A DISSERTATION ON

PARTIAL SEIZURES – CT BRAIN, EEG, CLINICAL CORRELATION

D.M. Degree

BRANCH – I
(NEUROLOGY)

THETAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

AUGUST 2006

CERTIFICATE

This is to certify that this dissertation titled "PARTIAL SEIZURES – CT BRAIN, EEG, CLINICAL CORRELATION" submitted by DR.V. SRIRAMAKRISHNAN to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement of the award of D.M. Degree Branch I (NEUROLOGY) is a original research work carried out by him under our direct supervision and guidance.

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ACKNOWLEDGEMENT

I thank the **DEAN,** Madurai Medical College, Madurai for permitting me to utilize the hospital materials for conducting this study.

It is with immense pleasure and privilege that I express my heartful gratitude, admiration and sincere thanks to **Prof. M. RAJAGURU.** M.D. D.M., Professor of Neurology, Madurai Medical College, Madurai for his supervision, help and constant guidance during this study.

I am extremely grateful to **PROF.V INBASEKARAN,M.ch.,** H.O.D., Department of Neurology & Neuro Surgery, Madurai Medical College, Madurai, for his constant encouragement and support while undertaking this study.

I express my sincere thanks and gratitude to our Asst. Professors, Dr. B. Sridharan, M.D. D.M., Dr. N. Muthuveeran, M.D. D.M., Dr. T.R. Gnanaeswaran, M.D. D.M., Dr.R. Kishore, M.D. D.M., Dr. P.K. Murugan, M.D. D.M., Dr. C. Justin. M.D. D.M for their valuable suggestions and support.

It is my pleasure to thank all my fellow Post-Graduates for their assistance.

I thank all the Patients who have ungrudgingly lent themselves to undergo this study without whom this study would not have seen the light of the day.

CONTENTS

INTRODUCTION

Epilepsy is as old as mankind. This disease is known to mankind even in the oldest civilization. About 3000 years ago a secondarily generalized major seizure was fully described in Akkadian, the oldest written language written in Mesopatomia (Now Iraq).[1] Epilepsy is the 8th leading cause of morbidity in children.

Hughling Jackson was the first to classify seizures into generalized and focal (partial) in the year 1870. This classification still holds its place in the clinical description of epilepsy and in its management. International leaque against epilepsy, classifies epileptic seizures broadly into

I - Partial (focal, local) seizures

II - Generalized seizures

III - Unclassified seizure

with sub division under each categories.

Partial seizures are those in which in general the first clinical and Electroencephalographic changes indicate initial activation of a system of Neurons limited to part of one cerebral hemisphere.

Many Investigations have suggested that people with partial seizures are more likely to have recurrence than generalized seizures. [2]

In the evaluation of partial seizure we the physician utilize various tools. First and foremost is the history of illness and then EEG and Neuro imaging. The incidence of structural abnormality in partial seizure is relatively high when compared to generalized seizure, and it is about 78% in a study by S. Misra et al, done at Banarus Hindu University, Varanasi. [3]

EEG helps us to identify the functional site of epileptogenesis even though the yield is low and also helps us to identify the mirror focus. In the era of epilepsy surgery a clinical approach which mixes the skillful history elicitation, EEG, Neuro imaging together helps us to localize the site of origin of seizure and thereby helps us to have a better cure rate.

In our present study we are intended to identify the correlation between the clinical history, EEG, and Neuroimaging in the identification of the site of lesion and also to study the incidence of structural lesion in partial seizures and also to identify clues in the clinical history and examination which points towards structural lesion.

AIM OF THE STUDY

- To study the incidence of structural lesion in partial seizures
- To identify the cause for partial seizures in different age groups
- Role of EEG in localizing the lesion in partial seizures
- Correlation of EEG, and Neuroimaging findings in partial seizures
- To identify clinical clues which predict a structural lesion in partial seizure.

REVIEW OF LITERATURE

Epilepsy a disease with a long history and lot of myths and stigma to its name.

Broadly seizures are classified in to partial, generalized, unclassified by International league against epilepsy 1981. Partial seizure is defined as a one in which in general the first clinical and electroencephalographic changes indicate initial activation of a system of Neurons limited to a part of the cerebral hemisphere. Partial seizure is further classified as simple partial and complex partial by the presence or absence of impairment in the level of consciousness.

I Simple partial

II Complex partial,

III Partial evolving to generalized seizure.

Simple partial is further categorized according to the presence of symptoms

into

A. Simple partial with

I Motor :

a) Focal motor without march

b) Focal motor with march

c) Versive

d) Postural

e) Phonatory (Vocalisation or speech arrest)

II - Somatosensory or special sensory

a) Samatosensory

b) Visual

c) Auditory

d) Olfactory

e) Gustatory

f) Vertiginous

III with autonomic symptoms

IV with psychic symptoms :

dysphasic, dysmnesic, cognitive, affective, illusion, structural hallucination.

B . Complex partial seizure is catagorised in to

1. Simple partial progressing into impaired consciousness
2. With impaired consciousness at the onset
   a) with impairment in the consciousness alone
   b) with automatism

Charcot used to name simple partial seizure a Bravais – Jackson seizure. The incidence of simple partial seizure among epileptic population is calculated to be around 17%.[4] Partial seizure may run in families. Recent evidence points to linkage of partial epilepsy to chromosome 10q.[5] Patients with history of febrile seizure have the chance of developing later seizure with temporal lobe focus in the adult life in about 5%. Most of the time correct history elicitation helps us to get in to the proper diagnosis and thereby effective management. Because proper history first helps us to localize the side of lesion then further probing regarding the type of seizure whether motor, sensory, psychic helps us to localize to a particular lobe. Then if we still go further in to the history the pattern of movements and the site of involvement and type of progression helps us to further localize to particular area of the lobe. But type of seizure may not always

necessarily correspond to location of lesion as evidenced by the study conducted by JMK Moorthy et al. [6]

Common seizure patterns and their localization : [7]

Clinical type	Localisation
Somatic motor	
Jacksonian (focal motor)	Pre rolandic gyrus
Masticatory, Salivation, speech arrest	Amygdaloid nuclei, opercular
Contra versive	Frontal
Head and eye turning associated with Arm movement or athetoid – dystonic posture	Supplementary motor cortex

Clinical type	Localisation
Somatic sensory	
Somato sensory	Post rolandic
Unformed images, lights, pattern	Occipital
Auditory	Heschl's gyrus
Vertiginous	Superior temporal
Olfactory	Mesial temporal
Gustatory	Insula
Visceral autonomic	Insular – orbital – frontal cortex
Formed hallucination	Temporal Neo cortex or Amygdaloid – hippocampal complex
Dyscognitive experience	Temporal
Affective states	Temporal

Todd's palsy, postictal nose wiping also helps in localization. Postictal nose wiping is usually carried out by ipsilateral hand to the seizure focus.

Patients with prolonged postictal disorientation to time and place – may have a right sided lesion.

Based on seizure and electroencephalographic (EEG) characteristics, age, and evidence of brain pathology, a patient with localization – related / symptomatic epilepsy often can be classified into one of four groups of epilepsy syndromes, according to the presumed cerebral lobe in which seizures originate : temporal lobe, frontal lobe, parietal lobe, or occipital lobe. Extensive and sometimes conflicting literature on cerebral localization using clinical and EEG data exists. This chapter contains a summary of features agreed on by the Commission on Classification and Terminology of the International League Against Epilepsy and some relatively recently described syndromes.

Epilepsies and epilepsy syndromes with onset at all ages and accompanying seizure types.

Localization – related / symptomatic (focal, partial) epilepsies

Temporal lobe epilepsy syndromes (SPS, CPS, TCS)

Frontal lobe epilepsy syndromes (SPS, CPS, TCS)

Pareital lobe epilepsy syndromes (SPS, CPS, TCS)

Occipital lobe epilepsy syndromes (SPS, CPS, TCS)

I. Temporal Lobe Epilepsies :

A. General Characteristics

Simplepartial, complex, or secondarily generalized seizures may occur with onset frequently in childhood or young adulthood. Seizures may occur randomly at intervals, or in clusters. Simple partial seizures are characterized by autonomic or psychic symptoms, or both, and by certain sensory phenomena, such as olfactory and auditory illusions or hallucination. The most common sensation is a rising epigastric discomfort.

B. Routine EEG characteristics :

Routine EEGs may shows

a) no abnormality

b) slight or marked asymmetry of the background activity or

c) temporal spikes, sharp waves, or slow waves (unilateral or bilateral), synchronous or asynchronous; may not be confined to temporal areas.)

