DISSERTATION ON

A STUDY OF CLINICAL AND ETIOLOGICAL PROFILE OF FOCAL SEIZURES

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations
for the award of the degree of

M.D. IN GENERAL MEDICINE

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THANJAVUR - 613 004

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

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CERTIFICATE

This is to certify that this dissertation entitled "**A STUDY OF CLINICAL AND ETIOLOGICAL PROFILE OF FOCAL SEIZURES**'' is the bonafide original work of **Dr. REVATHY.V** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu Dr.M.G.R. Medical University to be held in APRIL - 2016. The period of the study was from January – 2015 to August -2015.

Prof.Dr.K.NAGARAJAN, MD.,
Unit Chief M-I
Dept. Of Internal Medicine,
Thanjavur Medical College,
Thanjavur – 613004.

Prof.Dr.K.NAGARAJAN, MD.,
Head Of the Department,
Dept. Of Internal Medicine,
Thanjavur Medical College,
Thanjavur – 613004.

Prof.Dr.M.SINGARAVELU M.D.(Paed),DCH,
DEAN I/C,
Thanjavur Medical College,
Thanjavur – 613 004.

DECLARATION

I, **Dr.V.REVATHY,** solemnly declare that dissertation titled **"A STUDY OF CLINICAL AND ETIOLOGICAL PROFILE OF FOCAL SEIZURES"** is a bonafide work done by me at Thanjavur Medical College and Hospital during January 2015 to August 2015 under guidance and supervision of my unit chief **Prof.Dr.K.NAGARAJAN, M.D.,** Professor and head of the Department of Medicine.

This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

Place: Thanjavur

Date:

(Dr. V.REVATHY)
Postgraduate Student,
M.D. in General Medicine,
Thanjavur Medical College,
Thanjavur - 613 004.

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Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA - 613001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

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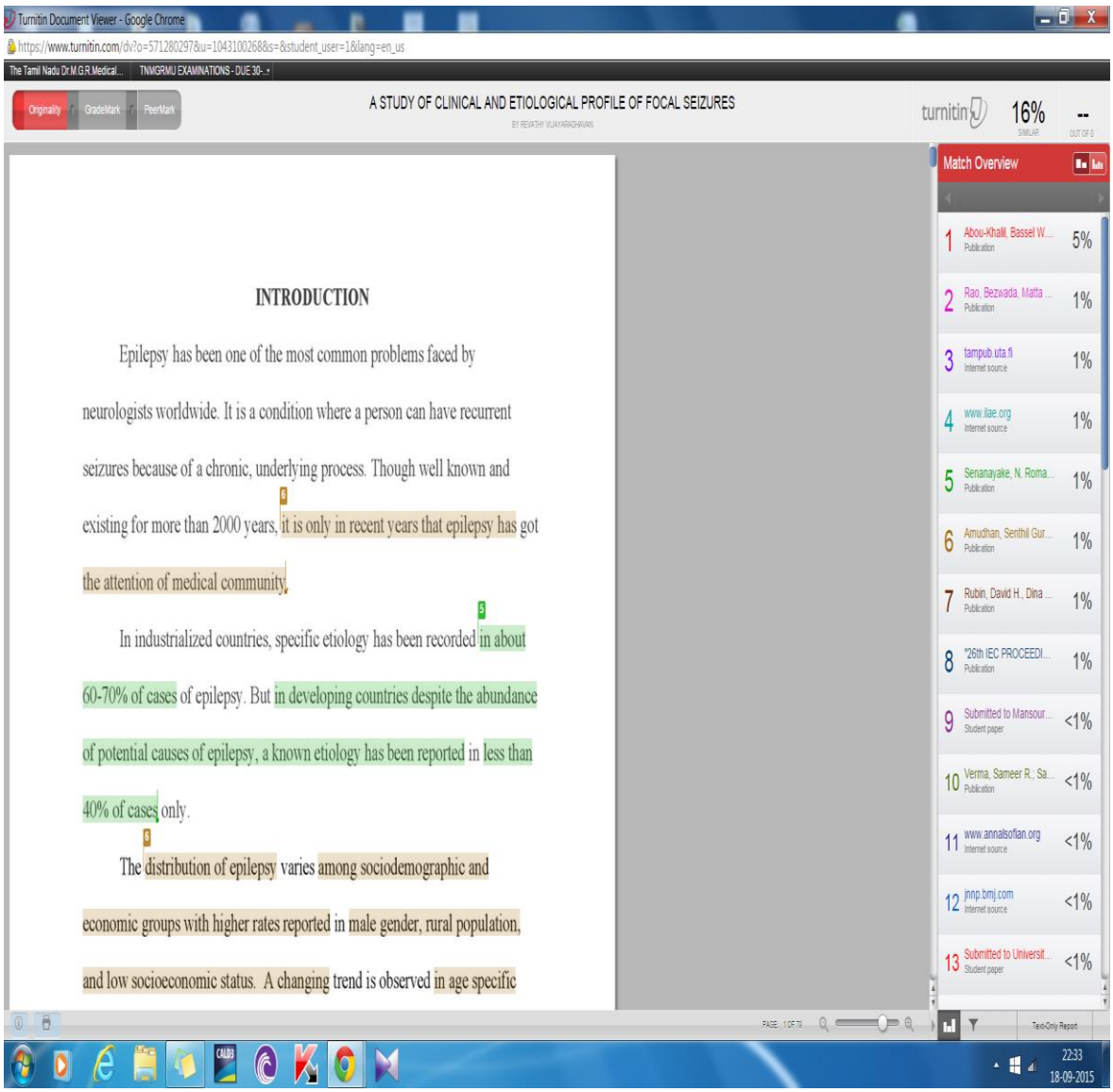
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In industrialized countries, specific etiology has been recorded in about 60-70% of cases of epilepsy. But in developing countries despite the abundance of potential causes of epilepsy, a known etiology has been reported in less than 40% of cases only.
The distribution of epilepsy varies among sociodemographic and economic groups with higher rates reported in male gender, rural population, and low socioeconomic status. A changing trend is observed in age specific
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INTRODUCTION

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The distribution of epilepsy varies among socio-demographic and economic groups with higher rates reported in male gender, rural populations, and low socioeconomic status. A changing trend is observed in age specific occurrence of epilepsy with a preponderance towards older age group.

The profile of epilepsy also varies across different cultures, and literature shows that in western countries about two-third of the cases are partial seizures. On the contrary, Indian studies report a reverse trend where generalized seizures constitute more than 70% of all seizures. The lower frequency of partial seizures in developing countries is attributed to the fact that they may be underreported in studies that use inadequate screening questionnaires.

ABBREVIATIONS

EEG: Electroencephalograph

CT: Computed tomography

MRI: Magnetic Resonance Imaging

PET: Positron emission Tomography

SPECT: Single photon emission computed tomography

WHO: World Health Organization

FDA: Food and Drug Administration

CNS: Central nervous system

ILAE: International League Against Epilepsy

GABA: Gamma amino butyric acid

CSF: Cerebrospinal fluid

PCR: Polymerase chain reaction

MEG: Magneto encephalography

MSI: Magnetic source image

FLAIR: Fluid attenuated inversion recovery

ATP: Adenosine triphosphate

ELISA: Enzyme linked immunosorbent assay

ELITB: Enzyme linked immunoelectron transfer blot

NCC: Neurocysticercosis

HIV: Human immunodeficiency virus

CVT: Cerebral venous thrombosis

AED: Antiepileptic drug

EPC: Epilepsia partialis continua

HT: Hypertension

DM: Diabetes mellitus

CKD: Chronic kidney disease

CLD: Chronic liver disease

TB: Tuberculosis

DNET: Dysembryoplastic neuroepithelial tumours

CVA: Cerebrovascular accidents

MTLE : Mesial Temporal Lobe Epilepsy

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INTRODUCTION

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In industrialized countries, specific etiology has been recorded in about 60-70% of cases of epilepsy. But in developing countries despite the abundance of potential causes of epilepsy, a known etiology has been reported in less than 40% of cases only[2].

The distribution of epilepsy varies among sociodemographic and economic groups with higher rates reported in male gender, rural population, and low socioeconomic status. A changing trend is observed in age specific occurrence of epilepsy with a preponderance towards older age group.

The profile of epilepsy also varies across different cultures, and literature shows that in western countries about two-third of the cases are partial seizures[3]. On the contrary, Indian studies report a reverse trend where generalized seizures constitute more than 70% of all seizures[4]. The lower frequency of partial seizures in developing countries is attributed to the fact that they may be underreported in studies that use inadequate screening questionnaires[5].

The etiology of seizures is directly linked to the type of the seizure[6]. In case of partial seizures, localisation of abnormalities is from 28% to 80% as observed in various studies. A proper history is essential to identify the type of seizure. Since focal seizures are more often associated with focal cerebral lesions, successful treatment of patients depends on identifying the cause and proper treatment thereby reducing morbidity and mortality.

Hence, this study is done to evaluate the clinical profile and etiology of new onset focal seizures in patients aged more than 12 years.

AIM OF THE STUDY

1. To study the clinical profile of focal seizures
2. To evaluate the etiological profile of focal seizures

REVIEW OF LITERATURE

HISTORICAL OVERVIEW:

Seizures was first mentioned in ancient Akkadian texts, the Sakkiku around 2000 BC which used the terms 'antasubba' and 'miqtu' for describing seizures[7]. Epilepsy was also described in texts from ancient Babylonians where patients with epilepsy were considered to be affected by evil spirits. Indian texts described epilepsy with premonitory symptoms and loss of memories as 'Abasmara'. Until around 200 years before, epilepsy was considered to be a disorder of supernatural origin due to demonic possession.

The first hallmarks in the history of epilepsy are the Hippocratic texts from fifth century B.C. which questioned the divine origin of the disease. Hippocrates gave an alternate thought that epilepsy is an ordinary medical condition with a natural cause[8].

John Hughlings Jackson, a British neurologist, considered the father of modern epileptology was the first one who studied epilepsy based on anatomy and pathology[9]. He reported that convulsions occurred due to a discharging lesion from damage to nerve cells and correlated the ictal behaviour with brain anatomy.

The description about partial seizure was first described by Bravais in 1827, by Jackson in 1860s and by Charcot as partial seizures in 1870s. Charcot called them Bravais-Jackson seizures. Though Bravais was the first to describe this type of seizure where convulsions was restricted to one side of the body which he called 'hemiplegic seizures', Jackson took this to a higher level and reasoned out that there should be a localized representation for this in cerebral cortex based on clinicopathologic

correlations[10]. Fritsch and Hitzig confirmed this experimentally in dogs when electrical stimulation of motor cortex gave rise to focal muscular contractions[11].

The human Electroencephalogram (EEG) discovered by the German Psychiatrist Hans Berger in 1929 made clear the presence of electrical discharges in brain and localisation of the site of epileptic discharges to certain extent[12]. EEG plays an important role in diagnosing and managing seizure disorders in addition to the various imaging techniques developed over the last 30 years.

EPIDEMIOLOGY

INCIDENCE:

According to the World Health Organization (WHO), there are 50 million people with epilepsy worldwide[13]. 80% of them reside in developing countries. Epilepsy was estimated to constitute 0.5% of the global burden of disease[14].

There is regional variation in incidence of epilepsy worldwide. Incidence is high in Africa and Latin America where it exceeds 100 per 1,00,000[15]. Among the few studies conducted in developing countries, two studies done in Mariana Islands have reported an annual incidence of 30-47.3 per 100 000 population[16,17].Partial seizures were found to constitute around 60% of seizures in developed countries[18]. In developing countries the incidence was relatively higher around 80%, which could be attributed to the higher rate of CNS infections[19].

PREVALENCE:

Prevalence is the measure of total number of persons with epilepsy at a specific moment of time. It is expressed as the number of persons with epilepsy per 1000 population. The prevalence of epilepsy in industrialized countries is found to be about 3-9 per 1000 population. Several African and Latin American countries showed prevalence more than 3-9 per 1000[20].

In a study in the United Republic of Tanzania, a Bantu population showed a prevalence of 20 per 1000 with a family history of epilepsy in 76.6% which supported

a strong genetic basis in this tribe[21,22]. The prevalence of epilepsy was also found to increase with age in many studies, reaching a peak in the third and fourth decades of life. In some studies in Ethiopia, Nigeria, and Sri Lanka the highest rates occurred in the second decade of life[23].

In India, there are more than 10 million people with epilepsy constituting a prevalence of 1%[24]. Rural population make up a prevalence of 1.9% while urban population constitutes only 0.6%[25,26].

DEFINITION

SEIZURES:[1]

A Seizure is a paroxysmal event due to abnormal excessive hypersynchronous neuronal activity in the brain.

EPILEPSY:

Epilepsy is a clinical phenomenon characterised by atleast two unprovoked seizures separated by a minimum duration of 24 hours. The word epilepsy comes from a Greek word meaning "to seize upon" or "taking a hold of".

OPERATIONAL CLINICAL DEFINITION OF EPILEPSY:[27]

Epilepsy is defined by any of the following:

1. At least two unprovoked seizures occurring >24 h apart
2. One unprovoked seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years
3. Diagnosis of an epilepsy syndrome

Epilepsy is considered resolved for those with an age-dependent epilepsy syndrome but who are now past the applicable age or when seizure-free for the last 10 years, without antiepileptic drugs for the last 5 years.

