A STUDY OF ASSOCIATION OF CLINICAL PROFILE WITH EEG AND NEUROIMAGING OF PATIENTS WITH PARTIAL AND GENERALISED SEIZURES

By

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Dissertation Submitted to the
Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar, Karnataka,
in partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE IN GENERAL MEDICINE

Under the guidance of

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I hereby declare that this dissertation titled **"A STUDY OF ASSOCIATION OF CLINICAL PROFILE WITH EEG AND NEUROIMAGING OF PATIENTS WITH PARTIAL AND GENERALISED SEIZURES"** is a bonafide and genuine research work carried out by me under the guidance and supervision of **Dr. VENKATARATHNAMMA P. N.** MD Professor, Department of General Medicine, Sri. Devaraj Urs Medical College, Tamaka, Kolar.

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Member-Secretary **Dr. Sanikop M. B.**

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Above all I thank the Almighty for all His guidance and blessings.

Date:

Place:

Signature of the Candidate

Dr. SRI SAI PRASAD M. MBBS

Dedicated to my youngest sister who was treated for idiopathic epileptiform seizures and got her life battered with anti-convulsants for 15 years without any relief before it was identified as psychogenic non epileptiform seizures.

LIST OF ABBREVIATIONS

AD	→	After the death of Christ
CT	→	Computerized Tomography
CVT	→	Corticovenous Thrombosis
CVA	→	Cerebrovascular Accident
CNS	→	Central Nervous System
CSF	→	Cerebrospinal Fluid
DKA	→	Diabetic Ketoacidosis
DF	→	Degrees of Freedom
ECG	→	Electrocardiography
EITB	→	Enzyme Linked Immuno Electro Transfer Blot
ELISA	→	Enzyme Linked Immunosorbent Assay
EEG	→	Electro Encephalograph
GTCS	→	Generalized Tonic Clonic Seizures
HIV	→	Human Immunodeficiency Virus
HONK	→	Hypersmolar Non-ketotic Coma

ICT	→	Intracranial Tension
MRI	→	Magnetic Resonance Imaging
MCA	→	Middle Cerebral Artery
PRL	→	Prolactin
SOL	→	Space Occupying Lesion
SD	→	Standard Deviation

ABSTRACT

A STUDY OF ASSOCIATION OF CLINICAL PROFILE WITH EEG AND NEUROIMAGING OF PATIENTS WITH PARTIAL AND GENERALIZED SEIZURES

Background

Patients who have experienced a seizure or have been suffering from epilepsy form a big chunk of clinical practice. Although a variety of factors influence the incidence and prevalence of seizures, approximately 5-10% of population will have at least one seizure with highest incidence occurring in early childhood and late adulthood. Seizures being a complex symptom of underlying disease, a detailed workup is generally required for a total evaluation of such cases and treatment. No fixed guidelines exist to evaluate the cases of seizures. In the past, a large number of cases used to be labelled as idiopathic epilepsy. The introduction of EEG, CT and MRI has substantially helped to identify the causes of epilepsy.

Need For The Study

In view of the above facts, this study would be undertaken to evaluate the role of clinical profile, EEG and CT scan as a diagnostic aid in various seizure disorders and also to determine various etiological factors in patients with focal and generalized seizures in adult patients.

Objectives of the study

- To study the correlation of clinical profile with EEG and neuroimaging in the diagnosis of seizures.
- To determine the etiological factors of seizures.

Methodology

Adult patients more than 18 years of age presenting with first time seizure to the Department of Medicine at Sri. R. L. Jalappa Hospital and Research Centre were included. Data was collected by interview to elicit clinical profile and also by performing EEG, CT scan and serum prolactin levels. Data collected was studied with respect to the association of clinical profile with EEG and neuroimaging in patients with focal and generalized seizures. Serum Prolactin Level estimation (Chemiluminiscence method) was done. Statistical tests employed were Chi-Square test, Fisher-Exact test and Pearson Correlation test. **Inclusion Criteria**: Patients who have seizures for the first time after 18 years of age. **Exclusion Criteria**: (a) Patients who have been diagnosed previously with a seizure disorder. (b) Patients whose onset of seizures before 18 years of age. (c) Patients who present with seizures with head injury.

Conclusion

Among the patients presenting with unprovoked seizures majority had generalized seizures than focal seizures.EEG was not very significant in the evaluation of seizures. CT scan was reliable in revealing structural abnormalities and was helpful in establishing the diagnosis in majority of cases. CT scan and EEG correlation is significant in seizures in general and as well as generalized seizures. CT scan and EEG did not correlate in focal

seizure group. In majority of cases of generalized seizures, a cause was detected. Neurocysticercosis was the most common cause of focal seizure. Though sensitivity and specificity of serum prolactin is low, it is fairly a good surrogate marker in diagnosing epilepsy. The search for other epilepsy markers continues.

Keywords

Seizures; Electroencephalography; Computed tomography; Serum prolactin levels

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DURING THE PROCESS OF THIS DISSERTATION STUDY, THE

"INTERNATIONAL LEAGUE AGAINST EPILEPSY"

HAVE UPDATED THE CLASSIFICATION OF SEIZURES AND HAVE

RECOMMENDED THE USAGE OF THE TERM "FOCAL SEIZURES"

INSTEAD OF "PARTIAL SEIZURES"

INTRODUCTION

Epilepsy is one of the most common disorder of the brain.[1] One of every ten people will have at least one epileptic seizure during a normal life span, and a third of these will develop epilepsy. According to World Health Organization (WHO) survey, epilepsy accounts for 1% of the global burden of the disease, a figure equivalent to the breast cancer in women and lung cancer in men.[2]

Patients who have experienced a seizure or have been suffering from epilepsy form a big chunk of neuromedical practice. Seizures' being a complex symptom of underlying disease, a detailed work up is generally required for the total evaluation of such cases and treatment. No fixed guidelines exist to evaluate the cases of seizures. Epilepsy is a group of conditions and not a single homogeneous disorder and seizure may be a symptom of both diverse brain disorders and an otherwise normal nervous system. It is neither possible nor desirable to develop inflexible guidelines for what constitutes a standard or minimal set of diagnostic tests.

In the past a large number of cases used to be labeled as epilepsy of unknown origin. The introduction of EEG and CT scan has really helped to sort out the causes of epilepsy. The evaluation of cases of seizures includes a detailed history, clinical examination, electroencephalography, advanced neuroimaging and functional neuroimaging. Various physicians use various combinations of these methodologies considering their cost factor and yield of information.

Localization abnormalities in cases of focal seizures are from 28% to 80% as observed in different studies.[3,4] Studies done on patients with generalized seizures also show similar abnormalities.[5]

In view of the above facts this study was conducted to determine various etiological factors in patients with focal and generalized seizures and also to evaluate role of EEG, CT scan and serum prolactin levels as a diagnostic aid in various seizure disorders in adult patients of more than 18 years.

AIMS AND OBJECTIVES

- To study the association of clinical profile with EEG and neuroimaging in the diagnosis of various seizures.

- To determine the etiological factors of seizures.

REVIEW OF LITERATURE

HISTORY

It is reasonable to assume that epilepsy is as old as mankind. The irregular and intermittent advance toward understanding epilepsy began with the first known book on epilepsy "On the Sacred Disease" about 2,400 years ago. The author of "On the Sacred Disease" is not known but is referenced as Hippocrates.[6] He rejected both the then current belief that individual Greek gods cause epilepsy and the superstitions and magic that were in use to avoid and cure epilepsy.

This earliest known treatise on epilepsy set the stage for the conflict between natural and supernatural concepts of the disease, which continued for over 2000 years. Temkin[7] concluded with an analysis of the conceptual changes during the 19th century culminating in the contributions of John Hughlings Jackson, who laid the foundation for contemporary ideas about epilepsy, and those of Jean-Martin Charcot, who separated epilepsy and hysteria more clearly than his predecessors had.[8]

The Hippocratic writings were known to Galen, the influential Greek Physician of the second century A.D. who dissected the brain and speculated that epilepsy resulted from an accumulation within the cerebral ventricles of two of the four Greek "humors" phlegm and bile. After this till fifteenth century, diseases such as epilepsy were generally attributed to supernatural control.

At the turn of the seventeenth century, William Gilbert abruptly accelerated a change in approach from mystical and supernatural to scientific particularly for magnetic and electric phenomena. In 1667, Thomas Willis the London physician and anatomist who originated the term neurology and became immortalized by describing the circle of Willis, reaffirmed Descarte's ideas that the source of both seizures and their auras was in the brain. Richard Caton established himself as the first person in the world to observe the continuous spontaneous electrical activity of the brain. He described "the existence of electrical currents ---------- of the grey matter" and noted that "Feeble currents of varying direction pass through the multiplier (amplifier) when the electrodes are placed on two points of the external surface, or one electrode on the grey matter, and one on the surface of the skull." Two years later, he gave more detailed accounts of his observations in a longer paper, also published in the British Medical Journal.[8]

In 1929, Dr. Hans Berger, a professor of psychiatry and chair of the psychiatric clinic at the university of Jena in Germany, published his discovery that spontaneous brain electrical activity in humans could be recorded from the scalp.[9] By 1931 he reported that interictal EEG changes were common in epilepsy and later that year he recorded human spike and wave activity.

In a research EEG laboratory set up at Boston City Hospital in 1934, Frederick Gibbs, Hallowell Davis and William G. Lennox in 1935[10] first demonstrated spike and wave complexes interictally and during clinical absences. Gibbs, Lennox and Gibbs and Jasper[11] demonstrated focal spikes in localization related epilepsy in 1936. In 1937 the first clinical department in US to formally perform and charge for EEG services[12] was

opened at Massachusetts General Hospital by Robert Schwab and others quickly followed.

However, the EEG years ago lost its place as a frontline non-invasive method for recognizing structural lesions. Structural neuroimaging prior to the introduction of CT and MRI were largely limited to X-ray skull, pneumoencephalography and conventional radioactive isotope imaging.[13,14]

The development of CT scan in the 1970's represented the beginning of contemporary structural neuroimaging. A large number of workers have worked on the role of CT in the evaluation of patients with epilepsy and to this the credit must certainly go to Hounsfields[15] description of the CT scan and clinical studies reported by Ambrose[16] who have shown CT to be a sensitive and accurate diagnostic modality with no risk or mortality whatsoever.

DEFINITIONS OF SOME TERMS

Seizure

A seizure (From the Latin sacire "to take possession of") is a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from an aggregate of central nervous system (CNS) neurons. Depending on the distribution of discharges, this abnormal CNS activity can have various manifestations, ranging from dramatic convulsive activity to experimental phenomena not readily discernible by an observer.[17]

Epilepsy

Epilepsy describes a condition in which a person has recurrent seizures due to a chronic, underlying process. The definition implies that a person with single seizure or recurrent seizures due to correctable or avoidable circumstances does not necessarily have epilepsy.[17]

The meaning of the term seizure needs to be carefully distinguished from that of epilepsy. Epilepsy describes a condition in which a person has recurrent seizures due to a chronic, underlying process. This definition implies that a person with a single seizure, or recurrent seizures due to correctable or avoidable circumstances, does not necessarily have epilepsy. Epilepsy refers to a clinical phenomenon rather than a single disease entity, since there are many forms and causes of epilepsy. However, among the many causes of epilepsy there are various epilepsy syndromes in which the clinical and pathologic characteristics are distinctive and suggest a specific underlying etiology.

Incidence and Prevalence

- In most developed countries incidence rates range from 40–70 per 100,000 but in developing countries the rates may be as high as 100–190 per 100,000.[18]
- Similarly the prevalence of active epilepsy defined as persons who take anticonvulsants or who have had a seizure in the past five years ranges from 4–10 per 10,000 in developed countries and upto 57 per 10,000 in developing countries.[19]
- Studies have estimated that 1.5% to 5% of any population will have a seizure at some time.[20]

- The estimated number of persons with epilepsy in India is approximately 5.5 million.[21]
- Based on a solitary study[22] which reported an incidence of 49.3 per 100,000 the number of new persons with epilepsy in India each year would be close to half million.

Classification of Seizures

Determining the type of seizure that has occurred is essential for focusing the diagnostic approach on particular etiologies, selecting the appropriate therapy, and providing potentially vital information regarding prognosis. The International League against Epilepsy (ILAE) Commission on Classification and Terminology, 2005–2009 has provided an updated approach to classification of seizures. This system is based on the clinical features of seizures and associated electroencephalographic findings. Other potentially distinctive features such as etiology or cellular substrate are not considered in this classification system, although this will undoubtedly change in the future as more is learned about the pathophysiologic mechanisms that underlie specific seizure types

1. **Focal seizures:** (Can be further described as having motor, sensory, autonomic, cognitive, or other features).

2. **Generalized seizures**
   a. Absence: (i) Typical (ii) Atypical
   b. Tonic clonic
   c. Clonic

d. Tonic

e. Atonic

f. Myoclonic

3. **May be focal, generalized, or unclear**

Epileptic spasms

A fundamental principle is that seizures may be either focal or generalized. Focal seizures originate within networks limited to one cerebral hemisphere **(note that the term partial seizures is no longer used).** Generalized seizures arise within and rapidly engage networks distributed across both cerebral hemispheres. Focal seizures are usually associated with structural abnormalities of the brain. In contrast, generalized seizures may result from cellular, biochemical, or structural abnormalities that have a more widespread distribution. There are clear exceptions in both cases, however.

Etiological factors of seizures

The literature contains a considerable number of studies devoted to analyzing etiological factors of seizures in adults.

Dam AM et al[21] (Denmark, 1985) studied 221 patients who had their first seizure after the age of 25 years. All patients had a clinical evaluation, electroencephalography (EEG) and computed tomography (CT) done. The major etiological group was the one where no cause could be detected (38%). Alcohol abuse as the etiology defined as cases with a history of long standing alcohol overuse, concomitant signs of alcohol intoxication

and spontaneous recurrent epileptic seizures made up a group of one fourth (25%) of all the patients with late onset epilepsy. Brain tumor was the cause in 16% and cerebrovascular infarction in 14%.

Perez Lopez JL et al[22] (1985) retrospectively studied 250 patients with late onset seizures. The ages of the patients ranged from 22 years to 88 years. All patients were evaluated clinically with EEG and CT scan. No cause could be identified in 49 patients (19.5%).Among the rest of the cases the most frequent etiology were chronic alcoholism 62 (25%), brain tumors 41 (16%), cerebrovascular disease 33 (13%) and post-traumatic epilepsy 28 (11%).

Tardy B et al[23] (1995) studied adult first seizure in an emergency department. This was a retrospective study of 3 years period during which 247 patients were studied. A CT scan had been performed in 247 patients and an EEG in 209. Etiologies were found to be (1) unknown (2) alcohol abuse (3) stroke (4) tumor.

Dr. R. A. Schoenenberger[24] (1994) studied 119 adult patients presenting to casualty with generalized seizure. The etiologies were withdrawal from alcohol, drug or drug intoxication in 48 (40%), cerebral infraction in 14 (11.7%), intracranial haemorrhage in 6 (5%), primary cerebral neoplasm in 10 (8.4%), metastases in 7 (5.8%), idiopathic in 17 (14.2%), remote trauma and HIV encephalopathy in four each, two each from Alzheimer's disease and toxoplasmosis, one each from viral encephalitis, bacterial meningitis, arteriovenous malformation and arachnoid cyst.

Medina MT et al[25] (1990) studied 100 consecutive patients with epilepsy that started after the age of 25 years. All patients underwent CT, EEG and additionally CSF analysis was performed in 82 of them. Neurocysticercosis or its sequelae were diagnosed in 50 patients.

Murthy JM, Yangala R[5] (1998) studied the putative etiology in 991 patients with symptomatic localization related epilepsies seen in university hospital in south India. Cerebrovascular diseases were the risk factors in 48% of patients with remote symptomatic epilepsy. Neurocysticercosis, single CT enhancing lesion (SCTEL), and small single cerebral calcific CT lesions (SSCCCTL) together accounted for 40% of etiological factors and neurotuberculosis for 10%.

Rogel-Ortiz FJ[26] (1999) prospectively studied 130 adult patients with adult onset epilepsy after 20 years. All the patients had CT scan brain and when necessary brain magnetic resonance imaging. They found structural brain lesion in 51% of patients. The most frequent causes of seizures were neurocysticercosis in 28% followed by cerebral infarct (11%) and brain atrophy (11%).

Marco T. Medina et al[27] (2005) studied an epilepsy survey in rural county of Salama, Honduras. Among 6473 residents surveyed 151 persons with epilepsy were identified. 100 of whom had active epilepsy. These patients underwent video EEG, CT brain and serum EITB for cysticercosis. Symptomatic epilepsy was primarily due to neurocysticercosis (37%), perinatal brain damage (8%), post-traumatic (3%) and past stroke (2%). Eight percent were idiopathic and 30% were cryptogenic.

Oscar H Del Brutto et al[28] (2005) did a door to door survey to detect subjects with epileptic seizures, in Atahualpa. Among 2,548 residents, patients with epilepsy as well as age and sex matched controls underwent a CT head and scalp EEG. Blood samples were also collected for determination of anti-cysticercal antibodies. Neurocysticercosis was associated with one third cases of epilepsy.

Vedantam Rajashekhar[29] (2003) who has studied more than 450 patients with single small enhancing CT lesions (SSECT) from 1991 to 1996 has opined that solitary cerebral cysticercous granuloma is one of the commonest causes of seizures in Indian patients.

Electroencephalography (EEG)

The EEG provides a dynamic record of electrical potentials of brain. This is recorded by the EEG acting like a powerful and complex amplifier which has the ability to amplify these potentials. These amplified potentials further cause deflections of ink writing pens strategically placed. The pens then produce a wave like pattern on a fast moving strip of paper. The EEG may be helpful in the interictal period by showing certain abnormalities that are highly supportive of the diagnosis of epilepsy.

The EEG is recorded from metal electrodes placed on the scalp. The placement of recording electrodes is generally based on the international 10-20 system.[30]

International 10-20 system

(i) The midline of the head is divided into portions of 10%, 20%, 20%, 20% 20% and 10% beginning at the nasion and ending at the inion.

(ii) The transverse line of the head is divided into portions of 10% 20% 20%, 20%, 20% and 10% beginning at the right preauricular point and ending at the left preauricular point.

(iii) Numerals or anatomical nomenclature are given to the points as shown in the figure 1.

(iv) Electrode pairs are interconnected in different arrangements called Montages to permit a comprehensive survey of the brain electrical activity. Typically montages are designed to compare symmetrical areas of the two hemispheres as well as anterior versus posterior regions or parasagittal versus temporal area in the same hemisphere. The EEG can be reconstructed after digital recording.

