View metadata, citation and similar papers at core.ac.uk

brought to you by CORE

provided by ePrints@TNMGRM (Tamil Nadu Dr. M.G.R. Medical University)

CLINICAL PROFILE AND EVALUATION OF NEW ONSET SEIZURE IN ADULTS

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI

In Partial Fulfillment of the Regulations

for the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI

April - 2014

BONAFIDE CERTIFICATE

This is to certify that **"CLINICAL PROFILE AND EVALUATION OF NEW ONSET SEIZURE IN ADULTS"** is a bonafide work performed by **Dr.DHANASEKAR.M.**, post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment of regulations of the Tamil Nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine) during the academic period from May 2011 to April 2014.

Prof. Dr. N. Gunasekaran M.D., DTCD
Medical Superintendent & Director INCD
Professor and HOD,
Department of Medicine
KMC & GRH
Chennai.

Prof. Dr.S.Ushalakshmi M.D.,FMMC.,
Professor and Unit chief,
Department of Medicine,
Kilpauk Medical College,
Chennai

Prof. P. Ramakrishnan M.D., D.L.O
The DEAN
Govt.Kilpauk Medical College
Chennai - 600 010

DECLARATION

I solemnly declare that this dissertation **"CLINICAL PROFILE AND EVALUATION OF NEW ONSET SEIZURE IN ADULTS"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Dr.S.Ushalakshmi M.D.**,**FMMC.,** Professor of Internal Medicine, Government Kilpauk Medical College and Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

Place: Chennai **(Dr.DHANASEKAR.M)**

Date:

ACKNOWLEDGEMENT

At the outset, I would like to thank my beloved Dean, Kilpauk Medical College **Prof. Dr. P. Ramakrishnan, M.D., D.L.O.,** for his kind permission to conduct the study in Kilpauk Medical College. I would like to express my special thanks to medical superintendent **Prof. Dr.Narayanababu.MD.,** for permitting to conduct this study in Kilpauk Medical College Hospital.

It gives me immense pleasure to express my sincere and deep gratitude to **Prof. Dr. N. Gunasekaran M.D., DTCD.,** Medical Superintendent and Director INCD, Professor and Head of the Department of Medicine, Kilpauk Medical College, for rendering permission to do this dissertation.

I would like to thank wholeheartedly, **Prof.Dr.S.Ushalakshmi M.D.,FMMC.,** my Unit chief and Professor of Medicine for her encouragement and guidance during the study.

With extreme gratitude, I express my indebtedness to **Prof. Dr.Sarala M.D., D.M.,** Former Professor and HOD, Department of Neurology, Kilpauk Medical College Hospital for her continuous motivation, timely advice and valuable criticism which enabled to complete the dissertation.

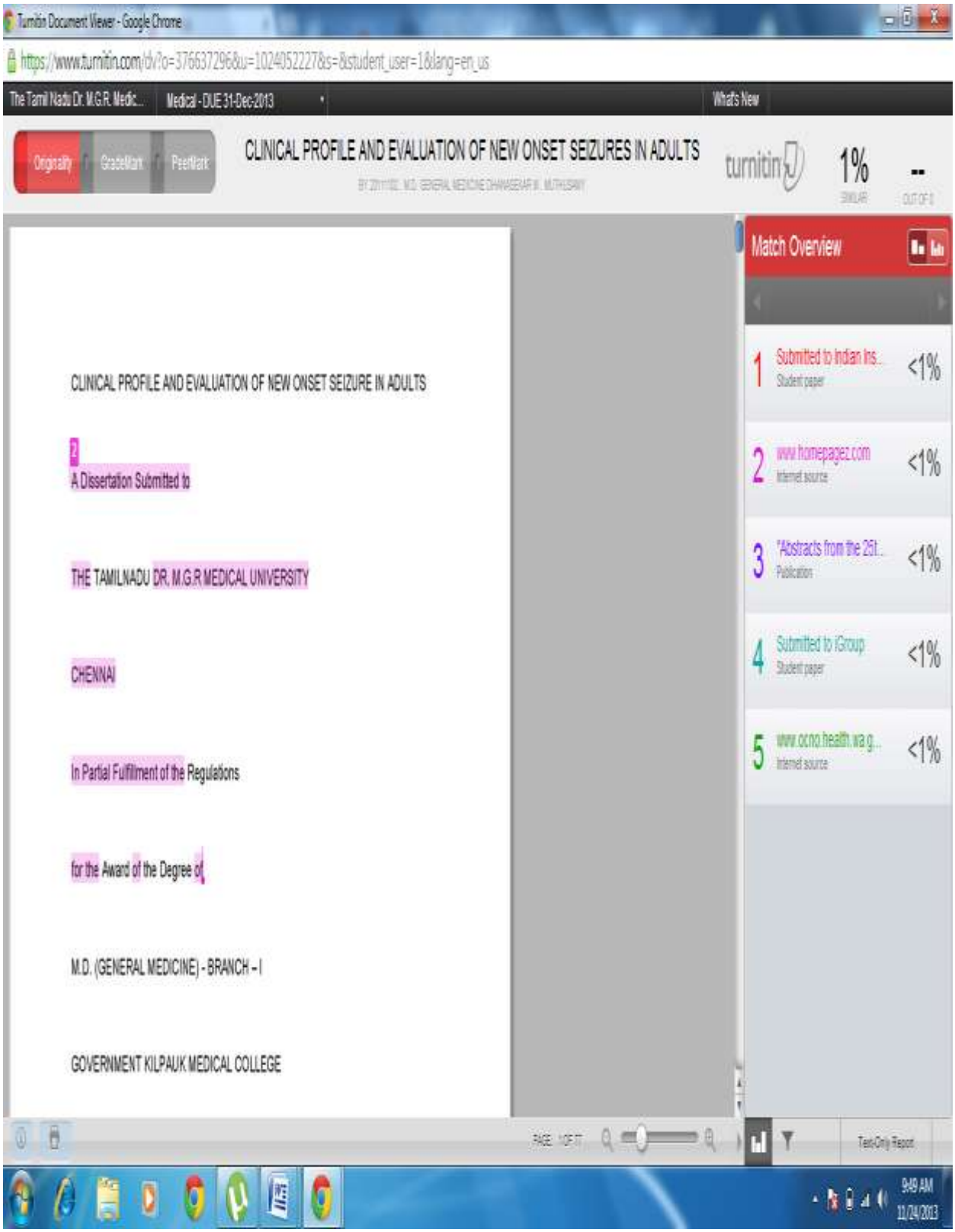
I also express my special thanks to **Prof. Dr.G.Balan M.D., Prof. Dr.T. Ravindran M.D., DNB., Dip Diabetology** and **Prof. D. Surendran M.D.,** I am extremely thankful to Assistant Professor of Medicine, **Dr.M.Bathragiri M.D.,**and **Dr.A.Marimuthu M.D.,** for their assistance and guidance.

I would like to take this opportunity to show gratitude to my Father **Mr.K.Muthusamy**, my Mother Smt**.M.Shanthamani,** my brother **Mr.M.Subramani** and my dear brother and best friend **Dr. S.Ganesh Aravind,** for their never ending support in finishing this thesis.

I would always remember with extreme sense of thankfulness for the co-operation and criticism shown by my fellow post graduate colleague and friends.I pray Almighty God to give me strength to achieve in all my endeavours.

Finally, I wholeheartedly thank all my patients for their active co-operation in this study, without which this would not have become a reality.

CONTENTS


Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?o=376637296&u=1024052227&s=&student_user=1&lang=en_us
The Tamil Nadu Dr. M.G.R. Medic...
Medical - DUE 31-Dec-2013
What's New
Originality
GradeMark
PeerMark
CLINICAL PROFILE AND EVALUATION OF NEW ONSET SEIZURES IN ADULTS
turnitin
1%
SIMILAR
--
OUT OF
CLINICAL PROFILE AND EVALUATION OF NEW ONSET SEIZURE IN ADULTS
A Dissertation Submitted to
THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY
CHENNAI
In Partial Fulfilment of the Regulations
for the Award of the Degree of
M.D. (GENERAL MEDICINE) - BRANCH – I
GOVERNMENT KILPAUK MEDICAL COLLEGE
Match Overview
1 Submitted to Indian Ins.. Student paper <1%
2 www.homepagez.com Internet source <1%
3 "Abstracts from the 25t.. Publication <1%
4 Submitted to iGroup Student paper <1%
5 www.ocnp.health.wa.g... Internet source <1%
PAGE 1 OF 77
Text-Only Report
9:49 AM
11/24/2013

Turnitin Originality Report

CLINICAL PROFILE AND EVALUATION OF NEW ONSET SEIZURES IN ADULTS by 20111102 . M.d. General Medicine DHANASEKAR M . MUTHUSAMY

From Medical (The Tamil Nadu Dr. M.G.R. Medical University)

Similarity Index	Similarity by Source	
1%	Internet Sources:	1%
	Publications:	0%
	Student Papers:	1%

Processed on 24-Nov-2013 09:14 IST
ID: 376637296
Word Count: 9746

sources:

1 < 1% match (student papers from 12-Sep-2013)

Submitted to Indian Institute of Science, Bangalore on 2013-09-12

---

2 < 1% match (Internet from 04-Apr-2008)

http://www.homepagez.com/shmily/floppy.htm

---

3 < 1% match (publications)

"Abstracts from the 25th International Epilepsy Congress Lisbon, Portugal October 12-16, 2003", Epilepsia, 10/2003

ABSTRACT

Background: Seizures beginning in adult life are likely to be an identifiable cause as compared to those beginning in childhood which are more likely to be idiopathic.