C) Sub Types :

1. Amygdala – Hippocampal Seizures

Amygdala - hippocampal seizures are the most common form of temporal lobe epilepsy and generally conform to the general description. Seizures are characterized by rising epigastric discomfort, nausea marked autonomic signs, and other symptoms including borborygmi, belching, pallor, fullness of the face, flushing, arrest of respiration, pupil dilation, fear, panic, and olfactory gustatory hallucinations. Scalp EEG often shows unilateral or bilateral spikes most prominent in the anterior temporal leads.

One variant of amygdale – hippocampal seizures is called the mesial temporal lobe epilepsy syndrome. Such patients demonstrate mesial temporal sclerosis on imaging studies. They typically have a strong family history of epilepsy showing an autosomal dominant inheritance with incomplete penetrance. The patient has seizures(often completed) during infancy or childhood. After a silent period lasting 2 to 15 years, unprovoked partial seizures begin in late childhood or early adolescence. The seizures are refractory to medical treatment in 20% to 30% of patients.

2. Lateral Temporal Seizures

Lateral temporal seizures begin as simple partial seizure characterized by auditory hallucinations or illusions, dreamy state, visual misperceptions, or language disorders (dominant- hemisphere focus). These may progress to complex partial seizures if propagation to mesial temporal or extra temporal structures occurs. Lateral temporal seizures usually lack several of the features typical of mesial temporal seizures, including automatisms, contralateral dystonia, swerving head movements, body shifting, hyperventilation, and postictal cough or sigh. The scalp EEG often shows unilateral or bilateral spikes most prominent in the middle or posterior temporal leads.

A special subtype termed autosomal dominant lateral temporal epilepsy(also called autosomal dominant nocturnal epilepsy) has been reported. Onset is in the second or third decade of life. The subtype is characterized by rare partial seizures, usually secondarily generalized, arising mostly in sleep, simple partial sensory phenomena of visual (lights, colors, simple figures) or auditory (buzzing or humming) sense may occur. Paroxymal activity may be seen in the EEG interictally in the temporal or occipital leads. The condition responds to antiepileptic drugs but may require prolonged administration. Genetic analysis has found linkage to chromosome 10q, locus EBN1, gene KCNQ2.

3. Lateralizing Features in temporal lobe epilepsy:

Useful lateralizing features for the temporal lobe seizures include unilateral clonic activity (seizure focus contralateral in all patients), unilateral dystonic or tonic posturing (seizure focus contralateral in 90% or 86% respectively), unilateral automatisms (seizure focus ipsilateral in 80%) and ictal speech preservation (seizures focus contralateral to the language – dominant hemisphere in 80%) Versive head rotation occurring less than 10 seconds before seizures secondarily generalize predicts a contralateral focus. Ictal speech arrest or postictal speech impairment are associated with a seizure focus ispilateral to the language dominant hemisphere in two-thirds of patients. Postictal speech preservation is associated with a seizure focus contralateral to the language dominant hemisphere in two-thirds of patients. Seizure manifestations not providing reliable lateralizing information include eye deviation, type of aura and versive head movements occurring at time other than immediately before seizures secondarily generalize.

II - Frontal Lobe Epilepsies

A. Clinical Characteristics

Frontal lobe epilepsies are characterized by simple partial, complex partial or secondarily geneararalized seizures or combinations of these. Features suggesting frontal lobe epilepsies are

a) Frequent seizures often in sleep

b) Short seizure duration

c) Minimal or no postictal confusion after complex partial seizure

d) Rapid secondary generalization

e) Prominent motor manifestations that are tonic or postural

f) Complex gestural automatisms (may be sexual) at onset;

g) Frequent falling during seizure; and

h) Frequent episodes of status epilepticus

B) EEG Characteristics

The interictal EEG of frontal lobe epilepsy patients may show

a) no abnormality

b) back ground asymmetry and

c) spikes, sharp waves or paroxymal fast activity that can be

unilateral or bilateral unilobular or multilobular. Patients whose seizures originate from the dorsolateral convexity tend to have interictal epileptiform abnormalities that localize to the region of seizure onset. Patients whose seizures begin in the medial frontal region tend to have either no epileptiform activity or multifocal epileptic form discharges. Vertex or midline epileptiform discharges also can be seen with medial frontal foci. Frontal foci not infrequently exhibit spikes or sharp waves in the temporal leads.

C. Sub types

1. Supplementary Motor Seizures

Supplementary motor seizures are typically brief, lasting only 10 to 40 seconds. The patient develops abrupt tonic posturing of one or more extremities; the arms are affected more often than the legs. Characteristically arms and legs are tonically adducted. During the tonic phase, the patient may cry or moan loudly. Consciousness is usually preserved, but the patient may be unable to speak. A versive movement, usually away from the side of ictal onset, may precede secondary generalization. The tonic posturing may be preceded by sensory symptoms in an extremity. Supplementary motor seizures occur frequently, and for a patient to experience five to ten episodes per day is not rare. Many seizures occur during sleep. Commonly, these seizures are medically intractable.

2. Cingulate :

Cingulate seizure patterns are complex partial with complex motor gestural automatisms at onset. Autonomic signs are common, as are changes in mood and affect.

3. Anterior Frontopolar Region :

Anterior frontopolar seizure patterns include forced thinking or initial loss of

contact and adversive movements of head and eyes, with possible evolution, including contraversive movements and axial clonic jerks, and falls and autonomic signs.

4. Orbitofrontal :

The orbitofrontal seizure pattern is one of complex partial seizures with initial motor and gestural automatisms, olfactory hallucinations and illusions, and autonomic signs. Automatisms may include unformed or formed speech (including expletives) and walking around the room.

5. Combined Mesial Frontal :

Seizures originating in any of the four mesial frontal structures described above sometimes show phenomena described for other mesial frontal structures. Functional spread of discharges likely occurs among the areas.

6. Dorsolateral

Dorsolateral seizure patterns may be tonic, or less commonly clonic with versive eye and head movements and speech arrest.

7. Opercular

Opercular seizure characteristics include mastication, salivation, swallowing,

laryngeal symptoms, speech arrest, epigastric aura, fear and autonomic phenomena. Simple partial seizures particularly partial clonic facial seizures are common and may be ipsilateral. If secondary sensory changes occur, numbness may be a symptom, particularly in the hands. Gustatory hallucinations are particularly common with seizures in this area.

8. Motor Cortex :

Motor cortex epilepsies are mainly characterized by simple partial seizures, and their localization depends on the side and topography of the area affected. In cases of the lower pre- rolandic area, speech arrest, vocalization or dysphasia, tonic clonic movements of the face on the contralateral side, or swallowing may occur. Generalization of the seizure frequently occurs. In the rolandic area, partial motor seizures with march or jacksonian, seizures occur, particularly beginning in the contralateral upper extremities. In the case of seizures involving the paracentral lobule, tonic movements of the ipsilateral foot may occur, as well as contralateral leg movements. Postictal paralysis is frequent.

9. Kojewnikow's Syndrome :

Kojewnikow's syndrome represents a particular form of rolandic partial

epilepsy both in adults and in children and is related to a variety of lesions in the motor cortex. Its principal features are motor partial seizures, always well localized ;

b) often late appearance of myoclonus in the same site at which somatomotor seizures occur

c) an EEG with normal background activity and a focal paroxysmal abnormality (spikes and slow waves)

d) occurrence at any age in childhood and adulthood

e) frequently demonstrable etiology (tumor, vascular) ; and

f) no progressive evolution of the syndrome (clinical EEG, or psychological, except in relation to the evolution of the causal lesion)

10. Rasmussen's Encephalitis

In Rasmussen's encephalitis, a previously normal child, usually approximately 6 to 10 years old, rapidly develops therapy resistant focal seizures, usually motor or sensory – motor, with a slowly progressive motor deficit implicating the same cerebral hemisphere. A mild or moderate mental deficit appears later. EEG shows prominent and persistant arrhythmic delta waves, loss of background features, and abundant spikes. Later, seizures may implicate widely separate portions of the same hemisphere. Pathologic specimens may

show gliosis, inflammation or spongiform changes. The disease may progress to death, stabilize, or improve over time.

