mg/kg/day) is recommended. The duration of antiparasitic therapy for the treatment of parenchymal NCC is 14-28 days. Adjunctive corticosteroid therapy is recommended to reduce seizures caused by degeneration of viable cysts due to anti-parasitic drugs. The most commonly used regimens are prednisone (1 mg/kg/day) or dexamethasone (0.1 mg/kg/day), and it is recommended that they are started at least one day prior to initiating antiparasitic therapy and should continue throughout the duration of therapy and are tapered over a few days.

CONCLUSION:

NCC should always be part of the differential diagnosis of adult onset epilepsy. The disseminated form, although rare, should particularly be kept in mind. The usefulness of a detailed physical examination, serological and radiological examination should be evaluated to get a conclusive diagnosis. Early diagnosis is helpful for a better outcome.

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