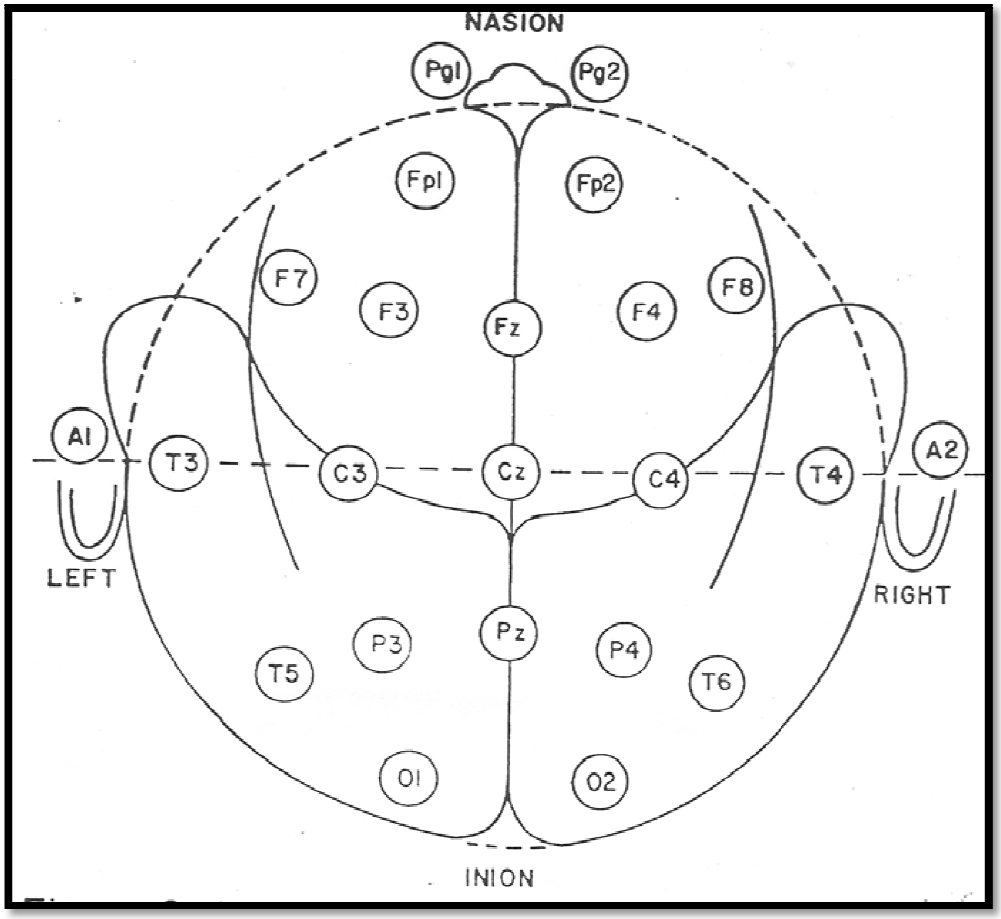


Fig. 1: The International 10 – 20 system of electrode placement

A - Earlobe, C – Central, F – Frontal, Fp – Frontal Polar, P – Parietal, Pg – Nasopharyngeal, T – Temporal, O – Occipital.

Right sided placements are indicated by even numbers, left sided placement by odd numbers and Midline placements by z.

DESIGNATIONS OF THE ELECTRODE POSITIONS

Electrode Number		International Symbol		Name
Left	Right	Left	Right	
1	2	FP1	FP2	Frontal Pole
3	4	F3	F4	Frontal
5	6	C3	C4	Central
7	8	P3	P4	Parietal
9	10	O1	O2	Occipital
13	14	F7	F8	Anterior Temporal
15	16	T3	T4	Middle Temporal
17	18	T5	T6	Posterior Temporal
19		Fz		Midline Frontal
24		Cz		Midline Central
20		Pz		Midline Parietal
11	12	A1	A2	Auricular

The electroencephalogram (EEG) is the depiction of the electrical activity occurring at the surface of the brain. This activity appears on the screen of the EEG machine as waveforms of varying frequency and amplitude measured in voltage (specifically microvoltages).

EEG waveforms are generally classified according to their frequency, amplitude, and shape, as well as the sites on the scalp at which they are recorded. The most familiar classification uses EEG waveform frequency (e.g., alpha, beta, theta, and delta).[31,32,33]

Information about waveform frequency and shape is combined with the age of the patient, state of alertness or sleep, and location on the scalp to determine significance.

Normal EEG waveforms, like many kinds of waveforms, are defined and described by their frequency, amplitude, and location.[34]

- Frequency (Hertz, Hz) is a key characteristic used to define normal or abnormal EEG rhythms.

- Most waves of 8 Hz and higher frequencies are normal findings in the EEG of an awake adult. Waves with a frequency of 7 Hz or less often are classified as abnormal in awake adults, although they normally can be seen in children or in adults who are asleep. In certain situations, EEG waveforms of an appropriate frequency for age and state of alertness are considered abnormal because they occur at an inappropriate scalp location or demonstrate irregularities in rhythmicity or amplitude.[35]

- Some waves are recognized by their shape, scalp location or distribution, and symmetry. Certain patterns are normal at specific ages or states of alertness and sleep.

- Some of the normal waveforms are K complex, V waves, lambda waves, positive occipital sharp transients of sleep (POSTS), spindles, mu rhythm, spikes, sharp waves, and certain delta waves (polyphasic and monophasic shapes).

Rhythm Frequency

The frequencies most brain waves range from are 0.5-500 Hz. However, the following categories of frequencies are the most clinically relevant:

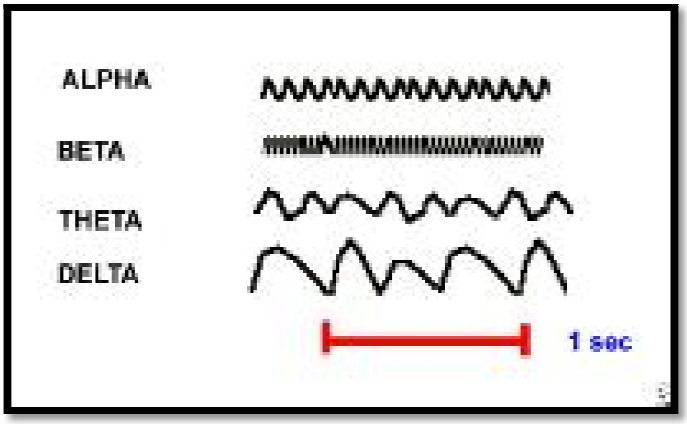


Fig. 2: Examples of alpha, beta, theta, and delta electroencephalography frequencies

- Alpha waves - 8-13 Hz
- Beta waves - Greater than 13 Hz
- Theta waves - 3.5-7.5 Hz
- Delta waves - 3 Hz or less

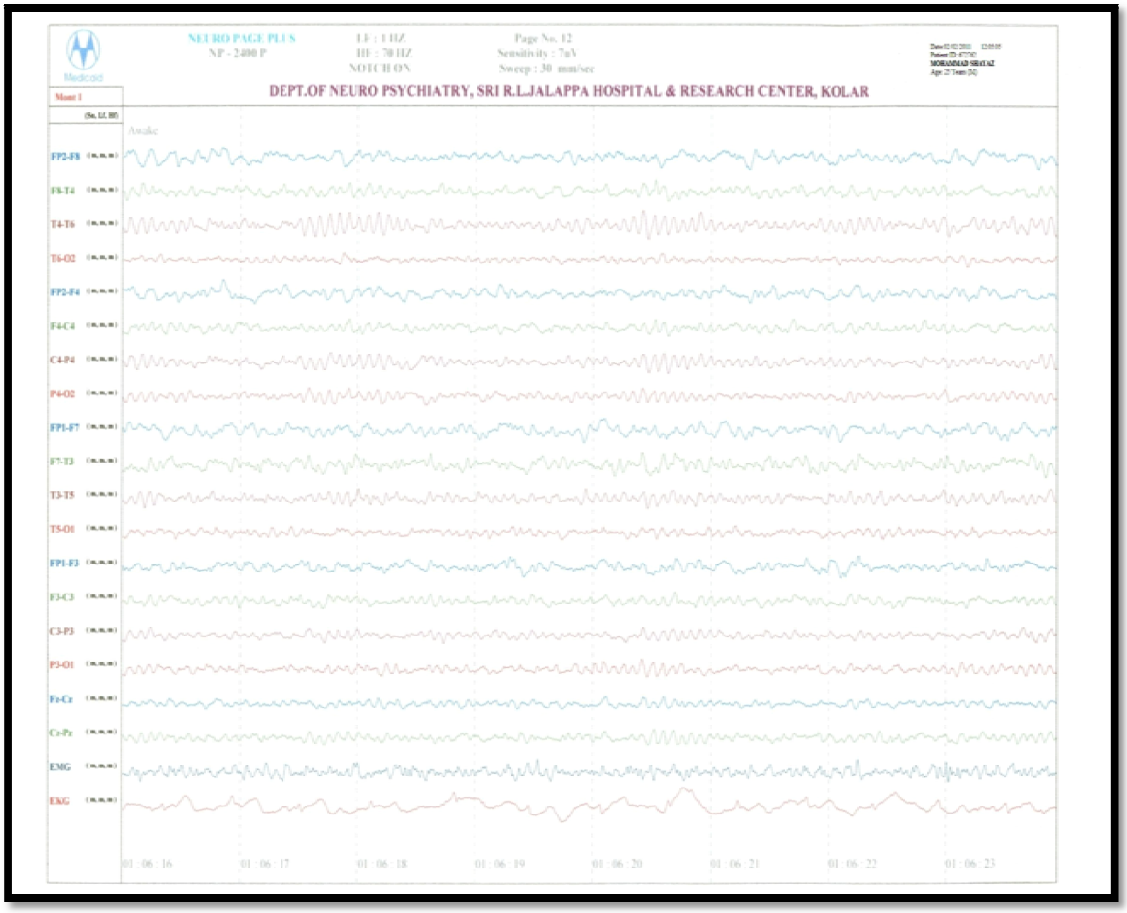


Fig. 3: Normal EEG

Alpha waves

- Alpha waves generally are seen in all age groups but are most common in adults. They occur rhythmically on both sides of the head but are often slightly higher in amplitude on the non-dominant side, especially in right-handed individuals. A normal alpha variant is noted when a harmonic of alpha frequency occurs in the posterior head regions. They tend to be present posteriorly more than anteriorly and are especially prominent with closed eyes and with relaxation.
- Alpha activity disappears normally with attention (e.g., mental arithmetic, stress, opening eyes). In most instances, it is regarded as a normal waveform.

- An abnormal exception is alpha coma, most often caused by hypoxic-ischemic encephalopathy of destructive processes in the pons (e.g., intracerebral hemorrhage). In alpha coma, alpha waves are distributed uniformly both anteriorly and posteriorly in patients who are unresponsive to stimuli.

Beta waves

- Beta waves are observed in all age groups.
- They tend to be small in amplitude and usually are symmetric and more evident anteriorly.
- Drugs, such as barbiturates and benzodiazepines, augment beta waves.

Theta waves

- Theta waves normally are seen in sleep at any age. In awake adults, these waves are abnormal if they occur in excess.
- Theta and delta waves are known collectively as slow waves.

Delta waves

- These slow waves have a frequency of 3 Hz or less.
- They normally are seen in deep sleep in adults as well as in infants and children.
- Delta waves are abnormal in the awake adult.

ABNORMAL RECORDING IN EPILEPTIC SEIZURES

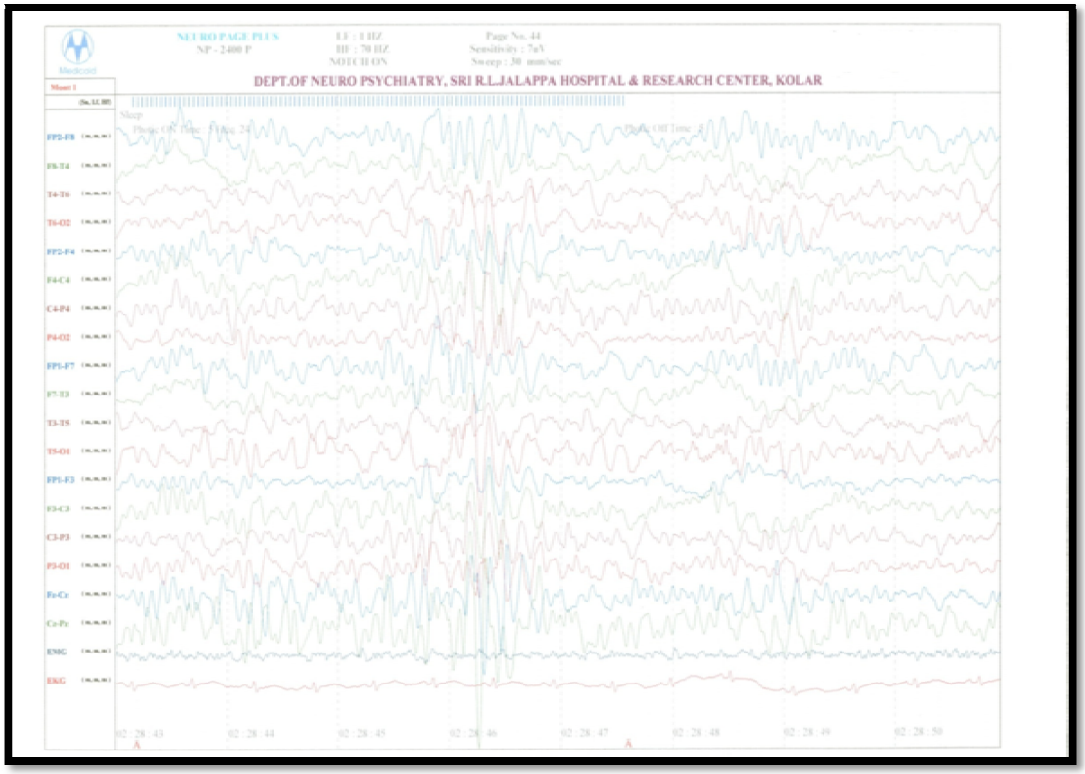


Fig. 4: Sharp and spike wave complexes followed by slow wave seizure record suggestive of GTCS

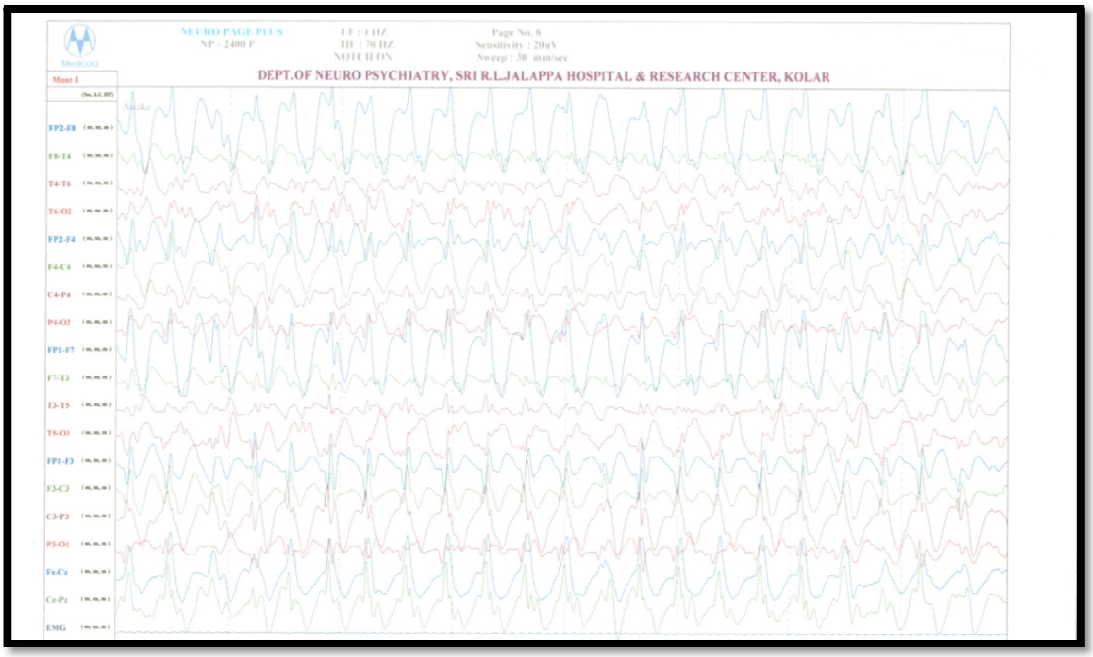


Fig. 5: Sharp and spike wave complexes three/second suggestive of absence seizures

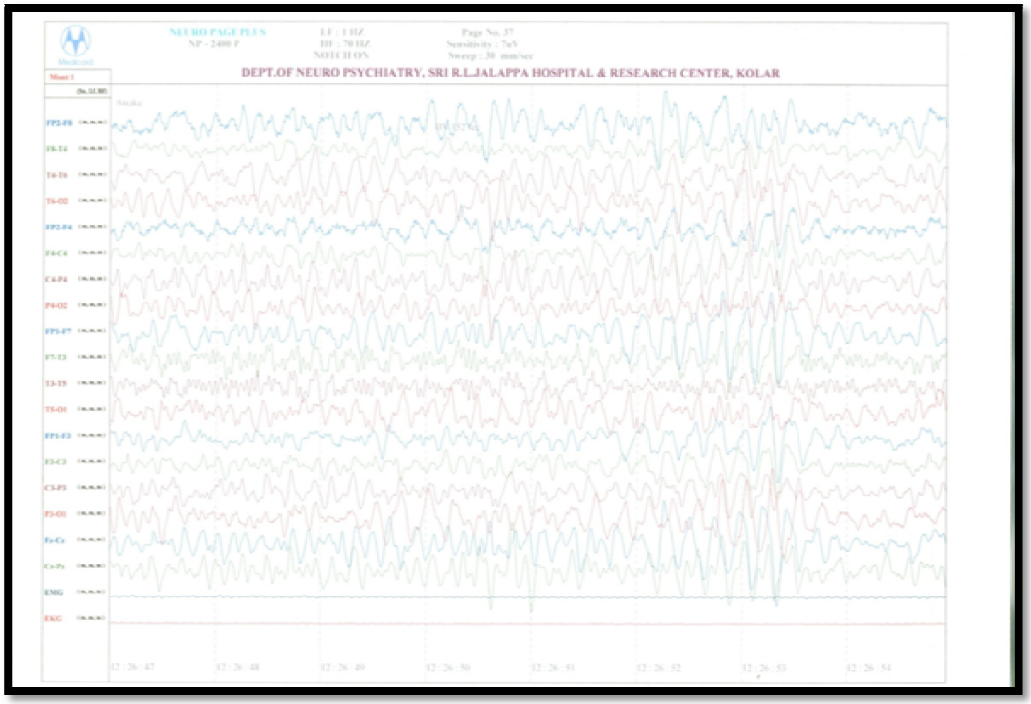


Fig. 6: Spike sharp waves with generalized epileptiform discharges suggestive of GTCS

