DISSERTATION

ON

"A STUDY ON GRANULOMA OF BRAIN"

Submitted in partial fulfilment of

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D.M. NEUROLOGY (BRANCH-I)

Of THE TAMILNADU Dr. M.G.R. MEDICAL

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CERTIFICATE

This is to certify that this dissertation entitled **"A study on Granuloma of Brain"** submitted by **Dr. G. BALAJI** appearing for **D.M. Neurology** Degree (Branch - I) examination in **August 2010** is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai.

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I solemnly declare that the dissertation titled **"A study on Granuloma of Brain" is** done by me at the Institute of Neurology, Madras Medical College & Govt. General Hospital, Chennai, during **2009-2010** under the guidance and supervision of Prof.R.M**.** Bhoopathy.

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CONTENTS

INTRODUCTION

Single ring enhancing cystic lesions may occur in several infectious and neoplastic diseases of the central nervous system and are the most common radiological abnormality seen in patients with acute-onset seizures in India and many other developing countries. Histopathological studies in India and in other countries revealed that neurocysticercosis (NCC) is the most likely cause of these lesions provided they fulfill a rigid set of clinical and radiological criteria. A single degenerating cyst is the most frequent finding associated with NCC in the Indian subcontinent[30].Single cysticercus granuloma measures less than 20 mm in diameter, may be associated with cerebral edema not severe enough to produce midline shift, and occur in patients with seizures, normal neurological status, and no evidence of active systemic disease. When these lesions resolve spontaneously, either disappearing or changing into a calcified nodule, the diagnosis of NCC is very likely. The second most common cause of these CT-detected lesions is tuberculoma; in patients with these lesions similar clinical and neuroimaging features are also present.

In a developing country like ours where both tuberculosis and Cysticercosis are common, it is difficult to differentiate between tuberculoma and a single cysticercal granuloma. The most interesting feature of these

solitary enhancing lesions is their spontaneous disappearance within weeks or months. Some lesions "heal" by becoming calcified. These patients require only antiepileptic therapy, and this medication may be withdrawn safely after the lesion has resolved on CT scanning. In several studies provision of anticysticercal drugs has been attempted, but because of conflicting results, their role in the management of these single lesions is uncertain.

Magnetization transfer, a new technique for improving image contrast in magnetic resonance (MR) imaging, is based on application of off-resonance radio-frequency pulses and observing their effects on MR images, as well as measuring the signal intensity with and without application of the pulses (i.e., magnetization transfer ratio. MTRs can be used to detect changes in the structural status of brain parenchyma that may or may not be visible with standard MR techniques.

AIM OF THE STUDY

To study the presentation and natural course of single ring enhancing Neurocysticercosis.

To assess the efficacy of Magnetization Transfer Imaging in differentiating Neurocysticercosis from Tuberculoma.

REVIEW OF LITERATURE

Single enhancing lesions visualized on CT scanning are the most common radiological abnormality in Indian patients with new-onset seizures[17]. In 1980, Tandon and Bhargava[48] first reported these lesions; at that time these CT-enhancing lesions were presumed to be tuberculoma and often were treated with empirical antituberculous drugs.

Subsequently, histopathological studies of brain tissue biopsy samples have suggested that, in majority, single CT-enhancing lesions represent dying cysticercal lesions (larval stage of tape-worm *Taenia solium).* [39] These single CT-enhancing lesions often resolve spontaneously.[17] Considerable controversy persists about their cause and appropriate management.

Epidemiology:

The precise incidence and prevalence of single enhancing CT-documented lesions, in India and other parts of the world, are not known. All data available in India are from hospital-based studies. Wadia, et al.,[50] studied 150 patients with simple partial seizures, and CT scanning revealed single enhancing

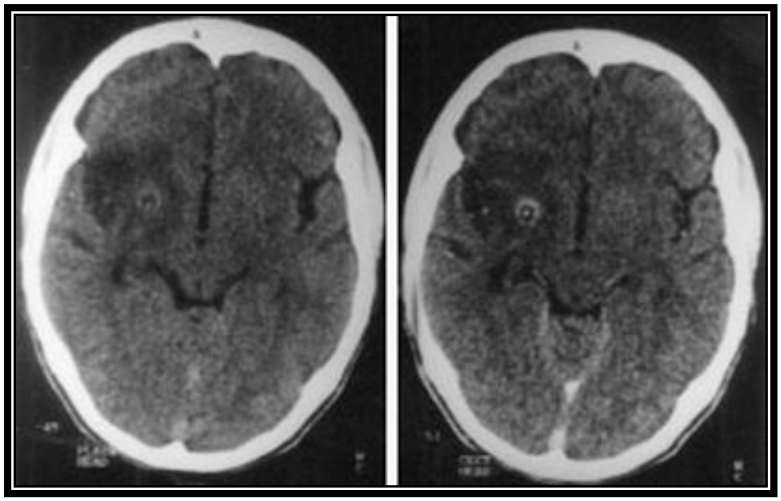
lesions in approximately 26%. The incidence of these lesions was higher among children; 40% of patients were younger than 15 years of age. Misra, et al.,[24]from North India, studied 1023 patients with partial seizures and obtained plain and contrast-enhanced CT scans in all. A single enhancing CT-demonstrated lesion was found in approximately 50% of patients (513 cases).[24] In a study from South India Murthy et al.,[26] reported these CT lesions in 23.4% of 2531 patients with all types of epilepsies. Garg and Nag[16] observed a higher incidence (72%) of single enhancing lesions in children and adolescents when CT scanning was performed after the first seizure.

Although initially it was thought that these lesions are found only in individuals from the Indian subcontinent, similar lesions were later reported from other parts of the world. Single enhancing lesions are frequently seen in Latin American countries and are invariably treated as cysticercal granuloma.[8] These lesions have also been reported in studies conducted in several developed countries such as Australia, the United States, and the United Kingdom.[20,23,47]Wadley, et al.,[51] recently reported a series of six patients from United Kingdom with single enhancing lesions. In all six patients the referral diagnoses were either tumor or tuberculoma. In the majority, the lesions had spontaneously disappeared. These authors concluded

that patients harboring single enhancing CT-documented lesions, even in nonendemic regions, need careful observation because their lesions may disappear spontaneously and patients may be saved from unnecessary neurosurgery.

Radiological Features:

The characteristic ring or disc-enhancing CT lesions are seen after intravenous contrast administration. Plain CT scans, at times, demonstrate some abnormality. The most common abnormality observed on plain scans is irregular low attenuation areas of vasogenic cerebral edema. Infrequently, a tiny speck of calcification is demonstrated within the area of hypodensity. Using newer-generation CT machines, it is possible to see the outline of complete cystic lesion with an extramural nodule.[13] Plain CT scanning may not reveal any abnormality.[6,15,17]

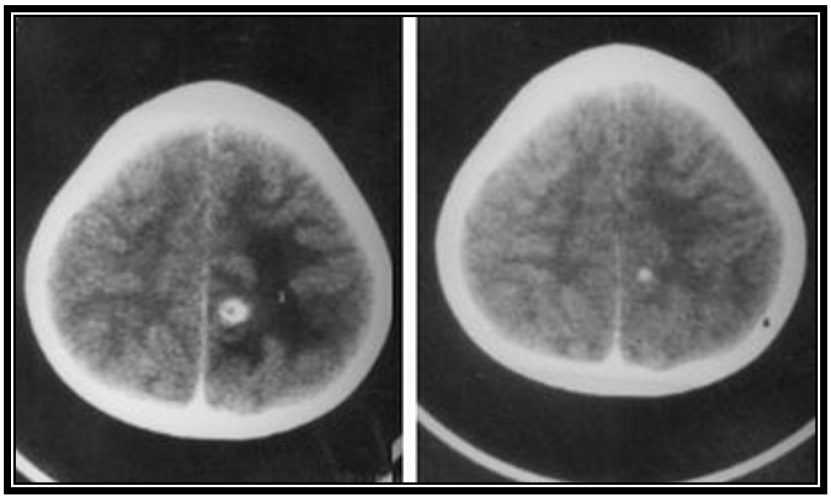


After contrast administration, there is a ring or a homogeneous disclike enhancement within the region of hypodensity. The enhancing lesions

observed on these CT scans are usually less than 20 mm in diameter; they are surrounded by a varying amount of perifocal edema. Occasionally, the edema may be severe enough to produce midline shift. An enhancing or a calcified eccentric dot (presumed to be a scolex of cysticercal larva) can be seen within the ring lesion (above Fig.). Single CT-documented lesions can be seen throughout the cerebral hemispheres, more commonly situated superficially in the gray matter or at the junction of gray matter and white matter. Parietal lobes are the most common location for these lesions. Frontal and occipital lobes are the other frequent sites.[6,15,17,31]

Radiological disappearance of Enhancing lesions:

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.[17,31] (above Fig.)

Etiopathogenesis:

Since these lesions were first described, their precise origin has remained controversial. Several origins have been postulated, from time to time, to explain the cause of these single enhancing lesions.

Common

Neurocysticercosis

Tuberculoma

Uncommon

Glioma

Secondaries

Cryptic AVM

Brain abscess

Sarcoidosis

In immunocompromised

Toxoplasmosis

CNS lymphoma

Fungal granuloma

Postictal Phenomenon

Until recently one of the major points of controversy was whether these lesions were cause or effect of the associated seizure disorder. Several biochemical and physiological changes occur at the site of abnormal neural activity during or following partial seizure. It was thought that the changes noted in neurons, blood vessels, and the blood-brain barrier had resulted in ring- or disclike enhancement on CT scanning.[18,19]

Intercranial Tuberculoma

In initial reports, several authors determined that single enhancing lesions were tuberculoma, based on several presumptions. Intracranial tuberculoma were common intracranial space-occupying lesions in this part of the world. Tuberculoma and tuberculous abscess constituted approximately 10 to 20% of all intracranial masses. Similar lesions documented on CT scanning were often seen in association with tuberculous meningitis. A favorable response to empirical antituberculous treatment was observed. Finally, tuberculosis is a highly endemic disease in India.[21,48]

Neurocysticercal Cysts

A major breakthrough in the understanding of these single CT-enhancing lesions came when Chandy, et al.,[5] 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.,[39] documented cysticercal granulomas in 25 patients and tuberculoma in six; in all cases these findings were confirmed. 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 NCC; however, other diseases such as tuberculoma should always be considered in the differential diagnosis.

Other Diseases Causing Single Enhancing Lesions

Various other presumptive diagnoses such as glioma, larva-migrans infection, microabscess, secondary metastasis, small infarct, focal encephalitis, and cryptic AVM have been suggested but none is consistently demonstrated in histopathological studies.[17,31]

Pathological charecteristics:

A parenchymal cysticercal cyst of the brain passes through four stages of natural evolution.

After entering in to 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.

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.[11]

Rajshekhar, et al.,[34] have attempted to demonstrate the presence of a viable parasite within the single CT-enhancing lesions. They performed histopathological examination of 43 brain tissue biopsy samples and were successful in demonstrating cystic lesions containing parts of an intact or degenerated larva in 22 patients. Inflammatory cavitary lesions, in which there was no definite evidence of parasite, were observed in 13 patients, whereas noncavitary hyalinized fibrous nodules with inflammation were found in the remaining eight. Of the 22 specimens in which there was definite evidence of cysticercosis, only lesions in two patients were shown to have an entire parasite within the granulomatous lesion. It was not possible to predict the presence of an intact parasite within the granuloma on the basis of clinical and radiological features.

In a more recent article, Chacko, et al.,[4] reported that, in a few patients, when no intact parasite or parasitic parts were observed within a granuloma, it was possible to demonstrate the presence of small ovoid masses corresponding morphologically to the intracorporeal vacuoles of a cysticercal larva. These structures were found to lie within the cavitary space of granuloma. The authors concluded that even the presence of calcareous residues of parasites may be the only evidence of cysticercal origin in some of the granulomas.

