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Neurocysticercosis

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I. Introduction

Cysticercosis caused by larval stage of the tapeworm Taenia solium, is a major public health problem.[1] Humans are only definitive host of T. solium harbouring the adult tapeworm in the intestine (referred to as, taeniasis). Both humans and pigs act as intermediate hosts and harbour T. solium larvae in different internal organs (referred to as, cysticercosis) including the brain (referred to as, neurocysticercosis - NCC).[2]

Neurocysticercosis is central nervous system (CNS) infection with T. Solium,it isidentified as the single most common cause of community acquired active epilepsy cases in the developing world including India.

It is also becoming more common in the developed world because of increased migration of people with the disease or Taenia solium carriers and frequent travel to the endemic countries.

Life- cycle of Taenia solium

Life cycle of T. solium comprises two natural hosts, humans as the definite and swine as the intermediate host. Human harbours the adult tapeworm; eggs produced by the worm are disseminated to the environment through faeces. The pig ingests some of these eggs, which develop into cysticerci in internal organs like muscle and brain.[3]

When human consumes contaminated pork containing cysticerci itresults in the transmission of viablecysticerci to the human small intestine, where by the action of bile and digestive enzymes, the scolexof a cysticercusevaginates and attaches to the intestinal wall. Proglottids multiply and the parasitebecomes a cestode that can be passed in the faeces as mature proglottids.

The two main sources from which humans contact cysticercosisare ingestion of food contaminated with taeniid eggs and the faeco-oral route in individuals harbouringthe intestinal cestode.[3] Once in the digestive tract, the eggs loose their coat due to action of gastricand pancreatic enzymes and liberate hexacanth embryos or oncospheres. Aided by their hooklets,onospheres cross the intestinal wall and local venules, enter the systemic circulation and are carriedto different organs of the host.

Aim of the study:
To study the clinical profile, neuroradiological features and outcome of patients presenting with neurocysticercosis.

Objectives:
To study the profile of patient with neurocysticercosis with respect to clinical presentation and neuro-adiological imaging, mode of treatment and outcome.

II. Materials and Methods:

The study was done on 40 patients who were referred to Radio-diagnosis department from neurology OPD, detected to have intra-cranial lesions on MRI of brain inMahatma Gandhi Medical College & Hospital, Jaipur betweenjan 2014 to may 2014.

Exclusion criteria:HIV patients.
Data Acquisition: Patients referred for MRI of brain, underwent the examination after contraindications for MRI were excluded.

Observation &Results: The present study was carried out to describe the clinical presentation and neuro-radiological imaging, mode of treatment and outcome of patients with NCC.

Data Analysis: 40 cases of NCC were included in the study. The observations of these 40 patients were compiled and analysed.

Age and sex distribution: In our study,maximum no of patients encountered in age group of 20 to 39 years (22),followed by 10 to 19 years(10) and>40 years (8). There were 21 males and 19 females.

Clinical Presentation: Seizure was the most common clinical presentation.18patientspresent with generalized tonic clonic seizures,13patientshad partial seizures and 9patientspresented with status epilepticus.

Others symptoms were headache with vomiting, double vision, reduced vision, sudden onset of weaknessand loss of consciousness. Some have combination of one or more symptoms.

Neuroradiological Investigations
MRI alone was used in diagnosing patients.

Single lobe

Lobes	No of patients
Parietal	16
Frontal	9
Temporal	4
Occipital	7

Multiple lobes

Lobes	No of patients
frontal + occipital	2
frontal + temporal	1
parietal + temporal	1

Stages:MRI findings

CT has a higher sensitivity and specificity and superior to MRI in identifying calcified granuloma. However intraventricular cysts might be difficult to identify with CT due to similar attenuation of CSF and cyst fluid.[4]

The main advantage of MR over CT is its higher contrast resolution, which makes for better lesion conspicuity. This higher resolution is particularly helpful in evaluation of ventricular involvement.