CLASSIFICATION OF SEIZURES:

Seizures can be classified as:

1) Based on etiology – primary (idiopathic) or secondary (symptomatic)

2) Based on site of origin – generalised or focal

3) Based on their frequency – isolated, cyclic or repetitive

INTERNATIONAL LEAGUE AGAINST EPILEPSY (ILAE)

CLASSIFICATION OF EPILEPTIC SEIZURES, 1981[28]

I. PARTIAL (FOCAL) SEIZURES

A. Simple partial seizures

B. Complex partial seizures

C. Partial seizures evolving to secondarily generalized seizures

II. GENERALIZED SEIZURES

A. Absence seizures

B. Myoclonic seizures

C. Clonic seizures

D. Tonic seizures

E. Tonic-Clonic seizures

F. Atonic seizures

III. UNCLASSIFIED EPILEPTIC SEIZURES

ILAE's CLASSIFICATION OF EPILEPSIES AND EPILEPTIC SYNDROMES:

Epileptic syndromes include constellations of epileptic seizures and concurrent or serially linked symptoms and signs. Epilepsies and epileptic syndromes are classified according to localisation into focal or generalized, which are in turn classified into idiopathic, symptomatic and cryptogenic (unclassified/mixed) syndromes.

1. Idiopathic epilepsy syndromes (focal or generalised)
    a. Benign neonatal convulsions
        i. Familial
        ii. Nonfamilial
    b. Benign childhood epilepsy
        i. With central midtemporal spikes
        ii. With occipital spikes
    c. Childhood /juvenile absence epilepsy
    d. Juvenile myoclonic epilepsy
    e. Idiopathic, otherwise unspecified
2. Symptomatic epilepsy syndromes (focal or generalised)
    a. West syndrome
    b. Lennox gastaut syndrome

c. Early myoclonic encephalopathy

d. Epilepsia partialis continua

e. Acquired epileptic aphasia

f. Temporal lobe epilepsy

g. Frontal lobe epilepsy

h. Post traumatic epilepsy

i. Other symptomatic epilepsy

3. Other epilepsy syndrome of uncertain or mixed classification

a. Neonatal seizures

b. Febrile seizures

c. Reflex seizures

d. Other unspecified

FOCAL SEIZURES:

The definition of focal seizures is updated as "originating within networks limited to one hemisphere". Though the origin may be broadly distributed, it is still confined to one hemisphere. Only limited cases of focal seizures can be localised based on clinical data alone. Along with scalp and intracranial EEG recording and MRI, the localization can be done reasonably accurately.

The ILAE Commission on Classification and Terminology, 2005-2009 has provided an updated approach to classification of seizures, where the term partial

seizures has been replaced by focal seizures. The term simple partial seizures and complex partial seizures are eliminated and focal seizures are classified as:

1. Focal seizures without dyscognitive features
2. Focal seizures with dyscognitive features
3. Focal seizures evolving into generalized seizures

FOCAL SEIZURES WITHOUT DYSCOGNITIVE FEATURES

Focal seizures without dyscognitive features, previously referred as Simple Partial Seizures is a type of focal seizure where the consciousness is not impaired. This is divided into four major categories:

1. With motor symptoms
2. With somatosensory or special sensory symptoms.
3. With autonomic symptoms or signs
4. With psychic symptoms

Focal motor seizures

The hallmark of focal motor seizures is focal motor activity which may be expressed as clonic, tonic, postural or phonatory activity. This type of seizure arises due to a focal discharging lesion in frontal lobe. The most common type arises from the supplementary motor area. This usually manifests as turning movement of the head and eyes to the side opposite the irritative lesion, mostly associated with tonic extension of limbs. This forceful sustained deviation of the head and eyes, sometimes with entire body is referred as versive seizures. Usually this occurs opposite to the side of irritative focus[29].

The characteristic features which may be seen in focal motor seizures are the abnormal motor movements which may begin in fingers and gradually progress to involve other areas. This phenomenon was described by Hughlings Jackson and known as Jacksonian march. Patients may also develop localized paresis involving the region for minutes to many hours which is known as Todd's paralysis. In rare instances, focal motor seizures may continue for hours to days and individual jerks occur no more than 10 seconds apart which is termed "epilepsia partialis continua", which may be refractory to medical therapy[30]. This usually occurs due to lesions involving the sensorimotor cortex due to stroke, tumor, trauma or metastasis[31].

The high incidence of movements involving hands, face and toes is probably due to disproportionately large representation of these parts in the cortex. The focus of excitation is at or near the motor cortex, Brodmann area 4. If there is associated sensory symptom, postrolandic area may also be involved. Lesions confined to the motor cortex are associated with clonic contractions while those confined to premotor cortex are associated with tonic contractions of limbs. Dystonic and choreoathetotic posturing are found in medial frontal lesions. Brief aphasic disturbance termed ictal aphasia may be seen when seizure discharges also involves language areas of cortex.

Focal sensory seizures

Focal sensory seizures were defined by Penfield and Jasper separately from autonomic and psychic symptoms[32] and they classified these sensory seizures as[33]

Somatosensory

Visual

Auditory

Olfactory

Gustatory

Vertiginous

Somatosensory: This usually presents as numbness, tingling, or pins-and-needles feeling and sometimes as crawling sensation. This may be focal or a marching sensation may occur from one part to other. The discharge usually occurs from the postcentral gyrus and in majority, onset occurs in lips, fingers or toes. Peculiar body sensations may occur from discharges in supplementary motor area.

Visual seizures are relatively rare but can be localized to striate cortex of occipital lobe. They may present as flashing lights, darkness or sparks, colorless or may be colored, may be stationary or moving. The most commonly reported colour was found to be red followed by blue, green and yellow[34]. While elementary visual hallucinations and visual loss are characteristic of occipital lobe epilepsy[35], more complex and formed hallucinations are features of temporal lobe lesions.

Auditory seizures are infrequent and if present are described as buzzing or drumming or a roaring sound in ears and are mapped to lesions in superior temporal convolution. Auditory hallucinations usually represent a psychotic disorder.

Olfactory seizures, called uncinate fits presents with an aura of disagreeable odour and the localization is invariably in the inferior or medial parts of temporal lobe involving the uncinate gyrus.

Gustatory hallucinations are seen in cases of temporal lobe diseases; may be associated with salivation and thirst sensation.

Vertiginous sensations described as dizziness or unsteadiness may on rare occasions present as a first symptom of seizure. Though the lesion may be localized to

superoposterior temporal region, this symptom has many different connotations and often is of little diagnostic value.

Focal autonomic seizures

Autonomic phenomena constitute around one third of partial seizure symptoms. 30% of simple partial seizures were classified as autonomic in a study conducted by Devinsky et al, out of which 33% was associated with motor seizures[36]. The most common autonomic manifestation described is abdominal sensations particularly in mesial temporal lobe epilepsy[37]. This is described as a rising sensation from epigastrium and may be associated with nausea, fear, pain and hunger. Ictal vomiting may be seen when discharges arise from opercular region which may be frequently misdiagnosed as an organic gastrointestinal disorder[38]. Abdominal pain may occur as the sole manifestation of seizures in children.

Palpitations and chest pain are the next common autonomic symptoms, usually associated with sinus tachycardia. Other symptoms like perspiration, urinary urgency or incontinence, flushing, cyanosis, lacrimation, hyperventilation may be seen. Genital symptoms like erotic feelings and orgasm are infrequent.

Psychic seizures[39]

Psychic symptoms may be perceptual hallucinations/illusions in the form of visual, auditory or olfactory; Odd internal feelings such as deja vu or jamais vu; Emotional changes like fear, sadness, pleasure, sexual arousal or anger. Cognitive changes like changes in reality and depersonalization or forced thinking may be associated. Psychic seizures are predominantly found in mesial temporal lobe epilepsy.

FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES

Focal seizures with dyscognitive features, previously referred as Complex Partial Seizures is a type of focal seizure with impairment of consciousness

The seizures mostly begin with an aura which is stereotypic for the patient. The ictal phase often starts with a sudden behavioural arrest or motionless stare, which marks the onset of the period of impaired awareness. The behavioural arrest is frequently accompanied by automatisms, which are involuntary, automatic behaviours with a wide range of manifestations. Automatisms may present as very basic behaviours such as chewing, lip smacking, swallowing, or "picking" movements of the hands, or more elaborate behaviours such as an emotional display or running. The seizure is typically followed by post ictal confusion with transition to a fully conscious state occurring after a few seconds to an hour. The seizures most commonly arise from the temporal lobe followed by frontal lobe.

FOCAL SEIZURES EVOLVING INTO GENERALIZED SEIZURES

These seizures may start as focal seizures with or without impairment of consciousness. The transition to secondary generalization usually involves versive head turning in a direction contralateral to the hemisphere of seizure onset and focal or lateralized tonic or clonic motor activity[40]. The generalized tonic phase is asymmetrical with flexion on one side and extension on the other. This has been named as figure of 4 posturing[41]. There is gradual evolution from tonic to clonic activity with a tremulous or vibratory phase with high frequency tremors. Clonic activity then decreases over time with longer intervals between jerks. Once the clonic

activity stops, the individual becomes limp with a loud snoring referred as stertorous respiration.

AURA:[42]

The term aura was introduced by Galen. Aura is a purely subjective sensation which is of short duration lasting for few seconds to few minutes. In case of simple partial seizures, the aura may constitute the entire seizure while it may be the initial symptom followed by loss of consciousness in a complex partial seizure.

Aura symptoms can be categorized as:[43]

Epigastric auras

Autonomic auras

Emotional auras

Vestibular auras

Psychic auras

Visual auras

Somatosensory auras

Dysphasic auras

Olfactory auras

Auditory auras

Whole body sensations

Cephalic sensations

Uncategorized

COMMON FOCAL SEIZURE PATTERNS

TYPE OF SEIZURE	LOCALIZATION
SOMATIC MOTOR	
Jacksonian(focal motor)	Prerolandic gyrus
Masticatory, salivation, speech arrest	Amygdaloid nuclei
Head and eye turning with arm movements	Supplementary motor cortex
SOMATIC AND SPECIAL SENSORY	
Somatosensory	Contralateral postrolandic
Unformed images, lights, patterns	Occipital
Auditory	Heschl gyri
Vertiginous	Superior temporal
Olfactory	Mesial temporal
Gustatory	Insula
Visceral	Insular, Orbitofrontal cortex
FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES	
Formed hallucinations	Temporal neocortex or amygdala, hippocampus
Affective states	Temporal
Automatism	Temporal and frontal

SYNDROMES ASSOCIATED WITH FOCAL SEIZURES

BENIGN FOCAL EPILEPSY OF CHILDHOOD:

Benign Epilepsy with centrotemporal spikes, Rolandic Epilepsy

This is the most common epileptic syndrome in children. Usual age of onset is between 5 and 9 years. This constitutes 15 to 25% of childhood epilepsies. Genetic factors play a role with autosomal dominant inheritance with variable penetrance. Presents as focal onset, nocturnal, tonic clonic seizures involving one side of face; less often one arm or leg may be involved. The diagnosis depends on clinical presentation and EEG. The typical EEG abnormality is central mid temporal blunt sharp waves activated in sleep. Seizures are readily controlled with a single antiepileptic drug and self limiting gradually disappearing during adolescence.

RASMUSSEN SYNDROME:

This is characterized by intractable focal motor seizures with frequent episodes of focal status or epilepsia partialis continua on one side of the body. Usually children affected are between 6 and 10 years of age. The disease progression leads to hemiplegia and focal brain atrophy. The disease is associated with intellectual decline and behavioural disturbances. An autoimmune hypothesis is postulated with the presence of autoantibodies to Glutamate receptor 3(GluR3). Imaging shows progressive hemiatrophy. The seizures are usually refractory to medical therapy. The use of high dose corticosteroids have proved beneficial in some cases when started early within the first year of the disease. Hemispherectomy is considered for refractory seizures when the disease is extensive and unilateral.

FAMILIAL MESIAL TEMPORAL LOBE EPILEPSY:

Familial MTLE is a benign condition, first identified in twins. The most prominent aura is déjà vu with frequent focal seizures without dyscognitive features, infrequent focal seizures with dyscognitive features and rare secondarily generalized seizures. Prior history of febrile seizures are uncommon and MRI is normal with no hippocampal sclerosis. This is very responsive to medical treatment. Familial MTLE is probably polygenic in inheritance, although no gene mutation has been identified so far.

MESIAL TEMPORAL LOBE EPILEPSY:

The seizures are typically focal seizures with dyscognitive features with aura and automatisms. The most common aura is an epigastric sensation. Most common presentation is in early childhood or late adolescence. Patients usually have a history of antecedent complex febrile seizures in upto 80%[44]. The febrile seizures are usually complex and prolonged. Memory impairment is present with longer duration of seizures. MRI shows hippocampal sclerosis with decreased volume and increased signal. Seizures are refractory to medical therapy with excellent response to surgery. 60 to 80% of patients become seizure free after temporal lobectomy.

PANAYIOTOPOULOS SYNDROME

This syndrome presents as focal seizures with dyscognitive features predominantly with autonomic manifestations mainly ictal vomiting, altered responsiveness and deviation of eyes to one side. Onset is between 1 and 14 years of age with a peak at 4 to 5 years. Seizures predominate during sleep. EEG shows multifocal spikes with posterior predominance. Though the seizures may be alarming, they are infrequent and associated with good prognosis. Remission usually occurs in 1 to 3 years.

