INTRODUCTION

Since the advent of CT scan in India, a large number of patients with focal or generalized seizures are being investigated for structural cerebral lesions. A significant number (26%) of these patients are found to have single, small (< 20 mm; some earlier studies mention < 10 mm) enhancing CT lesion (SSECTL) with varying amount of surrounding edema. Various presumptive diagnoses such as tuberculoma, cysticercosis, sarcoidosis, larva migrans, transient viral encephalitis, microabscesses, post ictal enhancement and vascular lesions have been considered as differential of SSECTL. Although first reported as ‘microtuberculomas’, their spontaneous resolution without administration of anti tubercular treatment gave a clue to an alternate etiology. Subsequently, with histopathological correlation lesions revealed that these were various stages of neurocysticercosis.

HISTORY

SSECTL were initially noted on computed tomographic (CT) studies performed in Indian patients with seizures, in the late 1970s and early 1980s, as a solitary small enhancing lesion. For various reasons, it was identified as an “immature tuberculoma” or “microtuberculoma,” and patients with the lesion were treated with antituberculous therapy (ATT). In 1985, Sethi et al. found curiously that these lesions resolved spontaneously and labeled them “appearing and disappearing abnormalities.” The etiology of the single, small, enhancing CT lesion (SSECTL) was revealed only in the late 1980s from pathological studies performed on excised lesions in a series of studies. Later elucidation of various aspects of this lesion including its natural history has been carried out.

EPIDEMIOLOGY

It is difficult to estimate the community prevalence of SSECTL. The most common method has been to carry out imaging studies during epilepsy surveys in patients with history of focal seizures. Estimated community prevalence of SSECTL by this method has been found to be around 4%, to 34% in more recent studies as a cause of ongoing seizures. A case series of over 2,500 patients from a tertiary care centre in South India observed SSECTL as the provoking factor in 61% of patients with an isolated seizure.

Table I. Causes of single enhancing CT lesions

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<thead>
<tr>
<th>Common Causes of SSECTL</th>
<th>Uncommon Causes of SSECTL</th>
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<tr>
<td>Neurocysticercosis</td>
<td>Glioma</td>
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<td>Tuberculoma</td>
<td>Larva migrans</td>
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<td>Cryptic AVM</td>
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<td>Brain Abscess</td>
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<td>In Immunocompromised patients</td>
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<td>Toxoplasmosis</td>
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<td>CNS lymphoma</td>
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<td>Fungal granuloma</td>
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<td>Secondaries</td>
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<td>Sarcoïdosis</td>
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<td></td>
<td>Small infarct</td>
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<td>Focal encephalitis</td>
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Neurocysticercosis remains the most common cause of SSECTL. Although the pig is the usual intermediate host of the tapeworm Taenia solium, human cysticercosis occurs when the eggs, which are excreted in the faeces of an individual carrying the parasite, are ingested. Ingestion of infected pork only causes adult tapeworm infestation (taeniasis), because infected pork contains the larval cysts that develop into adult worms in human intestine, and does not contain the eggs that cause cysticercosis (Fig. 1). Transmission is thus thought to be by indirect means, such as by the ingestion of vegetables irrigated with water contaminated with human faeces and thus T solium eggs. Epidemiological evidence also suggests that the most common source of infective eggs is a symptom-free tapeworm carrier in the household.

In human brain parenchyma, the larval form of Taenia solium undergoes four stages of evolution, vesicular, colloidal, granular-nodular and calcification. The term, cysticercus granuloma is used broadly to parasites in the colloidal or the granular-nodular stages and these two stages are together considered as transitional or degenerative phase of the

**Fig.1: Lifecycle of neurocysticercosis**

![Life cycle of neurocysticercosis diagram](http://www.cdc.gov/ncidod/dpkh/index.htm)
CLINICAL FEATURES
The usual presentation of SSECTL is in the form of focal seizures. Raised intracranial tension is seen in a fifth of them. In an early study of 753 patients with neurocysticercosis, epilepsy was the most common presentation (52.4%) followed by headache (43.4%), papilloedema (28.0%), pyramidal tract signs (27.2%), intellectual deterioration (21.5%), ataxic gait (15.8%), diminution of visual acuity (10.0%), optic atrophy (10.0%) and uncommon presentations like psychotic episodes, diplopia, vertigo, dysmetria or intention tremor, lower cranial nerve palsy (VII to XII), disturbances of behavior, hypoesthesia, decreased hearing, spinal cord compression, meningeal irritation signs, radicular syndrome and parinaud’s syndrome were reported in lesser number of patients. Seizures may occur in up to 70% of patients, as reported in another series.

