A STUDY ON CLINICAL PROFILE OF SEIZURES - CURRENT CLINICAL SCENARIO

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DECLARATION

I hereby declare that the dissertation entitled, "**A STUDY ON CLINICAL PROFILE OF SEIZURES – CURRENT CLINICAL SCENARIO"** was done by me at Stanley medical college hospital during the year October2009- October 2010, under the guidance and supervision of Prof. G. ELANGOVAN, M.D., Chief, UNIT IV , Department of Internal Medicine.

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical university towards the partial fulfillment of requirement for the award of M.D.DEGREE Branch-1 in General Medicine.

Place : Chennai

Date : **Dr. Namitha Narayanan**

CERTIFICATE

This is to certify that this dissertation entitled, **"A STUDY ON CLINICAL PROFILE OF SEIZURES – CURRENT CLINICAL SCENARIO"** submitted by Dr. NAMITHA NARAYANAN to The Tamilnadu Dr. M.G.R Medical University, Chennai is in partial fulfillment of the requirement of the award of M.D DEGREE BRANCH-1 (GENERAL MEDICINE) and is a bonafide research work carried out by her under direct supervision and guidance. The Period of study was from OCTOBER 2009 to OCTOBER 2010.

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INTRODUCTION & REVIEW OF LITERATURE

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DEFINITION

A seizure (meaning "to take possession of") is a paroxysmal event due to abnormal, excessive, hyper synchronous discharges from an aggregate of central nervous system neurons. Depending on the distribution of discharges, this abnormal CNS activity's manifestations can range from dramatic convulsive activity to experiential phenomena unidentified by the observer[1]. A seizure needs to be distinguished from epilepsy. Epilepsy is described as the condition in which the patient has recurrent seizures due to a chronic, underlying process. This definition implies that a person with a single seizures, or recurrent seizure due to correctable or avoidable circumstances, does not necessarily have epilepsy. Epilepsy can be defined as two or more unprovoked seizure[1].

TYPES

Seizures are separable as convulsive and non convulsive, depending on the prominence of motor features. They are also distinguished as *focal* or *non focal*. Seizures with an immediate or proximal cause, such as an acute metabolic disturbance, infection, or head trauma, are *symptomatic* or *provoked*. In other instances, seizures that may result from past brain injury can be described as *remote symptomatic*.

In large number of people, a cause is not identifiable. These seizures are classified to be either *idiopathic* or *cryptogenic*. The term idiopathic refers to disease from an unknown cause and is presumed to have a genetic basis. The term cryptogenic implies a symptomatic cause unidentifiable with current medical technology[2].

INCIDENCE AND PREVALENCE

Studies have estimated that 1.5% to 5% of persons in any population will have a seizure at some time of their lives. Incidence of epilepsy is 0.3- 0.5% in different populations throughout the world and prevalence has been estimated to at 5-10 persons per 1000[1].

In developed countries, the incidence rates range from 40-70 per 100000. But in developing countries, the rates may be as high as 100-190 per 100000[2]. Partial seizures, with or without secondary generalization, are the most common seizures, followed by generalised tonic-clonic seizures. Factors that contributes to higher prevalence of epilepsy in the developing countries include limited access to health care which compounds the problems of birth injury and head trauma. Poor sanitation leads to high incidence of CNS infection which causes seizures. Poverty and illiteracy increases the risk of social diseases like alcoholism and substance abuse which can contribute to development of seizures. Further influencing the treatment of seizure is combination of local social perceptions, government policies and anti-epileptic drug availability (Burneo et al., 2005[3]; de Bittencourt et al., 1996[4]; Preux and Dreut- Cabanac, 2005[5]).

The term "incidence-prevalence gap" refers to higher incidence of epilepsy in developing countries than in developed countries, whereas prevalence values are similar throughout the world (Bharucha NE et al, 2003[6]). Possible explanations are differences in methodology, namely, inclusion of acute symptomatic seizures as incidence in developing countries: higher mortality rates in developing countries: the higher rates of remission, which would imply more benign prognosis. There are no answers yet to these speculations, nor will be until, there are well conducted incidence studies and population based outcome studies from developing countries. Studies have estimated that 1.5% to 5% of persons in any population will have a seizure at some

time of their lives. Incidence of epilepsy is 0.3- 0.5% in different populations throughout the world and prevalence has been estimated to at 5-10 persons per 1000.

The incidence of epilepsy is bimodal in developed countries(Forsgren et al., 2005)[7]. Rates are high in the first decade, particularly before the age of one year, and decline during childhood, reaching a minimum between 20 and 30 years of age. A secondary rise in the incidence occurs after 60 years, increasing dramatically with age (Brodie and Kwan,2005[8]).

This bimodal distribution is not as evident in developing countries. The age-specific incidence remains high throughout adulthood, largely related to symptomatic seizures occurring as a result of infection and trauma (de Bittencourt et.al., 1996[4]).

Men are 1.0 to 2.4 times more likely to have seizures than women[2].

CLASSIFICATION OF SEIZURES:

International league against Epilepsy (ILAE) Classification of epileptic seizures[2]:

1. Partial (focal, local) seizures

A. Simple partial seizures (consciousness not impaired)

- Motor – Frontal lobe origin (tonic, clonic, tonic-clonic; jacksonian; benign childhood epilepsy; epilepsia partialis continua)
- Somato sensory or special sensory (visual, auditory, olfactory, gustatory, vertiginous)
- Autonomic
- Pure psychic

B. Complex partial seizures (with impairment of consciousness)

- ❖ Beginning as simple partial and progressing to loss of consciousness
- ❖ With impairment of consciousness at onset

C. Partial seizures evolving to secondarily generalized seizures

- ❖ Simple partial seizures evolving to generalized seizures
- ❖ Complex partial seizures evolving to generalized seizures
- ❖ Simple partial seizures evolving to complex partial and then to generalized seizures

2. Generalised seizures (convulsive or non convulsive)

A. Absence (petit mal) seizures

(1). With loss of consciousness only

(2). Complex – with brief tonic, clonic, or automatic movements

B. Tonic, clonic, or tonic-clonic (grand mal)

C. Lennox-Gastaut syndrome

D. Juvenile myoclonic epilepsy

E. Infantile spasms (West syndrome)

F. Atonic (astatic) seizures

3.Special epileptic symdromes

- ❖ Myoclonus and myoclonic seizures
- ❖ Reflex epilepsy

- Acquired aphasia with convulsive disorder
- Febrile and other seizures of infancy and childhood
- Hysterical seizures

PARTIAL SEIZURES

These occurs within the discrete regions of the brain. Simple partial seizures and complex partial seizures are distinguished solely on the basis of consciousness. Consciousness is usually assessed by the ability to respond to external stimuli.

This ability is intact in SPS but impaired in CPS.

SIMPLE PARTIAL SEIZURES (SPS)

This is further divided according to symptoms as motor, sensory, autonomic and psychic. Motor sensation can vary and include motor signs with or without march, versive movement, posturing and phonatory symptoms. SPS with sensory symptoms includes all five senses plus a vertiginous sensation. SPS with automatic symptoms includes the common rising epigastric sensation typically seen in mesial temporal lobe epilepsy (MTLE) and less frequent symptoms such as vasomotor phenomena or mydriasis. SPS with psychic symptoms are characterized by various experiences involving memory (eg., déjà-vu, jamaisvu), affect (fear, pleasure), or other complex, psychic phenomenon such as illusions.

COMPLEX PARTIAL SEIZURES (CPS)

CPS includes complex symptomatology like automatism and impairment of consciousness. The seizures consists of involuntary but coordinated motor activity that is purposeless or repetitive. Common automatisms include lip smacking, chewing, fidgeting and walking. CPS can begin as SPS and seizures with impaired consciousness. Electroencephalographically, partial seizures are characterized by focal epileptic form discharges interictally (spikes or sharp waves) and ictally.

GENERALISED SEIZURES

ABSENCE SEIZURES

These are characterized by sudden brief lapses of consciousness without loss of postural control. Absence seizures constitute 15- 20% of childhood seizures. In atypical absence, there is less abrupt onset, and termination with longer duration.EEG typically shows generalised symmetric 3 Hz spike and wave discharge that begins and ends suddenly on a normal EEG background.

MYOCLONIC SEIZURES

It is a sudden and brief muscle contraction that may involve one part of the body or the entire body.EEG shows bilateral synchronous spike and wave discharges.

CLONIC SEIZURES

These are repetitive rhythmic clonic movements that are bilateral and symmetric and associated with fast activity or spike-wave complex on EEG, which evolve over time typically decreasing in frequency while increasing in amplitude.

TONIC SEIZURES

These are characterized by stiffening of the musculature mostly axial, but also appendicular associated with low voltage paroxysmal fast activity.

GENERALISED TONIC CLONIC SEIZURES (GTCS)

This is the sequential combination of previous two types in which the tonic phase is gradually interrupted by quiescence evolving to the clonic phase with rhythmic spikes and spike-wave complexes decreasing in frequency on EEG.

ATONIC SEIZURES

Characterized by sudden loss of muscle tone lasting for 1-2 seconds. Consciousness is briefly impaired but usually there is no post- ictal confusion. EEG shows generalised epileptiform discharges (spike, spike-wave complexes) or an abrupt flattening on EEG.

UNCLASSIFIED SEIZURES

Not all seizure types can be classified as partial or generalised, especially true of seizures that occur in neonates and infants. The distinctive phenotypes of seizures at these early ages

likely result, in part, from differences in neuronal function and connectivity in the immature versus mature CNS.

EPILEPSY SYNDROMES

These are disorders in which epilepsy is a predominant feature, and there is sufficient evidence to suggest a common underlying mechanism. The important epilepsy syndrome includes the following

- **JUVENILE MYOCLONIC EPILEPSY(JME)**

  JME is a generalised seizure disorder of unknown cause that appears in early adolescence and is usually characterized by bilateral myoclonic jerks that may be single or repetitive. The myoclonic seizures are most frequent in the morning after awakening and can be provoked by sleep deprivation. Consciousness is preserved unless the myoclonus is especially severe.

- **LENNOX-GASTAUT SYNDROME**

  It occurs in children and is defined by the following triad.(1). multiple seizure types (2). An EEG showing slow spikes- and-wave discharges and a variety of other abnormalities; (3).impaired cognitive function in most but not all cases. The multifactorial nature of this syndrome suggests that it is a non specific response of the brain to diffuse neural injury. Many patients have poor prognosis due to the underlying CNS disease.

- **MESIAL TEMPORAL LOBE EPILEPSY (MTLE)**

  It is the most common syndrome associated with complex partial seizures, a symptomatic partial epilepsy with distinctive clinical, Electroencephalographic, and pathologic features. High-resolution MRI can detect the characteristic hippocampal sclerosis that appears to be essential in the pathophysiology of MTLE.

THE CAUSES OF SEIZURES AND EPILEPSY

Age is one of the most important factors determining both the incidence and the likely causes of the seizures or epilepsy[1]. Many causes of seizures and epilepsy results from a dynamic interplay between endogenous factors, epileptogenic factors, and precipitating factors. The cause of great majority of seizures and epilepsy in adult life will be symptomatic. But in studies of hospital and clinic-based populations, as well as in field studies, the etiology of epilepsy is identifiable in only one fourth to one third of the cases.

AGE	**CAUSES OF SEIZURES[1]**
Neonates (< 1 month)	Perinatal hypoxia and ischemia Intracranial hemorrhage and trauma Acute CNS infection Metabolic disturbances (hypoglycemia, hypocalcemia, hypomagnesemia, pyridoxine deficiency) Drug withdrawal Developmental disorders Developmental disorders and genetic factors

AGE	CAUSES OF SEIZURES
Infants and children (> 1 month and < than 12 years	Febrile seizures Genetic disorders (metabolic, degenerative, primary epilepsy syndromes) CNS infections Developmental disorders Trauma Idiopathic
Adolescents (12 – 18 years)	Trauma Genetic disorders Infection Brain tumor Illicit drug use idiopathic
Young adults (18-35 years)	Trauma Alcohol withdrawal Illicit drug use Brain tumor idiopathic
Older adults	Cerebrovascular disease Brain tumor Alcohol withdrawal Metabolic disorders (uremia, hepatic failure, electrolyte abnormalities,

	hypoglycemia Alzheimer's disease and other degenerative CNS diseases idiopathic

(1). ACUTE SYMPTOMATIC SEIZURES

These seizures, otherwise called reactive seizures/ provoked seizures/ situation related seizures[9]. These occurs at the time of systemic insult or in close temporal association with a documented brain insult. These seizures need only a short acting benzodiazepine as the treatment and do not require a long term treatment.

Acute symptomatic seizures typically occur within first week of an insult known to predispose to seizures[10] (eg, head trauma, CNS infection, cerebrovascular disease, brain tumor, metabolic causes, drug withdrawal). These seizures predispose later to unprovoked seizures, the incidence of it depend upon the initial cause and severity of the event.

(2). REMOTE SYMPTOMATIC SEIZURES

CEREBROVASCULAR DISEASE:

A chronic epileptogenic lesion at the stroke site may account for unprovoked seizures that occur more than 1 to 2 weeks after a clinical stroke. Such unprovoked seizures occur after a clinically detected stroke in 2.7 to 35% of patients . This variation in incidence may be due to difference in the methodology of the studies done. Regardless of the selection factors or length of follow-up, the risk of unprovoked seizure after stroke is at least three times (Hauser WA et al, Minnesota, 1935- 1985[11]).

Because stroke produces a focal brain injury that serves as a substrate focus for seizure development, it has been hypothesized that only unprovoked partial seizures should occur after stroke. Generalized seizures are, however, not uncommon after stroke, although some investigators include secondary generalized seizures in this category. Researchers who distinguish primary from secondary generalized seizures find that generalized seizures account for 4% to 69% of all unprovoked seizures after stroke. Undetected onset of partial seizures may account for this variability. Nonetheless, true primary generalized seizures do occur after stroke, perhaps as a result of persistent global alterations in neurotransmitter function after stroke or of factors that alter cerebral auto regulation in people with risk factors for stroke.

Cleary P et al, Lancet[12] 2004, in their studies state that the cumulative risk for stroke in patients with seizures was 10.0%, compared to 4.4% in patients without seizures. This increased prevalence of seizures in patients with stroke predicted that risk factors for stroke may also increase the risk factors for seizures. Studies state that hypertension, left ventricular hypertrophy independently increase the risk of unprovoked seizures (Herdorffer DC[13] et al, 1996).

Cortical venous stroke with underlying ischemia and infarction is highly epileptogenic. The same is true for hypertensive encephalopathy and Thrombotic Thrombocytopenic Purpura (TTP), which has strong tendency to cause non-convulsive status epilepticus. The rupture of saccular aneurysm may be marked by one or two generalized convulsions. Subcortical cerebral haemorrhages occasionally become a source of recurrent focal seizures.

BRAIN TUMORS:

Although neoplasms of the brain account for only 1% of cases of epilepsy, seizures occur in approximately 50% of children with supratentorial tumors and seizures develop in

approximately 35% to 40% of adults with brain tumors (LeBlanc FE[14] et al, 1974). The rate is much lower for tumors of the infratentorial or pituitary region, and consequently even higher for supratentorial lesions. Seizures occur especially commonly in association with oligodendrogliomas. Seizures are also commonly encountered in patients with meningiomas. In contrast, the incidence of seizures in patients with cerebral metastasis is much lower; most reports suggest an incidence of 20% at the time of presentation (Gamache FW[15], et al, 1979). Imaging increases the probability of finding an early malignancy. So it is recommended that every patient who presents with seizures for the first time undergoes brain imaging. EEG is also useful in assessing these patients. Findings correlate with tumor location, and in approximately 40% of the patients, the EEG abnormalities are lateralized to the side of the tumor. EEG is relatively inexpensive and helps to localize the epileptogenic foci by a method different from neuro imaging. Patients with brain tumors who undergo surgeries have increased risk of unprovoked seizures.12% to 16.3% of newly diagnosed epilepsy have brain tumors, indeed, seizures are often the first sign of brain tumor (Ludhorf K et al, 1986).

NEUROCYSTICERCOSIS:

Neurocysticercosis is caused by the larvae of tapeworm *Taenia solium* in the nervous system, a disease suffered by millions of people living in the developing countries. In these areas, the disease account for up to 12% of all admissions to neurological hospitals and is the leading cause of epilepsy in adults. More than 50,000 new cases of NCC- related deaths occur annually, and of the many more patients who suffer related neurological sequelae, most are affected at productive age. This makes NCC a large public health problem in the developing countries.

Stages of inflammation through which cysticerci pass through are colloidal, granular and calcified. Inflammatory reaction around cysticerci induce pathological changes in the CNS serving as a substrate for further development of seizures. It causes granular ependimitis which leads to obstructive hydrocephalus. Seizures, focal deficits, cognitive decline and increased intra cranial pressure are common manifestations of NCC. Seizures occur in about 70% of cases (Del Brutto OH et al, 1992 , Rajshekhar V[16&17] et al, 2004). NCC is often diagnosed on the basis of information provided by neuro imaging studies and serology. Imaging studies give information about the number and location of lesions as well as on the stage of evolution of cysticerci.

CNS INFECTIONS:

CNS infection increase the seizure risk by 11-fold. A 16-fold risk was associated with viral meningitis, a 4-fold risk with bacterial meningitis, a 2-fold risk with aseptic meningitis. Most of the unprovoked seizures occurred within the first 5 years of infection (Annegers JF[18] et al, 1988).

PYOGENIC MENINGITIS:

Pyogenic bacteria induce formation of a purulent exudate within the subarachnoid space related to migration of neutrophils and other immune cells.

Such exudates, together with direct effects of bacterial toxins may cause seizure by; (1) occlusion of small pial arteries, (2) venous thrombosis, (3) diffuse brain swelling, (4) toxic effects of bacteria in subpial space, (5) acute metabolic changes.

Acute symptomatic seizures occur in 40% of patients, more often generalized tonic clonic seizures. 2% to 7% of the patients developed chronic epilepsy, mostly in those with permanent neurological deficits (Pomeroy S[19]L et al, 1990).

VIRAL ENCEPHALITIS:

HSV-1 is the most common cause of sporadic viral meningitis. Viruses cause diffuse swelling, vascular congestion, demyelination, inflammatory infiltrates, microglial proliferation, diffuse necrosis of cerebral cortex and basal ganglia. Seizures are mostly related to development of cerebral infarcts. Seizures may be generalized or partial, more often recurrent and persistent after the acute disease (Knnedy PGE[20] et al, 2004).

CEREBRAL MALARIA:

Malaria is an endemic in our country. Acute cerebral malarial encephalopathy still has 22% mortality inspite of improved treatment. Patients usually presents with unarousable coma, evidence of P.falciparum infection and no other identifiable causes of coma. There is extravasation of erythrocytes resulting from endothelial damage causing cytokine release . There is also plugging of capillaries by parasitized erythrocytes. This causes brain damage as a result of obstruction to the cerebral vasculature and leads to ischemic hypoxia. Seizures occur in about 70% of cases, and are most often generalized tonic clonic, although some patients may present with partial seizures (RomanGC[21] et al, 1992).

CNS TUBERCULOSIS:

Tuberculous meningitis is a sub acute disease characterized by fever, malaise, behavioural changes, headache, seizures, focal neurological signs, and stupor or coma. Seizures occur in approximately 20% of the patients, are more common in children than in adults, and may represent predictors of poor outcome (Hosoglu S[22] et al, 2002). Intracranial tuberculomas presents as mass lesions with increased intracranial pressure, focal signs and seizures (Garcia-Monco JC[23] et al, 2005).

HEAD INJURY/ POST-SURGICAL:

Head injury increases the risk of unprovoked seizures, with the greatest risk occurring in survivors with severe injury. Severe injuries with more than 24 hours of unconsciousness or post traumatic amnesia, brain contusion or intracerebral hematoma increased the risk of seizures by 29-fold(RoccaWA[24] et al, 1987).

Analogous to seizures after penetrating injuries, unprovoked seizures may be a consequence of neurosurgical procedures to the head. Studies for unprovoked seizures after neurosurgical procedures are complicated by the nature of the underlying disorder, the presence of seizures before surgery, and the select nature of the population studied. Foy PM[25] et al,1981 in their study on natural history of post-operative seizures on 877 consecutive neurosurgical patients, unprovoked seizures developed in 175 of patients within 5 years.

DEMENTIA:

The underlying pathology of Alzheimers may be associated with increased susceptibility of seizures, with increase by 10-folds (Hauser WA[26] et al, 1986). All the seizures are usually generalized tonic clonic seizures (Romanelli MF[27] et al, 1990).

Hesdorfer[28] et al, 1996 evaluated the risk for seizures associated with dementia and found that dementia increased the risk of seizures by 8-fold.

MECHANISMS OF SEIZURE INITIATION AND PROPAGATION

The central hypothesis of the paper by Pinto[29] and coworkers is that epileptiform activity, as observed in a disinhibited slice, involves three stages, each with independent mechanisms: initiation, propagation, and termination. Electrophysiological analyses of cellular models of interictal spikes and epileptic seizures have long invoked these three particular stages in studies of epileptiform activity.

Typical disinhibited slice preparation involves reducing or blocking $GABA_A$receptors or the associated chloride conductance with a pharmacological treatment. The primary mechanisms assessed in these studies were synaptic excitation (i.e., glutamatergic non-*N*-methyl-D-aspartate [non-NMDA] receptors), synaptic inhibition (i.e., $GABA_A$ receptors), and intrinsic neuronal properties (specifically, depolarization block). The non-NMDA and $GABA_A$-receptor mechanisms were studied in experiments in which the concentrations of 6,7-dinitroquinoxaline-2,3-dione (DNQX), an α-amino-3-hydroxy-5-methyl-4-isoxazole

proprionic acid (AMPA)/kainate-receptor antagonist, and picrotoxin were varied systematically, as picrotoxin-induced epileptiform activity was recorded with the linear array of electrodes.