ETIOLOGY OF SEIZURES ACCORDING TO THE AGE GROUP

NEONATES: Perinatal hypoxia

Developmental disorders

Genetic disorders

Acute CNS infection

Intracranial haemorrhage and trauma

Hypoglycemia, hypocalcemia, hypomagnesemia

Pyridoxine deficiency, biotinidase deficiency

Drug withdrawal

INFANTS AND CHILDREN:

Genetic disorders

Developmental abnormalities

Febrile seizures

Trauma

CNS infection

Idiopathic

ADOLESCENCE (12-18YEARS):

Genetic disorders

CNS infection

Trauma

Tumour

Illicit drug abuse

Idiopathic

YOUNG ADULTS (18-35 YEARS):

Trauma

Tumour

Illicit drug abuse

Alcohol withdrawal

Idiopathic

OLDER ADULTS (>35YEARS):

Alcohol withdrawal

CNS infections

Cerebrovascular diseases

Metabolic disorders (uremia, hypoglycemia,

Hyperglycemia, hyponatremia, hepatic failure)

Degenerative disorders

Tumour

Drug induced

Idiopathic

MECHANISMS OF SEIZURES:

Focal seizures begin in a very discrete region of cerebral cortex. The burst activity in the neurons causing the seizure occurs due to a relatively long lasting depolarization of the neuronal membrane due to calcium influx intracellularly, which leads to opening of voltage dependent sodium channels resulting in influx of sodium and generation of action potentials repetitively. This is followed by a hyperpolarization mediated by GABA or K+ channels.

When the focal seizure activity spreads slowly into the surrounding regions, there is recruitment of surrounding neurons with sufficient activation which leads to propagation of currents into adjacent areas.

The recruitment of neurons occurs by various mechanisms like:

1) Blunting of hyperpolarisation because of increase in extracellular potassium (K^+)
2) Calcium accumulation in presynaptic terminals causing release of neurotransmitters
3) Activation of N-methyl-D-aspartate receptor which results in further calcium influx
4) Changes in tissue osmolarity and swelling of neuronal cells.

FACTORS INFLEUNCING THE NEURONAL EXCITABILITY:

1) Changes in ionic channel conductance
2) Second messengers
3) Cytoplasmic buffering
4) Response of receptors over neuronal membrane
5) Expression of proteins by genetic mechanisms
6) Changes in the neurotransmitter levels
7) Modulation of neuronal receptors

MECHANISM OF EPILEPTOGENESIS:

In epilepsy, a normal neuronal network is transformed to one that is chronically hyperexcitable. This occurs after a delay of months or years following an injury in the form of stroke, infection and trauma. The neurons of these epileptic focuses become susceptible to various stimuli like hypoxia, hypoglycaemia, hyperthermia, hyponatremia, hypocalcemia etc., Biochemical studies reveal following abnormalities.

1) Increased levels of extracellular potassium in the glial scars
2) Defects in calcium channel.
3) Deficiency in the neurotransmitter GABA
4) Increased glycine
5) Decreased taurine
6) Increase or decrease in glutamic acid.

PATHOLOGY OF EPILEPSY:

Pathological examination of autopsied specimen in most patients of primary generalised epilepsy show normal findings and also in cases where seizure occurs secondary to electrolyte disturbances, drug withdrawal, and drug intoxication since the primary defect is at the cellular level and neurotransmitters. Morphological changes like neuronal loss, gliosis, tumors, vascular malformations, hamartoma, heterotopias are noted in symptomatic epilepsies. In case of temporal lobe epilepsy,

pathological examination showed specific loss of neurons in the hippocampus and amygdala, distortion and loss of dendritic spines, and disruption of neuronal cells.

ROLE OF GENETICS:

Genetic studies show the presence of various gene mutations in association with epileptic syndromes. Single gene disorders account for 1-2% of all epilepsies and they present with additional neurological and systemic manifestations. Single gene disorders leading to pure epilepsy are rare conditions which are due to mutations involving ionic channels. Following are few such disorders,

1) Autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE)
2) Benign familial neonatal convulsions (BFNC)
3) Generalised epilepsy with febrile seizures plus (GEFS+)
4) Progressive myoclonus epilepsy (PME)
5) Dravet's syndrome

In addition to single gene disorders, epilepsy may be a part of various chromosomal and other genetic disorders like Down's syndrome, Angelman's syndrome, fragile X syndrome, glycogen storage disorders, aminoacid disorders, mitochondrial disorders, neurocutaneous disorders, and neurodegenerative disorders.

HISTORY AND EXAMINATION:

Evaluation of a patient presenting with focal seizures first involves detailed history of the onset and evolution of seizures and associated symptoms like presence of an aura, automatism, postictal features and postictal deficits. Patients should be

specifically asked for the presence of sensory symptoms and psychic auras. They are useful in localizing the seizure and etiology in many cases. The various risk factors and predisposing factors should be explored. The stereotypic nature of a seizure may be helpful in confirming a diagnosis of epilepsy. History of trauma, infections, tumour, stroke, systemic diseases, alcohol and drug abuse should be obtained. Identification of certain specific risk factors can help in prediction of the epileptogenic lesion. A history of febrile status epilepticus in infancy is associated strongly with hippocampal sclerosis[45]. Meningitis and encephalitis prior to age 5 is also associated with hippocampal sclerosis and temporal lobe epilepsy. Similarly, hippocampal sclerosis may also occur after head trauma.

Physical examination is done to identify neurocutaneous markers, signs of systemic illness or infection. Complete neurological examination should be done to assess memory status. Other system examinations are also carried out to rule out any organomegaly, evidences for chronic liver diseases, chronic renal diseases, and heart diseases.

LABORATORY EVALUATION:

Laboratory studies like complete blood count, blood sugar, renal function tests, liver function tests, electrolytes, thyroid function tests, and toxin screening in urine and blood should be done.

Lumbar puncture is done in those patients presenting with fever and signs of infection in whom meningitis or encephalitis are suspected. It is also indicated in cases of persistent alteration in mental status, elevated white blood cell count, and in

the absence of a precipitating factor. Lumbar puncture is particularly useful in cases of subarachnoid haemorrhage if CT brain is negative. CSF obtained through lumbar puncture should be sent for biochemical analysis, cytological analysis, cultures, PCR, and serological studies.

ELECTROPHYSIOLOGICAL STUDIES:

After initial examination and evaluation, a routine electroencephalography is the most commonly used neurodiagnostic test for evaluation of seizures since it is more informative to detect seizure activity. The absence of electrical activity does not exclude the diagnosis of seizure disorder because focal seizures arise from discrete areas of cortex which may not be detected by standard scalp electrodes. The detection of interictal spikes by scalp electrodes depends on the extent of epileptogenic zone. A 6 cm^2 critical area has to be involved to detect changes at the scalp electrodes.

It is not always possible to take ictal EEG, hence continuous monitoring by means of video EEG for prolonged duration can be obtained. About 50% of patients with epilepsy are found to show epileptic discharges in the first interictal EEG. Yield can be increased by the use of sleep studies[46]. The combination of sleep and wake records gives a yield of 80% in patients with clinically confirmed epilepsy. Yield can also be increased by the use of several special EEG procedures such as hyperventilation and photic stroboscopic stimulation.

The National Institute for Clinical Excellence guidelines for diagnosis and management of epilepsies in adults and children recommends an EEG to support a diagnosis of epilepsy in adults in whom history suggests epileptic seizures[47]. In

children, EEG is recommended after second or subsequent seizure. For those requiring an EEG, it should be performed within 4 weeks of request.

The EEG can also help determine prognosis after a first seizure. An abnormal EEG is consistently associated with an increased risk of recurrence of seizure. Video EEG monitoring is indicated for patients who continue to have seizures inspite of adequate treatment as the possibility of incorrect seizure diagnosis has to be ruled out.

IMAGING STUDIES:

Imaging plays an important role in focal seizures to localize the origin of focal seizures and to identify its cause which guides the treatment and prognosis of the patient. If a lesion is shown in a patient with seizures then there is high probability that seizures are due to that lesion[48]. According to guidelines given by American Academy of Neurology (1996), CT brain should be taken in new onset seizures in adults for acute emergency management[49].

Though CT brain remains the test most likely to be taken in the emergency room, MRI brain is more sensitive and is the imaging modality of choice. It is invaluable in identifying the cause of focal seizures, especially in seizures of temporal lobe origin as inferior temporal lobe lesions may not be seen even with contrast enhanced CT scan due to the beam hardening artefacts[50]. Physiologic imaging with SPECT and PET provides findings complementary to MRI but the physiologic changes are more widespread and less specific than MRI and EEG[51].

CAUSES OF FOCAL SEIZURES
STROKE:

Stroke is the most common cause of seizures in the elderly. Post stroke seizures are of two types. They can be early onset or late onset based on the occurrence of seizures after stroke. Early onset seizures occur within two weeks of stroke and late onset seizures occur after two weeks. In a multicentre prospective study, seizures were found in 8.9% of stroke patients. Of the post stroke seizures, 10.6% were due to hemorrhagic stroke, and 8.6% were due to ischemic stroke[52]. Risk of chronic epilepsy is highest in the first year after stroke. Approximately 30% of individuals who have early poststroke seizures develop epilepsy.

The incidence of seizures depends upon the type of stroke, the location of lesion and the severity of stroke. Seizures are more common in large haemorrhages involving cerebral cortex and less common in deep hematoma and supratentorial haemorrhage. The early onset post stroke seizures arise from the epileptic discharges of neurons in penumbra surrounding the central core of dead neurons whereas late onset post stroke seizures arise from the epileptic focus secondary to structural brain abnormalities[53].

In CVT, 40% of patients have seizures at the time of presentation of disease and 6.9% of patients develop seizures within 2 weeks of onset of illness[54]. Risk of developing seizures depends on the location of thrombosis and the associated parenchymal lesion[54].

METABOLIC ABNORMALITIES:

The metabolic and electrolyte abnormalities causing the seizures of adults include hypoglycemia, hyperglycemia, hyponatremia, hypernatremia, hypocalcemia, hypomagnesemia, hypokalemia, and hyperkalemia. They represent 9% of all acute symptomatic seizures. Though generalized tonic clonic seizures are more common,

focal seizures can also occur. Rapid changes in electrolyte levels are more likely to cause seizures than those occurring gradually[55].

Hyponatremia presents with seizures especially when the serum sodium levels are below 115mmol/L. The mortality rate in hyponatremia related seizures is very high around 50%. Hyponatremia related seizures can be stopped by rapid correction in the serum sodium concentration that average only 3 to 7 mEq/L[56]. In case of hypernatremia, seizures are common when there is acute elevation of serum sodium concentration to above 160mEq/L and also during rapid correction of hypernatremia. Brain shrinkage due to hypernatremia causes rupture of cerebral veins resulting in intracerebral and subarachnoid haemorrhages and seizures. Seizures may be the sole manifestation in hypocalcemia, occurring in 20-25% of acute cases. Although hypercalcemia results in reduced neuronal excitability, it can also cause seizures secondary to vasoconstriction and hypertensive encephalopathy[57]. Neurological manifestations occur in hypomagnesemia especially when serum concentrations are below 1mEq/L which includes confusion, tremors, seizures, agitation, hyperreflexia, tetany and myoclonus.

Hypoglycemia can cause focal or generalised seizures in addition to other neurological manifestations. The basic underlying mechanism involves reduced ATP, impaired Na-K ATPase channel leading to accumulation of sodium ions and subsequent cerebral edema. Seizures also manifest in hyperglycemic states especially in nonketotic hyperglycemic coma and less frequently in diabetic ketoacidosis[58].

Renal and hepatic failures are found to be associated with seizures due to the accumulation of the toxic compounds or due to the presence of co morbid illness. Seizures in renal failure also result due to the complications like malignant

hypertension, intracranial haemorrhage, dialysis disequilibrium syndrome, electrolyte and acid base disturbances[59]. Management of renal failure, control of hypertension and correction of electrolytes are needed to prevent the recurrence of seizures[60]. Generalised tonic clonic seizures, status epilepticus and nonconvulsive seizures complicate hepatic encephalopathy due to acute or chronic hepatic failure due to toxic effects of ammonia in brain[61].

Seizures due to metabolic abnormalities do not require treatment with antiepileptics as long as the underlying cause is rectified. Antiepileptics are usually ineffective in stopping the seizure if the metabolic disorder is not corrected.

BRAIN TUMORS

More than 30% of patients with brain tumors present as seizures[62]. Tumors mostly cause drug resistant epilepsy especially those in the temporal lobe. Excellent seizure control occurs after removal of benign tumors. Benign tumors like ganglioneuromas, dysembryoblastic neuroepithelial tumors(DNET) and low grade gliomas are commonly associated with seizures. Malignant brain tumors like anaplastic astrocytomas and glioblastoma multiforme can present with seizures.

Low grade tumors more commonly present with seizures than high grade tumors. Brain metastasis from cancers of lung, breast and malignant melanoma can present as seizure, although primary could not be identified in one third of the patients[63].

TUBERCULOMA

Tuberculoma is seen commonly in the developing countries. Around 20 to 30% of space occupying lesions in India are tuberculomas[64]. These develop in the parenchyma of brain from “Rich focus” when it does not rupture into subarachnoid

space and continue to grow walled off from the parenchyma and meninges by a thick fibrous capsule. Grossly, the lesions are well circumscribed masses with size varying from 1 cm to that of a small orange. Occasionally, tuberculomas may develop on the surface of the brain leading to a flat adherent mass called 'tuberculoma en plaque'[65].

