"SMALL SINGLE ENHANCING LESION IN CT BRAIN - CLINICAL AND RADIOLOGICAL OUTCOME"

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"learn to heal"

CERTIFICATE

This is to certify that the dissertation entitled **"Small single enhancing lesion in CT Brain - Clinical and radiological outcome"** is the bonafide original work of **DR.C.T.Suresh** in partial fulfillment of the requirements for D.M. Branch-I (Neurology) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2008.The period of post-graduate study and training was from August 2005 to July 2008.

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DECLARATION

I solemnly declare that the dissertation titled **"Small single enhancing lesion in CT Brain - Clinical and radiological outcome"** is done by me at Institute of Neurology, Madras Medical College & Govt. General Hospital, Chennai, during 2005-2008 under the guidance and supervision of Professor of Neurology, **Dr.V.NATARAJAN, D.M Neurology.**

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of **D.M., Degree in Neurology.**

Place: Chennai
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CONTENTS

INTRODUCTION

Small single enhancing lesion in CT (SSECT)scan as an entity came into existence in India in early 1980, with the advent of CT scan. Tandon etal[3] labeled them as intracranial tuberculomas. They identified two types of lesions based on the appearance of the lesion in the C T Scan brain. Small ring and disc lesions were called as immature tuberculomas and large lobulated lesions were called as mature tuberculomas. ATT was invariably started in these patients. Sethi etal[5] & Bansal etal[6] noted that these lesions disappeared spontaneously without treatment. Natarajan and Arjundas etal[37] at the Institute of Neurology, Chennai also noted spontaneous disappearance of these lesions without specific treatment.

The exact cause of these lesions could not be deciphered because apart from tuberculoma, cysticercus granuloma, pyogenic abscess, metastases, fungal granuloma, and glioma had been reported to cause similar appearance in CT scan brain as more centers acquired C.T.Scan facility. Hence the exact aetiology of these lesions became a subject of speculation. A significant breakthrough came in 1987 at Vellore [8] when stereo tactic biopsy was done on 15 patients with SSECT. Most of the lesions were identified as cysticercus and none were of tuberculous etiology. Bhargava and Tandon in 1988 and Wadia, Makhalle in 1986[3,4], found evidence of tuberculous lesions in histopathology.

However they failed in defining the size of the lesion. Rajshekhar etal[8] then defined a small single lesion based on CT appearance as “A solitary, contrast-enhancing lesion of less than 20-mm diameter lesion without severe cerebral edema (no midline shift)”.this definition is often equated to cysticercous granuloma but there is every possibility that these lesions could be a tuberculoma.

There is no consensus in terms of aetiology of these small lesions. The common presentation of SSECT is seizures. The seizures are acute symptomatic in nature and the duration of antiepileptic treatment is once again controversial. Murthy etal and Thussu A etal [34] [35] reported that AED can be tapered and stopped if the lesion had resolved radiologically. In patients who had radiological resolution AED was given for 6 months and then tapered over 3 months. They also construed that lesions which resolved with calcification is a risk factor for seizure recurrence. Gupta Met al [38] compared short term, (6 months AED) with long duration, (2years AED) and they found no significant recurrence in seizures with both forms of therapy. Though lot of work has been done with regards to aetiology of single lesion there is no consensus regarding the duration of AED requirement.

The purpose of this study is to treat SSECT symptomatically and follow the natural course of the lesion over a period of one year in terms of its resolution and symptom improvement. The end points were clinical and radiological outcome.

REVIEW OF LITERATURE

DEFINITION OF SSECT

Amongst single contrast enhancing lesions on CT Scan Brain, those less than 20 mm were classified as small and termed “single small enhancing computed tomography lesion” (or SSECT). The criteria for SSECT included patients who had acute symptomatic focal seizures with or without secondary generalization, minimal or no neurological deficit, no evidence of raised intracranial tension and no evidence of systemic disease[4, 7, 9, 10].

A Thailand based study evaluated 1,000 patients with various seizure disorders, of whom approximately 10% had seizure and a solitary lesion. Roughly 80% of patients had lesions more than 20 mm and were excluded. On follow-up of these excluded patients (> 20 mm), only 1 had spontaneous resolution while the rest had persistent lesion on CT scan. Of the remaining 20% included in the study, 90% had complete resolution by 6 months. Of the two patients with persistent lesions, one underwent excision biopsy which revealed eosinophilic granuloma. The other patient refused biopsy, anti-tubercular treatment was prescribed and lesion disappeared in 4 months. This study concluded that a small lesion of less than 20 mm in size is a cysticercous granuloma, because the lesions were classified based on Rajshekhar’s clinico radiological definition for Cysticercus granuloma.

CAUSES OF SINGLE ENHANCING LESION IN CONTRAST CT SCAN:

COMMON:

Cysticercus granuloma.

Tuberculoma.

UNCOMMON:

Glioma

Secondary disease

Cryptic AVM

Brain abscess

Larva migrans

Sarcoidosis

Small infarct

Focal encephalitis

IN IMMUNOCOMPROMISED PATIENTS:

Toxoplasmosis

CNS lymphoma

Fungal granuloma.

However in Indian subcontinent the commonest cause for a small single enhancing lesion is either a tuberculoma or a cysticercoma. An attempt was made to differentiate between these two entities based on clinical and radiological features.[3,4,6,8.]

CLINICORADIOLOGICAL DEFINITION

Since SSECT is a clinico radiological entity, the diagnostic dilemma begins when either an asymptomatic or symptomatic patient (usually with focal seizures) shows a characteristic small (< 20 mm) lesion on CT which enhances with contrast. The initial differentiating features are accompanying radiological characteristics of the lesion.

The characteristic lesion on a plain CT of neurocysticercosis is a small high attenuation lesion surrounded by peri-lesional edema or only low attenuation lesion depicting focal edema.

With contrast the lesion may enhance as a ring, disc, or target lesion. The shape may be ovoid, doughnut-like or bi-lobed in some cases. There is no or minimal mass effect. There is no specific site of predilection. Some studies indicate a predilection to frontoparietal regions. However, most of these lesions are situated at the gray-white matter junction [2. 3, 5, 9.]

DIFFERENCE BETWEEN CYSTICERCOMA &TUBERCULOMA AND THE CONTROVERSIES

Tuberculomas are usually irregular, solid and greater than 20 mm in size. They

are often associated with severe perifocal edema and focal neurological deficit. Regarding the diagnostic criteria of solitary cysticercus granuloma given by Rajshekhar and Chandy[7,8] there are several controversies which need to be highlighted. No study has been done to evaluate the spontaneous disappearance of lesions greater than 20 mm till now. Moreover, no histopathological study has ever demonstrated that all large lesions (>20 mm) are tuberculomas. On the contrary, every retrospective and prospective follow-up study of single enhancing CT lesions in patients with new-onset seizures observed the spontaneous resolution of the lesions irrespective of their size, shape and amount of surrounding edema. In fact, several series which included patients with CT lesions of varied sizes, shapes and perifocal edema, identical favorable clinical and radiological courses were obtained.

Some studies showed, small single enhancing CT lesions were treated with antituberculous treatment with excellent results and this adds to the controversy of the etiology of single enhancing CT lesions. Even if a patient does not fulfill the diagnostic criteria given by Rajshekhar and Chandy [7,8], it does not comprehensively exclude the possibility of a cysticercus etiology, and vice versa. In addition to the features suggested by Rajshekhar and Chandy [7,8], several other differentiating imaging features have been suggested from time to time. For example, in these contrast enhancing lesions, "target lesions" (lesions with central nidus of calcification or a dot enhancement) are frequently encountered. Earlier, target lesions were considered a pathognomonic feature of CNS

tuberculoma[3]. Del Brutto et al [28,29] and some other authors reported that visualization of an enhancing or a calcified eccentric dot which represented the scolex, could be considered a definite imaging feature of cysticercus etiology, unfortunately histopathology evaluation of these target lesions is not available [7,8].

SSECT, criteria as proposed by Chandy et al [7,8] in 1991. All criteria must be satisfied for a diagnosis of SSECT.

The clinical criteria are:

1. Seizures (partial or generalized) should be the initial symptom.
2. There should be no features of persistently raised intracranial pressure.
3. There should be no history of a progressive neurological deficit.
4. There should be no evidence of an active systemic disease.

The radiological criteria are:

1. CT scan should only show a solitary, contrast enhancing lesion.
2. The lesion should measure less than 20 mm in maximal diameter.
3. Edema may or may not be present, but is not severe enough to produce a shift of the midline structures.

These clinico-radiological criteria were found highly sensitive and specific in predicting benign outcome [7 8].