The study of Pinto and coworkers reports parametric differences between measures of burst initiation and propagation, including differences in the effects of various doses of the pharmacological antagonists; however, their results show that the initiation and propagation of synchronized bursts derive from the disinhibited transmission of glutamatergic synapses. The slow process underlying initiation appears to be sequential transmission through recurrent excitatory circuits in layer 5, while fast propagation over longer distances is presumably the result of conventional axonal conduction among pyramidal cells in all layers; thus, the mechanisms of initiation and propagation have both similarities and differences.

Pinto et al. found that although synaptic excitation and inhibition appeared to modulate termination, strong depolarizations (i.e., depolarization block) characterized termination. Termination would depend on different mechanisms than those responsible for initiation or propagation. According to the mechanism of action of the pharmacological/ionic treatments, different mechanisms of initiation, propagation, and termination are likely to be involved in the three different stages of epileptiform activity.

"Epileptiform" is a widely used term, with a long history in basic epilepsy research. The standard approach to investigations concerning epileptiform activity has been to use a pharmacological and ionic treatment protocol in an in vitro or in vivo preparation to induce

"hyperexcitability" or "hyperactivity," usually defined as increased electrical activity above what is considered normal. In most studies of this nature, the hyperexcitable or hyperactive events represent bursts of action potentials and then typically are called epileptiform events. Most of these events have durations in the order of tens of milliseconds, but some can persist for tens of seconds. Thus, these short-lasting epileptiform events during an EEG recording would be considered experimental models of the interictal spike rather than actual epileptic seizures, which are characteristically longer than several seconds and are often 2 or more minutes.

Thus, another issue is whether brief bursts of epileptiform activity in a disinhibited slice are actually relevant to the generation of spontaneous recurrent epileptic seizures in a patient or an animal model of chronic epilepsy. The events in the study of Pinto and coworkers were usually in the order of tens of milliseconds, although some lasted up to about 500 milliseconds; therefore, the epileptiform activity under investigation could be considered to be more analogous to an interictal than an ictal event. Thus, one would interpret these studies in the context of how spikes rather epileptic seizures occur in a cortical EEG.

The role of $GABA_A$ inhibition in the initiation, propagation, and termination of chronic epileptic seizures (or interictal spikes) may not be the same as for an epileptiform burst in a disinhibited slice.

APPROACH TO THE PATIENT

When a patient presents shortly after a seizure, the first priorities are attention to vital signs, respiratory and cardiovascular support, and treatment of seizures if they resume. Life-threatening conditions such as CNS infection, metabolic derangements, or drug toxicity must be recognized and managed appropriately.

When the patient is not acutely ill, the evaluation will initially focus on whether there is a history of earlier seizures. If this is the first seizure, then the emphasis will be to

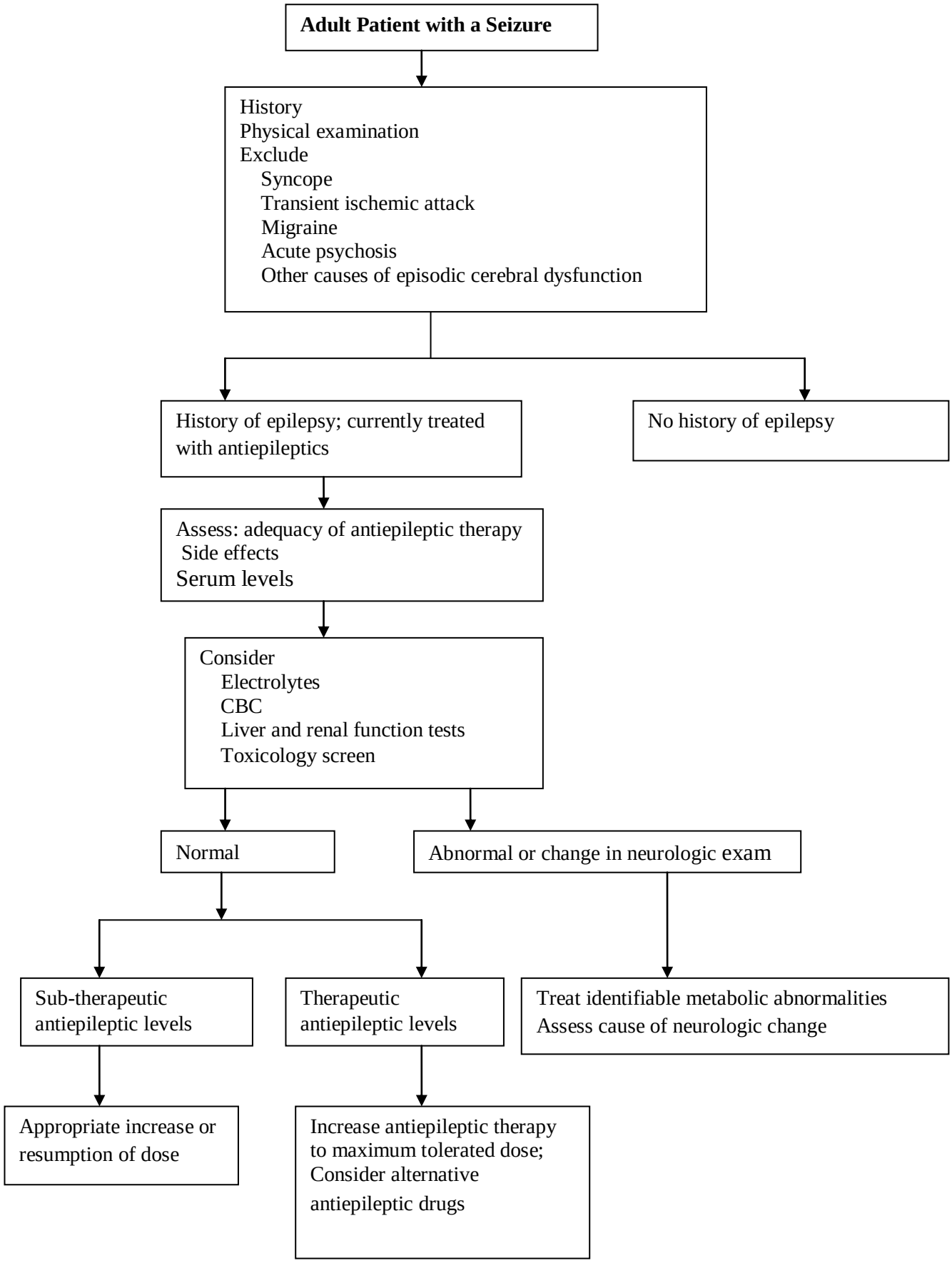
(1) establish whether the reported episode was a seizure rather than another paroxysmal event,
(2) determine the cause of the seizure by identifying the risk factors and precipitating events, and

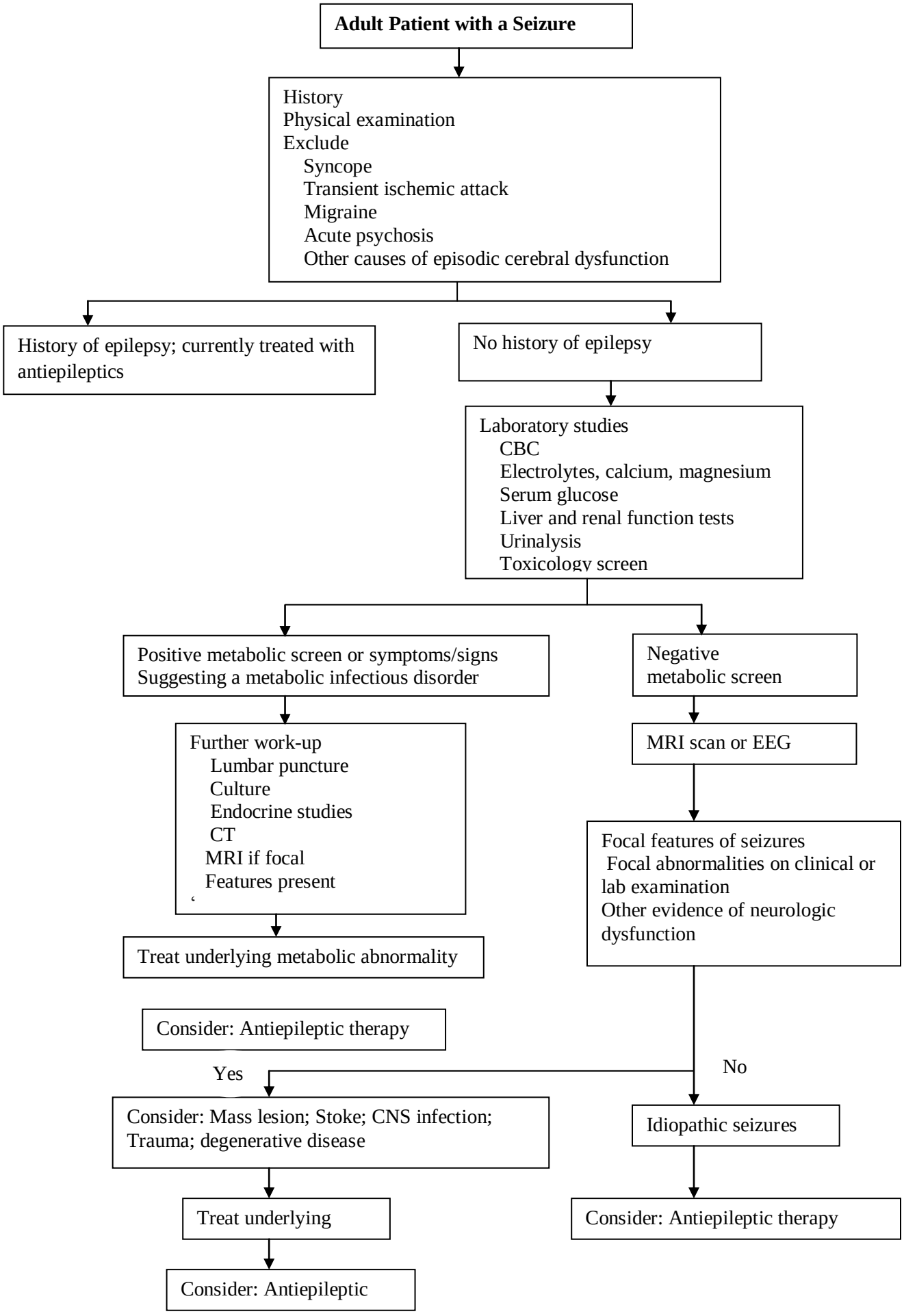
(3) decide whether anticonvulsant therapy is required in addition to the treatment of the underlying illness.

In the patients with prior seizures or a known history of epilepsy, the evaluation is directed toward

(1) identification of the underlying cause and precipitating factors, and

(2) determination of the adequacy of the patient's current therapy. A systematic approach towards the evaluation of the adult patients with a seizure is outlined below.


Adult Patient with a Seizure
History
Physical examination
Exclude
Syncope
Transient ischemic attack
Migraine
Acute psychosis
Other causes of episodic cerebral dysfunction
History of epilepsy; currently treated with antiepileptics
No history of epilepsy
Assess: adequacy of antiepileptic therapy
Side effects
Serum levels
Consider
Electrolytes
CBC
Liver and renal function tests
Toxicology screen
Normal
Abnormal or change in neurologic exam
Sub-therapeutic antiepileptic levels
Therapeutic antiepileptic levels
Treat identifiable metabolic abnormalities
Assess cause of neurologic change
Appropriate increase or resumption of dose
Increase antiepileptic therapy to maximum tolerated dose; Consider alternative antiepileptic drugs


Adult Patient with a Seizure
History
Physical examination
Exclude
Syncope
Transient ischemic attack
Migraine
Acute psychosis
Other causes of episodic cerebral dysfunction
History of epilepsy; currently treated with antiepileptics
No history of epilepsy
Laboratory studies
CBC
Electrolytes, calcium, magnesium
Serum glucose
Liver and renal function tests
Urinalysis
Toxicology screen
Positive metabolic screen or symptoms/signs Suggesting a metabolic infectious disorder
Negative metabolic screen
Further work-up
Lumbar puncture
Culture
Endocrine studies
CT
MRI if focal
Features present
MRI scan or EEG
Treat underlying metabolic abnormality
Focal features of seizures
Focal abnormalities on clinical or lab examination
Other evidence of neurologic dysfunction
Consider: Antiepileptic therapy
Yes
No
Consider: Mass lesion; Stoke; CNS infection; Trauma; degenerative disease
Idiopathic seizures
Treat underlying
Consider: Antiepileptic therapy
Consider: Antiepileptic

ELECTROPHYSIOLOGIC STUDIES

EEG is critical in the diagnosis of epileptic seizures, particularly of complex partial or absence status epilepticus, when EEG may be the most definitive indication of a seizure[30]. EEG may detect epileptiform abnormalities (spikes, sharp waves, spike and slow-wave complexes, polyspike and slow-wave complexes). Epileptiform abnormalities may be bilateral and generalized in patients with generalized seizures and may be localized in patients with partial seizures. EEG findings may include the following:

- Epileptiform abnormalities in temporal lobe foci between seizures (interictal) in complex partial seizures originating in the temporal lobe
- Interictal symmetric bursts of 4- to 7-Hz epileptiform activity in primarily generalized tonic-clonic seizures
- Focal epileptiform discharges in secondarily generalized seizures
- Spikes and slow-wave discharges at a rate of 3/sec in typical absence seizures
- Slow spike and wave discharges usually at a rate of < 2.5/sec in atypical absence seizures
- Bilateral polyspike and wave abnormality at a rate of 4- to 6-Hz in juvenile myoclonic epilepsy

However, normal EEG cannot exclude the diagnosis of epileptic seizures, which must be made clinically. EEG is less likely to detect abnormalities if seizures are infrequent. The initial EEG may detect an epileptiform abnormality in only 30 to 55% of patients with a known epileptic seizure disorder. Serial EEG may detect epileptiform abnormalities in up to 80 to 90% of such patients. Inpatient combined video-EEG monitoring, usually for 2 to 7 days, records

EEG activity and clinical behavior simultaneously. It is the most sensitive EEG testing available and is thus useful in differentiating epileptic from nonepileptic seizures.

Magnetoencephalography[42] (MEG) provides another way of looking non invasively at cortical activity. It measures the small magnetic fields that are generated by this activity. Epileptiform activity can be analyzed, and its source in the brain can be estimated using mathematical technique. These source estimates can then be plotted on an anatomic image of the brain, such as an MRI, to generate a magnetic source image (MSI). MSI can be useful to localize potential seizure foci.

BRAIN IMAGING

Neuroimaging[44] techniques are essential in the understanding, diagnosis and management of seizures. The rationale of neuroimaging[45&46] is mainly two fold:

(1). To identify underlying pathologies such as tumors, granulomas, vascular malformations, traumatic lesions or strokes that merit specific treatment.

(2). To facilitate the syndromic and aetiological diagnosis and subsequent accurate prognosis of the condition.

CT SCAN

CT scan[44] has a significant role in the acute seizure scenario and peri-operative period. The ability of the CT scan to detect trauma, hemorrhage, hydrocephalus and tumors can influence the mode of treatment. However the low sensitivity of CT especially with regards to

temporal fossa lesions, has led the International league against Epilepsy to suggest CT scan as the modality of choice only when MRI is not available or cannot be done due to some reason.

MRI

MRI is the investigation of choice in patients with epilepsy, especially if focal features are present on examination or EEG shows epileptic discharges or if seizures persists despite a previously normal CT scan. It has made a significant contribution in identifying Mesial temporal lobe sclerosis[47] in patients with intractable seizures, thus proves pivotal in the subsequent surgical treatment. It is also useful in the identification of the malformations of the cortical development (MCD). The diagnosis of malformations like Lissencephaly, Schizencephaly and Patchygyria and other lesions like Focal cortical dysplasia (FCD) have been rendered much easier by this technique.

The finding of a causative lesion on MRI in a new onset seizure makes treatment imperative. The MRI does have shortcomings[48] like high cost and its failure in identifying the electrical disorders of the neurons that cause epilepsy.

SINGLE PHOTON EMISSION CT (SPECT)

SPECT is a nuclear imaging technique[49] based on increased ictal region perfusion and decreased interictal region perfusion. It is useful in supporting the localization of epilepsy when performed in a carefully monitored ictal or early postictal examination. It is also used in presurgical evaluation and to guide the placement of intracranial electrodes in the context of other imaging data being equivocal. The evolution of Substraction Ictal SPECT has greatly augmented its accuracy.

POSITRON EMISSION TOMOGRAPHY (PET)

FDG-PET show hypometabolic areas in the epileptogenic region during interictal phase. They are useful in the lateralization of temporal lobe epilepsy, in whom there is discordance of data among MRI, EEG and other imaging modalities. Technique like ^{11}C Flumazenil(FMZ) PET may aid in precise localization in patients of medial temporal sclerosis with a negative MRI[50]. PET has no role in influencing therapeutic decisions.

MAGNETIC RESONANCE SPECTROSCOPY (MRS)

MRS is an excellent noninvasive biochemical tool for measuring brain metabolites[51], used primarily in temporal lobe epilepsy. The clinical utility of MRS has been compromised by the present limitation of spatial coverage limits. Recent studies have suggested that MRS evidence of disease progression might impact disease therapy in future.

FUNCTIONAL MRI (F MRI)

It utilizes very rapid scanning techniques that demonstrate alteration in blood oxygen[52], and is used in presurgical delineation of functional areas such as language, motor and visual cortical areas. The role of it in the medical management of epilepsy is obscure.

DIFFERENTIAL DIAGNOSIS OF SEIZURES

In most cases seizures can be distinguished from other conditions by meticulous attention to the history and relevant laboratory studies. Additional studies, such as video-EEG monitoring,

sleep studies, tilt-table analysis, or cardiac electrophysiology, maybe required to reach a correct diagnosis[31&32].

Syncope ▪ Vasovagal syncope ▪ Cardiac arrhythmia ▪ Valvular heart disease ▪ Cardiac failure ▪ Orthostatic hypotension	Metabolic disturbances ▪ Alcoholic blackouts ▪ Delirium tremens ▪ Hypoglycemia ▪ Hypoxia ▪ Psychoactive drugs
Psychological disorders ▪ Psychogenic seizure ▪ Hyperventilation ▪ Panic attack	Movement disorders ▪ Tics ▪ Nonepileptic myoclonus ▪ Paroxysmal choreoathetosis
Migraine ▪ Confusional migraine ▪ Basilar migraine	Sleep disorders ▪ Narcolepsy /Cataplexy ▪ Benign sleep myoclonus

Transient ischemic attack (TIA) ▪ Basilar artery TIA	Special considerations in children like breath holding spells, benign paroxysmal vertigo, apnea, night terror and sleep walking

The differential diagnosis of transient neurologic dysfunction is broad, and the elderly population that is at highest risk of stroke and transient ischemic attack (TIA) is also at risk of many conditions that can mimic seizures.

SYNCOPE

Syncope[33] may not be benign .Causes include:

- hypovolemia (e.g., blood loss, diuretics)
- decreased arterial or venous tone (e.g., vasodilators, autonomic dysfunction)
- limited cardiac output (e.g., aortic stenosis, arrhythmias)
- inappropriate baroreceptor reflexes (e.g., emotional situations, Valsalva maneuver)

Upright posture at onset and a typical warning of lightheadedness, nausea, warmth, and fading vision and hearing are common but not universal, and stroke patients may have difficulty reporting these sensations. Cardiac arrhythmias, some potentially fatal, may lead to sudden loss of consciousness, even in the supine position. In these patients, palpitations may be noted if onset is not sudden or at other times.

A few myoclonic jerks commonly accompany syncope, and tonic stiffening (as well as more complex movements) may also occur, especially if the head is kept upright. The pathophysiology

of such convulsive syncope is release of brain stem activity from cortical influence rather than an electrocortical seizure.

In addition, syncope can rarely occur as a vertebrobasilar TIA, especially when flow through one or both carotids is severely compromised.

MIGRAINE

The episodic headache and other symptoms of migraine sometimes are preceded by an aura, 5 to 60 minutes of cortical or brain stem dysfunction. Migraine auras are distinguished from seizures by their more gradual, often visual, warning and longer duration. Associated symptoms include nausea or vomiting, photophobia, and phonophobia. Headache usually, but not always, follows. "Migraine equivalents[34]" without headache are more common in the elderly and are occasional causes of TIA-like symptoms or of actual TIAs. Loss of consciousness is rare but may occur with so-called basilar migraine.

It must be recognized that migraine and epilepsy can coexist, that headaches often follow epileptic seizures, and that a migraine attack can, rarely, precipitate a seizure.

TRANSIENT ISCHEMIC ATTACKS

TIAs themselves can be confused with seizures[35], although they have characteristic symptoms and (if prolonged enough to persist to the time of evaluation) signs consistent with known vascular territories. They typically evolve over minutes and last minutes to hours.Jackson[41] was first to point out that seizures generally manifest "positive" symptoms, such as stiffening or shaking in the motor system or hallucinations in the special sensory modalities,

whereas ischemic symptoms are usually “negative” (e.g., weakness, sensory loss). Exceptions to this rule include ischemic paresthesias, rare motor inhibitory seizures, and “limb-shaking[36]” TIAs.