Clinical Features:

The patients in whom CT scanning reveals single enhancing lesions usually present with new-onset seizures. The seizures are often partial (motor > sensory) with or without secondary generalization. If a lesion is located in the occipital lobe, seizure is often preceded by visual aura, and in frontal lobe lesions an "adversive attack" is frequently observed. Cases of complex partial seizures are rare. Few patients present with partial status epilepticus. Several episodes of seizure occur in clusters, within a span of 2 to 3 days. Infrequently, Todd paralysis, which resolves within 24 hours, is observed.[15,35,50]Another major complaint in patients with single enhancing lesions is headache. A large number of patients presenting with seizure experience persistent or episodic headache during the interictal period.[15,17] In

some patients, severe episodic headache may be the only presenting complaint. In these patients the headache can be of such severe intensity so as to mimic that of a subarachnoid or an intracerebral hemorrhage. The fundus is usually found to be normal. Infrequently, the headache may be part of a frank increase in ICP. Patients experience headache, vomiting and papilledema. In this variety of headache the enhancing lesions have been noted in subcortical locations. Headache and other signs of raised ICP improve after spontaneous resolution of the lesion. Oral corticosteroid medication helps to relieve headache in the acute stage.[14] Patients in whom CT scanning demonstrates a single enhancing lesion in the brainstem invariably suffer headache and exhibit other brainstem signs.[22] Focal neurological deficits in the absence of seizures are rare and, at times, may be the sole manifestation.[14]

Diagnosis:- Cysticercal Granuloma and Tuberculoma

The distinction between cysticercal granuloma and tuberculoma is controversial, often associated with single enhancing CT-documented lesions. This is because the clinical and imaging features are quite similar; both diseases are common in endemic areas and may coexist in the same patient.

Rajshekhar, et al.,[39] have attempted to differentiate between these two entities on the basis of clinical and imaging features. Based on these findings

and their experience, Rajshekhar and Chandy[38]suggested that cysticerci are usually round in shape, 20 mm or smaller in size, with ring enhancement or a visible scolex; cerebral edema severe enough to produce midline shift or focal neurological deficit is not seen. Tuberculomas, by contrast, are usually irregularly shaped, solid, and greater than 20 mm in size. They are often associated with severe perifocal edema and focal neurological deficit[38] .

Diagnostic criteria for cysticercal granuloma*

Criteria
clinical
seizure (partial or generalized) as initial symptom
no persistent raised ICP
no progressive neurological deficit
no active systemic disease
CT picture
solitary, contrast-enhancing lesion
20-mm diameter lesion
no severe cerebral edema (no midline shift)

* As established by Rajshekhar and Chandy (1997).[33,35]

This distinction is important because parenchymal cysticercosis is a benign and self-limiting condition, whereas tuberculoma is an active infection

requiring prolonged therapy that involves potentially toxic drugs. Several authors firmly believe that this vital distinction, based on clinical and imaging features, is very difficult to make.[44] In addition to those features suggested by Rajshekhar and Chandy,[38] several other imaging features have been suggested to differentiate between these two entities. For example, in this setup a target lesion (a lesion with central or eccentric nidus of calcification or a dot of enhancement) is frequently visualized. In the past, these target lesions were considered a pathognomonic feature of CNS tuberculoma.[2] More recently, Del Brutto, et al.,[10] reported that visualization of an enhancing eccentric dot, which may possibly represent the scolex of cysticercosis, can be considered a definite imaging feature of cysticercus origin.

Magnetic Resonance Imaging.

Magnetic resonance imaging is frequently performed with the objective of differentiating between cysticercal granuloma and tuberculoma. In fact, the MR imaging features of both these conditions are also very similar and usually not helpful in this differentiation. Investigation entailing T_1-weighted MR images reveals a low signal center with isointense periphery. Granulomas are better visualized on T_2-weighted sequences where a low signal ring and high signal center are characteristic features. Surrounding edema is also best seen

on T_2-weighted MR images (above Fig). On contrast-enhanced MR imaging studies the granuloma shows marked peripheral enhancement and a low signal area in the center.[36]

Serological Studies.

The two principal serological tests are the enzyme-linked immunosorbent assay and the enzyme-linked immunotransfer blot. The latter is regarded as more reliable, with a specificity of 100% and a sensitivity of up to 97%, in tests of both blood and cerebrospinal fluid. Several authors, however, have demonstrated that these tests display poor sensitivity in detecting antibodies in cases of single lesions when sensitivity is very low (range 14-45%). Positive results obtained with these tests may help in confirming the diagnosis of cysticercal granuloma, but negative results cannot be used to exclude a diagnosis of cysticercosis.[40,41]

Diagnostic Criteria. Several diagnostic criteria for NCC have been proposed; however, in none of the criteria has the issue of differentiation between cysticercal granuloma and tuberculoma been addressed. This is the most difficult differentiation clinicians face in the developing countries. These

diagnostic criteria are based on clinical, epidemiological, imaging, and serological features along with response to anticysticercal drugs.

During a recent consensus meeting on cysticercosis held in August 2000 in Lima, Perú, a panel of experts agreed upon more accurate and stringent revised criteria for the diagnosis of neurocysticercosis.

Chart of diagnostic criteria and degrees of diagnostic certainty for human cysticercosis

Diagnostic criteria and degrees of certainty Criteria

Absolute criteria

1. Histologic demonstration of the parasite

2. Direct visualization of the parasite by funduscopic examination

3. Evidence of cystic lesions showing the scolex on CT or MRI

Major criteria

1. Evidence of lesions suggestive of neurocysticercosis on neuroimaging studies

2. Positive immunologic tests for the detection of anticysticercal antibodies

3. Plain X-ray films showing “cigarshaped” calcifications in thigh and calf muscles

Minor criteria

1. Presence of subcutaneous nodules (without histologic confirmation)

2. Evidence of punctuate soft-tissue or intracranial calcifications on plain X-ray.

3. Presence of clinical manifestations suggestive of neurocysticercosis

4. Disappearance of intracranial lesions after a trial with anticysticercal drugs

Epidemiologic criteria

1. Individuals coming from or living in an area where cysticercosis is endemic

2. History of frequent travel to cysticercosis-endemic areas

3. Evidence of a household contact with Taenia solium infection

Degrees of certainty

Definitive diagnosis

1. Presence of one absolute criterion

2. Presence of two major criteria

3. Presence of one major plus two minor and one epidemiologic criterion

Probable diagnosis

1. Presence of one major plus two minor criteria

2. Presence of one major plus one minor and one epidemiologic criterion

3. Presence of three minor plus one epidemiologic criterion

Possible diagnosis

1. Presence of one major criterion

2. Presence of two minor criteria

3. Presence of one minor plus one.

Unfortunately, none of the criteria proposed by Del Brutto, et al.,[9,10] seems to be helpful in differentiating between these two entities .

Other Diagnostic Possibilities

Several other diseases may also pose a diagnostic challenge in patients with a single enhancing CT-documented lesion. It is important to assess for the presence of a systemic disease such as pulmonary tuberculosis, malignancy, or a source of bacterial infection. A small CT-demonstrated enhancing brain lesion in an aged individual should arouse great suspicion of secondary malignancy and effort should be made to define primary site. When applicable (for example, in cases in which there is a history of sexual contact with a high-risk individual), serological tests for the human immunodeficiency virus infection should be performed because several human immunodeficiency virus related CNS complications such as toxoplasmosis, CNS lymphoma, and various fungal granulomas can present with single enhancing lesions.

Enlargement of lesions on follow-up CT scans is not necessarily indicative of a neoplastic nature because a small but significant number of single cysticercal granuloma may show paradoxical enlargement (exceeding 20 mm in diameter) after treatment with anticysticercal drugs.[37] Singh and associates[45] reported two patients in whom even enlarged CT-evidenced lesions resolved spontaneously.

Management:

Until recently, there was no consensus concerning the exact origin of these single enhancing lesions in India; thus, a uniform treatment has not been practiced. Various physicians continue to treat these lesions differently.

Antituberculous Treatment

In few initial series, patients received antituberculous treatment.[50]After histopathological demonstration of cysticercal disease origin in a majority of patients, as well as evidence of spontaneous resolution of these lesions, antituberculous treatment is now infrequently used.

Anticysticercal Treatment

In Latin American countries, CT-depicted single enhancing lesions are invariably treated either with albendazole or praziquantel. In an uncontrolled

study Del Brutto[8] 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 nontreatment group. The authors favored routine administration of anticysticercal drugs in patients with single enhancing lesions.[28]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.[29]

In India, studies involving anticysticercal treatment have provided conflicting results. In a placebo-controlled study Padma, et al.,[27]observed that 7-day treatment with albendazole did not hasten the resolution of CT-documented lesions. In a different double-blind placebo-controlled study, however, Baranwal, et al.,[1] 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.,[7] 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.

Antiepileptic Treatment

It has been argued that because CT-demonstrated enhancing lesions represent dying stages of cysticercal lesion, they require no anticysticercal treatment. Because these lesions disappear spontaneously, patients require only antiepileptic drugs to control the associated seizure disorder.[12] Chopra, et al.,[6] reported on 78 patients who underwent follow-up repeated CT scanning within 6 to 12 weeks; in 47 cases complete spontaneous resolution of the lesions was observed. Significant reduction in the size of the lesions and surrounding cerebral edema occurred in 24 patients; additional repeated CT studies revealed either complete disappearance or considerable regression in due course. All these patients received antiepileptic drugs only. In the only prospective study, Singh, et al.,[46] included 75 patients with single enhancing lesions. Follow-up CT scanning conducted after 2 months revealed complete

spontaneous disappearance of lesions in 55 patients (73.3%). In 11 patients (14.7%) the lesions became calcified. In the remaining nine patients in whom the lesions persisted or regressed, another follow-up CT study (after 6 months) revealed either complete disappearance or calcification. The majority of patients (87%) remained seizure free after 1-year follow-up examination.

Associated Seizure Disorder

Enough evidence is available to suggest that the prognosis of associated seizure disorder is better in cases in which single enhancing lesions are present than in those in which other forms of parenchymal NCC are found; in the latter there is very high incidence of seizure recurrence after withdrawal of antiepileptic drugs.[3] It has been suggested that patients with CT-documented single enhancing lesions do not require conventional prolonged therapy of 2 to 3 years. Antiepileptic drugs can safely be withdrawn once the lesion has disappeared. In a retrospective study, Murthy and Subba Reddy[25] studied 102 patients in whom CT scanning revealed a single enhancing lesion and in whom seizures were present. In 64 patients seizures did not recur once antiepileptic treatment was instigated. Twenty-eight patients (27.5%) continued to experience seizure recurrence for a median 2-month period before spontaneous remission was achieved. In the remaining 10 patients

seizures recurred after albendazole therapy (median period of seizure recurrence 8 months). The anti-epileptic drugs were withdrawn in all patients after the follow-up CT scan revealed complete resolution of the lesions. After withdrawal of antiepileptic drugs, only one patient experienced seizure recurrence during the mean follow-up period of 45 months (range 19-101 months). In this patient in whom seizures recurred, follow-up CT scanning revealed a gliotic scar at the site of the enhancing lesion.[25]

