Neurocysticercosis

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Neurocysticercosis occurs due to infection with intermediate stage or larval stage of Taeniasolium (pork tapeworm) is the most common inflammatory granuloma encountered world wide as well as in India. Usually human beings acquire the intermediate form by ingestion of food or water contaminated with eggs of TaeniaSolium. Sometimes individuals harboring the adult worms due to ingestion of undercooked pork may infect themselves with eggs by fecooral routes. In the small intestine, the egg releases an oncosphere that crosses the gut wall and spreads hematologenously to many tissues including the brain and give rise to neurocysticercosis.

Clinical Manifestation[1]:

Neurocysticercosis can present with various clinical manifestations depending upon the site of lesion. According to this, it is useful to classify neurocysticercosis as parenchymal, intraventricular, meningeal, spinal and ocular.

Parenchymal : (75 to 85%) most common variant & manifests as seizures- generalized or partial-simple or partial- complex, mainly with motor manifestation. Cerebral infarction can also occur due to obstruction of the small terminal arteries or due to associated vasculitis. Rarely massive or disseminated cysticerci in brain present with fulminant encephalitis.

Intraventricular : (5-10 %) usually presents with feature of raised intracranial tension, focal neurological deficit, & even with hydrocephalus. Most common site of obstruction is 4th ventricle.

Meningeal : (2-5 %) associated with sign of meningeal irritation & raised ICT.

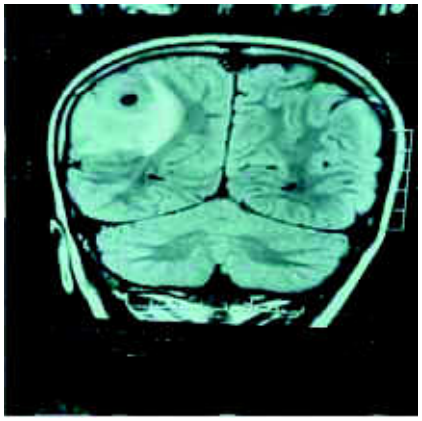
Spinal : (1-3%) extremely rare. Usually present with feature of cord compression, root pain, or rarely as a transverse myelitis.

Ocular (1-2%) present with features of decreased visual acuity & even blindness. Cause of this is either retinal or vitreal detachment or iridocyclitis.

Diagnosis[5,6]

Most useful diagnostic modality of neurocysticercosis is neuroimaging & MRI. MRI can also demonstrate all four stages- vesicular, colloid, granular & calcified stage of parenchymal neurocysticercosis. CT scan is better for identification of calcification. And MRI is superior in detecting basilar arachnoditis, intraventricular cyst, multiple cysts as well as those of spinal cysts. ELISA assay for detection of serum antibody is positive in almost half of the patient with single rings. CSF may also be examined for cells cysticercal antigens & PCR

a)



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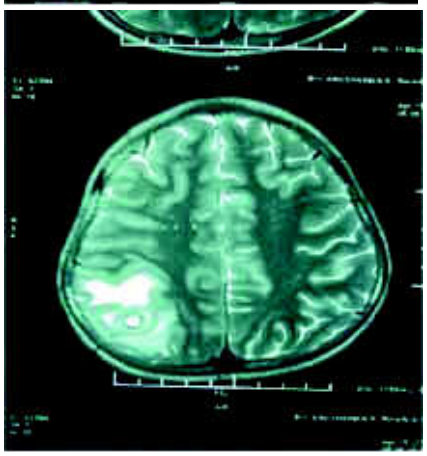

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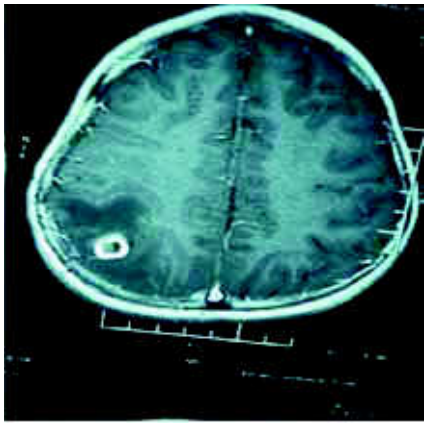

(a,b,c) MRI Brain suggestive of Ring enhancing lesion with calcified scolex noted in right parital region with mild surrounding edema S/O Neurocysticercosis (colloidal vesicular stage)

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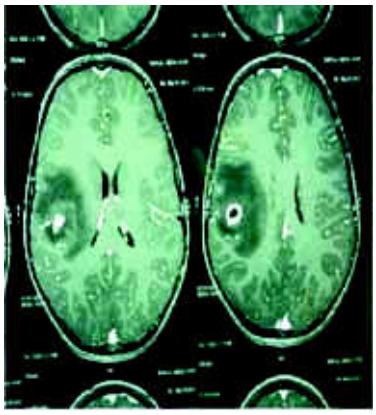
d) 

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Treatment[2,3,4]:

Symptomatic Management of seizures as anticonvulsant. Carmazepine or Ox-carbamazepine for partial seizures and sodium valproate for generalized and complex partial seizures.

Antiepileptic therapy : It may also be appropriate who are at high risk of seizures. Risk of seizures appear to be highest in multiple leision, particularly when the lesions are degenerating, and are surrounded by inflammation.

Calcified inactive lesion can also serve as a foci for seizures but in an otherwise asymptomatic patient are not generally considered an indication of prophylactic anti-epileptic drug therapy .

Optimal duration of AED therapy is uncertain. In general AED therapy may be appropriate in setting of calcified lesion as specially in those with recurrent seizures. Most of the experts favour administrating AED therapy for 6 to 12 months after radiographic resolution of active parasitic infection.

Anti-parasitic therapy : Though the natural history of parenchymal lesion is to resolve spontaneously with or without antiparasitic drug, cysticidal therapy is needed in case of multiple cysts and viable cysts. Perilesional edema in an MRI is indicative of non-viable cyst and does not demand a cysticidal therapy.

The potential benefit of antiparasitic therapy for treatment of neurocysticercosis is hastened resolution of active cyst, decrease risk for seizure and decrease recurrence of hydrocephalus.

Potential risk of treatment with antiparasitic therapy is exacerbation of neurological symptoms due to increase inflammation around the degenerating cyst, particularly in the patient with a large no of lesion. The inflammation can be so severe that it can lead disability or death.

Albendazol is aantiparasitic drug of choice (15mg/kg/day) orally to be given in 2 divided dose for 28 days. Maximum 800 mg/day. It can be taken with a fatty meal to improve the absorption.

Praziquantal is an alternative (50-100mg / kg / day) orally to be given in 2 divided dose for 28 days.

Corticosteroids for 2 to 3 days started before the therapy and continued thereafter can ameliorate this side effects. Dose of praziquantal should be increased if used with steroids as steroids decrease its level. In contrast, albendazol level is increased with concurrent use of steroids.

Follow-up

Patient with parenchymal and subarachnoid neurocysticercosis should undergone intermittent radiographic survellilance to evaluate further resolution of the cysticerci and development of calcification. Imaging may be performed 1 to 2 month and 6 month following the treatment. Imaging should be repeated prior to the discontinuation of antiepileptic drugs. New, worsening or persistent symptom should prompt evaluation.

Attention to personal hygiene, proper hand washing and avoidance of underwashed foods and raw vegetables in endemic zone for taeniasolium help prevent eggs and thus neurocysticercosis.

Conclusion

Seizures are the presenting findings in ~70% cases of parenchymal neurocysticercosis.

Most useful diagnostic study for parenchymal disease is MRI Head. MRI provides most information about cystic viability and associated inflammation.

References

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