"Limb-shaking TIAs" are rare manifestations of severe carotid stenosis. They can be distinguished from motor seizures mainly by

- their consistently postural character, usually occurring promptly on standing
- their involvement of arm, leg, or both, sparing facial muscles and cognition

On the other hand, rare seizure types, such as ictal amaurosis (total or hemianopic, not monocular) or aphasic status epilepticus, require EEG to be distinguished from TIAs.

Patients with cerebral amyloid angiopathy have been noted to have transient events for which the underlying pathophysiology has not been established; no evidence of microscopic bleeding, transient ischemia, or epilepsy has been discovered. The duration is more similar to that of TIAs than of the other potential etiologies.

MOVEMENT DISORDERS

Movement disorders can usually be readily distinguished from seizures because they are typically long-lasting and associated with preserved consciousness. Although usually bilateral, they may be unilateral after infarction, particularly infarction of the basal ganglia, thalamus, or subthalamus.

In patients with depressed mental status, toxic or metabolic processes may at times produce movement disorders, such as extrapyramidal reactions to neuroleptics or multifocal

myoclonus in uremia. Although the multifocality is not typical of seizures, and the movements are not time-locked to epileptiform discharges on EEG, such discharges are often present and imply "cortical irritability" that may later be manifest as clear-cut seizures.

Asterixis, an abrupt, repetitive loss of muscle tone during maintenance of certain postures, often occurs in patients with depressed mental status due to hepatic or other encephalopathies. After cerebral or brain stem stroke, it can occur unilaterally, contralateral to the lesion. Its positional nature usually distinguishes it from motor seizures, although rare cases of epileptic asterixis have been reported.

Antiepileptic drugs, especially at toxic levels, also can produce involuntary movements, such as dystonia with phenytoin or tremor with valproate.

SLEEP DISORDERS

Sleep disorders may result in microsleeps or more prolonged sleep attacks due to any cause of hypersomnolence. The most common cause is disrupted sleep from obstructive sleep apnea, a condition which (like stroke) is common among patients with hypertension, atherosclerosis, and obesity. Furthermore, many thrombotic strokes, in particular, occur during sleep and are characterized by patients' awakening with a new deficit.The second most common medical reason for sleep deprivation leading to sleep attacks is the movement disorder termed periodic limb movements[37] in sleep. These movements usually involve one or both lower limbs, with dorsiflexion of the ankle and flexion of the knee and hip, and are sustained for 1 to 2 seconds and repeated approximately every one-half minute. This condition is associated with

restless legs ssundrome, a need to walk around or otherwise move the legs, often in response to a crawling sensation felt when lying in bed or otherwise at rest.

Narcolepsy is a more dramatic but much less common cause of hypersomnolence, usually associated with symptoms of hypnagogic or hypnopompic hallucinations, sleep paralysis, and especially cataplexy. Onset is rare after early adulthood, although symptomatic cases related to brain stem trauma, demyelination, and, rarely, infarction have been reported. Although microsleeps may occur without warning, more prolonged sleep attacks are usually preceded by a subjective feeling of sleepiness. Unlike in complex partial seizures, the eyes are usually closed, and the patient may be awakened with stimulation.

Parasomnias can be difficult to distinguish from nocturnal seizures. The classic parasomnias of slow-wave sleep, sleepwalking, and night terrors are conditions of childhood, although the former sometimes persists into adulthood. They are not associated with stroke. In the population at risk for stroke, nocturnal wandering is more likely to occur after a complex partial seizure, and patients usually return to normal awareness rapidly, if stimulated.

A parasomnia of rapid eye movement (REM) sleep, REM behavior disorder, by contrast, typically begins late in life and may be associated with extrapyramidal syndromes such as Parkinson's disease. Cases in patients with stroke may be coincidental, given the typical ages for both disorders. These attacks consist of partial arousals from REM with a loss of the usual muscle atonia, resulting in "acting out" of dreams, often in a violent manner that may reflect defensive behavior prompted by a frightening dream. The timing of the spells later in the night, when REM periods are longer, can be a useful clue. Polysomnography with additional EEG electrodes may be necessary to distinguish this disorder from nocturnal partial seizures.

TOXIC METABOLIC DISTURBANCES

Altered behavior due to toxic-metabolic disturbances usually lasts much longer than changes due to seizures. The possibility of certain causes of encephalopathy (e.g., hyperglycemia, hypoglycemia, hyponatremia, hypocalcemia, hypomagnesemia) precipitating acute symptomatic seizures can further confuse the picture.

The EEG, although typically showing diffuse slowing, can, at times, display multifocal sharp waves or the triphasic wave pattern, which may be difficult to distinguish from the generalized sharp-slow complexes of nonconvulsive generalized SE.

PSYCHOGENIC NON-EPILEPTIC SEIZURE

Distinguishing psychogenic nonepileptic seizures (NESs), also known as pseudoseizures or psychogenic seizures, from epileptic seizures is a major undertaking of epilepsy monitoring units. Evidence suggests that this phenomenon is most common in young adults, especially women, but there are few data on the frequency and manifestations in elderly patients, and it may be under diagnosed.

Patients with a previous psychiatric history are likely to be at higher risk, as may be those with depression or other psychiatric complications of stroke, but data are unavailable.

In general, compared to epileptic seizures, psychogenic NESs display less stereotypy, longer duration, a more waxing and waning nature, and nonphysiologic progression[38]. Eyes tend much more often to be closed during unresponsive periods. Environmental precipitants are more likely and injuries less likely, although there are many exceptions. Unlike epileptic seizures, NES

do not arise from sleep, although they may arise from "pseudosleep," and video-EEG monitoring may be required.

INCREASED INTRACRANIAL PRESSURE

Transient increases in intracranial pressure can result in temporary alteration in awareness or, less often, focal neurologic dysfunction. The classic situations are a posterior fossa mass or intermittent obstruction of ventricular flow by a third ventricular tumor, but acute hydrocephalus can occur in patients after subarachnoid hemorrhage[39] or after ischemic or hemorrhagic stroke in the cerebellum.

Patients with cerebral edema as a result of hemispheric infarction are likely to show catastrophic focal deficits followed by progressive obtundation.

Headache is common in all of these scenarios, if the patient is alert and articulate enough to report it.

TREATMENT OF SEIZURES AND EPILEPSY

Therapy for a patient with a seizure disorder[40] is almost always multimodal. It includes treatment of underlying conditions that cause or contribute to the seizures, avoidance of precipitating factors, suppression of recurrent seizures by prophylactic therapy with antiepileptic medications or surgery. Treatment plans must be individualized, given the many different types and causes of seizures as well as the differences in efficacy and toxicity of antiepileptic medications for each patient.

Primary Generalized Tonic- Clonic	Partial	Absence	Atypical Absence Myoclonic, Atonic

FIRST- LINE

Valproic acid Lamotrigine Topiramate	Carbamazepine Phenytoin Oxcarbazepine Valproic acid	Valproic acid Ethosuximide	Valproic acid Lamotrigine Topiramate

ALTERNATIVES

Zonisamide Phenytoin Carbamazepine Oxcarbazepine Phenobarbital Primidone Felbamate	Levetiracetam Topiramate Tiagabine Zonisamide Gabapentin Phenobarbital Primidone	Lamotrigine Clonazepam	Clonazepam Felbamate

The following are few studies on the seizure disorders:

1.Clinical profile of Solitary Seizures

MJAFI 2004; 60 : 146-148

Solitary seizure is one of the controversies in neurology. This study evaluated the clinical profile of solitary seizure and the factors related to seizure recurrence with a view to evolve guidelines for management. 150 cases of solitary seizure were included in the study. All patients were males. The age varied from 18-52 years.

The diagnosis of seizure was confirmed with history. Apart from clinical neurological examination, blood counts, urinalysis and screening investigations to exclude possible underlying metabolic disorders were done. Inter-ictal EEG and contrast enhanced CT scan were done. CT scan was abnormal in 28 cases. EEG was abnormal in 33; clinical abnormality was noted in 15 cases. Median follow up duration was 1.2 years. 18 patients had seizure recurrence. Type of seizure, EEG findings (normal or abnormal) and treatment with antiepileptic drugs did not have any effect on seizure recurrence. Seizure recurrence was less common in presence of CT scan abnormality though it was not significant statistically. Routine laboratory tests of blood count, blood sugar and urinalysis were normal in all cases.

The study concluded that, in cases of solitary seizure, type of seizure, results of EEG and CT scan findings are not likely to predict seizure recurrence. Treatment with antiepileptic drugs does not prevent seizure recurrence. Laboratory investigations like blood counts, urinalysis and blood sugar estimation are unlikely to be of value in routine work-up of these cases.

2.Annals of Indian Academy of Neurology, 2005, vol.8

Seizures Disorders in Elderly

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Average age of our population is steadily increasing. This demographic change poses major challenges in the medical care of population; especially seizures disorders in the elderly. Seizures are frequently encountered in the elderly due to many medical/surgical diseases common in that age group as well as the treatment of these conditions. Incidence and prevalence of seizures increases with increasing age. Stroke, brain tumor, head injury and various metabolic diseases which are frequent in the elderly enhances the occurrences of seizures in them. About 35% seizures in patients above 75 years of age present with status epilepticus often with increased mortality. Frequency of seizures in the acute stroke may be 12-15%, while the risk of epilepsy may be as high as 30%.

Age and risk factors of stroke are also risk factors for epilepsy in the elderly. Differentiation of large number of non-epileptic conditions from seizures and epilepsy is a difficult task. EEG is less sensitive and specific than imaging studies. Cerebral atrophy and gray matter changes are common in the elderly; the pathological importance of seizures is not understood. PET & SPECT give functional information in addition.

3.Acta Neurol Scand. 1997 Aug: 96(2): 76-81.

Validation of diagnostic criteria for solitary cerebral cysticercus granuloma in patients presenting with seizures.

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The study was carried to evaluate a set of clinical and computed tomographic (CT) criteria (previously described by the authors) to predict the diagnosis of a solitary cerebral cysticercus granuloma (SCCG) at initial presentation, in patients presenting with seizures.

The diagnostic criteria were applied prospectively to patients presenting with seizures and solitary lesion on the CT scan. The clinical diagnostic criteria were as follows: seizures should be the presenting complaint; there should be no evidence of persistent raised intracranial pressure, progressive neurological deficit or an active systemic disease. The CT diagnostic criteria were: evidence of a solitary contrast enhancing lesion measuring 20 mm or less in its maximal dimension without a shift of the midline structures due to the surrounding oedema. A diagnosis of SCCG was made only when all the clinical and CT criteria were fulfilled. Over a period of 36 months, they managed 401 patients presenting with seizures and a solitary mass on the CT scan; 215 met the criteria for the diagnosis of an SCCG.

Of the 215 patients initially diagnosed to have an SCCG, 197 were ultimately determined to have that diagnosis (true positive diagnosis) while 16 were excluded because of lack of follow-up CT assessment. Two of the 215 patients with the initial diagnosis of an SCCG subsequently had histological diagnosis of a secondary metastasis and a pyogenic abscess (false

positive diagnosis). Our set of diagnostic criteria for SCCG had a sensitivity of 99.5%; specificity of 98.9%; a positive predictive value of 99%; and a negative predictive value of 99.5%. The likelihood ratios for the positive and negative tests were 92.99 and 0.005 respectively.

The diagnostic criteria helped them in not only accurately identifying an SCCG but also in differentiating it from a solitary tuberculoma and other brain masses. However, confirmation of the diagnosis of an SCCG was only obtained at follow-up evaluation and therefore careful clinical and CT re-evaluation is essential in all patients initially diagnosed to have an SCCG.

4.Mayo Clinic Proceedings November 2002 vol. 77 no. 11 1251-1264

Management of Seizure Disorders

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Neuroimaging is one of the most important advances made in the past decade in the management of seizure disorders. Magnetic resonance imaging (MRI) has increased substantially the ability to detect causes of seizure disorders, to plan medical or surgical therapy, and to prognosticate the outcome of disorders and therapy. However, MRI must be performed with techniques that will maximize the detection of potentially epileptogenic lesions, especially in candidates for epilepsy surgery. Functional imaging has an established role in evaluating patients for epilepsy surgery. It is relied on when results from standard diagnostic methods, such as clinical information, electroencephalography, and MRI, are insufficient to localize the seizure focus. Also, functional imaging is a reportedly reliable alternative to invasive methods for

identifying language, memory, and sensorimotor areas of the cerebral cortex. Despite the availability of multimodality imaging, the epileptogenic zone is not determined solely by a single imaging modality. Evidence and experience have shown that concordance of results from clinical, electrophysiologic, and neuroimaging studies is needed to identify the epileptogenic zone accurately. With modern techniques in image processing, multimodality imaging can integrate the location of abnormal electroencephalographic, structural, and functional imaging foci on a “map” of the patient's brain. Computer image-guided surgery allows surgically exact implantation of intracranial electrodes and resection of abnormal structural or functional imaging foci. These techniques decrease the risk of morbidity associated with epilepsy surgery and enhance the probability of postsurgical seizure control.

5.Epilepsia

Article first published online: 5 NOV 2007, DOI: 10.1111/j.1528-1157.1975.tb04757.x

Isolated Seizures: An EEG and Clinical Assessment

Michael Saunders, Clive Marshall.Departments of Neurology and Electroencephalography, Middlesbrough General Hospital, Middlesbrough, Cleveland, England

A study has been made of 39 patients with isolated fits presenting to an EEG department. Patients were excluded when there was a doubtful history, alcoholism, or drug abuse. The 39 patients were compared with a control group of patients with epilepsy matched for age and sex. No basic difference was found between the two groups. In particular the EEG examination showed similar changes. In conclusion, patients with isolated fits should be assessed in the same way as those with established epilepsy, while recognizing that in the majority of instances no serious underlying cause will be found

6. Postinfarction seizures. A clinical study

SR Gupta, MH Naheedy, D Elias and FA Rubino[51] Department of Neurology Veterans Administration Hospital, Hinesm Illinois[51].

They retrospectively studied 90 patients with potsinfarction seizures to determine the clinical feature (onset, number, type), prognosis, and electroencephalographic and computed tomographic findings; they included infarctions of all etiologies. Thirty-three percent of the 90 seizures appeared early (within 2 weeks after the infarction), and 90% of the 30 early seizures appeared within 24 hours after the infarction. Seventy-three percent of the 90 seizures occurred within the first year, and only 2% occurred greater than 2 years after the infarction. Fifty-six percent of the 90 seizures were more likely to be partial (57% of 30); late-onset seizures were more likely to be generalized (65% of 60). Thirty-nine percent of the 90 initial seizures recurred, and there was no significant difference in recurrence rate between early- or late-onset initial seizures. Twenty-two percent of the 90 initial seizures became multiple recurrent seizure, and they could identify a precipitating factor in 86% of the 35 recurrent seizures. The most common electroencephalographic abnormality in the 61 patients so examined was focal slowing (61%), but recurrent seizures occurred in 100% of the four patients with periodic lateralized epileptiform discharges and in 75% of the eight patients with diffuse slowing. Computed tomography in 61 patients showed that large infarction were associated with early ($p<0.021$) and multiple ($p<0.05$) seizures. Deep infarction on computed tomograms (cortical infarctions extending to sub cortical structures) tended to cause recurrent seizures ($p<0.057$).

AIM OF THE STUDY

AIM OF THE STUDY

To study the profile of the patients with seizures attending STANLEY MEDICAL COLLEGE HOSPITAL with relevance to age group, type of seizure, sex predilection, etiology with EEG and CT/MRI correlation.

To determine the prevalence of various type of seizures as in our hospital setup.

MATERIALS

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METHODS

MATERIALS AND METHODS

200 patients who attended department of Medicine/ Neuromedicine, Govt. Stanley Hospital with history of seizures from October 2009 to October 2010.

All patients in the age group > 12 yrs presenting with history of seizures were included in the study.

Patients with the below mentioned features were excluded from the study.

- Critically ill patients with acute Metabolic derangements
- Patients admitted with history of Poisoning
- Patients admitted with history of Attempted Hanging
- Patients with Hepatic Failure
- Patients with Renal Failure

A detailed history of Presenting Illness, Past history, Family, Personal and Social history were obtained along with occupation and socio economic status from the patient and reliable attendees. Details of the seizures included:

- First attack
- Last attack
- Type of seizures
- Part of body involved
- Duration
- Frequency
- Maximum period of freedom

- Time of attack
- Precipitating factors
- Aura
- Post-ictal symptoms
- automatism

Past history included

- Hypertension, including PIH
- Diabetes mellitus
- Head injury
- Previous stroke
- Cardiovascular illness (RHD/ MVP/ IHD)
- Respiratory illness (COPD/ PTB/ BA)
- Renal impairment
- Jaundice
- Psychiatric illness

Personal history included

- Smoking
- Drugs
- Alcohol
- Sleep
- Pork
- Snuff

- Other addictions

A detailed general examination, including vital signs recording, complete neurological examination and examination of other systems were done.

Neurological examination included

- Neurocutaneous markers
- Focal deficits
- Confusional state
- Fundal examination

Investigations like Blood counts, Random blood sugar, Renal function tests, Electrolytes, Chest X Ray, ECG, CT Scan Brain/ MRI Scan, and EEG were done for all patients.

In cases of acute febrile illness or signs of meningeal irritation associated with seizures,

- QBC- for malarial parasites and
- Cerebrospinal fluid analysis was done.

Lumbar puncture for CSF analysis was done under aseptic precautions. CSF was analysed for;

- Biochemistry: sugar, proteins
- Cell count: total, differential
- Cytology
- Culture and sensitivity
- Ziehl-Nielson staining
- When these showed abnormalities, anti HSV antibodies were done when relevant.

In suspicious patients, like those who had Tuberculoma- brain, Viral encephalitis, HIV- ELISA and VDRL were done.

Cardiac evaluation was done for patients with abnormal physical findings in cardiovascular system, abnormal ECG and for all patients with cerebrovascular etiology.

EEG was done for all the patients irrespective whether an underlying cause was found or not.

The clinical profile, brain imaging finding, EEG findings were all analysed to derive the various conclusions.

RESULTS

&

OBSERVATIONS

OBSERVATIONS

TABLE 1.

Age and Sex Distribution

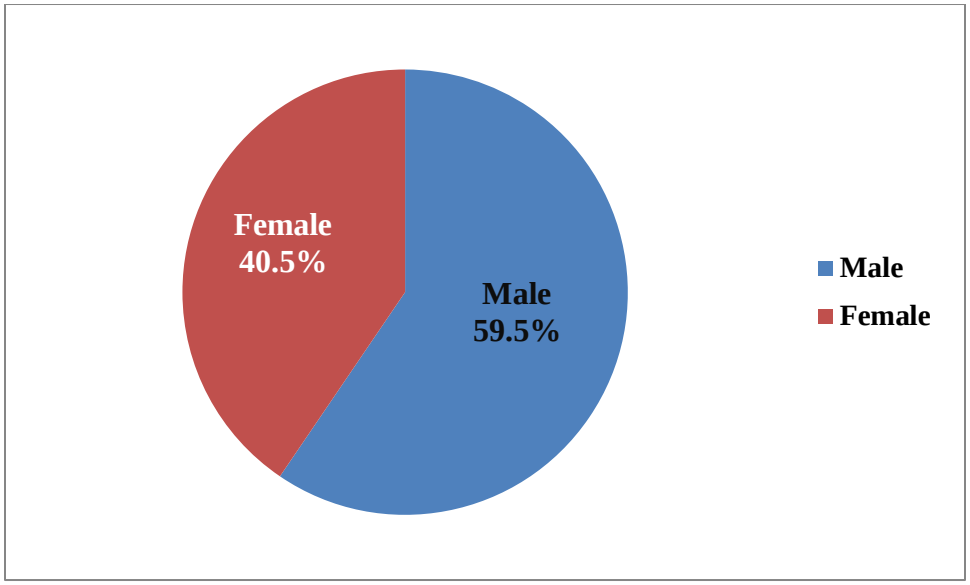
Age group	Male	Female	Total
12-20	25	20	45 (22.5%)
21-30	23	31	54 (27%)
31-40	26	8	34 (17%)
≥ 40	45	22	67 (33.5%)
Total	119 (59.5%)	81 (40.5%)	200

TABLE 2.