ROLE OF BIOPSY

A major breakthrough in the understanding of these single CT-enhancing lesions came when Chandy, et al.,[7,8]reported obtaining CT-guided stereotactic biopsy samples. Histopathological examination of these brain tissue samples showed cysticercal granuloma in the majority of patients. In another study of 51 patients Rajshekhar, et al.,[7,8] documented cysticercal granulomas in 25 patients and tuberculoma in six cases in biopsy. Of the remaining 20 patients, 12 patients harbored parasitic granuloma (cysticercal lesion not definite), six patients nonspecific inflammation, and one patient each had dystrophic calcification and secondary metastasis. The authors concluded that the majority of single enhancing CT lesions are caused by neurocysticercosis. However, other diseases such as tuberculoma should always be considered in the differential diagnosis.

A study carried out by Chacko et al showed on histopathology,

1. A cavitary inflammatory lesion,
2. Presence of eosinophils in inflammatory infiltrate,
3. Absence of caseous necrosis, and
4. Absence of acid-fast bacilli (AFB) or fungal elements.

They also performed 5 micrometre deeper sections in 6 patients which showed calcified ovoid bodies, structurally similar to intracorporeal vacuoles, which also suggested the

possibility of cysticercosis.

These different studies vindicated the stand that a small enhancing lesion is most likely to be a cysticercous granuloma [7, 8.]

ROLE OF BIOCHEMICAL TESTS

Immunological tests for tuberculosis, neurocysticercosis, or toxoplasmosis have limitations as these organisms are seen even in normal individuals in most countries of the developing world. The tuberculin test is of poor diagnostic use as 40% of the adult population may be positive due to prior exposure. The results of conventional ELISA for cysticercus antibodies in serum and CSF of patients with SSECT have been generally disappointing (sensitivity of 50% and a specificity of 65%). Serological tests are useful if positive, but negative test results do not rule out the disease. False positive serology can result from previous infection. False negative serology can result because of immune tolerance, inactive disease, or localized antibody production in the CSF. The newer enzyme-linked immunoelectrotransfer blot (EITB) assays on serum or CSF using purified glycoprotein antigens from T. solium cysticerci have claimed much higher sensitivity and specificity of 98% and 100% for multiple active cysts and extraparenchymal NCC, but sensitivity is less in calcifications or single cysts, respectively in LatinAmerica[21,26].

If there is associated tubercular meningitis, raised CSF proteins, normal sugar and lymphocytosis may be seen. CSF findings in neurocysticercosis include mononuclear pleocytosis, normal glucose levels, elevated protein levels, high immunoglobulin G index, and in some cases presence of oligoclonal bands.

Sotelo et al [24,25] in a study of 753 cases observed that demonstration of eosinophils in CSF, though not pathognomic, was valuable indirect evidence of neurocysticercosis. When CSF was inflammatory, eosinophils were found in 57.7% of cases. Far less specific, but still another frequent finding was hypoglycorrhachia detected in 17.7% of the cases with inflammatory CSF. The above findings were absent in non-inflammatory CSF.

However, if the disease is limited to the brain parenchyma, CSF examination may be normal, as is expected in the majority. CSF in effect may just say whether there is inflammatory or a noninflammatory lesion in the brain and does not aid in the diagnosis [24, 25].

LIFE CYCLE

Taeniasis and cysticercosis remain a global public health problem in both the

developing and developed countries. Infection is becoming increasingly common in the latter because of the increasing immigration and more frequent travel to regions of endemic disease. These parasitic diseases are related to poverty and poor sanitary infrastructure. Therefore, cysticercosis has been designated as a biological marker of the social and economic development of a community.

BIOLOGY

Humans are the only known host to harbor the adult cestode parasite, Taenia solium, in the intestine. Infection is acquired by ingesting undercooked pork infected with Taenia larvae (ie, cysticerci). The cysticerci evaginate into the intestines where they mature into adult worms. The worms consist of a scolex, which attaches itself to the intestinal wall, and numerous proglottids (ie, segments). Proglottids and eggs are shed intermittently into the stool.

The intermediate host, typically the pig, is infected by ingesting parasite eggs or proglottids containing eggs (ie, porcine cysticercosis). The oncospheres escape from the eggs, penetrate the intestinal mucosa, migrate through the bloodstream, and lodge in the

tissues. Over weeks to months, they evolve into larvae that enlarge and mature into cysticerci. The life cycle is completed when humans ingest pork contaminated with the cysts.

Human cysticercosis is acquired after eating food contaminated with fertilized eggs excreted in the feces of Taenia carriers. In humans, the most common routes of infection are ingestion of T solium eggs from contaminated food and rarely from fecal-oral autoinfestation from patients harboring the adult parasite in their intestines. While the cysts can develop in any human tissue, they have a predilection for the central nervous system (CNS), skeletal muscle, subcutaneous tissue, and eyes [9].

IMMUNE RESPONSE

In humans and pigs, the cysticerci may live within the host tissue without causing inflammation or disease. The immune response is unpredictable and may vary from a complete tolerance to an intense immune response. A single patient may show an intense inflammation around a cyst at any stage of the degeneration process, together with viable cysts with lack of inflammation and several calcifications scattered in the brain.

Autopsies of victims of warfare and road/traffic accidents have revealed that a large proportion of NC infection is asymptomatic and discovered incidentally at necropsy. Individuals who undergo computed tomography (CT) of the head for unrelated reasons (eg, head injury) may demonstrate multiple parenchymal calcifications.

Several studies have analyzed the mechanisms of the immune response elicited against T solium cysticercus, such as the heterogeneity of the humoral immune response, the existence of immune evasive mechanisms, and the fact that the immune response can both protect and harm the host.

The humoral immune response to antigens of T solium cysticerci is evident from the number of immunodiagnostic assays that have been developed using different types of antigens. Several immunoglobulin (Ig) classes are produced as specific antibodies against the parasite. The most frequent is immunoglobulin G (IgG), which can be detected in serum, CSF, and saliva and suggests that infection is of long duration. The immune response against T solium cysticerci appears to have components of both T helper type 1 cells (Th1) and T helper type 2 cells (Th2), although the underlying mechanisms are yet to be clarified. The parasite is probably killed by eosinophils, which

are attracted to the site by lymphoid cells. It is assumed that this specific response is mediated by Th2 cytokines [16, 21, 26].

PATHOPHYSIOLOGY

The natural history of cysticerci in the CNS is not entirely understood. CT scan and magnetic resonance imaging (MRI) have been useful in the study of the evolution of the cysticercus within the brain parenchyma. MRI is more useful than CT scan in detecting intraventricular and subarachnoidal cysts, as well as the accompanying signs of cyst degeneration and pericystic inflammatory reaction. However, CT scan is preferred for detection of parenchymal calcifications. Once the oncosphere has passed into the parenchyma, it grows and evolves through vesicular, colloidal, nodular-granular, and calcified phases.

After entering into the brain parenchyma, the parasite develops into a "vesicular stage" in which the cysts are viable and elicit very little inflammatory response in the surrounding brain tissue. On CT scanning viable cysticercal lesions appear as rounded, circumscribed, hypodense lesions, and contrast enhancement is absent. After a variable

period of time the parasite loses its viability either because of aging, inability of larva to become adult, or immunological factors particular to the host, and it enters into the next stage [9].

The second phase is the “colloidal stage” in which inflammatory changes develop in the cyst wall and surrounding brain parenchyma. Transparent cystic fluid is replaced by jellylike whitish material, which is surrounded by a fibrous capsule. This dying stage of larva is referred to as cysticercal granuloma. In this stage, CT scanning demonstrates a ring-enhancing lesion. Progressive reduction in the size of the cyst and scolex and mineralization of cystic fluid lead to development of a “granular-nodular” stage in which the larva appears as a disc-enhancing lesion on contrast-enhanced CT.

In the last "calcific stage" the lesion becomes completely mineralized and appears as a hyperdense calcified nodule on plain CT scanning. At this stage there is no contrast enhancement and surrounding edema is also absent because of abatement of inflammation [9].

CLINICAL FEATURES

Symptoms of nervous system involvement depend upon the (1) site of the cyst (2)

& number of the larva or (3) State of the lesion activity & (4) host immune response.

