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Case Report

A Rare Case Of Disseminated Toxic Neurocysticercosis Causing Threatening Of Life

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Abstract

Neurocysticercosis is the most common parasitic infection of the central nervous system. However, disseminated cysticercosis is rare. Cysticercosis is caused by the larvae of Taenia solium, a tapeworm. Humans are infected after eating contaminated food containing the eggs of the tapeworm. Its clinical manifestations are highly variable and depend on the number, stage, and size of the lesions and the host's immune response. We report a case of a 27-year-old lady, who presented with severe headache which was diagnosed as disseminated neurocysticercosis with characteristic imaging findings.

Keywords: Disseminated neurocysticercosis, severe headache, contaminated food.

Introduction

Cysticercosis is the most common parasite infection in the world, and CNS lesions eventually developed in 60 to 90 percent of patients with cysticercosis and it is known as neurocysticercosis (NCC), which is the most common parasite disease of the central nervous system.[1] Cysticercosis is highly endemic in most developing countries as a result of poor socioeconomic development and domestic pig raising. However, nowadays, the infestation is being seen more fre-quently in developed countries due to immigration and travel.[2]

Neurocysticercosis develops after the inges-tion of contaminated food containing the eggs of the tapeworm (ie, fecal-oral contamination).[3] A "cysticercus" cyst in the brain is actually the secondary larval form of the parasite. The "scolex" is head like part of the tape worm, bearing hooks and suckers in the larval form, invaginated into one end of the cyst.[4]

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Etiology

Most NCC are caused by encysted larvae of the pork tapeworm Taenia Solium. It is a two-host zoonotic cestode. The adult stage is a 2- to 4-m-long tapeworm that lives in the small intestine of humans. No other final hosts are known for T. solium tapeworms in nature. As in all cestodes, the gravid proglottids at the terminal end of the worm are full of eggs that are the source of infection with the larval stage, or cysticercosis. The natural intermediate host is the pig, harboring larval cysts anywhere in its body. Human become infected after ingestion of contaminated food with cysts by fecal-oral contamination.[6,7,8] Prevention- Hygiene and avoid raw food. When detected in the intestine praziquantel should be given.

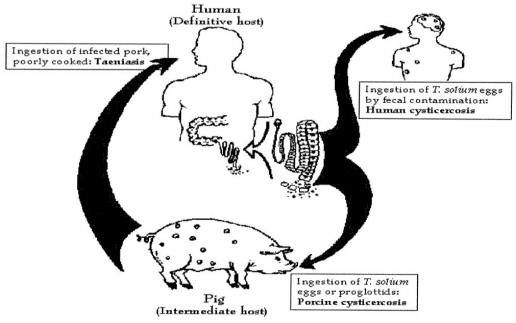

Figure 10. life cycle of T.soleum[8]

Gross pathology

There are four stages of NCC –first stage is vesicular stage which has a viable larvae (cysticerci) with no symptoms. In the second stage is known as colloidal vesicular stage the cyst is filled with fluid which is thick and turbid. A striking inflammatory response is initiated and characterized by a collection of multi nucleated giant cell, macrophages and neutrophils. A fibrous capsule develops and perilesional edema becomes prominent. Third stage is granular nodular stage represents involution with collapse and refraction of the cyst into a granulomatous nodule. The fourth stage is nodular and calcified stage.[5]

Case report

A 27 year old young lady was referred to the radio diagnosis department for CT for the complaints of headache since 4 months increased since 4 days. Emergency CT brain was done and showed multiple tiny calcific lesions about 1mm in diameter in average (old calcified scolex) and also detected multiple multiple small (2- 3 mm) cystic areas with surrounding edema and intrinsic hyperdense foci (scolex) measuring about 1 mm at the periphery in bilateral cerebral hemisphere and cerebellum with mild narrowing of the ventricles Fig.1, 2 & 3. Brainstem appears normal in the CT. Findings are suggestive of disseminated neurocysticercosis of both old and active forms. To reconfirm contrast was suggested after checking the renal functions (blood urea and creatinine), however the patient developed severe breathlessness and unconsciousness. Then patient was advised for MRI brain plain study. MRI findings shows multiple NCC in the cerebellar, cerebral hemispheres, brain stem of colloidal vesicular stage with mild narrowing of the ventricles and mild tonsillar herniation due to diffuse cerebral and cerebellar edema (fig 4,5 & 6) with mild dilatation of the perioptic subarachnoid stage. MRA shows spasticities of the distal cerebral vessels (middle cerebral, anterior cerebral and posterior cerebral arteries) suggestive of toxic vasculitis. MRV shows diminished flow involving superior and inferior sagittal sinuses and bilateral sigmoid and transverse sinuses. No flow is seen in the internal cerebral and vein of Galen (fig. 9).These above findings are suggestive of raised intra cranial pressure. However no evidence of subarachnoid hemorrhage which has worst prognosis.