Distribution of the variables studied

Seizure type	Number of patients	Male	Female	Age (12-20 yrs)	Age (21-30 yrs)	Age (31-40 yrs)	Age (≥ 40 yrs)	Image Normal	Image Abnormal	EEG Normal	EEG Abnormal
(A).GENERALIZED											
(1). Tonic clonic	137	84	53	33	38	27	39	108	29	88	49
(2). Absence	01	01		01	-	-	-	01	-	-	01
(3). Myoclonus	03	01	02	03	-	-	-	03	-	01	02
Total	141	86	55	37	38	27	39	112	29	89	52
(B). PARTIAL											
(1). SPS	09	05	04	01	05	02	01	01	08	05	04
(2). CPS	17	07	10	04	04	01	08	08	09	09	08
(3). SPSG	31	19	12	01	07	04	19	07	24	18	13
(4). CPSG	01	01	-	01	-	-	-	01	-	01	-
(5). Hemiconvulsion	01	01	-	01	-	-	-	-	01	-	01
Total	59	33	26	08	16	07	28	17	42	33	26

1. SEX DISTRIBUTION



2. AGE DISTRIBUTION

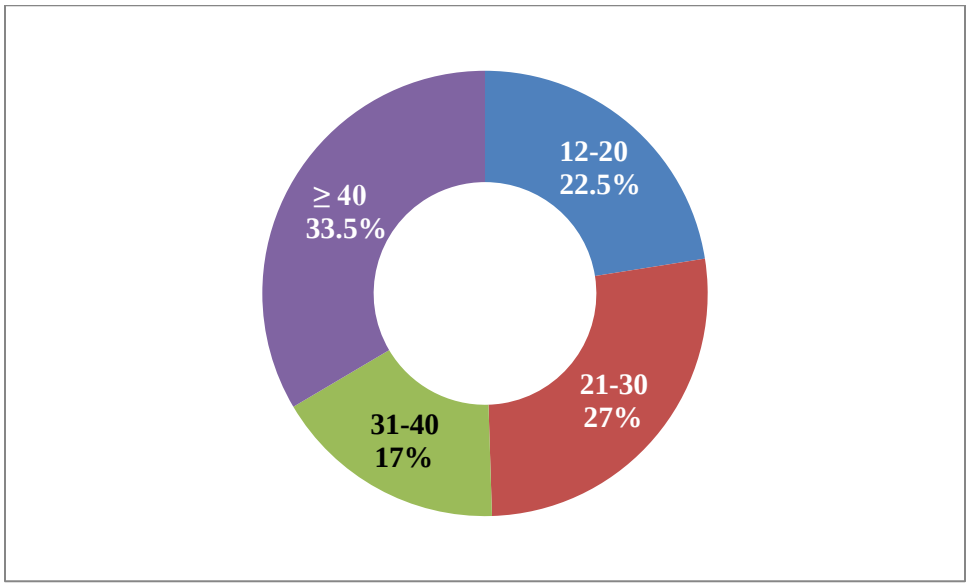


Table .3

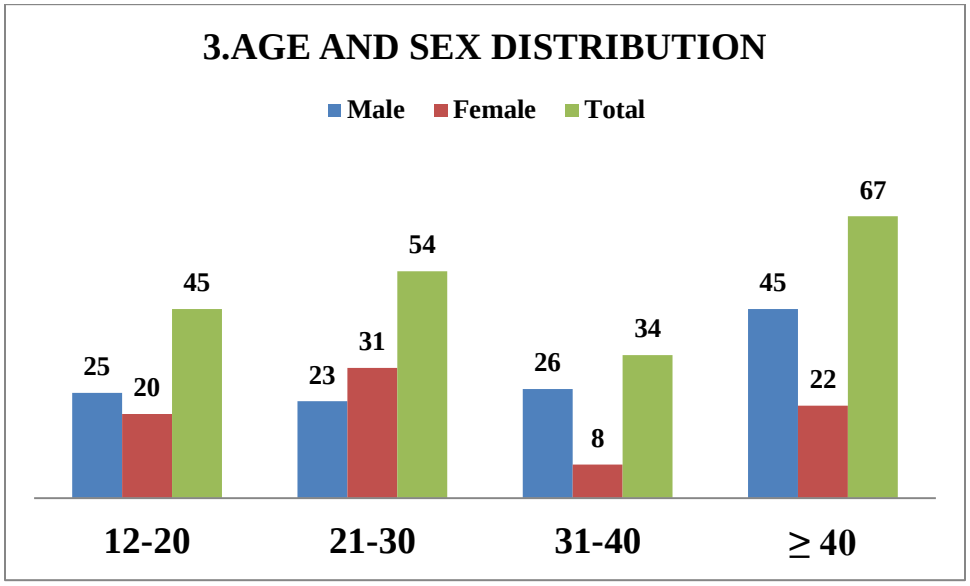
DISTRIBUTION OF DOMINANT SEIZURE TYPES CLASSIFIED BY CLINICAL PRESENTATION

Seizure type	Number of patients
(A).GENERALIZED	
(1). Tonic clonic	**137** (68.5%)
(2). Absence	**01** (0.5%)
(3). Myoclonus	**03** (1.5%)
Total	**141** (70.5%)
(B). PARTIAL	
(1). SPS	**09** (4.5%)
(2). CPS	**17** (8.5%)
(3). SPSG	**31** (15.5%)
(4). CPSG	**01** (0.5%)
(5). Hemiconvulsion	**01** (0.5%)
Total	**59** (29.5%)

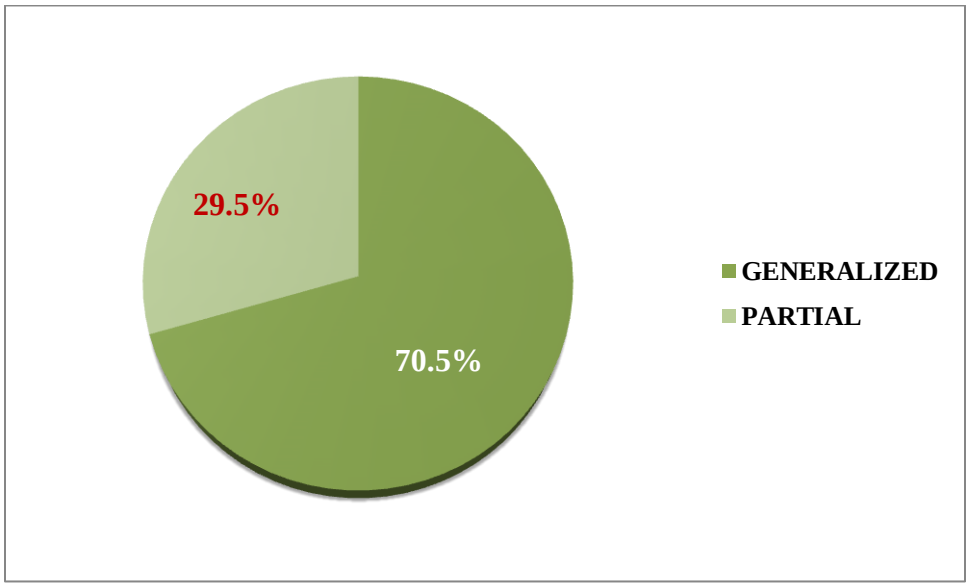
Table .4

SEIZURE TYPES AND IMAGING

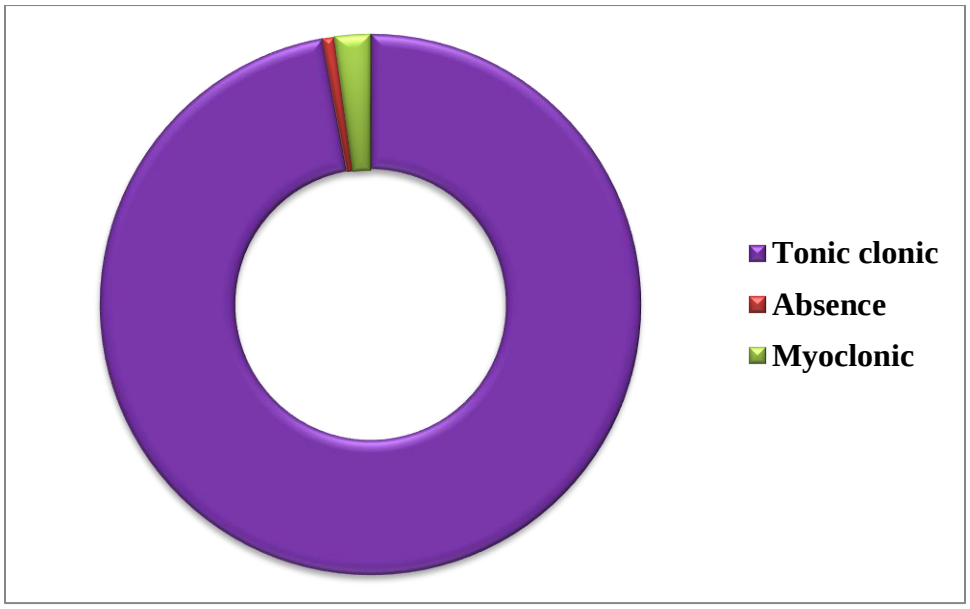
Seizure types	Number of patients	Image	Image
		Normal	Abnormal
(A).GENERALIZED			
(1). Tonic clonic	137	108	29
(2). Absence	01	01	0
(3). Myoclonus	03	03	0
Total	141	112	29
(B). PARTIAL			
(1). SPS	09	01	08
(2). CPS	17	08	09
(3). SPSG	31	07	24
(4). CPSG	01	01	0
(5). Hemiconvulsion	01	0	01
Total	59	17	42



4. DISTRIBUTION OF THE DOMINANT SEIZURES



5. DISTRIBUTION OF GENERALIZED SEIZURES



6. DISTRIBUTION OF PARTIAL SEIZURES

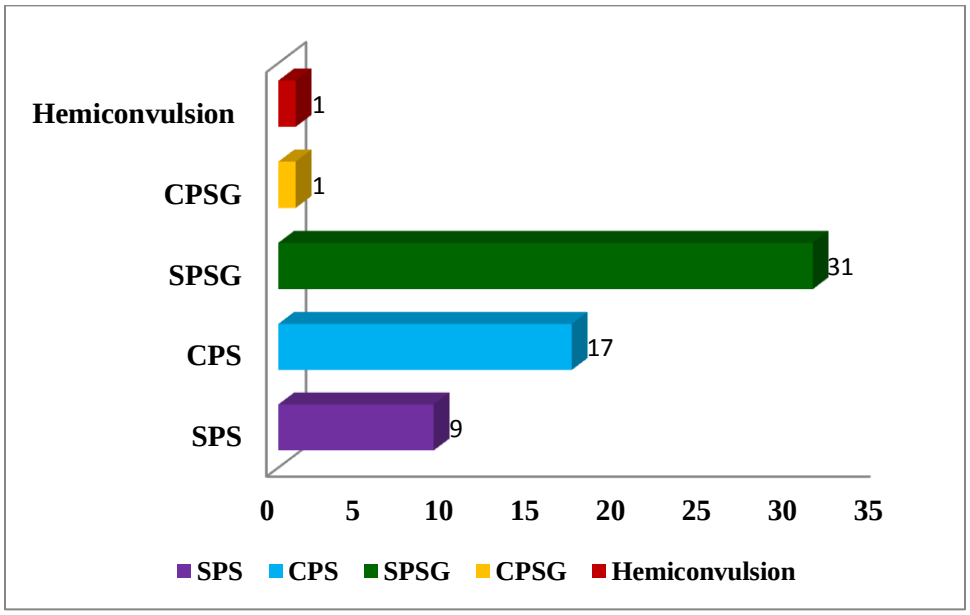


Table . 5

AURA

Aura	No of cases
Present	38
Absent	162

Table. 6

FAMILY HISTORY OF SEIZURES

PRESENT	10
NOT PRESENT	190

Table. 7

EEG FINDINGS

Normal	**122** (61%)
Abnormal	**78** (39%)

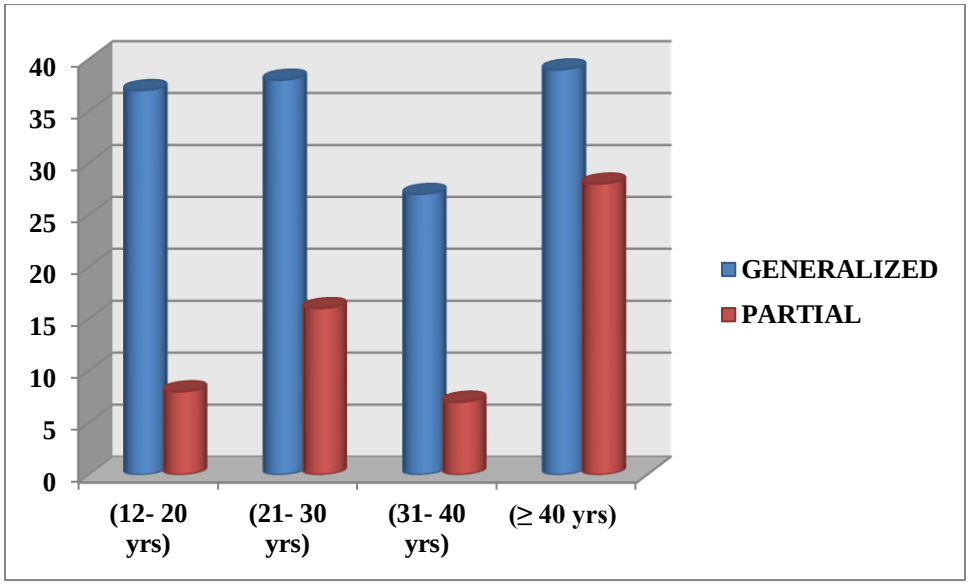
Table .8

EEG FINDINGS –

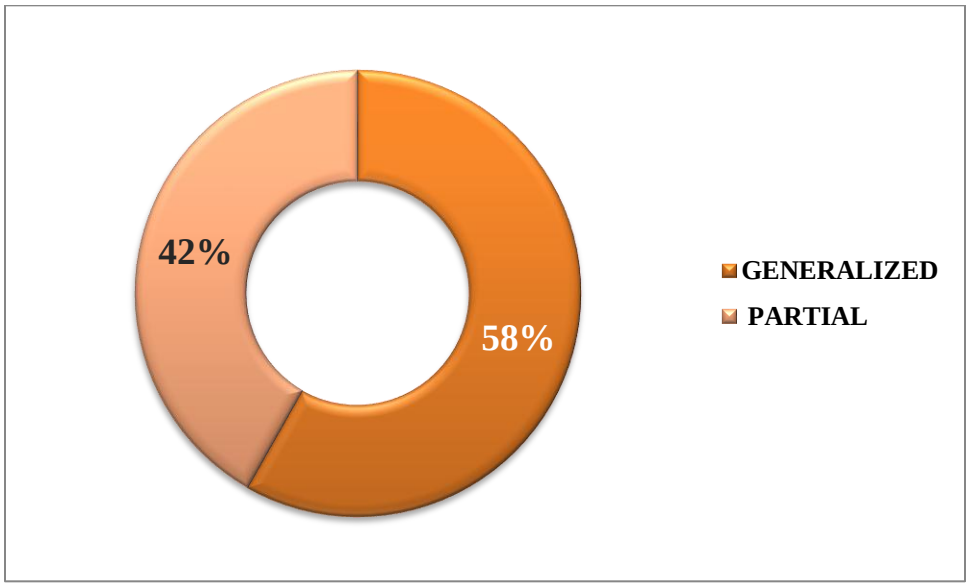
GENERALISED VS PARTIAL SEIZURES

SEIZURE TYPE	EEG NORMAL	EEG ABNORMAL
GENERALISED	89	52
PARTIAL	33	26

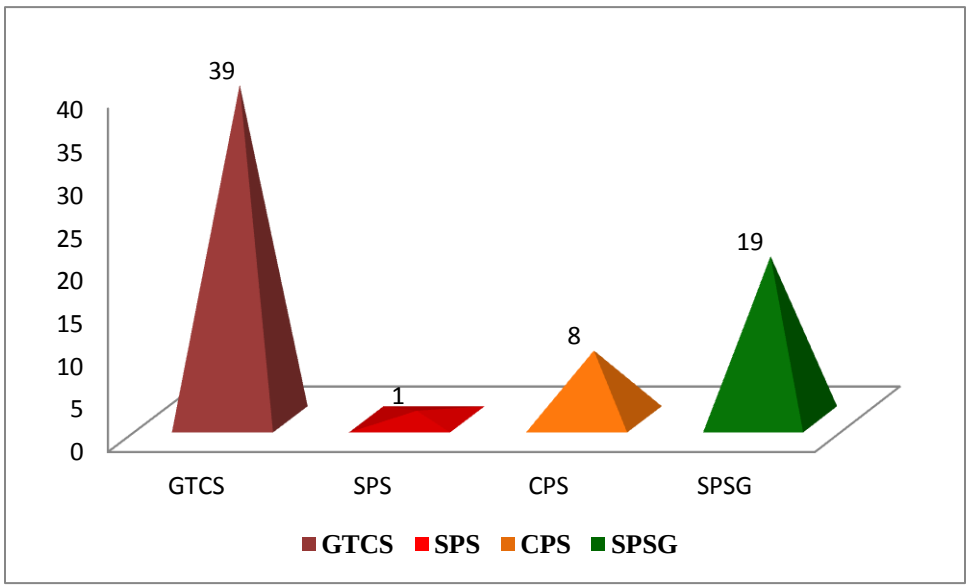
7. DISTRIBUTION OF DOMINANT SEIZURES TYPE AS A FUNCTION OF AGE



8. DISTRIBUTION OF GENERALISED AND PARTIAL SEIZURES IN PATIENTS AGED >40 YEARS



9. DISTRIBUTION OF DOMINANT SEIZURE TYPES IN PATIENTS AGED >40 YEARS



10. NEW ONSET SEIZURES VS RECURRENT SEIZURES

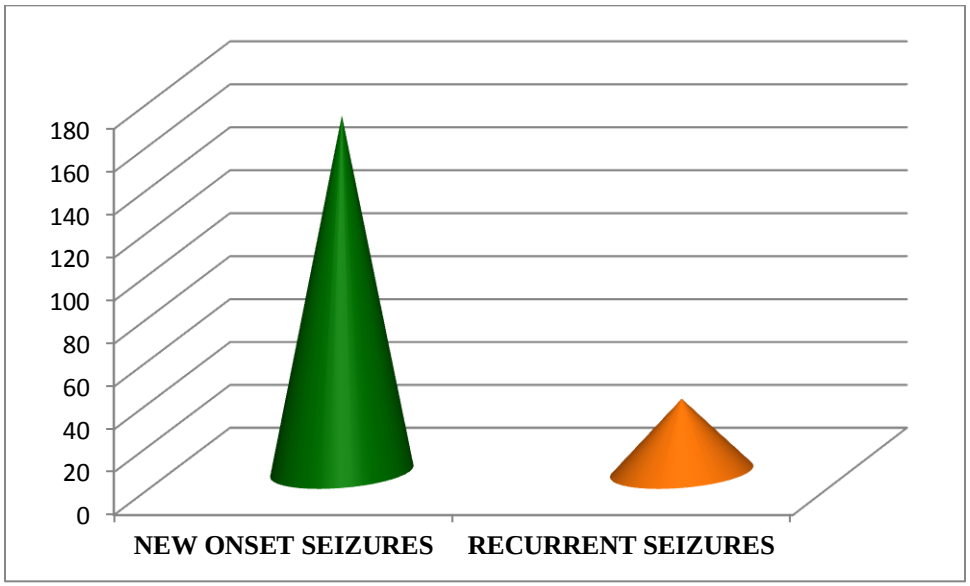


Table. 9

BRAIN IMAGING

Normal	**129** (64.5%)
Abnormal	**71** (35.5%)

TABLE . 10.