Common presentations in chronological order of incidence is given below,

Seizure

Headache

Papilloedema

Pyramidal tract signs

Intellectual deterioration

Ataxic gait

Diminution of visual acuity

Optic atrophy

Psychotic episodes

Diplopia

Vertigo

Dysmetria or intention tremor

Lower cranial nerve palsy (VII to XII)

Disturbances of behavior

Hypoaesthesia

Decreased hearing

Spinal cord compression

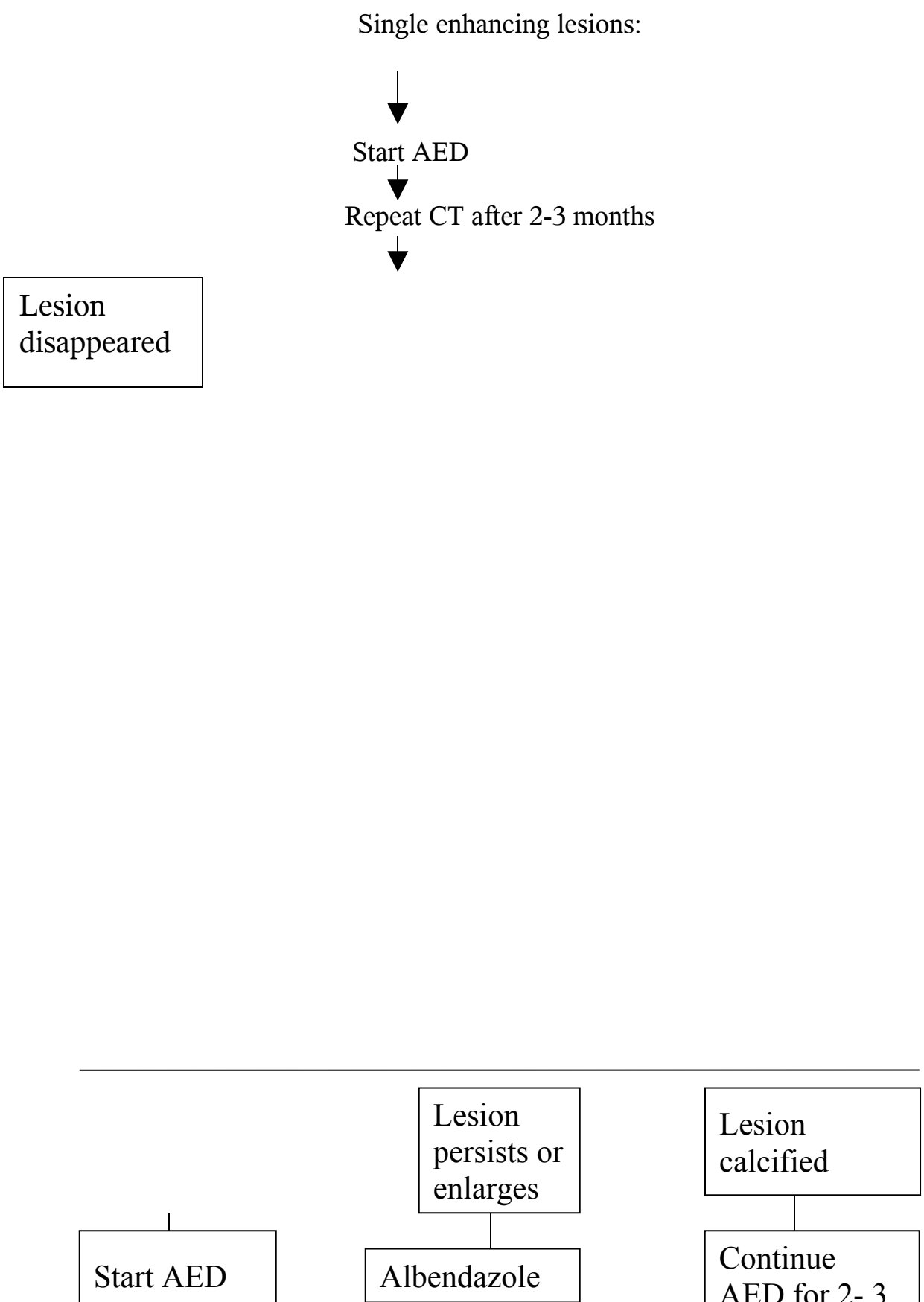
Meningeal irritation signs

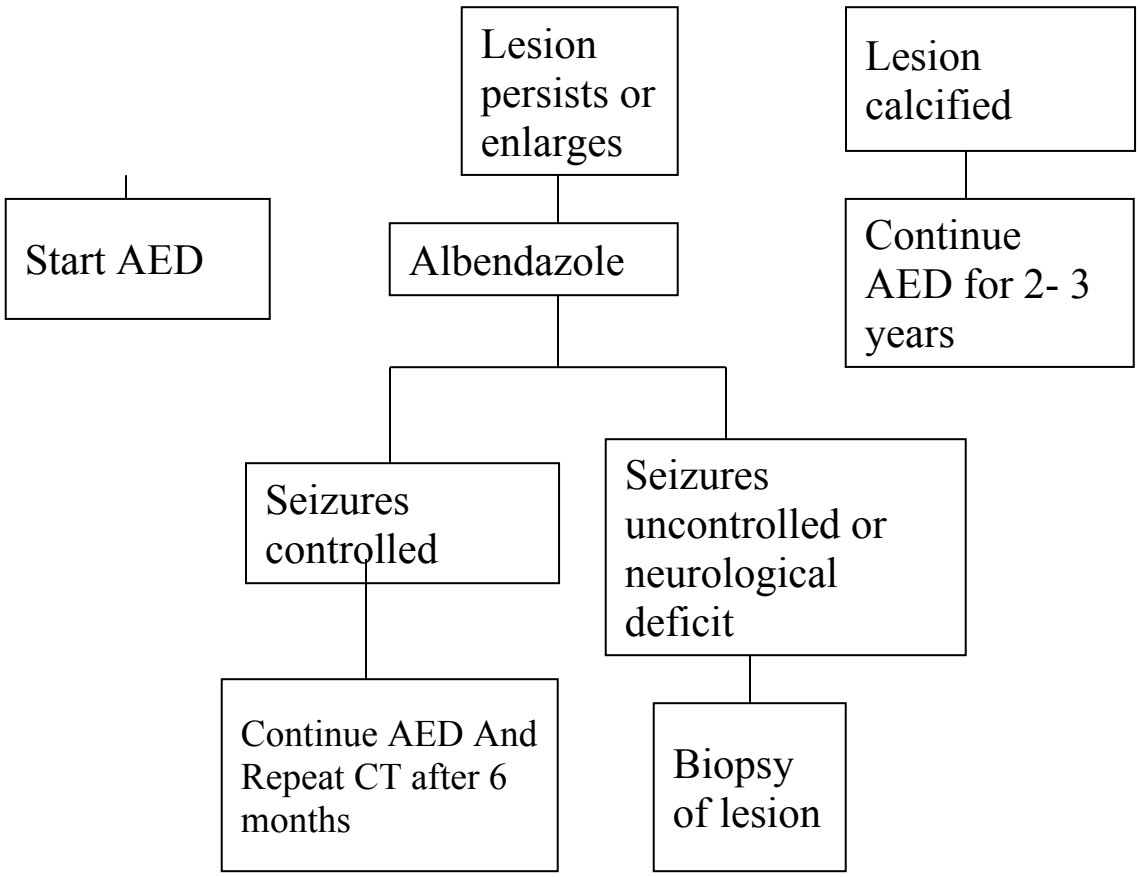
Radicular syndrome

Parinaud’s syndrome.

Epilepsy and headache formed 95% of the clinical manifestations and epilepsy accounted for 52.4% [1 2 4 6 7]

TO TREAT OR NOT TO TREAT





The algorithm says that a single lesion need not be treated with cysticidal drugs [10] [11] [18]. However in a study conducted by Albendazole therapy for single small enhancing CT lesion (SSECT) in brain in epilepsy A Thussu, etal [1],the conclusion drawn was patients presenting with seizures due to single parenchymal NCC, albendazole hastens resolution of SSECT if treatment is given in the early phase of illness [1].

The question then arises should a single lesion be treated with albendazole or

praziquantel or not at all. The question of treating or not treating a single lesion arose because of the radiological resolution which occurred spontaneously. The most remarkable feature of single enhancing lesions observed on CT scanning is their complete spontaneous disappearance in the majority of patients, as well as their occasional significant reduction in size in others. The edema surrounding the lesion is usually the first to resolve. Later, the lesion may disappear completely, leaving no residue, or it may leave a tiny speck of calcification at the former site of the lesion. In some patients the granulomatous lesion transforms into a calcified nodule. Controversy exists regarding the exact time that these CT-enhancing lesions disappear. In several retrospective studies the authors have suggested that spontaneous resolution may vary from as early as 6 weeks to as late as 75 weeks. Even greater persistence has been observed. Various authors have noted a wide variation in the rates of complete resolution. The rate of disappearance has ranged from 22 to 100% at 12 weeks after detection on the first CT scan. Addressing this same issue, Rajshekhar [7,8,] 32 in a prospective study of 210 patients, observed that single enhancing lesions completely resolved at different time intervals. At 3 months only 19% of lesion had completely resolved; at 1 year approximately 63% had disappeared. In another prospective study, which supports majority opinion, Singh, et al.,[4 6] observed that approximately 73% of similar-appearing lesions had disappeared within 2 months of their first CT documentation.