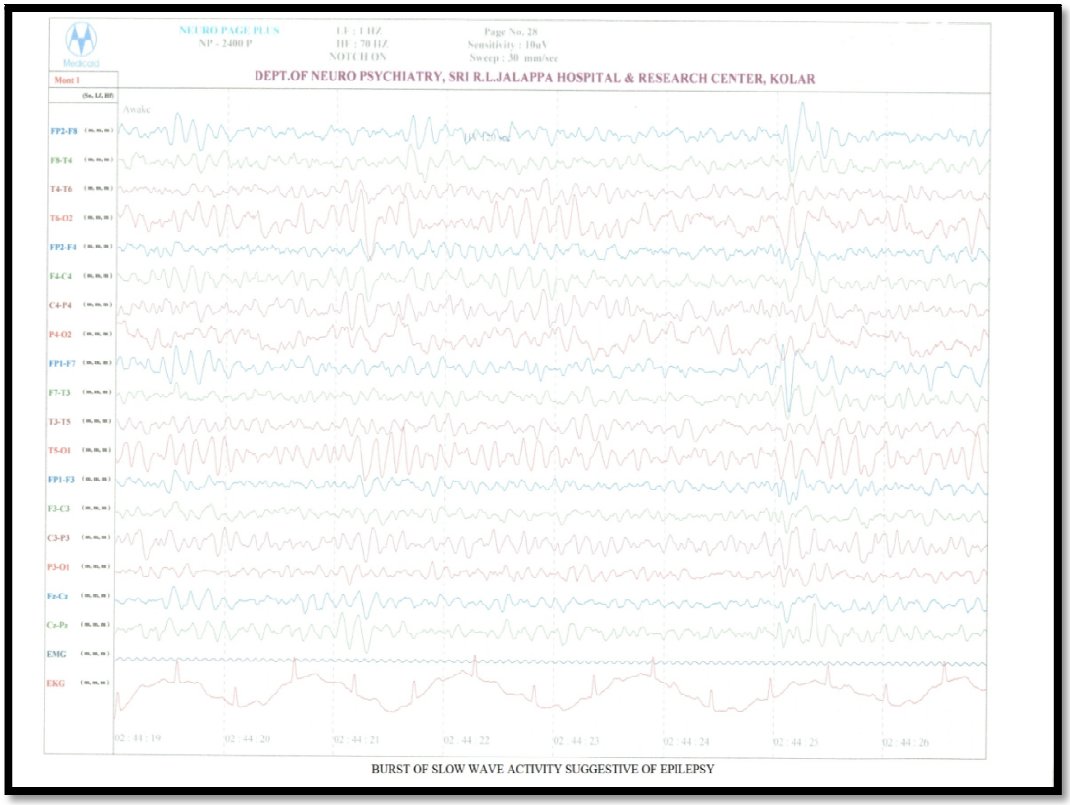


Fig. 7: Burst of slow wave activity suggestive of epilepsy

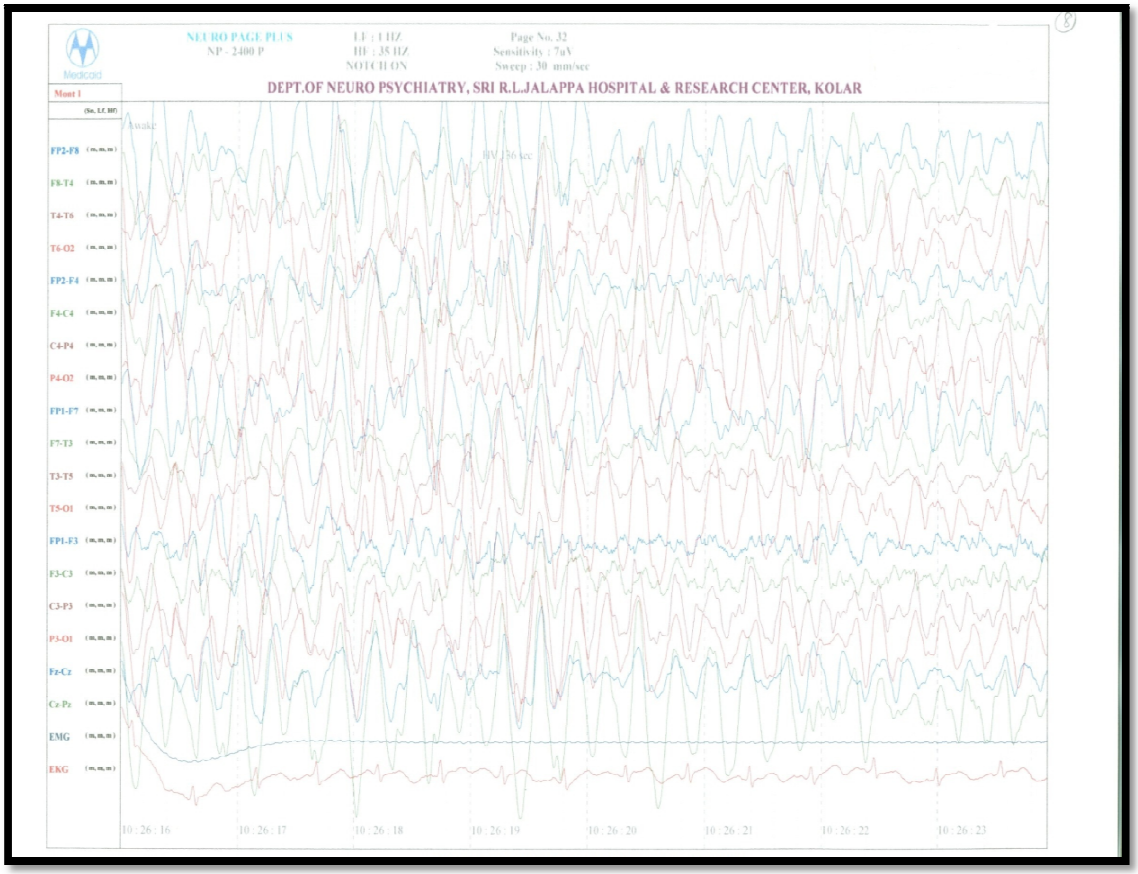


Fig. 8: Generalized slowing suggestive of epilepsy

The main types of epileptiform discharges are spikes, sharp waves and spike and wave discharges.

- Spikes are brief potentials having a steep ascending and descending limb with a duration of < 70 msec.
- Sharp waves are broader potentials with pointed peaks, having a duration that usually ranges between 70 and 200 msec.
- Spike and wave discharge consists of a spike followed by slow wave.
- Epileptic discharges may be focal or generalized.

EEG abnormalities indicating focal dysfunction

- Focal delta activity is the classic EEG sign of local disturbance in cerebral function. A structural lesion is most strongly suggested if the delta activity is continuously present, shows variability in waveform, amplitude, duration and morphology (so called “polymorphic” or “arrhythmic” activity) and persists during changes in physiological state.
- Periodic lateralized epileptiform discharges (PLED’s).
- Voltage attenuation.
- Intermittent rhythmic slow waves. When bursts of rhythmic slow waves occur (theta or delta) focally or are lateralized to one hemisphere usually indicate a structural abnormality

COMPUTED TOMOGRAPHY SCAN (CT SCAN)

The introduction of CT scan in 1970s has changed the diagnosis of cerebral lesion in a big way in patients suffering from seizures. A large number of workers have worked on the role of CT scan in evaluation of patients with epilepsy.

Guberman A[36] (1983) studied CT scan results in a consecutive series of 196 adult epileptics. In the consecutive series overall incidence of abnormal scans was 16% with the highest yield (44%) found in patients with partial seizures. In 25 of 51 cases with abnormal scans a specific lesion amenable to therapy was detected, including 16 neoplasms and 5 arteriovenous malformations. Other lesions included generalized or focal atrophy, infarcts, calcified lesion of tuberous sclerosis, unexplained calcifications and focal low density or enhancing lesions.

Reinikainen KJ et al[37] (1987) studied incidence and CT abnormalities and their correlates with clinical and EEG features were evaluated in a consecutive series of 202 adult patients with newly diagnosed epileptic seizures. Abnormal CT findings were found in 36% of the patients. The abnormalities consisted of brain tumors (17%), atrophic lesions (11%) and other finding (8%) such as arteriovenous malformations.

de la Sayette V et al[38] (1987) reviewed the CT findings of 387 patients with new onset seizures after the age and 50. CT scanning revealed cerebral atrophy in 113 cases, ischemic lesions in 75, cerebral neoplasm in 20 and no abnormality in 177 cases.

Bajaj S et al[39] (1991) studied results of CT findings in 170 patients who developed seizures at PG Department of Medicine, SRN Hospital, Allahabad. The commonest abnormality was a focal ring or disc enhancing lesion in 66 patients (62.3%) followed by calcification in 18 (16.9%) cerebral atrophy in 9 (8.5%) vascular lesions in 7 (6.6%) tumors 4 (3.8%) and congenital hydrocephalus in 2 (1.5%).

Lt. Col. S. K. Jha[40] (2004) studied 150 consecutive cases of solitary seizures who reported to the neurology services between 1995 and August 1997 at their neurology department. CT scan head was done in 119 patients. Out of the total of119 CT scans, 91 cases (76.4%) were normal and was abnormal in 28. The abnormalities were disc or ring enhancing lesion in 8, calcified or nodular lesion in 9 and small cystic lesion in 2.

Single enhancing lesions visualized on CT scanning are the most common radiological abnormality in Indian patients with new onset seizures.

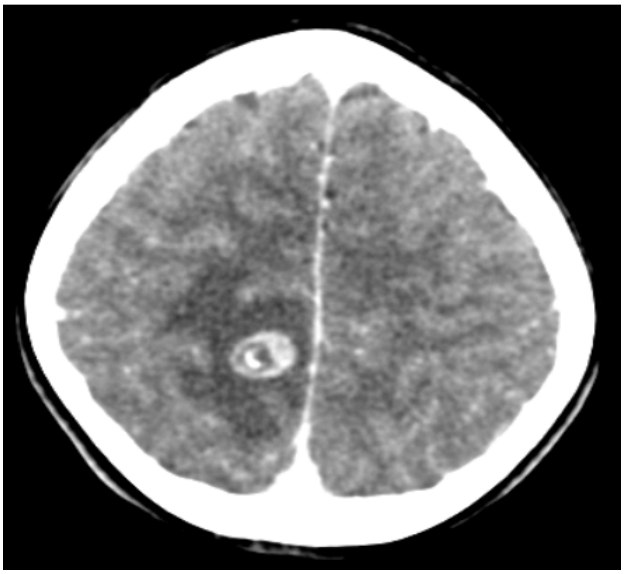


Fig. 9: Ring enhancing lesion with calcification noted in left parietal region with perilesional edema – suggestive of granulomatous lesion Neurocysticercosis / Tuberculoma

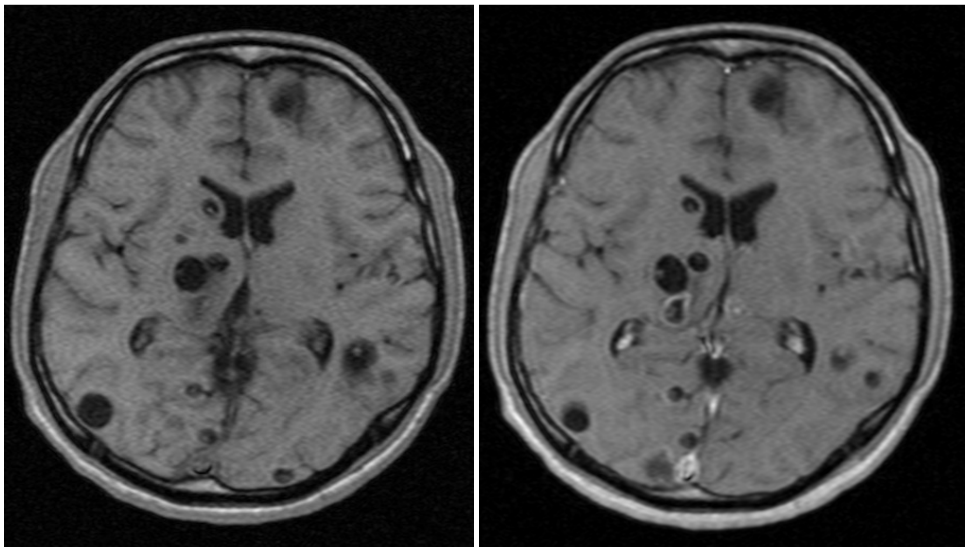


Figure 10: T1 pre and post-contrast images showing multiple scattered neurocysticercosis in the brain parenchyma. Note that the lesions are better appreciated in post contrast images

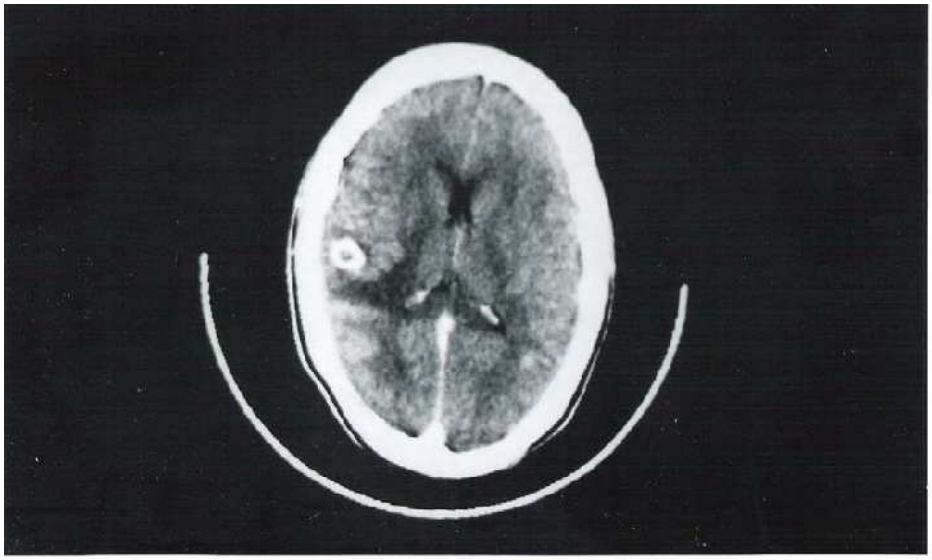


Figure 11: Ring enhancing lesion of neurocysticercosis

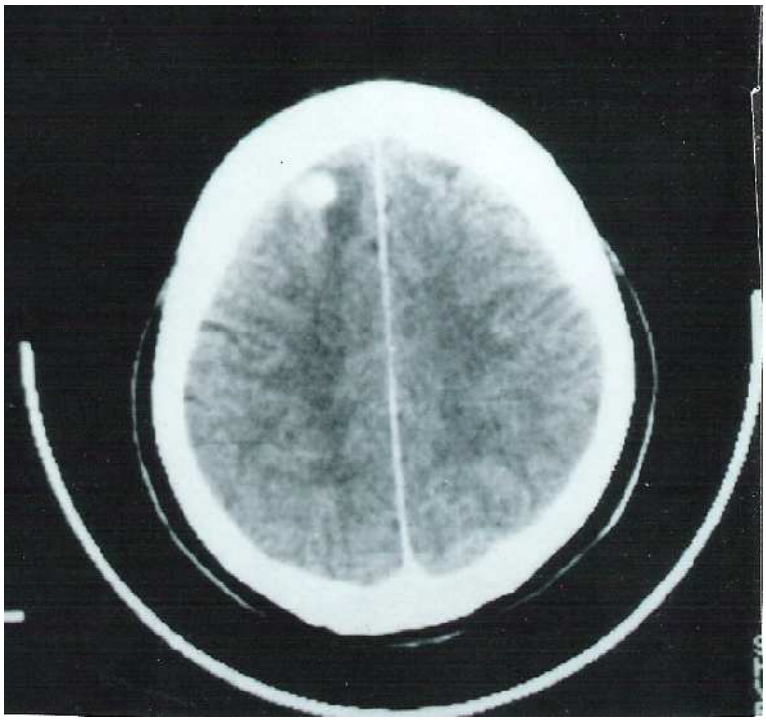


Figure 12: Disc enhancing lesion of neurocysticercosis

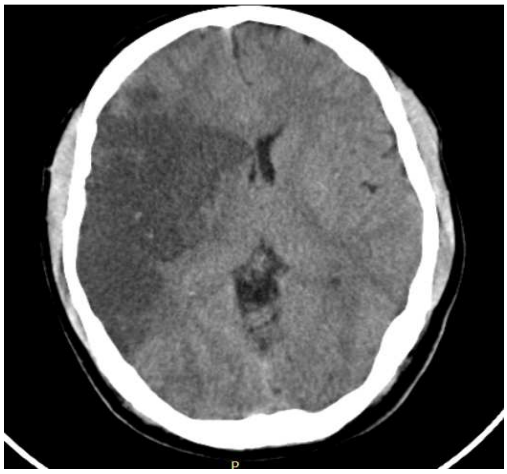


Figure 13: Acute infarct in right MCA territory with hemorrhagic transformation

SINGLE RING ENHANCING COMPUTERIZED TOMOGRAPHY LESION

Neurocysticercosis and tuberculoma both can present with seizure and CT scan picture of single enhancing lesion.

Rajshekar V, Chandy MJ[42] based on their experience proposed diagnostic criteria for cysticercus granuloma.