11. Autosomal Dominant Frontal Lobe Nocturnal Epilepsy :

Autosomal dominant frontal lobe nocturnal epilepsy is a syndrome in which patients develop nocturnal seizures within the first two decades of life, although the seizures often persist throughout adulthood. The seizures usually occur, during sleep, but in severe cases seizures may occur while awake. The clinical features are similar to other frontal epilepsies. A nonspecific aura that may include somatosensory, special sensory, psychic, or autonomic, phenomena is commonly present. After the aura, gasping, groaning, other vocalization, prominent motor phenomena (thrashing, hyperkinetic movements, tonic stiffening,clonic jerking) or reflex agitation with rapid changes in position may occur.

The gene defect has been mapped to chromosome 20q, 13.2q 13.3 is one family, although other affected families do not share this locus. The known mutation results in phenylalanine replacing serine at codon 248 (ser 248 phe) in the gene for the 2-4 subunit of the neuronal nicotinic acetylcholine receptor, which may deleteriously affect receptor function.

III PARIETAL LOBE SEIZURES :

A. General characteristics :

Parietal lobe epilepsy syndromes usually are characterized by simple partial and secondarily generalized seizures. Most seizures remain simple and exhibit sensory phenomena. Most frequently, seizures are of the anterior parietal subtype.

B. EEG Characteristics :

Interictal EEGs may show a) Normal results b) focal slowing or c) focal spikes and sharp waves that are unilateral or bilateral synchronous or asynchronous. Slow and sharp activity spreading beyond parietal leads is not common. Vertex or midline epilepti form abnormalities can be seen with somatosensory seizures arising from the mesial surface of the parietal lobe.

C . Subtypes :

1. Anterior Parietal Seizures

Anterior parietal seizures involve the posterior central gyrus and are predominantly sensory with positive or negative phenomena. Positive phenomena may include tingling; a feeling of electricity ; desire to move a body part; the sensation that a body part is being moved ; tongue or facial sensations, or both and pain. Negative phenomena include loss of muscle tone, numbness, the feeling that a body part is absent, or loss of awareness of a part or one half of the body (asomatognosis).

The parts most frequently involved are those with the largest cortical

representation (hand, arm, face) and seizures may spread along the posterior central gyrus, producing a jacksonian march of symptoms as adjacent structures are progressively affected.

2. Posterior Parietal Seizure :

Posterior parietal seizures are frequently accompanied by prominent staring and relative immobility. Visual phenomena may occur, including formed hallucinations and metamorphopsia (visual distortions and confusion.

3. Inferior Parietal Seizures

Inferior parietal seizures may demonstrate severe vertigo and dis orientation in space, and abdominal sensations.

4. Paracentral Seizures :

Paracentral seizures may demonstrate contralateral genital sensations or rotary or postural motor activity, and have a tendency to become secondarily generalized.

5. Dominant Hemisphere Parietal Seizures.

Seizures arising from the dominant parietal lobe may demonstrate receptive or conductive language disturbances

6. Nondominant Hemisphere Parietal Seizures

Metamorphopsia and asomatognosia often indicate involvement of the nondominant parietal lobe.

IV – OCCIPITAL LOBE SEIZURES

A. General Characteristics :

Occipital lobe seizures are characterized by positive and negative visual phenomena. Positive phenomena include elementary visual hallucinations often described as bright lights or colored lights. Negative phenomena include amaurosis, scotomas, and hemianospsia. The visual phenomena usually are contralateral to the side of the seizure and may remain stationary or move across the field. Persistent (hours) amaurosis can be a postictal phenomenon.

Other occipital seizure manifestations include tonic and clonic eye deviation, head deviation, blinking, a sensation of eye movement, and nystagmoid eye movements. Eye and head movements usually are contralateral to the side of the seizure focus in occipital seizures (this may not be the case for seizures arising in other areas)

B. ECG Characteristics

Surface EEGs most often demonstrate extensive posterior temporal occipital paroxysmal activity. This pattern may be difficult to distinguish from temporal lobe epilepsy of posterior temporal origin.

C. Subtypes

Seizure discharges within the occipital lobe produce a limited number of signs and symptoms. However, such discharges may spread to the temporal, frontal, supplementary motor, or parietal areas and produce seizures typical of these areas. The most common mode of spread is infrasylvian to the ipsilateral temporal lobe, producing automatisms typical of temporal lobe seizures (psychoparetic, psychomotor). Visual signs or symptoms at onset of a seizure suggest occipital origin.

D. Evaluation :

The approach to benign occipital epilepsy of children is discussed. Symptomatic (due to a structural lesion) occipital epilepsy is suggested by the presence of neurologic deficits, limbic spread of seizures, and short and frequent seizures. Structural lesions causing occipital seizures include developmental malformations, perinatal lesions, post traumatic lesions, strokes, tumors, Sturge Weber syndrome, epilepsy with bilateral occipital calcification, microchondrial

disorders (mitochondrial encephalomyelopathy with ragged red fibers and stroke like episodes (MELAS), myoclonic epilepsy and ragged red fibres (MERRF) and Lafora's disease (neural ceroid lipofuscinosis).

If a structural lesion is suspected, magnetic resonance imaging is preferred. Developmental anomalies (a common finding) do not show well on computed tomography, and magnetic resonance imaging is the preferred imaging technique.

Regarding the etiological aspect of partial seizure, if review we find some interesting features. Etiology almost encompase the following headings (developmental anomalis, CNS infection, Trauma, Brain Tumor, Cerebrovascularaccidents, degenerative and others. Percentage of these groups among themselves as a cause of partial seizure varies in different age groups.

Age in years	AnomalyDev.	Infection	Trauma	Tumor	CVD	rativeDe gene	Others
0-4	43	26	14	0	8	0	9
5-14	32	38	15	10	5	0	0
15-24	16	20	44	10	4	0	6
25-44	11	12	23	30	14	10	0
45-64	1	0	10	25	54	8	2
Above 65	0	0	4	6	78	10	2

In a study by Armstrong DD, Mizrali EM et al particularly in children in a western population they found more than 50% had mesial temporal sclerosis as an etiology for partial seizures. MTS refers to the pathologic entity of hippocampal sclerosis and atrophy which is often visible on imaging studies with loss of Neurons in the CA1 region and end folium CA3 / CA4 but with relative sparing of the CA2 region. The etiology of mesial temporal sclerosis is controversial. Evidence suggests that prolonged seizures including prolonged febrile seizures may cause mesial temporal sclerosis. Congenital lesions principaly hamartomas, heterotopias and focal cortical dysplasia (FCD) account for 15% to 20% of recognized lesions and are particularly common in children. Neoplasms account for 10-15% of recognized lesions particularly glial based. Trauma accounts for 5-10% of pathological findings. This is a pathological exam based study.

An Indian study based on CT examination in partial seizure by Misra et al in the year 1994, showed that CT was abnormal in 79.3% of patients with partial seizure. Contrast enhancing granuloma was the most common lesion and accounts for about 63.3%. Next comes calcification which is noted in 11.8%

Cerebral atrophy - 5.9%

Mass - 4.6%

Infarct - 4%

Oedema - 1.9%

Vascular malformation- 0.8%

Haemorrhage - 0.8%

Gliosis - 0.5%

In a study by Hussian et al in partial seizures in children contrast enhancing granulomas account for about 73.5% of patients.

In an Indian study of 100 consecutive surgical specimen from cases operated for medically refractory CPS, by Radhakrishnan et al showed 58 patients had Ammon horn sclerosis, corpora amylacea deposition in 54 patients, 6 patients had neoplasm, 31 patients had non specific changes. [8]

PROGNOSIS OF PARTIAL SEIZURES AND LOCALIZATION RELATED / SYMPTOMATIC EPILEPSIES

A. First Unprovoked Seizure :

The risk of seizure recurrence by 36 months is 25% in persons with no risk factors after having a first unproved seizure. In persons with risk factors, the risk of seizure recurrence usually is much greater. Risk factors include evidence of prior neurologic insult (determined by history, neurologic examination, imaging studies), abnormal EEG, and multiple seizure or status epilepticus as initial event.

Treatment after first partial seizures remains controversial because of the uncertainty regarding the risk of another seizure and the side effects of antiepileptic medication. However, randomized clinical trails do indicate that antiepileptic drugs reduce risk of seizure recurrence.

B. After two or More Unprovoked Seizures :

Persons with two or more unprovoked seizures almost always are treated. The two Veterans Administration Cooperative studies indicate that 35% to 60% of adult patients with partial seizures will have complete seizure control after 1 year with carbamazepine or phenytoin monotherapy as the initial and only treatment.