Objectives: To study the clinical profile and analyze the etiological agents of New Onset Seizures.

Material and Methods: This Descriptive study done in the KMC hospital to know the various etiologies in patients presented with new onset seizures.In these cases history and clinical examination and special investigations like CT BRAIN, MRI BRAIN,EEG, SEROLOGY, CSF ANALYSIS were done to find out the etiology.

Results: Out of 100 patients 55% were males,45%were females with male to female ratio of 1.2:1.Majority of males were in 2^{nd} decade and females were in 4^{th} decade.Patients age ranged from 18 yrs to 80 yrs,with the mean of 40.11years with 77% of the patients were in the below 50 yrs. Alcohol withdrawal was the leading cause of seizures which account for 34% followed by idiopathic seizures (29%),neuro infection (16%),CVA 12% and metabolic (9%).

Conclusion: Alcohol withdrawal is the most common cause of seizure in new onset seizure patients who coming to KMCH.

Keywords: New onset seizure, Alcoholwithdrawal,Tuberculoma, Neurocysticercosis,Meningitis, Metabolic seizure.

INTRODUCTION

Epilepsy describes a condition in which a person has recurrent seizures due to chronic underlying process..

Epilepsy of late onset (*Epilepsia tarda, late onset epilepsy*) may be simply defined as epilepsy beginning in adult life.

Epilepsy beginning in adult life is due to progressive brain disease as compared to idiopathic epilepsy, which has, it's onset in childhood or adolescence.

With proper history and clinical examination, analysis of etiology is made with available investigations, the epilepsy can be treated accordingly thus reducing the morbidity and mortality associated with it.

Hence, this study is aimed to evaluate the clinical profile and etiological analysis of new onset epilepsy in adults of more than 18 years of age.

REVIEW OF LITERATURE

HISTORY OF EPILEPSY :

A *seizure* (from the Latin *sacire*, " to take possession of ") is a paroxysmal event due to abnormal excessive or synchronous neuronal activity in the brain.

From the Greek word “epilambanein ” the term “epilepsy” comes which means “to seize” or “ to take hold of ”.

Approximately 500 years before Hippocrates, Atreya, the father of Indian Medicine, recognized that disturbance of mind is epilepsy, rather than an effect of supernatural phenomena. Hippocrates, in his book “on the scared disease”noted, “The brain is the seat of epilepsy as it is of every violent disease”.

The idea that epilepsy was caused by demons, humors, and toxic substances persisted through the middle ages.

The ideas of supernatural causation slowly died out only to be replaced by another set of bizarre misnomers. Obstetrician to Queen Victoria, Sir Charles Locock, Credited crowded teeth, menstruation and masturbation with causing

seizures. Dr.Locock pioneered the use of potassium bromide the first modern antiepileptic drug treatment.He had found that women with “hysterical”seizures associated with menstruation,was thought to curb the sexual desires.

In 1857 after the start of bromide treatments, modern epilepsy treatment proceeded at a quick pace. In 1912 Phenobarbitone was introduced and 24 years later to that, pioneer work on phenytoin was carried out. Drugs like Carbamazepine, sodium valproate and vigabatrin followed in 1954, 1973 and 1990 respectively. Now people with seizures are confronted with an array of confusing but it often effective, treatments.

We can look forward in future to a deeper understanding of alternative treatments and advances in diagnosis and treatment through progress in surgery ,biochemistry,and possibly even neural grafts and molecular genetics[(1)].

EPIDEMIOLOGY OF EPILEPSY

PREVALENCE :

It is the proportion of people with seizure in given population at a specified time. The worldwide prevalence of active seizure is between 4-10 per 1000 population[(2)]. The prevalence rate in India is 5.59 per 1000.There is no statistically different rates between women and men or urban and rural residence[(3)].

INCIDENCE:

The number of new cases of seizure occurring during a given time interval, usually one year, in a specified population is called as incidence. Incidence rate varies from 38 to 49.3 per 1, 00,000 population per year from two community based studies in India.[4]

Type of seizure pattern showed maximum number of cases belonged to generalized seizures, which is different from western countries where partial seizure is the commonest variety. (The proportion of generalized seizures and partial seizures was 58.8% and 30.6% respectively)[5].

The prognosis for seizure control is good and over 70% will enter remission. There is an increased risk of premature death particularly in patients with chronic epilepsy[6].

DIAGNOSIS OF EPILEPSY

It is rare to observe a seizure directly at the first medical examination or at an outpatient clinic. The confirmation and diagnosis of the seizure type usually based on the history taken from the patients or caregivers. First we have to distinguish epileptic seizures from the non- epileptic attacks, such as psychogenic seizures and syncopal attacks. In distinguishing a syncopal attack from an epileptic seizure, we must pay attention to the sensation of faintness or feeling of “blackouts” immediately before loss of consciousness and the presence of provoking factors,

such as noxious stimuli, sudden unexpected pain or standing for a long time,. Next we must ask the following questions directly to the patients or indirectly to the care givers to make a precise seizure diagnosis including the aura, asymmetry of the seizures, content, clouded consciousness, presence of automatism, deviation of the head and eyes and a dystonic arm posture[7].

Table 369-1 Classification of Seizures

1. **Focal seizures**

   (Can be further described as having motor, sensory, autonomic, cognitive, or other features)

2. **Generalized seizures**
   - a. Absence

     Typical

     Atypical
   - b. Tonic clonic
   - c. Clonic
   - d. Tonic
   - e. Atonic
   - f. Myoclonic

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

   Epileptic spasms

A fundamental principle is that seizures may be either focal or generalized. Generalized seizures involve diffuse regions of the brain simultaneously. Focal seizures are those in which the seizure activity is restricted to discrete areas of the cerebral cortex. Generalized seizures may result from biochemical, cellular abnormality or structural abnormalities that have a more widespread distribution. .

In contrast, focal seizures are usually associated with structural abnormalities of the brain.

Focal Seizure

Focal seizures arise from a neuronal network either discretely localized within one cerebral hemisphere or more broadly distributed but still within the hemisphere.The new classification system, described it as focal seizures with or without dyscognitive features.

Focal seizures can also evolve into generalized seizures. In the past this was referred to as *focal seizures with secondary generalization*, but the newer system relies on specific descriptions of the type of generalized seizures that evolve from the focal seizure.

Focal Seizures Without Dyscognitive Features

Focal seizures can cause autonomic, motor, sensory or psychic symptoms without impairment of cognition.The EEG recorded with scalp electrodes during the seizure may show abnormal discharges in a very limited region over the appropriate area of cerebral cortex if the seizure focus involves the cerebral convexity. Seizure activity from deeper brain structures is often not recorded by the standard EEG, it may require intracranial electrodes for its detection.

Three additional features of focal motor seizures are,

1. In some patients the abnormal motor movements may begin in a very restricted region such as the fingers and gradually progress to include a larger portion of the extremity. This phenomenon known as a "Jacksonian march" which represents the spread of seizure activity over a progressively larger region of motor cortex.
2. Patients may experience a localized paresis (Todd's paralysis) for minutes to many hours in the involved region following the seizure.
3. In rare instances the seizure may continue for hours or days.It termed *epilepsia partialis continua*, also it is often refractory to medical therapy.

Focal seizures may also manifest as changes in vision (flashing lights or formed hallucinations), equilibrium (sensation of falling or vertigo), or autonomic function (flushing, sweating, piloerection) somatic sensation (e.g., paresthesias). Focal seizures arising from the temporal or frontal cortex may also cause alterations in olfaction, hearing, or higher cortical function sensation of unusual, intense odors (e.g., burning rubber or kerosene) or an epigastric sensation that rises from the stomach or chest to the head. Patients describe odd, internal feelings such as fear, detachment, a sense of impending change, depersonalization, déjá vu, or illusions that objects are growing smaller (micropsia) or larger (macropsia). These

subjective, "internal" events that are not directly observable by someone else are referred to as *auras*.