Tuberculomas can be seen in the cerebral hemispheres, basal ganglia, brainstem and cerebellum. They can also develop in the meninges where it mimics a meningioma[66]. The usual presentations are seizures (60-100%), focal neurological deficits (33-68%) and features of raised intracranial tension (56-93%). Pathologically these tuberculomas are caseating granuloma which can be solitary or multiple[67].

CT appearance varies based on the stage of the lesion showing low attenuated areas in the early stages of edema and necrosis. When granuloma develops, it is visualised as high attenuated area with ring enhancement. These ring enhanced lesions contain central calcification surrounded by hypodense region called target sign. MRI Brain shows isointense lesion in T1 and T2 weighted sequence in the initial stage[68]. As the lesion advances, it may be either hyper or hypointense in T2, based on the relative proportions of cellular infiltrates, macrophages and fibrosis. The granulomas appear hyperintense in T2 if it is rich in cellular infiltrates and scanty macrophages and less fibrosis, hypointense in T2 if it is rich in macrophages and fibrosis. These lesions also enhance with contrast administration and produce solid or ring enhancement. Magnetisation transfer technique is another newer method to improve the tissue specificity and useful in differentiating tuberculoma from other lesions like neurocysticercosis. In tuberculoma, the MT ratio of T2 is hypointense than in cysticercosis[69].

NEUROCYSTICERCOSIS:

Neurocycticercosis is the most common parasitic disease of the central nervous system caused by larval forms (cysticercus cellulosae) of tapeworm, taenia solium. These cysts are situated in various areas like intracerebral, subarachnoid, intraventricular, spinal cord, and also in various organs, muscles and subcutaneous tissues outside the brain.

The parenchymal cyst tends to remain dormant for several years where it can cause minimal or no symptoms. The four stages of parenchymal cysts are (1) vesicular, (2) colloidal, (3)granular-nodular and (4) calcific[70]. The parasites are usually alive in the first two stages, while the last two stages represent the dead parasites. Patients become symptomatic only when the larva inside the cyst dies and releases its antigen, resulting in an intense inflammatory reaction.

Seizures are the most common clinical manifestation of neurocycticercosis[71]. Seizures are reported to occur in 70 to 90% of patients with neurocysticercosis[72]. Other symptoms include headache and focal neurological deficits. In a community based study done in South India (Vellore district) the prevalence of active epilepsy caused by NCC was 1.3 per 1000 population[73]. The lesions of neurocysticercosis can present as single or multiple, although multiple is less common. The single cyst infection (47.7% - 53.4%), is the most common manifestation in Indian subcontinent[74].

Diagnosis is done by clinical, radiological and immunological methods. Serological diagnosis is done by complement fixation test, indirect haemagglutination tests and ELISA. A newer gold standard test is enzyme linked immunoelectron

transfer blot (ELITB) assay which is done with CSF or serum samples based on seven glycoproteins specific to the pathogen.

Western blot assay have also been developed for diagnosis of NCC[75]. Neuroimaging studies are done using MRI Brain which is used to differentiate various stages of the parasite. CT Brain is helpful to detect calcified lesion[76]. Treatment with short course of antiheminthic (albendazole) for 28 days is given along with corticosteroids.

HIV INFECTION:

Patients of acquired immunodeficiency disease and other immunocompromised states have high risk of seizures either by direct effects of HIV virus on CNS or through opportunistic CNS infections[77]. Common opportunistic infections presenting as seizures include tuberculosis, toxoplasmosis, cryptococcosis, cytomegalovirus encephalitis and progressive multifocal leukoencephalopathy[78]. A study conducted in a tertiary care centre in India, showed that new onset seizures occurred in 5% of HIV patients out of which 26% were focal motor seizures and 8.6% were focal seizures with secondary generalization and majority of them were due to cerebral toxoplasmosis, followed by cryptococcosis and tuberculoma[79].

POST TRAUMATIC SEIZURES:

Post traumatic seizures can present as three types, immediate, early and late. Seizures that occur within the first 24 hours of trauma are called as immediate, and those that occur in the first week are known as early seizures. Late seizures occur beyond the first week of trauma. Early seizures contribute to 5% of hospitalised

patients. Among the trauma patients with seizures, about 2-12% of cases were due to closed head injury.

Those injuries with less than 30 minutes of amnesia and without skull fractures are considered as mild injuries which are less commonly associated with seizures. Moderate head traumas are those in which there are associated skull fractures and more than 30 minutes of amnesia. 1-4% of moderate injuries were associated with seizures. In severe head trauma with more than 24 hours of amnesia, cerebral contusion and intracranial hematoma, seizures are noted in 10-15%. In conditions of open head injury and penetrating injuries, seizures are seen in 30-50% of patients[80].

Changes in the brain reflecting the process of epileptogenesis are likely to occur in the latent period between the head injury and onset of chronic epilepsy. None of the several therapeutic measures tested for prevention of seizures after head injury were proven effective.

MANAGEMENT:

MEDICAL MANAGEMENT:

The first line treatment for epilepsy is medical treatment. The goal of treatment is a complete seizure free state in the absence of side effects of medications. In around 70% of patients with epilepsy, seizures are completely or almost completely controlled by medications. In another 20 to 25%, the attacks are significantly reduced with drugs in number and severity. The choice depends on the type of seizure and epilepsy syndrome along with factors like age, sex comorbid conditions and interactions with other drugs. Pharmacotherapy is not always necessary after a single unprovoked seizure, especially when the risk of recurrence is low.

For focal seizures, carbamazepine or phenytoin may be used as first line drugs but newer drugs without enzyme induction and better pharmacokinetics have supplanted the older ones. Though topiramate and oxcarbazepine are the only drugs with official FDA indications, lamotrigine, gabapentin and levitiracetam have evidence supporting their use as initial monotherapy. A study by Marson et al has shown that lamotrigine was found to be significantly better than carbamazepine, gabapentin and topiramate. However, lamotrigine requires slow titration and hence it is not an appropriate first choice when rapid onset of action is required. Oxcarbazepine and levitiracetam may be the drugs of choice when rapid therapeutic effect is required.

For all epilepsy indications, treatment is initiated with an AED monotherapy. In the absence of urgency, it is preferred to start a low dose antiepileptic and titrate slowly. The initial target dose is usually the minimum effective dose demonstrated in

clinical trials which can be titrated gradually until clinical efficacy is established. Medication has to be titrated to the highest tolerated dose before it is considered ineffective. Replacement monotherapy has to be considered if the initial therapy has been completely ineffective or if there is lack of tolerability and the new AED has to be added before withdrawing the old agent. Adjunctive therapy should be considered based on the pharmacodynamic and pharmacokinetic interactions. All AEDs are approved for combination therapy although some combinations have a synergistic effect; like lamotrigine and valproate when used together have greater efficacy than when used alone. The combination of lamotrigine and levitiracetam is also favoured.

TABLE 1.COMMON ANTIEPILEPTICS USED FOR FOCAL SEIZURES

DRUGS	**ADULT DOSE MG/D**
VALPROIC ACID	1000-3000
PHENYTOIN	300-400
CARBAMAZEPINE	600-1200
OXCARBAZEPINE	900-2400
PHENOBARBITAL	90-200
LAMOTRIGINE	300-500
LEVITIRACETAM	500-3000
TOPIRAMATE	400

Discontinuation of antiepileptic therapy:

The decision to withdraw medications must balance the potential consequences of seizure relapse and the potential benefits of eliminating medication side effects and costs on an individual basis. The potential risk factors associated with greater risk of recurrence are:

i. presence of EEG abnormality,
ii. partial seizures,
iii. longer than 5 years to attain seizure freedom,
iv. longer duration of active disease,
v. shorter number of years of remission,
vi. abnormal psychiatric examination,
vii. presence of hippocampal atrophy,
viii. abnormal neurological findings
ix. older age of seizure onset.

Most neurologists require patients to be seizure free for 2 to 4 years before discontinuing AEDs, and the drugs are generally discontinued over a 2 to 6 month period. A prospective study by Callaghan and colleagues showed that in patients who had been seizure free during 2 years of treatment with a single drug, one third of patients relapsed after discontinuation of the drug. The relapse rate was higher in patients with focal seizures compared to absence and generalized seizures[81].

SURGICAL TREATMENT OF EPILEPSY[82]

Surgical excision of epileptic focus which has not responded to prolonged and intensive medical treatment is being increasingly used in specialised epilepsy centers. It has been proposed that approximately 25% of all patients with epilepsy are candidates for surgery. A perspective that may increase surgery is the observation that around 60% of patients with focal seizures will respond to a conventional

anticonvulsant and among the remainder only few will respond to the addition of second and third line drugs.

To determine the location of epileptic focus, careful clinical, EEG and imaging findings are needed including long term video/EEG monitoring. Sometimes intraparenchymal depth electrodes for intracranial EEG recording may be needed. Introduction of functional imaging, magnetoencephalography and specialized EEG techniques have supplanted the previous available techniques.

The candidates who are most favourable for surgery are those with focal seizures that induced altered consciousness and a unilateral temporal lobe focus, in whom rates of cure and significant improvement approach 90%. The most common surgical approach has been a temporal lobectomy in which lateral temporal cortex is resected first and then followed by resection of amygdala and hippocampus. In a randomized controlled trail by Wiebe and colleagues, 58% of patients remained seizure free for 1 year after temporal lobectomy while only 8% were seizure free on medication alone. In patients with clear hippocampal sclerosis, an alternative is amygdalohippocampectomy with less risk to language functions and an equal outcome.

Surgery like callosotomy and hemispherectomy are of value in highly selected cases of intractable partial seizures. Removal of entire cortex of one hemisphere along with amygdala and hippocampus has been of value in children and some adults in severe unilateral disease and intractable contralateral motor seizures with hemiplegia like in Rasmussen encephalitis and Sturge weber syndrome.

Vagal nerve stimulation is a new technique which has found some favour in cases of intractable partial generalizing seizures, where a pacemaker like device is

implanted in the anterior chest wall and electrodes are connected to vagus at left carotid bifurcation[83]. Several trials have demonstrated around 25% reduction in frequency of seizures among those resistant to anticonvulsants. However they may be considered experimental currently.

MATERIALS AND METHODS

STUDY POPULATION:-

A total number of 50 patients who are presenting with new onset focal seizures during the study period from January 2015 to August 2015 in Thanjavur Medical College Hospital, fulfilling the inclusion criteria were taken for the study.

PLACE OF STUDY: Thanjavur Medical College Hospital, Thanjavur.

STUDY DESIGN: A hospital based, cross sectional observational study.

INCLUSION CRITERIA:

1) Patients with new onset focal seizures
2) Age of onset of seizures >12 years

EXCLUSION CRITERIA:

1) Age of onset of seizures <12 years
2) Onset before 12 years but continued to have seizures even after 12 years
3) Pseudo seizures

METHODS OF COLLECTION OF DATA:

Patients who presented with new onset focal seizures were subjected to detailed history, clinical examination and investigations. Patients and the attenders/bystanders were enquired in detail about the onset of seizure, progression of seizure, presence of aura, automatisms, any associated sensory or autonomic symptoms and postictal phenomenon. Family history of seizures and presence of any comorbid illnesses were also noted. Detailed general and systemic examination was

carried out and presence of any residual focal deficits was noted. The following investigations were done.

1) Blood sugar
2) Blood urea
3) Serum creatinine
4) Serum electrolytes- Na^{+} , K^{+} , Mg^{++}
5) Complete hemogram
6) CT Brain
7) MRI Brain
8) EEG
9) Special investigations like MR spectroscopy in selected cases.

OBSERVATION AND RESULTS

Total number of subjects studied- 50

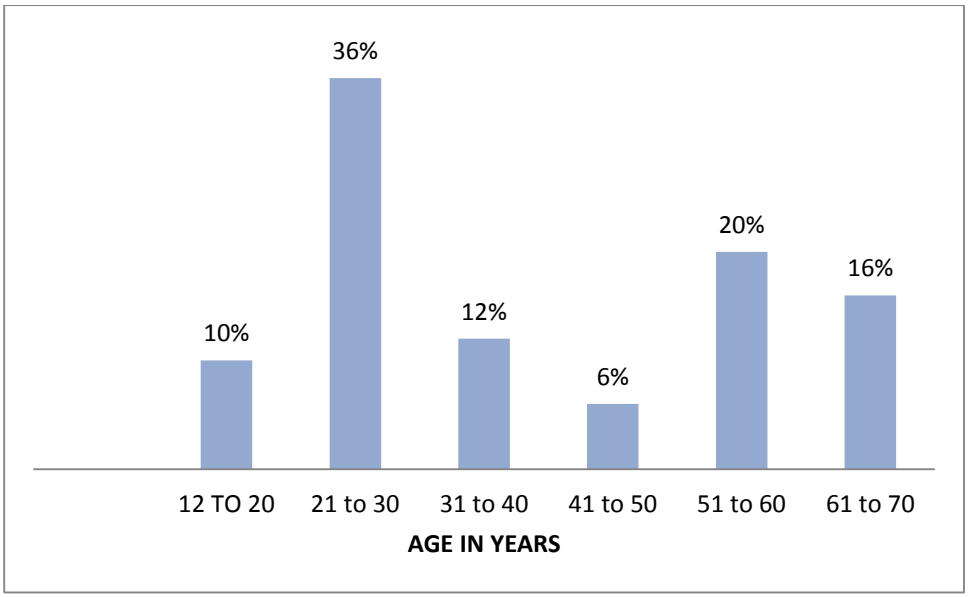
The baseline characteristics were observed and the results were shown in tables.