BRAIN IMAGING FINDINGS

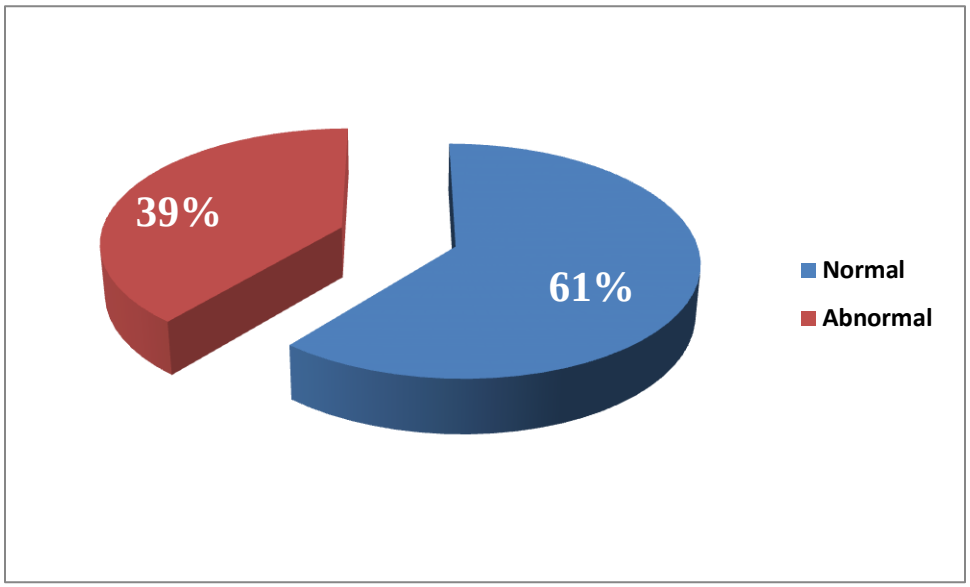
Findings	No of Cases
Normal	129
Cerebral infarction	24
Intra cerebral haemorrhage	7
CVST	10
Granuloma – TB	7
Granuloma - NCC	4
SOL- primary	7
SOL- secondary	2
Diffuse cerebral atrophy	3
Gliotic changes	3
Subdural hematoma	1
Temporal Hypodensity (s/o HSV)	2
Enlarged Cisterna Magna	1

Table .11

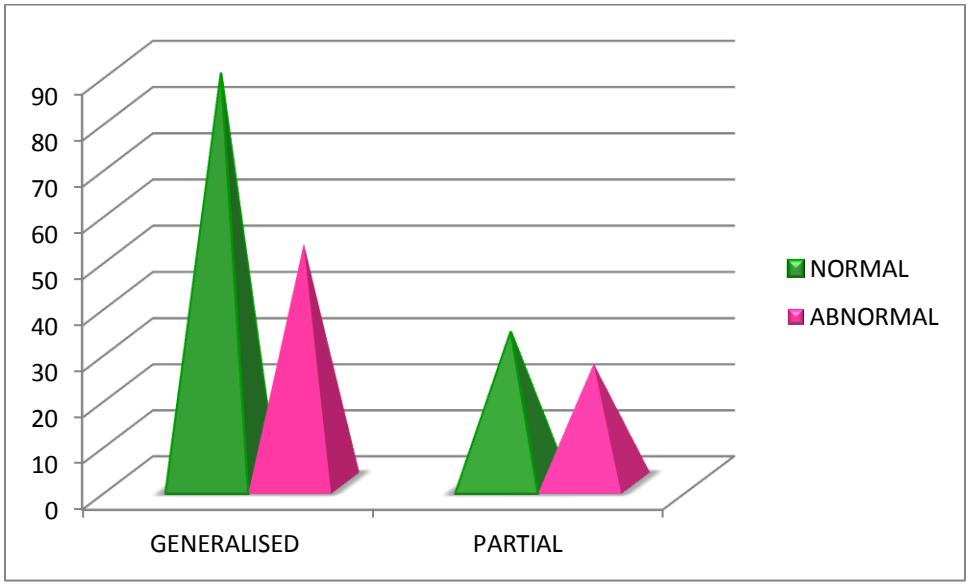
IMAGING & EEG CORELATION

IMAGING	EEG	No of Cases
Normal	Normal	77
Normal	Abnormal	52
Abnormal	Normal	45
Abnormal	Abnormal	26

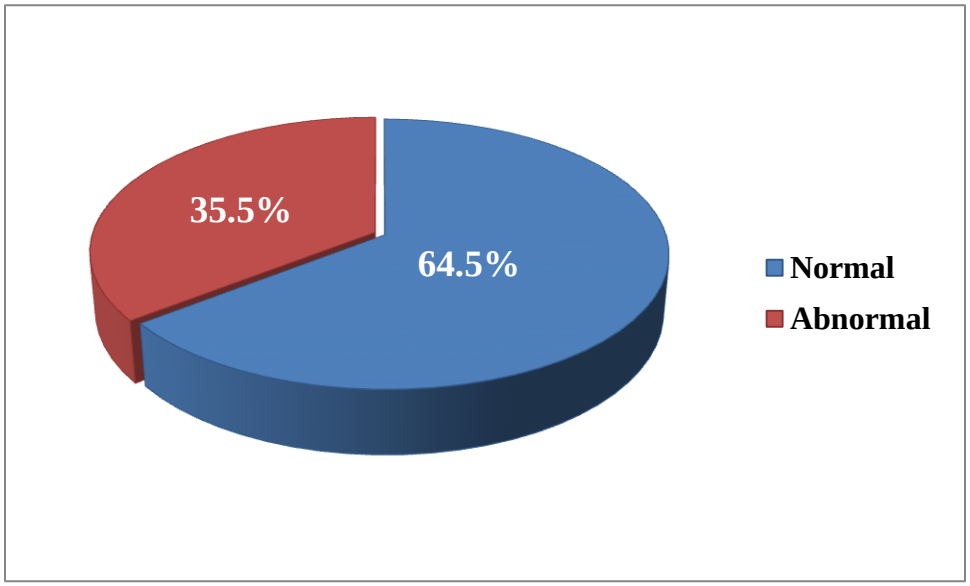
11.EEG FINDINGS



12. EEG FINDINGS IN GENERALISED AND PARTIAL SEIZURES



13. BRAIN IMAGING



14. IMAGING FINDINGS IN SEIZURES SUBTYPES

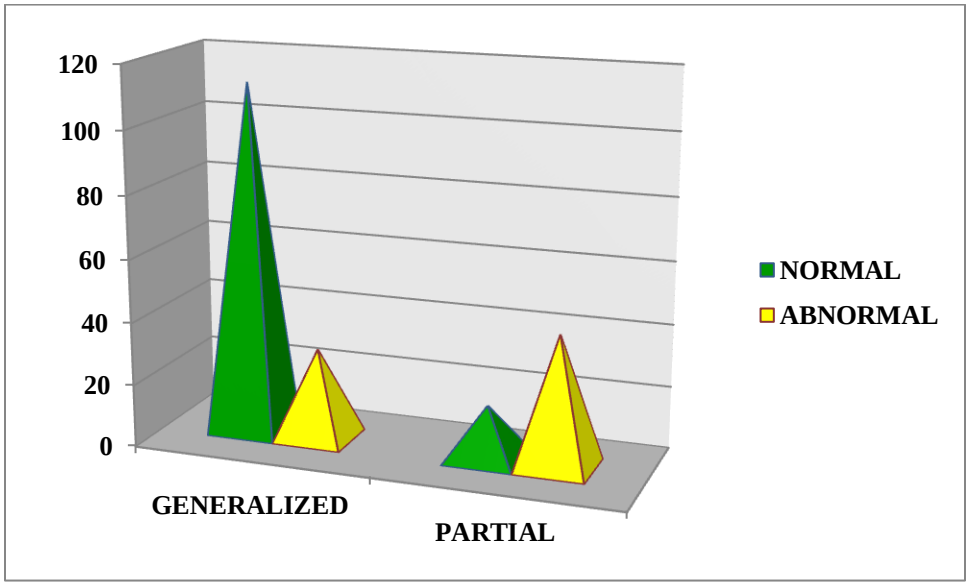


Table . 12

ETIOLOGY AND SEIZURE TYPES CORRELATION

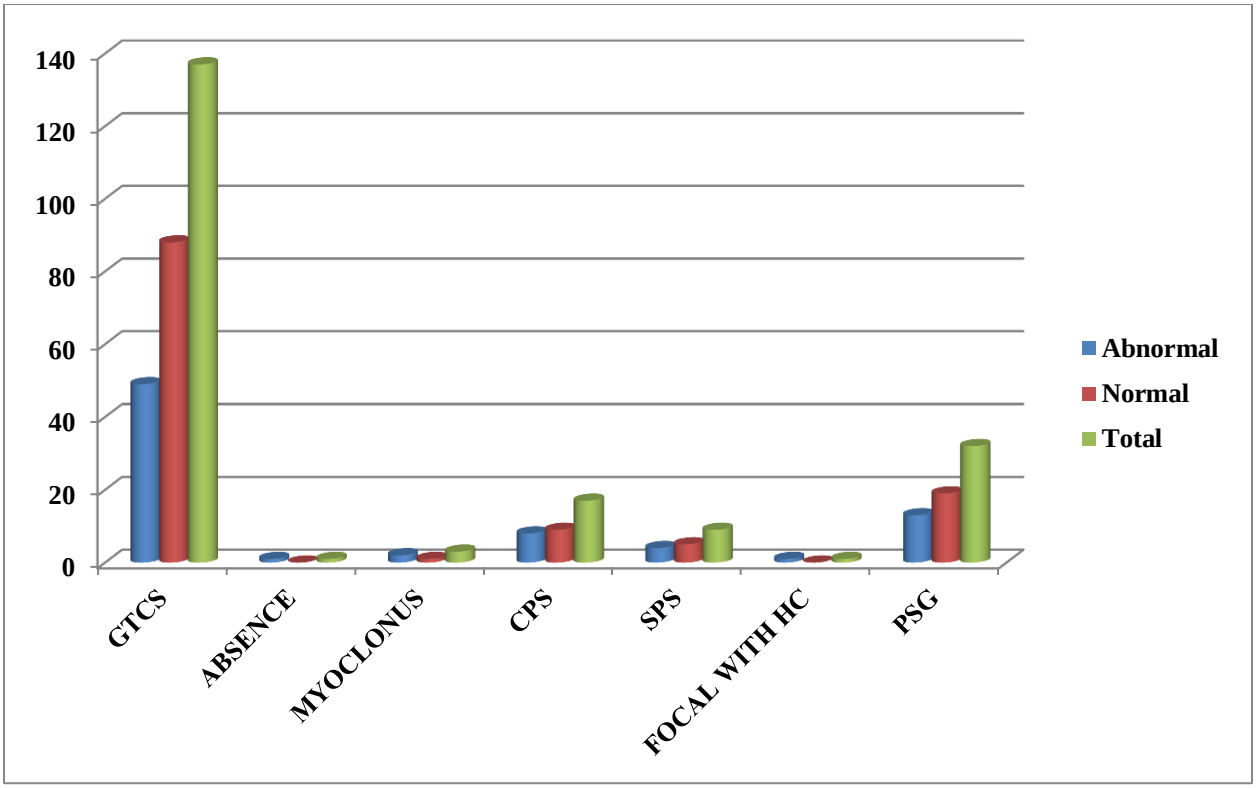
ETIOLOGY	GTCS	ABSENCE	MYOCLONUS	CPS	CPSG	SPS	PSG	FOCAL H C
Unknown	100	1	3	8	1	1	7	0
Cerebral infarct	9	0	0	3	0	0	12	0
CVST	6	0	0	0	0	3	1	0
Cerebral haemorrhage	2	0	0	2	0	0	3	0
SOL- primary	4	0	0	1	0	2	0	0
SOL- secondary	0	0	0	0	0	0	2	0
Granuloma – TB	0	0	0	2	0	2	3	0
Granuloma – NCS	2	0	0	1	0	1	0	0
Cerebral malaria	4	0	0	0	0	0	0	0
Cerebral atrophy	4	0	0	0	0	0	2	1
HSV	2	0	0	0	0	0	0	0
Meningitis	4	0	0	0	0	0	0	0
Subdural hematoma	0	0	0	0	0	0	1	0

Table . 13

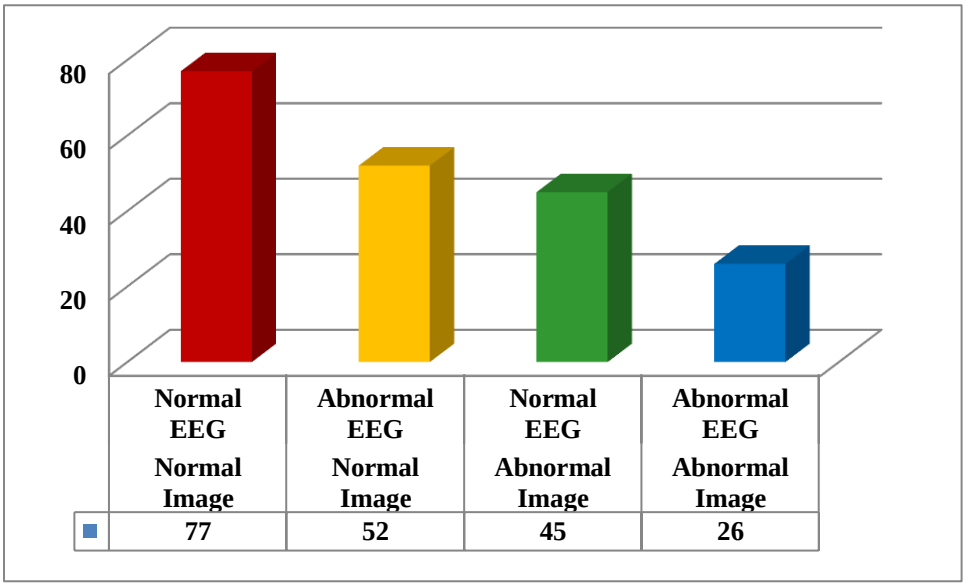
EEG AND SEIZURE TYPES CORRELATION

EEG	GTCS	ABSENCE	MYOCLONUS	CPS	SPS	FOCAL WITH HC	PSG
Abnormal	49	01	02	8	4	01	13
Normal	88	-	01	9	5	-	19
Total	137	01	03	17	9	01	32

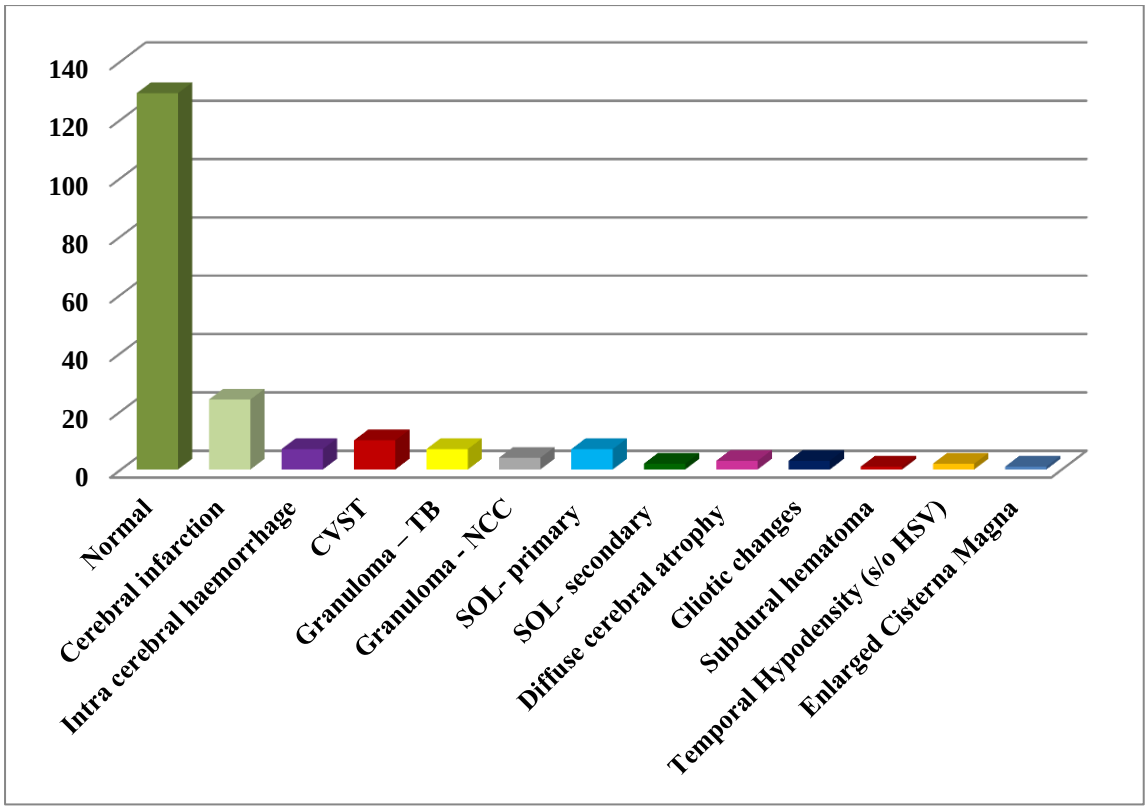
15. SEIZURE TYPES AND EEG CORRELATION



16. BRAIN IMAGING – EEG CORRELATION



17. BRAIN IMAGING - FINDINGS



18. DISTRIBUTION OF SEIZURES WITH UNDETERMINED ETIOLOGY

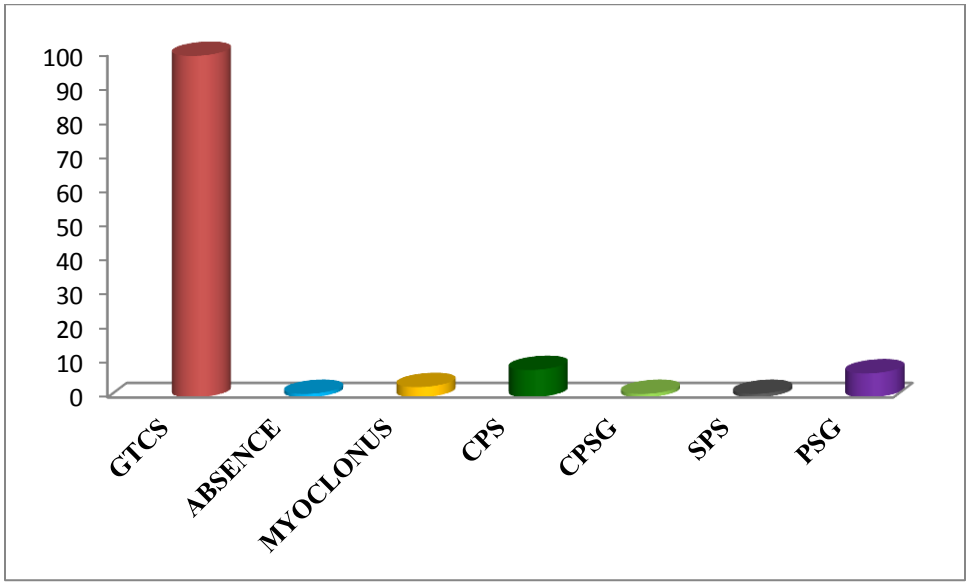


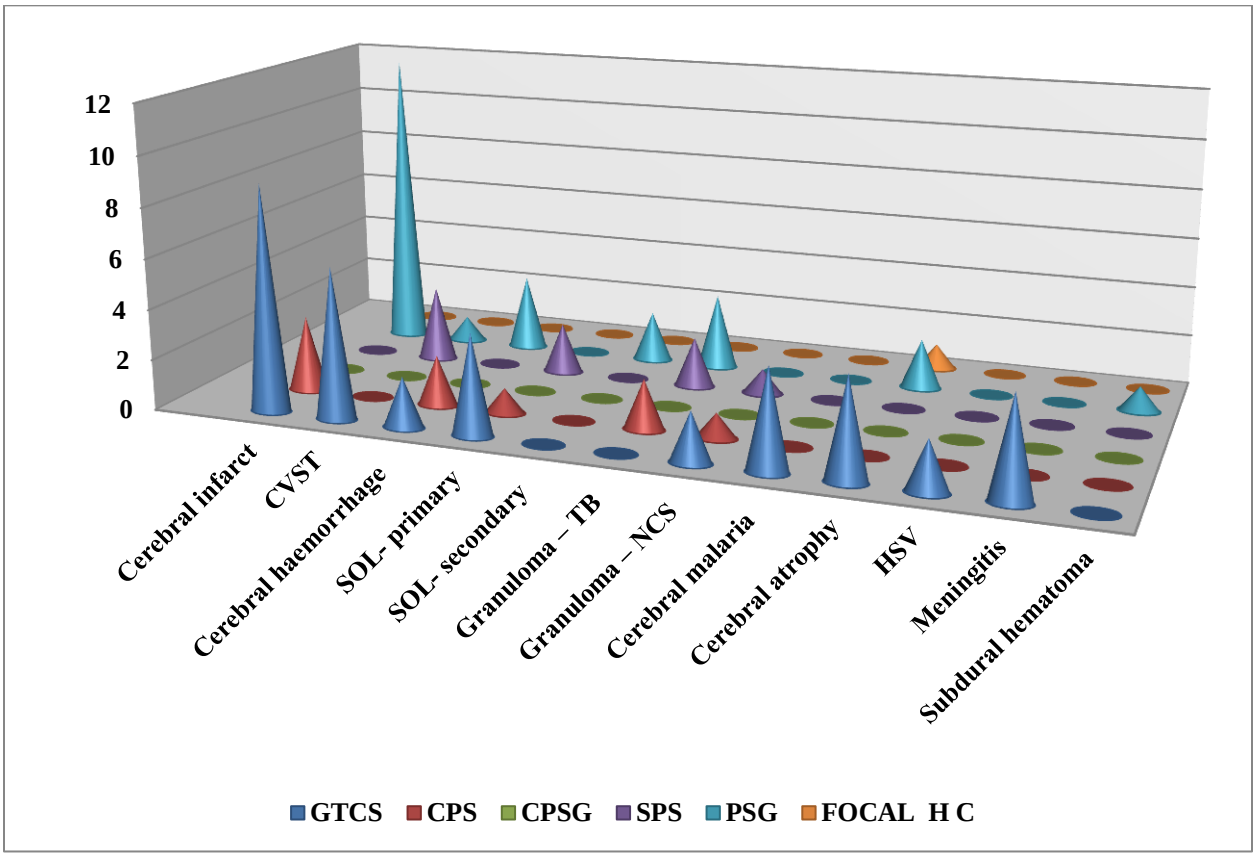
Table . 14

ETIOLOGY OF SEIZURES - CLINICAL, EEG

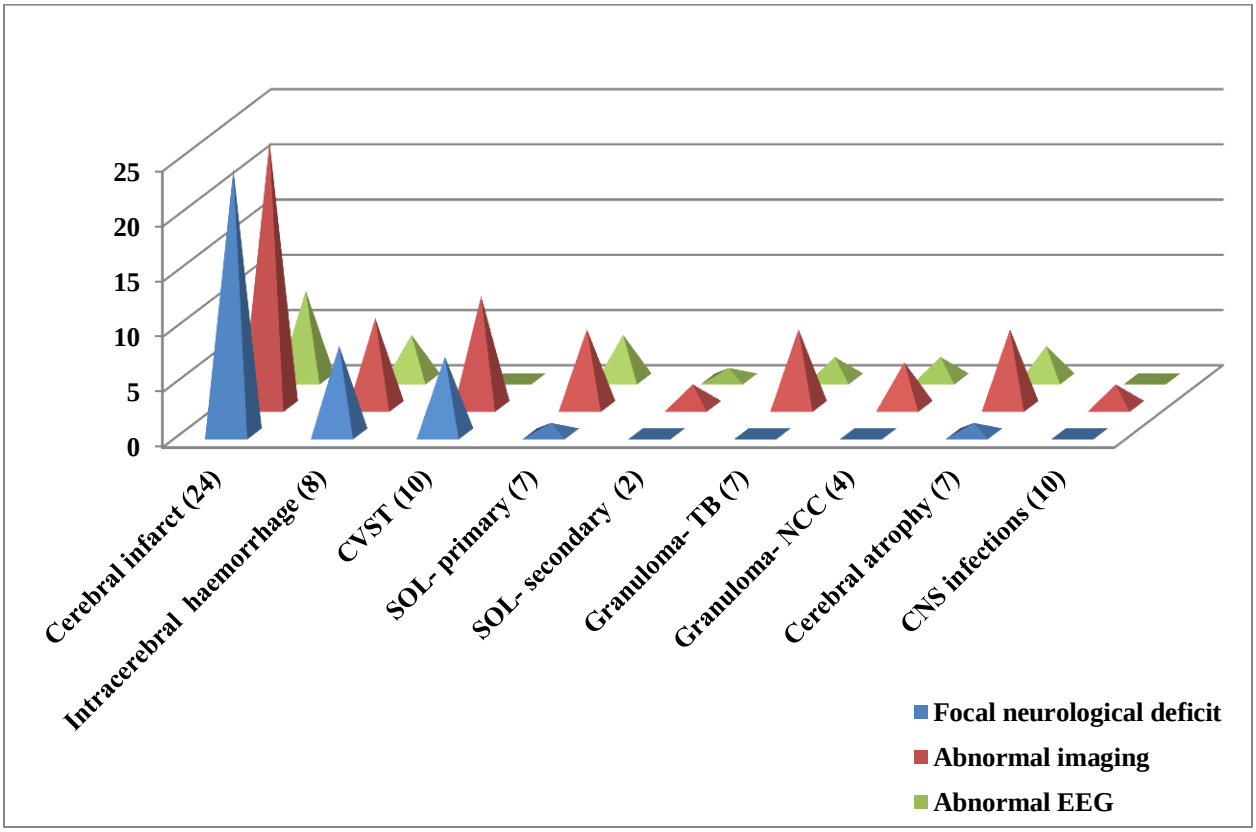
AND IMAGING CORRELATION

Etiology	Focal neurological deficit	Abnormal imaging	Abnormal EEG
Cerebral infarct (24)	24	24	8
Intracerebral hemorrhage (8)	8	8	4
CVST (10)	7	10	0
SOL- primary (7)	1	7	4
SOL- secondary (2)	0	2	1
Granuloma- TB (7)	0	7	2
Granuloma- NCC (4)	0	4	2
Cerebral atrophy (7)	1	7	3
CNS infections (10)	0	2	0

19. DISTRIBUTION OF SEIZURES WITH DEFINITE ETIOLOGY



20. SEIZURES – ETIOLOGY, IMAGING, EEG AND CLINICAL CORRELATION



DISCUSSION

Discussion

The incidence of epilepsy in developed countries is 50 per 100,000. In developing countries like India, the incidence is almost double up to 100 per 100,000. A meta-analysis of studies shows an overall prevalence rate of 5.59 per 1000 (International journal of Neurology, vol 9, 2008).