Focal Seizures with Dyscognitive Features

Focal seizures accompanied by a transient impairment of the patient's ability to maintain normal contact with the environment. These patients unable to respond appropriately to verbal or visual commands during the seizure and has impaired awareness or recollection of the ictal phase. Seizures frequently begin with an aura that is stereotypic for the patient. The start of the ictal phase is a sudden behavioral arrest or motionless stare. The behavioral arrest is usually accompanied by *automatisms*, which are involuntary, automatic behaviors such as chewing, lip smacking, swallowing, or "picking" movements of the hands, or elaborate behaviors such as a display of emotion or running. These patients typically confused following the seizure, and the full recovery of consciousness may range from seconds up to an hour. Immediately following the seizure may show anterograde amnesia or, in cases involving the dominant hemisphere, a postictal aphasia.

Evolution of Focal Seizures to Generalized Seizures: Focal seizures can spread to involve both cerebral hemispheres and produce a generalized seizure, usually of the tonic-clonic variety . This evolution is observed following focal seizures

arising from a focus in the frontal lobe, but may also be associated with focus elsewhere in the brain. A focal seizure that evolves into a generalized seizure is often difficult to distinguish from a primary generalized-onset tonic-clonic seizure.

Generalized Seizures

Generalized seizures are arise at some point in the brain but immediately and rapidly that engage neuronal networks in both cerebral hemispheres..

Typical Absence Seizures

It characterized by sudden, brief lapses of conscious without loss of postural control. It typically lasts for only seconds, consciousness returns as suddenly as it was lost, and there is no postictal confusion. It usually accompanied by subtle,bilateral motor signs such as chewing movements, rapid blinking of the eyelids, or small-amplitude, clonic movements of the hands. In typical absence seizures the onset usually in childhood (ages 4–8 years) or early adolescence and are the main seizure type in 15–20% of children with epilepsy. Seizures can occur 100 of times per day, but the child may be unaware or unable to convey their existence. Absence epilepsy is often unexplained "daydreaming" and a decline in school performance recognized by a teacher. The EEG of typical absence seizures

is a generalized, symmetric, 3-Hz spike - and - wave discharges that begins and ends suddenly, superimposed on a normal EEG background.

Atypical Absence Seizures

The loss of consciousness is usually of longer duration and slow in onset and cessation.It also associated with motor signs that includes focal or lateralizing features.EEG shows a generalized,2.5Hz slow spike-and-wave pattern.These are usually associated with diffuse or multifocal structural abnormalities of the brain.So associated with other signs of neurologic dysfunction such as mental retardation.Atypical absence seizures are less responsive to anticonvulsants compared to typical absence seizures.

Generalized, Tonic-Clonic Seizures

These are the main seizure type in 10% of all persons with epilepsy.Also a common seizure type resulting from metabolic derangements. The seizure usually begins abruptly without warning. The initial phase is usually tonic contraction of muscles throughout the body. Tonic contraction of the muscles of expiration and the larynx will produce a loud moan or "ictal cry." Contraction of the jaw muscles may cause biting of the tongue. After 10–20 seconds, the seizure typically evolves into the clonic phase, produced by the periods of muscle relaxation on the tonic

muscle contraction. It progressively increase until the end of the ictal phase. The postictal phase is characterized by muscular flaccidity, unresponsiveness, and excessive salivation. Bladder or bowel incontinence may occur. Patients gradually regain consciousness over minutes to hours, and in this transition there is typically a period of postictal confusion. The duration of unconsciousness in the postictal phase can be extremely long in patients with prolonged seizures or underlying CNS diseases such as alcoholic cerebral atrophy.The EEG during the tonic phase shows a progressive increase in generalized low-voltage fast activity, followed by high-amplitude, polyspike discharges. The high-amplitude activity is interrupted by slow waves to create a spike-and-wave pattern in the clonic phase.

Atonic Seizure

These are characterized by sudden loss of postural muscle tone lasting 1–2 seconds. Brief impairment in Consciousness, but there is usually no postictal confusion. The EEG shows brief, generalized spike-and-wave discharges followed by diffuse slow waves that correlate with the loss of muscle tone.

Myoclonic Seizures

Myoclonus is a sudden and brief muscle contraction that may involve one part of the body or the entire body. A common physiologic form of myoclonus is

the sudden jerking movement observed during asleep. Pathologic myoclonus is commonly seen in association with anoxic brain injury, metabolic disorders and degenerative CNS diseases. The EEG shows bilaterally synchronous spike-and-wave discharges synchronized with the myoclonus

Table 369-2 Examples of Genes Associated with Epilepsy Syndromes[a]

Gene (Locus)	Function of Gene	Clinical Syndrome	Comments
CHRNA4 (20q13.2)	Nicotinic acetylcholine receptor subunit; mutations cause alterations in Ca2+ flux through the receptor; this may reduce amount of GABA release in presynaptic terminals	Autosomal dominant nocturnal frontal lobe epilepsy (ADNFLE); childhood onset; brief, nighttime seizures with prominent motor movements; often misdiagnosed as primary sleep disorder	Rare; first identified in a large Australian family; other families found to have mutations in CHRNA2 or CHRNB2, and some families appear to have mutations at other loci
KCNQ2 (20q13.3)	Voltage-gated potassium channel subunits; mutation in pore regions may cause a 20–40% reduction of potassium currents, which will lead to impaired repolarization	Benign familial neonatalconvulsions (BFNC); autosomal dominant inheritance; onset in 1st week of life in infants who are otherwise normal; remission usually within weeks to months; long-term epilepsy in 10–15%	Rare; other families found to have mutations in KCNQ3; sequence and functional homology to KCNQ1, mutations of which cause long QT syndrome and a cardiac-auditory syndrome
SCN1B (19q12.1)	β-subunit of a voltage-gated sodium channel; mutation disrupts disulfide bridge that is crucial for structure of extracellular domain; mutated β-subunit leads to slower sodium channel inactivation	Generalized epilepsy with febrile seizures plus (GEFS+); autosomal dominant inheritance; presents with febrile seizures at median 1 year, which may persist >6 years, then variable seizure types not associated with fever	Incidence uncertain; GEFS+ identified in other families with mutations in other sodium channel subunits (SCN1A and SCN2A) and GABAA receptor subunit (GABRG2 and GABRA1); significant phenotypic heterogeneity within same family, including members with febrile seizures only
LGI1 (10q24)	Leucine-rich glioma-inactivated 1 gene; previous evidence for role in glial tumor progression; protein homology suggests a possible role in nervous system development	Autosomal dominant partial epilepsy with auditory features (ADPEAF); a form of idiopathic lateral temporal lobe epilepsy with auditory symptoms or aphasia as a major simple partial seizure manifestation; age of onset usually between 10 and 25 years	Mutations found in approximately 50% of families containing two or more subjects with idiopathic localization-related epilepsy with ictal auditory symptoms, suggesting that at least one other gene may underlie this syndrome. LGI1 is the only gene identified so far in temporal lobe epilepsy
CSTB (21q22.3)	Cystatin B, a noncaspase cysteine protease inhibitor; normal protein may block neuronal apoptosis by inhibiting caspases directly or indirectly (via cathepsins), or controlling proteolysis	Progressive myoclonus epilepsy (PME) (Unverricht-Lundborg disease); autosomal recessive inheritance; age of onset between 6 and 15 years, myoclonic seizures, ataxia, and progressive cognitive decline; brain shows neuronal degeneration	Overall rare, but relatively common in Finland and Western Mediterranean (>1 in 20,000); precise role of cystatin B in human disease unknown, although mice with null mutations of cystatin B have similar syndrome
EPM2A (6q24)	Laforin, a protein tyrosine phosphatase (PTP); involved in glycogen metabolism and may have antiapoptotic activity	Progressive myoclonus epilepsy (Lafora's disease); autosomal recessive inheritance; onset age 6–19 years, death within 10 years; brain degeneration associated with polyglucosan intracellular inclusion bodies in numerous organs	Most common PME in Southern Europe, Middle East, Northern Africa, and Indian subcontinent; genetic heterogeneity; unknown whether seizure phenotype due to degeneration or direct effects of abnormal laforin expression
Doublecortin (Xq21-24)	Doublecortin, expressed primarily in frontal lobes; directly regulates microtubule polymerization and bundling	Classic lissencephaly associated with severe mental retardation and seizures in males; subcortical band heterotopia with more subtle findings in females (presumably due to random X-inactivation); X-linked dominant	Relatively rare but of uncertain incidence, recent increased ascertainment due to improved imaging techniques; relationship between migration defect and seizure phenotype unknown

Juvenile Myoclonic Epilepsy

It is a generalized seizure disorder of unknown cause that occurs in early adolescence.It is usually characterized by single or repetitive bilateral myoclonic

jerks. The myoclonic seizures can be provoked by sleep deprivation and most frequent in the morning after awakening. Consciousness is preserved unless the myoclonus is especially severe.

