ORIGINAL RESEARCH PAPER
Volume - 12 | Issue - 01 | January - 2023 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A CASE OF NEUROCYSTICERCOSIS PRESENTED WITH NEW ONSET SEIZURE: A CASE REPORT

General Medicine

Dr. Pradeep K. Dudhrejia M.D. Medicine And Superintendent At General Hospital, Morbi.

KEYWORDS

Epilepsy; Neurocysticercosis; Taenia solium; Computed tomography; Contrast study of Magnetic resonance imaging; Cerebrospinal fluid.

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 can 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 consist of gelatinous material that exhibits ring enhancement and edema on imaging which is associated with increased epileptogenic potential. Many patients also presents 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 65 year old male muslim patient with complaining of high grade fever with head ache, nausea and vomiting for 3days followed by involuntary tonic and clonic movements of all four limb, up rolling of eyeball, loss of consciousness and frothing from mouth, such 2 episode at home. Then patient brought to civil hospital morbi on 3rd December,2022. No any past history of similar illness. No any past history of major medical and surgical illness. No any significant family history noticed. Patient is mixed (vegetarian and non veg.) by diet.

On examination, patient was unconscious, not responding to deep painful stimulation. Frothing from mouth present. No pallor, cyanosis, icterus, clubbing, edema, lymphadenopathy seen.

On admission vital, Temp-104 F, pulse rate- 118/min, BP-148/82mmHg, RR- 38/min, spo2- 87%on room air, RS- bilateral crepitation presents, CVS- S1S2+, CNS- unconscious & not responding to deep pain stimulation, all four limb hypotonia, both pupil normal in size and reactive to light, planter reflex extensor both limb.

On investigation, HB-13.2, WBC-18,400, Platelet-2.72lacs, Mp card-negative, PS for MP-negative, dengue card negative for NS-1 and IgM, ESR-24, creatinine-0.78, s.urea-12, s.sodium-139, s.pottasium-4.5, s.calcium-8.2, s.billirubin-1.0(direct-0.8/indirect-0.2), SGPT- 18, SGOT-20,ALP-42, S.widal- negative for O and H antigen. 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 negative. CSF sugar- 152, CSF protein-91.

CXR and USG-chest— NAD, USG-abdomen— NAD.

MRI BRAIN WITH CONTRAST study performed suggestive of mildly and peripherally enhancing focal lesion of 7.9*4.6 cm size seen in left high frontal cortex without diffusion restriction, and mild blooming on GRE seen which favours calcification. Another such lesions seen in right anterior temporal, left anterior frontal, left parietal lobe. S/o chronic granulomatous lesion p/o neurocysticercosis.

Patient was treated with Iv steroids, Albendazole, antibiotics, anti-epileptic drugs and mannitol for 28days 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. Anti-parasitic 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 better outcome.

REFERENCES:

1. Harrison's principles of internal medicine, 21st edition
2. API textbook of medicine, 11th edition
3. Mansion's tropical disease, 22nd edition
4. Infectious disease book by Jonathan cohen
5. Principles and Practice of Infectious Diseases, 8th Edition