

The Silent Epidemic: Neurocysticercosis and Its Impact on Neurological Health

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Abstract: *Introduction:* Cysticercosis occurs worldwide, affecting more than 2.5 million people. It is the most common parasitic infection of the nervous system, occurring in up to 3.6% of individuals with systemic infection. It is an old disease. In Mexico, for instance, cysticercosis accounts for approximately 25% of intracranial tumors. Humans are infected when they become the intermediate host after ingestion of *Taenia Solium*, the pork tapeworm. *Case Report:* In this paper we discuss a case of a 10 - year - old female child patient with no any past medical history who was diagnosed with Neurocysticercosis (NCC) after initially presenting with focal seizures. In addition, we also discuss how this patient was non - surgically managed after radiological and serological confirmation of NCC. Besides case details, we also elaborate on the parasitology of the tapeworm *Taenia Solium* along with potential sources of infection in this case. Finally, we also discuss, different ways of classifying NCC based on literature review. *Conclusion:* Thus, this case report highlights a classic presentation of NCC due to eating poorly cooked meat and inappropriate personal hygiene along with image findings and clinical management.

Keywords: Albendazole, Faecal - Oral transmission, Neurocysticercosis, Seizures, NCC

1. Introduction

Neurocysticercosis is a parasitic infection caused by the tapeworm *Taenia Solium*. It is a leading cause of epilepsy in developing countries including India, Africa, Latin America and China [1]. In India contributes to 1.2% of all space occupying lesions and 5.2% of late onset epilepsies. Acute seizures in neurologically normal children over 3 years, 36.7% revealed inflammatory granulomas. Teniasis not uncommon in school children. Pork eating is not the only cause, Contamination of vegetables and water by cysts acquired from human excreta is the most important cause of the infestation. The CNS involvement in cysticercosis is protean and may involve the parenchyma, meninges, subarachnoid space, ventricles and the spinal cord. **In India parenchymal cysticercosis is most common.**

Active neurocysticercosis is confined to the following three stages: (1) when larvae enter the brain and oedema can be detected; (2) when a thin - walled cyst forms; and finally (3) during an inflammatory stage when the cyst wall begins to degenerate and larvae are released. Inactive disease occurs when the cyst becomes calcified and nonviable. (Figure 5) Frequently patients with multiple cysts may have active and inactive lesions concurrently. In one large series, 70% of patients were considered to have active disease (37% with stage 1 and 2; 33% with stage 3); the remaining 30% had inactive disease [6]. Sero Diagnosis of NCC not satisfactory, IgM ELISA more specific than IgG ELISA, IgG ELISA sensitivity and specificity 67% and 64%. CSF/SERUM not recommended for routine diagnosis of NCC except in meningeal/ intraventricular. The findings on CSF are rarely helpful, and findings range from normal to isolated high protein levels with or without an inflammatory pleocytosis. In approximately one - third to two - thirds of cases, CSF eosinophilia is present, A lumbar puncture should not be done in the presence of suspected increased intracranial pressure. **Neuroimaging: is currently the method of**

diagnosis. CT scan suffices in most cases. Non contrast CT scan identifies active/live cysts as hypodense cystic lesions. Contrast enhanced CT scan enhances the ring stage, better identification. **MRI is superior in identifying lesion detail** (Cost is 5 - 6 times as compared to CT scan), MRI distinguishes cysticercosis from tuberculomas better, both are common inflammatory granulomas that merit clinical distinction. Staging of Cysticerci can be also characterized by MRI: Vesicular cysts are stage one and colloidal cysts are stage two [2]. 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 [2].

Cysticercosis may be asymptomatic and identified during incidental neuroimaging. When symptomatic, seizures are the commonest symptom (90%) - two thirds are partial, one third generalized. Most commonly patients present with new onset partial seizures with or without secondary generalization due to cyst formation in various areas of the brain parenchyma. 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 oedema on imaging which is associated with increased epileptogenic potential **Seizures in NCC - acute symptomatic (70%) or remote symptomatic (30%).** [1]. Alternatively, patients may present with a generalized headache secondary to increased intracranial pressure and meningitis. Other manifestations are raised intracranial pressure (15%), neurological deficits (10%), subcutaneous nodules and ocular cysts (5 - 7%). Meningeal variety uncommon in India. Disseminated neurocysticercosis uncommon.