Lennox-Gastaut Syndrome

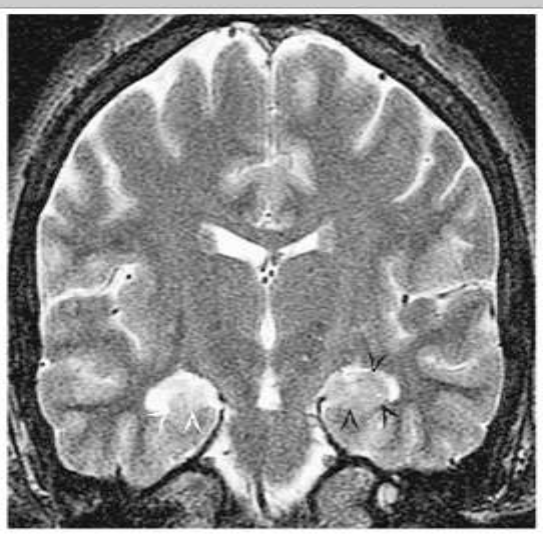
It occurs in children and is defined by the following triad: (1) multiple seizure types (including atonic, generalized tonic-clonic and atypical absence seizures); (2) impaired cognitive function in most but not all cases,(3) an EEG showing slow (<3 Hz) spike-and-wave discharges.It is associated with CNS disease or dysfunction due to developmental abnormalities, trauma, infection, perinatal hypoxia/ischemia, and other acquired lesions. Many patients have a poor prognosis due to the underlying CNS disease.

Mesial Temporal Lobe Epilepsy Syndrome

It is the most common syndrome associated with focal seizures with dyscognitive. High-resolution MRI can detect hippocampal sclerosis that appears to be essential in the pathophysiology of MTLE for many patients.

Table 369-3 Characteristics of the Mesial Temporal Lobe Epilepsy Syndrome	
History	
History of febrile seizures	Rare generalized seizures
Family history of epilepsy	Seizures may remit and reappear
Early onset	Seizures often intractable
Clinical observations	
Aura common	Postictal disorientation
Behavioral arrest/stare	Memory loss
Complex automatisms	Dysphasia (with focus in dominanthemisphere)
Unilateral posturing	
Laboratory studies	
Unilateral or bilateral anterior temporal spikes on EEG	
Hypometabolism on interictal PET	
Hypoperfusion on interictal SPECT	
Material-specific memory deficits on intracranial amobarbital (Wada) test	
MRI findings	
Small hippocampus with increased signal on T2-weighted sequences	
Small temporal lobe	
Enlarged temporal horn	
Pathologic findings	
Highly selective loss of specific cell populations within hippocampus in most cases	

Figure 369-1



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: Harrison's *Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

Copyright © The McGraw-Hill Companies, Inc. All rights reserved.

Mesial temporal lobe epilepsy. The EEG suggested a right temporal lobe focus. Coronal high-resolution T2-weighted fast spin echo magnetic resonance image obtained through the body of the hippocampus demonstrates abnormal high-signal intensity in the right hippocampus (*white arrows*; compare with the normal hippocampus on the left, *black arrows*) consistent with mesial temporal sclerosis.

The Causes of Seizures and Epilepsy

Shift in the normal balance of excitation and inhibition within the CNS causes seizures. Three clinical observations emphasize how a variety of factors determine why certain conditions may cause seizures or epilepsy in a given patient.

1) The normal brain is capable of having a seizure under the appropriate circumstances, and individuals vary in the susceptibility or threshold for seizures. For eg, seizures may be induced by high fevers in children who are otherwise normal and they may never develop other neuro logic problems, including epilepsy.Febrile seizures occur only in a relatively small proportion of children.It implies there are various underlying, endogenous factors which influence the threshold for having a seizure.

2) There are varieties of conditions that have an extremely high likelihood of resulting in a chronic seizure disorder. One of the examples of this is severe, penetrating head injury, which is associated with upto a 45% risk of subsequent epilepsy.A process is known as epileptogenesis which is, high propensity for severe traumatic brain injury lead to epilepsy suggests that the injury results in a long-lasting, pathologic changes in the CNS that transforms a presumably normal neural network into a abnormally hyperexcitable. Other processes associated with

epileptogenesis include infections, stroke, and abnormalities of CNS development.

3) Seizures are episodic. Patients with epilepsy have seizures intermittently.Depending on the underlying cause, many patients are completely normal for months to years between seizures. It shows there are important precipitating or provocative factors that induce seizures in patients with epilepsy.

Causes According to Age

Table 369-4 Causes of Seizures	
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 Genetic disorders
Infants and children (>1 month and <12 years)	Febrile seizures Genetic disorders (metabolic, degenerative, primary epilepsy syndromes) CNS infection 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 (>35 years)	Cerebrovascular disease Brain tumor Alcohol withdrawal Metabolic disorders (uremia, hepatic failure, electrolyte abnormalities, hypoglycemia, hyperglycemia) Alzheimer's disease and other degenerative CNS diseases Idiopathic

Table 369-5 Drugs and Other Substances that Can Cause Seizures
Alkylating agents (e.g., busulfan, chlorambucil)
Antimalarials (chloroquine, mefloquine)
Antimicrobials/antivirals
β-lactam and related compounds
Quinolones
Acyclovir
Isoniazid
Ganciclovir
Anesthetics and analgesics
Meperidine
Tramadol
Local anesthetics
Dietary supplements
Ephedra (ma huang)
Gingko
Immunomodulatory drugs
Cyclosporine
Tacrolimus
Interferons
Psychotropics
Antidepressants
Antipsychotics
Lithium
Radiographic contrast agents
Theophylline
Sedative-hypnotic drug withdrawal
Alcohol
Barbiturates (short-acting)
Benzodiazepines (short-acting)
Drugs of abuse
Amphetamine
Cocaine
Phencyclidine
Methylphenidate
Flumazenil[a]

[a]In benzodiazepine-dependent patients.

Mechanisms of Seizure Initiation and Propagation

Focal seizure activity can begin in a discrete region of cortex and then spread to neighboring regions. There is a *seizure initiation* phase followed by a *seizure propagation* phase.

Initiation phase characterized by the following the two concurrent events,

(1) high-frequency bursts of action potentials and (2) hypersynchronization.

The bursting activity is caused by a long-lasting depolarization of the neuronal membrane due to influx of extracellular Ca^{2+},this leads to the opening of voltage-dependent Na^{+}channels, influx of Na^{+} & generation of repetitive action potentials. Followed by a hyperpolarizing afterpotential mediated by GABA receptors or K^{+} channels, depending on the cell type. The spike discharge on the EEG is due to synchronized bursts from a sufficient number of neurons.Normally, intact hyperpolarization and a region of "surround" inhibition created by Inhibitors prevents the spread of bursting activity. There is a recruitment of surrounding neurons via one of the following four mechanisms, including:

1)An increase in extracellular potassium, which blunts hyperpolarization and depolarizes neighboring neurons;

(2) Enhanced neurotransmitter release due to accumulation of calcium in presynaptic terminals;

(3) Depolarization-induced activation of the NMDA receptor, which causes additional calcium influx and neuronal activation;

(4) Changes in tissue osmolarity and cell swelling. Propagation of seizure activity into contiguous areas via local cortical connections due to recruitment of a sufficient number of neurons and to distant areas through the long commissural pathways such as the corpus callosum.

Accidental ingestion of domoic acid, which is an analogue of glutamate causes profound seizures via direct activation of excitatory receptors throughout the CNS. Commonly used Penicillin, which can lower the seizure threshold in humans but in experimental models it reduces inhibition by antagonizing the effects of GABA and act as a potent convulsant.

Basic mechanisms of other precipitating factors of seizures such as fever, alcohol withdrawal, infection, sleep deprivation and hypoxia are not as well understood. The generalized spike-and-wave discharges in absence seizures is due to oscillatory rhythms which is normally generated during sleep by the circuits connecting the thalamus and cortex. This involves an interaction between $GABA_B$

receptors, T-type calcium channels, and potassium channels located within the thalamus. This modulation of these receptors and channels can induce absence seizures.

Alcohol causes intoxication through effects on diverse ion channels and neurotransmitter receptors, including $GABA_A$ receptors—particularly those containing δ subunits that are localized extrasynaptically and mediate tonic inhibition and N-methyl-*D*-aspartate (NMDA) receptors.

Alcohol dependence results from compensatory changes during prolonged alcohol exposure, including internalization of $GABA_A$ receptors, which allows adaptation to these effects.

Withdrawal seizures are due to reflect unmasking of these changes and may also involve specific withdrawal-induced cellular events, such as rapid increases in α4 subunit–containing $GABA_A$ receptors that confer reduced inhibitory function.