In Latin American countries, CT-depicted single enhancing lesions are invariably treated either with albendazole or praziquantel. In an uncontrolled study Del Brutto [28,29] observed early resolution of lesions on CT scans following treatment with albendazole. More recently, in a controlled study, Pretell, et al.,[28] included 26 patients with single enhancing lesions. The patients were openly assigned to receive either single-day praziquantel therapy (three doses of 25 mg/kg at 2-hour intervals) or no treatment. In praziquantel-treated patients, complete resolution occurred in 11 and partial resolution in two, in the remaining patient the lesion was later diagnosed as AVM. Conversely, the lesions persisted unchanged in six of 12 patients in the non treatment group. The authors favored routine administration of anticysticercal drugs in patients with single enhancing lesions. Although this single-day praziquantel therapy has been found particularly useful for single lesions, poor response has been noted in those with multiple cysticercal lesions [11 12 13 14 17].

In India, studies involving anticysticercal treatment have provided conflicting results. In a placebo-controlled study Padma, et al., observed that 7-day treatment with albendazole did not hasten the resolution of CT-documented lesions [18]. In a different double-blind placebo controlled study, however, Baranwal, et al., observed a significantly faster and higher incidence of complete disappearance of lesions in children who underwent 28-day albendazole treatment (15mg/kg/day). The conflicting results of these two studies also fueled the controversy of the ideal dosage regimen of

albendazole. A comparative study is needed to evaluate 7- or 8-day albendazole treatment in a 30-day course in patients with single CT-enhancing lesion. In patients with other forms of NCC, Cruz, et al, [14] have already demonstrated that 8 day albendazole treatment is as effective as 15- or 30-day therapy. These authors concluded that there is no benefit to extending albendazole treatment beyond 7 or 8 days [11 12 13 14 17].

PRAZIQUANTEL OR ALBENDAZOLE

As demonstrated by experiments in animals, praziquantel and albendazole are both effective antiparasitic drugs against T. solium cysticerci. The issue is which drug is better, or how long is it to be given, and at what dosage it has to be taken. Initial studies began treatment with praziquantel with low doses like 5-10 mg/kg/day for 2 weeks, while later studies uniformly adopted 50 mg/kg/day for 2 weeks. Some studies even used a single dose praziquantel treatment, and there was no difference in the rate of disappearance of the cysts. When Albendazole became available, no dose ranging studies were performed for it and the dose used in Hydatid cyst (15 mg/kg/day) was also used for cysticercosis. The advantages of albendazole over praziquantel are that the former is orally available as a once daily dose, has a better penetration into the CSF, its concentration is not affected when given with steroids, and it is cheaper as compared to the latter [11 12 13 14 17].

PROS AND CONS FOR ATT/ANTI NCC TREATMENT

1) Rapid disappearance of cysts – no conclusive evidence in better epilepsy control.
2) Severe cases seen less frequently now – may be not due to cysticidals but due to improved sanitations and fewer mass infections.
3) Series of albendazole or praziquantel treated patients have better resolution (fewer seizures) than untreated patients seen at the same centres - inadequate control groups in initial studies.
4) Fewer residual calcifications - No evidence that anti-cysticercal therapy results in fewer calcifications.

PROS AND CONS FOR SYMPTOMATIC TREATMENT

1) NCC becomes symptomatic after a period of years as a result of onset of process of death of parasite. – Questionable methodology as some patients persist with symptoms and live with cysts for years.
2) Anti-cysticercal therapy leads to acute cerebral Inflammation and this therapy is thus severe and unnecessary. Inflammation can be controlled with steroids; chronic inflammation, and moderate inflammation may lead to scars similar to or worse than those from a short, acute, severe process.
3) Adverse reaction to treatment [27].

DURATION OF ANTIEPILEPTIC DRUGS IN SMALL SINGLE ENHANCING LESIONS IN CT BRAIN

The duration of antiepileptic drug therapy in patients with Small single enhancing lesion in CT brain has not been clearly worked out. SSECT is the commonest cause of acute symptomatic seizures.

Thussu etal [35] compared short term (6months) antiepileptic treatment with long term antiepileptic treatment (2 years) in two groups of patients. In both the groups patients who had resolution in CT brain at 3 months had no further seizures. They also found that calcified lesions were associated with seizure recurrence.

Among 34 patients with calcifications in both the groups, 11 patients had recurrent seizures. Among these 11 patients 8 patients belonged to short term AED group while 3 patients belonged to long term AED group. The difference between both the groups did not attain statistical significance. They concluded that calcified lesions have a high frequency of seizure recurrence and the duration of AED treatment in these cases is conjectural at present.

The adequate duration of treatment in such cases is an individual prerogative of the treating physician[35]. Murthy et al expressed similar views in a retrospective analysis of 102 patients with small single enhancing lesions.[34] Antiepileptic drugs are the principal therapy for seizures in SSECT. In general, seizures should be managed in a manner similar to the management of acute symptomatic seizure. However, after resolution of the lesion with normalization of imaging studies, most patients who are seizure-free can eventually discontinue antiepileptic drugs. Antiparasitic drugs should not be regarded as an alternative for antiepileptic drug therapy [36].

AIMS AND OBJECTIVES

1) To study the natural course of SSECT without giving cysticidal or anti tuberculous treatment and give only symptomatic treatment as needed.

2) To observe the seizure outcome in patients with SSECT, duration of persistence or subsidence of symptoms and to find out the duration of symptomatic treatment needed.

3) To follow the patients with SSECT radiologically at 3months,6 months and 1 year to assess the size, morphology, occurrence of calcification and resolution characteristics in CT brain. Identifying the aetiology was not part of this study as tissue study by biopsy is the only conclusive test for a definitive diagnosis.

MATERIALS AND METHODS

The study was done at the Institute of Neurology Government General Hospital, Chennai. The study had collaborations with the Institutes of Internal Medicine, Biochemistry, Radiology and Microbiology attached to government general hospital.

This study was observational in nature designed to analyze patients in age group more than 12 years of age and who presented with seizures and CT brain showing SSECT. The sample size was 26 and the study period was from Dec 2005 to Jan 2008,including the follow up.

Inclusion criteria:

1. All patients who have SSECT lesions in the brain, detected by CT scan.

2. The patients should not have other neurological problems.

Exclusion criteria:

1. Patients who have neurological problems other than due to the small single enhancing lesion of the brain.

2. More than 2 lesions on CT brain.

3. Patients age less than 12 years and pregnant women.

METHOD OF THE STUDY

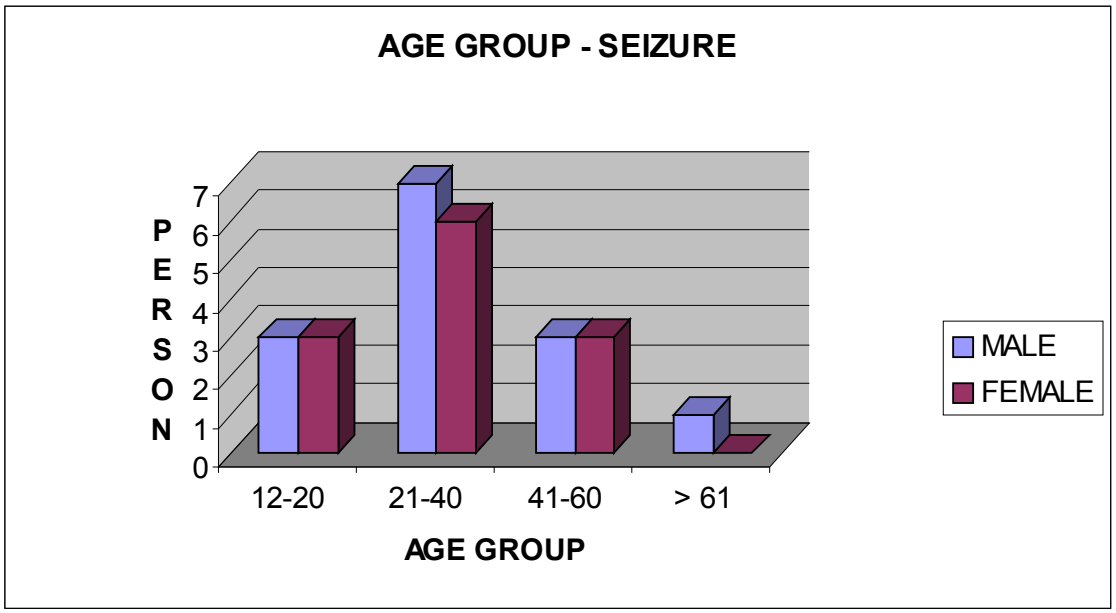
The clinical diagnosis on the seizure type, whether partial or generalized was made. A detailed history was taken and recorded. Significant past medical history if any were noted. A thorough clinical examination was performed at the time of admission and relevant findings were recorded. Laboratory work up, which included blood sugar, urea, serum creatinine, electrolytes and liver function tests (if indicated), were done at the time of enrollment into the study .Patients with CT brain showing single enhancing lesion were enrolled in the study after taking consent. Consent was taken after explaining the therepeutical aspects.