DIAGNOSIS
The diagnosis of SSECTL as neurocysticercosis or otherwise is difficult because clinical manifestations are nonspecific, most neuroimaging findings are not pathognomonic, and some serologic tests have low sensitivity and specificity. A set of diagnostic criteria was proposed for neurocysticercosis in 1996 and revised later (Table II). These criteria provide two degrees of diagnostic certainty:
- **Definitive diagnosis**, in patients who have one absolute criterion or in those who have two major plus one minor and one epidemiologic criteria;
- **Probable diagnosis**, in patients who have one major plus two minor criteria, in those who have one major plus one minor and one epidemiologic criteria, and in those who have three minor plus one epidemiologic criteria.

The most common neuroimaging examination done in endemic areas is CT. Although new CT machines have fairly good diagnostic sensitivity, some small lesions, especially those in the posterior fossa, close to the bone, or those inside the ventricles or basal cisterns, may be missed. MRI has better accuracy, although it may miss some small calcifications, and is more expensive and less available in areas where the disease is endemic.

Serological assays to detect specific antibodies have been used for decades with different results. Currently, most centres use an enzyme-linked immunoelectrotransfer blot (EITB) with purified glycoprotein antigens (western blot), which can be done in serum samples or in CSF. An advantage of EITB is that its sensitivity in serum samples is equal to or better than that in CSF samples. Although EITB has 100% specificity and an overall sensitivity of 98%, a major problem is that approximately 30% of patients with a single brain parasite may test negative.

MANAGEMENT
The questions that need to be answered in terms of treatment are:

1. **How long to give AEDs?** Rajshekhar et al. in their study of 185 patients found that AEDs can be safely withdrawn within 2-12 week of disappearance of lesion if there is no break through seizure, 2 or less seizures and no CT calcification. The spontaneous resolution of lesion occurs in 18.8% at 3 months, 36.4% at 6 months, and 62.5% at one year.

2. **Should we give cysticidal?** A recent Cochrane review found that for viable lesions in children, there were no trials and in adults, no difference was detected.

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**Table II. Diagnostic criterion for neurocysticercosis**

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<tr>
<th>Absolute</th>
<th>Major</th>
<th>Minor</th>
<th>Epidemiologic</th>
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<tr>
<td>Histologic demonstration of the parasite from biopsy of a brain or spinal cord lesion</td>
<td>Lesions highly suggestive of neurocysticercosis on neuroimaging studies</td>
<td>Lesions compatible with neurocysticercosis on neuroimaging studies</td>
<td>Evidence of a household contact with T. solium infection</td>
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<td>Cystic lesions showing the scolex on CT or MRI</td>
<td>Positive serum immunoblot for the detection of anticycstercical antibodies</td>
<td>Clinical manifestations suggestive of neurocysticercosis</td>
<td>Individuals coming from or living in an area where cysticercosis is endemic</td>
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<tr>
<td>Direct visualization of subretinal parasites by fundoscopic examination</td>
<td>Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel</td>
<td>Positive CSF ELISA for detection of anticycstercical antibodies or cystercical antigens</td>
<td>History of frequent travel to disease-endemic areas</td>
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<tr>
<td>Spontaneous resolution of small single enhancing lesions</td>
<td>Cysticercosis outside the central nervous system</td>
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for albendazole compared with no treatment for recurrence of seizures; but fewer participants who received albendazole had lesions at follow up (RR 0.56, 95% CI 0.45 to 0.70). For non-viable lesions in children, seizures recurrence was less common with albendazole when compared with no treatment (RR 0.49, 95% CI 0.32 to 0.75). There was no difference detected in the persistence of lesions at follow up. For non-viable lesions in adults, there were no trials. In trials including viable, non-viable or mixed lesions (in both children and adults), headaches were more common with albendazole alone (RR 9.49, 95% CI 1.40 to 64.45), but no difference was detected in one trial giving albendazole with corticosteroids.

3. **What should be the duration of treatment?** The Cochrane metaanalysis has shown that short (3 days or 8 days) versus long (more than 8 days) duration treatment does not make any difference in outcome 26.

4. **Which agent should be used?** Earlier trials have shown that albendazole and praziquantel had comparable efficacy 27 which was replicated in subsequent trials 28. However a recent study conducted in North India in pediatric patients showed that a combination therapy for albendazole and praziquantel was statistically comparable to sole therapy with albendazole in eradicating lesions and preventing seizures 29.

5. **What is the role of steroids?** Corticosteroids may help in alleviating symptoms related to SSECTL 30 but may not help in rapid resolution of solitary cysticercus granuloma 31.

**CONCLUSION**

SSECTL remains a challenge in terms of accurate identification and clear guidelines in terms of management, especially as they have varied symptoms. Diagnostic criteria do aid in a better diagnosis. The flowchart given below is proposed as a approach to a patient presenting with SSECTL and seizures (Fig.2).

![Flowchart](image)

**Fig.2: Approach to SSECTL**


32. CDC. page title here [Internet]. [cited 2010 Sep 23];Available from: http://www.cdc.gov/ncidod/dpd/parasites/cysticercosis/default.htm