Management of Seizures :

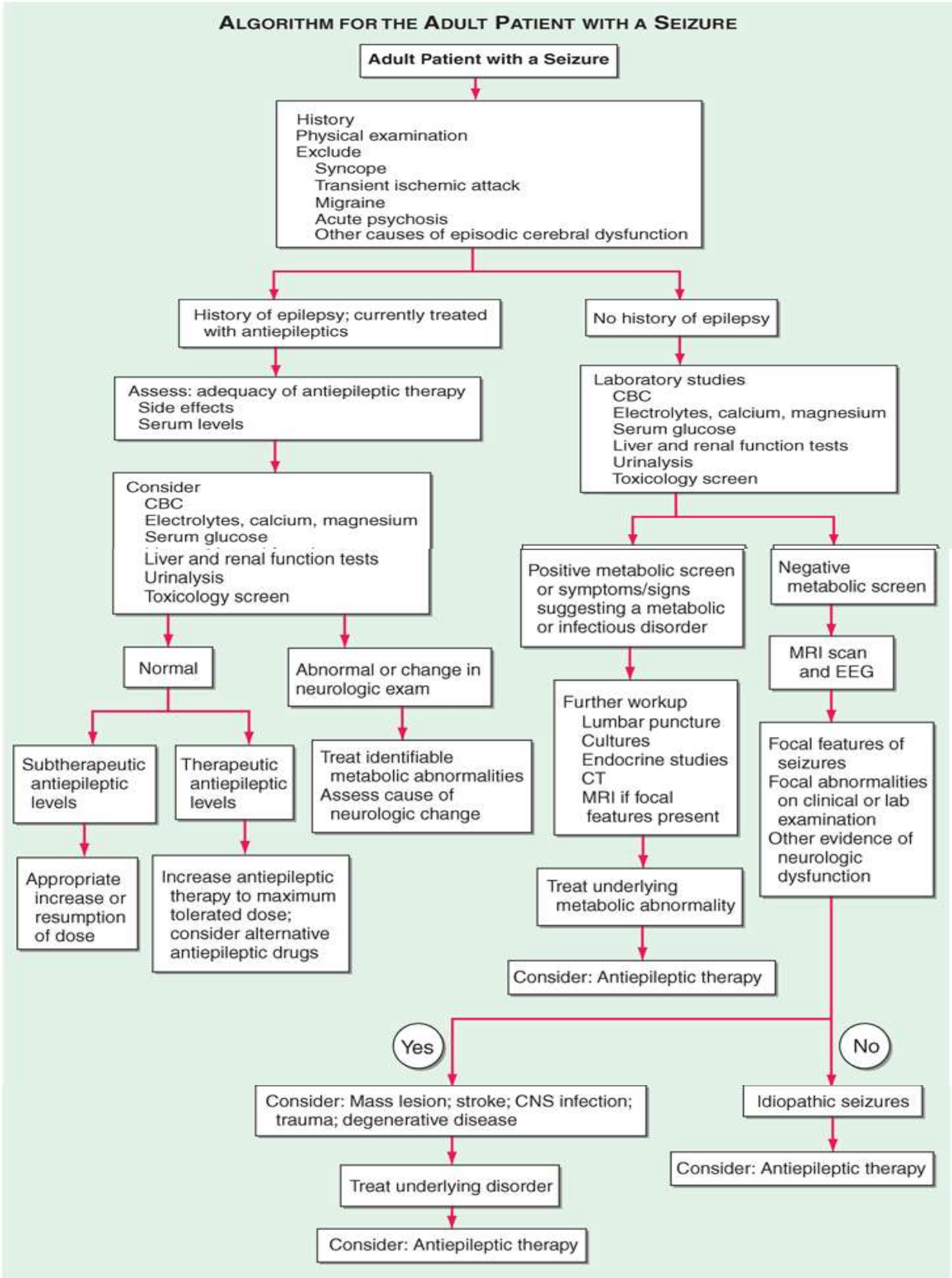
History and Examination

The first goal is to determine whether the event was a true seizure. An in-depth history is important, for *in many cases the diagnosis of a seizure is based solely on clinical grounds—the examination and laboratory studies are often normal*. Focused on the symptoms before, during, and after the episode of seizure the questions should be asked, in order to differentiate a seizure from other paroxysmal events.

The history should also focus on predisposing events and risk factors. Epileptogenic factors such as prior stroke, head trauma, tumor, or CNS infection should be identified. History of febrile seizures, brief seizures not recognized as such,earlier aura and a family history of seizures should be asked to get a clues regarding predisposition.Ask for developmental milestones in children,it may provide evidence for underlying CNS disease. Precipitating factors such as systemic diseases, sleep deprivation, electrolyte or metabolic derangements, drugs that lower the seizure threshold, acute infection, or illicit drug or alcohol use should also be identified.

On general examination look for signs of infection or systemic illness. Look for signs of neurocutaneous disorders such as neurofibromatosis, tuberous sclerosis or chronic liver or renal disease. Presence of organomegaly may indicate a metabolic storage disease.Presence of limb asymmetry may gives a clue to brain injury early in development. Look for signs of head trauma, alcohol or illicit drugs uses. Auscultation of the CVS and carotid arteries may help to identify an abnormality that predisposes to cerebrovascular disease.

All patients need a complete neurologic examination. Careful assessment of mental status (including memory, abstract thinking, language function) may suggest lesions in the anterior frontal, temporal or parietal lobes. Testing of visual fields will help to screen for lesions in the optic pathways and occipital lobes.Pronator drift,abnormal deep tendon reflexes, gait, and inco-ordination may suggest lesions in motor (frontal) cortex, and cortical sensory testing may detect lesions in the parietal cortex.


ALGORITHM FOR THE ADULT PATIENT WITH A SEIZURE
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
Laboratory studies
CBC
Electrolytes, calcium, magnesium
Serum glucose
Liver and renal function tests
Urinalysis
Toxicology screen
Consider
CBC
Electrolytes, calcium, magnesium
Serum glucose
Liver and renal function tests
Urinalysis
Toxicology screen
Positive metabolic screen or symptoms/signs suggesting a metabolic or infectious disorder
Negative metabolic screen
Normal
Abnormal or change in neurologic exam
MRI scan and EEG
Subtherapeutic antiepileptic levels
Therapeutic antiepileptic levels
Treat identifiable metabolic abnormalities
Assess cause of neurologic change
Further workup
Lumbar puncture
Cultures
Endocrine studies
CT
MRI if focal features present
Focal features of seizures
Focal abnormalities on clinical or lab examination
Other evidence of neurologic dysfunction
Appropriate increase or resumption of dose
Increase antiepileptic therapy to maximum tolerated dose; consider alternative antiepileptic drugs
Treat underlying metabolic abnormality
Consider: Antiepileptic therapy
Yes
No
Consider: Mass lesion; stroke; CNS infection; trauma; degenerative disease
Idiopathic seizures
Treat underlying disorder
Consider: Antiepileptic therapy
Consider: Antiepileptic therapy

LABORATORY EVALUATION :

Routine blood investigations are indicated to identify the common metabolic causes of seizures like abnormalities in electrolytes, glucose, magnesium or calcium, and hepatic or renal disease. A screen for toxins in urine and blood should also be obtained when no clear precipitating factor has been identified. A lumbar puncture is indicated in suspicion of meningitis or encephalitis and even in the absence of symptoms or signs suggesting infection it is mandatory in all patients infected with HIV.

Electroencephalography(EEG):

By recording from electrodes placed on the scalp, the EEG measures electrical activity of the brain. The potential difference between pairs of electrodes is amplified and displayed on a computer monitor, oscilloscope, or paper. The characteristics of the normal EEG depend on the patient's level of arousal and age. The recorded activity is the postsynaptic potentials of pyramidal cells in the cerebral cortex and is characterized by its frequency.

The EEG is best recorded by several different electrode arrangement in turn, and the following activating procedures like hyperventilation (for 3 or 4 min), sleep, sleep deprivation on the night pior to the recording and photic stimulation are usually performed in an attempt to provoke abnormalities.

In a patient with suspected epilepsy during evaluation, the presence of electrographic seizure activity like abnormal, repetitive, rhythmic activity with an abrupt onset and termination, clearly establishes the diagnosis. But , absence of electrographic seizure activity does not exclude a seizure disorder. Because simple or complex seizures may originate from a region of cortex that is not within range of the scalp electrodes. During generalized tonic – clonic seizures the EEG is always abnormal.

The EEG may also be supportive of the diagnosis of epilepsy during the inter-ictal period by showing certain abnormalities. Epileptiform activity consists of bursts of abnormal discharges containing spikes or sharp waves.The EEG is also used for classifying seizure disorders and for the selection of anticonvulsant medications . The routine scalp - recorded EEG is also used to assess the prognosis of seizure disorders; A normal EEG implies a better prognosis, whereas profuse epileptiform activity suggests a poor outlook.

Brain Imaging

Patients with new- onset seizures should have a brain imaging study to rule out an underlying structural abnormality that is responsible . For the detection of cerebral lesions associated with epilepsy MRI has been shown to be superior to computed tomography (CT). MRI will identify lesions such as vascular malformations, tumors, or other pathologies that need immediate therapy. Fluid-

attenuated inversion recovery (FLAIR) is the newer MRI method, which has increased the sensitivity for detection of abnormalities of cortical architecture like abnormalities of cortical neuronal migration and hippocampal atrophy associated with mesial temporal sclerosis. In such cases, they do provide an explanation for the patient's seizures and point to need for possible surgical resection or the need for chronic anticonvulsant therapy .