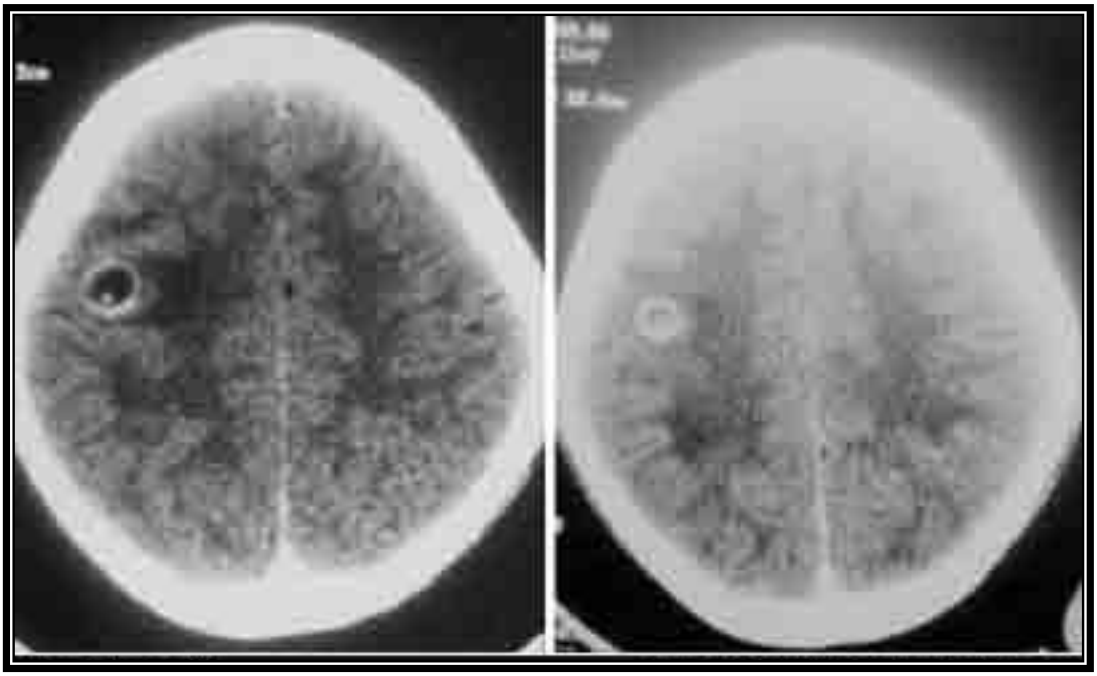
In one review Carpio, et al.,[3] suggested that seizures in patients with CT-demonstrated single enhancing lesions should be considered as acute symptomatic seizure disorder and such patients require antiepileptic treatment for the acute-stage period (perhaps for several months, during which the inflammatory reaction of the lesion is most active). Once the lesion has disappeared, antiepileptic therapy may be gradually withdrawn. If seizures recur, the antiepileptic therapy treatment should be reinitiated and the patient may be treated for 2 to 3 years. If lesions have become calcified, the antiepileptic therapy should be provided for a longer period (2-3 years) before being tapered.

Persistence of Enhancing Lesions:

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 (Fig. 5). 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 brain biopsy sampling is necessary.[31] 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[32] 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[16] 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.[16] 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.[6,15,25,26,32,46] In several uncontrolled series, albendazole therapy has been shown to produce complete resolution of persisting lesions.[30,49]

Role of Neurosurgery:

The mainstay of treatment in patients with CT-evidenced single enhancing lesions is seizure control with antiepileptic drugs. Seizures can often be very well controlled. Small cortical granulomas do not require biopsy sampling or removal of the lesion because the parasite is in the stage of dying and will disappear spontaneously. The principal indications for surgical intervention in patients with NCC are treatment of hydrocephalus, the removal of mobile intraventricular cysts, spinal cysts, accessible racemose cysts in the basal cisterns, and large supratentorial cysts causing mass effects.

Stereotactic brain biopsy sampling is often difficult because of the toughness and mobility of the cysticercal lesion, and it can also be hazardous because of the typical site of lesions at the junction of gray-white matter and possible risk of intracerebral hemorrhage. Moreover, because the lesion is benign, obtaining a biopsy sample is not justified in every patient. In very rare cases in which a lesion enlarges and causes increasing neurological deficit refractory to albendazole treatment, other diagnoses such as abscess, tuberculoma, or tumor (primary or secondary metastasis) are likely. Later in the course neurosurgery may be required. Even in developed countries where these lesions are infrequent this same nonsurgical approach has proven successful.[51]

Initially single enhancing lesions demonstrated on CT scanning were presumed to be tuberculoma and were treated with empirical antituberculous drugs. Later, histopathological examination of brain tissue biopsy samples suggested that, in majority, these lesions are single cysticercal granuloma. Tuberculoma may be present in a few of these patients. It is very difficult to differentiate cysticercal granuloma from tuberculoma based on clinical and radiological characteristics. The authors of several prospective and retrospective studies have convincingly demonstrated that, regardless of origin, single enhancing CT-documented lesions tend to disappear spontaneously. Patients require antiepileptic drugs to prevent seizure

recurrence. The role of albendazole or praziquantel in hastening the resolution of lesions is uncertain. Antiepileptic drugs may safely be withdrawn after CT evidence that the lesion has disappeared. Neurosurgical intervention may be considered if lesions increase in size and produce uncontrolled seizure or progressive focal neurological deficit.

MAGNETIZATION TRANSFER IMAGING:

Introduction:

Magnetization transfer (MT) in a magnetic resonance imaging (MRI) context was first discovered accidentally by Dr Bob Balaban *et al.* These investigators were attempting to perform a spin transfer experiment by selective saturation of urea looking for small signal suppression in water. Instead, they found a significant loss of image intensity from the proton signal in tissue, which did not depend on the specific offset frequency of the irradiation.

This generalized signal suppression, now known as MT, has become accepted as an additional way to generate unique contrast in MRI that can be used to advantage in a variety of clinical applications. The detailed underlying biophysics of MT is quantitatively understood, enabling MT to be optimally exploited in MRI.

HOW MT WORKS?

Proton MRI detects signal only from mobile protons which have sufficiently long *T*2 relaxation times (i.e. greater than 10 ms) so that spatial

encoding gradients can be played out between excitation and acquisition before the signal has completely decayed. The *T*2 of the less mobile protons associated with macromolecules and membranes in biological tissues are too short (i.e. less than 1 ms) to be detected directly in MRI. However, coupling between the macromolecular protons and the mobile or 'liquid' protons allows

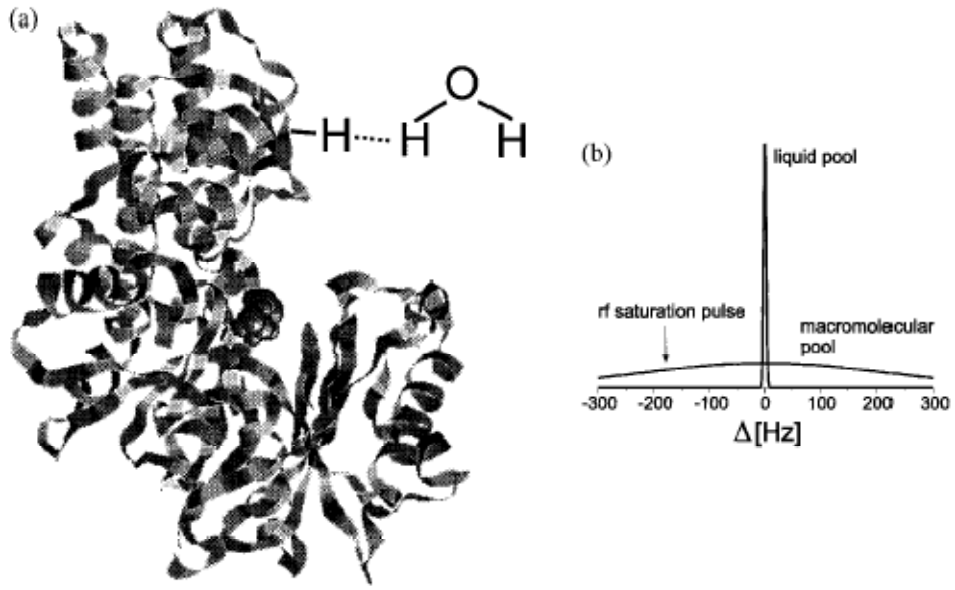


Figure 1. (a) Magnetization transfer exchange between macromolecular and water protons. (b) The macromolecular spins, exhibiting much broader absorption lineshape than the liquid protons, can be preferentially saturated using an off-resonance RF pulse

the spin state of the macromolecular protons to influence the spin state of the liquid protons through exchange processes. As shown in the figure below it is

possible to saturate the macromolecular spins preferentially using an off-resonance radio frequency pulse. The macromolecular spins have a much broader absorption line shape than the liquid spins, making them as much as

106 times more sensitive to an appropriately placed off-resonance irradiation. This preferential saturation of the macromolecular spins can be transferred to the liquid spins, depending on the rate of exchange between the two spin populations, and hence can be detected with MRI.

Applications in imaging

Magnetization transfer is more than just a probe into the proton spins interactions within tissues. It can be used to provide additional advantageous contrast in MR images. One universally agreed upon MT application is in magnetic resonance angiography (MRA). MRA uses specific imaging sequences to suppress the signal from static tissues while enhancing signal from blood by means of inflow or phase effects. The signal contrast between the blood and other tissue can always be enhanced by using MT (which need not affect blood) to further suppress the background tissue signal. Better contrast between blood and tissue leads to better angiograms. The improvement produced by MT in MRA is predicted to become even greater at higher fields because of the larger MT effect.

The second major application of MT is characterization of white matter disease in the brain, principally multiple sclerosis (MS). MS is a diffuse, progressive disease, grossly characterized by the presence of lesions in brain white matter tissue with pathological characteristics\ that vary as the lesions

evolve. The evolution and history of specific MS lesions is difficult to resolve with conventional *T*1-weighted or *T*2-weighted MRI, and some lesions are unobservable. Using MT imaging for regionof- interest analyses, however, MS lesions are more conspicuous and MTR values provide information on lesion evolution. More recently, the diffuse characteristics of MS have been characterized by plotting the MTR histogram of the whole brain. This process indicates that there are significant differences between the MTR ratio of the so-called 'normal-appearing white matter' in MS patients and the white matter of healthy individuals. Histogram-based measures of MTR show strong correlation with cognitive decline in MS patients and may provide a useful method to study the natural course of MS or to evaluate the effect of drug treatments.

Magnetization transfer is a unique contrast mechanism in MRI that has been known for the past decade. Over this period, researchers have characterized the underlying NMR physics, exchange and relaxation rates that govern MT, although detailed understanding of the chemistry and molecular interactions is still needed. Full models of MT have allowed for confident optimization of MRI pulse sequences for MT. MT has shown its value in

MRA and white matter disease and holds continuing promise for use in imaging other tissues and diseases.

MATERIALS AND METHODS

Design of the Study: Prospective

Study Centre: Institute of Neurology and Barnard Institute of Radiology, Madras Medical College and Government general Hospital, Chennai-3

Study Period: January 2009 to April 2010

Material and selection of subjects: Consecutive patients who attended outpatient department / got admitted in the Institute of Neurology or in medical wards fitting into the criteria stated below.

Inclusion criteria: Patients presenting with seizures and a cystic ring enhancing lesion of Brain and with a visualized scolex in MRI as reported by a radiologist were grouped as Neurocysticercosis.

Exclusion criteria: All patients with calcified granuloma

Patients with primary malignancy

HIV positive patients

Patients with Claustrophobia

STUDY PROCEDURE:

Ethical Consideration:

The study was commenced after obtaining approval from the Institutional Ethical Committee. Written informed consent was obtained from those who were willing to participate in the study after explaining to them about the nature of study, additional sequences of imaging and the need for follow up in the prescribed format and in the regional language. Left thumb impression was obtained from those patients who are illiterate.

Detailed histories were obtained including the semiology of seizure, seizure clusters if any before presentation, treatment details before presentation, previous history of seizures and were entered in the pro forma. A thorough clinical examination was performed at the time of admission and was recorded.

All patients were treated with anti epileptic drugs for the entire study period. A course of steroids of 1 mgm/kg was started on patients who had signs of increased intra cranial tension or imaging evidence of profound peri lesional edema.

Patients who were already started on Tab.Albendazole for the current illness by their primary physicians before enrolment into this study were continued on the same for 8 days. Patient who were treatment naïve until enrolment in the study were not treated with Tab.Albendazole.