Satisfactory results (an acceptable number of seizures or none, acceptable side effects) are obtained in approximately 70% of patients with a single drug, either the initial choice or an alternative. Thirty percent of patients have inadequate control despite trials of several drugs used alone. When a second drug is added, another 10% are satisfactorily controlled. With a third drug, another 5% are satisfactorily controlled. Approximately 15% of patients are not controlled after trails of three or more drugs. Such patients are considered medically refractory.

Risk factors for poor control of partial seizures include :

Abnormal EEG,

Evidence of a structural brain lesion,

Number and duration of seizures before diagnosis and before control with medication,

Neurologic deficit from birth, and

Secondarily generalized tonicclonic seizures.

C. Mortality :

1. General :

Available studies are not optimal but generally report increased mortality in patients with symptomatic epilepsies. This mortality is caused, at least in part, by the underlying symptomatic disease (congenital malformations, tumors, cerebrovascular disease) and its complications. Studies regarding increased rate of suicide are conflicting.

2. Sudden Unexplained Death :

The risk of sudden unexplained death is between 1 in 500 and 1 in 1,100 person years for all persons with epilepsy, and 1 in 200 person – years for patients with refractory seizures. The risk is greater for persons between the ages of 15 to 45 years with poorly controlled tonic-clonic seizures (usually secondarily generalized). Structural lesions and severe or frequent seizures appear to be risk factors. Available evidence suggests that most sudden deaths are temporally

related to seizures and often occur in sleep. Postulated mechanisms include cardiac arrhythmias, pulmonary edema, and suffocation.

D. Neuropsychologic Function :

Animal studies suggest that repeated partial seizures may result in neuronal damage. Human studies are difficult to evaluate because of the confounding effects of the original structural lesion, antiepileptic drug effects, and impaired social adjustment. The effects of repeated partial seizures on neuropsychologic function remain unknown.

Investigations in Partial Epilepsy :

Electroencephalography :

Next thing which helps us to localize in the evaluation of seizures is Electroencephalography, EEG is complementary to CT & MRI. Friederick Gibbs, Halloweel Davis (1934) and William G. Lennox (1935) first demonstrated spike and wave complex interictaly and also ictally. We are all indebted to Prof. Hans Berger, a Professor of psychiatry of university of Gena for his discovery that spontaneous electrical activity of brain can be recorded from the scalp in the year 1929.

Gibbs & Lennox demonstrated focal spikes in localization related epilepsy – 1936

First use of closed circuit TV for simultaneous recording of the EEG and seizure was reported by Golden sohn – 1966.

EEG spikes at the brain surface were found to be the summation of depolarising and hyper polarising post synaptic potentials.

Paroxysmal depolarization shifts are the essential feature of the spike focus. By intra cellular recording Matsumo in the year 1964 demonstrated that PDS from a column of Neo cortex as small as 2 mm is sufficient as a focus.

Focal spikes, focal polymorphic delta waves, frequency difference between 2 hemisphere > 1 Hz, Focal slow waves (FIRDA), occipital sharp waves, phase reversal, onset of spike wave in EEG have localizing value.

Polymorphic delta waves	-	Superficial space occupying lesion
Rhythmical slow waves	-	Deep SOL
FIRDA	-	Midline tumours
Frontal sharp waves	-	Jacksonian or versive
Parietal sharp waves	-	Versive or sensory
Anterior Temporal sharpwave	-	Complex partial
Midline sharp waves	-	Simple partial
PLEDS	-	Epilepsia partialis continua

When a patient has got only partial motor seizure the chance of EEG being abnormal is only 33% if he has pure simple partial with only sensory chance of

being abnormal is only 15%.

If EEG of a person shows anterior temporal spike or sharp wave it is strongly associated with the occurrence of clinical focal onset of seizure. When this pattern is seen on EEG likely hood of the person developing seizure is 98%. Converse is not true – proved in study by Joseph F.Hulihan M.D. mentioned in his article focal EEG wave form abnormalities.[9]

Focal polymorphic delta waves were often (68%) associated with focal structural lesion in CT brain. Stroke being most frequent etiology. [10]

In patients with normal CT brain in a study of 100 cases of focal delta activity convulsion itself was the most common cause with the exception of Mesial Frontal lobe epilepsy. Ictal EEG recordings are very useful in the localization / lateralization of focal seizures. Even during Ictal EEG recording occipital lobe epilepsy is often localized falsely (28%).[11] Some studies revealed that many patients with generalized tonic clonic seizure have focal features. Structural, functional imaging studies as well as histopathological studies have shown presence of focal brain abnormality in patients with generalized epilepsy. [12]

In many situations EEG in partial seizure may be totally Normal. Hence if it is abnormal above mentioned abnormal wave forms may help us to localize. On

the contrary Normal EEG doesn't rule out focal onset. Many a time the surface EEG is normal in partial seizure, because a critical area of 6 cm^2 has to be involved for the scalp electrode to pick up focal abnormality in the EEG. In a study by Quesney et al who has examined surface and subdural recording from 34 patients with frontal lobe epilepsy who were subsequently rendered seizure free after surgical resection of epileptogenic foci, they found focal spikes in 9%, No spikes in 12%, 60% had more widespread or generalized spikes.

When a patient has got only partial motor seizure the chance of EEG being abnormal is only 33%, if he has pure simple partial seizure with only sensory, the chance of EEG being abnormal is only 15%.[13]

EEG as a tool for prognosis assessment has been studied in some papers published in International Journals, both with drugs and epilepsy surgery cases. Focal spikes and focal slow waves in every area is much frequent in patients with uncontrol seizures than in patients who has very good control with antiepileptic drugs. People with frontal spikes also had poorly controlled seizures. This is shown in a work done by Jhon Hughes by reviewing 804 EEGs. [14] Patients with MRI and EEG findings which are concordant are more likely to be seizure free when compared to those who has a discordant when compared to those who has a discordant relationship. About 72% of patients were seizure free after surgery

when they have concordant finding compared to 41% those who had discordant findings. [15]

When the EEG of a patient with partial seizure is abnormal the chance of getting an abnormal CT brain is about 57.8 %.[16] Limitations of EEG with evaluation of seizures.

1. Standard scalp electrodes record from as little as 1/3 of cortex
2. Deep cortex are after too distant for scalp electrodes.
3. Scalp and skull serve as special averages further hindering EEG interpretation

Wicket rhythm may be mistaken for epileptiform changes in temporal lobe. [17]

Magneto encephalogram and EEG complement each other for the detection of interictalepileptiform discharges. EEG offers the advantage of long term recording significantly increasing its diagnostic yield which is not feasible with MEG. MEG is more sensitive for the detection of Neocortical spike sources and can clarify the spatial relationship between the irritative zone and structural lesion.

In the evaluation of focal seizure one interesting feature in EEG is mirror focus. Mirror focus does not alter the prognosis after the epileptic surgery

Neuro Imaging :

Next investigation which comes to our help is neuroimaging. Both CT brain and MRI brain help us to identify the lesions producing epilepsy.

Prior to the advent of CT and MRI plain skiagrams, pneumoencephalgraphy, radioactive isotope imaging and arteriography were used to identify structural lesion. Many of them were invasive and therefore with complication. In 1970's with computerized tomography being introduced into the world of Neurosciences it has revolutionized the diagnostic aspects in Neurology. We can identify many etiologies causing epilepsy baring a few like Neuronal migrational disorders. Now with the availability of CT scan throughout the country it has been utilized to screen patients with epilepsy. There were many studies with CT scan brain with regard to epilepsy both Nationally and internationally.

In one study by Hussein et al published in the year 2004, CT brain was abnormal in 68% of their study population ie., patients with partial seizure. [18]

In their study 75/102 positive CT lesion cases showed single ring enhancing lesion. Parietal lobe being the commonest site for SCECTL in their study. Partial seizures with or without secondary generalisation being the commonest seizure type in patients with single contrast enhancing granuloma. This is shown by Chopra et al 1992. [19]

Vedhantham Rajasekar, Chandy et al has proposed following CT criteria

to diagnose Neuro cysticercosis.