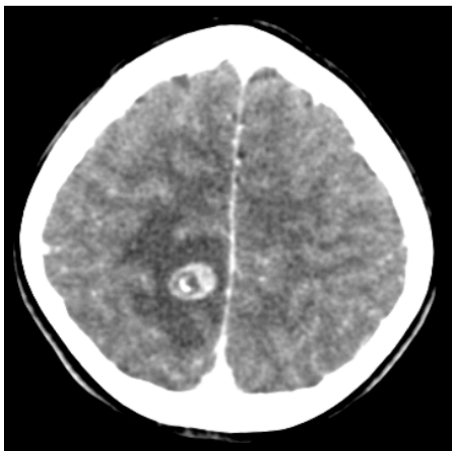


Figure 14: Ring enhancing lesion with calcification noted in left parietal region with perilesional edema – suggestive of granulomatous lesion neurocysticercosis / tuberculoma

Clinical Criteria

- Seizures (partial or generalized) as initial symptom.
- Absent persistent raised ICT.
- No progressive neurological deficit.
- No active systemic disease

CT criteria

- Solitary contrast enhancing lesion.
- Lesion less than or equal to 20 mm in diameter.
- Absence of severe cerebral edema (no midline shift)

However, none of these features are specific enough, for the diagnosis of Neurocysticercosis.

REVISED DIAGNOSTIC CRITERIA OF NEUROCYSTICERCOSIS (DEL BRUTTO et al)[43]

Absolute

1. Histological demonstration of parasite.
2. CT or MRI showing cystic lesions with scolex.
3. Fundoscopic visualization of parasite.

Major

1. Lesions suggestive of neurocysticercosis on CT or MRI.
2. Positive serum EITB.
3. Resolution of cyst after therapy.
4. Spontaneous resolution of single enhancing lesions.

Minor

1. Lesions compatible with neurocysticercosis on CT or MRI.
2. Suggestive clinical features.
3. Positive CSF ELISA.
4. Cysticercosis outside CNS.

Epidemiologic

1. Household contact with Taenia solium infection.
2. Immigration from or living in an endemic area.
3. Travel to an endemic area.

Definite: One absolute; or two major + one minor + one epidemiologic.

Probable: One major + two minor; one major + one minor + one epidemiologic; three minor + one epidemiologic.

SERUM PROLACTIN LEVELS

Prolactin (PRL) release from the pituitary is controlled by the hypothalamus via a PRL inhibitory factor, now believed to be dopamine.[44] It has been hypothesized that ictal epileptic activity in the mesial temporal structures may propagate to the hypothalamus, altering the hypothalamic regulation of PRL release.[45]

Trimble first demonstrated that generalized tonic–clonic seizures, but not non-epileptic seizures (NESs), could raise serum PRL.[46] Despite subsequent confirmatory findings, the sensitivity and specificity of serum PRL assay for diagnosis of epileptic seizures (ESs) remain uncertain. Utility of PRL assays for diagnosis of seizures depends upon the study design, standard of seizure classification, and criteria for abnormal PRL elevation. Additional uncertainty arises from the circadian fluctuations of serum PRL, demonstrating surges of 50 to 100% prior to awakening from sleep, although PRL serum levels otherwise are stable during the waking state.[47] PRL concentrations usually are higher in females than in males,[48] and higher in persons with epilepsy than in healthy individuals.[49] Psychogenic or physiologic non-epileptic events also can influence serum PRL level. Several studies[50-53] have suggested that serum PRL can increase after syncope, a common imitator of epilepsy.

In primarily or secondarily generalized tonic clonic seizures and partial complex seizures of temporal lobe origin, the serum prolactin level rises at least threefold, and often more (fivefold to twentyfold).[54] This transient post-ictal elevation has been used clinically to distinguish these seizures from non-epileptic seizures. Prolactin levels, unlike cortisol levels,[55] are seldom elevated in non-epileptic seizures. Rises that do occur are limited to the twofold level that can accompany stress or occur postprandially.

Thus, a post-ictal prolactin elevation greater than threefold suggests the presence of epileptic seizure. The lack of such an elevation makes it unlikely that an ictal event was epileptic if the event was a tonic-clonic seizure.

There are several limitations on the usefulness of post-ictal serum prolactin elevation as a test to distinguish epileptic from non-epileptic seizures:

- Post-ictal serum prolactin elevation cannot be used to differentiate focal seizures without dyscognitive features or absence seizures from non-epileptic seizures.
- Prolactin levels may increase during syncope, demonstrated during tilt table studies.[56]
- Focal seizures with dyscognitive features that do not arise from the temporal lobe do not lead to prolactin elevation.
- 10% to 20% of patients with tonic-clonic seizures may not show a post-ictal prolactin rise.
- Ambiguous test results, such as a twofold elevation, are difficult to interpret.
- Prolactin level rises predictably only after a single seizure. Patients who have more than 2 seizures in 12 hours have progressively smaller elevations, presumably because stored prolactin from the pituitary lactotrophs is exhausted.

With these caveats, however, the test is a useful one. One simple protocol involves checking serum prolactin level 20 minutes and 12 hours post-ictally (24 hours in outpatient settings) in patients whose seizure frequency is less than 1 in 12 hours. The value measured 12 or 24 hours after the seizure serves as the patient's own post-hoc baseline.

Recent interest in the use of PRL has diminished with the availability of video-EEG monitoring. However, a serum marker continues to be of potential clinical utility, especially when video-EEG is not readily available. Most laboratories report PRL upper normal limits of 18 to 23 ng/mL.[57,58] However, prior literature does not specify a precise and commonly accepted cut off PRL level as an indicator of epilepsy.[59]

METHODOLOGY

STUDY DESIGN

A prospective comparative study with 82 patients of seizures, in which 20 patients having focal seizures and 62 patients with generalized patients is undertaken to study the association of clinical profile and neuroimaging with EEG and neuroimaging in patients with focal and generalized seizures.

SOURCE OF DATA

The study was under taken over a duration of one year, of adults presenting for the first time with the seizure disorder and was admitted to **SRI R. L. JALAPPA HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR** attached to Sri Devaraj Urs Medical College and Research Centre.

DURATION

The study was undertaken for a period of one year from January 2011 to December 2011.

INCLUSION CRITERIA

Patients who had seizures for the first time after 18 years of age.

EXCLUSION CRITERIA

- Patients who had been diagnosed previously with a seizure disorder.
- Patients whose onset of seizures before 18 years of age.
- Patients who present with seizures with head injury.

METHOD OF COLLECTION OF DATA

Patients of more than 18 years of age with first time seizure disorder were included in the study presenting at Sri. R. L. Jalappa Hospital and Research Centre.

Data was collected by interview to elicit clinical profile and also by performing EEG, CT scan and serum prolactin levels.

EEG was performed with 24 channel digital EEG machine (NEUROPAGE PLUS) in Department of Neuropsychiatry, R. L. Jalappa Hospital and Research Centre.

Fig. 15: 24 channel digital EEG machine (NEUROPAGE PLUS)

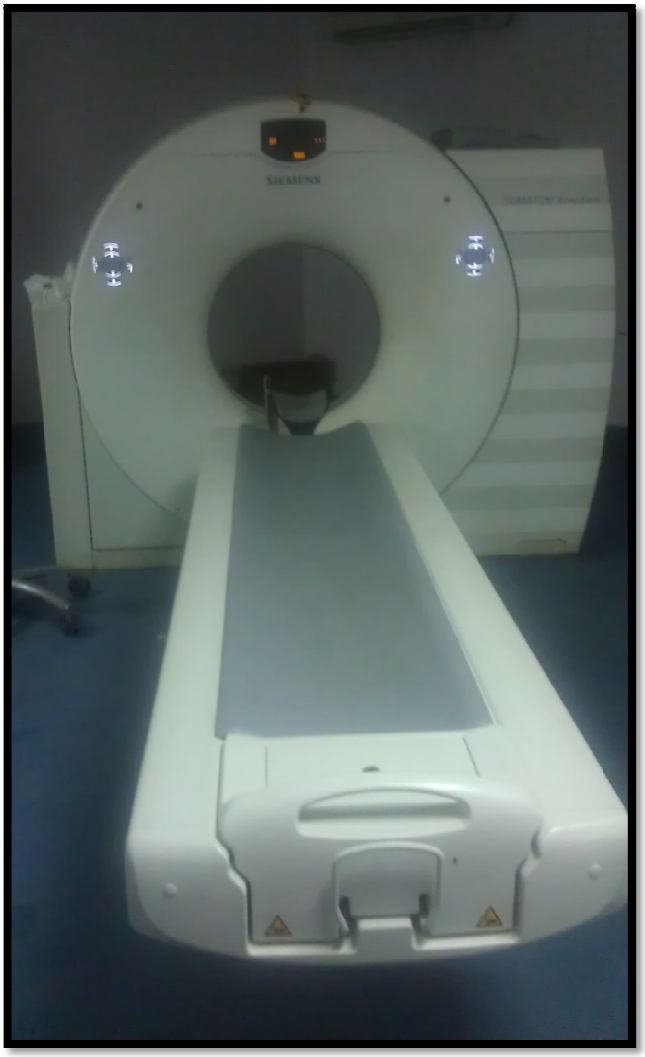


Fig. 16: Computerized Tomography was performed with 16 slice CT machine (SOMOTOM MOTION-SEIMENS) by sequence films

Data collected was studied with respect to the association of clinical profile with EEG and neuroimaging in patients with focal and generalized seizures.

Serum Prolactin Level estimation (Chemiluminiscence method) was done.

INVESTIGATIONS

The study required the following investigations:

1. EEG.
2. CT scan.
3. Serum Prolactin Levels (Chemiluminiscence method).
4. ECG, Chest X-ray, 2D-ECHO.
5. Serological tests for Syphilis and HIV detection and CSF study if required.
6. Complete blood count.
7. Routine urine examination.
8. Blood Sugar.
9. Renal profile tests.

STATISTICAL METHODS

Descriptive and inferential statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5% level of significance. The following assumptions on data is made, **Assumptions**: 1.Dependent variables should be normally distributed, 2.Samples drawn from the population should be random, Cases of the samples should be independent.

Chi-square/Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups.

1. Sample Size estimation

Proportion Known populations

$n = [(z^2 * p * q) + ME^2] / [ME^2 + z^2 * p * q / N]$

Proportion Unknown population

$n = [(z^2 * p * q) + ME^2] / (ME^2)$

ME: is the margin of error, measure of precision.

and Z is 1.96 as critical value at 95%CI

N: population size

n: Sample size

σ: Standard deviation

z: Critical value based on Normal distribution at 95% Confidence Interval

Standard deviation: $SD = \sqrt{\frac{\sum (x - \bar{x})^2}{n-1}}$

2. Chi-Square Test: The chi-square test for independence is used to determine the relationship between two variables of a sample. In this context independence means that the two factors are not related. In the chi-square test for independence the degree of freedom is equal to the number of columns in the table minus one multiplied by the number of rows in the table minus one

$\chi^2 = \frac{\sum (Oi - Ei)^2}{Ei}$, Where Oi is Observed frequency and Ei is Expected frequency

With (n-1) df

The Assumptions of Chi-square test

The chi square test, when used with the standard approximation that a chi-square distribution is applicable, has the following assumptions:

- **Random sample**: A random sampling of the data from a fixed distribution or population.
- **Sample size (whole table)**: A sample with a sufficiently large size is assumed. If a chi square test is conducted on a sample with a smaller size, then the chi square test will yield an inaccurate inference. The researcher, by using chi square test on small samples, might end up committing a Type II error.
- **Expected Cell Count**: Adequate expected cell counts. Some require 5 or more, and others require 10 or more. A common rule is 5 or more in all cells of a 2-by-2 table, and 5 or more in 80% of cells in larger tables, but no cells with zero expected count. When this assumption is not met, Fisher Exact test or Yates' correction is applied.

3. **Fisher Exact Test:** The Fisher Exact Test looks at a contingency table which displays how different treatments have produced different outcomes. Its null hypothesis is that treatments do not affect outcomes-- that the two are independent. Reject the null hypothesis (i.e., conclude treatment affects outcome) if p is "small".

The usual approach to contingency tables is to apply the χ^2 statistic to each cell of the table. One should probably use the χ^2 approach, unless you have a special reason. The most common reason to avoid χ^2 is because you have small expectation values

	Class 1	**Class 2**	**Total**
Sample1	A	B	a+b
Sample2	C	D	c+d
Total	a+c	b+d	N

2x2 Fisher Exact Test statistic= $$\sum p = \frac{(a+b)!(c+d)!(a+c)!(b+d)!}{n!}\frac{1}{\sum a!b!c!d!}$$

Fisher Exact test (rxc tables)

Let there exist two such variables X and Y, with m and n observed states, respectively. Now form an $m \times n$ matrix in which the entries a_{ij} represent the number of observations in which $x = i$ and $y = j$. Calculate the row and column sums R_i and C_j, respectively, and the total sum

$$N = \sum_i R_i = \sum_j C_j$$

of the matrix. Then calculate the conditional probability of getting the actual matrix given the particular row and column sums, given by

$$P_{\text{cutoff}} = \frac{(R_1!\,R_2!\cdots R_m!)(C_1!\,C_2!\cdots C_n!)}{N!\prod_{i,j} a_{ij}!},$$

which is a multivariate generalization of the hypergeometric probability function.

4. Significant figures

\+ Suggestive significance (p value: $0.05<p<0.10$)

* Moderately significant (p value: $0.01<p\leq 0.05$)

** Strongly significant (p value: $p\leq 0.01$)

Statistical software: The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1, Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

RESULTS

The present study was a prospective comparative study undertaken over a period of one year from January 2011 to December 2011 conducted on 82 patients.

Age distribution

Table 1: Age distribution of patients studied

Age in years	Number of patients	%
18-20	6	7.3
21-30	27	32.9
31-40	15	18.3
41-50	9	11.0
51-60	13	15.8
61-70	9	11.0
>70	3	3.6
Total	82	100.0

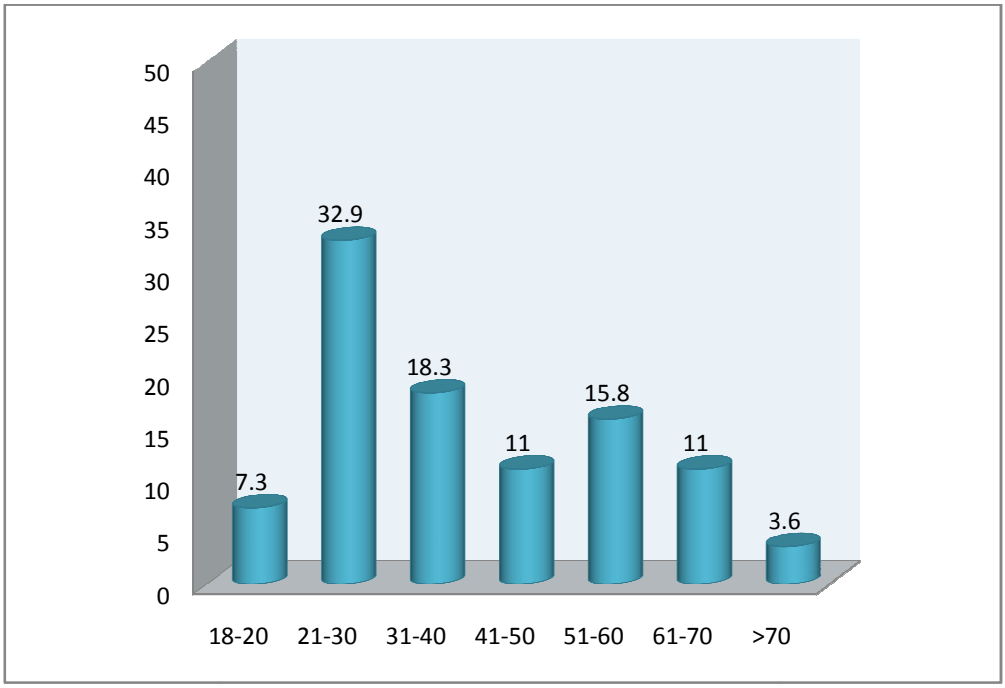


Fig. 17: Age distribution of patients studied

The youngest patient was 18 years old and the eldest patient was 74 years old Maximum numbers of patients were in the age group 21 – 30 years (32.90%).

Gender distribution

Table 2: Gender distribution

Gender	Number of patients	%
Male	42	51.2
Female	40	48.8
Total	82	100.0

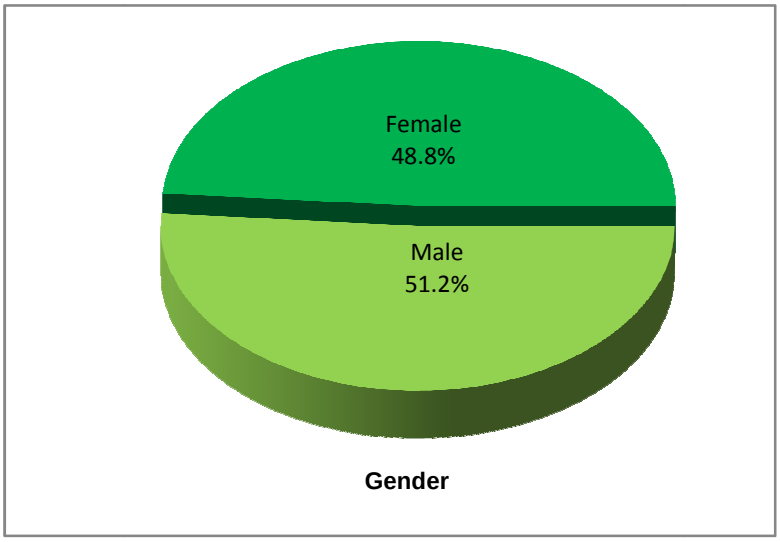


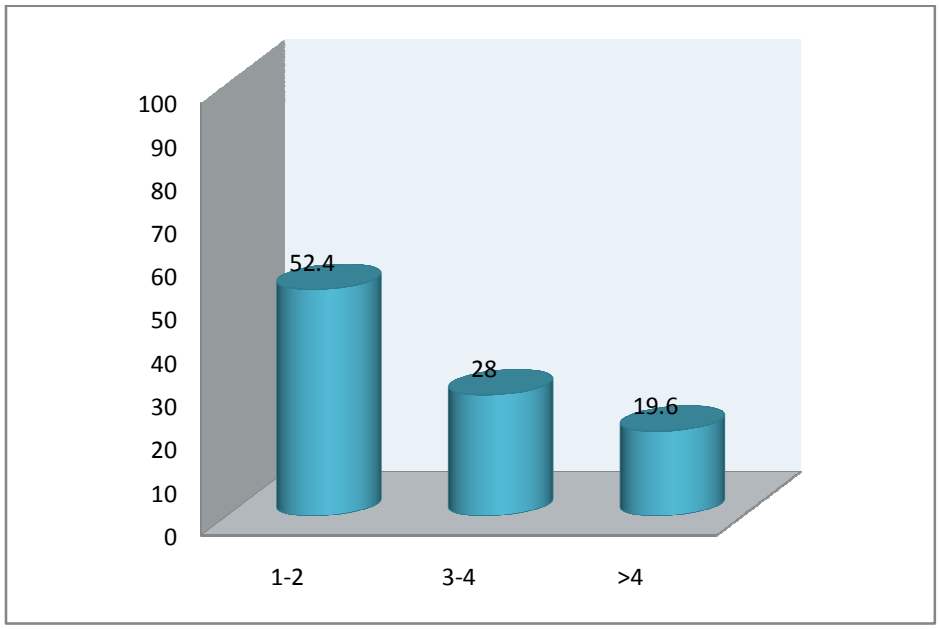
Fig. 18: Gender distribution of patients studied

Among the 82 patients studied 42 [51.2%] were males and 40 [48.8%] were females. There was no gender preponderance in the study.