It is well recognized that in neurocysticercosis in any location, the attenuation or signal intensity of the cyst fluid depends on the viability of the parasite. Live cysts have a signal intensity and (as mentioned earlier) attenuation similar to those of CSF. However, with degeneration, the cyst fluid becomes more proteinaceous and gelatinous. This change manifests as increased signal intensity on T1-weighted MR images.[5]

MRI appearance of various stages in MRI

Vesicular stage-Cyst signal intensity similar to that of CSF on T1- and T2-weighted images; cyst wall is well defined and thin, with little or no enhancement on gadolinium-enhancedimages; scolex (hole with dot appearance)

Colloidal vesicular - Cyst contents hyperintense on T1- and T2-weighted images (proteinaceous fluid), cyst wall is thick and hypointense, pericystic edema, pericystic enhancement on gadolinium-enhanced images.
Granular nodular- Similar to colloidal vesicular stage but with more oedema and thicker ring enhancement.
Calcified nodular- Hypointense nodules, no edema, no enhancement

Treatment

Out of 40 patients 23 were treated with antiepileptics,steroids and albendazole.6 were treated with antiepileptic only and 11 were treated with antiepileptic and steroids.

Follow Up

36 patients followed for 1 month, 27 patients showed no recurrence of presenting symptomsin the month of treatment, while 9 patientshad recurrent seizures in spite of the treatment given.

III. Discussion

Neurocysticercosis is one of the leading causes of adult-onset seizures in developing countries cause by Tinea solium.

Seizures in NCC are commonly due to inflammation caused by degenerating cysticerci and uncommonly due to vasculitis etc. In our study which was done on 40 patients, majority were males 21 (52.5%) with most common age group of 20-39 years. 18patientspresent with generalized tonic clonic

seizures,13patientshad partial seizures and 9patientspresented with status epilepticus.[6,7] These findings are similar to the ones found in our study.

Majority of the studies done have dealt with single lesion neurocysticercosis. These have shown a presence of a solitary granuloma in 60.8-76% of their patients.[8,9] In ourstudy, 90% patients had presence of solitary nodule.

Parietal lobe was the commonest site of nodule in ourstudy (44.4%), followed by frontal lobe (25%).

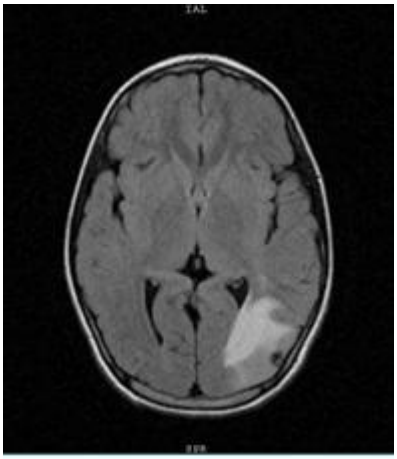
Studies done by Baranwal AK et al and Singhi Pet al had reported a 41% and 57.3% parietal lobe involvement, respectively.[10,11]

In our study, antiepileptics, steroids and albendazole were treatment ofchoice with 57% patients receiving it.

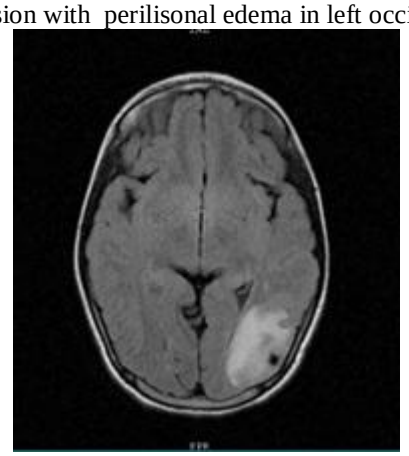
Steroids have a dual role as they not only reduce the perilesionaledema but also increase the plasma concentration of albendazole.