1. Solitary
2. Enhancement with contrast
3. Less than 20 mm
4. Perilesional sedema may be present or may not be but if present it must be with out midline shift

With thin slice contrast CT scan brain sensitivity of detecting such lesions is about 98%. Hence he concluded contrast CT is a reliable and cost effective modality to diagnose one of the commonest cause of seizure in this part of world. He even, quotes that "use of CSF for Immunological test to diagnose NCC is not recommended when clinical and CT features are generally straight forward. [20] Misra et al in the year 1994 in their study of CT observation in partial seizures done at BHU – varanasi, they found CT was abnormal in 79.3 % of patients with partial seizures. Commonest lesion being focal disc or ring enhancing lesion (63.3%) followed by calcification (11.8%)[21] Zee et al in the year 1980 were the first to report solitary contrast enhancing granuloma on CT scan brain. SCG are classified as disc enhancing, ring enhancing, and doughnut lesions.

Disc lesion - Uniform enhancement

Ring lesion - Peripheral enhancement with central hypodensity

Doughnut lesion - Peripheral enhancement occupies much greater area leaving a small central hypodense area.

Dot inside the lesion represents the scolex. Cases with scolex inside the lesion responds better to albendazole therapy.

All these lesions if solitary are known as Type A lesions.

If there is a combination of disc and ring or 2 discs or 2 rings in a single CT brain it is called as Type B lesion with regard to NCC.

MRI, PET, SPECT are also used in the field of epileptology, particularly with the introduction of epilepsy surgery. Role of MRI is very useful in identifying lesions which could be easily missed in CT evaluation like Neuronal Migrational disorders and vascular malformations. Sensitivity of MRI approaches 100 % in tumour, vascular malformations, infarcts, granuloma.

But not only conventional MRI but also some newer techniques like quantitative T2 relaxometry, diffusion Tensor imaging, double inversion recovery, fast flair T2 image, Magnetization transfer technique, MR spectroscopy has to be used in patients particularly having refractory partial seizures before subjecting to epilepsy surgery, because conventional MRI may fail to identify a cerebral lesion in 20% of patients with refractory partial seizure. Quantitative evaluation of T2

images is more sensitive and objective than visual assessment for identification of subtle pathologies. [22,27] Role of MRI is valuable in the diagnosis of mesial temporal sclerosis, Focal cortical dysplasia. 90% of patients with non lesional temporal lobe epilepsy localization of ictal onset zone is in the amygdala or hippocampus.[23] Visual inspection of the MRI for atrophy of Mesial Temporal lobe has been shown by many investigators to be satisfactory in most patients with mesial temporal lobe sclerosis. Signal intensity is mesial temporal lobe is altered in quantitative T2 relaxometry even if no visible change in the size of mesial temporal lobe.

Patients with MRI identifiable structure lesion may be triaged to epilepsy surgery early in the course of treatment if it is clear that the initial response to anti epileptic drugs are disappointing.

PET studies utilizing FDG is mainly used in Pre surgical evaluation of patients with refractory partial seizures. It shows diffuse / regional hypometabolism in 90% interictal recording and some regional hypo perfusion. Periictal PET shows diffuse / regional hyper metabolism. Presence of temporal hypometabolism and absence of extra temporal cortical hypometabolism predicts best outcome in temporal lobe epilepsy.[24] Pseudo PLEDS on scalp EEG can be associated with focal hypermetabolism even in the absence of overt seizure. This suggests in some

who are not experiencing clinical seizure manifestation of PLEDS may be an ictal rather than a interictal EEG pattern. [25]

f MRI has also been utilized in Pre surgical evaluation to identify eloquent areas.

^{11}C Flumazanil PET is a newer technique which gives higher yield than the Advanced MRI technique in picking up the epileptogenic areas. Carbon 11 labeled flumazenil is a marker for the functional integrity of the GABAnergic inhibitory system. Loss of GABAnegic blinding by ^{11}C flumazanil PET shows the areas of epileptogenic zone. But its high yield is not directly transferable in assessing the surgical results. There is a study by Mathias J Koepp et al. Among 102 patients, with MRI negative partial seizures 71 showed ^{11}C flumazanil PET abnormality but these findings were of use for surgery only in 25% of patients. [26] Because these picks up some white matter abnormalities like micro dysgenesis (increased density of heterotopic white matter neurons) which are not easily respectable by surgery. Moreover epileptogenic zone is the area of cortex necessary for seizure generation, which according to Rosenow no technique helps us to measure it directly and accurately. [27] Area of seizure onset might be same or smaller and sitting inside the epileptogenic zone.

Management :

The incidence of focal epilepsy in Western population is estimated to be about 0.4 %. Patients with partial seizure who doesn't have an identifiable lesion have best prognosis.

Satisfactory results (an acceptable number of seizures or none, acceptable side effects) are obtained in approximately 70% of patients with a single drug, either the initial choice or an alternative. Thirty percent of patients have inadequate control despite trials of several drugs used alone. When a second drug is added, another 10% are satisfactorily controlled. With a third drug, another 5% are satisfactorily controlled. Approximately 15% of patients are not controlled after trails of three or more drugs. Such patients are considered medically refractory.

Besides patients without structural lesion patients with SCG also respond well to Medical Management. Focal seizures in younger age groups have a better control over older age group.

Usually patients with SCG needs short term AED only for about 6 months. [28] But people with calcified granuloma need prolonged AED. In such cases therapy has to be individualized. Role of albendazole in SCG is been much debated. CT brain with ring enhancing lesions with dot inside respond better with albendazole. In other cases spontaneous revolution is possible, needs only AED

and repeat CT brain after 3-6 months as per Rajashekar et al studies at CMC vellore. Gliosis around focal cerebral calcification as seen in T1 Magnetisation Transfer MRI is a prediction of poor seizure control.

Patients with acute symptomatic partial seizures due to metabolic insults like hyperosmolar nonketotic coma, hypocalcemia responds very well to the correction of underlying abnormality. They don't need long term AED therapy.

The following anti epileptic drugs are commonly used in the treatment of partial seizures. They are carbamazepine, oxcarbazepine, Gabapentin, phenytoin, Sodiumvalproate. Topiramate, Lamotrigine, Tiagabine, Leviteracetam. Carbamazepine or phenytoin is currently the initial drug of choice for the treatment of partial seizures including those with secondary generalization. Carbamazepine is preferred because of its pharmaco kinetics and toxicity profile. [29] Valproic acid is an effective alternative for some patients when particularly the seizure secondarily generalize.

Gabapentin as an add on therapy in partial seizure in patient who are not responding to monotherapy is been well studied. In India, Prof. Dhanraj has established its role as an add on therapy in partial seizure in his paper published in the year 1998. [30] Gabapentin is unique in that it does not have any significant drug interaction.

Lamotrigine is almost effective in all sub types of partial seizure. [31] Lamotrigine appears to have an over all efficacy profile similar to the more standard drugs and is now being used as mono therapy and also can be used as an add on therapy. When used as an add on therapy dose should be started as a lowest possible and slowly titrated up.

Antiepileptic Drugs of Choice :

Seizure type	Drugs of first choice	Drugs of second choice	Alternative drugs
Partial (simple, complex, secondarily generalized tonic-clonic)	Carbamazepine Phenytoin	Gabapentin Lamotrigine Topiramate Valproic acid	Phenobarbital Primidone Tiagabine

	Responder Rate	No Serious toxicity	No Nuisance toxicity	No drug inter action	Adminis trations
Gabapentin	30-40%	+	±	+	t.i.d
Lamotrigine	30-40%	-	±	±	b.i.d
Phenobarbital	?	+	-	-	q.d
Primidone	?	+	-	-	t.i.d.
Tiagabine	20-30%	-	-	-	b.i.d or t.i.d
Topiramate	40-50%	-	-	+	b.i.d
Valproic acid	30-40%	-	-	-	b.i.d or t.i.d

Patients with MRI identified structural lesions may be triaged to epilepsy surgery early in the course of treatment if it is clear that the initial response to AED is disappointing for the following reasons.

1. The longer the patient lives as a disabled epileptic, the more difficult it is to become fully functional after successful epilepsy surgery.
2. Evidence suggests that seizures may cause brain damage and worsen a seizure disorder and neuropsychological handicaps.
3. Evidence also suggests that seizures may have adverse effects on the developing brain

Epileptic Surgery :

The following surgeries are done for patients with medically refractory seizures.

1. Temporal lobectomy
2. Non temporal resections
3. Corpus callosotomy
4. Hemispherectomy
5. Subpial resections
6. Lesionectomy

Indications and Contraindications for epilepsy surgery procedures

Indications	Medically intractable seizures (necessary) Seizures significantly reduce quality of life (necessary) Localised seizures focus (helpful) Biological predictors of seizures persistence (helpful)
Contraindications	Benign, self-limited epilepsy syndromes. Neurodegenerative and metabolic disorders Non compliance with medication Severe family dysfunction Psychosis.