ADULT ONSET IDIOPATHIC GENERALIZED EPILEPSY (AIGE)

It is generally thought to have a focal basis and symptomatic etiology[8]. However in some patients, IGE is suspected because of typical generalized tonic - clonic, myoclonic or absence seizures, a family history of seizures, the generalized spike- wave complexes on EEG, normal brain imaging[9].Two hospital based studies recently reported that 34.8% and 13.4% of IGE cases had seizure onset in adulthood[10,11] .The IGE typically appears within first two decades of life.

The annual age specific incidences of IGE patients aged 15- 24 years were 3.6 per 1,00,000 and 25- 34 years were 3.5 per 1,00,000.Whereas the incidences of IGE patients aged 5- 9 years were 10.7 per 1,00,000 and 10- 14 years were 15.3 per 1,00,000[12].Patients with AIGE have a good prognosis, with good to excellent seizure control with a single AED[13] .

The patients with AIGE can be divided into three groups on the basis of seizure type

1) **Adult onset absence epilepsy:** absence seizures as well as tonic – clonic seizures occurs in these patients.
2) **Adult onset myoclonic epilepsy:** myoclonic jerks and tonic- clonic seizures occurs in these patients.
3) **Adult onset tonic – clonic epilepsy:** only tonic clonic seizures occurs in these patients

POSTSTROKE EPILEPSY :

Most frequent causes of seizures in adulthood, particularly in the old age is stroke. Incidence of seizures after stroke varies from 4.1% to 12.5%[14]. Post stroke epilepsy incidence in India is13%[15].

Temporal relation of stroke and seizures: Seizure may occur before or at the onset of or weeks to months after a stroke.

The incidence of epilepsy prior to stroke was 4.5% when compared to 0.6% in the matched control group[16]. These seizures occur weeks or even years before the presenting stroke. This increased incidence of epilepsy in stroke patients may be

attributed to subclinical cerebral vascular disease Thus, the onset of seizures in adult or elderly population may be warning sign for further strokes.

Post stroke seizures classification:

Early onset seizures: seizures occurring within 2 weeks following stroke onset

Late onset seizures*: seizures* occurring after 2 weeks. This differentiation helps to determine the need and duration of treatment these patients with an AED's[14].

POST STROKE EARLY SEIZURES:

The incidence of epileptic seizures in acute stroke is 4.4% in patients with transient ischemic attacks (TIA), lobar infarcts and extensive hemorrhages. Seizures occur within 24 to 48 hours after the stroke. In a prospective study of 1000 patients with stroke and TIA the incidence of early seizures was (15.4%) in patients with supratentorial lobar or extensive hemorrhages, followed by SAH(8.5%), carotid artery cortical infarction (6.5%), and hemispheric TIA's (3.7%)[18].The commonest site of infarction among the patients with cortical infarcts and early epilepsy is in the MCA territory. Predictably, subcortical and deep cerebral, brain stem infarcts, and infratentorial hemorrhages are not associated with increased risk for seizures. Incidence of seizures is low (1%) in lacunar infarcts. Cerebral embolism patients experience more seizures than

thrombotic infarct patients[19]. Patients with stroke and early seizures had larger lesions(>10 mm) on cerebral CT scan. In acute stage, almost 60% of seizures are focal and 40% are of generalized tonic - clonic type. Of the focal seizures, 75% are simple focal motor while remaining 25% become generalized. In 0.7 to 1.1% of patients with stroke early seizures presenting with status epilepticus (SE).

Predictive factors of early seizures:

The presence of precipitating factors like hypoglycemia, hyperglycemia, hypernatremia ,hyponatremia, hypomagnesemia, hypocalcemia, infections and renal failure increases the incidence of seizures. Increased risk of early seizures also been found in patients with history of diabetes and atrial fibrillation. However, the major predictors of early seizures in new onset stroke patients are cortical involvement in neuro- imaging studies, initial stroke severity and acute confusional state at the onset of stroke[20]. Primary generalized seizure is common with late onset seizures (56%).But early onset seizures, which are generally simple partial in nature.Status epilepticus is more common in early onset than late onset seizures.[24]

Pathophysiology of early post stroke seizures:

In infarct, in the ischemic penumbra ionic imbalances, enhanced release of excitotoxic glutamate, breakdown of membrane phospholipids and release of FFA play an important role in epileptogenesis[14].

Risk of recurrence – There is 11% to 39% risk of recurrent seizures in patients with early stroke seizures. Lesions involving more than one lobe, patients with large hemorrhagic strokes, and cortical infarcts are at higher risk of developing seizures later[21].

Management:

Seizures can be controlled with monotherapy alone. [22]

If the stroke patient presents with status epilepticus or if there is early recurrence of seizures treatment with AED therapy is indicated.

POST STROKE LATE SEIZURES:

It occur after first 2 weeks of stroke.It also may begin months to a year after a stroke. The incidence is about 15%[23]. Nearly 24% of seizures occur within three weeks and 93% in two years.

Pathophysiology of late post stroke epilepsy: it occurs due to structural brain abnormalities leading to the development of an epileptic focus.

Management:

Carbamazepine and Phenytoin have high treatment success in post stroke epilepsy.

SINGLE SMALL ENHANCING CT LESIONS (SSECTL)

In Indian patients with new onset seizures the commonest imaging abnormality is spontaneously resolving single, small, enhancing lesion in CT[25].

Atleast 26% of Indian patients with focal epilepsy have SSECTL reported by Wadia et al[26] . The lesion could have either a disc or a ring like enhancement of size less than 20mm. The surrounding perifocal oedema may be mild to moderate and usually there is no mass effect. In 1985 Setiet al[27], however reported the spontaneous resolution of these lesions.

By the help of stereotactic brain biopsy Rajshekar et al[28] have made an attempt to answer the controversy regarding the etiology of these lesions. He showed after histopthological diagnosis that, majority of these lesions are cysticercus granuloma and few of them are tuberculoma. They form the clinical and radiological criteria of SSECTL to diagnose the lesion to be a solitary

cysticercus granuloma.These lesions itself acts as antigen and production of inflammatory cytokines, causing cytotoxic and vasogenic oedema, which acts as an epileptogenic foci in dying phase of cyst.

Diagnostic criteria for solitary cerebral cysticercus granuloma (SCCG)[29]

Clinical criteria

1. Seizure as initial symptom (partial or generalized).

2. No features of persistent raised ICT.

3. No evidence of progressive neurologic deficit

4. No evidence of active systemic illness like tuberculosis and/or focus of pyogenic infections, primary malignancy.

CT criteria:

1. solitary lesion

2. Should enhance after contrast injection

3. <20mm in diameter

4. Absence of severe cerebral edema.

Management of SSECTL: AED therapy is the mainstay of treatment. An addition of short course of oral prednisolone (1mg/kg/day for ten days followed by tapering over 2 weeks) helps in prevention of seizure recurrence and also in early disappearance of lesion.[25] After a period of 10- 12 weeks follow up CT scan should be done in every patient to document the resolution of granuloma. If the patient has not had a seizure in the preceding 3 months, soon after a documented resolution of granuloma early discontinuation of AEDs is recommended.[29]

If the lesion enlarges in size on follow up CT scan Anti tuberculous therapy (ATT) may be considered.

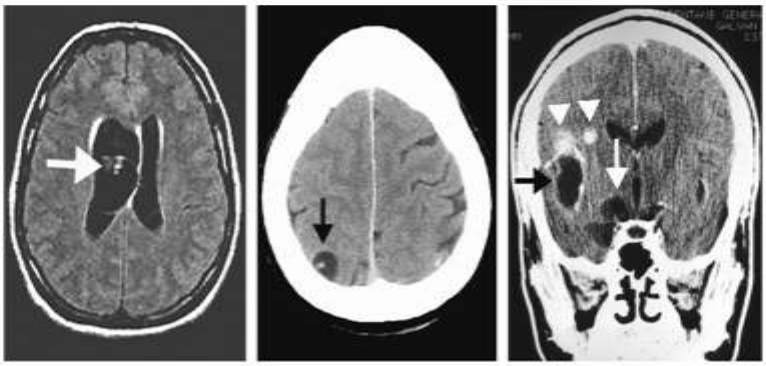
If clinically malignancy is suspected Surgical excision of the enlarging lesion for histopathological examination is recommended, as SSECTL in few patients could be due to meningioma and other primary bone tumours.[29]

NEUROCYSTICERCOSIS

It is a common parasitic disease of the CNS. Neurocysticercosis is caused by *'cysticercus cellulose'* the encysted larval stage of the tapeworm TaeniaSolium. For many years the parenchymal cysts may remain dormant and the death of larva and subsequent intense inflammatory reaction induced by larval antigens produces symptoms (e.g. seizures). Subsequently, the cyst transforms into active granuloma.