The patient was shifted to intensive care unit and started immediate treatment (steroids and mannitol) to reduce cerebellar edema and focal toxins of the cysticerci in addition to respiratory supports of ventilator. However ECG was normal. Patient was treated in the intensive care unit (ICU) for about 2 days with ventilator support. The next day patient became conscious with further treatment improved and shifted to general ward.

Discussion

This is a rare case of disseminated toxic neurocysticercosis presenting with severe headache. Many cases of neurocysticercosis had been reorted without any toxix signs. However in this case patient developed uncon-

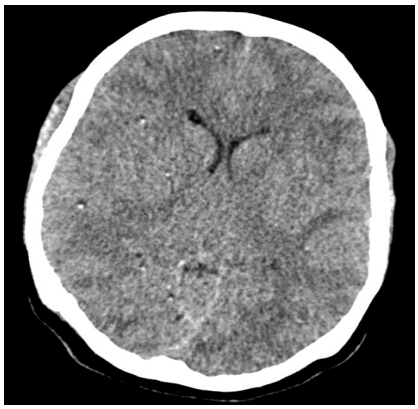

Fig 1. CT axial section at the level of lateral ventricles.

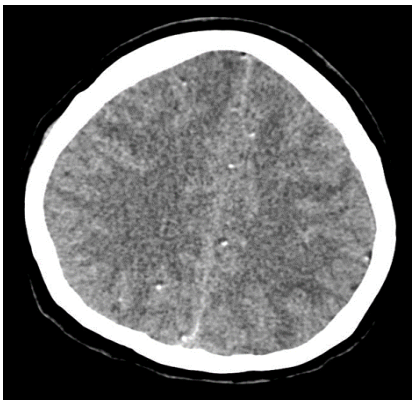

Fig 2. CT axial section at high parietal region.