AGE DISTRIBUTION:

TABLE 2: AGE DISTRIBUTION

AGE IN YEARS	NO. OF PATIENTS	PERCENTAGE
12- 20	5	10%
21 to 30	18	36%
31 to 40	6	12%
41 to 50	3	6%
51 to 60	10	20%
61 to 70	8	16%
TOTAL	50	100%

FIGURE 1: AGE DISTRIBUTION



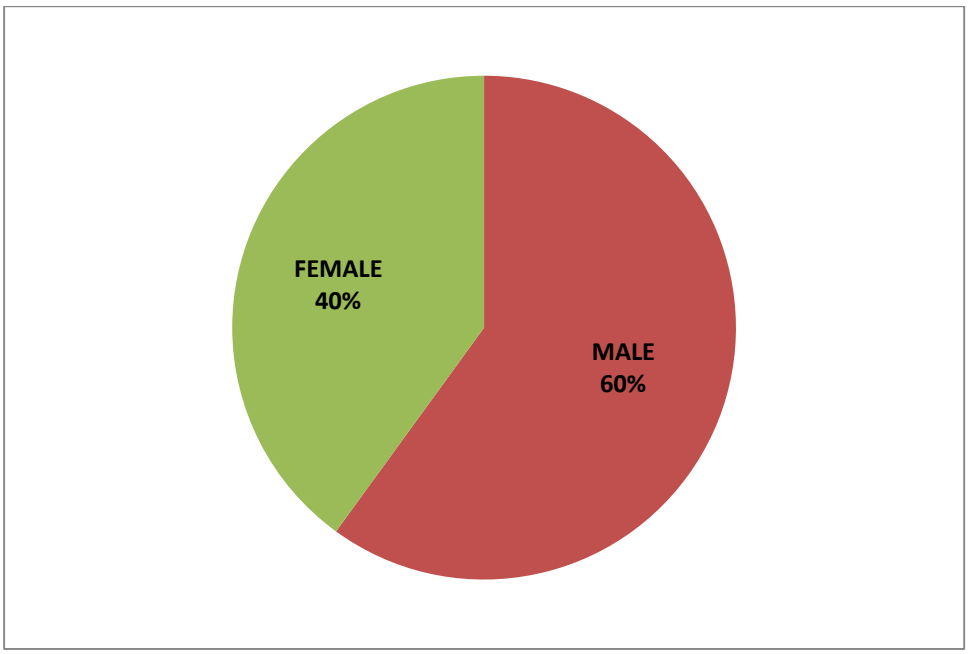
In this study, patients of age >12 years were included. The youngest age observed was 14 years and oldest was 70 years. The majority of patients belong to age group 21-30 years (36%), followed by the age group 51-60 years (20%). About 74% of patients were between 2nd -5th decade, and 16% of patients were above 60 years of age.

SEX DISTRIBUTION:

TABLE 3: SEX DISTRIBUTION

SEX	NO.OF PATIENTS	PERCENTAGE
MALE	30	60%
FEMALE	20	40%

FIGURE 2: SEX DISTRIBUTION



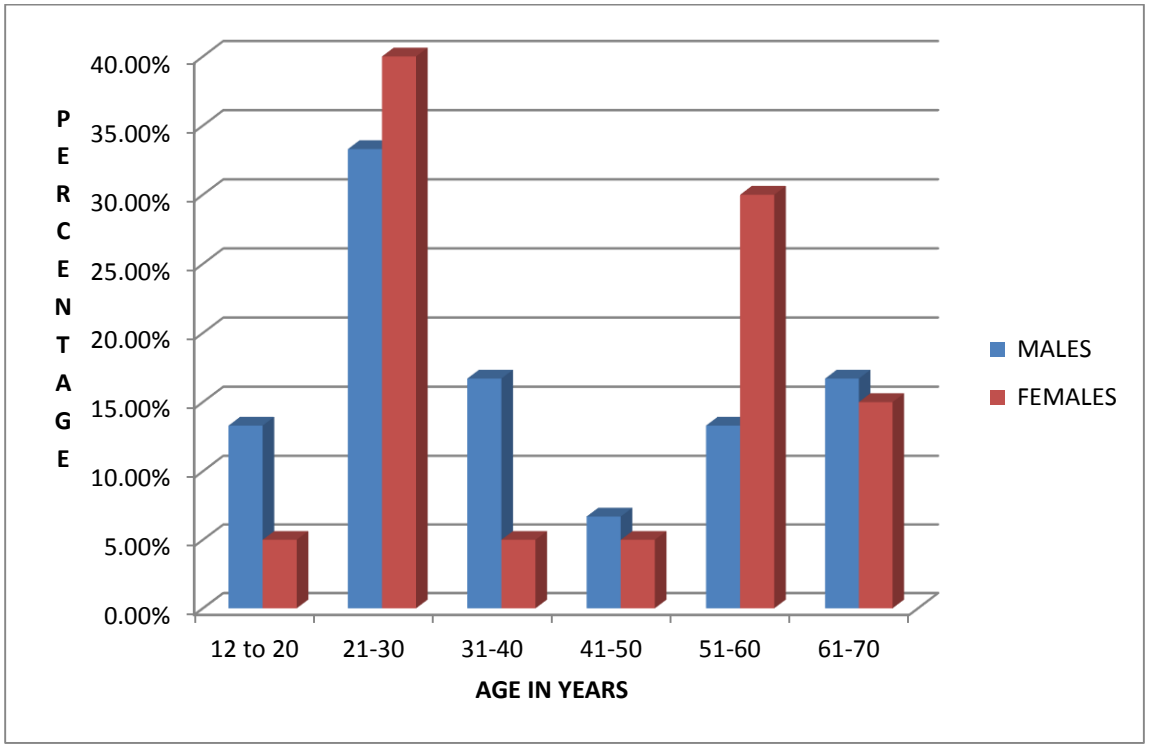
In the present study, 30 of 50 patients were males, 20 of 50 patients were females.

TABLE 4: COMPARISON OF SEX AND AGE IN YEARS

AGE IN YEARS	MALES		FEMALES		TOTAL	
	NO.	% among males	NO.	% among females	NO.	%
12-20	4	13.3%	1	5%	5	10%
21-30	10	33.3%	8	40%	18	36%
31-40	5	16.7%	1	5%	6	12%
41-50	2	6.7%	1	5%	3	6%
51-60	4	13.3%	6	30%	10	20%
61-70	5	16.7%	3	15%	8	16%
TOTAL	30	100%	20	100%	50	100%

The male to female ratio is 1.5:1 in the present study.

FIGURE 3:COMPARISON OF SEX AND AGE IN YEARS



Out of males, the majority belong to the age group 21-30 years (33.3%), and out of females also, the majority belong to the age group 21-30years (40%). Mean age is 40 years.

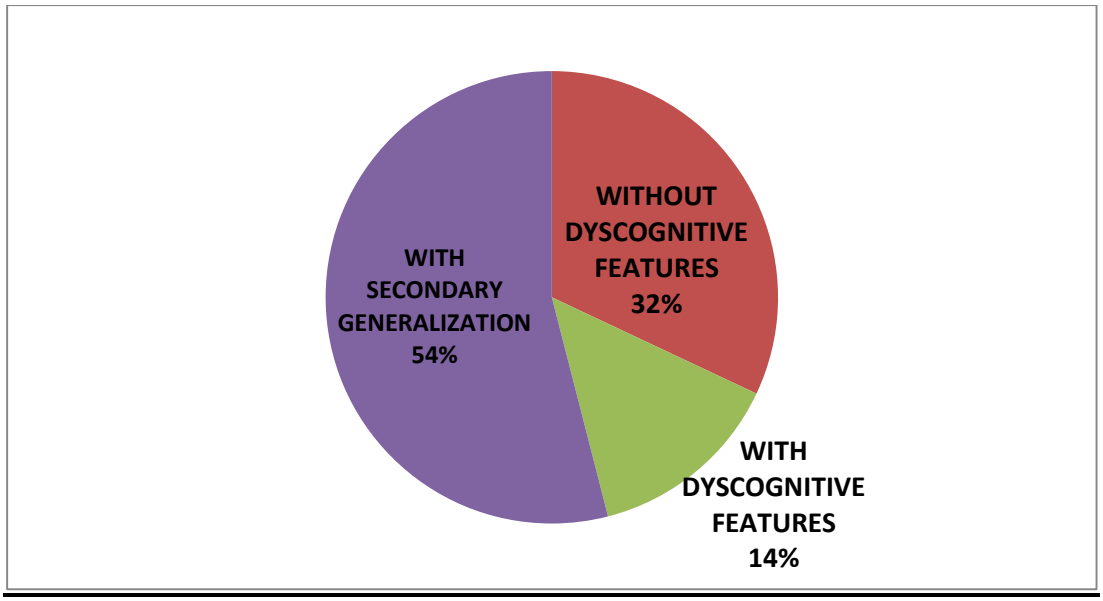
TYPE OF FOCAL SEIZURES:

TABLE 5: TYPE OF FOCAL SEIZURES

TYPE OF FOCAL SEIZURES	NO.OF PATIENTS	PERCENTAGE (100%)
WITHOUT DYSCOGNITIVE FEATURES	16	32.0
WITH DYSCOGNITIVE FEATURES	7	14.0
WITH SECONDARY GENERALIZATION	27	54.0

In this study, the most common type of focal seizures observed was focal seizures with secondary generalization (54%) followed by 32% focal seizures without dyscognitive features and 14% focal seizures with dyscognitve features. Out of 16 patients with focal seizures without dyscogntive features 2 patients had epilepsia partialis continua.

FIGURE 4: DISTRIBUTION OF TYPE OF SEIZURES



AURA:

Among the 50 patients, 16 patients(32%) had symptoms suggestive of aura, out of which 8 patients had somatosensory aura, 5 patients had autonomic aura, 2 patients had emotional aura in the form of fear 1 patient had cephalic sensation in the form of pressure on the head and lightheadedness.

AUTOMATISM:

Among 7 patients with focal seizures with dyscognitive features, 3 patients had automatism**.**

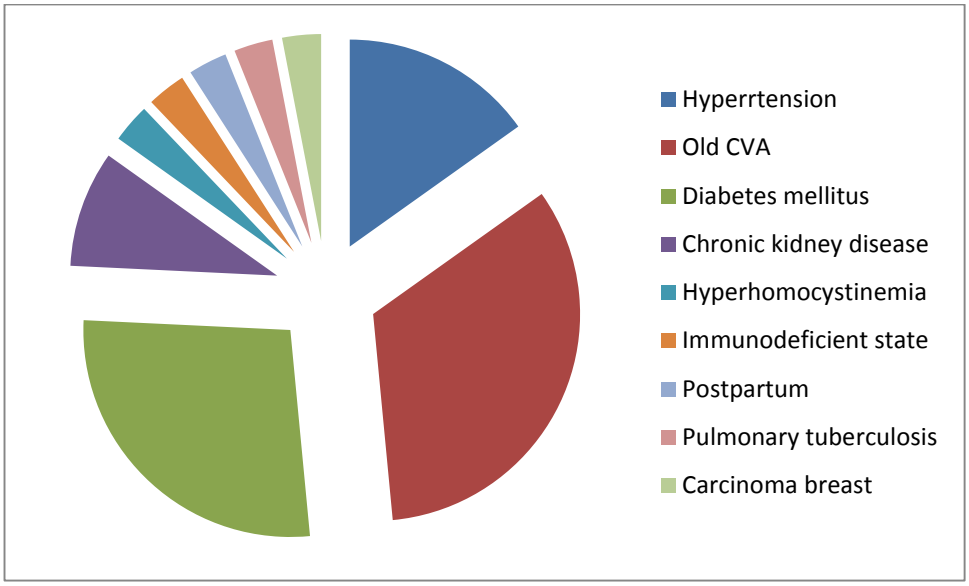
COMORBID CONDITIONS:

The various comorbid conditions observed among the patients of new onset focal seizures were hypertension, diabetes mellitus, chronic kidney disease, pulmonary tuberculosis. Majority of the patients had previous stroke followed by diabetes mellitus and chronic kidney disease. 5 patients had diabetes, hypertension and chronic kidney disease. One patient with tuberculoma was found to have associated pulmonary tuberculosis and another patient with tuberculoma was diagnosed with immunodeficiency.

TABLE 6: DISTRIBUTION OF CO MORBID CONDITIONS

COMORBID CONDITIONS	NO.OF PATIENTS
Hyperrtension	5
Old CVA	11
Diabetes mellitus	9
Chronic kidney disease	3
Hyperhomocystinemia	1
Immunodeficient state	1
Postpartum	1
Pulmonary tuberculosis	1
Carcinoma breast	1

FIGURE 5: DISTRIBUTION OF COMORBID CONDITIONS



IMAGING STUDIES:

CT Brain and MRI Brain were taken for the patients presenting with focal seizures. CT brain showed abnormality in 35(70%) patients. In case of old cerebrovascular diseases, CT Brain showed gliotic changes suggestive of old vascular etiology in 11 patients. Calcified granuloma with surrounding edema is seen in 4 patients, hypodense lesions present in 5 patients pointing to a diagnosis of tuberculoma with MRI. MR spectroscopy was done in 2 patients to differentiate the tuberculoma lesions from neurocysticercosis which showed decreased choline peak in tuberculoma patients.