The cyst then shrinks and granuloma eventually calcify or frequently disappears completely.[30]

Figure 220-1



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: Harrison's Principles of Internal Medicine, 18th Edition: www.accessmedicine.com

Copyright © The McGraw-Hill Companies, Inc. All rights reserved.

Neurocysticercosis is caused by *Taenia solium*. Neurologic infection can be classified on the basis of the location and viability of the parasites. When the parasites are in the ventricles, they often cause obstructive hydrocephalus. ***Left:*** MRI showing a cysticercus in the lateral ventricle, with resultant hydrocephalus. The arrow points to the scolex within the cystic parasite. ***Center:*** CT showing a parenchymal cysticercus, with enhancement of the cyst wall and an internal scolex (*arrow*). ***Right:*** Multiple cysticerci, including calcified lesions from prior infection (*arrowheads*), viable cysticerci in the basilar cisterns (*white arrow*), and a large degenerating cysticercus in the Sylvian fissure (*black arrow*). *(Modified with permission from JC Bandres et al: Clin Infect Dis 15:799, 1992. © The University of Chicago Press.)*

In brain parenchyma, the cysticercus cyst typically goes through four stages of involution

1) Vesicular
2) Colloidal
3) Granular – nodular and
4) Calcific.

The first 2 stages are considered to represent the live parasite, and last two stages, the dying or dead forms of the parasite. A live cyst is asymptomatic, evolving no or minimal host immune response.

In India, majority of patients of neurocysticercosis have single enhancing lesions but the multiple enhancing CT/MRI lesions are also not uncommon[31]. These single or multiple lesions pose a challenge to clinicians and radiologists. The clinical features and imaging of neurocysticercosis and tuberculoma are exceedingly similar and it is difficult to differentiate these two conditions.[32]

The distinction between tuberculoma and single cysticercus granuloma is important because single cysticercus granuloma is a benign and self limiting condition, but tuberculoma is an active infection which requires prolonged therapy with potentially toxic drugs.[33]

Table 220-1 Diagnostic Criteria for Human Cysticercosis[a]
1. Absolute criteria
a. Demonstration of cysticerci by histologic or microscopic examination of biopsy material
b. Visualization of the parasite in the eye by funduscopy
c. Neuroradiologic demonstration of cystic lesions containing a characteristic scolex
2. Major criteria
a. Neuroradiologic lesions suggestive of neurocysticercosis
b. Demonstration of antibodies to cysticerci in serum by enzyme-linked immunoelectrotransfer blot
c. Resolution of intracranial cystic lesions spontaneously or after therapy with albendazole or praziquantel alone
3. Minor criteria
a. Lesions compatible with neurocysticercosis detected by neuroimaging studies
b. Clinical manifestations suggestive of neurocysticercosis
c. Demonstration of antibodies to cysticerci or cysticercal antigen in cerebrospinal fluid by ELISA
d. Evidence of cysticercosis outside the central nervous system (e.g., cigar-shaped soft-tissue calcifications)
4. Epidemiologic criteria
a. Residence in a cysticercosis-endemic area
b. Frequent travel to a cysticercosis-endemic area
c. Household contact with an individual infected with *Taenia solium*

[a]Diagnosis is confirmed by either one absolute criteria or a combination of 2 major criteria, one minor criteria, and one epidemiologic criteria. A probable diagnosis is supported by the fulfillment of (1) one major criteria plus two minor criteria; (2)

one major criteria plus one minor criteria and one epidemiologic criteria; or (3) three minor criteria plus one epidemiologic criteria.

Unequivocal evidence of neurocysticercosis is histopathological demonstration of the parasite.

Following lesions are highly suggestive of neurocysticercosis in neuroimaging, solitary cysticercus granuloma ,spontaneous resolution or eventual calcification after several months.[34]

Electroimmuno transfer blot (EITB)assay is the current serological assay of choice for the diagnosis of neurocysticercosis. This assay has a specificity of 100% and a sensitivity of 94% to 98% for patients with 2 or more cystic or enhancing lesions. But frequent false negative results in patients with a solitary intracranial cysticercus lesion, in whom less than 50% test positive. The sensitivity and specificity of EITB is also low in patients with calcified lesions.[35]

CSF ELISA for neurocysticercosis is 95% specific and 87% sensitive . It is a useful supportive tool for the diagnosis . But in contrast Serum ELISA has a large number of false negative and false positive results.

TUBERCULOMA OF BRAIN

It account for 20 to 30% of intracranial space occupying lesions in India[36].Tuberculoma develop in brain when the initial "Rich focus" does not rupture into the meninges but expands locally with in the brain parenchyma. It may also originate in the meninges, and may be found in the superficial cortex. This meningeal form may resemble a meningioma.[37]

Patients with tuberculoma most often present with seizures (60 to 100%), signs and symptoms of raised ICT (56- 93%) and focal neurological deficits (33-68%).Tuberculomas may also be multiple or military.[38]

There may be multiple caseating granulomas in the brain, although most of the patients (66- 73%) have single or multiple large granulomas with necrotic centre.[39]

Imaging features:

During the initial phase, edema and necrosis may appear as low attenuating areas on CT scan., There may be high attenuation, contrast enhancement and calcification associated with ring enhancement and a surrounding edema when the granuloma has began to organize. This may be homogenous enhancement or there

may be a central radiolucent area which corresponds to a central zone of necrosis.[40]

MRI is more sensitive than CT in detecting cerebral parenchymal tubercuoma. Tuberculoma are isointense with gray matter on T1 weighted images. Lesions show central hyper intensity on T2 weighted images.A hypointense ring is present within the wall of tuberculoma on T2 weighted MR images in some cases. Tuberculomas typically "enhance" after administration of IV gadopentetate dimeglumine in a solid or ring pattern.[41]

SEIZURES IN PATIENTS WITH HIV:

CNS involvement in AIDS patients may first be manifested by seizures[42]. seizure threshold is often lower than normal in these patient due to the frequent presence of electrolyte abnormalities Seizures are seen in 15- 35% of patients with primary CNS lymphoma 15- 40% of patients with cerebral toxoplasmosis, 7- 50% of patients with HIV encephalopathy and 8% of patients with cryptococcal meningitis[43]. The most common cause of enhancing CT/MRI lesions in patients with AIDS isToxoplasmosis. Seizures have been reported as an early symptom of CNS tuberculosis in 50% patients[44] .

Both generalized and focal seizures were noted in patients with focal brain lesions and in those where no cause for seizures could be identified. Presence of focal seizure did not necessarily imply the presence of focal brain lesion.[45]

Recurrences of seizures have been observed in 68% of patients despite of anticonvulsant therapy. It is difficult to predict which patient is likely to have recurrent seizures. While looking for a cause of seizures in patients with HIV infection, investigation should include CT/MRI scan, serological test for toxoplasmosis and CSF examination for cryptococcal antigen, adenosine deaminase and fungal culture.[45]

Differential Diagnosis of Seizures

In most cases seizures can be distinguished from other conditions by careful attention to the history and relevant laboratory studies. Additional studies such as video-EEG monitoring, cardiac electrophysiology, sleep studies, tilt-table analysis may be required to reach a correct diagnosis. Disorders that may mimic seizure are listed below,

Table 369-6 Differential Diagnosis of Seizures
Syncope
Vasovagal syncope
Cardiac arrhythmia
Valvular heart disease
Cardiac failure
Orthostatic hypotension
Psychological disorders
Psychogenic seizure
Hyperventilation
Panic attack
Metabolic disturbances
Alcoholic blackouts
Delirium tremens
Hypoglycemia
Hypoxia
Psychoactive drugs (e.g., hallucinogens)
Migraine
Confusional migraine
Basilar migraine
Transient ischemic attack (TIA)
Basilar artery TIA
Sleep disorders
Narcolepsy/cataplexy
Benign sleep myoclonus
Movement disorders
Tics
Nonepileptic myoclonus
Paroxysmal choreoathetosis
Special considerations in children
Breath-holding spells
Migraine with recurrent abdominalpain and cyclic vomiting
Benign paroxysmal vertigo
Apnea
Night terrors
Sleepwalking

Table 369-7 Features that Distinguish Generalized Tonic-Clonic Seizure from Syncope		
Features	**Seizure**	**Syncope**
Immediate precipitating factors	Usually none	Emotional stress, Valsalva, orthostatic hypotension, cardiac etiologies
Premonitory symptoms	None or aura (e.g., odd odor)	Tiredness, nausea, diaphoresis, tunneling of vision
Posture at onset	Variable	Usually erect
Transition to unconsciousness	Often immediate	Gradual over secondsa
Duration of unconsciousness	Minutes	Seconds
Duration of tonic or clonic movements	30–60 s	Never more than 15 s
Facial appearance during event	Cyanosis, frothing at mouth	Pallor
Disorientation and sleepiness after event	Many minutes to hours	<5 min
Aching of muscles after event	Often	Sometimes
Biting of tongue	Sometimes	Rarely
Incontinence	Sometimes	Sometimes
Headache	Sometimes	Rarely

Mechanisms of Action of Antiepileptic Drugs

AED's appear to act primarily by blocking the initiation or spread of seizures.Through a variety of mechanisms by which they modify the activity of ion channels or neurotransmitters and these drugs also have pleiotropic effects in most of the cases. The mechanisms include

1) inhibition of sodium dependent action potentials in a frequency-dependent manner (e.g., carbamazepine, phenytoin, lamotrigine, rufinamide, topiramate, zonisamide, lacosamide),

2) inhibition of voltage-gated calcium channels (phenytoin, pregabalin,gabapentin),
3) attenuation of glutamate activity (lamotrigine, felbamate, topiramate),
4) potentiation of GABA receptor function (benzodiazepines and barbiturates),
5) increase in the availability of GABA (valproic acid, tiagabine,gabapentin), and
6) modulation of release of synaptic vesicles (levetiracetam).