Patients in whom treatment should be considered are those with hydrocephalus, arachnoiditis, recurrent seizures in the presence of multiple or mixed active and inactive lesions, encephalitis, increased intracranial pressure, intraventricular

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cysts, and spinal neurocysticercosis. Both praziquantel and Albendazole is approved in USA. (1) **Albendazole (preferred)** - [[at]15 mg/Kg/day] Short course - 8 days, long course - 28 days. (2) Praziquantel (expensive) – [[at]50 mg/kg/ day] - 15 days. Phenytoin and carbamazepine significantly decrease concentrations of praziquantel, and may be responsible praziquantel treatment failure [8]. Most authorities suggest that albendazole may be more appropriate for initial treatment. Corticosteroid (oral Dexamethasone [at] 0.6 mg/kg/day) must be given for 2 days before and 5 days after starting cysticidal therapy. Initiation of cysticidal therapy may enhance perilesional oedema and worsen symptoms. Surgical intervention may be required to alleviate mass effect, to remove cysts causing obstruction of the ventricles, or for shunt Placement. Ocular cysticercosis may also require surgical excision of the cyst.

The prognosis of neurocysticercosis is extremely variable, depending on the sort of infection. Patients with single enhancing lesions generally do well and often are seizure free and discontinue therapy within 1 to 2 years. Patients with calcified lesions may require chronic antiepileptic therapy. Patients with numerous subarachnoid Cysticerci often require repeated courses of therapy and even repeated surgeries. However, with optimal management of hydrocephalus, cases are rarely fatal. [7]

2. Case Report

Our patient is a 10 - year - old female child (Figure 4) with no any past medical history who was brought to the emergency room with a new onset right sided tonic clonic seizure witnessed by her mother which was last for 10 - 15 mins, Up - on arrival, patient was postictal and drowsy. On physical examination, she was semi - conscious, responsive but confused with a right sided tongue bite without any focal neurological deficits. Initial treatment with 100% oxygen and inj. Valproate was loaded Intravenously, kept nil per oral and supportary treatment given. On further interview, patient's mother reported that they have occupation of Meat shop and the child used to eat meat like chicken and fish on daily basis from Last 6 year. This may reveal higher possibility of an infectious etiology towards NCC, hence MRI of brain was Planned. **MRI demonstrated a well - defined ring like lesion seen in left parietal paramedian cortical - subcortical region with mild perilesional oedema and represent infective granulomatous lesion like Neurocysticercosis.** (Figure 1, 2 and 3) . Blood tests revealed

elevated IgE levels along with (+) CSF ELISA testing. Awake EEG was performed with interictal bilateral frontal temporal spike wave epileptiform discharges although no discharges were noted during photic stimulation with uneventful sleep recordings as well. **Stool culture and analysis was positive for adult tapeworms.** Then Patient was Review by the Paediatric Neuro Physician and started oral Prednisolone [at] 2mg/kg/day for 5days, oral Albendazole[at]15mg/kg/day for 14days which was advised to start after 3day, consequently, patient was started on i. v. Valproate, then switched to oral Phenytoin for seizure control and advised to continue till 1 year. In addition, patient was advised initial follow up every two weeks for first six months. During initial follow up patient had no recurrent seizures. In addition, patient has been seizure free for more than one year after initial treatment. In addition, patient has regularly followed up with good medication compliance, improved personal hygiene and returning to a vegetarian diet.

3. Discussion

Based on this case report, we believe the two possible sources of tapeworm infection are from the contamination of the poorly cooked meat and inappropriate personal hygiene.

In the southwestern US and third world countries, NCC has been a major public health problem and also one of leading causes of epilepsy [1]. Croker et. al determined that there were about 300 cases of NCC in Southern California in 2009, in which men were more likely to be affected as compared to women [3].

Like other infectious diseases, Neurocysticercosis outbreaks financially burden countries that are economically well off as well as underdeveloped nations [4], [5].

Neurocysticercosis poses a major health hazard in close communities where people have a high likelihood of infection because of exposure to infected faeces that contains tapeworm eggs. Due to increasing global migration, NCC has a great potential for rapid spread. Since carriers may be asymptomatic, tracking the infectious source can become very difficult and expensive for health investigators. Prevention strategies must include proper food surveillance via stricter controls implemented via WHO standards and most importantly thorough history and physical examination of patients with attention to their diet & lifestyle. [9]