No Serological or CSF tests for Neurocysticercosis were included in the study. All patients were followed up during the entire study period. They were asked to report for recurrence of seizures in addition to a monthly follow up. CT scans with contrast were done at the end of 6 months for regression, persistence, enlargement, resolution and calcification. In patients with unresolved / partially resolved cysts CT scans were repeated at 12 months. Repeat CT scans were also planned for patients presenting with a recurrence of seizure with semiology different from that of the presenting one at any point during the follow up.

All patients were imaged with MRI Brain and opinions obtained from radiologists.

Sequences used:

Conventional spin ECHO T1-Weighted plain and contrast, T2-Weighted non contrast axial MR images.

Ps3d/3d CISS sequence – for demonstration of scolex.

Signal intensity from rim of the granuloma (Region of Interest) was obtained with a single pixel from the conventional T1WI without an off resonance pulse and MT images with an off resonance pulse.

For each region of interest (ROI), Magnetisation Transfer Ratio (MTR) was calculated using the formula:

$$MTR = \frac{(Mo - Mt)}{Mo} \times 100$$

Mo – Signal intensity with saturation pulse off.

Mt – Signal intensity with saturation pulse on.

Patients with Tuberculomas were recruited and the MT ratios from their walls were calculated for comparison with that of the above studied Neurocysticercosis granulomas.

Inclusion criteria for Tuberculoma:

Patients presenting with seizures and MRI evidence of cystic ring enhancing lesions of Brain with coexistence at least one of the following,

Miliary tuberculosis on chest X-ray

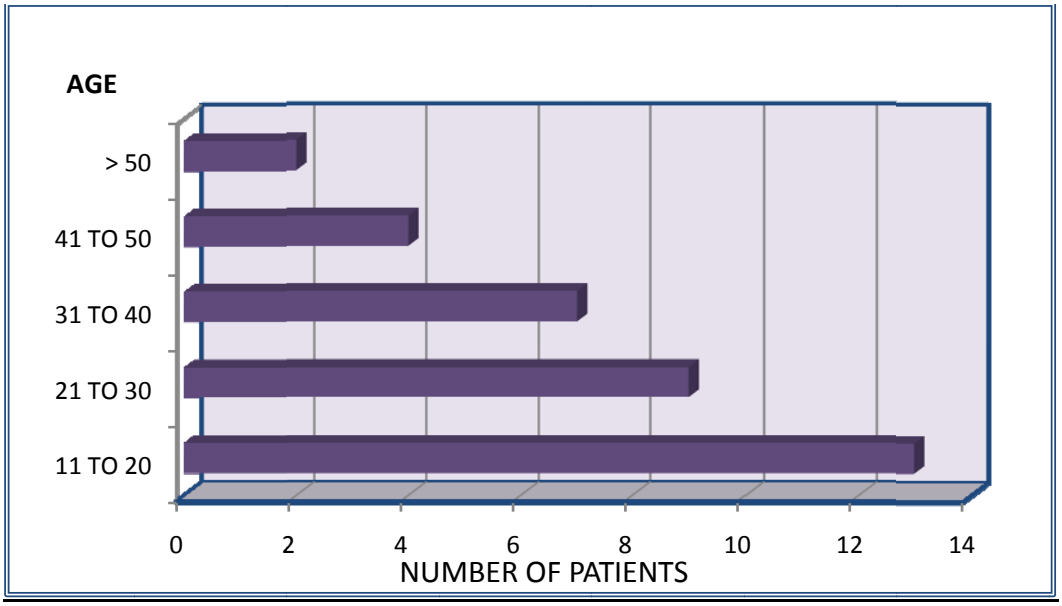
Sputum positive and cavitating pulmonary tuberculosis.

RESULTS

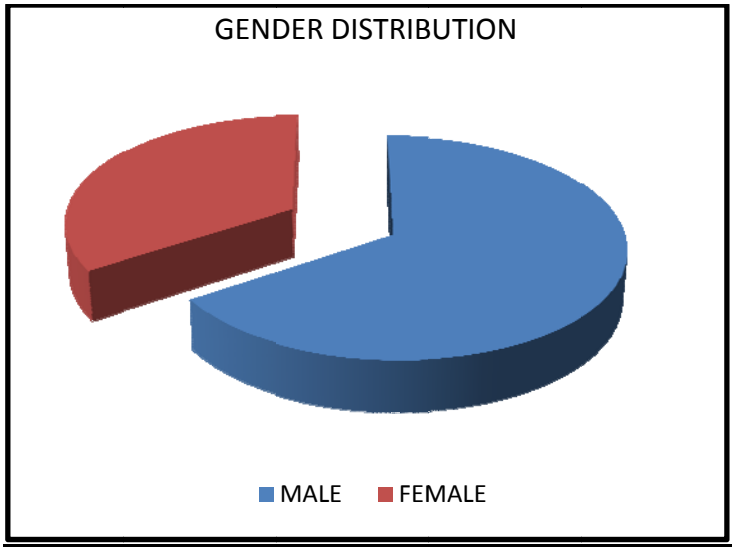
A total of 38 patients with solitary cystic Neurocysticercosis lesion with a visualized scolex (satisfying absolute criteria) were enrolled. 3 patients were lost during follow up leaving 35 patients who were studied for 12 months.

Age distribution

Age	Incidence
11 to 20	**13**
21 to 30	**9**
31 to 40	**7**
41 to 50	**4**
➢ 50	**2**



Gender distribution:.



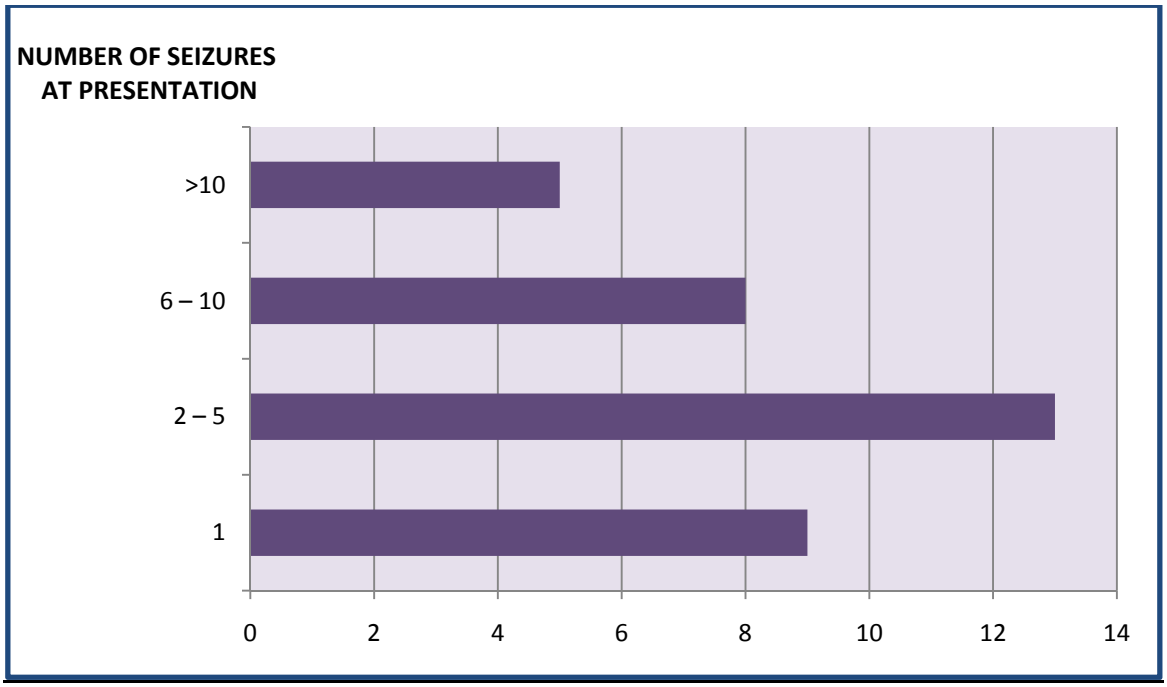
Seizure semiology at presentation:

Seizure type	N
Partial	**21**
Rt Focal Motor	**9**
Lt Focal Motor	**3**
Rt Focal Sensory	**2**
Sensori Motor	**5**
Speech arrest	**1**
Occipital seizures	**1**
Secondary Generalisation	**8**
Complex partial	**2**
Cps Secondary Generalisation	**1**
Generalised from Onset	**12**

Number of seizures before presentation

Number of seizures at presentation	Number of patients
1	9
2 – 5	13
6 – 10	8
>10	5

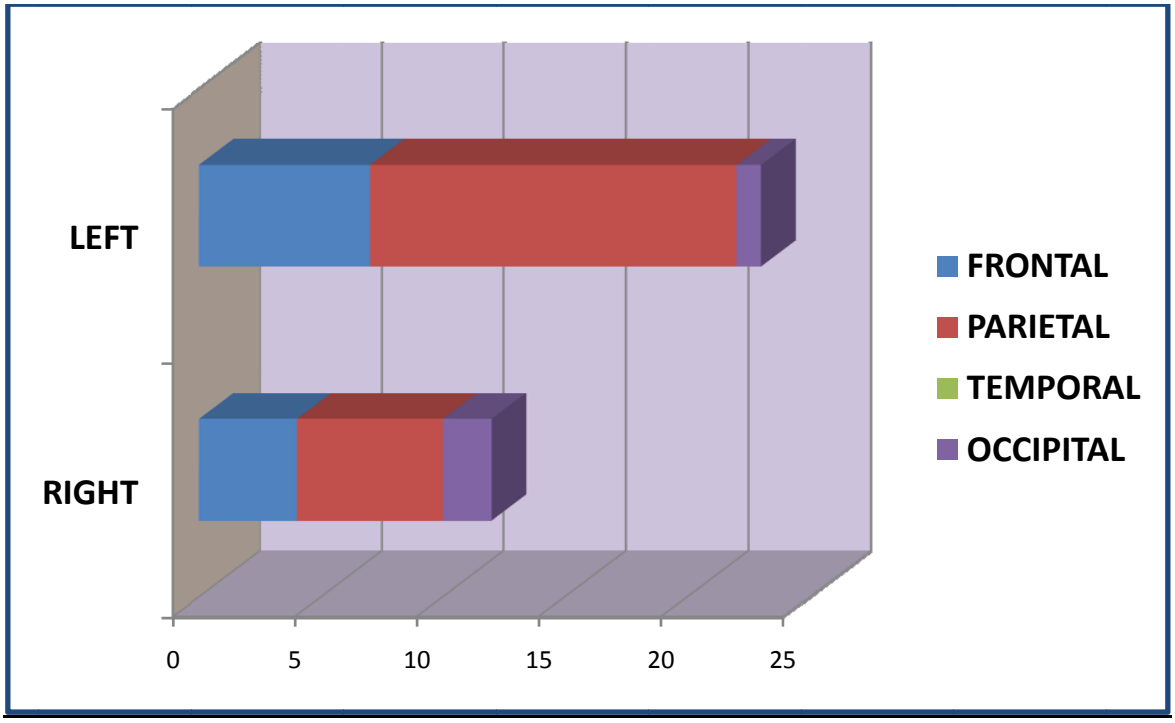
9 patients had a solitary seizure at presentation.13 patients presented with more than 5 / clusters of seizures and one of them presented with status epilepticus.



Location of cyst in the MRI

LOCATION	N
Frontal	11
Parietal	21
Temporal	-
Occipital	3

The lateralization and localisation of cystic granulomas in the imaging studies.