Number of seizures at presentation

Table 3: Number of seizures at presentation

No. of episodes	Number of patients	%
1-2	43	52.4
3-4	23	28.0
>4	16	19.6
Total	82	100.0



At the time of presentation 43 patients [52.4%] had less than 2 seizures, 23 [28%] had 3-4 seizures and 16 patients [19.6%] had greater than 4 seizures.

Distribution of seizures of patients studied

Table 4: Distribution of seizures of patients studied

	Clinical seizure types	Numbers	Percentage
Focal	Without dyscognitive features	5	6.1
	With dyscognitive features	15	18.3
	With secondary generalization	0	0
	Total	20	24.2
Generalized	Tonic clonic (Grandmal)	62	75.6
	Absence (Petit mal)	0	0
	Tonic	0	0
	Atonic	0	0
	Myoclonic	0	0
	Total	62	75.6

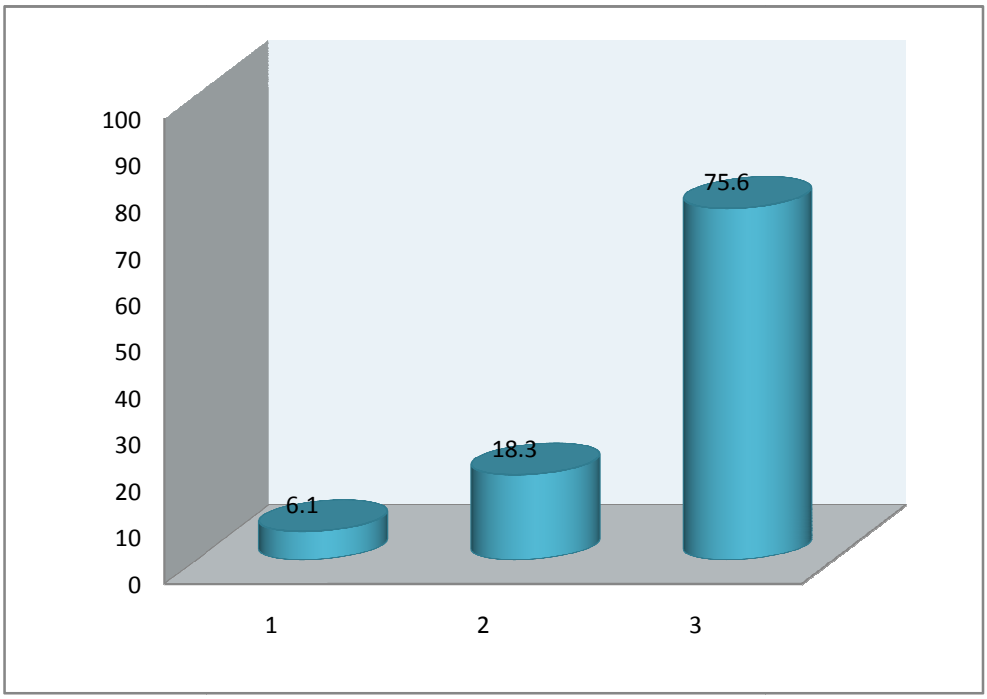


Fig. 19: Distribution of seizures

Among the 82 patients 62 (75.6%) had generalized tonic clonic seizures, 15 (18.3%) had focal seizures with dyscognitive features, 5 (6.1%) had focal seizures without dyscognitive features. Among the seizure types generalized tonic clonic seizure were the most common type.

Distribution of EEG findings of patients studied

Table 5: Distribution of EEG findings of patients studied

EEG			**Cases**	**Percentage**
Normal			47	57.30
Abnormal	Epileptiform discharges	Generalized	24	29.26
		Focal	4	4.88
		Total	28	34.14
	Focal slowing		2	2.46
	Diffuse slowing		5	6.1
	Total		35	42.7
Total			82	100

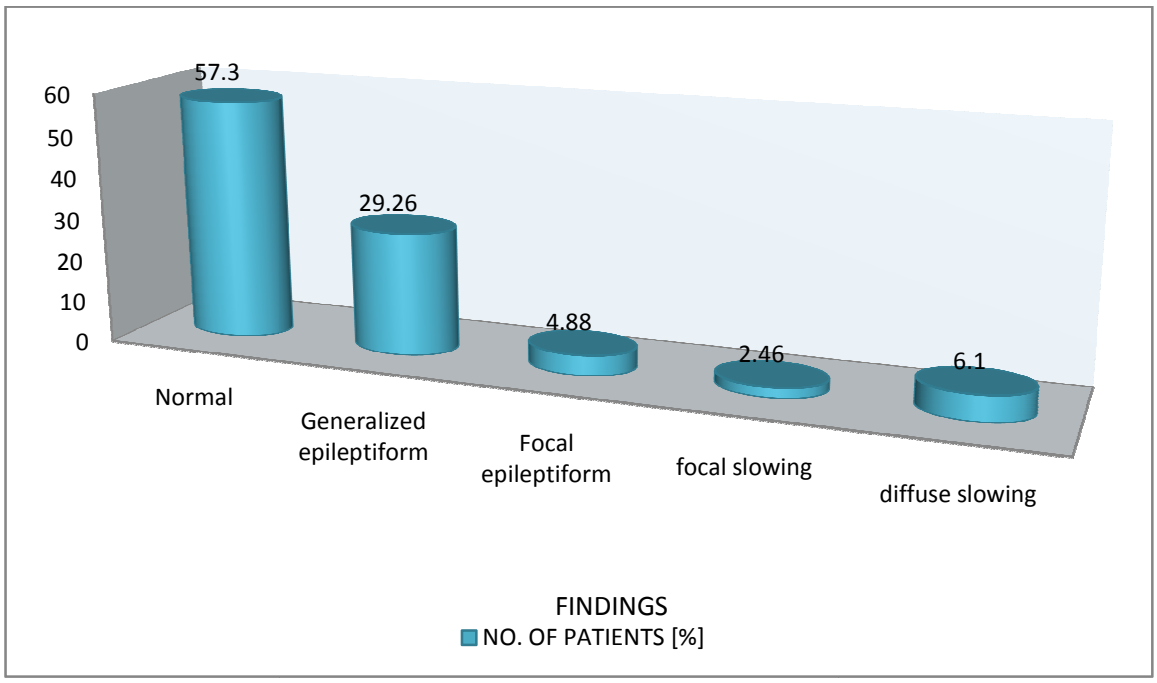


Fig. 20: Distribution of EEG findings

Overall, EEG was abnormal in 35 [42.7%, chi square=0.355, DF=1, p=0.551, not significant] patients. It showed epileptiform discharges which were generalized in 24 (29.26%) and were focal in 4 (4.88%). It showed focal slowing in 2 (2.46%) patients and diffuse slowing in 5 (6.1%) patient. Overall, generalized epileptiform discharges were the most common EEG abnormality.

EEG in generalized seizures

Table 6: EEG in generalized seizures

EEG			Cases	Percentage
Normal			40	64.52
Abnormal	Epileptiform discharges	Generalized	16	25.80
		Focal	1	1.61
		Total	17	27.42
	Focal slowing		1	1.61
	Diffuse slowing		4	6.45
	Total		22	35.48
Total			62	100

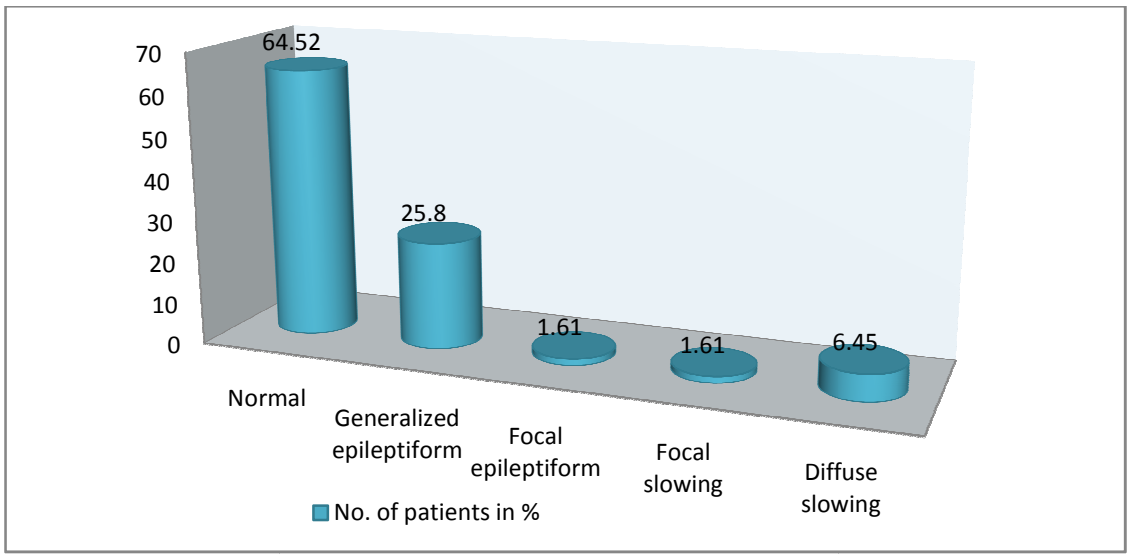


Fig. 21: EEG in generalized seizures

In generalized seizures group, EEG was abnormal in 22 (35.48%) patients. It showed epileptiform discharges which were generalized in 16 (25.80%) patients. It showed Focal epileptiform discharges in 1 [1.61%]. It showed focal slowing in 1 (1.61%) patients and diffuse slowing in 4 (6.45%) patient. Generalized epileptiform discharges where the most common EEG abnormality.

EEG in focal seizures

Table 7: EEG in focal seizures

EEG			Cases	Percentage
Normal			11	55.0
Abnormal	Epileptiform discharges	Generalized	4	20.0
		Focal	3	15.0
		Total	7	35.0
	Focal slowing		1	5.0
	Diffuse slowing		1	5.0
	Total		9	45.0
Total			20	100

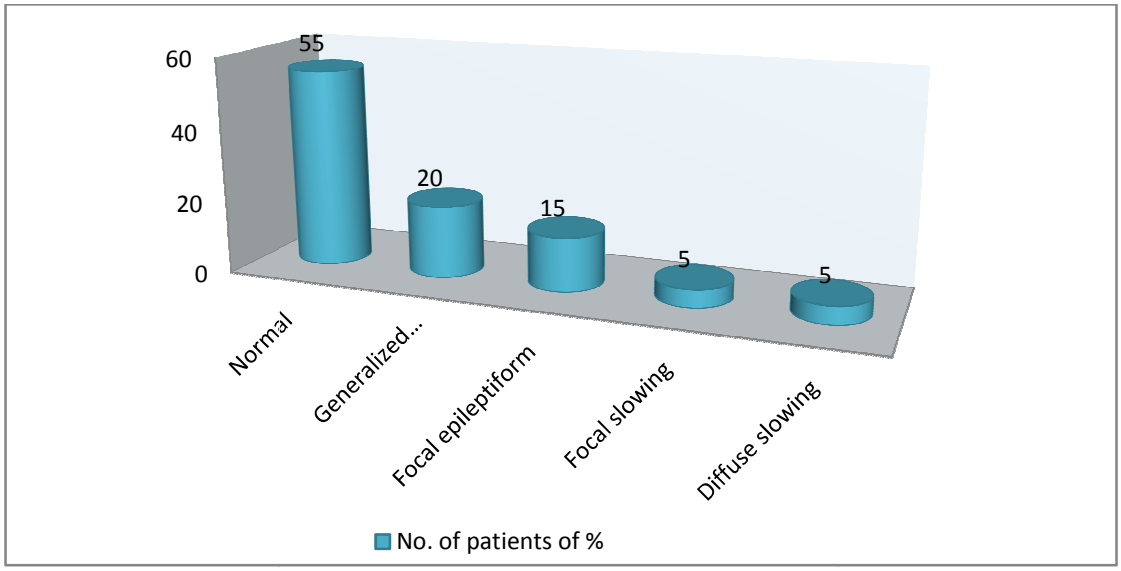


Fig. 22: EEG in focal seizures

EEG in partial seizures group was abnormal in 9 (45.0%) patients. It showed Generalized epileptiform discharges in 4 [20.0%]. It showed focal epileptiform discharges in 3 (15.0%) patient and focal slowing in 1 (5.0%) patients and diffuse slowing in 1[5.0%] patients. Generalized epileptiform was the most common EEG abnormality.

Distribution of CT brain of patients studied

Table 8: Distribution of CT brain of patients studied

CT Scan		**Cases**	**Percentage**
Normal		36	44.0
Abnormal	Ring / Disc enhancing lesions[Irrespective of size]	17	20.73
	Cerebral infarct	12	14.63
	Raised ICT	7	8.53
	Old healed, calcified granulomatous lesion	4	4.83
	Brain tumor	2	2.44
	Cortical venous thrombosis	1	1.21
	Diffused cerebral atrophy	1	1.21
	Leukoencephalopathy	1	1.21
	Tuberculoma	1	1.21
	Total	46	56.0
Total		82	100

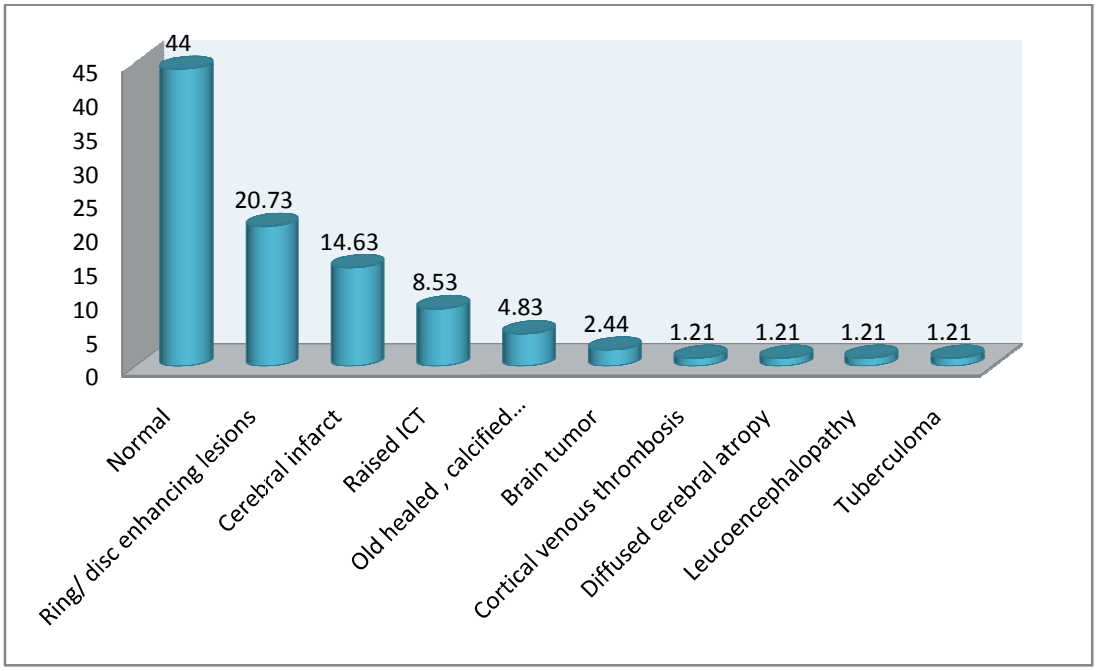


Fig. 23: Distribution of CT brain

CT scan was abnormal in 46[56.0%, chi square=14.992, DF=1, p<0.001, highly significant] patients. It showed Ring / Disc enhancing lesions (Irrespective of size) in 17 [20.73%], Cerebral infarct in 12 [14.63%], Raised ICT in 7 [8.53%], Old healed, calcified granulomatous lesion in 4 l[4.83%], Brain tumor in 2 [2.44%],and one each [1.21%] in Cortical venous thrombosis, Diffused cerebral atrophy, Leukoencephalopathy, and Tuberculoma.

Overall, ring or disc enhancing lesion was the most common abnormality.