They were followed for 1 year with respect to seizure control and CT evolution of the lesion. All patients were treated with antiepileptic drugs(AED). Steroids at 1mg per kg body weight were added to patients who had lesions with oedema. It was tapered over a period of 15 days given. Follow up CT scans with contrast was done at the end of 3rd month, 6th month and 1 year if required. The lesions were followed in terms of regression, persistence, enlargement, resolution and calcification.

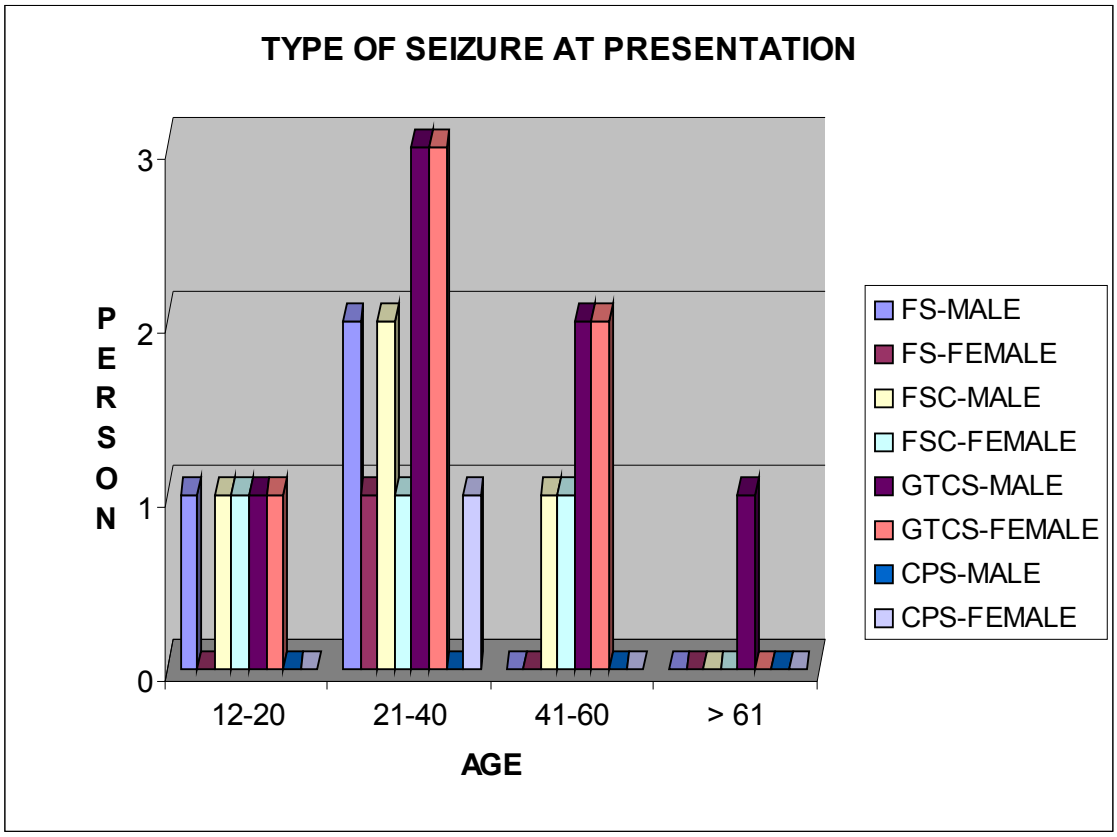
RESULTS

SEIZURE TYPE - AGE DISTRIBUTION

TOTAL NUMBER OF PATIENTS - 25



TYPE OF SEIZURE AT THE TIME OF PRESENTATION



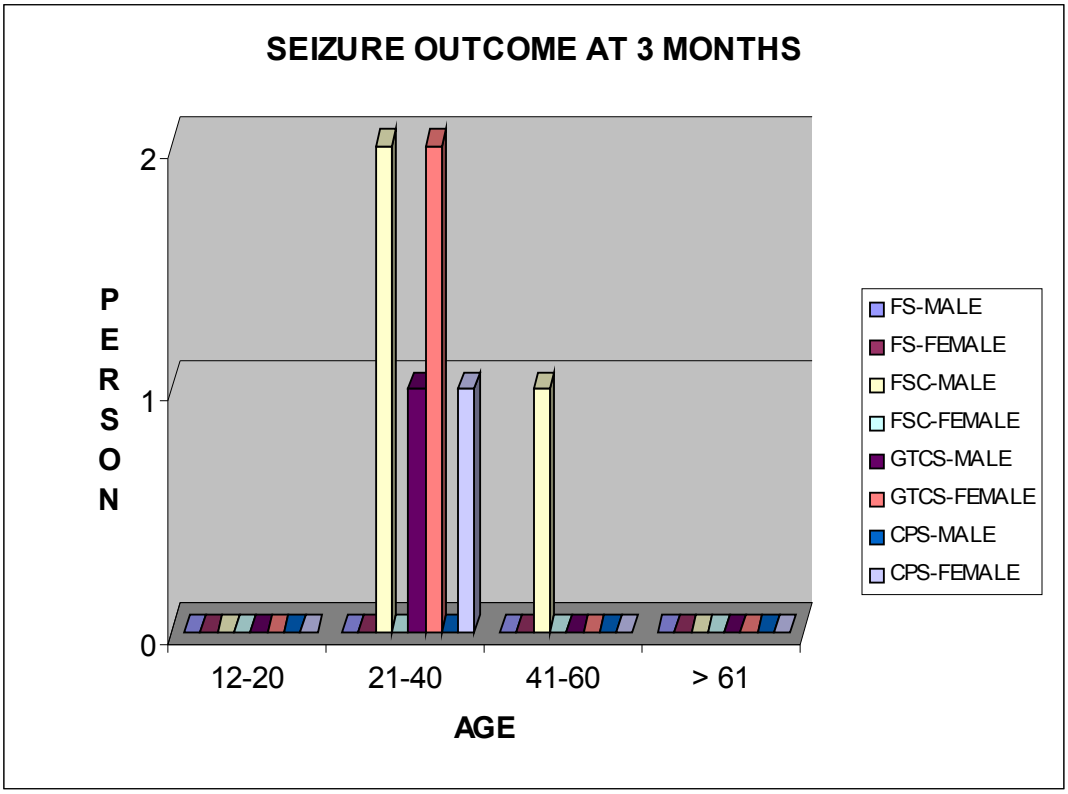
FS : Focal Seizure

FSC : Focal Seizure with Secondary Generalisation

GTCS : Generalised Tonic Clonic Seziure

CPS : Complex Partial Seizure

SEIZURE OUTCOME AT THREE MONTHS



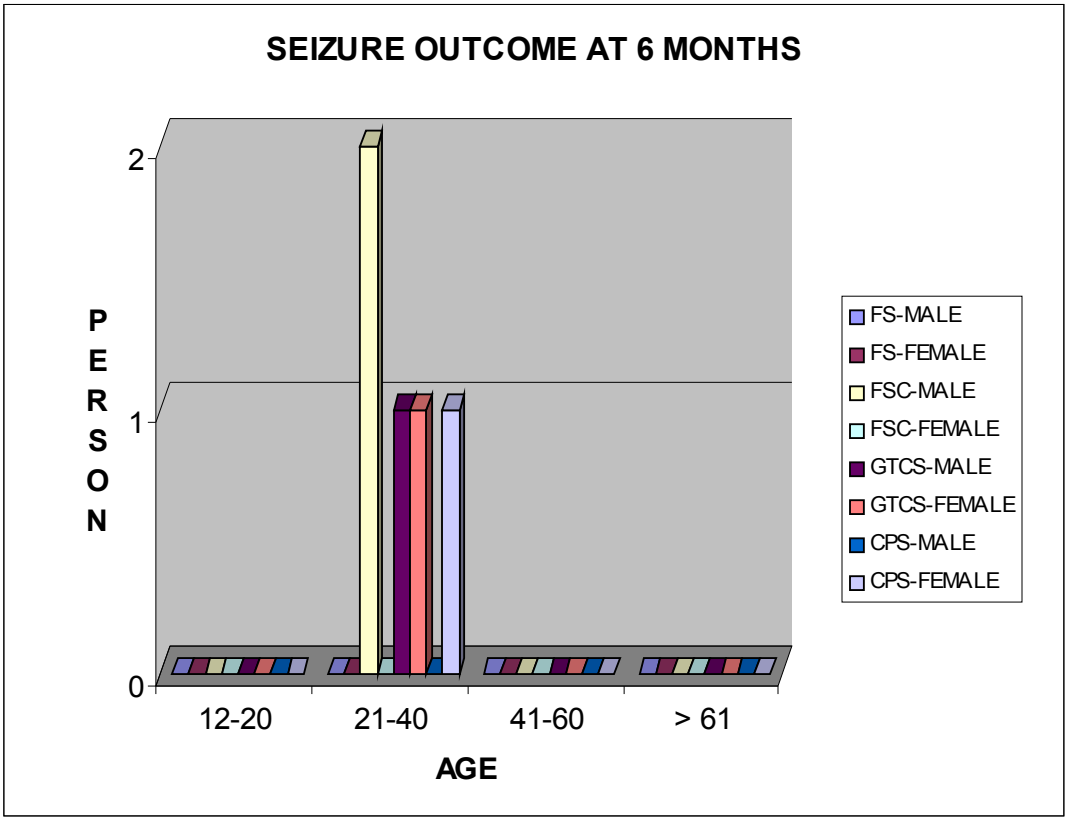
FS : Focal Seizure

FSC : Focal Seizure with Secondary Generalisation

GTCS : Generalised Tonic Clonic Seziure