The present study was done to analyze the distribution of dominant seizure type according to clinical symptoms, as a function of age and sex, etiology, imaging and EEG correlation. Idiopathic seizures are more common in early years of life. As age increases, it is more likely that the episodes are symptomatic rather than being idiopathic. The goal of evaluation of a patient with paroxysmal episodes of seizures are to determine whether the patient has epilepsy, characterize the type of seizures, identify the potential cause of seizures, determine the course of treatment and provide the patient and the family about the course of the disorder[9].

The following are the inferences derived from the observations made in the study.

AGE DISTRIBUTION

In this study, 22.5% of patients were in the 12-20 yrs age group, 27% in 21-30yrs, 17% in 31-40 yrs and 33.5% in more than 40 yrs age group respectively. Second and third decades together constituted 49.5% of the study population[54] (Tables 1 & 2, Charts 2&3).

R.Sridharan, B.N. Murthy et al 1999 state that prevalence and incidence rates is higher in the first three decades of life[53].

SEX DISTRIBUTION

In this study males were more in number than females, men contributed to 59.5% of the study population (Tables 1&2, Charts 1&3).

The prevalence of epilepsy in men were 6.05 and in women 5.18 per 1000 according to *R.Sridharan, B.N. Murthy et al 1999.*

TYPE OF SEIZURES

Generalised seizures were found in 70.5% and partial seizures in 29.5% of the study population. GTCS was the most common type of seizure[55] and was found in 68.5% of cases. Partial seizures with secondary generalization constituted 15.5% of cases. Complex partial seizures (8.5%), Simple partial seizures (4.5%), Myoclonus (1.5%), Absence seizures (0.5%) and Complex partial seizures with secondary generalization (0.5%) formed the remaining cases. (Tables 2&3, Charts 4,5&6). The distribution of various seizures types according to age group is shown in chart 7. Classification of seizures in this study was made according to ILAE classification[56].

The frequency of SPS was less in this study. This might be because patients ignore episodes of insignificant seizures like partial seizures without gross disturbances in consciousness especially in a country like ours. Absence seizure was observed in one case. It is more common in 5-15 year age group. This study population consisted mostly of adult patients and only 22.5% of patients were in 12-20 years age group.

Patients aged more than 40 years formed 33.5% of the study population. Among them 58% presented with Generalised seizures and 42% presented with Partial seizures (Charts 8&9).

This study excluded patients who presented with seizures of metabolic etiology and alcohol withdrawal. A secondary rise in incidence occurred after 40 years, increasing with advancing age[8].

PAST HISTORY OF SEIZURES

Of the study population, 83% had new onset seizures and 17% had recurrent episode of seizure (Chart 10). Among the patients who had recurrent seizure, 11 patients had childhood seizures of which three patients had febrile seizure. One patient was mentally retarded. The most common precipitating factor for recurrent seizure was poor drug compliance. Infections also precipitated seizure .

AURA

It was seen in 38 cases in the study population. Headache was most common symptom preceding seizure. The other patients could not recollect the prodromal symptoms (Table 5).

FAMILY HISTORY OF SEIZURES

Family history of seizures was found only in 10 (5%) patients (Table 6). Family history of epilepsy in first degree relative has been observed in 5.2 – 8.9% [70].

Sathishchandra et al recorded a history of epilepsy in first and second degree relatives in 13.7% of their study population[71].

POST ICTAL STATE

All patients with GTCS and Partial seizures with secondary generalization had post ictal state. Post -ictal confusion, disorientation, headache and myalgia were the most common

symptoms in the study population. Patients who presented with status epilepticus were found to have prolonged post-ictal state lasting for more than 24 hours. Of the 11 patients who presented with status, 9 patients had structural brain lesion.

EXAMINATION FINDINGS

In this study neurological deficit was found in 41 patients (20.5%). 31 patients had CVA and had neurological deficit according to the vascular territory affected. 7 patients were found to have Cerebral sinus venous thrombosis out of which four female patients presented with post-partum seizures. One patient had primary space occupying lesion, Schwannoma and one patient had Hemiplegia hemiconvlsion (Table 14,Chart 20).

Other findings seen in the study were signs of meningeal irritation (10 cases), features of dementia (02 cases), and confusional state, papilledema (17 cases).

BRAIN IMAGING

Normal imaging was seen in 64.5% (129) of cases and abnormal imaging seen in 35.5% (71) of cases in this study. Among the patients who presented with generalised seizures 79.5% of cases had normal imaging and 20.5% had abnormal imaging. Among patients with partial seizures 71% had abnormal imaging pointing to an etiology and the 29% of cases had normal imaging (Table 9, Charts 13&14).

The image related abnormalities are as follows – Infarct (33.8%), Cerebral Venous Sinus Thrombosis (14%), Tuberculoma (9.8%), Primary Brain Tumor (9.8%), Intracerebral hemorrhage (9.8%), Neurocysticercosis (5.6%), Gliotic changes (4.2%), Cerebral atrophy

(4.2%), HSV Encephalitis (2.8%), Brain Metastasis (2.8%), Enlarged cistern magna and Subdural hematoma (1.4%) each (Table 10, Chart 17).

Hence imaging is very important in the management of seizures especially in partial seizures. Every patient with new onset seizure should undergo imaging.

CEREBRAL INFARCTION

In the study population 24 patients had cerebral infarct. Of these, three patients presented with status epilepticus. 5 patients had seizures that occurred within a period of 1-4 weeks of stroke (Early post-stroke seizures).

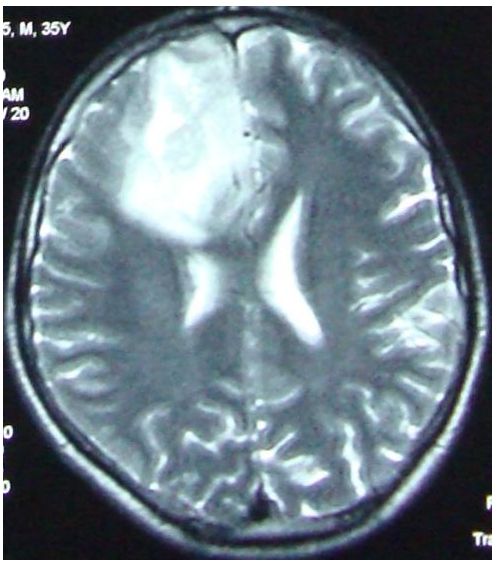
Gupta[56] *et al*, in their study of 90 patients with ischemic stroke stated that 33% presented with early post-stroke seizures. *Labovitz*[57] *D L et al*, state that 5-10% of individuals with CVA experience seizure at the time of onset.

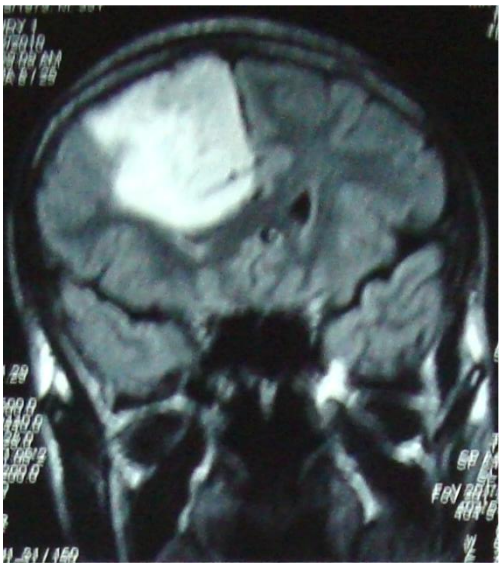
16 patients had post-stroke seizures after an interval varying from 3-6 months. Of these 7 patients presented with GTCS, 6 patients presented with Partial seizures with secondary generalization and 3 patients presented with CPS. *Panayiotis Varelas et al*[58] state that late post-stroke seizures occurs in 2.5-67% and recurrent seizures occur in 49% of post-stroke patients.

CEREBRAL HEMORRHAGE

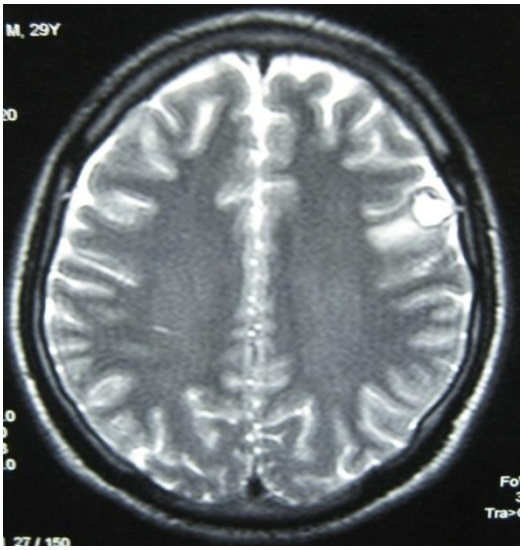
Seven patients presented with intracerebral hemorrhage and one patient presented with Subdural hematoma. Of these, two patients presented with GTCS, two patients with CPS and four patients had partial seizures with secondary generalization. *Panayiotis Varelas et al*[58] also state that about 31% of patients with ICH present with seizures at the onset.

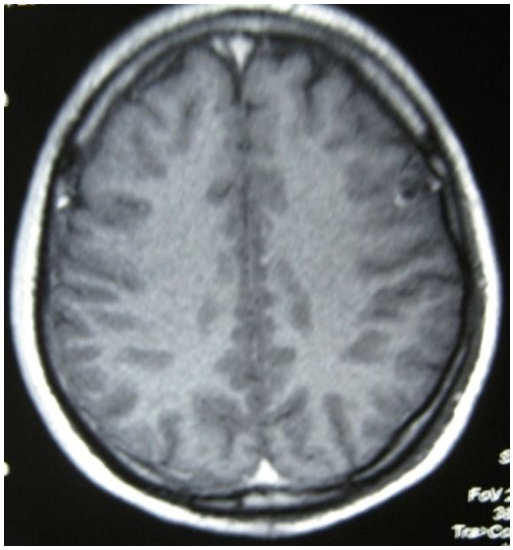
ASTROCYTOMA - MRI IMAGES





NEUROCYSTICERCOSIS – MRI IMAGES





GRANULOMAS

(A). NEUROCYSTCERCOSIS (NCC)

NCC usually presents as single enhancing lesion, though there are various presentations. *Garcia et al*[59] state that Single Cysticercal Granuloma (SCG) are the most frequent clinical presentation of NCC in India. They also state that in the imaging studies performed in the general population of endemic villages and other asymptomatic populations brain calcifications were more common than viable cyst or SCG. Of the four patients presented with NCC, three had single calcified granuloma and one patient had multiple ring enhancing lesions. Two patients presented with GTCS, one patient with SPS and one with CPS.

(B). TUBERCULOMA

In the study population seven patients were found to have Tuberculoma. All patients presented with partial seizures, CPS (2), SPS (2), and PSG (3). In a study " *Brain CT and MRI findings in* 100 *consecutive patients with intracranial tuberculoma" Wassy M*[60] *et al* state that multiple lesions and infratentorial lesions were more common.

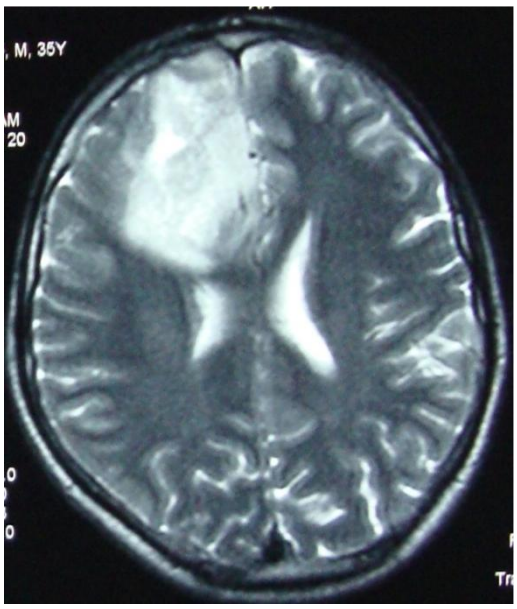
Patwari AK et al[61]*, 1996* state that moderate and to marked perilesional oedema is frequently associated with parenchymal Tuberculoma providing substrate for seizures.

CEREBRAL VENOUS SINUS THROMBOSIS (CVST)

In the study 10 patients were found to have CSVT. 6 patients presented with GTCS, three patients with SPS and one patient with PSG. Of these, 4 patients had post-partum seizures.

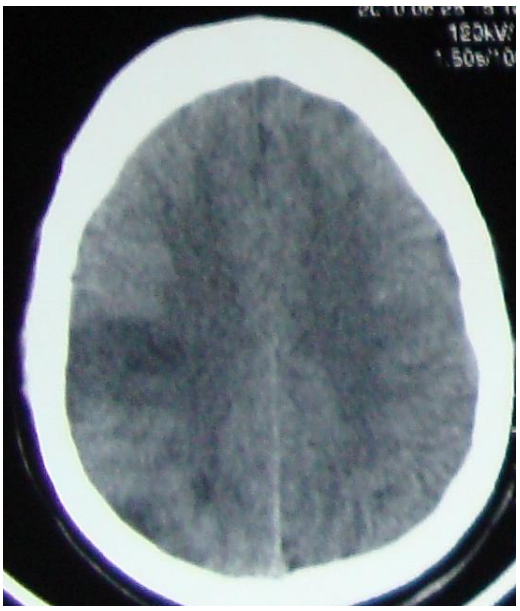
SCHWANNOMA

MRI – IMAGE

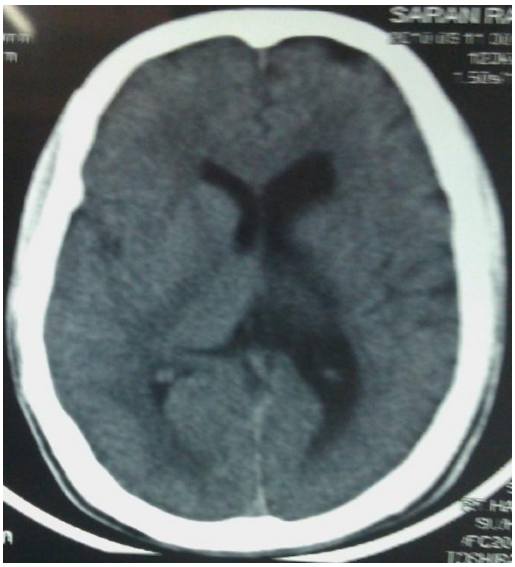


(R) PARIETAL INFARCT

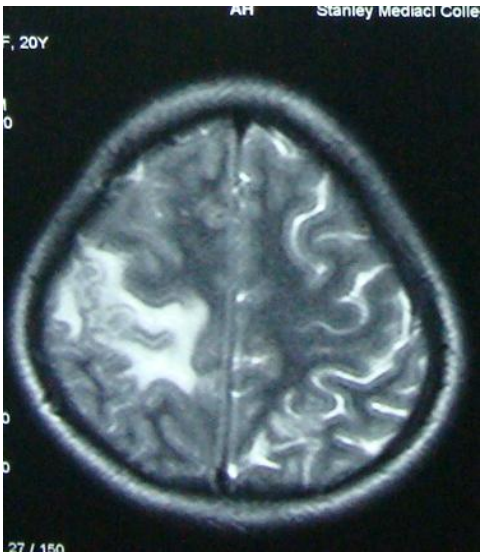
CT - IMAGE



(L) CEREBRAL HEMIATROPHY



TUBERCULOMA



CVST is rather a rare disease which accounts for less than 1% of all stroke. Diagnosis is frequently overlooked or delayed due to wide spectrum of clinical symptoms and often subacute onset[62].

Ferro et al[63] reported that early seizures occurred in 40% of patients with CVST and 7% of them had a higher risk of recurrent seizures within two weeks.

Most frequently affected location are Superior Sagittal sinus, Transverse sinus, Sigmoid sinus, and Sinus Cavernosus[64]. Similar findings were found in the present study.

BRAIN TUMORS

In the study 7 patients had primary brain tumors and 2 patients had brain secondaries. Among the patients four cases presented with GTCS, one patient presented with CPS, and two patients presented with SPS. Among the patients with brain metastasis, one patient presented with status epilepticus, and other patients had PSG. Both patients had bronchogenic carcinoma. Among the primary tumours 3 patients were diagnosed to have Meningioma, one patient had CP angle tumour, one patient had Schwannoma, one had Astrocytoma and one patient had Glioma.

Although neoplasms of the brain account for only 1% of cases of epilepsy, seizures occur in approximately 50% of children with supratentorial tumors and seizures develop in approximately 35% to 40% of adults with brain tumors (*LeBlanc FE*[14] *et al,* 1974). Seizures occur especially commonly in association with Oligodendrogliomas. Seizures are also commonly encountered in patients with Meningiomas. In contrast, the incidence of seizures in patients with cerebral metastasis is much lower; most reports suggest an incidence of 20% at the time of presentation (*Gamache FW*[15]*, et al,* 1979).

CATAMENIAL EPILEPSY

In this study population one patient was found to have increased frequency of seizures in the premenstrual period. Catamenial epilepsy is due to either the effects of estrogen and progesterone on neuronal excitability. Acetazolamide (250 – 500mg/d) may be effective as adjunctive therapy when started 7-10 days prior to the onset of menses and continued until bleeding stops[1, 65].

HEMICONVULSION HEMIPLEGIA EPILEPSY (HHE)

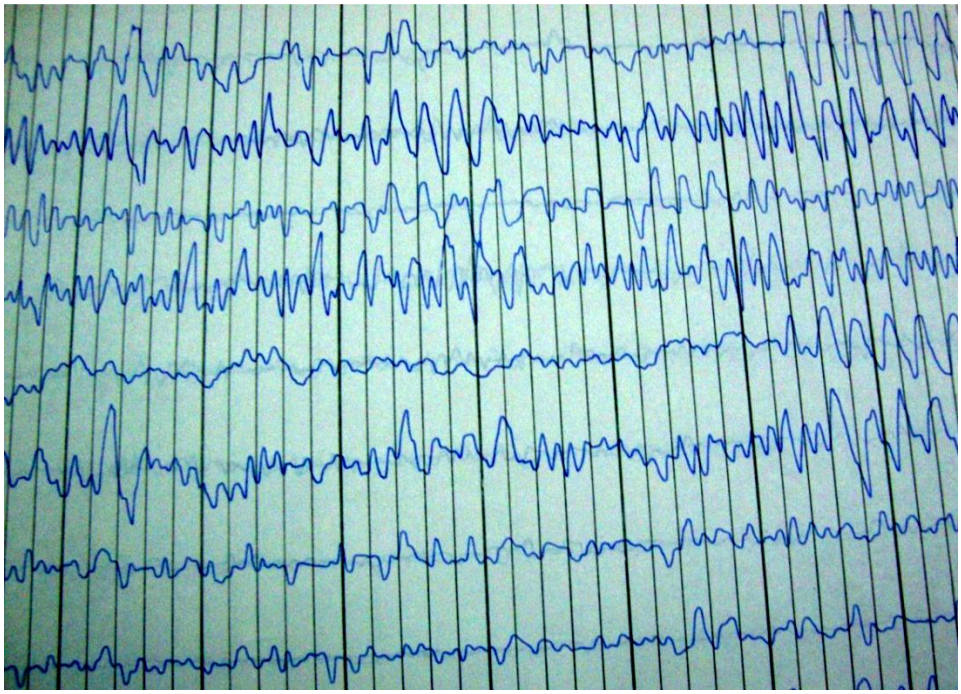
One patient had focal seizures followed by convulsions in one half of the body. Hemiconvulsion hemiplegia is often used to describe the initial stage of the syndrome[66,67&68]. Using ILAE classification criteria for the definition of epilepsy syndrome, HHE cannot be considered as a epilepsy syndrome. Usually this occurs during the first two years of life and very few patients present more than four years of age.