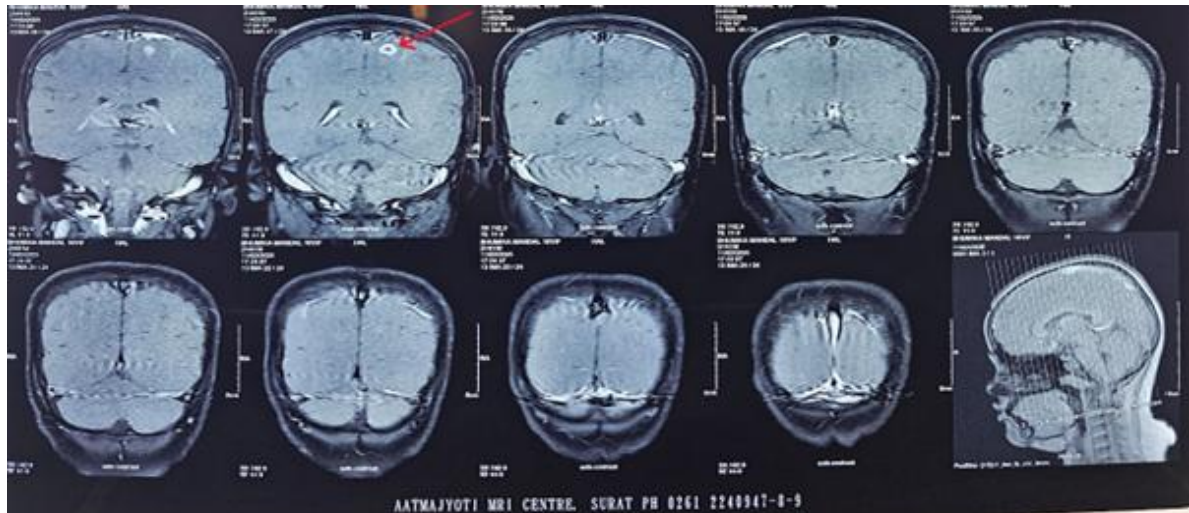


Figure 1

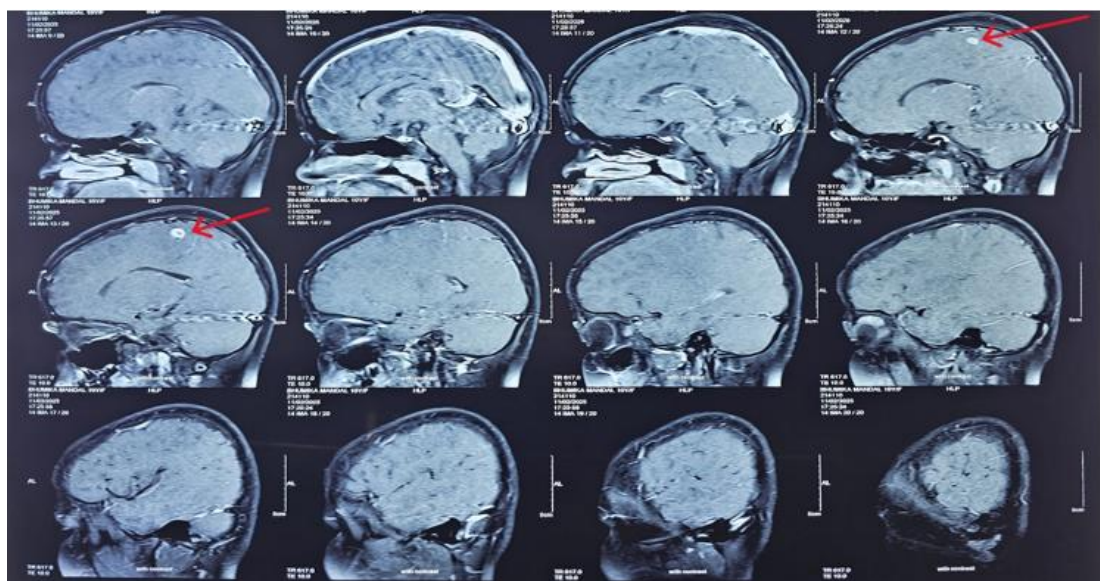


Figure 2

Figure 1 & 2: Well - defined ring like lesion seen in left parietal paramedian cortical - subcortical region with mild perilesional oedema, may represent infective granulomatous lesion like Neurocysticercosis