IV. Conclusion:

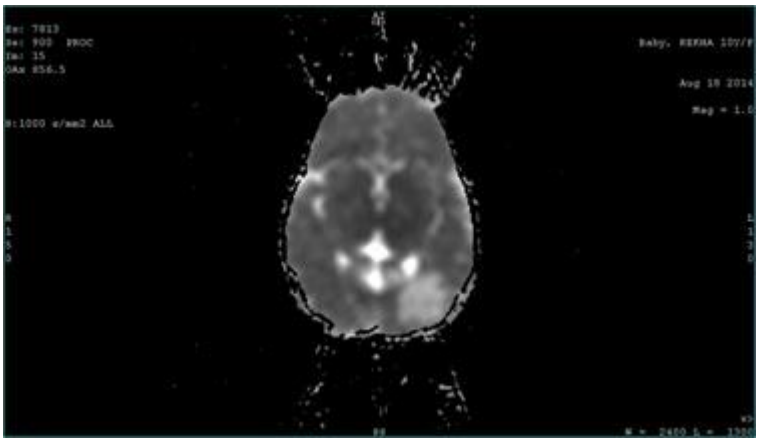
In a developing country like India most of the middle age group patients presented with new onset seizures, on neuroimaging showing a solitary or multiple ring enhancing lesion one should always be suspect for neurocysticercosis and managed on albendazole, steroids and antiepileptics.



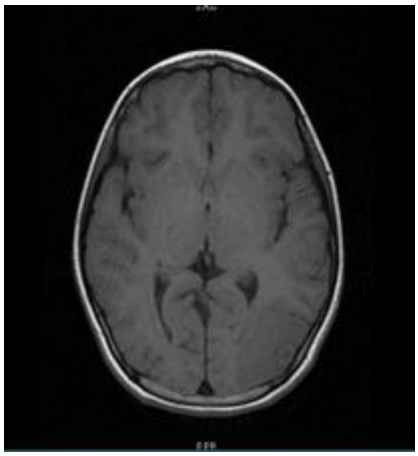
MRI FLAIR axial image showing lesion with perilisonal edema in left occipital lobe



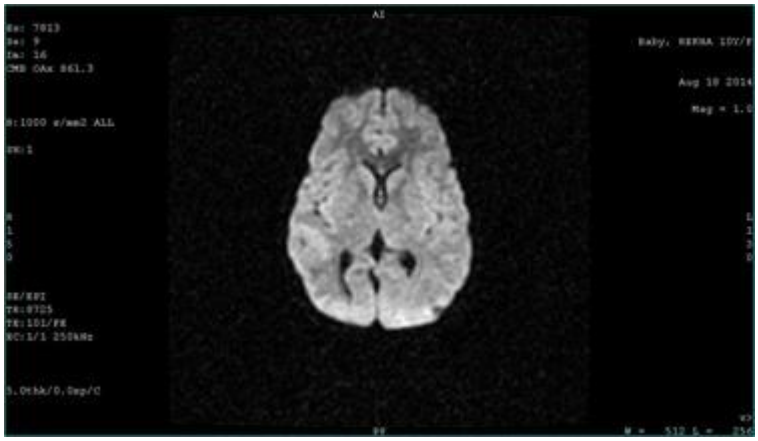
MRI FLAIR axialimage showing lesion with perilisonal edema in left occipital lobe



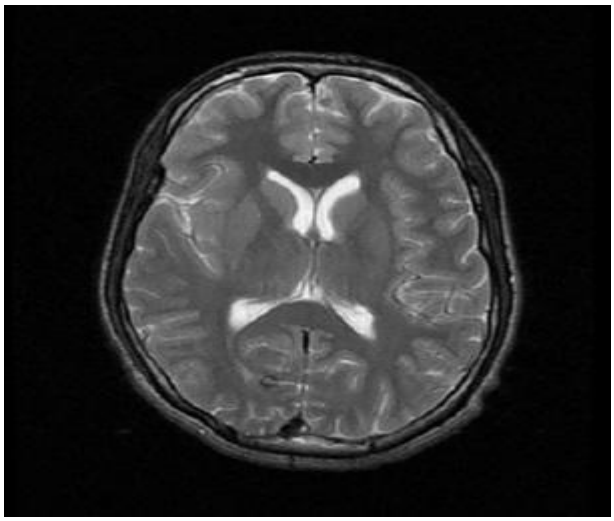
MRI ADC axialimage showing lesion with perilisonal edema in left occipital lobe