In a statistics from a tertiary referral centre in India showed that about 74% of patients with intractable seizure referred to surgery were suffering from partial seizures. [32]

Clear identification and complete resection of epileptogenic focus will result in good out come. Almost 90% of people will become seizure free in Mesial temporal lobe epilepsy. [33] In children operated for epilepsy with tumours, after the resection of tumour almost 90% of them became seizure free. [34] In the study

at CMC vellore by Danial et al over 40 years period showed, total or neartotal control in 53% of patients and worth while outcome in another 25%.[35] In western centres they have noted 70-80% seizures control after epileptic surgery.

Post operatively patient generally need to remain on antiepileptic drug therapy but the marked reduction of seizures following surgery can have a very beneficial effect on their quality of life

MATERIALS AND METHODS

This study was conducted in the Department of Neurology, Govt. Rajaji Hospital, Madurai Medical College, Madurai in the year 2004 – 05.

Cases are selected on a Random basis attending our OPD. Detailed history and clinical examination is carried out to ensure the organic nature of epilepsy.

A proforma formulated by the Post graduate and accepted by Professor of Neurology is used to collect the data – proforma enclosed.

An eight channel EEG machine present in our department is utilized for recording of EEG with all routine activation procedures. All the EEGs are critically analyzed for the presence of focal, localized or generalized changes by montage wise analysis. Individual abnormalities are recorded in the proforma.

CT scan brain plain and contrast axial section with routine slice thickness performed in all cases. Radiologist's opinion obtained. abnormalities noted.

Finally the data was analyzed combining the clinical, EEG and CT scan brain findings and conclusion arrived.

OBSERVATION

In our study I have examined 76 patients of which 3 did not turn up for CT brain and EEG, So a drop out of 3 cases. Finally the study included 73 cases, in them detailed history, clinical examination and Investigations were completed.

In our study population, children under 13 years were 28 in number. Adults under 45 years were 38 in number. Adults more than 45 years were 7 in number. Study group youngest patient was 9 months old baby. Eldest person was 70 years old.

Among the Total 73 cases 29 patients had simple partial seizures, 42 patients had complex partial seizure and 2 patients had both simple and complex partial attacks.

Duration of illness before reporting for Medical advice

Less than 1 week	-	18
Less than 1 month	-	33
More than 1 month	-	22

Right focal seizure was noted as 43 patients and left focal seizure is 30 patients. When we analyzed the symptomatology of our patients headache was the most frequent symptom and it was reported in 25 cases. 31 patients out of the 73 cases

had clinical signs of deficit. (42.5%).

Among the clinical signs hemiparesis was seen in 14 patients (19.2%) among these patients CT scan brain was abnormal in 12 patients (85.87%).

5 patients had papilloedema(6.8%). CT brain was abnormal in all the 5 patients (100%).

3 patients had hemisensory deficit (4.1%) and CT brain was abnormal in 2 patients (67%)

3 had facial weakness of upper motor neuron type (4.1%). CT brain was abnormal in 3(100%)

3 had extensor plantar (4.1%) –CT was abnormal in all the three patients. (100%)

2 patients had homonymous hemianopia (2.7%) in these CT brain was abnormal in both (100%)

One patient had paraparesis 1.4% in whom CT was abnormal showing a suprasellar mass lesion.

In toto among the 73 patients who were examined 31 patients had deficit which amounts to 42.5%.

28 / 31 patients with signs of Neurological deficits postictally had structural lesions in their CT brain.

STATISTICAL SIGNIFICANCE OF POSTICTAL NEUROLOGICAL DEFICITS IN PREDICTING CT BRAIN LESIONS

CT BRAIN LESIONS

CLINICAL SIGNS OF DEFICITS

	PRESENT	ABSENT	TOTAL
PRESENT	28	3	31
ABSENT	19	23	42
TOTAL	47	26	73

X^2 - 15.01

P - 0.000072

P < 0.01 Significant

Sensitivity - 59.57 %

Specificity - 88.46%

In our study, CT brain was abnormal in 47 patients (64.4%). 28 patients among these 47 patients had deficits on clinical examinations (59.6%). 19 patients with CT brain abnormality didn't show any deficit.

EEG was abnormal in 40 cases (54.79%) Among these generalized changes were present in 14 patients (19%) lateralizing changes were present in 26 cases (35.6%)

Among the patients with granuloma EEG was positive in 16 cases. So

Totally among 26 cases of ring enhancing granulomas as evidenced in CT brain, 16 patients had EEG abnormality (61.5%). Interestingly 15 patients had shown lateralizing EEG abnormalities. (93.75%). Predominantly granulomas were seen in younger population. Among the 26 cases 23 cases

were at or under 18 years of age, 3 were in their 20-35 years of age group.

Infarct was seen in 10 cases here to parietal lobe was the commonest site. 8/10 cases shown the infarct in the parietal lobe

EEG was abnormal in 4 cases (40%) of which 3 had lateralising EEG changes and one showed generalized changes.

Among the 4 cases with mass lesion, EEG was abnormal in 2 cases both of them showed generalized changes.

Patient with tuberous sclerosis also showed generalized EEG changes.

Patient with AVM showed lateralizing EEG changes. Patient with calcification and gliosis didn't show any EEG abnormality.

So, totally among the 47 patients with CT abnormality 25 had shown abnormal EEG (53%)

Post ictal oedema is reported in 2 cases. Both in young age group. One confirmed by repeat CT brain and other with MRI scan. Both didn't show any EEG abnormality.

Among the CT brain lesion – Ring enhancing granuloma was the most common lesion reported in 26 cases. (55.3%). Among these single contrast enhancing ring lesion was seen in 18 patients, disc enhancing lesion in 5, Double ring enhancing lesion in 2 and multiple ring enhancing lesion in 1. 2 patients had shown scolex.

Infarct was seen in 10 cases (21%). Mass lesion was reported in 4 cases (8.5%). Post ictal oedema in 2 cases (4.2%), gliosis, calcification, Arterio-venous malformation, Tubers, diffuse gyral enhancement each in one case (others 10.6%)

Granulomas are most commonly reported in the parietal lobe.

Among the 26 cases with granuloma, 21 patients had lesions in the parietal lobe. Distribution of granuloma was in the following order, Right parietal in 9 and left parietal in 12. In 3 patients they were seen in the temporal lobe of which one was on the right side and other 2 on the left side. Among the other two cases, one case had the lesion in the right frontal lobe, other showed multiple lesions.

S.No.	Clinical Deficit	No.of patients	% of study
1	Hemiparesis	14	45.16
2.	Papilloedema	5	16.13
3.	Hemisensory impairment	3	9.68
4.	UMN facial weakness	3	9.68
5.	Extensor plantar response	3	9.68
6.	Homonymous hemianopia	2	6.45
7.	Paraparesis	1	3.22

CT Lesion

S.No.	CT lesion	No.of patients	% of study
1.	Contrast enhancing granuloma	26	55.31
2.	Infarct	10	21.27
3.	Mass	4	8.51
4.	Postictal oedema	2	4.25
5.	Calcification	1	2.12
6	AVM	1	2.12
7.	Tubers	1	2.12
8.	Gliosis	1	2.12
9.	Diffuse gyral enhancement	1	2.12
		47	100

Among 26 contrast enhancing granulomas

Single ring enhancing lesion - 18

Disc enhancing lesion - 5

Double ring enhancing lesion - 2

Multiple ring enhancing lesion - 1

Distribution of granulomas

Right parietal - 9

Left parietal - 12

Temporal - 3

Frontal - 1

Multiple - 1

Following were the EEG abnormalities noted

1. Phase reversal - 11 cases
2. Bilateral spike, sharp waves - 14 cases
3. Focal or unilateral sharp waves- 8 cases
4. Focal slow waves - 7 cases

Distribution of infarcts in the CT brain

Parietal - 8

Temporal - 1

Occipital - 1

Distribution of mass lesion

Suprasellar - 1

Frontal - 1

Parieto occipital - 1

Fronto temporal - 1

Others

AVM - Parietal

Calcification - Parietal

Gliosis - Occipital

Tubers - Periventricular

Interestingly among the 47 cases with CT lesions almost 31 cases lesions were seen in the parietal lobe.