CT scan in generalized seizures

Table 9: CT scan in generalized seizures

CT Scan		**Cases**	**Percentage**
Normal		35	56.45
Abnormal	Ring / Disc enhancing lesions[Irrespective of size]	7	11.29
	Cerebral infarct	7	11.29
	Raised ICT	6	9.68
	Old healed, calcified granulomatous lesion	2	3.24
	Brain tumor	1	1.61
	Cortical venous thrombosis	1	1.61
	Diffused cerebral atrophy	1	1.61
	Leukoencephalopathy	1	1.61
	Tuberculoma	1	1.61
	Total	27	43.55
Total		62	100

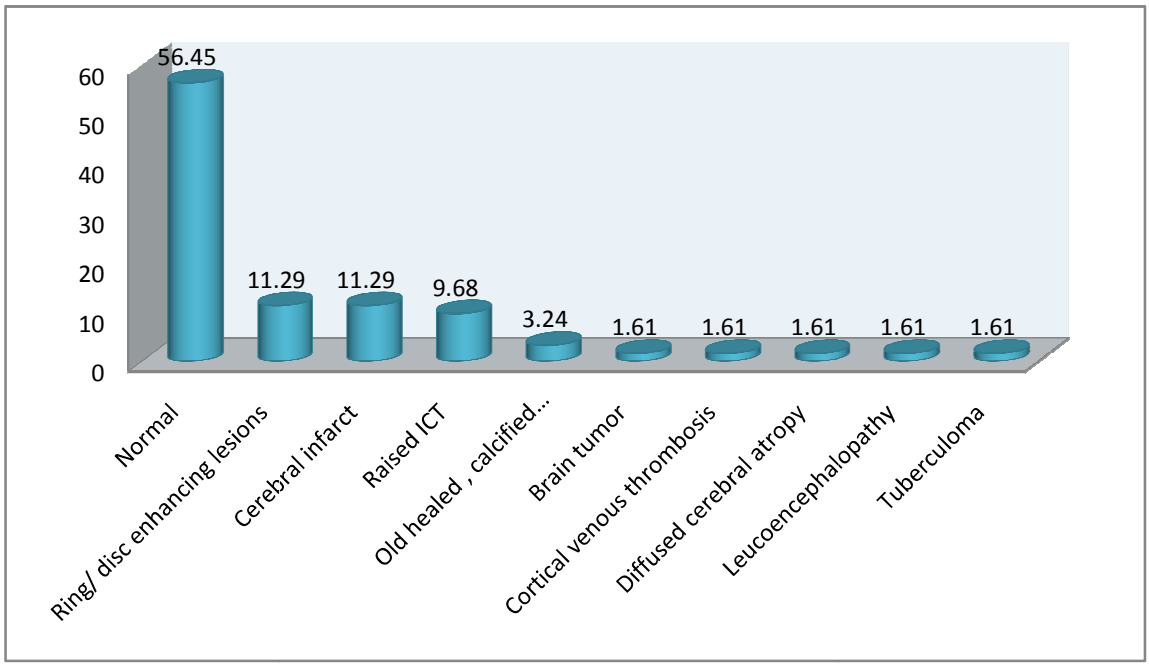


Fig. 24: CT scan in generalized seizures

CT scan was abnormal in 27 (43.54%) patients. It showed Ring / Disc enhancing lesions in (Irrespective of size) 7 [11.29%], Cerebral infarct in 7[11.29%], Raised ICT 6 [9.68%], Old healed, calcified granulomatous lesion 2 [3.24%], and one each [1.61%] in brain tumor, cortical venous thrombosis, diffused cerebral atrophy, Leukoencephalopathy and tuberculoma

Overall, ring or disc enhancing lesion was the most common abnormality.

CT scan in focal seizures

Table 10: CT scan in focal seizures

CT Scan		Cases	Percentage
Normal		1	5.0
Abnormal	Ring / Disc enhancing lesions[Irrespective of size]	13	65.0
	Cerebral infarct	2	10.0
	Raised ICT	2	10.0
	Old healed, calcified granulomatous lesion	1	5.0
	Brain tumor	1	5.0
	Total	19	95.0
Total		20	100

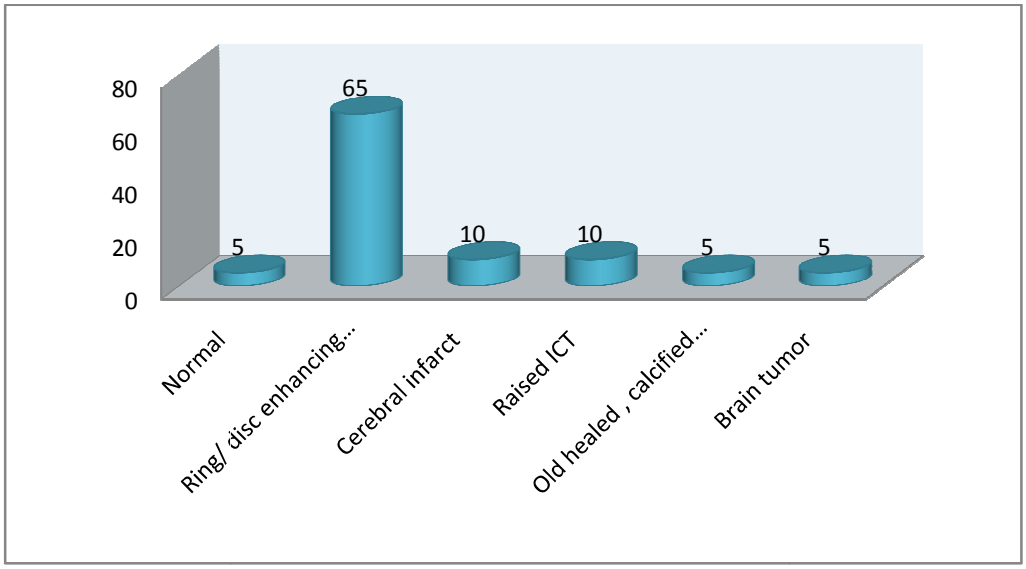


Fig. 25: CT scan in focal seizures

CT scan was abnormal in 19 (95.0%) patients. It showed Ring / Disc enhancing lesions [Irrespective of size] in 13 [65%], Cerebral infarct in 2 [10%], Raised ICT in 2 [10%], one each [5%] in old healed, calcified granulomatous lesion and brain tumor.

Overall, ring or disc enhancing lesion was the most common abnormality.

CT SCAN and EEG correlation

Table 11: CT SCAN, EEG correlation in seizures [n=82]

	Normal EEG	**Abnormal EEG**
Normal CT scan	26[31.70%]	10[12.2%]
Abnormal CT scan	22[26.83%]	24 [29.27%]

Chi square=4.952, DF=1,p=0.0261, statistically significant

Table 12: CT SCAN and EEG correlation generalized seizures [n=62]

	Normal EEG	**Abnormal EEG**
Normal CT scan	26 [41.93%]	9[14.52%]
Abnormal CT scan	12[19.35%]	15[24.20%]

Chi square=7.1195,DF=1,p=0.0076, statistically significant

Table 13: CT SCAN and EEG correlation partial seizures [n=20]

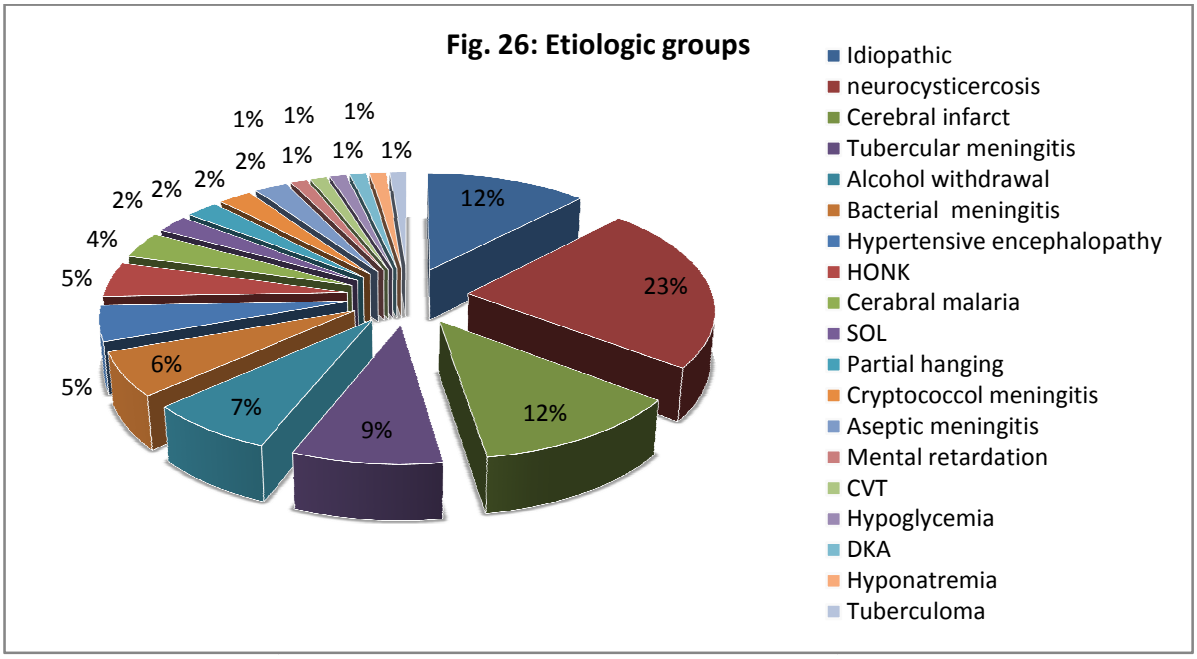
	Normal EEG	**Abnormal EEG**
Normal CT scan	0	1[5%]
Abnormal CT scan	10[50%]	9[45%]

Chi square=1.0526, DF=1,p=0.3049, not significant

Etiological Groups

Table 14: Etiological Groups

		Number	%
I. Idiopathic (No cause detected)		10	12.20
II. Cause detected in		72	87.80
1	Neurocysticercosis	19	23.17
2	Cerebral infarct	10	12.20
3	Tubercular meningitis	7	8.54
4	Alcohol withdrawal	6	7.32
5	Bacterial meningitis	5	6.10
6	Hypertensive encephalopathy	4	4.88
7	HONK	4	4.88
8	Cerebral malaria	3	3.66
9	SOL	2	2.44
10	Partial hanging	2	2.44
11	Cryptococcol meningitis	2	2.44
12	Aseptic meningitis	2	2.44
13	Mental retardation	1	1.21
14	CVT	1	1.21
15	Hypoglycemia	1	1.21
16	DKA	1	1.21
17	Hyponatremia	1	1.21
18	Tuberculoma	1	1.21



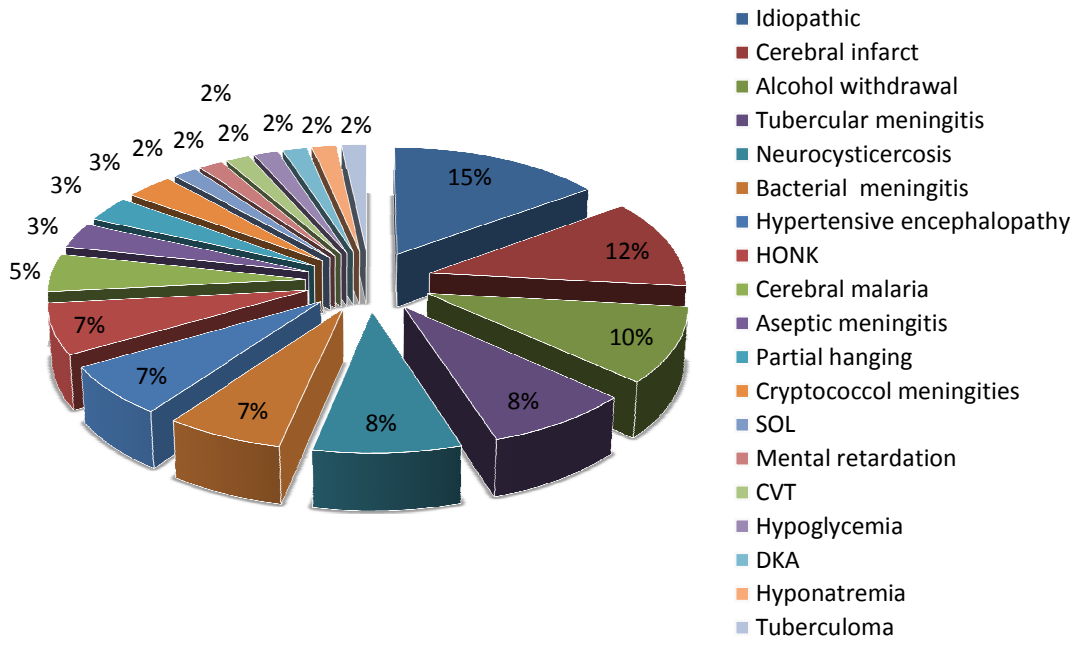
Overall, the cause was not found in 10(12.2%), etiology was found in 72(87.8%) with neurocysticercosis being the most commonest cause. The other common causes are CVA in 10(12.2%), Neurotuberculosis in 7(8.54%), Alcohol abuse in 6(7.32%) followed by others.

Etiological Groups in Generalized Seizures

Table 15: Etiological Groups in Generalized Seizures (n=62)

		Number	%
I. Idiopathic (No cause detected)		9	14.51
II. Cause detected in		53	85.48
1	Cerebral infarct	7	11.29
2	Alcohol withdrawal	6	9.68
3.	Tubercular meningitis	5	8.06
4.	Neurocysticercosis	5	8.06
5.	Bacterial meningitis	4	6.45
6.	Hypertensive encephalopathy	4	6.45
7.	HONK	4	6.45
8.	Cerebral malaria	3	4.84
9.	Aseptic meningitis	2	3.23
10.	Partial hanging	2	3.23
11.	Cryptococcol meningitis	2	3.23
12.	SOL	1	1.61
13.	Mental retardation	1	1.61
14.	CVT	1	1.61
15.	Hypoglycemia	1	1.61
16.	DKA	1	1.61
17.	Hyponatremia	1	1.61
18.	Tuberculoma	1	1.61

Fig. 27: Etiologic groups of generalized seizures

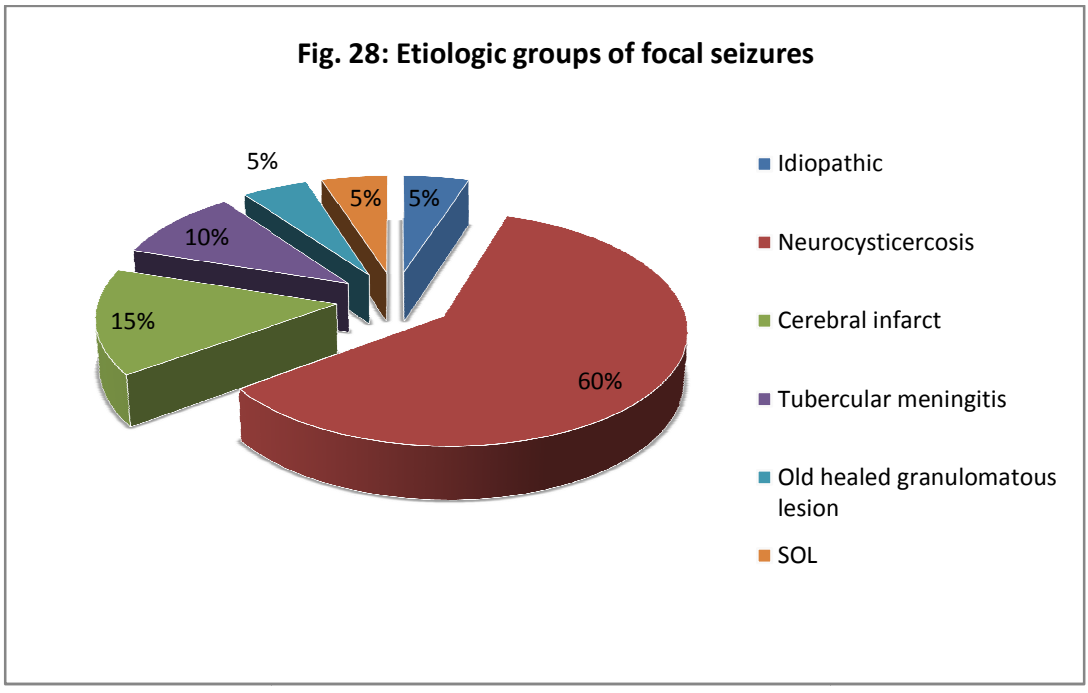


Overall, cause was not found in 9(14.51%) and cause was found in 53(85.48%) in generalized seizures. The commonest cause was CVA in 7 (11.29%), Alcohol abuse in 6(9.68%), Neurotuberculosis in 5(8.06%), Neurocysticercosis in 5(8.06%) and followed by others.

Etiological Groups in Focal Seizures

Table 16: Etiological Groups in Focal Seizures (n =20)

		Number	%
I. Idiopathic (No cause detected)		1	5.0
II. Cause detected in		19	95.0
1	Neurocysticercosis	12	60.0
2	Cerebral infarct	3	15.0
3.	Tubercular meningitis	2	10.0
4.	Old healed granulomatous lesion	1	5.0
5.	SOL	1	5.0



The etiology was found in majority of cases i.e., 19(95%) and the most commonest cause being neurocysticercosis in 12(60%).

Serum Prolactin Levels

Serum Prolactin Levels were done within 20 minutes of the suspected ictal event to differentiate GTCS or Focal seizures with dyscognitive features from psychogenic non epileptic seizures, though serum prolactin levels does not distinguish epileptic seizures from syncope. The use of serum prolactin assay has not been established in evaluation of status epilepticus, repetitive seizures and neonatal seizures.

Serum prolactin levels were done by chemiluminiscence assay method in the present study.

Table 17: Serum prolactin levels

Seizure type	Serum prolactin levels		Total
	Normal	Abnormal	
Tonic-clonic	22	40	12
Focal	13	07	19

Sensitivity: 64.5%, Specificity: 37%

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DISCUSSION

Clinical Profile

1. Age Distribution

There is rising trend of epilepsy with increasing age, the peak being the third and fourth decade of life.[60] But there is no consensus on this issue as different trends have been witnessed across various countries.[61] The age of onset of epilepsy has been reported in the first two decades of life in 75-80% patients[62,63] and in 68.8% before 15 year of age in another study[64,65] Troster[66] has found that 68% cases had age of onset before 20 years. In Ethiopia,[67] Nigeria,[61,68] and Sri Lanka,[69] the highest prevalence was in the second decade of life; in Guam[70] in the third decade; and in Ecuador[79] in the fifth decade. The onset of epilepsy is in the first two decades of life in 80% of the patients in Mathai's study.[92] In rural Kashmir, 90% of the surveyed patients reported onset of epilepsy before the age of 30 years,[64] whereas, Bharucha et al[72] in their study of the Parsi community of Bombay, noted that the median age of onset was 22 years. Age-specific prevalence rates were highest in the second decade in men and in the third and fourth decades of women. The possibility of under ascertainment of seizure disorders leading to lower prevalence rates in the elderly cannot be ruled out. The age-specific prevalence was found to be higher with increasing age.[73,74] In the present study, the highest prevalence was in third decade of life.

2. GENDER

Most studies of epilepsy in industrialized countries report that males are more frequently affected than females, although the difference is seldom statistically significant. Results from developing countries are similar, although, some studies in Nigeria,[61,68] and Latin America,[71,75,76] have found higher prevalences for females.Many studies have shown higher prevalences for males[64,72,77-81], though studies by Sohi et al[82] and Senanayke et al[15] did not show any difference in the two sexes. Similarly, the present study also did not show any differences in the two sexes.

3. POPULATION PROFILE

The literature reveals that rural populations are at higher risk than urban populations to have epileptic seizures.[83-85] Various studies on the epidemiology of epilepsy in India have been published from 1964 until date. Nine studies were done with urban or semi urban populations, 14 with rural ones, and three with both rural and urban ones.[86] Present study was based entirely on rural population.