Recurrence of seizures

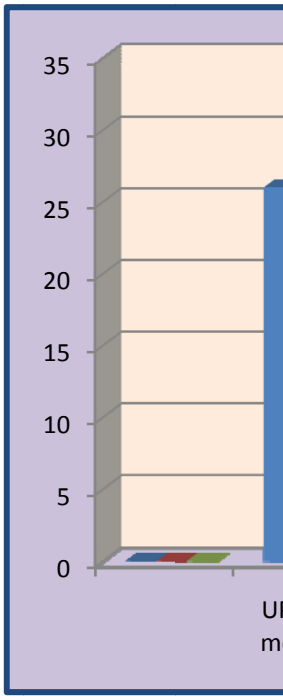
On follow up visits the recurrence of seizures if any was recorded.19 seizures recurred in 11 patients during the study period, the semiology of which were the same as that of the initial ones in all of them.

Period	Total patients seizure free n=35	Albnendazole Treated n=14	Albendaznole NOT Treated n=21
UPTO 3 months	26	12	14
4 TO 6 months	28	11	17
7 TO 9 months	32	14	20
10 TO 12	33	12	20*

months

* New ring enhancing lesion at a different site from the initial one

SEIZURE RECURRENCE	Albendazole		Total
	Treated	**NOT Treated**	
Yes	4 28.6%	7 33.3%	11 31.4%
No	10 71.4%	14 66.7%	24 62.9%
	14 100.0%	21 100.0%	35 100.0%



Seizure recurrence with and without Albendazole treatment

Pearson Chi-Square value **0.134**

Status of the Cyst on Follow up Scans

Patients were subjected to CT scans with contrast study around 6 and 12 months of the study.48% of the total granulomas completely resolved and 11% of them calcified at 6 months. At 12 months 65% of all granulomas disappeared while 17 % were found calcified. At 12 months one of the patients was found to have a new granuloma from a site different from the initial one. This patient had recurrence of seizures, the semiology of which was the same as that of presenting one (GTCS).

Complete Resolution of cysts at 12 months with / without Albendazole Treatment:

Of the 35 patients followed up 23 patients had a complete resolution of their cysts at the end of 12 months 9 of the 14 patients treated with Albendazole and 14 of the 21 patients treated without Albendazole had a complete resolution.

The complete reolution of cysts in association with Albendazole was looked into:

Complete resolution	Observed N	Expected N	Residual

Albendalole Treated	9	11.5	-4.5
Albendalole NOT Treated	14	11.5	-4.5
Total	23		

	COMPLETE RESOLUTION
Chi-Square	3.522
Df	1
p	**.061**

Calcification of cysts at 12 months with / without

Albendazole Treatment:

Of the 35 patients followed up 6 patients had a calcification of their cysts at the end of 12 months 2 of the 14 patients treated with Albendazole and 4 of the 21 patients treated without Albendazole had a residual calcification.

The calcification of cysts in association with Albendazole was looked into:

Residual calcification	Observed N	Expected N	Residual

Albendalole Treated	2	4	-2.5
Albendalole NOT Treated	4	4	2.0
Total	6		

	RESIDUAL CALCIFICATION
Chi-Square	2.000
Df	1
p	**.157**

PREDICTORS OF SEIZURE RECURRENCE- clinical

FEATURE	**N**	**SEIZURE RECURRENCE**	**"P"**
SEX			
MALE	23	8	**0.182**
FEMALE	12	3	
MEDIAN AGE AT DIAGNOSIS			
<24	16	7	**0.257**
>24	15	4	

TODDs PARALYSIS	11	3	**0.19**
NUMBER OF SEIZURES AT PRESENTATION			**0.12**
<5	21	7	
>5	13	4	
SEIZURE TYPE			**0.163**
PARTIAL	13	5	
PARTIAL TO GEN	9	3	
GENERALIZED	12	3	
LOCATION OF CYSTS			**0.184**
PARIETAL	21	8	
FRONTAL	11	3	
OCCIPITAL	3	-	
ANTI HELMINTHIC TREATMENT			**0.134**
ALBENDAZOLE TREATED	14	7	
NOT TREATED	21	4	

ASSOSIATIONS OF SEIZURE RECURRENCE- Follow up Radiology

Of the 35 patients followed up, 11 patients had a recurrence of seizures in the entire follow up period. The follow up radiological study in these patients with recurrence of seizures showed a residual calcification in 6, partial resolution in 4 and complete resolution of cysts in one patient at the end of 12 months

The recurrence of seizure during the study period in association with the residual calcification / partial resolution at 12 months were looked into:

	Observed N	Expected N	Residual
Residual calcification / partial resolution	10	5	3.0
Complete resolution	1	5	-3.0
Total	11		

	SEIZURE RECURRENCE
Chi-Square	4.500
Df	1
p	**.034**

Magnetisation Transfer Ratio:

PARAMETER	N	Mean MT Ratio	"p"
Number of seizures at presentation			0.83
Single	9	16.4	
>10 / clusters	5	15.8	

Recurrence in the follow up period Yes No	11 24	16.1 17.0	0.54
Complete Resolution at 12 months Yes No	23 12	15.4 16.9	0.34

The Magnetisation transfer ratio from the cyst walls of all patients was calculated at the time of inclusion into the study.

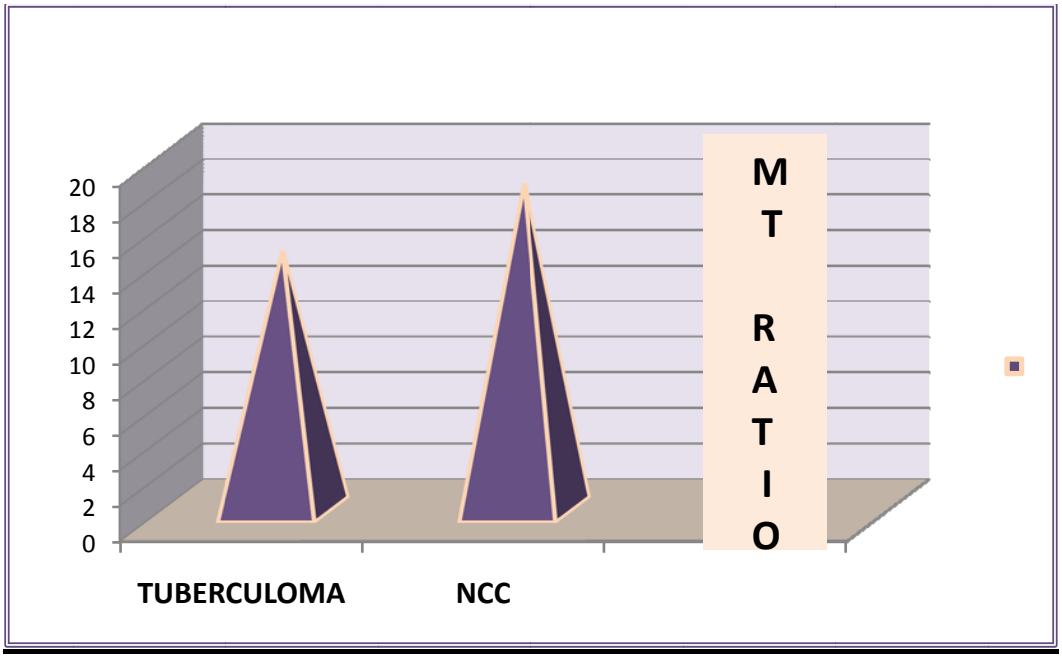
The MT ratio of all the patients ranged from 15.6 to 17.56.

Despite this narrow range, the MT ratio calculated at presentation was tested for any association with the number of seizures (single / >10) at presentation, recurrences in the follow up period and complete resolution at 12 months.

Magnetisation Transfer Ratio - Neurocysticercosis and Tuberculoma:

Tuberculoma	**14.6 to 19.3**
Neurocysticercosis	**18.4 to 22.90**

Finally the Magnetisation Transfer ratios that were calculated from the cyst walls of the Neurocysticercosis patients were compared to that of Tuberculomas selected as per the inclusion criteria



DISCUSSION

The study was determined to include only the unequivocal cases of Neurocysticercosis. The latest consensus statement available in the literature is the one published following the meeting of a panel of experts in August 2000 at Lima, Peru which states 3 absolute criteria that permit an unequivocal diagnosis of Neurocysticercosis. The practically feasible criterion of "Evidence of cystic lesions showing the scolex on CT or MRI" was chosen as the essential pre requisite for inclusion of patients in this study.

Age and Sex incidence:

Degenerating neurocysticercosis lesions can occur at any age; the youngest reported patient in literature was 2 years old. This study included patients between 11 and 62 years with a mean age of 24 and with a gender distribution of 23 males and 12 females. The above data from this study are compared with other similar studies. It is unclear as to why NCC is more common in the young adults; it might be the result of a relative lack of immunity to the infection at that age. With age, repeated exposure of the parasite might lead to some degree of immunity in the

Age and sex incidence in comparison with major studies

Author	Year	Age (years)		Sex		Total
		<20	>20	M	F	
Tandon and Bhargava	1985	12	10	10	12	22
Sethi et al	1985	5	6	7	4	11
Goulatia et al	1987	Mean 16.4		24	22	46
Wadia et al	1987	18	21	-	-	39
Van Dyck	1988	5	0	3	2	5
Bhatia and Tandon	1988	17	8	19	6	25
Ahuja et al	1989	-	-	16	22	38
Kumar et al	1990	56	0	-	-	56
Sachdev et al	1991	20	1	-	-	20
Chopra et al	1992	74	48	77	45	122
Misra et al	1994	-	-	-	-	513
Murthy and Reddy	1998	Mean 21.9		68	34	102
V.rajashekar et al	2001	Mean 20.8		129	81	210
Goel.D	2010	Mean 21.4		244	101	345
This study	***2010***	***Mean 24.1***		***23***	***12***	***35***

individual which prevents the development of disease even if the individual consumes ova or parasite. The higher predilection of males in many of the series including this could be because of the fact that men are more likely to consume foods prepared outside their homes. Foods prepared outside the home might not be hygienically prepared and stored and the chances of several people handling then are high.

Seizure types at presentation:

Partial seizures are by far the commonest presentations in all the studies to the magnitude of 100% in Kumar et al and Wadia et al studies and above 90% in the studies of Tandon,Bhatia and Sachdev.

This study also revealed partial seizures as the most common type (60%) of which 40% generalised.Among the partial seizures right focal motor semiology was the commonest observed. The high percentage generalisation from onset (35%) is similar to the one observed in Chopra et al study. One patient had speech arrest as the only neurological presentation. The rare occurrence of complex partial seizures is a similar observation in most of the cited studies. The commonest sites of lesion location in this study were in the parietal lobes as in most of the studies.

Seizure types in comparison with major studies

Author	Year	Simple Partial	Complex partial	Generalised From onset	Others	Total
Tandon and Bhargava	1985	14	1	1	-	16
Sethi et al	1985	9	-	2	-	11
Goulatia et al	1987	38	-	6	2	46
Wadia et al	1987	39	-	-	-	39
Bhatia and Tandon	1988	16	-	4	-	20
Kumar et al	1990	56	-	-	-	56
Sachdev et al	1991	19	-	1	-	20
Chopra et al	1992	86	4	31	1	122
Srinivas et al	1994	23	-	7	-	30
Sethi et al	1995	164	5	17	-	186
Del Brutto	1995	37	-	17	-	54
Murthy and Reddy	1998	15	49	13	25*	102
This study	***2010***	***21***	***2***	***12***	**-**	***35***

* Simple or complex partial with secondary generalization

RECURRENCE OF SEIZURES:

The arbitrary "window period"

The recurrences of seizures as defined in the other similar studies were the one following a window period of 1 week following the first seizure. This is in accord with strategies used for classification of acute symptomatic seizures from other causes[51] such as stroke or head injury. In Neurocysticercosis, the seizures that occur **both in** the so called window period as well as in the early months (the periods of maximal recurrences) are due to an ongoing activity in the cysts and hence this situation is not comparable to strokes and head injuries in stratification of the window period. Despite being arbitrary, the window period defined for recurrence in this study too was 1 week in accordance with other similar studies[22].