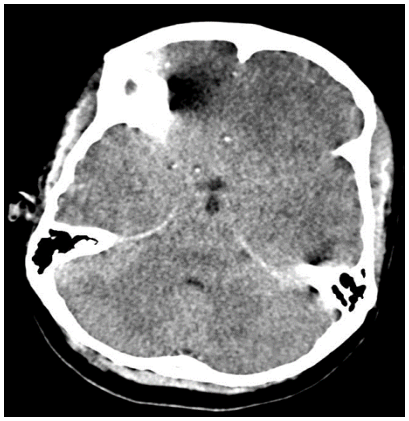

Fig 3. CT axial section at the level of cerebellum

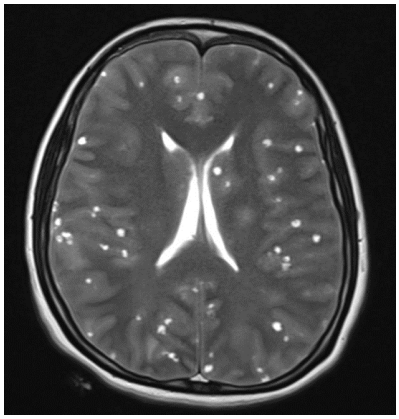

Fig 4. MRI T2WI at the ventricular level

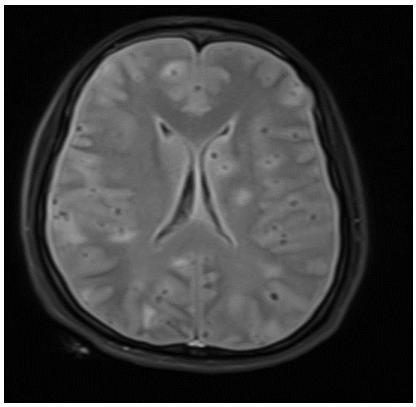

Fig 5. Axial MRI T1WI at the ventricular level

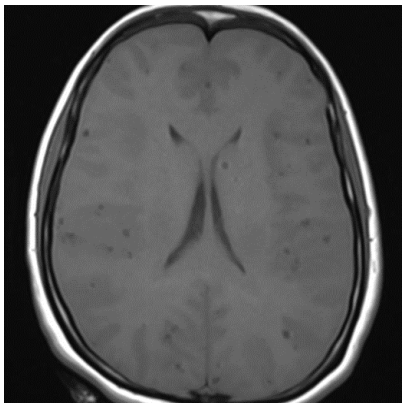

Fig 6. MRI FLAIR image

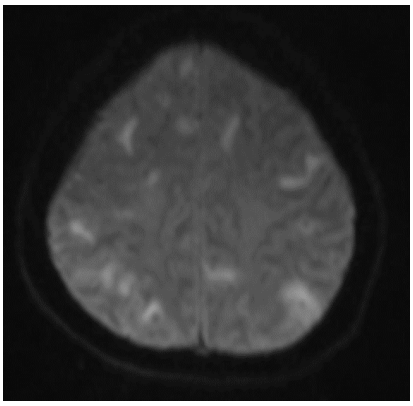

Fig 7. MRI DWI

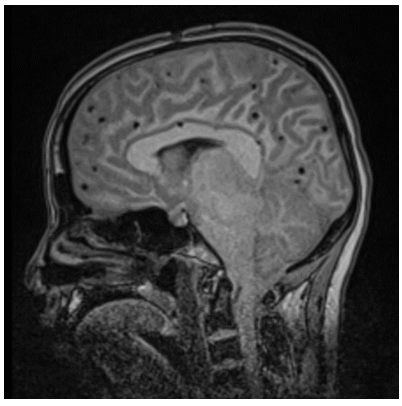

Fig 8. MRI sag MP rage

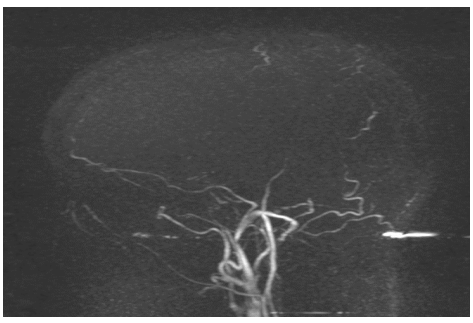

Fig 9. MRV

sciousness after 2 days of hospitalization. In most of developing countries neurocysticercosis is common parasite disease, especially in Latin-America, Asia and Africa.[8] Prevalence of cysticercosis is high in these countries due to unhygienic practices, consumption of undercooked meat and vegetables and close contact between humans and pigs.[10] The larvae of taenia solium cause neurocysticercosis and the ova of the tapeworm spread via the fecal-oral route. The intermediate host is the pig, which harbors the larvae after eating ova, while the definitive host is the human being. Once the human ingests egg-contaminated food or water, the embryosphere is dissolved by gastric acid and enzymes, oncosphere is liberated. The embryo invade the intestinal wall and enters into the blood circulation then extend into central nervous system (CNS), eyes, muscle, heart, fat tissues and skin.[7] In our case, the patient had the habit of eating undercooked meat and uncooked vegetables, stayed in poor sanitation and poor personal hygiene. Neuroimaging studies are now evolved, and MRI can even detect the different stages of cysticercal larvae better than CT.[9]

Clinical presentations of our patient was only severe headache. The radiologic findings depend on several factors, including pathologic stage, location, and associated complications such as vascular involvement (ie, arteritis with or with-out infarction), inflammatory response (ie, edema, gliosis, or arachnoiditis), and, in ventricular forms, degree of obstruction. Therefore, the diagnos-tic approach, management, and prognosis differ widely depending on the type of infection. Neurocysticercosis is a preventable disease, linked to poverty and ignorance, and eradication programs should target all the stages of larvae, including human carriers, infected pigs, and eggs in the environment.[10]

Conclusion

It is a very rare case of disseminated and toxic NCC involving multiple parts of the brain causingdiffuse vasogenic edema, increased intracranial pressure and respiratory failure due to toxins produced by the cysticerci. Immediate treatment is required in a highly equipped intensive care unit with experienced doctors which can save the life of the patient. It is highly important to detect the early stage of the raised intracranial pressures to save the live after intensive medical treatment with ventilators correctly. Even though the disseminated NCC is very rare infectious disease, we recommend to investigate other parts of the body Eg. heart, lungs, spine etc. Financial and sponsorship support- Nil.

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