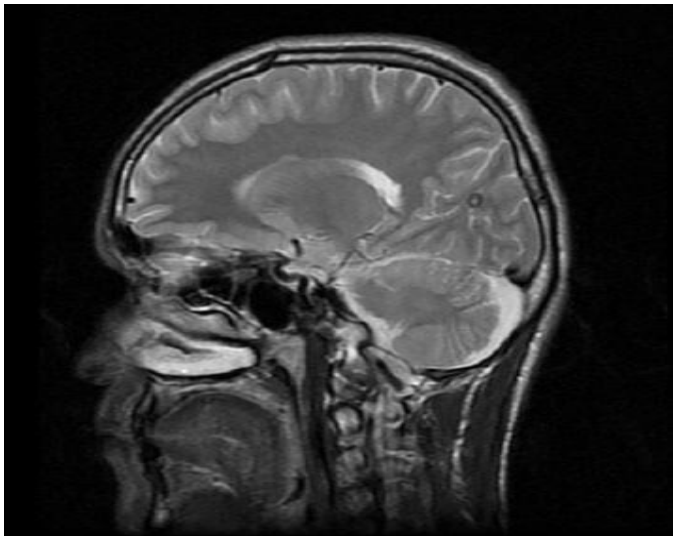
MRI T1W axialimage showing hypointense lesion in left occipital lobe



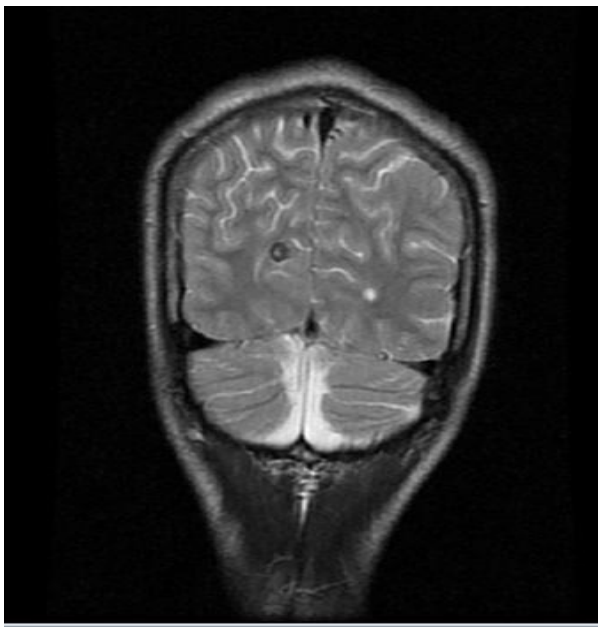
MR DWI axialimage showing lesion with no diffusion restriction in left occipital lobe



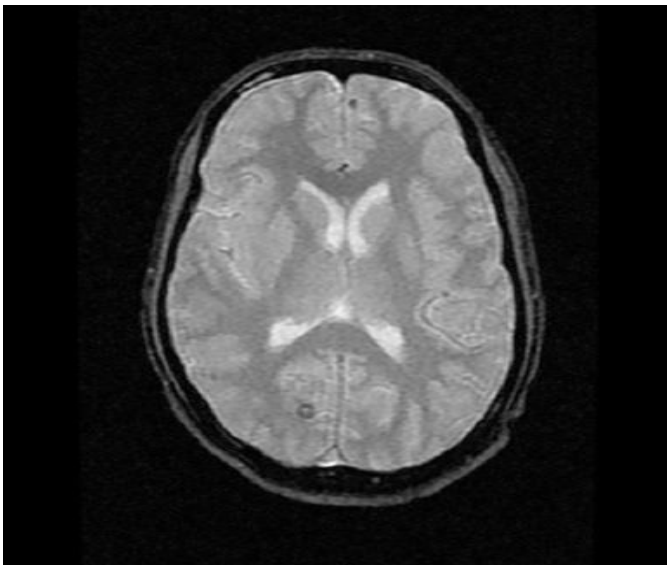
MRI T2W axialimage showing lesion in right occipital lobe