Seizures recurred in 11 of the 35 patients (31%) of patients in the follow up period of 12 months. This seizure recurrence rate of 31% in this study is also in the same range as that of the recurrence rate observed among individuals with structural brain abnormalities and acute symptomatic seizures [53, 54, 55, 56, 57]. A similar study of solitary Neurocysticercosis granuloma from Ecuador by Carpio and Hauser[52] is compared below for the cumulative seizure recurrences in relation to the follow up period.

Follow up period	Cumulative Seizure recurrence (%)	
	This Study	Carpio and Hauser
At 6 months	27.4%	22%
At 12 months	31%	32%

The cumulative seizure recurrence in this study was a marginal 5% high at 3 months period but was almost similar at 12 months compared to Carpio and Hauser study, which included twice as many patients as that of this study.

A total of 14 seizures occurred among this 11 patients. The semiology of recurrences in all patients was the same as that of the initial ones which implies that the same lesions were the ones causing recurrences.

Associations of seizure recurrence:

The cause for only a group of patients developing recurrences, 11 out of 35 patients as in this study is an issue of intensive research and postulations. Knowledge of the risk of seizure recurrence is a necessary prerequisite for making rational decisions regarding short-term treatment of the acute condition and the need for long-term treatment with antiepileptic drugs (AED).An array of variables from patients with and without seizure recurrences in this study were analyzed for their potential associations with recurrence (Table following) and **the only**

Feature	N	Seizure recurrence	"p"
Sex			
male	**23**	**8**	**0.182**
female	**12**	**3**	
Median age at diagnosis			
<24	**16**	**7**	**0.257**
>24	**15**	**4**	
Todds paralysis	**11**	**3**	**0.19**
Number of seizures at presentation			
<5	**21**	**7**	**0.12**
>5	**13**	**4**	
Seizure type			
partial	**13**	**5**	**0.163**
partial to gen	**9**	**3**	
generalized	**12**	**3**	
Location of cysts			
parietal	**21**	**8**	**0.184**
frontal	**11**	**3**	
occipital	**3**	**-**	
Anti helminthic treatment			
albendazole treated	**14**	**7**	**0.134**
not treated	**21**	**4**	

parameter associated significantly (p=0.034) was the persistence of abnormalities at 12 months CT scan (partial resolution / calcification). This implies that patients with either partial resolution or calcification in their repeat scans at 12 months period need a longer anti epileptic course than the other group of patients. A number of studies have found correlations between the persistence of viable or degenerating cysts and increased risks of seizures[58, 59, and 60]

Seizure Recurrence in Albendazole treated and untreated groups:

The studies on effectiveness of antiparasitic treatments in solitary degenerating NCC have been variable.

A non blinded follow-up evaluation of Albendazole treated and non treated subjects by Baranwal AK et al[63] showed an equalization of seizure frequency and an overall good prognosis in both groups. This study followed patients over a 2 year period.

In 2006 a Meta analysis of treatment trials of enhancing NCC lesions by Del Bruto and Garcia et al showed an overall benefit of treatment[64]including that of risk of seizure recurrences.

This study though of a small sample size and a short follow up period did not show any association between seizure recurrence in patients with or without Albendazole treatment (p=0.134).

Follow up Radiology:

There is a wide variation in the rates of complete resolution mentioned by different authors[65 – 69], ranging from 22% to 100% at 6 months / 12 months after the first CT scan.

In this study the follow up contrast CT revealed a complete resolution of 17 cysts (48%) at 6 months and 23cysts (65%) at 12 months.

Study	Number of patients followed	Follow up period	Complete resolution (%)
V.Rajashekar	210	6 months 12 months 24 months	36 62 88
Garg & Nag	101	6 months	84
Jain et al	156	6 months	53
Goel et al	345	6 months	61
This study	35	6 months 12 months	48 65

The rates of resolution of cysts at 6 months in studies from north India are high compared to this study. The rate of resolution of cysts in this study was similar to that of the Vedantam Rajshekhar et al study.

Timing of the follow-up scan:

Patients with solitary NCC have a good seizure prognosis after withdrawal of antiepileptic drugs soon after the resolution of the granuloma[70, 1]. Hence it is important to know the natural history of a solitary NCC to decide on the timing of the follow-up scan after the initial investigation. From the trends of resolution (near about 50% resolve in 6 months) in this study it is reasonable to repeat imaging studies at 6 months. The caveat to this statement is that the patient should be clinically evaluated frequently to look for symptoms and signs of a progressive lesion.

It is mentioned in a few studies, as the one by Mukherjje et al that in a small proportion of cases (10%), the cysts actually increase in size on follow-up CT scans. No such phenomenon was observed in this study.

Complete Resolution of cysts at 12 months with / without Albendazole Treatment:

Of the 35 patients followed up 23 patients had a complete resolution of their cysts at the end of 12 months 9 of the 14 patients treated with Albendazole and 14 of the 21 patients treated without Albendazole had a complete resolution. The association of complete resolution with / without Albendazole treatment was not statistically significant.

Disappearance and reappearance of cysts:

One patient in this study had a recurrence of granuloma at a site other than the presenting one in the last quarter of the study. The initial one was in the right parietal lobe which had resolved completely in the CT scan taken at 6 months and the recurrence was in the right frontal lobe. The initial as well as the recurrent seizure in this patient was a Generalised clonic seizure. This phenomenon of disappearance and reappearance of cystic as well as calcified granulomas at sites different from the initial one have been described in earlier studies from our institute by Prof.S.Kalyanaraman followed by others.

`**Residual calcifications:**

Among the 11 patients with seizure recurrences, 6 had calcifications (54.5%), 4 had partial resolution (36%) and one patient (9%) had complete resolution of granulomas in the CT scans done at 12 months. The high incidence of calcific lesions among patients with recurrences is an observation differing from other studies52.This has implications for the following reasons.

- Calcified brain lesions typical of those in patients with Neurocysticercosis are commonly seen in CT images of patients who present with seizures in our regions.

- In population-based studies that report the findings of CT examinations, calcified lesions are much more common than are viable cysts and they are more prevalent in symptomatic patients than they are in asymptomatic patients62.
- Lastly, when symptoms appear in individuals with only calcified lesions, some of these patients have perilesional brain oedema around one or more of the calcifications62.

Magnetisation Transfer ratio:

The Magnetisation transfer ratio from the cyst wall of the neurocysticercosis granuloma at the time of inclusion in ranged from **18.4 to 22.90.** Despite being in a narrow range the variation in ratios between patients were analyzed statistically for any associations with an array of variables. None of the variables such as multiplicity of seizures at presentation, location of cysts, seizure recurrence and resolution of cysts had any statistical associations with the magnetization transfer ratios.

The Magnetisation transfer ratios from the cyst walls of Tuberculoma patients selected as per the inclusion criteria ranged from **14.6 to 19.3**. The observed MT ratios from the walls of Tuberculomas are in a lesser range

observed in the study by Rakesh.K.Gupta84 which ranged from 18.67 to 23.34.

In this study the MT ratios from walls of degenerating Neurocysticercosis were in a fairly distinct range compared to Tuberculomas.However the MT ratios of two of the NCC cysts and one of the Tuberculomas were observed in an overlapping range.

Using MT ratio the sensitivity for differentiating Tuberculoma and NCC was 78% and specificity was 73% in this study.

The unresolved management issue:

Carpio and colleagues studied 138 patients prospectively with cystic neurocysticercosis in an open-label study of steroids alone or together with either albendazole or praziquantel80. Overall, there were no significant differences in the proportion of patients free of cysts at 6 months or 1 year, in the proportion free of seizures for 2 years, or in the rates of sequelae.

Among many case series only three randomized, controlled trials of Albendazole have been reported on patients with single enhancing lesions. Two studies (**Padma, M. V** et al and **Singhi, P** et al) demonstrated no significant difference in radiographic resolution69, 82. The other (**Baranwal)**

demonstrated more rapid resolution with albendazole but no significant difference in the frequency of clinical events83.

In this study the treatment with Albendazole when subjected to statistical analysis did NOT "reduce the incidence of recurrent seizures" or "shorten the time to cyst disappearance."

The proponents of Albendazole treatment accept the above facts that have already been brought out in other studies but argue that they treat to kill larvae and live cysts seen or not seen on neuroimaging73, 74 which if not treated will eventually become inflammatory and symptomatic. They also believe that such therapy may reduce the chance of calcification in the lesions during healing75.

In contrary the number of calcifications in this study, though not statistically significant (p=0.157) occurred more in the Albendazole treated group. The studies of Hauser et al also do not support the argument that antihelminthic treatment reduces calcification.

The non advocators of the use of antihelminths are aware that such therapy results in death and resolution of viable cysts, 76-79 but question the clinical benefit of this treatment50.They argue that the antihelminthic treatment predictably injures cysts and initiates at times an intense inflammatory response81 that mimics the natural evolution of cysts. Seizures,

headaches, and uncommonly death can occur secondary to cysticidal treatments.

There are some conservative groups which use Albendazole only when cysts do not regress after 6 months, in enlarging cysts, and in a few patients with “disappearing/reappearing” lesions.

Evidence for a convincing clinical outcome from more trials of anticysticercal treatment is keenly awaited.

CONCLUSIONS

1. The observed mean age of incidence of single ring enhancing Neurocysticercosis was 24 with a male female ratio of 2:1.
2. Partial seizures were the most common seizure type at presentation of which right focal motor semiology was the commonest.
3. Majority of granulomas occurred in the parietal lobes (Left > Right).
4. Seizures recurred in 11 of the 35 patients (31%) in the follow up period of 12 months.
5. Follow up contrast CT revealed a complete resolution of 17 cysts (48%) at 6 months and 23cysts (65%) at 12 months.
6. Treatment with Albendazole did NOT statistically "reduce the incidence of recurrent seizures" or "shorten the time to cyst disappearance."
7. The only parameter associated with seizure recurrence statistically ($p=0.034$) with was the persistence of abnormalities at 12 months CT scan (partial resolution / calcification).
8. The high incidence of a resultant calcific lesions among patients with recurrences is an observation different from other studies.

9. The Magnetisation transfer ratio from the cyst wall of the neurocysticercosis granuloma at the time of inclusion in ranged from 18.4 to 22.90.
10. MT ratio differentiated Tuberculoma and degenerating NCC with a sensitivity of 78%, and specificity of 73%.