Age wise distribution of various pathologies :

Neuro infection - 2 to 32 years

Most of them below 18 years old only 2 cases > 25 years

Median - 12 years

Mean - 12.5 years

Infarct - 20 to 61 years

Median - 35 years

Mean - 43 years

Mass - 5 – 50 years

Median - 30 years

Mean - 29 years

Post ictal oedema - < 21 years

AVM - 34 years

Calcification - 15 years

Gliosis - 25 years

Tuber - 6 years

Diffuse gyral Enhancement - 10 years

Incidence of structural lesion - Morethan 45 years - 86%

14 – 45 years - 56.4 %

< 13 years - 63.3%

EEG :

Among the patients with phase reversal CT was abnormal in 81%

Among the patients with Focal / unilateral sharp waves

- CT was abnormal- 75%

Among the patients with Focal slow waves

- CT was abnormal - 71%

Among the patients with Bilateral spike / sharp waves

- CT was abnormal - 42.8 %

EEG was abnormal in 40 cases (56.2%). Lateralizing EEG changes were noted in 26 cases, among these patients CT brain was abnormal in 20 cases. (76.92%)

Bilateral changes were noted in 14 cases, among these CT was abnormal in 5 (35.7%).

ST/ STICAL SIGNIFICANCE OF EEG CHANGES IN PREDICTING CT BRAIN LESIONS

LATERISING EEG CHANGGES

CT BRAIN LESIONS

	PRESENT	ABSENT	TOTAL
PRESENT	20	6	26
ABSENT	27	20	47
TOTAL	47	26	73

X^2 - 8.71

P - 0.0032

P < 0.05 Significant

Lateralising EEG changes predicting CT brain lesion

Sensitivity of predicting CT brain lesion - 42.55 %

Specificity for predicting CT brain lesion- 76.92 %

GENERALISED EEG CHANGES PREDICTING CT BRAIN LESIONS

CT Brain Lesions

GENERLISED EEG CHANGGES

	Present	Absent	Total
Present	5	9	14
Absent	42	17	59
TOTAL	47	26	73

X^2 - 6.21

P - 0.0127

Significant P < 0.05

Generalised EEG changes predicting CT brain lesion

Sensitivity - 10.64%

Specificity - 65.38 %

EEG was most commonly abnormal in patients with infective pathology than any other conditions. Lateralising EEG changes were also common in patients with infective pathology.

DISCUSSION

Overall we have studied 73 cases. Clinical history is taken as the tool for the diagnosis. Strictly conversion and other seizure mimics have been excluded.

8 channel EEG which is available in our department was utilized in the study. Generalized EEG epileptiform activity and lateralizing EEG abnormalities were particularly scrutinized. Among the lateralizing changes, phase reversal as defined as abnormal wave forms of opposite polarity in adjacent Bipolar montages. Focal spikes, sharp waves as defined by the duration < 70 m/sec, 70-200 m/sec respectively. Slow waves as defined by frequency less than 8 / sec were taken into consideration while interpreting the abnormality.

Computerized Tomography of brain was done in all 73 cases. Both plain and contrast CT brain axial section with normal window length / breadth with 10mm anterior and 5mm posterior cuts were taken. Radiologists opinion

obtained in all cases. Vedhantham Rajasekar's criteria for NCC utilized. Ring enhancing lesion is defined as peripheral thin rim of enhancement with central hypodensity and disc enhancing lesion is defined as uniform enhancing lesion in entirety. Mass lesions are not categorized in to individual tumour.

The incidence of partial seizures is almost equally distributed among male and female population. Complex partial motor seizure was the commonest entity. CPS : SPS = 1.5 : 1

Incidence of partial seizure is more common in younger population than in older. More frequent in young adults and children.

When compared to generalized seizures, partial seizures are known to produce more clinical signs when evaluated postictally. In our study 31 cases out of the 73 (42.46%) had positive clinical signs. Hemiparesis was the most frequent deficit noted. Hemisensory, UMN facial weakness, homonymous hemianopia, papilloedema, extensor plantar response alone, paraparesis were also noted. We have statistically analysed whether people who has a deficit or sign on clinical examinations have a higher chance of harbouring a structural lesion in their brain when compared to those who don't have any deficit. And it was proved that those who have a deficit have more incidence of structural

lesion than those who don't. Among the patients with clinical signs or deficit postictally, 28/31 had structural lesion on CT brain. So, we conclude that patients with deficits are more likely to have abnormal CT brain with sensitivity of 60% and specificity of 88% with a statistically significant p value of 0.000072. Headache was the most common symptom reported by our patients. This history was given in 25 cases.

Among the 73 cases studied, EEG was abnormal in 40 cases (56.2%). 25 out of 40 patients with EEG abnormality had CT brain lesions. (62.5%). This is almost comparable to an Indian Study done in 2003 by Ramesh Bahti et al. In their study 57.9% of cases with EEG abnormality had abnormal CT brain. [15]

Generalized EEG changes were noticed in 14 cases and lateralizing EEG changes in 26 cases. We have analyzed the statistical significance of both these changes independently in predicting structural lesion in the CT brain. Analysis showed that EEG showing lateralizing changes are more specific in picking up structural lesion than EEG with generalized changes. Patients with EEG showing lateralized changes have a sensitivity of about 42.5% and specificity of 76.9% in predicting CT brain abnormality. With a very significant p value (p=0.0032). Patients with generalized EEG changes have a sensitivity of

10.6% and specificity of 65.3% in predicting a structural lesion. So In general patient with EEG changes in partial seizure are more likely to have structural lesion than those who don't. But in specific patient with lateralizing changes have more chance of having structural lesion than generalized changes. Among the localized EEG changes phase reversal is more predictive of structural lesion (81%) followed by focal spike, sharp and slow waves accounting for about 70%.

In the etiological aspect contrast enhancing granuloma was the most frequent lesion. We have encountered contrast enhancing granuloma in 55.3% of our study populations. In our study CT brain was abnormal in 64.4%. When compared to a study by Misra et al in the year 1994 this is a bit low. In their study structural lesions were noted in 79.3%.[3]

Comparison of CT observation in Misra et al study in the year 1994 to our study. [3]

	Misra et al 1994 %	Our study 2004-05 %
Enhancing granuloma	63.3%	55.3%
Infarct	4.0%	21.3%
Mass	4.6%	8.5%
Calcification	11.8%	2.1%
Cerebral atrophy	5.9%	Nil
Oedema	1.9%	4.3%
Haemorrhage	0.8%	Nil
Vascular malformation	0.8%	2.1%
Gliosis	0.5%	2.1%
Tubers	Nil	2.1%

Similar study in children was done in the year 2004 by Hussian et al. In their study the incidence of structural lesion was 68% which is almost close to our study. In their study also contrast enhancing granuloma was the most common lesion.

We have observed that patients with granulomatous contrast enhancing lesions have more chance of their EEG being abnormal when compared to other patients with structural lesions. Incidence of lateralizing EEG abnormality was also more in patients with granulomatous lesions.

Lesion	EEG Abnormality
Contrast enhancing granuloma	62%
Infarct	40%
Mass	50%
Others	43%

Distribution of granuloma was most commonly noted in parietal lobe 21/26 (81%) of which left side was more common than right with 30% more on the left compared to the right side.

Overall 34 cases out of the 47 cases with structural lesion had their lesion in the parietal lobe (72.3%). So it shows that parietal lobe lesions are the most common cause of partial motor seizure.

CONCLUSION

- Partial seizures are equally distributed among Men and Women
- Patients with post ictal deficit have more chance of structural lesion. Our study has shown 28 out of 31 patients who has postictal clinical signs or deficit had abnormal CT brain (90%)
- Incidence of structural lesion in CT brain is more in patients who has an abnormal EEG. Moreso if EEG has lateralizing abnormality (43%)
- Contrast enhancing granuloma was the most common cause for partial seizure (55%) followed by CVA (21%)
- Contrast enhancing granuloma were predominantly situated in the parietal lobe.
- Parietal lobe lesions were the most common cause of partial seizure.
- EEG was most often abnormal in patients with contrast enhancing granuloma (62%) when compared to other lesions (44.3%).