CT brain also showed single calcified lesions in 3 patients, hypodense lesion with calcification in 2 patients and multiple ring enhancing lesions involving both cerebral hemispheres suggestive of neurocysticercosis. Calcified granuloma with no active lesion was found in 4 patients for which no further etiology could be found as they were healed lesions.

Those patients in whom cerebral venous thrombosis was suspected, MR angiogram and MR venogram were taken which showed thrombosis in superior sagittal sinus in 1 case and thrombosis in transverse sinus in other case.

CT Brain and MRI Brain also revealed tumors namely glioma in one patient and secondaries in brain in other patient. In a patient with post traumatic seizure, hemorrhagic contusion in left temporoparietal region was noted. Both CT brain and MRI brain was found to be normal in 14 patients out of which 7 patients had metabolic causes and 7 patients no cause could be found with the available investigations.

TABLE 7. CT BRAIN ABNORMALITIES

CT BRAIN	NO.OF PATIENTS	PERCENTAGE
NORMAL	15	30%
ABNORMAL	35	70%
TOTAL	50	100%

TABLE 8. CORRELATION OF CT BRAIN WITH MRI BRAIN

MRI BRAIN	CT BRAIN						P VALUE
	Normal		Abnormal		Total		
	(n=15)	(100%)	(n=35)	(100%)	(n=50)	(100%)	
Normal	14	93.3%	0	.0%	14	28.0%	.000
Abnormal	1	6.7%	35	100.0%	36	72.0%	

In this study, statistical significance with the p value of < 0.05 was obtained with the imaging findings, indicating that the MRI Brain was found to be superior to CT Brain in detecting the abnormal findings.

EEG:

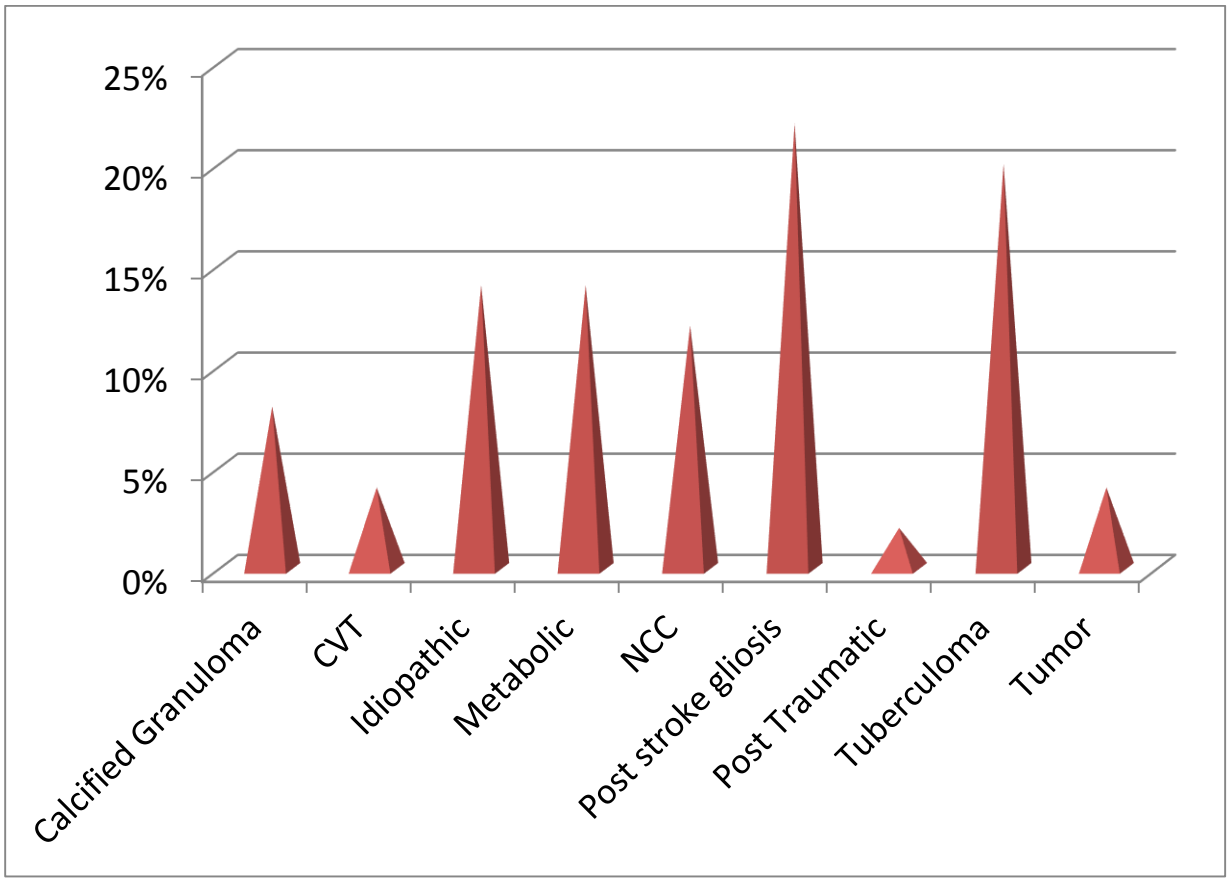
Electroencephalography was done in all 50 patients, out of which 26 (52%) of them had abnormal EEG recordings, and 24 patients(48%) patients had normal EEG recordings. Most of the EEG was taken during the interictal period.

ETIOLOGY OF SEIZURES:

TABLE 9. DISTRIBUTION OF ETIOLOGY

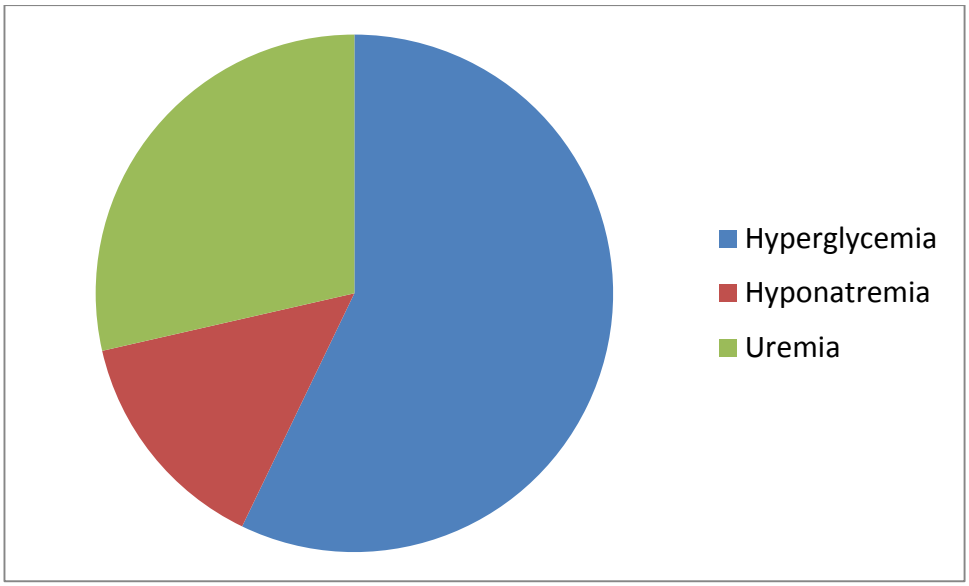
ETIOLOGY	NO.OF PATIENTS	PERCENTAGE
Calcified Granuloma	4	8%
CVT	2	4%
Idiopathic	7	14%
Metabolic	7	14%
NCC	6	12%
Post stroke gliosis	11	22%
Post Traumatic	1	2%
Tuberculoma	10	20%
Tumor	2	4%
Total	50	100%

FIGURE 6: DISTRIBUTION OF ETIOLOGY



The commonest cause was due to cerebrovascular diseases causing poststroke gliosis (22%) followed by tuberculoma (20%), metabolic (14%), idiopathic (14%), neurocysticercosis (12%), calcified granuloma (8%) cerebral venous thrombosis(4%) and tumor(4%) and posttraumatic (2%).

FIGURE 7: DISTRIBUTION OF METABOLIC CAUSES



Among the metabolic causes, 4 patients had hyperglycemia, 2 patients had uremia and 3 patients had hyponatremia.

ETIOLOGY ACCORDING TO SEX DISTRIBUTION:

MALES:

Total number of males: 30

The commonest etiology of focal seizures in males are neuroinfections which includes tuberculoma and neurocysticercosis accounting for 33.3% followed by poststroke gliosis(20%) followed by idiopathic(16.7%), metabolic(13.3%), calcified granuloma(10%).

The less common etiologies include CVT(3.3%) and posttraumatic (3.3%).

FEMALES:

Total number of cases: 20

The commonest etiology in females is also Neuroinfections (30%), followed by poststroke gliosis(25%), metabolic (15%).

The less common etiologies are idiopathic (10%), tumors(10%), calcified granuloma(5%),CVT (5%).

TABLE 10:ASSOCIATION OF SEX WITH ETIOLOGY

ETIOLOGY	SEX						P VALUE
	MALE		FEMALE		TOTAL		
	(n=30)	(100%)	(n=20)	(100%)	(n=50)	(100%)	
Calcified Granuloma	3	10.0%	1	5.0%	4	8.0%	.781>0.05 Not Significant
CVT	1	3.3%	1	5.0%	2	4.0%	
Idiopathic	5	16.7%	2	10.0%	7	14.0%	
Metabolic	4	13.3%	3	15.0%	7	14.0%	
NCC	4	13.3%	2	10.0%	6	12.0%	
Post stroke gliosis	6	20.0%	5	25.0%	11	22.0%	
Post Traumatic	1	3.3%	0	.0%	1	2.0%	
Tuberculoma	6	20.0%	4	20.0%	10	20.0%	
Tumor	0	.0%	2	10.0%	2	4.0%	

Statistical significance was found for association of sex and etiology of seizure with Chi square test and there was no significant association found between etiology of seizure and sex of the patients**.** (p value > 0.05)

ETIOLOGY ACCORDING TO AGE DISTRIBUTION:

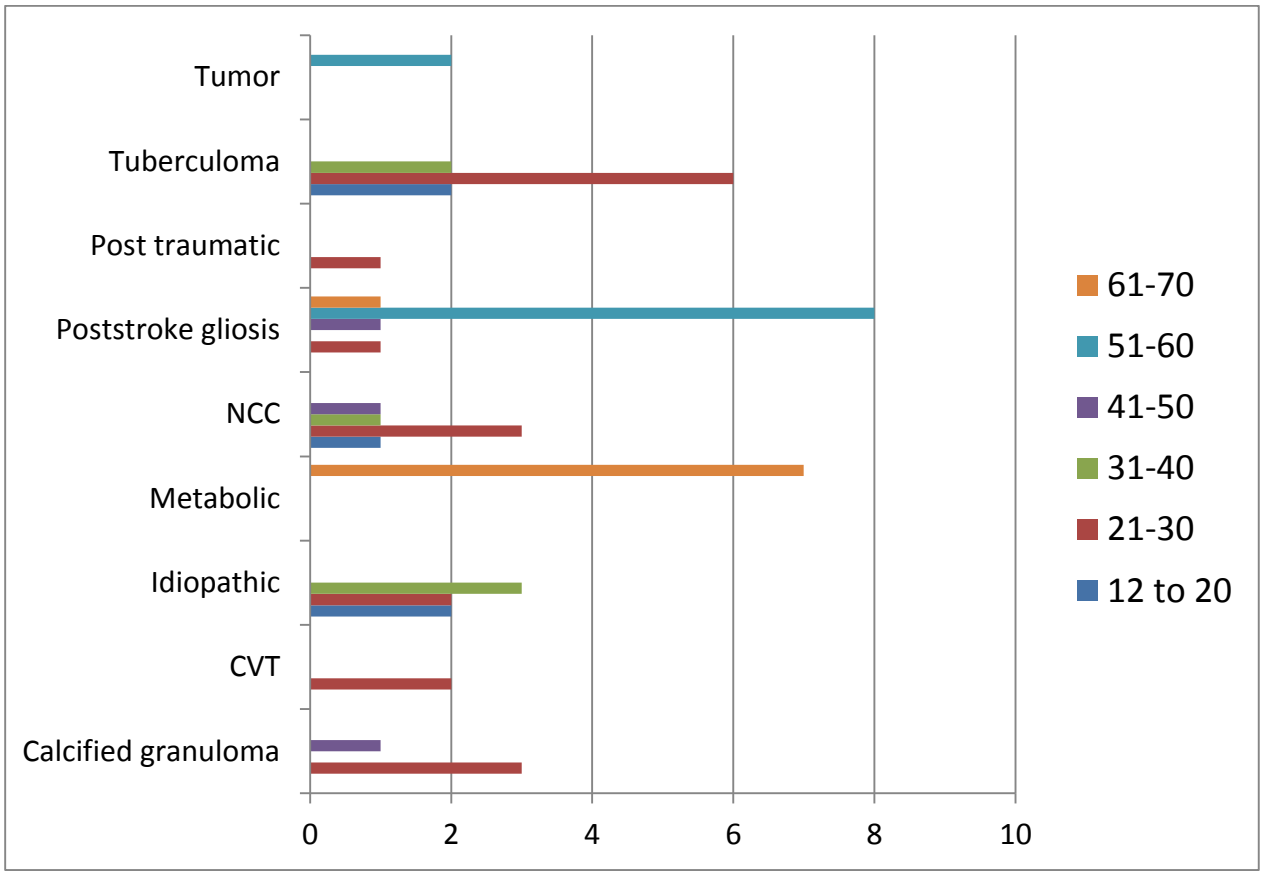
Most common etiology among 12-20 years is neuroinfections both tuberculoma and neurocysticercosis together (60%), followed by idiopathic (40%). Among 21-30 years, most common etiology is neuroinfections ,tuberculoma (33.3%), followed by neurocysticercosis (30.4%). Among age group 31-40 years, idiopathic (50%) followed by tuberculoma (33.3%).