BIBILIOGRAPHY

1. Baranwal AK, Singhi PD, Khandelwal N, et al: Albendazole therapy in children with focal seizures and single small enhancing computerized tomographic lesions: a randomized placebo-controlled double blind trial. Pediatr Infect Dis J 17:696-700, 1998
2. Bargallo J, Berenguer J, Garcia-Barrionuevo J, et al: The "target sign": is it a specific sign of CNS tuberculoma? Neuroradiology 38:547-550, 1996
3. Carpio A, Escobar A, Hauser WA: Cysticercosis and epilepsy: a critical review. Epilepsia 39:1025 1040, 1998
4. Chacko G, Rajshekhar V, Chandy MJ, et al: The calcified intracorporeal vacuole: an aid to the pathological diagnosis of solitary cerebral cysticercus granulomas. J Neurol Neurosurg Psychiatry 69:525-527, 2000
5. Chandy MJ, Rajshekhar V, Prakash S, et al: Cysticercosis causing single, small CT lesions in Indian patients with seizures. Lancet 1:390-391, 1989
6. Chopra JS, Sawhney IMS, Suresh N, et al: Vanishing CT lesions in epilepsy. J Neurol Sci 107:40-49, 1992
7. Cruz I, Cruz ME, Carrasco F, et al: Neurocysticercosis: optimal dose treatment with albendazole. J Neurol Sci 133:152-154, 1995
8. Del Brutto OH: The use of albendazole in patients with single lesions enhanced on contrast CT. N Engl J Med 328:356-357, 1993
9. Del Brutto OH, Rajshekhar V, White AC Jr, et al: Proposed diagnosed criteria of neurocysticercosis. Neurology 157: 177-183, 2001
10. Del Brutto OH, Wadia NH, Dumas M, et al: Proposal of diagnostic criteria for human cysticercosis and neurocysticercosis. J Neurol Sci 142:1-6, 1996
11. Escobar A: The pathology of neurocysticercosis, in Palacios E, Rodriguez-Carbajal J, Taveras JM (eds): Cysticercosis of the Central Nervous System. Springfield, IL: Charles C Thomas, 1983, pp 27-54
12. Garg RK: Solitary cysticercus granulomas. J Neurosurg 82: 911 912, 1995 (Letter)
13. Garg RK, Karak B, Sharma AM, et al: Single CT (ring) lesion in epilepsy patients: a new observation. Indian J Pediatr 66: 155-157, 1999
14. Garg RK, Misra S: Non-epileptic manifestations in patients with single enhancing computed tomography lesions. J Assoc Physicians India 48:788-793, 2000

15. Garg RK, Nag D: Single enhancing CT lesions in Indian patients with seizures: clinical and radiological evaluation and follow-up. J Trop Pediatr 44:204-210, 1998
16. Garg RK, Nag D: Single ring- or disk-enhancing computed tomographic lesion in Indian children and adolescents after first seizure. Arch Pediatr Adolesc Med 157:632-634, 1997
17. Garg RK, Singh MK, Misra S: Single-enhancing CT lesions in Indian patients with seizures: a review. Epilepsy Res 38: 91-104, 2000
18. Goulatia RK, Verma A, Misra NK, et al: Disappearing CT lesions in epilepsy. Epilepsia 28:523 527, 1987 19. Jayakumar PN, Taly AB, Mohan PK: Transient computerised tomographic (CT) abnormalities following partial seizures. Acta Neurol Scand 72:26-29, 1985
19. Kennedy A, Schon F: Epilepsy: disappearing lesions appearing in the United Kingdom. BMJ 302:933-935, 1991
20. Kumar R, Kumar A, Kohli N, et al: Ring or disc-like enhancing lesions in partial epilepsy in India. J Trop Pediatr 36:131-134, 1990
21. Lath R, Rajshekhar V: Solitary cysticercus granuloma of the brainstem. Report of four cases. J Neurosurg 89:1047-1051, 1998
22. Minguetti G, Ferreira MV: Computed tomography in neurocysticercosis. J Neurol Neurosurg Psychiatry 46:936-942, 1983
23. Misra S, Verma R, Lekhra OP, et al: CT observations in partial seizures. Neurol India 42:24-27, 1994
24. Murthy JMK, Subba Reddy YV: Prognosis of epilepsy associated with single CT enhancing lesion: a long term follow up study. J Neurol Sci 159:151-155, 1998
25. Murthy JMK, Yangala R, Srinivas M: The syndromic classification of the International League Against Epilepsy: a hospital-based study from South India. Epilepsia 39:48-54, 1998
26. Padma MV, Behari M, Misra NK, et al: Albendazole in single CT ring lesions in epilepsy. Neurology 44:1344-1346, 1994
27. Pretell EJ, Garcia HH, Custodio N, et al: Short regimen of praziquantel in the treatment of single brain enhancing lesions. Clin Neurol Neurosurg 102:215-218, 2000
28. Pretell EJ, Garcia HH, Gilman RH, et al: Failure of one-day praziquantel treatment in patients with multiple neurocysticercosis lesions. Clin Neurol Neurosurg 103:175-177, 2001
29. Rajshekhar V: Albendazole therapy of persistent, solitary cysticercus granulomas in patients with seizures. Neurology 43: 1238-1240, 1993

30. Rajshekhar V: Etiology and management of single small CT lesions in patients with seizures: understanding a controversy. Acta Neurol Scand 84:465-470, 1991
31. Rajshekhar V: Rate of spontaneous resolution of a solitary cysticercus granuloma in patients with seizures. Neurology 57: 2315-2317, 2001
32. Rajshekhar V: Severe episodic headache as the sole presenting ictal event in patients with a solitary cysticercus granuloma. Acta Neurol Scand 102:44-46, 2000
33. Rajshekhar V, Chacko G, Haran RP, et al: Clinicoradiological and pathological correlations in patients with solitary cysticercus granuloma and epilepsy: focus on presence of the parasite and oedema formation. J Neurol Neurosurg Psychiatry 59: 284-286, 1995
34. Rajshekhar V, Chandy MJ: Clinical manifestation of solitary cysticercus granuloma, in Chandy MJ, Rajshekhar V (eds): Cysticercus Granuloma: The Disappearing Lesion. Hyderabad: Orient Longman, 2000, pp 29-39
35. Rajshekhar V, Chandy MJ: Comparative study of CT and MRI in patients with seizures and a solitary cerebral cysticercus granuloma. Neuroradiology 38:542-546, 1996
36. Rajshekhar V, Chandy MJ: Enlarging cysticercus granulomas. J Neurosurg 80:840-843, 1994
37. Rajshekhar V, Chandy MJ: Validation of diagnostic criteria for solitary cysticercus granuloma in patients presenting with seizures. Acta Neurol Scand 96:76-81, 1997
38. Rajshekhar V, Haran RP, Prakash GS, et al: Differentiating solitary small cysticercus granulomas and tuberculomas in patients with epilepsy. Clinical and computerized tomographic criteria. J Nerurosurg 78:402-407, 1993
39. Rajshekhar V, Oommen A: Serological studies using ELISA and EITB in patients with solitary cysticercus granuloma and seizures. Neurol Infect Epidemiol 2:177-180, 1997
40. Rajshekhar V, Wilson M, Schantz PM: Cysticercus immunoblot assay in Indian patients with single enhancing small CT lesions. J Neurol Neurosurg Psychiatry 54:561-562, 1991
41. Sethi PK, Kumar BR, Madan VS, et al: Appearing and disappearing CT scan abnormalities and seizures. J Neurol Neurosurg Psychiatry 48:866-869, 1985
42. Sethi PP, Wadia RS, Kiyawat DP, et al: Ring or disc enhancing lesions in epilepsy in India. J Trop Med Hyg 97:347-353, 1994

43. Shah GV: Central nervous system tuberculosis: imaging manifestations. Neuroimaging Clin N Am 10:355-374, 2000
44. Singh G, Sobti P, Bhatia RS, et al: Enlarging single CT lesions can also spontaneously resolve. Neurol India 48:85-87, 2000
45. Singh MK, Garg RK, Nath G, et al: Single small enhancing computed tomographic (CT) lesions in Indian patients with new-onset seizures. A prospective follow-up in 75 patients. Seizure 10:573-578, 2001
46. Stamos JK, Rowley AH, Hahn YS, et al: Neurocysticercosis: report of unusual pediatric cases. Pediatrics 98:974-977, 1996
47. Tandon PN, Bhargava S: CNS tuberculosis: lessons learnt from CT studies. Neurol India 28:207-212, 1980
48. Thussu A, Arora A, Lal V, et al: Albendazole therapy for solitary persistent cysticercus granuloma. Neurol India 49:95-97, 2001
49. Wadia RS, Makhale CN, Kelkar AV, et al: Focal epilepsy in India with special reference to lesions showing ring or disc-like enhancement on contrast computed tomography. J Neurol Neurosurg Psychiatry 50:1298-1301, 1987
50. Wadley JP, Shakir RA, Rice Edwards JM: Experience with neurocysticercosis in the UK: correct diagnosis and neurosurgical management of the small enhancing brain lesion. Br J Neurosurg 14:211-218, 2000
51. Commission on Epidemiology and Prognosis of the International LeagueAgainst Epilepsy. Guidelines for epidemiological Studies on epilepsy. Epilepsia1993;34:592–596
52 Prognosis for seizure recurrence in patients with newly diagnosed neurocysticercosis Arturo Carpio, MD; and W. Allen Hauser, MD NEUROLOGY 2002;59:1730–1734
52. Burn J, Dennis M, Bamford J, et al. Epileptic seizures after a first stroke: the Oxfordshire Community Stroke Project. Br Med J 1997;315:1582–1587.
53. Reith J, Jorgensen HS, Nakayama H, Raaschou HO, Olsen TS. Seizures in acute stroke: predictors and prognostic significance.
54. Stroke 1997;28:1585–1589.
55. Salazar AM, Jabbari B, Vance SC, et al. Epilepsy after penetrating
56. head injury. I. Clinical correlates: a report of the Vietnam Head Injury Study. Neurology 1985;35:1406–1414.
57. Annegers JF, Hauser WA, Beghi E, Nicolosi A, Kurland LT. The risk of unprovoked seizures after encephalitis and meningitis. Neurology 1988;38:1407–1410.

58. Annegers JF, Hauser WA, Coan SP, Rocca WA. A population based study of seizures after traumatic brain injuries. N Engl J Med 1998;338:20–24.
59. 58 Carpio A, Santillan F, Leon P, Flores C, Hauser WA. Is the course of
60. neurocysticercosis modified by treatment with antihelminthic agents? Arch Intern Med 1995;155:1982–1988.
61. 59 Baranwal AK, Singhi PD, Singhi SC, Khandelwal N. Seizure recurrence in children with focal seizures and single small enhancing computed tomographic lesions: prognostic factors on long-term follow-up. J Child Neurol 2001;16:443 445.
62. Rajshekhar V, Jeyaseelan L. Seizure outcome in patients 2004;62:2236 2240.with a solitary cerebral cysticercus granuloma. Neurology
63. Nash TE, Del Brutto OH, Butman JA, et al. Calcifi c neurocysticercosis and epileptogenesis. *Neurology* 2004; 62: 1934–38.
64. Baranwal AK, Singhi PD, Singhi SC, Khandelwal N. Seizure recurrence in children with focal seizures and single small enhancing computed tomographic lesions: prognostic factors on long-term follow-up. J Child Neurol 2001;16:443–445.
65. Del Brutto OH, Roos KL, Coffey CS, Garcı´a HH. Meta-analysis: cysticidal
66. drugs for neurocysticercosis: albendazole and praziquantel. Ann Intern Med 2006;145:43–51.
67. Disappearing CT scan abnormalities and seizures. J Neurol Neurosurg Psychiatry 198;48:866–869.
68. Wadia RS, Makhale CN, Kelkar AV, Grant KB. Focal epilepsy in India with special reference to lesions showing ring or disclike enhancement on contrast computed tomography. J Neurol Neurosurg Psychiatry 1987;50:1298–1301.
69. Sethi PP, Wadia RS, Kiyawat DP, et al. Ring or disc enhancing lesions in epilepsy in India. J Trop Med Hyg 1994; 97:347–353.
70. Chopra JS, Sawhney IMS, Suresh N, Prabhakar S, Dhand UK, Suri S. Vanishing CT lesions in epilepsy. J Neurol Sci 1992;107:40–49.
71. Padma MV, Behari M, Misra NK, Ahuja GK. Albendazole in single CT ring lesions in epilepsy. Neurology 1994;44:1344– 1346
72. .Del Brutto OH. Single parenchymal brain cysticercus in the acute encephalitic phase: definition of a distinct form of neurocysticercosis with a benign prognosis. J Neurol Neurosurg Psychiatry 1995;58:247–249.