PROFORMA

Name : Age / Sex: No.: Date :

Seizure :

Onset -

Type → Simple partial

Type → Complex partial

Duration of Seizure :

Side :

Site :

Status if any :

Other symptoms :

Head ache

Paresis / Plegia

Sensory impairment :

Cranial Nerve dysfunction :

Behaviour abnormality :

Signs :

Fundus :

Fever :

Meningeal signs :

Focal Neurological deficit :

Investigations :

EEG :

CT Brain :

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MASTER CHART

Name	Age /Sex	IP.No.	Types of Seizure			Side of the Seizure		Deficit		EEG			CT Scan Brain		
			Simple	Complex	Both	Rt	Lt	Present	Absent	Abnormal		Normal	Abnormal		Normal
										Generalised	Localised		Rt	Lt	
	27/F	6369/04	-	Y	-		Y	Y	-	Y	-	-	-	-	Y
ppal	58/M	6625/04	Y	-	-	-	Y	-	Y	-	-	Y	Y	-	-
an	35/M	6940/04	-	Y	-	-	Y	Y	-		Y	-	Y	-	-
asu	4 ½ M	6946/04	Y	-	-	-	Y	Y	-	-	-	Y	Y	-	-
haran	16/M	6542/04	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y
ammal	50/F	6975/04	-	Y	-	-	Y	Y	-	Y	-	-	Y	-	-

levi	1 ½ F	6649/05	Y	-	-	-	Y	-	Y	Y	-	-	-	-	Y	
raj	32/M	8017/04	Y	-	-	Y	-	-	Y	-	-	Y	-	-	Y	
	6/F	7234/04	-	Y	-	Y	-	Y	-	Y	-	-	Bil		-	
ja	8/F	7374/04	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y	
a	15/F	7580/04	-	Y	-	Y	-	-	Y	Y	-	-	-	-	Y	
lakshmi	12/F	314621	-	-	Y	-	Y	-	Y	-	-	Y	Y	-	-	
	35/F	7664/04	Y	-	-	-	Y	Y	-	-	Y	-	Y	-	-	
ka	14/F	8563/04	Y	-	-	-	Y	Y	-	-	-	Y	Y	-	-	
	15/F	7049/04	-	Y	-	Y	-	-	Y	Y	-	-	-	-	Y	
kshmi	20/F	7966/04	Y	-	-	Y	-	Y	-	-	-	Y	-	Y	-	
kshi	5/F	7910/04	Y	-	-	-	Y	Y	-	Y	-	-	Sup			
priya	10/F	8642/04	Y	-	-	Y	-	-	Y	-	-	Y	-	Y	-	
n	29/M	8942/04	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y	
	35/F	6028/04	Y	-	-	Y	-	Y	-	Y	-	-	-	Y	-	
apoor	18/M	316069	-	-	Y	Y	-	Y	-	-	Y	-	-	Y	-	
pandi	32/M	9718/04	Y	-	-	Y	-	-	Y	Y	-	-	-	Y	-	
mari	9/F	9810/04	-	Y	-	Y	-	-	Y	-	-	Y	-	Y	-	
ponnu	21/F	9644/04	-	Y	-	Y	-	-	Y	-	-	Y	-	Y	-	
adran	11/M	101361/04	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y	
n prakash	8/M	9558/04	Y	-	-	Y	-	-	Y	-	Y	-	-	Y	-	
kumar	9/12 M	8539/04	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y	
riselvi	7/F	10170/04	-	Y	-	Y	-	-	Y	-	-	Y	-	Y	-	
samy	17/M	10507/04	-	Y	-	Y	-	-	Y	Y	-	-	-	-	Y	
a selvi	15/F	10569/04	Y	-	-	-	Y	-	Y	-	-	Y	Y	-	-	
ka	13/F	10577/04	Y	-	-	Y	-	Y	-	Y	-	-	-	-	Y	
lakshmi	18/F	10704/04	-	Y	-	-	Y	Y	-	-	Y	-	Bil		-	
urugan	28/M	6425/04	-	Y	-	Y	-	-	Y	-	-	Y	-	Y	-	
mmal	55/F	10644/04	Y	-	-	-	Y	Y	-	-	-	Y	Y	-	-	

aj	15/M	11265/04	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y
ndi	25/M	10915/04	-	Y	-	Y	-	-	Y	-	-	Y	Y	-	-
l hameed	45/M	10946/04	Y	-	-	Y	-	Y	-	-	Y	-	-	Y	-
ran	34/M	1127/05	-	Y	-	-	Y	Y	-	-	Y	-	Y	-	-
	10/F	670/05	Y	-	-	-	Y	-	Y	-	-	Y	Y	-	-
	6/F	1469/05	-	Y	-	Y	-	Y	-	-	Y	-	-	Y	-
a	2/F	1649/05	Y	-	-	Y	-	Y	-	-	Y	-	-	Y	-
an	1/M	1436/05	Y	-	-	-	Y	-	Y	Y	-	-	-	-	Y
elvi	50/F	1625/05	-	Y	-	-	Y	-	Y	-	-	Y	-	-	Y
ni	10/F	1405/05	-	Y	-	Y	-	-	Y	Y	-	-	Bil		-
r	18/F	6349/05	Y	-	-	Y	-	Y	-	-	Y	-	-	Y	-
	35/F	1786/05	-	Y	-	Y	-	-	Y	-	Y	-	-	-	Y
k	15/M	1637/05	-	Y	-	-	Y	-	Y	Y	-	-	-	-	Y
an	30/M	1923/05	-	Y	-	-	Y	Y	-	-	Y	-	Y	-	-
lkumar	29/M	2618/05	Y	-	-	Y	-	Y	-	-	-	Y	-	Y	-
th	33/M	1466/05	-	Y	-	-	Y	Y	-	-	-	Y	Y	-	-
avalli	15/F	2651/05	Y	-	-	Y	-	-	Y	-	-	Y	-	-	Y
eswaran	35/M	3922/05	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y
	13/M	3895/05	-	Y	-	-	Y	-	Y	-	Y	-	Y	-	-
an	61/M	3635/05	Y	-	-	-	Y	Y	-	-	-	Y	Y	-	-
ni	16/F	2509/05	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y
yee	9/F	6005/05	Y	-	-	Y	-	-	Y	-	Y	-	-	Y	-
ah	14/M	364876	-	Y	-	-	Y	Y	-	-	Y	-	Y	-	-
	14/M	6640/05	-	Y	-	-	Y	-	Y	-	Y	-	-	Y	-
j	50/M	7689/05	-	Y	-	-	Y	-	Y	-	-	Y	Y	-	-

	11/F	7887/05	Y	-	-	Y	-	Y	-	-	Y	-	-	Y	-	
vel	12/M	7584/05	-	Y	-	-	Y	Y	-	-	Y	-	Y	-	-	
	70/M	8216/05	-	Y	-	-	Y	Y	-	-	-	Y	-	-	Y	
amy	14/M	8571/05	Y	-	-	Y	-	-	Y	-	Y	-	-	Y	-	
	4/M	8549/05	-	Y	-	Y	-	Y	-	-	Y	-	-	Y	-	
a	8/F	8689/05	-	Y	-	Y	-	-	Y	-	Y	-	-	-	Y	
ini Devi	13/F	396016	-	Y	-	Y	-	Y	-	-	Y	-	-	Y	-	
inmary	32/F	399582	Y	-	-	-	Y	Y	-	-	-	Y	Y	-	-	
r nisha	9/F	408349	Y	-	-	-	Y	Y	-	-	Y	-	Y	-	-	
a vel	4/M	10834/05	-	Y	-	Y	-	-	Y	-	Y	-	-	-	Y	
bramani	9/M	2553/05	-	Y	-	Y	-	-	Y	-	Y	-	-	Y	-	
r	35/M	2252/05	-	Y	-	Y	-	-	Y	-	-	Y	-	-	Y	
pandian	29/M	6834/05	-	Y	-	-	Y	-	Y	-	Y	-	-	-	Y	
r	5/M	8550/05	Y	-	-	Y	-	-	Y	-	-	Y	-	-	Y	

MASTER CHART – ABBREVATION

P - PATIENT

M - MALE

F - FEMALE

F - FRONTAL

O - OCCIPITAL

Bil - BILATERAL

I - INFARCT

T - TUBERS

PV - PERI VENTRICULAR

PI - POST ICTAL OEDEMA

VI - VENOUS INFARCT

TP - TEMPERO PAREITAL

SOL- SPACE OCCUPYING LESION

CG - CONTRAST ENHANCIG GRANULOMA

SUP - SUPRA SELLAR

MCEL- MULTIPLE CONTRAST ENHANCING LESIONS

CALGR - CALCIFIED GRANULOMA

GL - GLIOSIS

GYE - GYRAL ENHANCEMENT

FT - FRONTO TEMPORAL

FP - FRONTO PARIETAL

PO - PARIETO OCCIPITAL

AVM - ARTERIO VENOUS MALFORMATIONS