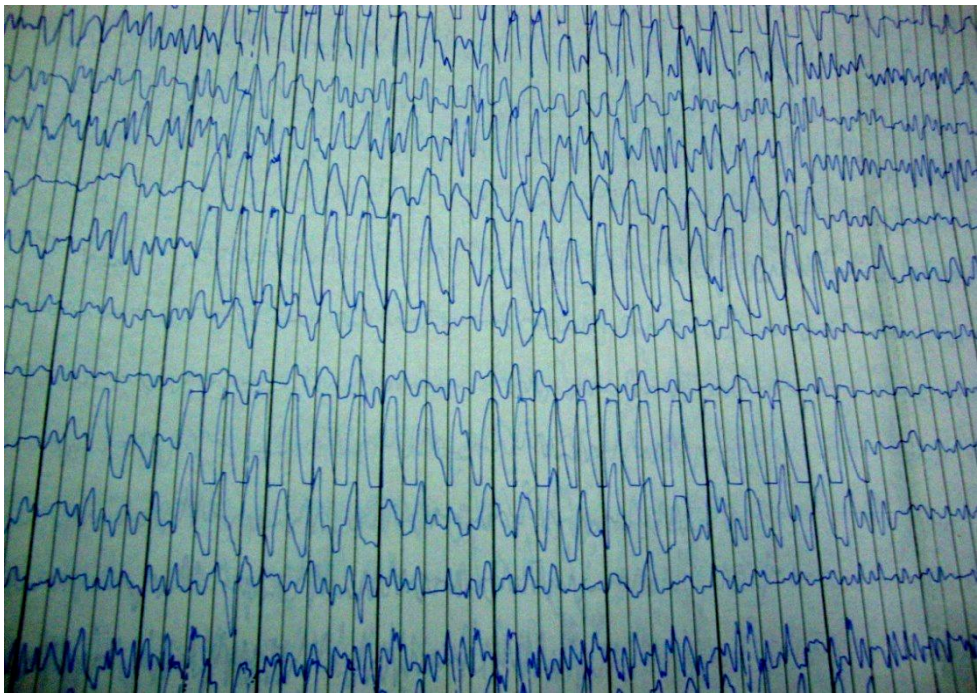
EEG FINDINGS

In this study, 61% of patients had normal EEG findings and 39% had abnormal EEG findings. Abnormal EEG findings were present in 39% of patients with GTCS where as 51% of patients with partial seizures had EEG abnormalities. Patients with partial seizures were found to have more EEG abnormalities than patients presented with GTCS (Tables 7, 8&13 Charts 11, 12 & 15).

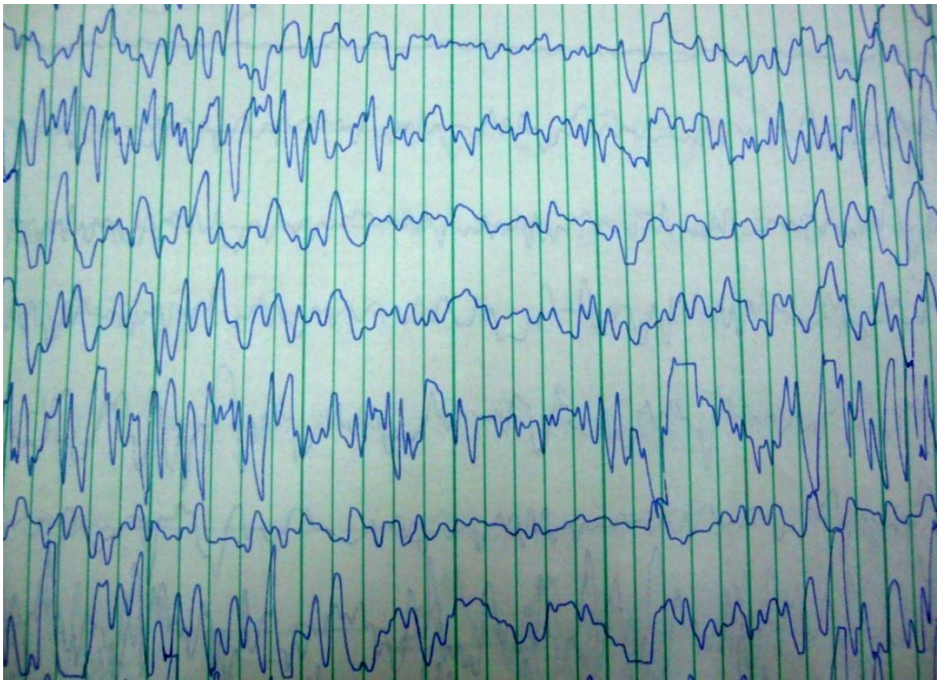
A single EEG tracing obtained during the inter-ictal state is abnormal to some degree in 30-50% of epileptic patients. This figure rises to 60-70% if patients are subjected to three or

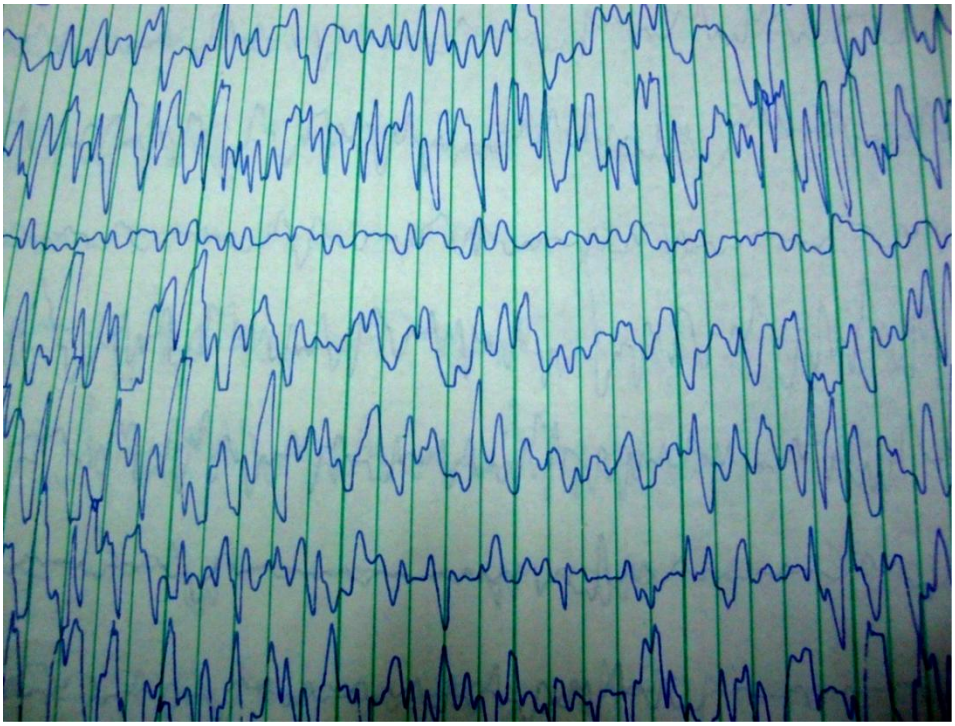
EEG – SPIKE AND WAVE PATTERN (RIGHT FOCAL BECOMING GENERALISED





MYOCLONUS (BURSTS OF SPIKES, POLY SPIKES AND WAVES)





more studies utilizing standard activating measures (hyperventilation, photic stimulation and sleep)[69].

BRAIN IMAGING AND EEG CORRELATION

In the study population 77 (38.5%) patients had normal imaging and EEG, 52 (26%) had normal imaging and abnormal EEG findings. 45 (22.5%) had abnormal imaging and normal EEG findings. 26 (13%) patients had abnormalities both in imaging and EEG (Table 11, Chart 16).

ETIOLOGY OF SEIZURES

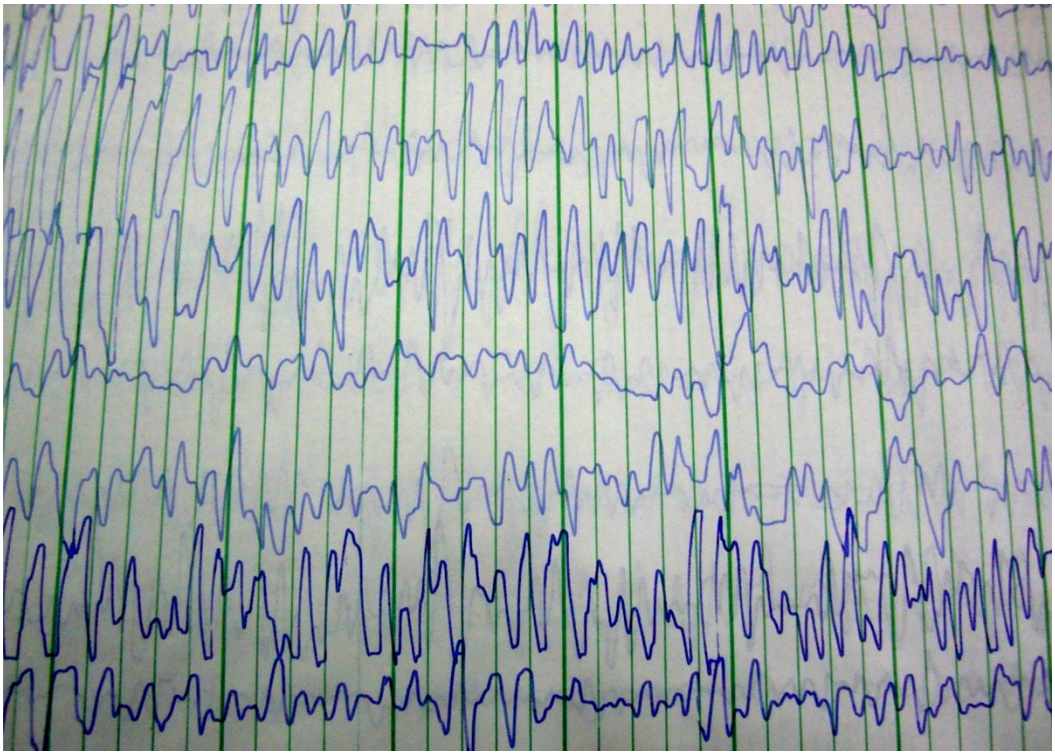
After complete evaluation with history, clinical findings, investigations and neuroimaging an etiology could not be arrived at in 121 (60.5%) patients (Table 12, Chart 18). Among them 104 patients had generalised seizures and the remaining 17 patients had partial seizures.

An etiology could be identified in 79 patients (Tables 12 & 14). Distribution of seizures with definite etiology is shown in chart 19&20.

The most common etiology identified was cerebrovascular accidents comprising 40.5%. Of this 30%, had cerebral infarction and 10.5% had haemorrhagic disease.

Granulomas were the next common etiology found in the study population. 11(14%) patients had CNS granulomas out of which 7 patients had Tuberculoma and 4 patients had NCC. Acute CNS infections and CVST were the next common etiologies found in the study (13% each). Brain tumors were found in 9 patients. 7 patients had primary tumors and 2 patients had secondaries in the brain. Cerebral atrophy was found in 7 patients.

GTCS



CONCLUSION

CONCLUSION

- Generalised seizures (70.5%) were more common than partial seizures (29.5%).
- GTCS was the commonest seizure type (68.5%).
- Among the partial seizures, partial seizures with secondary generalization (15.5%) was most common.
- Males (59.5%) were more in number than females in patients presenting with seizures.
- Patients aged more than 40 years constituted a significant proportion (33.5%) of patients presenting with seizure disorder. An organic cause for seizures could be identified in most of those patients.
- A secondary rise in incidence of new onset seizures occurred in patients aged more than 40 years.
- Imaging was normal in 64.5% of cases and abnormal in remaining cases.
- Neuroimaging was abnormal in 71% of patients who presented with partial seizures. Hence imaging plays a crucial role in the management of patients presenting with partial seizures.
- Abnormal EEG tracings were found only in 39% of patients presented with seizures in this study. 44% of cases with partial seizures had EEG abnormalities as against 37% in patients with generalised seizures. In most of the patients EEG was done at a later date after the seizures were well controlled. Studies indicate that a higher yield of abnormalities and more precise definition of seizure type can be obtained during the ictal or in the immediate post-ictal state.
- In 38.5% of the cases who presented with seizures both neuroimaging and EEG studies were normal.

- The study revealed that most common etiology for seizures that occurred in patients more than 40 years of age was cerebrovascular diseases, ischemic events more commonly causing seizures than hemorrhagic events.

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PROFORMA

PROFORMA:

NAME:

AGE: SEX:

ADDRESS:

EDUCATIONAL STATUS:

OCCUPATION:

Ht (cm):

Wt (kg):

BMI (kg/m^2):

CLINICAL PRESENTATION:

Presenting Complaints

Details about Seizures

- First attack
- Last attack
- Type of Seizures

(a) Generalized –

Tonic

Clonic

Tonic-Clonic

Absent

Atonic

Myoclonic

(b) Partial

Simple partial seizures (with motor, sensory, autonomic or psychic signs)

Complex partial seizures

Partial seizures with secondary generalization

- Part of body involved
- Duration
- Frequency
- Time of attack
- Precipitating factors
- Maximum period of freedom
- Altered level of consciousness
- Aura
- Automatism
- Bladder/bowel incontinence
- Post-ictal symptom

PAST HISTORY

- Hypertension, including PIH
- Diabetes mellitus
- Head injury
- Previous stroke
- Cardiovascular (RHD, MVP, IHD)
- Respiratory illness (COPD, PTB, BA)
- Psychiatric illness
- Jaundice
- H/O Febrile seizure

FAMILY HISTORY:

PERSONAL HISTORY

- Smoking
- Drugs
- Alcohol
- Snuff
- Other addictions
- Pork
- Sleep
- Sexual Promiscuity

ON EXAMINATION:

GENERAL EXAMINATION:

- BUILT
- BP: PR: RR: Temp:
- Anemia

- Jaundice
- Cyanosis
- Clubbing
- JVP
- Lymphadenopathy
- Pedal edema
- Vitamin deficiency
- Neurocutaneous markers

Cardiovascular system:

Respiratory system

Abdomen

Central nervous system

- Higher functions
- Cranial nerves
- Spinomotor system
- Bulk
- Tone
- Power
- Superficial reflexes
- Deep tendon reflexes
- Cerebellar signs
- Involuntary movements
- Sensory system
- Focal deficits
- Bladder/bowel
- Spine and cranium
- Meningeal signs

- Fundus

INVESTIGATIONS:

- CBC
- RBS
- RFT
- Serum Electrolytes
- QBC-MP
- HIV-ELISA
- VDRL
- CSF Analysis
- URINE ROUITNE EXAMINATION
- CHEST X-RAY
- EEG
- CT/MRI Brain

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

வலிப்பு நோயின் காரணங்களையும் அதன் தன்மைகளையும் கண்டறிவதற்கான ஓர் ஆய்வு

ஆராய்ச்சி நிலையம் : அரசு ஸ்டான்லி
மருத்துவமனை,
சென்னை - 600 001.

பங்கு பெறுபவரின் பெயர் :
பங்கு பெறுபவரின் எண் :

பங்கு பெறுவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்தகாரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன் இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத வழக்கத்திற்கு மாறான நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு இரத்தம், சிறுநீர், எக்ஸ்ரே, ஸ்கேன் பரிசோதனை செய்துகொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம்..... இடம் ..... தேதி

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....

ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளரின் பெயர் .....

ANNEXURE

MASTER CHART

ABBREVATION

SD	-	Seizure disorder
GTCS	-	Generalized Tonic Clonic Seizure
CPS	-	Complex partial seizure
SPS	-	Simple partial seizure
PSG	-	Partial seizure with secondary generalization
METS	-	Metastatic deposits
NCS	-	Neurocysticercosis
MR	-	Mental retardation
CSF	-	Cerebro spinal fluid
HSV	-	Herpes simplex virus
N	-	Normal
M	-	Male
F	-	Female
R	-	Right
L	-	Left
AU	-	Aura
PI	-	Post ictal
Fev	-	Fever
V	-	Vomiting
HA	-	Headache
Rel	-	Relevant
Inv	-	Investigations
CS	-	Childhood seizure

S/O	-	Suggestive of
TBM	-	TB Meningitis
Bac M	-	Bacterial Meningitis
MCA	-	Middle Cerebral Artery
PRE EPI	-	Previous episodes
LCS	-	Lymphocytic Pleocytosis
Sig	-	Significant
f/h	-	Family history
SCSD	-	Subcortical Seizure disorder
PIC	-	Post-ictal Confusion
Prolg	-	Prolonged
FND	-	Focal neurological deficit
Alt	-	Altered

CERTIFICATE FOR APPROVAL OF ETHICAL COMMITTEE

To

Dr.Namitha Narayanan, PG in M.D(GM)

Dear **Dr.Namitha Narayanan, PG in M.D(GM)**

The Institutional Ethics Committee reviewed and discussed your application for approval of the project entitled

"A study of clinical profile of seizures – current scenario in Govt.Stanley Hospital "

The following members of the ethics committee were present at the meeting held on 28.01.2010 at the Council Hall, Stanley Medical College, Chennai-1 at 10.00AM

Dr.C.B.Tharani, Director of Pharmacology,
Madras Medical College, Chennai-3 - Chairman of the Ethics Committee
Dr.S. Chitra, Vice-Principal,
Stanley Medical College, Chennai – 1- Member Secretary of the Ethics Committee

MEMBERS

Dr.Jayanthi
Prof.of Medical Gastroenterology
Dr.Madhavan
Prof.of Pharmacology
Dr.E.Dhandapani
Prof.of Medicine
Dr.Sujatha Sridharan
Prof.of Paediatrics
Thiru.Pachaiappan,
Junior Administrative Officer,
Thiru.A. Senthil Manoharan,
Advocate

We approve the project to be conducted in its presented form.

The Institutional Ethics Committee/Independent Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

Yours sincerely,

Chairs

Member Secretary,
Ethics Committee

Sl No	Name	Age	Sex	Sig History & Findings	Type of seizure	Rel inv	CT/MRI Findings	EEG	CS	PRE EPI
1.	Devi	16	F	PIC+	GTCS		CT- Normal	Normal	NIL	NO
2.	Ameena Banu	60	F	PIC+	GTCS		CT- Normal	Normal	NIL	NO
3.	Sasi Kala	29	F	Catamenial epilepsy	GTCS		MRI- Normal	Abnormal	NIL	YES
4.	Selvaprakasam	60	M	Prolg PIC+	(R)Focal with secondary generalization, status		CT-Diffuse hypodensity R temperooccipital region. MRI- Multifocal heterogenous nodule with perilesional oedema. Minimal contrast enhancement to both cerebral hemispheres- s/o METS	Abnormal	NIL	NO
5.	Kasthuri	47	F	Prolg PIC+	GTCS with status		CT- Normal	Abnormal	NIL	YES
6.	Devi	28	F	Aura+	(R) Focal with secondary generalization		CT- Normal	Abnormal	NIL	YES
7.	Marimuthu	60	M	Prolg PIC, FND+	GTCS, status		CT- A/C Infarct (L) MCA Territory	Abnormal	NIL	NO
8.	Munirudheen	20	M	Aura+, PIC+	CPS with secondary generalization		CT- Normal MRI- NORMAL	Normal	NIL	YES
9.	Hajira	34	F	F,HA,V PIC+	GTCS	CSF- s/oTB M	CT - NORMAL	Normal	NIL	NO
10.	Vijayalakshmi	58	F	PIC+	GTCS		CT- Normal	Normal	NIL	NO
11.	Padma	70	F	PIC+,FND+	(R) Focal with secondary generalization		CT- Chr Infarct (L) MCA Territory	Normal	NIL	NO
12	Dhanasekhar	29	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
13	Vijayakumar	14	M	PIC+	GTCS		CT – CP angle tumor, (L) Arachanoid cyst with hydrocephalus,	Abnormal	YES	YES
14	JOTHI	39	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
15	NITHYA	23	F	PIC+, Aura+	(R) FOCAL SEIZURE WITH SECONDARY GENERALIZATION		CT BRAIN- CALCIFIED GRANULOMA (L) FRONTO PARIETAL CORTEX AND (L) OCCIPITAL REGION. (s/o Tuberculoma)	Normal	NIL	NO
16	KAMALA KANNAN	20	M	PIC	GTCS		CT - NORMAL	Abnormal	NIL	YES
17	KARTHICK\	16	M	PIC+	GTCS		CT-NORMAL	Normal	NIL	NO
18	PUSHPA	30	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	YES
19	AMMU	29	F	PIC+	GTCS \		CT-NORMAL	Normal	NIL	YES