CPS : Complex Partial Seizure

SEIZURE OUTCOME AT SIX MONTHS



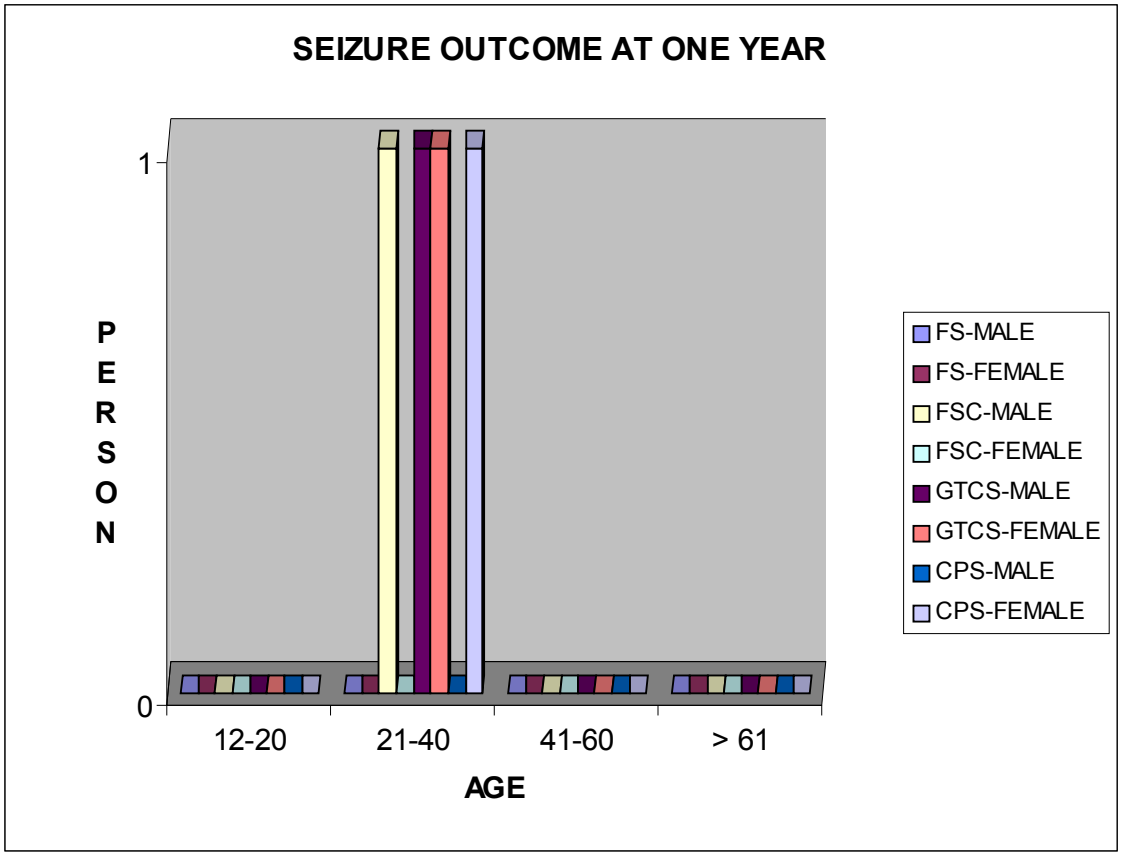
FS : Focal Seizure

FSC : Focal Seizure with Secondary Generalisation

GTCS : Generalised Tonic Clonic Seziure

CPS : Complex Partial Seizure

SEIZURE OUTCOME AT 1 YEAR



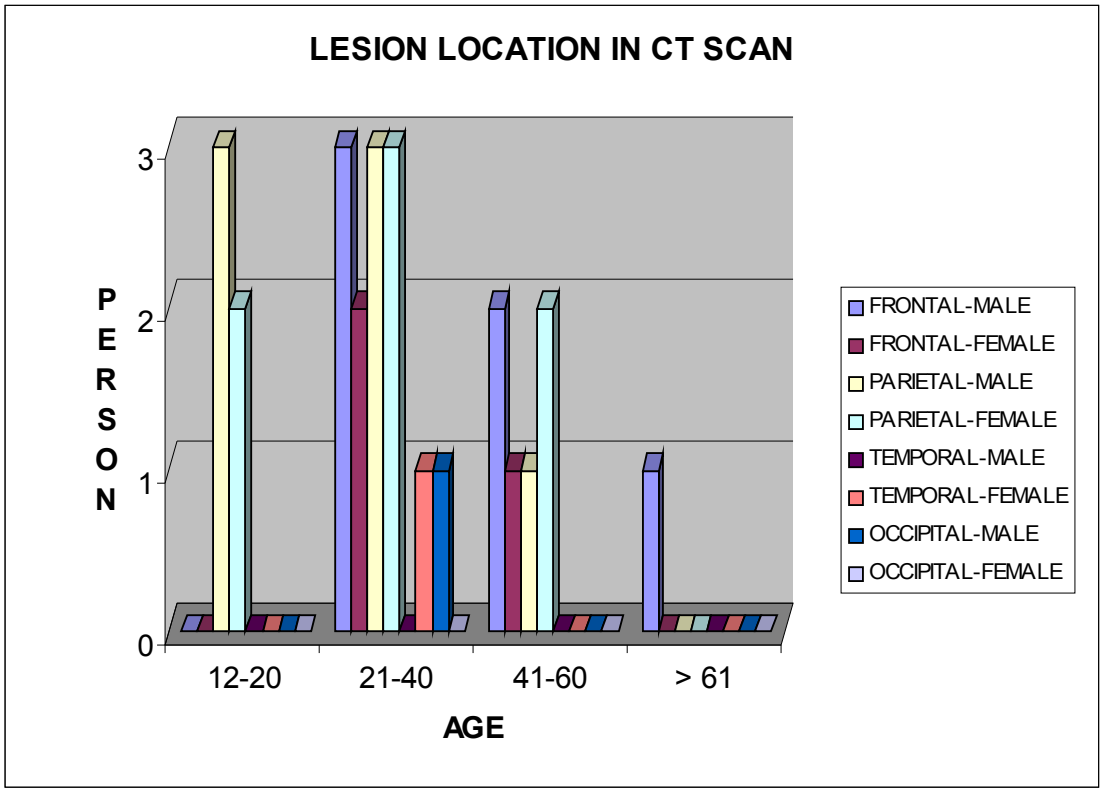
FS : Focal Seizure

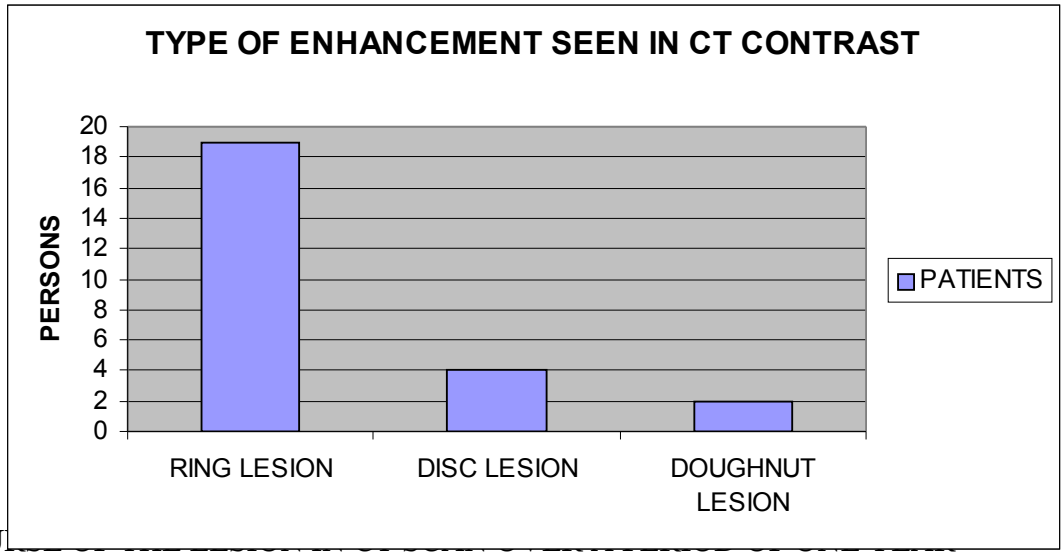
FSC : Focal Seizure with Secondary Generalisation

GTCS : Generalised Tonic Clonic Seziure

CPS : Complex Partial Seizure

LESION LOCATION IN CT SCAN – PLAIN AND CONTRAST





COU

	COMPLETE RESOLUTION	INCREASE IN SIZE	DECREASE IN SIZE	CALCIFICATION	NEW LESIONS

AT 3 MONTHS	12	2	8	3	1 (excluded from study)
AT 6 MONTHS	4	0	2	4	
AT ONE YEAR	1	0	0	1	

COMPLETE RESOLUTION WITHOUT CALCIFICATION - 17

RESOLUTION WITH CALCIFICATION - 8

DISCUSSION

The study included 25 patients of different age and sex. Sex ratio was almost even (men-14–56% women 11 –44%). The youngest patient was 13, and the oldest was 65 years old.