TABLE 11. ETIOLOGY ACCORDING TO AGE GROUP

S.NO	ETIOLOGY	12-20	21-30	31-40	41-50	51-60	61-70	Total n(100%)
1.	Calcified granuloma	0	3	0	1	0	0	4(8%)
2.	CVT	0	2	0	0	0	0	2(4%)
3.	Idiopathic	2	2	3	0	0	0	7(14%)
4.	Metabolic	0	0	0	0	0	7	7(14%)
5.	NCC	1	3	1	1	0	0	6(12%)
6.	Poststroke gliosis	0	1	0	1	8	1	11(22%)
7.	Post traumatic	0	1	0	0	0	0	1(2%)
8.	Tuberculoma	2	6	2	0	0	0	10(20%)
9.	Tumor	0	0	0	0	2	0	2(4%)
	TOTAL	5 (10%)	18 (36%)	6 (12%)	3 (6%)	10 (20%)	8 (16%)	50(100%)

In the age group 41-50 years, calcified granuloma, neurocysticercosis and poststroke gliosis each contributed 33.3%.

FIGURE 8. ETIOLOGY ACCORDING TO AGE DISTRIBUTION



Among 51-60 years, 80% was due to poststroke gliosis followed by tumors(20%). In the age group 61-70 years, 87.5% cases was due to metabolic etiology follwed by poststroke gliosis(12.5%).

TABLE 12. CORRELATION OF CT BRAIN FINDINGS WITH ETIOLOGY

ETIOLOGY	CT BRAIN FINDINGS						P VALUE
	Normal		Abnormal		Total		
	(n=15)	(100%)	(n=35)	100.0%	50	100.0%	
Calcified Granuloma	0	.0%	4	11.4%	4	8.0%	.000<0.05 Significant
CVT	1	6.7%	1	2.9%	2	4.0%	
Idiopathic	7	46.7%	0	.0%	7	14.0%	
Metabolic	7	46.7%	0	.0%	7	14.0%	
NCC	0	.0%	6	17.1%	6	12.0%	
Post stroke gliosis	0	.0%	11	31.4%	11	22.0%	
Post Traumatic	0	.0%	1	2.9%	1	2.0%	
Tuberculoma	0	.0%	10	28.6%	10	20.0%	
Tumor	0	.0%	2	5.7%	2	4.0%	

The majority of cerebrovascular diseases (31.4%) and neuroinfections (45.7%) showed abnormal CT Brain findings when compared to other etiologies. The metabolic causes (46.7%) had normal CT Brain imaging. This was statistically significant (p value <0.05).

ETIOLOGY AND TYPE OF SEIZURES

The most common seizure type is focal seizures with secondary generalization followed by focal seizures without dyscognitive features and focal seizures with dyscognitive features.

Among focal seizures with dyscognitive features, around 71.4% was idiopathic and 28.6% was due to tuberculoma.

Among seizures without dyscognitive features, 31.3% was due to tuberculoma and 25% was due to poststroke gliosis followed by other causes..

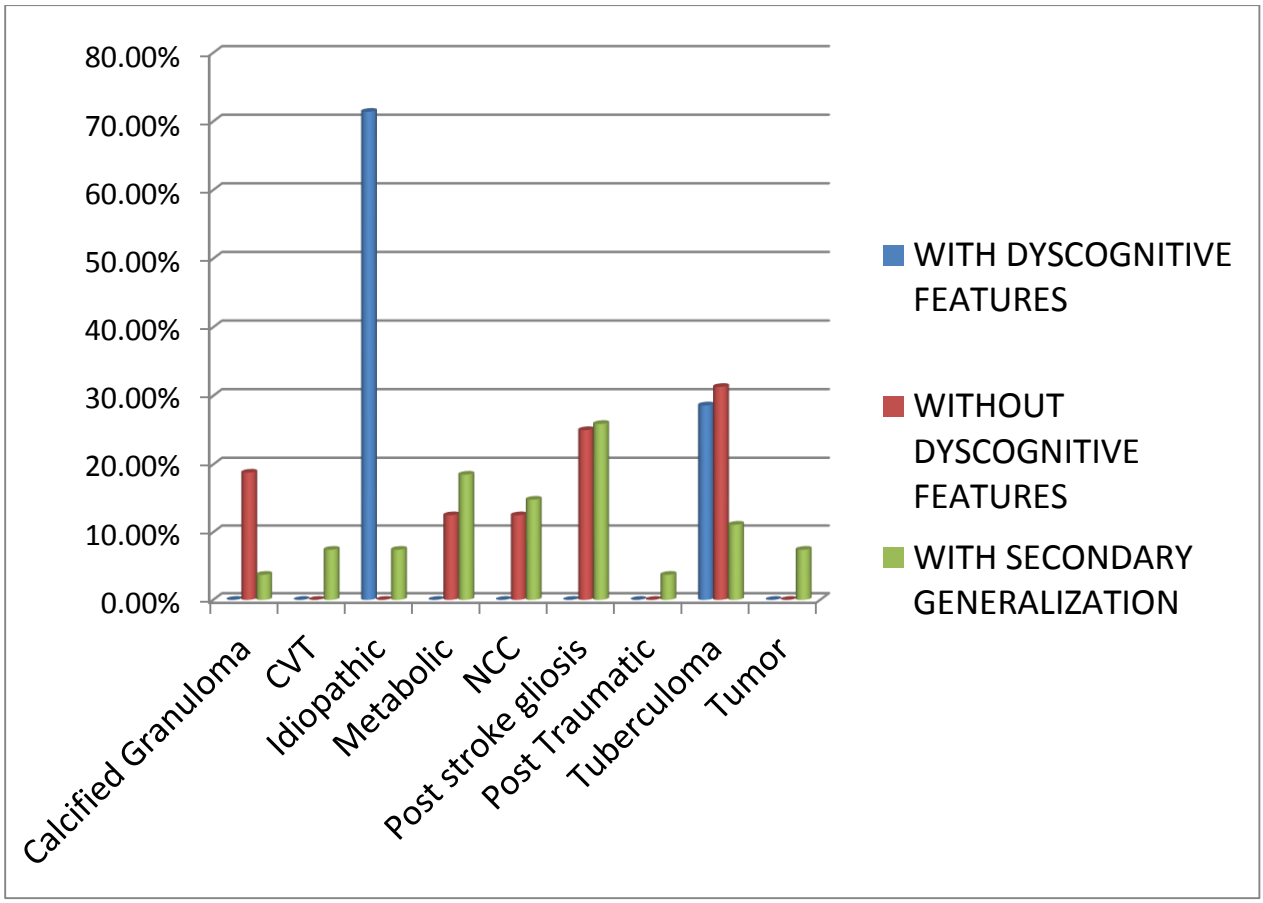
Among focal seizures with secondary generalization, 25.9% was due to poststroke gliosis.

TABLE 13: ASSOCIATION OF ETIOLOGY WITH TYPE OF SEIZURES

ETIOLOGY	TYPES OF FOCAL SEIZURE								P VALUE
	With dyscognitive features		Without dyscognitive features		With secondary generalization		TOTAL		
	(n=7)	(100%)	(n=16)	(100%)	(n=27)	(100%)	(n=50)	(100%)	
Calcified Granuloma	0	.0%	3	18.8%	1	3.7%	4	8.0%	.006<0.05 Significant
CVT	0	.0%	0	.0%	2	7.4%	2	4.0%	
Idiopathic	5	71.4%	0	.0%	2	7.4%	7	14.0%	
Metabolic	0	.0%	2	12.5%	5	18.5%	7	14.0%	
NCC	0	.0%	2	12.5%	4	14.8%	6	12.0%	
Post stroke gliosis	0	.0%	4	25.0%	7	25.9%	11	22.0%	
Post Traumatic	0	.0%	0	.0%	1	3.7%	1	2.0%	
Tuberculoma	2	28.6%	5	31.3%	3	11.1%	10	20.0%	
Tumor	0	.0%	0	.0%	2	7.4%	2	4.0%	

There is significant association between type of seizures with the etiology of seizures with the p value of <0.05.

FIGURE 9: ETIOLOGY AND TYPE OF SEIZURES



DISCUSSION

In our study, 50 cases of new onset focal seizures were included as per the inclusion and exclusion criteria and the observations were compared with similar other studies.

Age and sex distribution:

In the present study, out of 50 patients, 30 were males and 20 were females and the male to female ratio is M: F = 1.5:1. This was similar to a study done by AMARAVATHI et al (2015) in Hyderabad, India[84]. In a study by DILLI RAM KAFLE (2013) the male to female ratio was 1.3:1[85]. Among both males and females, most of them belong to the age group 21 to 30 years.

In this study, majority of the patients belong to the age group 21-30 years (36%), and 74% were in the age group 2^{nd}-5^{th} decade. In the study by AMARAVATHI et al, majority (26%) patients were in the age group 28- 37 years followed by 18% each in 18 to 27 years and 57-68 years. In a study by DILLI RAM KAFLE (2013), the majority 38% belonged to age group less than 20 years, probably because in the present study only new onset seizures were included.

Type of focal seizures:

In the present study, focal seizures with secondary genreralization was the most common type (54%) followed by focal seizures with and without dyscognitive features. This was similar to the study by DILLI RAM KAFLE (2013), where focal seizures with secondary generalization was seen in 43% followed by focal seizures

with and without dyscognitive features 29% each, while in AMARAVATHI et al(2015), focal seizure without dyscognitive features was the most common (54%). 5% patients had epilepsia partialis continua in this study. In AMARAVATHI et al (2015), 12% of pateints had epilepsia partialis continua. In this study, 16 patients (32%) had symptoms of aura and somatosensory aura was most commonly present, while in AMARAVATHY et al, aura was reported in 48%.

Etiology of seizures:

In the present study, the majority of cases 22% were due to poststroke gliosis. This was similar to AMARAVATHY et al (2015) which reported 38% of cases due to poststroke gliosis. The study by DILLI RAM KAFLE (2013) (55%) showed calcified granuloma as the most common cause, while cerebral infarction was the next common contributing 17%. Calcified granuloma constitutes around 8% of cases in the present study.

Among the neuroinfections, tuberculoma constitutes 20% while neurocysticercosis accounts for 12%. In AMARAVATHI et al (2015) study, tuberculoma was reported around 10% while neurocysticercosis was around 14%. While in DILLI RAM KAFLE (2013), tuberculoma constituted 8%. In a study by WASHIMKAR et al (1996), tuberculoma constituted 65.9% of cases of partial seizures[86].

Metabolic cause of seizures constitutes 14% in our study out of which majority are due to hyperglycemia (57%) followed by uremia and hyponatremia. In a study by

NARAYANAN JT and MURTHY JMK (2007) hyponatremia was the most common metabolic cause of seizures (71.4%)[87].

Tumors were observed in 6% of cases in the study by AMARAVATHI et al (2015). In the present study, tumor constitutes 4% of cases.

Seizures are found to be idiopathic in 14% in the present study. In AMARAVATHI et al (2015), 10% were found to be idiopathic. DILLI RAM KAFLE reported mesial temporal sclerosis in 4% while no case of mesial temporal sclerosis is diagnosed in the present study.

Etiology according to age:

In our study, majority of poststroke gliosis occured in 51 to 60 years, while neuroinfections mainly tuberculomas and neurocysticercosis are the major causes in 21 to 30 years age group. This was similar to a study by HAUSER et al, where 50% of neuroinfections were observed in 2^{nd}-3^{rd} decades and metabolic causes were common in the age group above 40 years[88].

Imaging findings:

EEG taken during interictal period showed abnormality in 52% of patients and 48% had normal EEG findings.

CT brain is found to be abnormal in 70% of patients. The most common lesion in CT brain was gliosis due to old infarct followed by hypodense lesion with edema. In DILLI RAM KAFLE, 74% had imaging abnormality while in AMARAVATHY et al (2015), 86% had imaging abnormality. In a study by RAVINDRA KUMAR GARG et al (1998), 71.4 % had abnormal imaging[89]. In a study by WADIA et al, 68% had

CT abnormalities and the most common lesion was a ring ehancing lesion[90]. The majority of cerebrovascular diseases (22%) and neuroinfections (32%) showed abnormal CT Brain findings when compared to other etiologies like metabolic causes with a significant p value of <0.05.

It was very difficult to differentiate between between neurocysticercosis and tuberculoma with CT brain alone. MRI brain was taken in all these patients. Chest x ray was also done in all suspected cases of tuberculoma. Overall, MRI Brain was found to be superior to CT Brain in detecting abnormal findings (p <0.05).

In this study, despite careful investigations, 10% were diagnosed with idiopathic epilepsy. More sensitive scanning techniques may help to further sort this group of idiopathic epilepsy into other categories.