73. Murthy JMK, Reddy YVS. Prognosis of epilepsy associated with single CT enhancing lesion: a long term follow up study. J Neurol Sci 1998;159:151–155.
74.. Mukherjje A, Chakravarty A. Partial and complex partial seizures. In: Singhal BS, Nag D, eds. Epilepsy in India 2000. Indian Epilepsy Association, 2000;50–75.
75.. Cuetter AC, Garcia-Bobadilla J, Guerra LG, Martinez FM, Kaim B. Neurocysticercosis, focused on intraventricular disease. Clin Infect Dis 1997;24:157–164.
76.. Cuetter AC, Andrews RJ. Intraventricular neurocysticercosis: a series of 18 consecutive patients and review of the literature. J Neurosurg (serial online) 2001;12:1–7.
77..Ambar Chakravarty, MD, FRCP, *Calcutta*, *India* Ambar Chakravarty, MD, FRCP, *Calcutta, India* NEUROLOGY 60 June (1 of 2) 2003
78. Sotelo J, del Brutto OH, Penagos P, Escobedo F, Torres B, Rodriguez-Carbajal J, et al. Comparison of therapeutic regimen of anticysticercal drugs for parenchymal brain cysticercosis. J Neurol 1990;237:69–72.
79. Garcia HH, Pretell EJ, Gilman RH, et al. A trial of antiparasitic treatment to reduce the rate of seizures due to cerebral cysticercosis. N Engl J Med 2004;350:249–258.
80. Alarcon F, Duen˜ as G, Diaz M, Cevallos N, Estrada G. Short course of
81. Albendazole therapy for neurocysticercosis: a prospective randomized trial comparing three days, eight days and the control group without albendazole. Rev Ecuat Neurol 2001;10:1–6.
82. Sotelo J, Penagos P, Escobedo F, Del Brutto OH. Short course of albendazole
83. Therapy for neurocysticercosis. Arch Neurol 1988;45:1130–1133.
84. Carpio A, Santillan F, Leon P, Flores C, Hauser WA. Is the course of neurocysticercosis modified by treatment with antihelminthic agents? Arch Intern Med 1995;155:1982–1988.
85. Del Brutto OH, Roos KL, Coffey CS, Garcı´a HH. Meta-analysis: cysticidal drugs for neurocysticercosis: albendazole and praziquantel. Ann Intern Med 2006;145:43–51.
86. Singhi, P., M. Ray, S. Singhi, and N. Khandelwal. 2000. Clinical spectrum of 500 children with neurocysticercosis and response to albendazole therapy. J. Child. Neurol. 15:207-213
87. Baranwal, A. K., P. D. Singhi, N. Khandelwal, and S. C. Singhi. 1998. Albendazole therapy in children with focal seizures and single small enhancing computerized tomographic lesions: a randomized, placebo-controlled, double blind trial. Pediatr. Infect. Dis. J.17:696-700.

88. Magnetization Transfer MR Imaging Correlation with Histopathology in Intracranial Tuberculomas RAKESH K. GUPTA*, N. HUSAIN{, M. K. KATHURIA*, S. DATTA{, R. K. S. RATHORE{, M. HUSAIN} Clinical Radiology (2001) 56: 656±663

PROFORMA FOR EVALUATION

Name **age** **sex**

Address

Occupation **Contact no**

Medical unit prof **op/ip no** **ward** **DOA** **DOD**

Neuro unit prof **min no** **EEG no**

Referred from

Elaboration of seizure

Witnesse of seizure **Reliability**

Before the episode

Recent illness (headache / fever)

Unusual stress

Medications

Last alcohol intake

Last meal

Sleep deprivation

Activity just before seizure

During the episode

Time of day

Aura

Duration

Ability to talk & comprehend

Ability to recall events

Movements of eyes face arms legs

Tongue bite frothing

Bowel / bladder incontinence

Bodily injuries sustained

After event

Confusion duration

Focal neurological deficits

Headache

Any other significant symptoms

SIGNIFICANT PAST HISTORY

Diabetic : yes / no duration & treatment

Hypertension CAD CKD

tuberculosis

any others

alcohol intake y / n duration freq quantity

last intake

smoking

family h/o seizures

Clinical Examination

General exam

Neuro cut markers

Vitals : BP Pulse RR Temp

CNS :

at presentation Time after seizure

signs of meningial irritation

higher functions

motor system

sensory system

cranial nerves

cerebellum

CVS : RS: P/A :

COURSE DURING HOSPITAL STAY

INVESTIGATIONS :

Hematology TC : DC: P L E B HB : ESR:

Biochemistry sugar urea creatinine Na k Ca

Others :

CXR :

CT BRAIN:

MRI BRAIN:

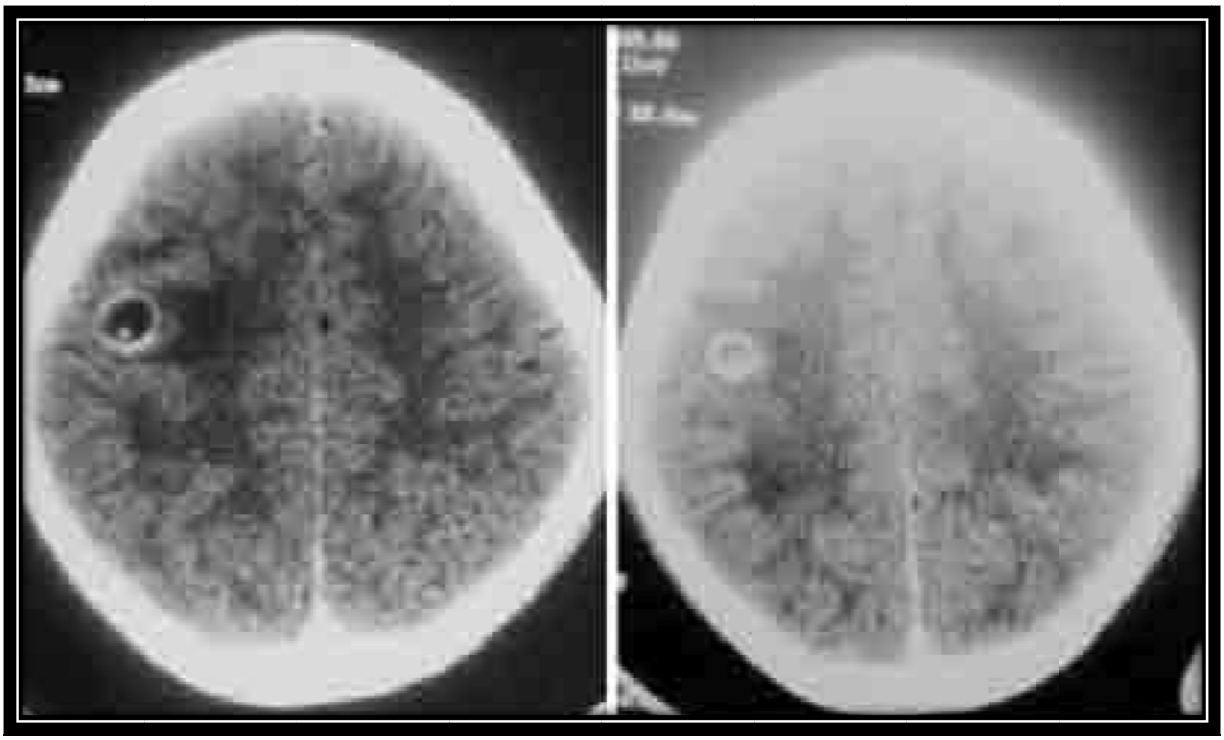
Treatment

MASTER CHART

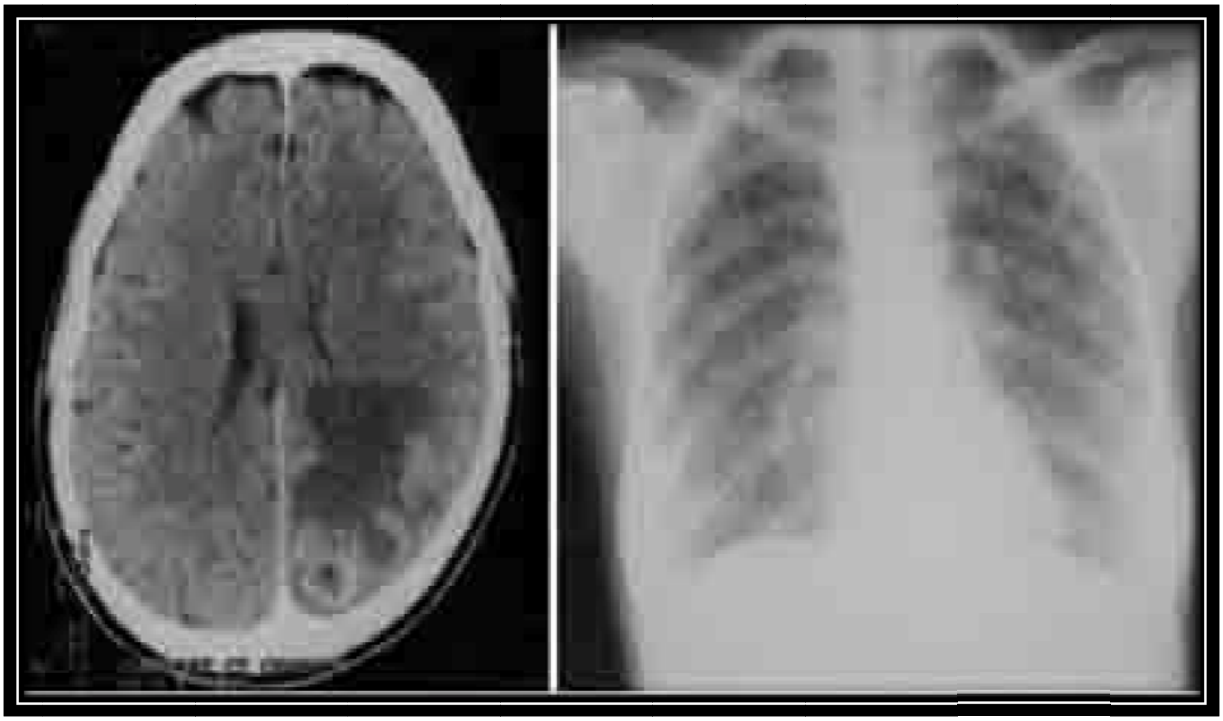
Patient Number	Age	Sex	Seizure Semiology	Number of seizures at presentation	Location Of cyst	Alb	Recurren
1.	13	M	GO	2 - 5	F	Y	R
2.	25	F	PG	>10	PA	N	R
3.	39	M	P	6 - 10	O	N	NR
4.	23	M	GO	1	F	Y	NR
5.	17	M	CPG	1	PA	N	NR
6.	21	M	P	>10	F	N	R
7.	14	M	GO	2 - 5	F	Y	N R
8.	28	F	P	1	F	N	NR
9.	18	F	GO	2 – 5	PA	Y	R
10.	13	M	PG	1	PA	Y	NR

11.	19	F	P	6 - 10	PA	N	NR
12.	40	F	P	6 - 10	PA	N	NR
13.	39	M	P	1	PA	N	NR
14.	16	M	P	>10	PA	N	R
15.	23	F	GO	2 - 5	PA	Y	NR
16.	41	M	P	6 - 10	F	N	R
17.	38	M	GO	2 - 5	PA	Y	NR
18.	18	F	GO	2 – 5	O	N	NR
19.	27	M	PG	2 – 5	PA	N	NR
20.	19	M	P	2 – 5	PA	Y	R
21.	33	F	PG	6 - 10	PA	N	NR
22.	47	M	GO	1	PA	Y	NR
23.	24	M	GO	>10	PA	N	R
24.	40	F	CP	2 – 5	PA	N	NR

25.	15	M	PG	6 - 10	PA	Y	NR
26.	64	M	P	2 – 5	F	N	R
27.	32	M	GO	6 - 10	O	N	NR
28.	24	F	PG	1	F	Y	NR
29.	17	M	GO	2 – 5	PA	Y	R
30.	19	F	GO	>10	PA	N	NR
31.	38	M	PG	1	PA	Y	NR
32.	28	F	P	6 - 10	F	N	NR
33.	33	M	P	2 – 5	PA	N	NR
34.	11	M	P	1	F	N	NR
35.	60	M	PG	2 - 5	PA	Y	NR



SOLITARY CYSTICERCOSIS LESION AT PRESENTATION (LEFT) PARTIAL RESOLUTION AT 6 MONTHA FOLLOW UP RADIOLOGY (RIGHT)



TUBERCULOMA BRAIN WITH MILIARY SHADOWS IN CHEST X-RAY