The analysis of the age groups showed that the incidence of SSECT is more common among the younger age group. As the age advances the incidence decreases. This data is concordant with other studies [1 2 4 6].

SEIZURE SEMIOLOGY AND SEIZURE OUTCOME

Among the 25 patients encountered in this study, 13(52%) patients presented with generalized tonic clonic convulsions and 7(28%) presented with focal seizure with secondary generalization. 3 patients presented with complex partial seizure with secondary generalization, while two patients presented with focal seizures. Several studies implicated focal seizures with secondary generalization as a common presentation of SSECT. A Thussu, A Chattopadhyay, IM S Sawhney in their study of albendazole as monotherapy in SSECT also noted focal seizure with secondary generalization as the commonest presentation [1].

In some studies GTCS was the commonest presentation. Venkataraman et al[19], in their study found GTCS as a commonest presentation. In our study GTCS was the common presentation. 4 patients presented with cluster seizures and 3 patients presented with status. All these patients had significant edema in CT brain. The occurrence of cluster seizures and status could be attributed to the presence of vasogenic edema, which by itself is due to the immunological reaction of the dying parasite and break down of the blood brain barrier.

Patients were treated as acute symptomatic seizures. Steroids were added to patients who had significant oedema in CT. All the patients were followed for 1 year.14 (56%) patients became seizure free by 3 months. Among these 14 patients ,12 patients had complete resolution of their lesion in CT scan.11 patients were having seizures. Among these patients 3 had calcifications, 2 patients had an increased size while the others had decreased size of the lesion. Seizure semiology did not change in any of the patients who continued to have seizures. At 6 months follow up only 5 patients had seizures. All these patients had calcifications in C.T. scan. Patients who had their sizes increased in CT at 3 months decreased in size when compared to the initial CT scan taken at the time of seizure occurrence for the first time. At 1 year only 5 patients continued to have seizures. Their seizure semiology were the same as before. AED's were continued in these patients.

In summary predominant presentation in our patients was GTCS followed by focal seizures with secondary generalization. Seizures are acute symptomatic in nature and most of the patients had adequate seizure control. Recurrent seizures were common in calcified lesion. All calcifications did not cause seizures. Why some patients with calcifications cause seizures while others remain asymptomatic is a matter of debate?. Recent evidence suggest that mild inflammation may persist in the calcified stage in some cases, and may be associated with an increased risk for ongoing seizures [33].

There are several hypotheses that might explain this apparent relapse of disease. Cerebral edema may have resulted from the death of parasites that were viable and incompletely calcified. Alternatively, antigen release from several dead and involuted, calcified parasites might have produced cerebral edema. However, it is difficult to understand why numerous parasites would have died or released antigen simultaneously. A more plausible hypothesis is that a spontaneous increase in immunity, possibly triggered by the release of antigen from a live or dying parasite, led to an intense immune response to antigen at the site of a calcified lesion.

Alternatively, another unrelated event, the immunologic reaction, might have led to a generalized enhancement of cellular immunity, which in turn was directed against

cysticercal antigen associated with several dead calcified lesions. This upgrading of immunity would be analogous to the treatment-induced or spontaneous enhancement of cell-mediated immunity associated with the reversal reaction of leprosy. This phenomenon might help to explain why several calcified lesions might simultaneously develop a surrounding inflammatory response associated with edema. These are several hypothesis which are given for seizures occurring in a calcified lesion, by Tarang N.et al, in their case report of reactivation of neurocysticercosis [33].

Another feature which was noted was the presence of headache in patients with calcification. Among the patients who had calcification in their CT developed headache which was had the characteristics of migrainous headache without aura. Headache started after 2-4 months of the first episode of seizure in 6 patients and the headaches occur at a frequency of 2-4 episodes per month. All these patients underwent EEG testing to rule out a ictal headache. All the EEG's were normal. Headaches occurred only in patients with calcifications. The presence of headache could be the calcified lesion acting as a trigger for the trigemino thalamic pain pathway by irritating the arachnoid granulations or dural sinus which are pain sensitive. Another plausible hypothesis is headache occurring as an ictal phenomenon, but EEG was obtained in our patients were normal. However these recordings were interictal recordings. The occurrence of headache is discussed by Dr.Garg etal and Delbrutto etal [10,11,28,29].

RADIOLOGICAL FEATURES

16 out of 25 patients had SSECT in parietal cortex, while 7 had in frontal lobe, 1 each in occipital and temporal lobe.

The predeliction for parietal lobe for SSECT corresponds to most other studies [4,8,10].Among the 25 patients 12 patients had scolex in their initial CT while 4 patients had central dot sign. When contrast was given the enhancement pattern of the lesions were 19 had ring enhancement,4 had disc enhancement and 2 patients had dough nut enhancement. The presence of disc enhancement and doughnut enhancement corresponds to nodular granular stage of neurocysticercosis. The different patterns of enhancement is similar to what was noticed by Chandy etal [7,8] and their pathological correlation is similar to the study by Escobar etal [9]. All patients had a complete ring enhancement on contrast examination and their sizes ranged from 10-20 mm in size. The presence of central dot sign which was initially thought to be a pointer to tuberculoma but it does occur in metastasis as well as neurocysticercosis.

At 3 months, 12 (48%) patients had complete resolution, 3(12%) patients had calcifications in the corresponding areas while two patients(8%) showed increase in size while in the other patients sizes had reduced. Resolution by calcification is a well known occurrence in SSECT and could be considered as natural evolution of the lesion. Why some lesions disappear while some calcify, depends on the immunity of the

patient.

The exact reason is not known. Rajshekhar [7,8], in a prospective study of 210 patients, observed that single enhancing lesions completely resolved at different time intervals. At 3 months only 19% of the lesion had completely resolved; at 1 year approximately 63% had disappeared. In another prospective study, which fits with the majority opinion, Singh, et al.,[10] observed that approximately 73% of similar-appearing lesions had disappeared within 2 months of their first CT documentation. Because of these conflicting observations, it is very difficult to recommend guidelines concerning the need and timing of obtaining follow-up CT scans in these patients. Hence controversy exists regarding the exact time that these CT-enhancing lesions disappear. In several retrospective studies the authors have suggested that spontaneous resolution may vary from as early as 6 weeks to as late as 75 weeks [9]. Even greater persistence has been observed. Various authors have noted a wide variation in the rates of complete resolution.

Two patients had size of lesion increased at 3 months and we had one patient who at 3 months developed multiple ring enhancing lesions in CT and hence excluded from the study. Paradoxical response is well known in tuberculomas and it is a hypersensitivity reaction to ATT. But in our patients ATT was not started and still 1 patient had this response. Infact it could not be called a paradoxical enlargement. Two

patients who had increase in size of lesion where still continued only on AED's and were followed up.

Singh and associates reported two patients in whom even enlarged CT-evidenced lesions resolved spontaneously. Rajshekhar and Chandy [7,8] postulated two mechanisms for the enlargement of the cysticercus granulomas. One was simply an increasing amount of inflammatory reaction giving rise to an increase in the size of the ring enhancing lesion as well as the surrounding oedema. The second mechanism involved two cysticercus cysts located in proximity to each other, the degeneration of one following the other very closely.

Rajshekhar and Chandy, based on their experience of 3 patients, suggested that the enlarging cysticercus granulomas may be conservatively managed with a trial of albendazole therapy. On the contrary, Singh etal believe that albendazole therapy was the cause of enlargement of the CT lesions. In our patients, at 6 months their sizes decreased and both calcified at 1 year. Paradoxical enlargement of the lesion in our patients was noted even without treatment. The reason could be the patient became symptomatic when the cyst was evolving in its size. This finding is important because some lesions which are bigger than 20mm in size could be cysticercosis and some lesions which are less than 20mm could be tuberculomas and the clinico radiological classification is just a clinical pointer and each patient may behave differently and it

depends upon the relationship between the host and the cyst/tubercle bacilli. In effect in our two patients we had not treated them with albendazole initially or even when they had enlargement at 3 months. On follow up they calcified. The exact reason for the enlargement is still debatable and that it can occur even without treatment gives credence to the postulations made by Rajasekhar etal [7,8.]