4. BEHAVIOURAL CHANGES

Epilepsy can be accompanied by changes in cognition, personality, and other elements of behaviour. There is no true epileptic personality complex, the only unifying theme to the behavior in epilepsy is diversity.[87] Some patients may be irritable and aggressive, whereas, others may be timid and athletic. Psychosis, depression, paranoia and personality disorders may represent a negative role of epilepsy related behavioural changes. Common behavioural features in epilepsy include changes in emotional state with deepening or increase in emotionality.[87] The association of specific behavioural

changes with epileptic patients has been questioned for hundreds of years,[88-89] contrary to this, Koul et al[64] found behavioural changes in 15.2% cases. In the present study, 17% of patients had behavioural changes.

5. SEIZURE TYPE

The profile of epilepsy varies across various cultures and the review shows that in western countries about two-third of the epileptic patients have partial seizures.[91,92] Similar trend has been shown in some developing countries like Nigeria.[93] Predominance of partial seizures (52.1%) over generalized seizures (47.11%) though of lesser magnitude has been reported from Peru.[94] On the contrary, reverse trend has been reported in Indian studies, where generalized seizures constitute more than 70% of all seizures.[64,95,96] However, Bharucha et al[72] have reported higher incidence of partial seizures (54.5%) than generalized seizures (45.4%) in Parsi community from Mumbai in India. The lower frequency of partial seizures in developing countries has been attributed to ascertainment problems than to any specific geographical trend. Shorvon and Farmer[97] are of the opinion that partial seizures may be underreported in studies that use inadequate screening questionnaires. They have illustrated their view with the help of an example of a study from China[65] where 81% epileptic patients showed generalized convulsive seizures. In the present study we report higher incidence of generalized seizures (76.8%) than focal seizures(24.2%)which correlates with the Indian studies quoted above.

Electroencephalograph (EEG)

Ghazy et al[98] studied 89 patients with epileptic seizure disorder and found EEG to be abnormal in 89% of patients.

Newfeld MY et al[99] studied 91 patients with generalized seizure and the EEG was taken within 48 hours of the seizures. Abnormal EEGs were obtained in 69% of patients. Epileptiform activity was present in 21% of patients, (10% focal, 9% generalized, 2% focal and generalized), slowing in 58% (21% focal, 31% generalized, 7% focal and generalized) and both epileptiform activity and slowing in 10%.

Forsgren L et al[100] studied 103 adult persons over 17 years with newly diagnosed epileptic seizures, with EEG. Epileptiform activity was recorded in 18% of patients and was more common in partial than generalized seizures.

Lt. Col. SK Jha[40] studied 150 cases of solitary seizures. EEG was done in all patients, and was abnormal in 22% of patients.

Ramesh Baheti et al[101] found an abnormal EEG in 73% and 76% of patients with partial and generalized seizures respectively.

In the present study EEG was abnormal in 42.69% [chi square=0.355, DF=1, p value=0.551,not significant] of patients. It was abnormal in 35.48% (n=22) of patients with generalized seizures. But in this group epileptiform discharges were generalized in 25.8%(n=16) of patients and helped to confirm the diagnosis. It showed focal slowing in 1.61%(n-=1) of patients where it correlated with underlying structural lesion in the form

of brain tumor. It showed diffuse slowing in 6.45% (n=4) of patients where in there was diffuse cerebral abnormality.

EEG was abnormal in 45% (n=9) of patients with focal seizures. But in this group generalized epileptiform activity was seen in 20% (n=4), there was focal slowing in 5% (n=1) and focal epileptiform discharge in 15% (n=3) which correlated with underlying structural lesion.

Thus, EEG helps us to confirm the diagnosis of generalized seizures and in case of structural lesions, though not sensitive enough, focal abnormalities gave a clue to the underlying focal structural abnormalities.

CT-SCAN

Scollo-Lavizzari G[102] et al have found that CT was abnormal in 62.5% of patients with partial seizures and 34% of patients with generalized seizures.

Guberman A[36] (1983) studied CT scan results in a consecutive series of 196 adult epileptics. In the consecutive series overall incidence of abnormal scans was 16% with the highest yield (44%) found in patients with partial seizures. In 25 of 51 cases with abnormal scans a specific lesion amenable to therapy was detected, including 16 neoplasms and 5 arteriovenous malformations. Other lesions included generalized or focal atrophy, infarcts, calcified lesion of tuberous sclerosis, unexplained calcifications and focal low density or enhancing lesions.

Reinikainen KJ et al[37] (1987) studied incidence and CT abnormalities and their correlates with clinical and EEG features were evaluated in a consecutive series of 202 adult patients with newly diagnosed epileptic seizures. Abnormal CT findings were found in 36% of the patients. The abnormalities consisted of brain tumors (17%) atrophic lesions (11%) and other finding (8%) such as arteriovenous malformations.

De La Sayette V et al[109] (1987) reviewed the CT findings of 387 patients with new onset seizures after the age and 50. CT scanning revealed cerebral atrophy in 113 cases, ischemic lesions in 75, cerebral neoplasm in 20 and no abnormality in 177 cases.

Bajaj S et al[39] (1991) studied results of CT findings in 170 patients who developed seizures at PG department of Medicine, SRN Hospital, Allahabad. The commonest abnormality was a focal ring or disc enhancing lesion in 66 patients (62.3%) followed by calcification in 18 (16.9%) cerebral atrophy 9 (8.5%) vascular lesions 7 (6.6%) tumors 4 (3.8%) and congenital hydrocephalus 2 (1.5%).

Lt. Col. SK Jha[40] (2004) studied 150 consecutive cases of solitary seizures who reported to the neurology services between 1995 and August 1997 at their neurology department. CT scan head was done in 119 patients. Out of the total of 119 CT scans, 91 cases (76.4%) were normal and was abnormal in 28. The abnormalities were disc or ring enhancing lesion in 8 calcified or nodular lesion in 9 and small cystic lesion in 2.

Rogel-Ortiz FJ[26] (1999) prospectively studied 130 adult patients with adult onset epilepsy after 20 years. All the patients had CT scan brain and when necessary brain magnetic resonance imaging. They found structural brain lesion in 51% of patients. The most frequent causes of seizures were neurocysticercosis in 28% followed by cerebral infarct (11%) and brain atrophy (11%).

Overall in present study CT scan was found to be abnormal in 56% [chi square=14.992, DF=1, p value<0.001, highly significant] of patients. It was abnormal in 43.54% (n=27) of generalized seizures and 95% (n=19) of partial seizures. Compared to the previous studies the percentages of CT scan abnormalities were higher in present study.

Single, small ring or disc enhancing lesion was the most common abnormality in our study and is consistent with other studies done by Wadia et al[104], Misra et al[105], Murthy et al.[106] Only the incidence was higher in present study compared to these studies.

The incidence of other abnormalities like brain tumors, cerebral infarct, calcification and cerebral atrophy were comparable to other studies.

The high incidence CT scan abnormalities as well as ring or disc enhancing lesions can be explained because of selection bias.

Etiology of seizures

The present study has revealed an etiology of seizures in 87.80% (n=72) of patients in general, 85.49% (n=53) of patients with generalized seizures and 95% of patients (n=19) with partial seizures. High percentage of etiology detected in the present study is due to exclusion of psychogenic non epileptiform seizures by subjecting the patients to serum prolactin level estimation within 20 minutes of ictal event by chemiluminiscence method.

The studies of Perez Lopez JL et al[20], Dam AM et al[19], Schoenenberger RA[22] who revealed high percentage of seizures where in etiology was detected, alcohol abuse formed a significant etiological factor accounting for 19.5%, 38% and 14.2% of seizures in their studies respectively.

Medina MT et al[23] Rogel-Ortiz FJ[24], Marco T. Medina et al[25] have found that Neurocysticercosis was the main etiology and accounts for 50%, 28% and 37% cases of seizures, in their studies respectively. It is similar to present study where neurocysticercosis accounts for 23.17% (n=19) of seizures.

In the present study, neurocysticercosis (23.17%, n=19) was the main etiology followed by cerebrovascular accident (12.1%, n=10), tubercular meningitis (8.53%, n=7) and alcohol abuse formed 7.31% (n=6).

CT scan and EEG correlation

In the overall group of seizures, when the CT scan was normal EEG was abnormal in 12.2% (n=10) of cases, and when CT scan was abnormal EEG was normal in 26.82% (n=22) of cases. Both were abnormal in 29.26% (n=24) of cases. Both were normal in 31.7% (n=26) of cases. Their correlation was studied with chi square test and showed moderate correlation as p=0.0261.

In the generalized seizure group when the CT scan was normal EEG was abnormal in 14.51% (n=9) of cases, and when CT scan was abnormal EEG was normal in 19.35% (n=12) of cases. Both were normal in 41.93% (n=26) of cases. Both were abnormal in 19.35% (n=12) of cases. Their correlation was studied by chi square test and showed mild correlation as p=0.0076.

In the focal seizure group when the CT scan was normal EEG was abnormal in 5% (n=1) of cases, and when CT scan was abnormal EEG was normal in 50% (n=10) of cases. Both were abnormal in 45% (n=9) of cases. Both were normal in 0% of cases. Their correlation was studied by chi square test and showed poor correlation as p=0.3049.

This is consistent with studies done by Chayasirisobhon et al[107] and Kuruvilla A et al[108] wherein they studied the correlation between neurocysticercosis lesions and EEG abnormalities. These authors have concluded that the EEG abnormality does not depend on the number of lesions, but rather on location, host response and viability of cysts.

CONCLUSIONS

- Among the patients presenting with unprovoked seizures majority had generalized seizures than focal seizures.

- EEG was not very significant in the evaluation of seizures. However, abnormal EEG helped to confirm the diagnosis of seizures and when it showed focal abnormalities gave a clue to the underlying structural abnormality.

- Overall, CT scan was abnormal in 56% of patients ($p<0.01$), 43.55% of generalized seizure group and 95% of focal seizure group patients. CT scan was reliable in revealing structural abnormalities and was helpful in establishing the diagnosis in majority of cases.

- CT scan and EEG correlation is significant in seizures in general and as well as generalized seizures. CT scan and EEG did not correlate in focal seizure group. In majority of cases of generalized seizures, a cause was detected.

- Neurocysticercosis was the most common cause of focal seizure.

- The main advantage of using serum prolactin as a surrogate marker is that the physician can be fairly sure that if the prolactin level is increased two fold after an event, it’s probably an epilepsy or syncope. If the EEG shows epileptic activity, then a diagnosis could be established—but it would have been established without prolactin analysis.

- Moreover, serum prolactin levels are low in specificity and sensitivity in detecting epileptic seizures, added to which are the practical problems of collecting blood samples within twenty minutes of ictal event.

- The search for other epilepsy markers continues.

SUMMARY

The present study was conducted in 82 patients among whom 62 patients had generalized seizures and 20 patients had focal seizures. Overall, among all the patients presenting with unprovoked seizures, generalized seizures were seen in 76.8% of cases and focal seizures were seen in 24.20% of cases. EEG was not very useful in the evaluation of seizures. However, abnormal EEG helped to confirm the diagnosis of seizures and when it showed focal abnormalities gave a clue to the underlying structural abnormality.

CT scan of brain was very reliable in detecting structural brain abnormalities. It was normal in majority of patients with generalized seizures and abnormal in majority of patients with focal seizures. In focal seizure group single, small ring enhancing lesion was the most common structural abnormality. On complete evaluation most common cause of generalized seizures was idiopathic whereas most common cause of focal seizures was neurocysticercosis. Serum prolactin levels are low in specificity and sensitivity in detecting epileptic seizures, added to which are the practical problems of collecting blood samples within twenty minutes of ictal event.

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ANNEXURE A

PROFORMA

Title of the Topic

"A STUDY OF ASSOCIATION OF CLINICAL PROFILE WITH EEG AND NEUROIMAGING OF PATIENTS WITH PARTIAL AND GENERALIZED SEIZURES"

Name:

Age:

Sex:

Religion:

Address:

DOA:

DOD:

Occupation:

Final Diagnosis:

A. PRESENT HISTORY: Seizure Pattern

1. First Seizure

2. Frequency

3. Last Seizure

4. Type of Seizure:

a. Focal

i. Without dyscognitive features

ii. With dyscognitive features

iii. Evolution to generalized seizures

b. Generalized

i. Tonic-clonic

ii. Tonic

iii. Atonic

iv. Myoclonic

v. Absence

c. Others:

5. Detailed description of one such seizure:

ASSOCIATED NEUROLOGICAL PROBLEMS

1. Symptoms of raised intracranial tension

a. Headache

b. Blurred vision

c. Projectile vomiting

d. Impaired / altered consciousness

2. Focal neurological deficits

3. Symptoms of neuro infection

a. Fever

b. Neck stiffness

c. Focal deficits

4. Trauma

5. Features of degenerative neurological disease

6. Any others

ASSOCIATED NON-NEUROLOGICAL PROBLEMS

1. Infection

a. Fever

b. Ear discharge

c. Nasal discharge

d. Osteomyelitis

e. Productive cough

2. Cardiovascular disease

a. Hypertension

b. Chest pain

c. Palpitations

3. Metabolic disease

a. Diabetes mellitus

b. Uraemia

4. Collagen Vascular disease

a. Rash

b. Muscular aches

c. Joint pains

5. Malignant disease

a. Anorexia

b. Cachexia

B. <u>PAST HISTORY</u>

1. Mental retardation

2. Febrile convulsions

3. Childhood seizures

4. Infections (Encephalitis, Meningitis)

5. Treatment history

a. Drug

b. Dosage

c. Duration d. Response

C. FAMILY HISTORY

1. Epilepsy
2. Neurological disease

D. PERSONAL HISTORY

1. Diet
2. Appetite
3. Sleep
4. Bowel and Bladder
5. Habits
6. Alcohol
7. Stimulant drugs (Amphetamine/cocaine)

PHYSICAL EXAMINATION

I. General

1. Pallor
2. Jaundice
3. Cyanosis

4. Clubbing

5. Lymphadenopathy

6. Pedal oedema

7. Cutaneous markers of neuroepidermal syndromes :

    a. Café-au-lait spots

    b. Malar rash

    c. Facial port wine spot

    d. Depigmented spots on trunk

II. Vitals

Pulse:

BP:

Respiratory Rate:

Temperature:

III. Systemic examination

A) NEUROLOGICAL

a. Higher mental function

b. Cranial Nerves

c. Motor system

d. Sensory system

e. Signs of meningeal irritation

f. Cerebellar signs

g. Skull and spine

B) CARDIOVASCULAR SYSTEM

C) RESPIRATORY SYSTEM

D) ABDOMINAL SYSTEM

INVESTIGATIONS

- CBC
- Routine urine examination
- Blood sugar

- Blood urea
- Serum Creatinine
- Serum electrolytes
- S. Prolactin levels [chemiluminiscence method]
- ELECTROENCEPHALOGRAPHY

Normal

Abnormal

a. Focal

b. Diffuse

c. Epileptiform discharges

i. Generalized

ii. Focal

d. Provocative technique

CT-SCAN BRAIN:

1. Normal:

2. Abnormal:

   a. Site of lesion:

   b. Nature of lesion:

   c. Diagnosis:

Other investigations as necessary such as

- Serological tests

a. Syphilis:

b. HIV:

- LP CSF study
- ECG:
- CXR:
- Echocardiography

KEY TO THE MASTER CHART

IP. No.	→	Inpatient Number
ng/dl	→	Nanograms per deciliter
EEG	→	Electroencephalography
CT	→	Computed Tomography
CVA	→	Cerebrovascular Accident
DM	→	Diabetes Mellitus
CVT	→	Corticovenous Thrombosis
ICSOL	→	Intra-cerebral Space Occupying Lesion
HTN	→	Hypertension
M	→	Male
F	→	Female

MASTER CHART

SL. No.	NAME	AGE	SEX	IP NUMBER	SEIZURE PATTERN	FOCAL SEIZURES WITH OUT DYSCOGNITIVE FEATURES	FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES	GENERALISED SEIZURES	SERUM PROLACTIN LEVELS(ng/dl)	EEG	CT-BRAIN	DIAGNOSIS
1	VENKATESH	25	M	766619	4 EPISODES			TONIC-CLONIC	11	NORMAL	NORMAL	MENTAL RETARDATION
2	ABDUL BASHEER	60	M	666629	2 EPISODES			TONIC-CLONIC	40.33	GENERALISED EPILEPTIFORM DISCHARGES	SUB ACUTE INFARCT IN RIGHT SIDED OCCIPITO-FRONTAL REGION.IMPRESSION:CVA	CVA(CEREBROVASCULAR ACCIDENT) WITH TYPE 2 DM
3	GOPALAKRISHNA	45	M	738577	1 EPISODE		YES		8.9	NORMAL	RING ENHANCING LESION IN RIGHT SIDED HIGH PARIETAL AREA.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
4	USHA	25	F	667432	2 EPISODES	YES			19	LATERALISED ABNORMAL ELECTRICAL ACTIVITY	NORMAL	IDIOPATHIC SEIZURE DISORDER
5	VENKATESH	50	M	782111	3 EPISODES			TONIC-CLONIC	16	NORMAL	NORMAL	ALCOHOL WITHDRAWAL SEIZURES
6	CHINNAPPA	70	M	694542	1 EPISODE			TONIC-CLONIC	11.36	NORMAL	ACUTE INFRACT IN RIGHT FRONTAL LOBE.IMPRESSION:CVA	CVA
7	MUNIYAMMA	65	F	701955	12 EPISODES			TONIC-CLONIC	52.66	GENERALISED EPILEPTIFORM DISCHARGES	SAGITTAL SINUS THROMBOSIS.IMPRESSION:CVT	CVT(CEREBROVENOUS SINUS THROMBOSIS) WITH TYPE 2 DM
8	VARALAKSHMI	25	F	758789	1 EPISODE		YES		16	NORMAL	RING ENHANCING LESION IN RIGHT SIDED HIGH PARIETAL AREA.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
9	AMEENA BEE	55	F	672762	2 EPISODES			TONIC-CLONIC	18.6	NORMAL	LEUCOENCEPHALOPATHIC CHANGES.IMPRESSION:HYPERTENSIVE ENCEPHALOPATHY	HYPERTENSIVE ENCEPHALOPATHY , UNCONTROLLED TYPE 2 DM WITH DIABETIC NEPHROPATHY
10	CHANDRASHEKAR	25	M	559339	3 EPISODES			TONIC-CLONIC	33.62	NORMAL	NORMAL	IDIOPATHIC SEIZURE DISORDER
11	GIDDAMMA	58	F	695806	3 EPISODES			TONIC-CLONIC	29.88	NORMAL	RIGHT PARIETAL LOBE OLD HEALED GRANULOMA.IMPRESSION:?NEUROCYSTICERCOSIS	TUBERCULAR MENINGITIS
12	SARASWATHI	37	F	761490	3 EPISODES		YES		16.36	FOCAL SLOWING	RIGHT SIDED FRONTO PARIETAL INFARRCT.IMPRESSION:CVA	CVA
13	RAJANNA	35	M	776221	8-10 EPISODES			TONIC-CLONIC	26	NORMAL	BILATERAL FRONTAL,HIGH PARIETAL & LEFT TEMPORAL CALCIFIC LESIONS .IMPRESSION:OLD HEALED GRANULOMATOUS LESION	DIFFUSE OLD HEALED GRANULOMATOUS LESION
14	SHANKARAMMA	53	F	675601	3 EPISODES			TONIC-CLONIC	14.36	NORMAL	NORMAL	ACUTE BACTERIAL MENINGITIS
15	SURESH	25	M		6 EPISODES		YES		16.86	DIFFUSE SLOWING	LEFT FRONTO TEMPORAL REGION- FOCAL AREA OF CEREBRAL EDEMA.IMPRESSION:RAISED INTRACRANIAL TENSION	TUBERCULAR MENINGITIS
16	BHAVANI	55	F	678194	3 EPISODES			TONIC-CLONIC	51.62	DIFFUSE SLOWING	RIGHT FRONTO TEMPORAL REGION- FOCAL AREA OF CEREBRAL EDEMA.IMPRESSION:RAISED INTRACRANIAL TENSION	TUBERCULAR MENINGITIS
17	MUNIYAMMA	22	F	757514	1 EPISODE		YES		18.36	NORMAL	RING ENHANCING LESION IN LEFT FRONTAL LOBE.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS

MASTER CHART

SL. No.	NAME	AGE	SEX	IP NUMBER	SEIZURE PATTERN	FOCAL SEIZURES WITH OUT DYSCOGNITIVE FEATURES	FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES	GENERALISED SEIZURES	SERUM PROLACTIN LEVELS(ng/dl)	EEG	CT-BRAIN	DIAGNOSIS
18	MUNILAKSHMAMMA	60	F	665631	3 EPISODES			TONIC-CLONIC	32	NORMAL	NORMAL	IDIOPATHIC SEIZURE DISORDER
19	MANJULAMMA	23	F	682712	4 EPISODES		YES		36	GENERALISED EPILEPTIFORM DISCHARGES	B/L BASAL GANGLIA INFARCT.IMPRESSION:CVA	CVA-BILATERAL BASAL GANGLIA INFARCT
20	MANGALA	22	F	732012	1 EPISODE	YES			29	NORMAL	RING ENHANCING LESION IN RIGHT POSTERIOR TEMPOROPARIETAL AND LEFT TEMPORAL.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
21	KAVITHA	25	F	683327	1 EPISODE			TONIC-CLONIC	12.89	DIFFUSE SLOWING	NORMAL	HYPOGLYCEMIA
22	KANTHAMMA	38	F	684404	1 EPISODE		YES		12.89	FOCAL EPILEPTIFORM DISCHARGES	NON ENHANCING RIGHT FRONTO TEMPARO PARIETAL LESION.IMPRESSION:GLIOMA	RIGHT SIDED ICSOL?GLIOMA
23	NAGABHUSHAN	26	M	685093	5 EPISODES			TONIC-CLONIC	22	NORMAL	NORMAL	IDIOPATHIC SEIZURE DISORDER
24	RATNAMMA	23	F	652350	6 EPISODES	YES			16	NORMAL	ING ENHANCING LESION IN RIGHT SIDED FRONTO PARIETAL REGION.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
25	GURURAJ M V	30	M	688698	1 EPISODE		YES		19	NORMAL	RING ENHANCING LESION IN LEFT PARIETAL LOBE.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS WITH LEFT LOWER LOBE PNEUMONIA
26	SHANKARAPPA	47	M	689030	3 EPISODES			TONIC-CLONIC	44.21	NORMAL	CHRONIC INFARCT IN RIGHT PARIETAL REGION.IMPRESSION:CVA	DIABETICKETOACIDOSIS WITH OLD CVA
27	RAFIQ	41	M	704213	2 EPISODES			TONIC-CLONIC	36.22	GENERALISED EPILEPTIFORM DISCHARGES	DIFFUSE CEREBRAL EDEMA.IMPRESSION:RAISED INTRACRANIAL TENSION	SEPTIC ENCEPHALOPATHY
28	CHANDRA SHEKAR	32	M	559339	1 EPISODE			TONIC-CLONIC	19	NORMAL	NORMAL	CEREBRAL MALARIA
29	GULZAL	32	F	695454	8 EPISODES			TONIC-CLONIC	14.8	GENERALISED EPILEPTIFORM DISCHARGES	NORMAL	SEPTIC ENCEPHALOPATHY
30	GIDDAMMA	50	F	695806	1 EPISODE			TONIC-CLONIC	36	GENERALISED EPILEPTIFORM DISCHARGES	RIGHT FRONTAL AND LEFT PARIETAL MULTIFOCAL VASOGENIC ODEMA.IMPRESSION:RAISED INTRACRANIAL TENSION	TUBERCULAR MENINGITIS WITH CVA
31	KAVITA	28	F	695986	8 EPISODES		YES		16	GENERALISED EPILEPTIFORM DISCHARGES	RING ENHANCING LESION IN LEFT TEMPORAL REGION.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
32	RAMACHANDRAPPA	36	M	697002	7 EPISODES		YES		9.31	NORMAL	LEFT PARA SAGITTAL REGION AND LEFT FRONTAL MULTIPLE CALCIFIED LESIONS.IMPRESSION:OLD HEALED NEUROCYSTICERCOSIS	OLD HEALED MULTIPLE CALCIFIED NEUROCYSTICERCOSIS
33	NAGARAJ	35	M	697058	1 EPISODE		YES		9.86	NORMAL	RING ENHANCING LESION IN LEFT HIGH PARIETAL LOBE.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
34	AMBRISH	25	M	697352	4 EPISODES			TONIC-CLONIC	8	NORMAL	NORMAL	ALCOHOL WITHDRAWAL SEIZURES

MASTER CHART

SL. No.	NAME	AGE	SEX	IP NUMBER	SEIZURE PATTERN	FOCAL SEIZURES WITH OUT DYSCOGNITIVE FEATURES	FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES	GENERALISED SEIZURES	SERUM PROLACTIN LEVELS(ng/dl)	EEG	CT-BRAIN	DIAGNOSIS
35	JAMES PETER	18	M	698024	8 EPISODES	YES			7.8	NORMAL	RING ENHANCING LESION IN LEFT PARIETAL REGION.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
36	LAKSHMI	24	F	614940	1 EPISODE		WITH SECONDARY GENERALIZATION	TONIC-CLONIC	18.2	DIFFUSE SLOWING	TEMPARO PARIETAL FOCAL AREA OF CEREBRAL EDEMA.IMPRESSION:RAISED INTRACRANIAL TENSION	TUBERCULAR MENINGITIS
37	NANJAMMA	62	F	747144	4 EPISODES			TONIC-CLONIC	18.2	GENERALISED EPILEPTIFORM DISCHARGES	LEFT FRONTO TEMPERO PARIETAL INFARCT.IMPRESSION:CVA	CVA-RIGHT SIDED HEMIPARESIS(LEFT FRONTO TEMPERO PARIETAL INFARCT)
38	PAPAMMA	70	F		1 EPISODE			TONIC-CLONIC	12.1	NORMAL	LACUNAR INFARCT IN RIGHT BASAL GANGLIA.IMPRESSION:CVA	RIGHT BASAL GANGLIA LACUNAR INFARCT WITH ESSENTIAL HTN
39	VENKATA REDDY	60	M	699990	3 EPISODES			TONIC-CLONIC	32	GENERALISED EPILEPTIFORM DISCHARGES	NORMAL	HYPONATREMIA
40	THOMAS	21	M	705327	3 EPISODES			TONIC-CLONIC	26.72	NORMAL	NORMAL	IDIOPATHIC SEIZURE DISORDER
41	SHASHIKALA	18	F	705814	4 EPISODES			TONIC-CLONIC	16	NORMAL	NORMAL	TUBERCULAR MENINGITIS
42	RUKMINI	20	F	709799	3 EPISODES			TONIC-CLONIC	57.24	GENERALISED EPILEPTIFORM DISCHARGES	DIFFUSE CEREBRAL EDEMA.IMPRESSION:RAISED INTRACRANIAL TENSION	PARTIAL HANGING
43	SARASWATHI	35	F	761490	1 EPISODE			TONIC-CLONIC	22.91	DIFFUSE SLOWING	NORMAL	TUBERCULAR MENINGITIS
44	DEKAPPA	65	M	692368	6 EPISODES			TONIC-CLONIC	19.66	GENERALISED EPILEPTIFORM DISCHARGES	NORMAL	ALCOHOL WITHDRAWAL SEIZURES
45	SHIVAKUMARR	18	M	709947	1 EPISODE			TONIC-CLONIC	21	GENERALISED EPILEPTIFORM DISCHARGES	NORMAL	CRYPTOCOCCAL MENINGITIS
46	KAMALAMMA	50	F	711812	4 EPISODES		YES		12.9	NORMAL	RING ENHANCING LESION IN RIGHT PARIETAL LOBE.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
47	SUSHMA	18	F	713388	1EPISODES			TONIC-CLONIC	22	NORMAL	RING ENHANCING LESION IN LEFT TEMPORAL REGION.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
48	AYASHA	22	F		2 EPISODES			TONIC-CLONIC	46.31	NORMAL	NORMAL	IDIOPATHIC SEIZURE DISORDER
49	UMA DEVI SA	51	F	715094	1 EPISODE			TONIC-CLONIC	19.22	NORMAL	CORTICAL WITH HIPPOCAMPAL ATROPHY	DEMENTIA WITH HYPERTENSION
50	RAMALINGA REDDY	74	M	681544	1 EPISODE			TONIC-CLONIC	17	NORMAL	RIGHT PARIETAL LOBE GRANULOMATOUS LESION.IMPRESSION:NEUROCYSTICERCOSIS	OLD HEALED GRANULOMATOUS LESION IN RIGHT PARIETAL LOBE WITH HTN,TYPE 2 DM
51	MUNIRATHNAMMA	50	F	717643	1 EPISODE			TONIC-CLONIC	42.16	NORMAL	NORMAL	HYPERTENSIVE ENCEPHALOPATHY

MASTER CHART

SL. No.	NAME	AGE	SEX	IP NUMBER	SEIZURE PATTERN	FOCAL SEIZURES WITH OUT DYSCOGNITIVE FEATURES	FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES	GENERALISED SEIZURES	SERUM PROLACTIN LEVELS(ng/dl)	EEG	CT-BRAIN	DIAGNOSIS
52	REDDAPPA	28	M	719222	2EPISODES			TONIC-CLONIC	33.21	GENERALISED EPILEPTIFORM DISCHARGES	HYPODENSE AREA IN LEFT PARIETAL REGION WITH OEDEMA.IMPRESSION:TUBERCULOMA	TUBERCULOMA
53	NAGARAJAPPA	40	M	720564	2EPISODES	YES			26	FOCAL EPILEPTIFORM DISCHARGES	HYPODENSE AREA IN LEFT TEMPOROFRONTO PARIETAL REGION .IMPRESSION:CVA	CVA -RIGHT HEMIPLEGIA WITH CEREBRAL PALSY(RIGHT INFANTILE HEMIPLEGIA)
54	AMMU	28	F	724639	1EPISODES		YES		26	GENERALISED EPILEPTIFORM DISCHARGES	RIGHT PARIETIAL REGION WITH RING ENHANCING LESION WITH PERILESIONAL EDIMA.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
55	VENKATARAMANA	40	M	676209	5 EPISODES			TONIC-CLONIC	10	GENERALISED EPILEPTIFORM DISCHARGES	NORMAL	ASEPTIC MENINGITIS
56	MADAPPA	55	M	759529	5 EPISODES			TONIC-CLONIC	15.5	NORMAL	NORMAL	ALCOHOL WITHDRAWAL SEIZURES
57	ESHWARAMMA	35	F	668889	3 EPISODES			TONIC-CLONIC	39	GENERALISED EPILEPTIFORM DISCHARGES	DIFFUSE CEREBRAL EDEMA.IMPRESSION:RAISED INTRACRANIAL TENSION	PARTIAL HANGING
58	SHANKAR	38	M	671692	3 EPISODES			TONIC-CLONIC	11	NORMAL	NORMAL	ACUTE PYOGENIC MENINGITIS
59	VENKATA GIRIYAPPA	72	M	738169	1EPISODES			TONIC-CLONIC	39	NORMAL	NORMAL	TYPE 2 DM WITH HYPEROSMOLAR NON KETOTIC COMA
60	PRAKASH. V	45	M	740297	1 EPISODE			TONIC-CLONIC	10	NORMAL	NORMAL	ALCOHOL WITHDRAWAL SEIZURES WITH ALCOHOLIC LIVER DISEASE
61	REDDYAMMA	24	F	740888	1EPISODES			TONIC-CLONIC	25	FOCAL SLOWING	RIGHT OCCIPITIAL REGION,RING ENHANCING LESION WITH PERILESIONAL EDEMA.IMPRESSION:NEUROCYSTICERCOSIS.	NEUROCYSTICERCOSIS
62	SATISH KUMAR M S	38	F	741024	1 EPISODE			TONIC-CLONIC	29.11	NORMAL	NORMAL	IDIOPATHIC SEIZURE DISORDER
63	BASAPPA	60	M	742382	3EPISODES			TONIC-CLONIC	42.11	GENERALISED EPILEPTIFORM DISCHARGES	LEFT PARIETAL REGION CALCIFICATION PRESENT.IMPRESSION:OLD HEALED CALCIFIED LESION	SEPTIC ENCEPHALOPATHY WITH ACUTE RENAL FAILURE
64	MUNIRAJA	28	M	745243	1 EPISODE			TONIC-CLONIC	16	NORMAL	NORMAL	IDIOPATHIC SEIZURE DISORDER
65	VENKATESHAPPA	70	M	745886	1 EPISODE			TONIC-CLONIC	11	NORMAL	NORMAL	TYPE 2 DM ,HYPEROSMOLAR NON KETOTIC COMA
66	DORAI SWAMY	45	M	747114	1EPISODES			TONIC-CLONIC	62	NORMAL	LEFT FRONTAL REGION HYPODENSE LESION.IMPRESSION:CVA	CVA
67	MUNEGOWDA	22	M	749484	1 EPISODE			TONIC-CLONIC	29.43	NORMAL	NORMAL	CEREBRAL MALARIA
68	RAMAN	82	M	750166	1 EPISODE			TONIC-CLONIC	9	NORMAL	NORMAL	IDIOPATHIC EPILEPSY

MASTER CHART

SL. No.	NAME	AGE	SEX	IP NUMBER	SEIZURE PATTERN	FOCAL SEIZURES WITH OUT DYSCOGNITIVE FEATURES	FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES	GENERALISED SEIZURES	SERUM PROLACTIN LEVELS(ng/dl)	EEG	CT-BRAIN	DIAGNOSIS
69	KRISHNAPPA	65	M	750163	1 EPISODE			TONIC-CLONIC	18	NORMAL	NORMAL	HYPEROSMOLAR NON KETOTIC COMA
70	LALITHAMMA	30	F	684301	2 EPISODES			TONIC-CLONIC	19	NORMAL	NORMAL	IDIOPATHIC EPILEPSY
71	REDDIAMMA	35	F	750854	5 EPISODES			TONIC-CLONIC	38.92	GENERALISED EPILEPTIFORM DISCHARGES	RING ENHANCING LESION IN LEFT PARIETAL REGION AND RIGHT FRONTAL REGION WITH PERILESIONAL EDEMA.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
72	RANGAPPA	35	M	711482	2 EPISODES			TONIC-CLONIC	5	GENERALISED EPILEPTIFORM DISCHARGES	NORMAL	ASEPTIC MENINGITIS
73	NARAYANAPPA	80	M	765296	1 EPISODE			TONIC-CLONIC	13.4	NORMAL	LEFT SIDED OCCIPITO FRONTAL REGION INFARCT.IMPRESSION:CVA	CVA
74	SRIDEVI	18	F	691728	3 EPISODES		YES		18	GENERALISED EPILEPTIFORM DISCHARGES	TWO RING ENHANCING LESION IN RIGHT PARIETAL REGION WITH PERILESIONAL EDEMA.IMPRESSION:NEUROCYSTICERCOSIS	NEUROCYSTICERCOSIS
75	BABU	32	M	754895	5 EPISODES			TONIC-CLONIC	15	NORMAL	NORMAL	ALCOHOL WITHDRAWAL SEIZURES
76	MANJULAMMA	24	F	580238	6 EPISODES			TONIC-CLONIC	26	GENERALISED EPILEPTIFORM DISCHARGES	RING ENHANCING LESION IN LEFT FRONTAL REGION.IMPRESSION:NEUROCYSTICERCOSIS.	NEUROCYSTICERCOSIS
77	LAKSHMAMMA	70	F	652283	2 EPISODES			TONIC-CLONIC	14	NORMAL	NORMAL	UNCONTROLLED TYPE 2 DM WITH HYPEROSMOLAR NON KETOTIC COMA
78	MEENASAMMA	60	F	725266	3 EPISODES			TONIC-CLONIC	19	NORMAL	RIGHT INTERNAL CAPSULE, LACUNAR INFARCT	HYPERTENSIVE ENCEPHALOPATHY
79	RAMESH.K	28	M	758551	4 EPISODES			TONIC-CLONIC	31	FOCAL DISCHARGES	RIGHT FRONTAL REGION,RING ENHANCING LESION WITH PERILESIONAL EDEMA.IMPRESSION:NEUROCYSTICERCOSIS.	NEUROCYSTICERCOSIS
80	GIDAMMA	55	F	695806	6EPISODES			TONIC-CLONIC	16	NORMAL	NORMAL	CEREBRAL MALARIA(FALCIPARUM POSITIVE)
81	NANJAMMA	55	F	747144	2 EPISODES			TONIC-CLONIC	29	GENERALISED EPILEPTIFORM DISCHARGES	LEFT FRONTAL REGION MASS.IMPRESSION:GLIOBLASTOMA MULTIFORME	GLIOBLASTOMA MULTIFORME
82	SHIV KUMAR	18	M	709947	1EPISODES			TONIC-CLONIC	19	GENERALISED EPILEPTIFORM DISCHARGES	NORMAL	CRYPTOCOCCAL MENINGITIS