20	KAMAKSHI	48	F	PIC+ Papilledema	GTCS		CT- (L) PARASAGITTAL MENINGIOMA	Abnormal	NIL	NO
21	DURGADEVI	16	F	Aura	COMPLEX PARTIAL SEIZURE.		CT- NORMAL	Normal	NIL	NO
22	MURUGAN	29	M	f/h SD+, aura	(R) FOCAL WITH SECONDARY GENERALIZATION		CT - NORMAL	Abnormal	NIL	YES
23	PRABHAKAR	35	M	PIC+	GTCS		CT - NORMAL	Abnormal	NIL	NO
24	PARVATHY	17	F		MYOCLONIC SEIZURE.		CT- NORMAL	Abnormal	NIL	YES
25	MOHAN	45	M	Fever ,chills, alt sensorium	GTCS	QBC MP +	CT- NORMAL	Normal	NIL	NO
26	RAJKUMAR	32	M	Aura +, PIC+	(R) FOCAL with secondary generalization		CT- NORMAL	Abnormal	NIL	NO
27	SHANKAR	42	M	PIC+, FND+	GTCS		CT- (R)MCA INFARCT - Chr	Normal	NIL	NO
28	GOVINDARAJ	38	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
29	GANGADHARAN	45	M	PIC+, FND+	GTCS		CT- (R) Chr MCA INFARCT	Normal	NIL	NO
30	PRAKASH	13	M	PIC+, F, HA, V, Neck stiffness	GTCS	CSF- s/o BactM	CT- NORMAL	Normal	NIL	NO
31	BALAKRISHNAN	55	M	PIC+, FND+	GTCS		CT- Chr INFARCT IN (L) FRONTO-TEMPERO-PARIETAL REGION	Normal	NIL	NO
32	CHRISTIDAS	29	M	PIC+,	GTCS		CT- NORMAL	Normal	NIL	YES
33	SETHURAMALINGAM	27	M	Prolog PIC+ f/h SD+	GTCS, status		CT- NORMAL	Normal	NIL	YES
34	RAJA	60	M	Aura+, prolog PIC+, FND+	(R) FOCAL with secondary generalization, status		CT- (L) MCA INFARCT- Chr	Normal	NIL	NO
35	RAJKUMAR	15	M	Aura+	(R) FOCAL with secondary generalization		CT- NORMAL	Abnormal	YES	YES
36	ELUMALAI	40	M	F, HA, V, Aura+ pic+	(R) FOCAL with secondary generalization	CSF- s/o BactM	MRI- NORMAL	Normal	NIL	NO
37	YUVANTHI	33	F	PIC+, f/h SD+	GTCS		CT- NORMAL	Normal	NIL	NO
38	MOHAN KUMAR	38	M	PIC+	GTCS		CT -NORMAL	Abnormal	NIL	NO
39	SEKHAR	43	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
40	DEVI	28	F	Aura+	(R) FOCAL with secondary generalization		CT- NORMAL	Normal		

41	BHARATH	17	M	f/h SD+, aura+	CPS		CT- NORMAL	Abnormal	FS +	YES
42	VASANTHI	26	F	Aura+	CPS		CT- NORMAL	Abnormal	NIL	NO
43	SANDHYA	14	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
44	PUSHPA	30	F	PIC+	GTCS		CT- NORMAL	Normal	YES	yes
45	KAMALAMMA	70	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
46	KAVERI	37	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	YES
47	JESSINTHA	29	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	YES
48	ASMATHULLAH	37	M	AURA +	(R) FOCAL SEIZURE		MRI- (L) PARIETAL LOBE MENINGIOMA	Abnormal	NIL	NO
49	PAVITHRA	21	F	PIC+	GTCS		CT- NORMAL	Normal	YES	YES
50	PAUL DHARMARAJ	52	M	FND+ , PIC+, aura+	(L) FOCAL seizure with secondary generalization		CT- GLIOTIC CHANGES (R)TEMPERO-PARIETAL REGION	Normal	NIL	YES
51	PRABHAKAR	28	M	Aura +	(L) FOCAL seizure		CT- (R) PARIETAL CALCIFIED GRANULOMA (NCC)	Abnormal	NIL	NO
52	VINODH	15	M	Prolo PIC+ FND+,papilledema	GTCS, status		CT- Schwanoma frontal region	Abnormal	YES	YES
53	GUNASEKAR	28	M	PIC+, papilledema	GTCS		MRI- CORTICAL VENOUS SINUS THROMBOSIS	Normal	NIL	NO
54	AMEER KHAN	29	M	PIC+, papilledema, FND+	(R) FOCAL SEIZURE		MRI- (R) LATERAL, SIGMOID AND JUGULAR VENOUS SINUS THROMBOSIS, (L) FRONTO-PARIETAL CORTICAL AND SUBCORTICAL HAEMATOMMA	Normal	NIL	NO
55	BABU	51	M	PIC+, FND+ Papilledema	FOCAL SEIZURE FACE AND (L) UL		MRI,MRA AND MRV- SUPERIOR SAGITTAL BILATERAL TRANSVERSE AND SIGMOID SINUS THROMBOSIS	Normal	NIL	NO
56	SIGAMANI	35	M	PIC+, papilledema, FND+	GTCS		MRI, MRA AND MRV- VENOUS INFARCT IN (L) FRONTAL AND PARIETAL LOBE WITH SUPERFICIAL CORTICAL VEIN THROMBOSIS	Normal	NIL	NO.
57	RAJAMANI	20	F	Prolong PIC+, papilledema,	GTCS-, status		MRI,MRA AND MRV- SIGMOID SINUS THROMBOSIS (L).	Normal	NIL	NO

				FND+, post partum						
58	VAJITHA BEGUM	21	F	Post partum PIC+, papilledema, FND+	GTCS		MRI- BL PARIETAL INFARCT WITH DIFFUSE CEREBRAL OEDEMA, SUPERIOR SAGITTAL SINUS AND (R) TRANSVERSE SINUS THROMBOSIS.	Normal	NIL	NO
59	ELLAMMAL	28	F	PIC+, papilledema, FND+,post partum	FOCAL SEIZURE (L) UL		BL HIGH PARIETAL VENOUS INFARCT, (L) FRONTAL INFARCT , SUPERIOR SAGITTAL SINUS AND (R) TRANSVERSE SINUS THROMBOSIS.	Normal	NIL	NO
60	SELVI	25	F	PIC+, papilledema, FND+,post partum	GTCS		MRI, MRA AND MRV- VENOUS INFARCT (R) FRONTO-PARIETAL LOBE WITH THROMBOSIS OF SUPERIOR SAGITTAL SINUS , STRAIGHT SINUS,I NFERIOR CEREBRAL VEIN AND (L) TRANSVERSE SINUS.	Normal	NIL	NO
61	RAMACHANDRAN	27	M	PIC+, papilledema, aura+	(L) FOCAL SEIZURE		MRI, MRA AND MRV- ACUTE (R) FRONTAL HAEMARRHAGIC INFARCT , THROMBOSIS OF SSS	Normal	NIL	NO
62	YUVARAJ	26	M	PIC+	GTCS WITH MR		MRI- PITUTARY GLIAL HYPOINTENSITY	Abnormal	YES	YES
63	SHAHUL HAMEED	60	M	Prolong PIC+. FND +	GTCS, status		CT- (R) PARIETO-OCCIPITAL REGION HAEMORRHAGIC INFARCT- A/C	Normal	NIL	NO
64	ELANGO	15	M		Absence		CT- NORMAL	Abnormal	NO	YES
65	MUTHU	24	M	PIC+, f/h SD	GTCS		CT- NORMAL	Normal	NIL	NO
66	MUNIVEL	39	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
67	MUTHUSAMY	42	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
68	AMALDASS	60	M	PIC+, FND+	GTCS		CT- INTRA-CEREBRAL HAEMORRHAGE	Normal	NIL	NO
69	THIRUMURUGAN	15	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
70	KARTHICK	16	M	F,HA, V,	GTCS	QBC MP+	CT- NORMAL	Normal	NIL	NO
71	AMALA	23	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
72	AMEENA BANU	25	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
73	DINESH KUMAR	38	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
74	SUDHA	21	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO

75	RAJAKANNAN	23	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
76	AJAY	13	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
77	SURESH	13	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
78	KALAI	17	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
79	VIJAYAKUMAR	15	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
80	SAHAYAMARY	26	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
81	AMEER	14	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
82	JOTHY	23	F	Aura+	CPS		CT- NORMAL	Abnormal	NIL	NO
83	DIVYABHARATHI	19	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
84	GAYATHRI	13	F	Aura+ , f/h SD+	CPS		CT- NORMAL	Abnormal	NIL	NO
85	KAMALA	20	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
86	SANTHIYA	16	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
87	GUNASUNDARI	19	F	Aura+,	(L) FOCAL SEIZURE		CT- (R) Parietal Granuloma s/o Tuberculoma	Abnormal	NIL	NO
88	MEENAKSHI	20	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
89	SELVI	26	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
90	MANIKANDAN	I4	M	PIC+, f/h SD+	GTCS		CT - NORMAL	Abnormal	NIL	NO
91	RAMESH	30	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
92	KATHIRAVAN	33	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
93	AMUDHAVALLI	16	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
94	MURUGAN	29	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
95	ANITHA	15	F	Aura+	CPS		CT- NORMAL	Abnormal	NIL	NO
96	RABINDAR	25	M	PIC, f/h SD+	GTCS		CT- NORMAL	Abnormal	NIL	NO
97	SRINIVASAN	15	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
98	VIGNESH	17	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
99	SARASWATHI	19	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
100	VANITHA	32	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO

101	KUMAR	31	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
102	SUMAN	21	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
103	JENNIFAR	39	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
104	KONDALA RAO	28	M	PIC+	GTCS		CT - NORMAL	Normal	NIL	NO
105	SITTAIYAH	65	M	PIC+, FND+	GTCS		CT- (L) MCA INFARCT-Chr	Normal	NIL	NO
106	PARAMESWARI	24	F	PIC+, f/h SD+	GTCS		CT- NORMAL	Normal	NIL	NO
107	MOORTHY	26	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
108	PARVEEN	28	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
109	SOUNDARI	48	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
110	JAMUNA	25	F	PIC+, f/h SD+	GTCS		CT- NORMAL	Normal	NIL	NO
111	ALAMELU	34	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
112	GOPAL	30	M	Aura+	CPS		CT- NORMAL	Normal	NIL	NO
113	SHANTHI	23	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
114	NANDAKUMAR	39	M	PIC+, F. HA V, Alt sensorium	GTCS		CT- NORMAL	Normal	NIL	NO
115	MAHALAKSHMI	19	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
116	JANCY	29	F	Aura +	CPS		CT- NORMAL	Abnormal	NIL	NO
117	DURGA DEVI	30	F	PIC+, f/h SD+	GTCS		CT - NORMAL	Normal	NIL	NO
118	KULASEKHAR	28	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
119	RAMESH	25	M	PIC+, F, HA , V,	GTCS	CSF-LCS, HSV, IgM +	MRI – HYPODENSITY BL Temporal region s/o HSV Encephalitis	Normal	NIL	NO
120	KAVITHA	30	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
121	KAYAMMA BEE	48	F	PIC+	GTCS		CT - NORMAL	Abnormal	NIL	YES
122	ANTONY	30	M	PIC+	GTCS		CT - NORMAL	Normal	NIL	YES
123	DHANASEKHAR	24	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
124	SARAVANAN	13	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
125	SUDHAVARSHI	19	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
126	RAJESWARI	15	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	YES
127	RAJA	37	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO

128	JAYANTHY	13	F	FS	GTCS		CT- NORMAL	Normal	YES	YES
129	CHANDRASEKHAR	18	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
130	JAFAR	20	M	PIC+	GTCS		CT-NORMAL	Normal	NIL	NO
131	MUTHU KRISHNAN	36	M	PIC+	(L) FOCAL with secondary generalization		MRI – GLIOTIC CHANGES INVOLVING PCA TERRITORY	Abnormal	YES	YES
132	LOGESWARI	20	F	FS+	Myoclonus		MRI – NORMAL	Abnormal	YES	YES
133	KANNAN	35	M	Prolong PIC+	GTCS, status		CT- WELL DEFINED HYPO INTENSITY INVOLVING WHITE MATTER OF (R) FRONTAL REGION. MRI – S/O ASTROCYTOMA	Abnormal	NIL	NO
134	VADIVELU	20	M		MYOCLONUS		CT- NORMAL	Abnormal	NIL	YES
135	SANKAR	32	M	PIC+	GTCS		MRI- S/O NEUROCYSTICERCOSIS	Normal	NIL	NO
136	SARAN RAJ	20	M		(R) FOCAL PROGRESSING TO HEMICONVULSION		MRI- Atrophy of left cerebral hemisphere	Abnormal	YES	YES
137	MAHALINGAM	41	M	FND+	(L) FOCAL WITH SECONDARY GENERALIZATION		CT- (R) PARIETAL INFARCT-A/C	Normal	NIL	NO
138	PREMA	22	F	Aura+ Papilledema	(L) FOCAL with secondary generalization		MRI- TUBERCULOMA, BL PARIETAL LOBE, (L) TEMPORAL AND (R) OCCIPITAL REGION	Normal	NIL	NO
139	SAKTHIVEL	35	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
140	GOVIND	36	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
141	KANDASAMY	57	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	YES
142	KUPPAN	68	M	PIC+, FND+	GTCS		CT- CHRONIC INFARCT IN (L) PARIETAL REGION	Normal	NIL	NO
143	GAJENDRAN	51	M	FND+, PIC+	GTCS		CT- (R) ACUTE INFARCT CAPULO GANGLIONIC AREA WITH SORROUNDING OEDEMA	Abnormal	NIL	NO
144	VINAYAGAM	43	M	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
145	DEVAKI	58	F	FND+, PIC+	PARTIAL SEIZURE with secondary generalization		CT-Chr INFARCT (R) PARIETO-OCCIPITAL REGION	Normal	NIL	NO
146	GOVID SWAMY	43	M	aura+	CPS		CT- MUTIPLE RING ENHANCING LESION , S/O NEUROCYSTICERCOSIS	Normal	NIL	NO
147	VAJRAVEL	44	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
148	MARIAMMA	73	F	Aura + FND+	CPS		CT- CHRONIC INFARCT (R) PARIETAL CORTEX	Abnormal	NIL	NO

149	MUNIAMMAL	52	F	Aura+ , FND+	PARTIAL SEIZURES with secondary generalization		CT- CHRONIC INFARCT (L) FRONTO-PARIETAL CORTEX	Abnormal	NIL	NO
150	SHAJAHAN	58	M	PIC+, FND+	GTCS		CT- ACUTE HAEMORRHAGE (L) CAPSULO-GANGLIONIC REGION	Normal	NIL	NO
151	MOHAN RAJ	42	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
152	MOHAMED YUSAF	69	M	FND+	(L) FOCAL SEIZURES with secondary generalization		CT- ACUTE INFARCT (R)PARIETO-TEMPORAL REGION	Normal	NIL	NO
153	RAMASWAMY	53	M	FND+	CPS		CT- Chr INFARCT IN (L) PARIETAL CORTEX AND CORONA RADIATA	Normal	NIL	NO
154	MUTTHU	64	M	Aura+ , FND+, PIC+	PARTIAL SEIZURES with secondary generalization		CT- CHRONIC INFARCT (L) CAPSULO-GANGLIONIC REGION	Abnormal	NIL	NO
155	JAYARAMAN	72	M	FND+	PARTIAL SEIZURES with secondary generalization		CT- ACUTE INFARCT PARIETO-TEMPORAL CORTEX	Normal	NIL	NO
156	PANDIAN	42	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
157	VISHALATCHI	62	F	Aura+ , FND+	PARTIAL SEIZURES with secondary generalization		CT- CHRONIC HAEMORRHAGE IN (R) PARIETAL REGION	Abnormal	NIL	NO
158	THOMAS	56	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
159	RAMANATHAN	68	M	Aura+			CT- MASS IN PARIETO OCCIPITAL REGION- GLIOMA	Normal	NIL	NO
160	SHANMUGAN	39	M	PIC+, F, HA, Alt sensorium	GTCS		CT- NORMAL, CEREBRAL MALARIA, QBC MP- POSITIVE	Normal	NIL	NO
161	SELVAM	72	M	PIC+ dementia+	GTCS		CT- DIFFUSE CORTICAL ATROPHY	Normal	NIL	NO
162	NATARAJAN	66	M	PIC+	(L) PARTIAL SEIZURES with secondary generalization		CT- MULTIPLE HYPODENSE LESIONS (R) FRONTO-PARIETAL REGION-BRONCHOGENIC CARCINOMA WITH BRAIN SECONDARIES	Normal	NIL	NO
163	LAKSHMI	46	F		CPS		CT- (R) FRONTAL TUBERCULOMA	Normal	NIL	NO
164	SARADHA	50	F	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
165	PADMAVATHY	68	F	FND+	PARTIAL SEIZURES		CT- ACUTE HAEMORRHAGE	Abnormal	NIL	NO

					with secondary generalization		CAPSULI-GANGLIONIC REGION			
166	RENUKA	38	F	PIC+, FND+	(L)) PARTIAL SEIZURES with secondary generalization	Mx- +	CT- (R) FRONTO-PARIETAL TUBERCULOMA with peri-lesional edema	Normal	NIL	NO
167	VINAYAGAM	56	M	FND+	PARTIAL SEIZURES with secondary generalization		CT- ACUTE INFARCT IN (R) PARIETO-OCCIPITAL REGION	Normal	NIL	NO
168	CHINNA SWAMY	53	M	FND+	PARTIAL SEIZURES with secondary generalization		CT- INTRA CEREBRAL HAEMORRHAGE	Abnormal	NIL	NO
169	MANI	44	M	PIC+	GTCS	CSF- S/0 TBM	CT- NORMAL, TB- MENINGITIS, CSF ADA +,PROTEIN – INCREASED, SUGAR -DECREASED	Normal	NIL	NO
170	CHELLA DURAI	56	M	Alt sensorium	PARTIAL SEIZURES with secondary generalization		CT- (L) CHRONIC SUBDURAL HAEMATOMA	Normal	NIL	NO
171	PERIA SWAMY	54	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
172	PUNIYA KODI	52	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
173	CHANDRA	59	F	Aura+, FND+	CPS		CT- CHRONIC (L) FRONTO-PARIETAL IMFARCT	Normal	NIL	NO
174	SHANEENA BEE	54	F	Aura+ , FND+	CPS		CT- CHRONIC HAEMORRHAGE (R) FRONTO-PARIETO REGION	Normal	NIL	NO
175	RAMAKRISHNAN	32	M	Aura+	CPS		CT- (L) FRONTO PARIETAL TUBERCULOMA	Normal	NIL	NO
176	NEELA MEGAM	53	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
177	RAHMATULLAH	70	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
178	GANESHAN	35	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
179	LALITHA	43	F	PIC+, F, HA, Alt sensorium	GTCS		CT- NORMAL, CEREBRAL MALARIA FALCIPARUM +	Normal	NIL	NO
180	RAVANAIYYA	33	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
181	MEENAKSHI	74	F	PIC+	GTCS		CT- DIFFUSE CEREBRAL ATROPHY	Normal	NIL	NO
182	MANI MEGHALAI	59	F	Aura+	GTCS,		CT- NORMAL	Normal	NIL	NO
183	MARY	56	F	Aura+	SIMPLE PARTIAL SEIZURE,		CT- MENINGIOMA (L) PARASAGITTAL REGION	Normal	NIL	NO
184	GHOUSE	62	M	FND+	PARTIAL SEIZURES with secondary generalization		CT- CHRONIC INFARCT (L) PARIETAL CORTEX AND CORONA RADIATA	Abnormal	NIL	NO
185	SEETHA	35	F	Aura+	SIMPLE PARTIAL		CT- NORMAL	Normal	NIL	NO

					SEIZURE					
186	MUTHAIAH	50	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
187	JAMUNA	46	F	PIC+, F, HA	GTCS	CSF- LC, HSV Ig M +	MRI- HYPODENSITY (L) TEMPORAL REGION/ CSF LYMPHOCYTIC PLEOCYTOSIS/ HSV Ig M +	Abnormal	NIL	NO
188	SUNITHA	28	F	PIC+	GTCS		CT- GRANULOMA (NCC)	Normal	NIL	NO
189	KRISHNAN	51	M	Aura+	CPS,		CT- CHRONIC HAEMORRHAGE IN (R) PARIETAL REGION	Abnormal	NIL	NO
190	KALPANA	25	F	PIC+	GTCS		CT- NORMAL	Abnormal	NIL	NO
191	RAJESH	22	M	Aura+	(L) FOCAL SEIZURE,		CT- TUBERCULOMA	Abnormal	NIL	NO
192	PARTHIBAN	19	M	PIC+	GTCS		MRI- NORMAL	Abnormal	NIL	YES
193	RAJENDRAN	37	M	PIC+	GTCS		CT- NORMAL	Abnormal	YES	YES
194	JAMES	45	M	PIC+, FND+	GTCS		CT-(R) MCA INFARCT Chr	Normal	NIL	NO
195	ANTONY RAJ	38	M	PIC+	GTCS		CT- NORMAL	Normal	NIL	NO
196	JAYANTHI	25	F	Aura+	(R) FOCAL with secondary generalization		CT- NORMAL	Normal	NIL	NO
197	GANGA	27	F	FND+	(L) FOCAL with secondary generalization		CT- (R) PARIETO-OCCIPITAL INFARCT- A/C	Normal	NIL	NO
198	MANIKIAVEL	70	M	FND+	(L) FOCAL with secondary generalization		CT- Chr (R) TEMPERO-PARIETAL INFARCT	Abnormal	NIL	NO
199	SARAVANAN	20	M	PIC+	GTCS		CT- ENLARGED CISTERNA MAGNA	Abnormal	NIL	YES
200	DAMODHARAN	35	M	PIC+, HA, papilledema+	GTCS		MRI- DURAL SINUS THROMBOSIS WITH CVT WITH PARENCHYMAL HAEMORHAGE	Normal	NIL	NO