The common denominator in all patients with SSECT was almost all the lesions either resolved without any residua in CT or resolved with calcification. Between calcification and complete resolution by resorption complete resolution was more common rather than calcification, in our study. This conforms with most other studies which have been done previously.

Until recently, if single enhancing lesions did not disappear or regress within a reasonable time period (usually within 6 months), they were viewed with suspicion and often alternative diagnoses such as tuberculoma, pyogenic abscesses, or metastatic lesions were considered. Some authors have contended that "persistence" of lesions indicates that more aggressive treatment or brain biopsy sampling may be necessary. Currently it is very difficult to set a cutoff period after which these lesions may be termed persisting. In a recent prospective follow-up study, Rajshekhar [7,8] noted that the longer the follow-up period the higher the number of cases in which spontaneous disappearance of the granuloma occurred. He observed that at 6 months in only 19% of

210 patients had complete resolution occurred whereas at the end of 1 year and 2 years, respectively, in approximately 63 and 89% of patients CT scans revealed normal findings. Garg and Nag [10 11] also reported similar observations in a retrospective study. They observed that in 16 of 101 patients the lesions did not disappear or regress after 6 months. Additional follow-up scans in these 16 patients, however, revealed that the lesion eventually calcified in four patients, the ring lesions changed to disc lesion and degree of associated edema was considerably less in four, and the lesion persisted unchanged in the remaining eight.

Although concern is often expressed, in none of the prospective and retrospective follow-up studies has either clinical deterioration or significant enlargement of lesions been noted. In several uncontrolled series, albendazole therapy has been shown to produce complete resolution of persisting lesions. In our study lesions which had persisted and which showed paradoxical enlargement either disappeared or calcified by one year in all these patients. Hence a longer follow up is essential even in patients who have single enhancing lesions.

There are conflicting views about the role of anticysticercal drugs in the management of single enhancing CT lesions. Prospective randomized studies on the role of steroid and albendazole are at best rare. Our study clearly suggests that therapy is not necessary for single enhancing CT lesions. Seizure recurrences were infrequent. However a large sample size study may be required to substantiate this study.

CONCLUSIONS

1) By three months most of the SSECT resolve without calcification and by 1 year all lesions resolve either by resolution or calcification.

2) Enlargement of a lesion may not be always paradoxical and it can be a natural course in the evolution of the lesion.

3) SSECT present as generalized tonic clonic convulsions or focal seizure with secondary generalization.

4) Seizure caused by SSECT are well controlled and most of them don't have seizures beyond 3 months.

5) Patients who have seizures are usually secondary to calcified lesions.

6) Specific treatment in terms of anticysticidal or ATT is unnecessary as all lesions have better seizure control and radiological resolution when followed for 1 year.

7) Steroids might be useful in patients who have lesion with significant oedema.

8) Headache is a feature of calcified lesion.

9) The commonest location of SSECT in brain is parietal cortex.

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ABBREVIATIONS AND ACRONYMS

- SSECT : Small Single Enhancing Lesion in CT
- EEG : Electro Encephalo Gram.
- CSF : Cerebro Spinal Fluid.
- CT : Computerized Tomogram.
- MRI : Magnetic Resonance Imaging.
- GTCS : Generalized Tonic-Clonic Seizure
- FS : Focal seizure
- FSGS : Focal seizure with secondary generalisation.
- CPS : Complex partial seizure.
- AED : Antiepileptic Drug

MADRAS INSTITUTE OF NEUROLOGY

PATIENT PROFORMA FOR SSECT LESION OF THE BRAIN, CLINICAL AND RADIOLOGICAL OUTCOME

NAME AGE SEX IP.,NO., MIN.,NO.,

ADDRESS

CLINICAL HISTORY

SYMPTOMS.

HEADACHE

SEIZURES:

GTCS :

SPS.

CPS.

FOCAL WITH SECONDARY GENERALISATION:

OTHER NEUROLOGICAL DEFICITS:

PAST AND PERSONAL HISTORY:

FAMILY HISTORY

ANY OTHER DETAILS

CLINICAL FINDINGS

PROBABLE DIAGNOSIS

INVESTIGATIONS

HAEMOGRAM, URINE STOOL.

MANTOUX, SPUTUM FOR AFB CHEST XRAY.

CT SCAN PLAIN

CONTRAST

EEG

OTHER INVESTIGATIONS

FOLLOW UP

SYMPTOMS.

HEADACHE

SEIZURES

NEUROLOGICAL DEFICITS

SYMPTOM ANALYSIS

REPEAT CT SCANS AT 3 MONTHS AND 6 MONTHS

MASTER CHART

Sl.No.	Name	Age	Sex	AT THE TIME OF PRESENTATION		
				Type of Seizure	Site of Lesion	Type of enhancement
1	Karthik	14	m	SPS	PARIETAL	RING

2	Senthamarai	49	f	GTCS	PARIETAL	RING
3	Vinodh	16	m	GTCS	FRONTAL	RING
4	Prasadh	32	m	GTCS	PARIETALRING	
5	Durai	62	M	GTCS	FRONTAL	RING
6	Kani	48	f	FSGS	PARIETAL	DISC
7	Nandhini	46	f	GTCS	PARIETALRING	
8	Ashwini	34	f	GTCS	PARIETAL	RING
9	Chellamal	36	f	GTCS	FRONTAL	RING
10	Kannan	15	m	FSGS	PARIETAL	RING
11	Selvam	33	m	FSGS	PARIETAL	DISC
12	Kanmani	19	f	GTCS	FRONTAL	RING
13	Suresh	28	m	SPS	PARIETAL	DISC
14	Sherin	18	f	FSGS	PARIETAL	RING
15	Vijaya	22	f	CPS	TEMPORAL	RING
16	Yasodha	16	f	GTCS	PARIETAL	RING
17	Prabhu	26	m	GTCS	FRONTAL	RING
18	Chandran	48	m	FSGS	PARIETAL	DOUGHNUT
19	Megathoth	38	m	GTCS	OCCIPITL	DISC
20	Sundar	51	m	GTCS	FRONTAL	RING
21	Kalaiarasan	30	m	FSGS	FRONTAL	RING
22	Vetriselvi	33	f	GTCS	PARIETAL	RING
23	Kalai	48	f	FSGS	PARIETAL	DOUGHNUT
24	Gnanam	52	m	GTCS	FRONTAL	RING
25	Sankar	36	m	SPS	FRONTAL	RING

Sl.No.	Name	THREE MONTHS		SIX MONTHS	
		Type of Seizure	Radiological Outcome	Type of Seizure	Radiological Outcome
1	Karthik		Resolution		
2	Senthamarai		Decreased		Resolution
3	Vinodh		Resolution		

4	Prasadh		Decreased		Resolution
5	Durai		Resolution		
6	Kani		Decreased		Calcified
7	Nandhini		Resolution		
8	Ashwini	GTCS	Increased	GTCS	Decreased
9	Chellamal	GTCS	Decreased		Resolution
10	Kannan		Resolution		
11	Selvam	FSGS	Decreased	FSGS	Calcified
12	Kanmani		Resolution		
13	Suresh		Decreased		Resolved
14	Sherin		Resolution		Calcified
15	Vijaya	CPS	Calcified	CPS	
16	Yasodha		Resolution		
17	Prabhu	GTCS	Calcified	GTCS	
18	Chandran	FSGS	Decreased		Calcified
19	Megathoth		Resolution		
20	Sundar		Decreased		Resolution
21	Kalaiarasan	FSGS	Increased	FSGS	Decreased
22	Vetriselvi		Resolution		
23	Kalai	GTCS	Calcified		
24	Gnanam		Resolution		
25	Sankar		Calcified		

Sl.No.	Name	ONE YEAR		FINAL OUTCOME IN CT
		Type of Seizure	Radiological Outcome	
1	Karthik		Resolution	
2	Senthamarai		Resolution	
3	Vinodh		Resolution	
4	Prasadh		Resolution	
5	Durai		Resolution	
6	Kani		Calcified	
7	Nandhini		Resolution	
8	Ashwini	GTCS	Calcified	Calcified
9	Chellamal		Resolution	
10	Kannan		Resolution	
11	Selvam	FSGS		Calcified
12	Kanmani			Resolution
13	Suresh			Resolution
14	Sherin			Calcified
15	Vijaya	CPS		Calcified
16	Yasodha			Resolution
17	Prabhu	GTCS		Calcified
18	Chandran			Calcified
19	Megathoth			Resolution
20	Sundar			Resolution
21	Kalaiarasan		Resolution	Resolution
22	Vetriselvi			Resolution
23	Kalai			Resolution
24	Gnanam			Resolution
25	Sankar			Calcified