TABLE 14: COMPARISON OF ETIOLOGICAL SPECTRUM OF THE PRESENT STUDY WITH OTHER STUDIES

ETIOLOGY	AMARAVATHY et al (2015)	DILLI RAM KAFLE (2013)	PRESENT STUDY
Tuberculoma	10%	6%	20%
NCC	14%		12%
Calcified granuloma	14%	55%	8%
Post stroke gliosis	38%	17%	22%
Metabolic			14%
Idiopathic	10%	12%	14%
Tumors	6%	4%	4%
CVT			4%
Posttraumatic		2%	2%
Brain abscess	4%		
Meningitis	4%		
Mesial temporal sclerosis		4%	

All patients were started on antiepileptic drugs. Most of the patients had adequate control of seizures with a single antiepileptic drug. For patients with tuberculoma, Antituberculous therapy was started.

Follow up scan was advised after 12 weeks to look for regression of lesions. For patients with neurocysticercosis, Albendazole was given for 28 days along with steroids. Metabolic factors were corrected in those with metabolic etiology. Patients with brain tumors were referred to neurosurgery.

SUMMARY

This study was the cross sectional observational study carried out with 50 patients of new onset focal seizures admitted in Thanjavur Medical College Hospital fulfilling the inclusion and exclusion criteria.

Of the total 50 patients, 60% were males and 20% were females, with the ratio of M: F=1.5:1. Majority of the patients belong to the age group 21-30 years (36%), and 74% were in the age group 2^{nd}-5^{th} decade.

Focal seizures with secondary genreralization was the most common type (54%) followed by focal seizures without dyscognitive features (32%) and focal seizures with dyscognitve features (14%). Two patients had epilepsia partialis continua. 16 patients (32%) had symptoms of aura.

Old cerebrovascular disease was seen in 22%, diabetes mellitus in 18%, hypertension in 10%, chronic kidney disease in 6%.

The commonest cause was due to cerebrovascular diseases causing poststroke gliosis (22%) followed by tuberculoma (20%), metabolic (14%), idiopathic (14%), neurocysticercosis (12%), calcified granuloma (8%) cerebral venous thrombosis(4%), tumor(4%) and posttraumatic (2%).

CONCLUSION

The following conclusions were made from the present study,

- Majority of focal seizures were noted in the age group 2nd to 5th decade with male preponderance.
- Most of the focal seizures were focal seizures with secondary generalization (54%)
- The spectrum of etiology varies which include old cerebrovascular diseases, Neuroinfections, metabolic causes, tumors, and idiopathic.
- In younger age group less than 30 years, neuroinfections were the most common cause.
- In age more than 50 years, metabolic causes and cerebrovascular diseases predominate.
- Hence, in all cases of new onset focal seizures, complete evaluation is required to identify the etiology and hence to initiate proper treatment, thereby the morbidity and mortality in the community can be reduced.

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CONSENT FORM

I _____ hereby give consent to participate in the study conducted by **DR. REVATHY.V,** Post graduate in the Department of General Medicine, Thanjavur Medical College & Hospital, Thanjavur – 613004 and to use my personal clinical data and result of investigation for the purpose of analysis and to study the nature of disease. I also give consent for further investigations.

Place :

Date :

Signature of participant

A STUDY OF CLINICAL AND ETIOLOGICAL PROFILE

OF FOCAL SEIZURES

PROFORMA

Name: D.O.A:
Age: D.O.D:
Sex: Male/Female Address:
Occupation:

CHIEF COMPLAINTS:

HISTORY OF PRESENTING ILLNESS:

1. Convulsions:

   a) Duration:

   b) No. of episodes:

   c) Type of focal seizure:

   d) Aura: Yes / No

   e) Type of aura:

   f) Automatism: Yes/No

   g) Loss of consciousness: Yes / No

   h) Tongue bite: Yes / No

   i) Autonomic symptoms:
      Frothing / Sweating / Salivation/ Urination/ Defecation

   j) Postictal state: Confusion / Amnesia / Paralysis

2. Other associated complaints:
   a. Fever:
   b. Headache:
   c. Nausea/ vomiting:

d. Focal deficits:
e. Others:

PAST HISTORY:

- Previous history of seizures: yes / no
- H/o stroke / infections / trauma / recent delivery:
- H/o ht / dm / tb / rheumatic heart disease/ psychiatric disorder
- Others:

FAMILY HISTORY:

- Any similar complaints in other family members
- Comorbid illness: yes / no

PERSONAL HISTORY:

- Habits-
  - Smoking: yes/ no. if yes, duration:
  - Alcohol: yes/ no. if yes, duration:
  - Tobacco chewing: yes/ no
  - Drug abuse: yes/ no. if yes, drug:
  - Exposure to STDs: yes/ no.

EXAMINATION OF THE PATIENT

GENERAL EXAMINATION:

- Build and nourishment:
- Pallor / icterus/ clubbing / cyanosis/ pedal edema / lymphadenopathy
- Any neurocutaneous markers:

VITAL SIGNS:

Pulse: RR:
BP: Temp:

CENTRAL NERVOUS SYSTEM EXAMINATION:

A) Higher mental functions

a. Consciousness:
b. Orientation to : time/ place / person
c. Appearance and behaviour:
d. Attention:
e. Vigilance:
f. Language:
g. Memory
h. Intelligence

B) Cranial nerves examination: Right Left

CN I: Sense of smell

CN II: Visual acuity
Field of vision:
Colour vision:
Fundus:

CN III, IV, VI: Ptosis: present / absent
Extraocular movements:
Pupils- size, shape:
Light reflex: direct / indirect

CN V: Motor:
Sensory:
Reflexes:

CN VII: Motor:
Taste sensation:

CN VIII: Rinnes test:
Webers test:

CN IX:

CN X: Uvula
Palatal reflex
Pharyngeal reflex:

CN XII: Sternocleidomastoid:
Trapezius:

CN XII: Tongue movements
Wasting/Fasciculations

C) Motor system: **Right** **Left**

Bulk:
Tone
Power
- Shoulder
- Elbow
- Wrist
- Hand grip
- Hip:
- Knee:
- Ankle:

Reflexes:
- Superficical : Abdominal
  Cremasteric
  Plantar
- Deep : Biceps
  Triceps
  Supinator
  Knee
  Ankle
  Jaw jerk

Any abnormal movements:

D) Sensory system:
- Touch
- Pain
- Temperature
- Vibration
- Joint position sense
- Cortical sensation:
  - Stereognosis
  - Tactile localisation
  - Two point discrimination
  - Romberg's sign

E) Cerebellar signs:

F) Gait:

G) Signs of meningeal irritation:

H) Spine and Cranium:

OTHER SYSTEMS:

Cardiovascular system:
Respiratory system:
Abdomen:

INVESTIGATIONS:

1) Urine
   - Albumin
   - Sugar
   - Deposits
   - Others
2) Blood sugar:
3) Blood urea:
4) Serum creatinine:
5) Serum electrolytes:
   a. Sodium
   b. Potassium
6) Complete hemogram
   a. Hb%:
   b. TC:
   c. DC:
   d. RBC:
   e. Platelets:
   f. PCV:
   g. ESR:
7) VCTC:
8) ECG:
9) Chest X ray:
10) CT brain:
11) MRI brain:
12) EEG:
13) Others:

MASTER CHART

S.NO.	NAME	AGE	SEX	TYPE OF FOCAL SEIZURE	AURA	TYPE OF AURA	AUTOMATISM	COMORBIDITY	EEG	CT BRAIN	MRI BRAIN	ETIOLOGY	ETIOLOGY OF METABOLIC SEIZURES
1	THENMOZHI	24	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	YES	SOMATOSENSORY			NORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
2	LAKSHMI	60	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			CA BREAST	ABNORMAL	ABNORMAL	ABNORMAL	TUMOR	
3	VALARMATHY	29	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	YES	CEPHALIC SENSATION			NORMAL	NORMAL	NORMAL	IDIOPATHIC	
4	RAJENDRAN	25	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO				ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
5	VIJAY	18	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO				ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
6	MURUGANANDHAM	63	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			SHT, CVA	ABNORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
7	SHANMUGAVALLI	56	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO			CVA	NORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
8	ARASAN	30	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	YES	AUTONOMIC			NORMAL	ABNORMAL	ABNORMAL	NCC	
9	LAKSHMIKANDHAN	70	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO			DM	NORMAL	NORMAL	NORMAL	METABOLIC	HYPERGYCEMIA
10	JEGANNATHAN	40	M	**FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES**	YES	AUTONOMIC	YES		NORMAL	NORMAL	NORMAL	IDIOPATHIC	
11	JAGADAMBAL	65	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO			DM	NORMAL	NORMAL	NORMAL	METABOLIC	HYPERGYCEMIA
12	REVATHY	26	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	YES	SOMATOSENSORY			ABNORMAL	ABNORMAL	ABNORMAL	CALCIFIED GRANULOMA	
13	SATISH KUMAR	21	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			HYPERHOMOCYSTINEMIA	NORMAL	NORMAL	ABNORMAL	CVT	
14	DURAIRAJ	27	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO				NORMAL	ABNORMAL	ABNORMAL	CALCIFIED GRANULOMA	
15	SHEIK BABU	35	M	**FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES**	YES	EMOTIONAL AURA	YES		ABNORMAL	NORMAL	NORMAL	IDIOPATHIC	

16	MAHADEVAN	14	M	**FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES**	NO		NO		NORMAL	NORMAL	NORMAL	IDIOPATHIC	
17	MUTHUKUMAR	25	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO				ABNORMAL	ABNORMAL	ABNORMAL	POSTTRAUMATIC	
18	SULOCHANA	68	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			DM, CKD	NORMAL	NORMAL	NORMAL	METABOLIC	UREMIA
19	MUTHAIYAN	30	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO				ABNORMAL	ABNORMAL	ABNORMAL	CALCIFIED GRANULOMA	
20	THENNARASI	56	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO				ABNORMAL	ABNORMAL	ABNORMAL	TUMOR	
21	MUTHULAKSHMI	52	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO			SHT, DM, CVA	NORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
22	RAVI	21	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	YES	SOMATOSENSORY		PT	ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
23	AJITH	20	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO				NORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
24	MURUGAN	42	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	YES	AUTONOMIC			ABNORMAL	ABNORMAL	ABNORMAL	CALCIFIED GRANULOMA	
25	PAPPA	50	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			CVA	NORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
26	ARIVAZHAGAN	28	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	YES	AUTONOMIC			ABNORMAL	ABNORMAL	ABNORMAL	NCC	
27	KRISHNAMOORTHY	65	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			DM, CKD	NORMAL	NORMAL	NORMAL	METABOLIC	HYPONATREMIA
28	RAJATHI	60	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	YES	SOMATOSENSORY		SHT, CVA	NORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
29	KUNJAMMAL	67	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			DM	NORMAL	NORMAL	NORMAL	METABOLIC	HYPERGLYCEMIA
30	POTTU	31	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO				ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	

31	NADHIYA	25	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			POSTPARTUM	NORMAL	ABNORMAL	ABNORMAL	CVT	
32	CHELLAPPA	26	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO				ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
33	JAYABAL	32	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO				NORMAL	NORMAL	NORMAL	IDIOPATHIC	
34	VANITHA	16	M	**FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES**	YES	EOMTIONAL AURA	NO		ABNORMAL	NORMAL	NORMAL	IDIOPATHIC	
35	ARUN KUMAR	28	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	YES	SOMATOSENSORY			ABNORMAL	ABNORMAL	ABNORMAL	NCC	
36	PITCHAIYAN	70	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			DM	NORMAL	NORMAL	NORMAL	METABOLIC	HYPERGLYCEMIA
37	RAMAN	32	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	YES	AUTONOMIC		IMMUNODEFICIENT STATE	ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
38	ANJALAI	56	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	YES	SOMATOSENSORY		CVA	ABNORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
39	MANIMEGALAI	26	F	**FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES**	NO		NO		NORMAL	NORMAL	NORMAL	IDIOPATHIC	
40	RANI	59	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO			CVA	ABNORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
41	SOWMYA	22	F	**FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES**	NO		YES		ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
42	VADIVEL	58	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			CVA	ABNORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
43	MURUGANANDHAM	55	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			CVA	ABNORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
44	KALPANA	32	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO				NORMAL	ABNORMAL	ABNORMAL	NCC	
45	PITHCHAIYAMMAL	60	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			SHT, DM, CVA	NORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
46	BABU	56	M	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	YES	SOMATOSENSORY		SHT, CVA	NORMAL	ABNORMAL	ABNORMAL	POSTSTROKE GLIOSIS	
47	KANNAYIRAM	68	M	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO			DM, CKD	NORMAL	NORMAL	NORMAL	METABOLIC	UREMIA
48	RAJESHWARI	45	F	FOCAL SEIZURES WITH SECONDARY GENERALIZATION	NO				NORMAL	ABNORMAL	ABNORMAL	NCC	
49	ANBARASI	28	F	**FOCAL SEIZURES WITH DYSCOGNITIVE FEATURES**	YES	SOMATOSENSORY	NO		ABNORMAL	ABNORMAL	ABNORMAL	TUBERCULOMA	
50	PRIYANGA	18	F	FOCAL SEIZURES **WITHOUT** DYSCOGNITIVE FEATURES	NO				ABNORMAL	ABNORMAL	ABNORMAL	NCC	