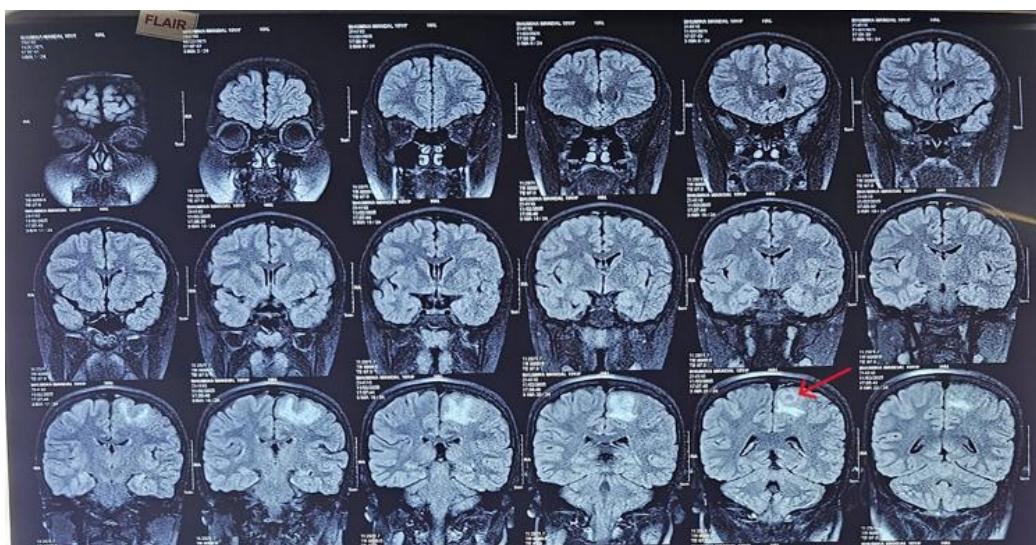


Figure 3

Figure 3: Mild perilesional oedema noted in adjacent left fronto - parietal white matter, which shows ill - defined hyperintense signal on FLAIR images



Figure 4

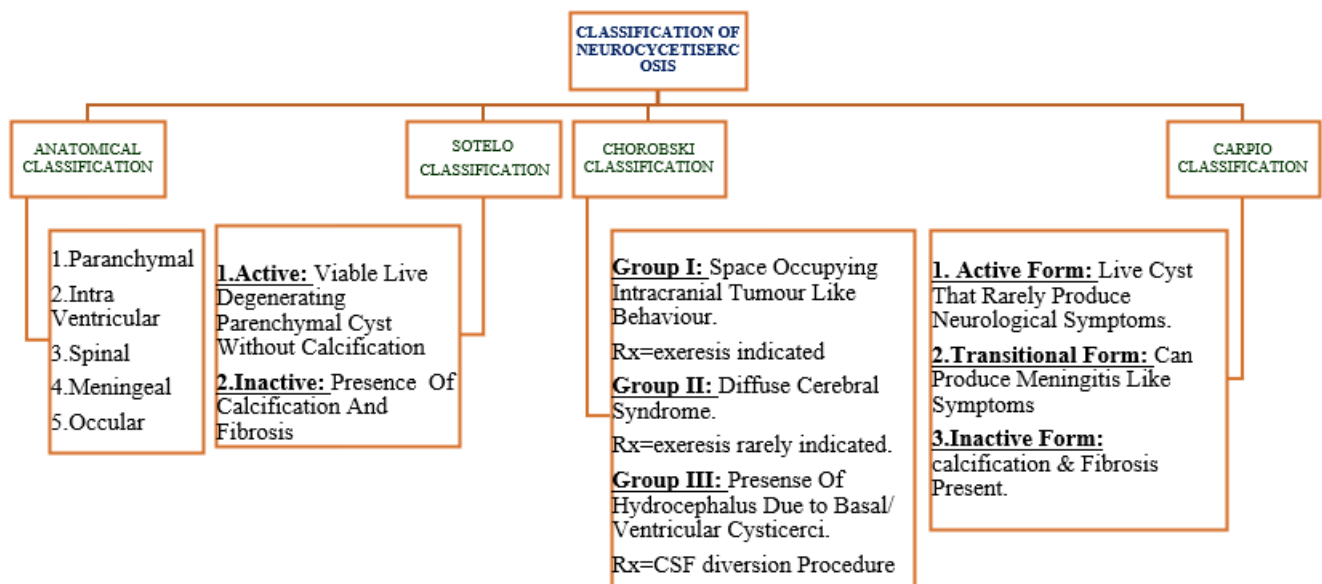


Figure 5

Various classification of neurocysticercosis

4. Conclusion

Neurocysticercosis has the potential of becoming a much more prevalent disease with high societal and individual costs. The spread of disease may be decreased by careful attention to the safety of the global food supply chain and public education campaigns.

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