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Epilepsy with Single Small CT Scan Enhancing Lesion in India-Do we know all the Answers Yet?

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Editorial

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Editorial

So do we know all the answers when it comes to epilepsy with single small CT enhancing lesion (SSCTEL)? The answer is an empathetic no. In 1985, we were the first to draw attention to these lesions in a publication titled "Appearing and disappearing CT abnormalities and seizures" [1]. Our small series of 11 patients aroused considerable interest nationally and internationally in these lesions. The patient reported were treated with no specific medicines except anticonvulsants and in all these, lesions disappeared spontaneously after a variable periods of 2-3 months. Prior to our publication, due to high incidence of tuberculosis in our population and response of these lesions to anti-tubercular therapy (ATT) it was widely believed that these lesions were tuberculomas. In absence of any biopsy in our series, by a process of exclusion, we thought that these lesions represented some sort of spontaneously resolving infection, peculiar to the Indian subcontinent and referred to it as a "focal encephalitis". In hind sight the choice of our word was inappropriate and a better term would have been "focal encephalitides". Following our publication others reported presumptive diagnoses of these lesions as tuberculoma, cysticercosis, sarcoidosis, larva migrans, transient viral encephalitis, microabscess, post ictal enhancement and even vascular lesions. Rajshekhar documented stereotactic biopsy findings in 6 such cases. In all cases the biopsy was reported as "non specific chronic inflammatory lesions" or "focal encephalitides" [2]. Five of these lesions were followed up with anti-convulsant therapy and in all of them the lesion reportedly disappeared in three months. The authors disagreed with policy regarding starting these patients on ATT preferring to treat only with anti-convulsants as advised by us. A subsequent paper published in 1991 which included stereotactic biopsy in some of the cases, it was concluded that in Indian epileptic patients with SSECTL, cysticercosis is the commonest etiology. A critical analysis of Chandy's and Rajshekhar's paper reveals that in a significant number of their patients, the lesion disappeared or turned into calcific dots with no specific therapy. The authors concluded that disappearing lesions are nothing but a manifestation of a "very benign form of neurocysticercosis" [3]. While the disappearance or death and calcification of these parasites in the brain is a natural event in the evolution of most types of benign cerebral cysticercosis, the process can take anywhere from 18 months to 10 years from the time of manifestation. The rapid resolution of the granulomas (disappearance in 4 to 6 weeks) in some patients and the long duration of symptoms in others is difficult to explain except by postulating that this is determined by the individual host immune status and the host-parasite reaction. A point to ponder here is that why in cases reported from the Indian subcontinent 60 to 80% of SSECT lesions disappear while in South America, where neurocysticercosis is rampant, disappearing lesions have not been commonly reported. How do we explain this paradox?

Currently in India when it comes to SSCTEL pendulum has swung from tuberculosis to cysticercosis. In-fact

SSCTEL lesion has become synonymous with cysticercosis. This is unfortunate since many things remain unexplained. No critical analysis of published case series has been carried out. In many cases biopsies were not done to confirm or refute the diagnosis of NCC. Neither was immunoblot assay for cysticercosis antibodies carried out. ELISA was done which has a poor sensitivity. Should we treat these patients with anticonvulsants alone or anticonvulsants along with albendazole? Which patients should be treated with anti-tubercular therapy (ATT)?

We want to stress that SSCTEL is not synonymous with cysticercosis. It is a mixed bag. Lesions suggestive of NCC on CT, in patient with compatible clinical picture residing in endemic areas are and should usually be diagnosed as NCC. Differentiating NCC from tuberculoma though remains a diagnostic challenge. On CT scan or MRI unless you have a scolex, etiology at best is an educated guess. Ancillary investigations such as ESR, Mantoux test (PPD), lymphadenopathy on X-ray or CT chest may help to identify a small group of patients. Serology is not usually helpful. In SSCTEL with negative initial ancillary investigation for tuberculosis the lesion in 60-80% may disappear with no other treatment except anticonvulsants. Either this is a non-specific infection or a very benign form of cysticercosis. Lesion may persist in 20-30%. In these cases we advise to repeat ancillary investigations. Of note some lesions may disappear further in 3-6 months. Albendazole may be tried. If lesion does not disappear or patient clinically worsens, ATT should be initiated. Prognosis is excellent in the large number of cases in which the lesion spontaneously disappears, needing no specific treatment other than AED and is thus aptly described as the "syndrome of disappearing CT lesions". Question though remains when to do stereotactic biopsy for definitive diagnosis.

The syndrome of seizure with SSCTEL is mainly confined to the Indian subcontinent, age group affected is mostly children or young adults and type of epilepsy is mostly focal (partial). Severity of epilepsy is benign responding usually to one AED. The diagnosis is retrospective and made when the lesion disappears and etiology is nonspecific infection vs. benign form of cysticercosis. When one encounters a case with seizure and SSCTEL, there is no way to predict whether the lesion will disappear on its own or not. Disappearing lesions do not require any other treatment except AED which can be stopped after 1 to 3 months of disappearance of lesion. There is no role of albendazole in such a lesion. Addition of steroids may theoretically reduce edema but exposes the patient to risk of having flare up of tuberculosis, if it turns out to be tuberculosis. Why some lesions do not disappear and persist is not known. Other questions remain unanswered. Is the etiology of disappearing lesion same as that of persistent lesion? Why some lesions heal clean, why others get calcified? In patients with seizure and calcified lesion how long should we administer AED? Do we operate on these lesions or not?

We conclude that the mystery of epilepsy with SSCTEL is far from solved, be it etiopathogenesis, diagnosis, treatment or prognosis. Unfortunately, interest in solving this mystery is already waning. We need large consecutive biopsy studies to answer some of these questions. This a problem unique to our country and one of commonest cause of symptomatic epilepsy in children and young adults. If we do not solve it, no one will. The battle is far from over, in fact it has just begun. We need to have reappearing interest in disappearing lesions.

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