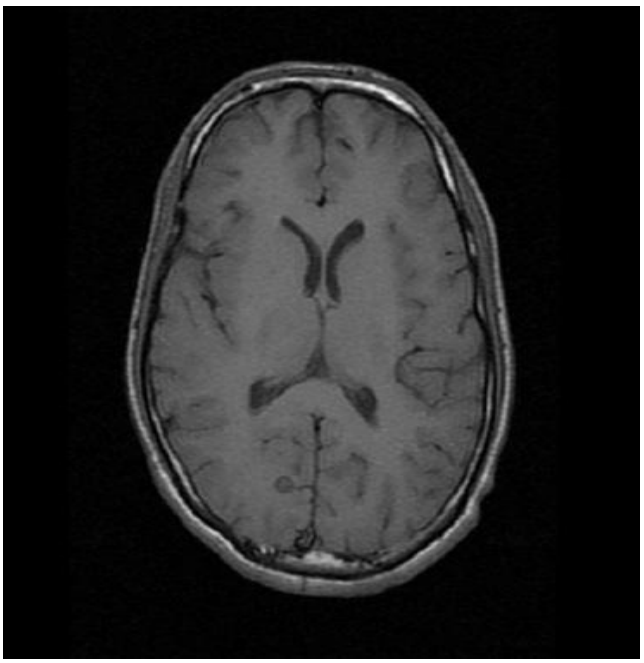
MRI T2W segital image showing lesion in right occipital lobe



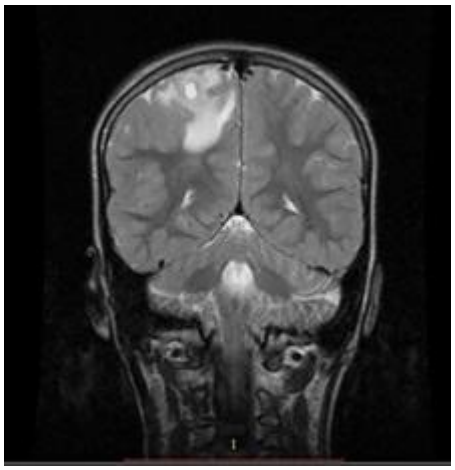
MRI T2W coronal image showing lesion in right occipital lobe



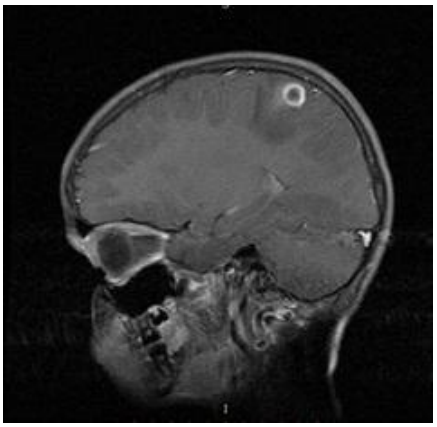
MRI FLAIR axial image showing lesion in right occipital lobe



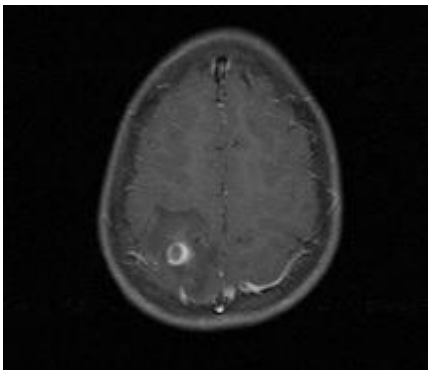
MRI T1W segital image showing lesion in right occipital lobe



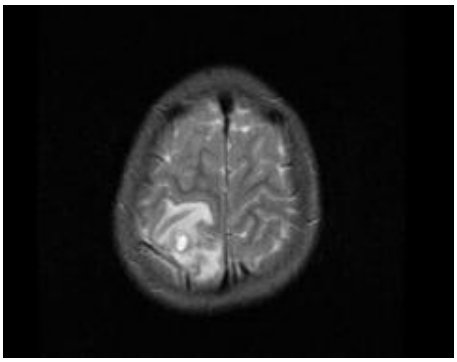
MRI T2W coronal image showing lesion in right hi parital region.



MRI FLAIR segital image showing lesion in right hi parital region.



MRI T1W axial image showing lesion in right hi parital region.



MRI T2W axial image showing lesion in right hi parital region.

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