The most effective drugs for absence seizures, valproic acid and ethosuximide probably act by inhibiting T-type calcium channels in thalamic neurons.

Treatment of Seizure:

When a patient presents shortly after a seizure,we should give first priorities to vital signs, cardiovascular and respiratory support, and treatment of seizures. Life-threatening conditions such as metabolic derangement, CNS infection, or drug toxicity must be managed appropriately.

If patient is not acutely ill,the initial evaluation focus on whether there is a history of earlier seizures. If this is the first seizure, then the emphasis will be : (1) to establish whether the reported episode was a seizure or any another paroxysmal

event, (2) to determine the cause of the seizure by identifying precipitating events and risk factors, and (3) to decide whether anticonvulsant therapy is required.

Table 369-8 Selection of Antiepileptic Drugs

Generalized-onset Tonic-Clonic	Focal	Typical Absence	Atypical Absence, Myoclonic, Atonic
First-Line			
Valproic acid Lamotrigine Topiramate	Lamotrigine Carbamazepine Oxcarbazepine Phenytoin Levetiracetam	Valproic acid Ethosuximide	Valproic acid Lamotrigine Topiramate
Alternatives			
Zonisamide[a] Phenytoin Carbamazepine Oxcarbazepine Phenobarbital Primidone Felbamate	Topiramate Zonisamide[a] Valproic acid Tiagabine[a] Gabapentin[a] Lacosamide[a] Phenobarbital Primidone Felbamate	Lamotrigine Clonazepam	Clonazepam Felbamate

[a]As adjunctive therapy.

AED Selection for Focal Seizures

Carbamazepine, lamotrigine and phenytoin is currently the initial drug of choice for the treatment of focal seizures, including those that secondarily generalize. Overall, these drugs have very similar efficacy, but differences in toxicity and pharmacokinetics are the main determinants for use in a given patient.

Phenytoin offers the advantage of once or twice daily dosing because it has a relatively long half- life compared to two or three times daily dosing for carbamazepine and lamotrigine. An advantage of carbamazepine is that the relationship between drug dose, serum levels, is linear because of its metabolism follows first- order pharmacokinetics.By contrast, small increases in dose of phenytoin above a standard maintenance dose can precipitate marked side effects because its properties of saturation kinetics. This is the main causes of acute phenytoin toxicity. The long- term use of phenytoin is associated with cosmetic effects (e.g., hirsutism, gingival hypertrophy and coarsening of facial features), and it is often avoided in young patients who require the drug for many years because of its effects on bone metabolism. Carbamazepine can cause aplastic anemia, leucopenia, or hepatotoxicity.Therefore it would be contraindicated in patients with predispositions to these problems. Lamotrigine may cause skin rash during the initiation of therapy. Sometimes it can be extremely severe and may lead to Stevens - Johnson syndrome if the symptoms unrecognized and if the medication is not discontinued immediately. Slow introduction and titration may decreases the risk.

Valproic acid is an effective alternative for some patients with focal seizures, especially when the seizures secondarily generalize. GI side effects are

fewer when using the valproate semisodium formulation. Valproic acid also rarely causes hepatotoxicity and reversible bone marrow suppression and laboratory testing is required to monitor toxicity.In patients with preexisting bone marrow or liver disease this drug should be avoided.

Topiramate, levetiracetam, tiagabine, zonisamide, gabapentin, and oxcarbazepine are additional drugs currently used for the treatment of focal seizures with or without secondary generalization.Previously, phenobarbital and other barbiturate compounds were commonly used as first - line therapy for most of epilepsy[58] .

AED Selection for Generalized Seizures

Currently Valproic acid is the best initial choice for the treatment of primarily generalized, tonic- clonic seizures. Followed by Lamotrigine, phenytoin and carbamazepine are suitable alternatives. Valproic acid is also effective in absence, atonic and myoclonic seizures.Therefore it is the drug of choice in patients with generalized epilepsy syndromes having mixed seizure types.Both carbamazepine and phenytoin can worsen certain types of generalized seizures, including myoclonic, absence, tonic, and atonic seizures.In uncomplicated absence seizures ethosuximide is a particularly effective drug for the treatment, but it is not

useful for tonic- clonic or focal seizures. Periodic monitoring of blood cell counts is required in ethosuximide therapy because it rarely causes bone marrow suppression.Zonisamide, topiramate, and felbamate may have similar broad efficacy.

Initiation and Monitoring of Therapy

Patients should be carefully educated about the approach to therapy because the response to any antiepileptic drug is unpredictable. The goal is to prevent seizures and minimize the side effects of therapy; determination of the optimal dose may take months or longer if the baseline seizure frequency is low. Most anticonvulsant drugs[(59)] need to be introduced relatively slowly to minimize side effects. Patients should expect that minor side effects such as slight changes in cognition,mild sedation or imbalance would typically resolve within a few days. Subsequent increase in dose should be made only after achieving a steady state with the previous dose.

Monitoring of serum AED levels can be very useful for establishing the initial dosing schedule.The key determinants are the clinical measures of seizure frequency and presence of side effects and not the laboratory values. Serum drug levels measured by Conventional assays measure the total drug (that is both free

and protein- bound forms).The concentration of free drug that reflects extracellular levels in the brain and correlates with efficacy. Patients with decreased levels of serum proteins (i.e., decreased serum albumin due to impaired liver or renal function) may have an increased ratio of free to protein bound drug, yet the concentration of free drug may be adequate for seizure control. These patients may have a "subtherapeutic" drug level, but the dose should be changed only when seizures remain uncontrolled, not just to achieve a "therapeutic" level.

If seizures continue despite gradual increases to the maximum tolerated dose and documented compliance, then it becomes necessary to switch to another AED. This is usually done by maintaining the patient on[(60)] the first drug while a second drug is added. The second drug should be adjusted to reduce the seizure frequency and also without causing toxicity. If this is achieved, the first drug can be gradually withdrawn. The dose of the second drug is then further optimized based on seizure response and side effects. Anyhow monotherapy should be the goal.

When to Discontinue Therapy

Overall, about 70% of children and 60% of adults who have their seizures completely controlled with AED's can eventually discontinue therapy. The

following profile of patient yields the greatest chance of remaining seizure- free after the drug withdrawal:

(1) complete medical control of seizures for one to five years;

(2) single seizure type, either generalized or partial;

(3) normal neurologic examination;

(4) normal EEG.

The appropriate seizure- free interval is unknown and it varies for different forms of epilepsy. However, it seems reasonable to attempt withdrawal of therapy after 2 years in a patient who meets all of the above criteria, is motivated to discontinue the medication. In most cases, it is preferable to reduce the dose of the drug gradually over two to three months. Most recurrences occur in the first three months after discontinuing therapy. So patients should be advised to avoid potentially dangerous situations such as swimming or driving during this period.[61]

Treatment of Refractory Epilepsy

Approximately one - third of patients with epilepsy do not respond to treatment with a single antiepileptic drug, so it becomes necessary to try a combination of drugs to control seizures. Patients with multiple seizure types and

developmental delay or those who have focal epilepsy related to an underlying structural lesion are particularly likely to require multiple drugs.The initial combination therapy combines first- line drugs, i.e., carbamazepine, valproic acid, phenytoin and lamotrigine. If these drugs are unsuccessful, then add a newer drug such as topiramate or levetiracetam. Patients with myoclonic seizures resistant to valproic acid then add clonazepam, and those with absence seizures may respond to a combination of ethosuximide and valproic acid.

Surgical Treatment of Refractory Epilepsy

Approximately 20% of patients with epilepsy are resistant to medical therapy. For some, surgery can be extremely effective in substantially reducing seizure frequency and even providing complete seizure control. Surgical options are Lesionectomy, Temporal Lobectomy Hemispherectomy Corpus CallosotomySelective Amygdalohippocampectomy Minimal Access, Stereotactic ablations, Cerebellar stimulation, and Vagus nerve stimulation.

Status Epilepticus

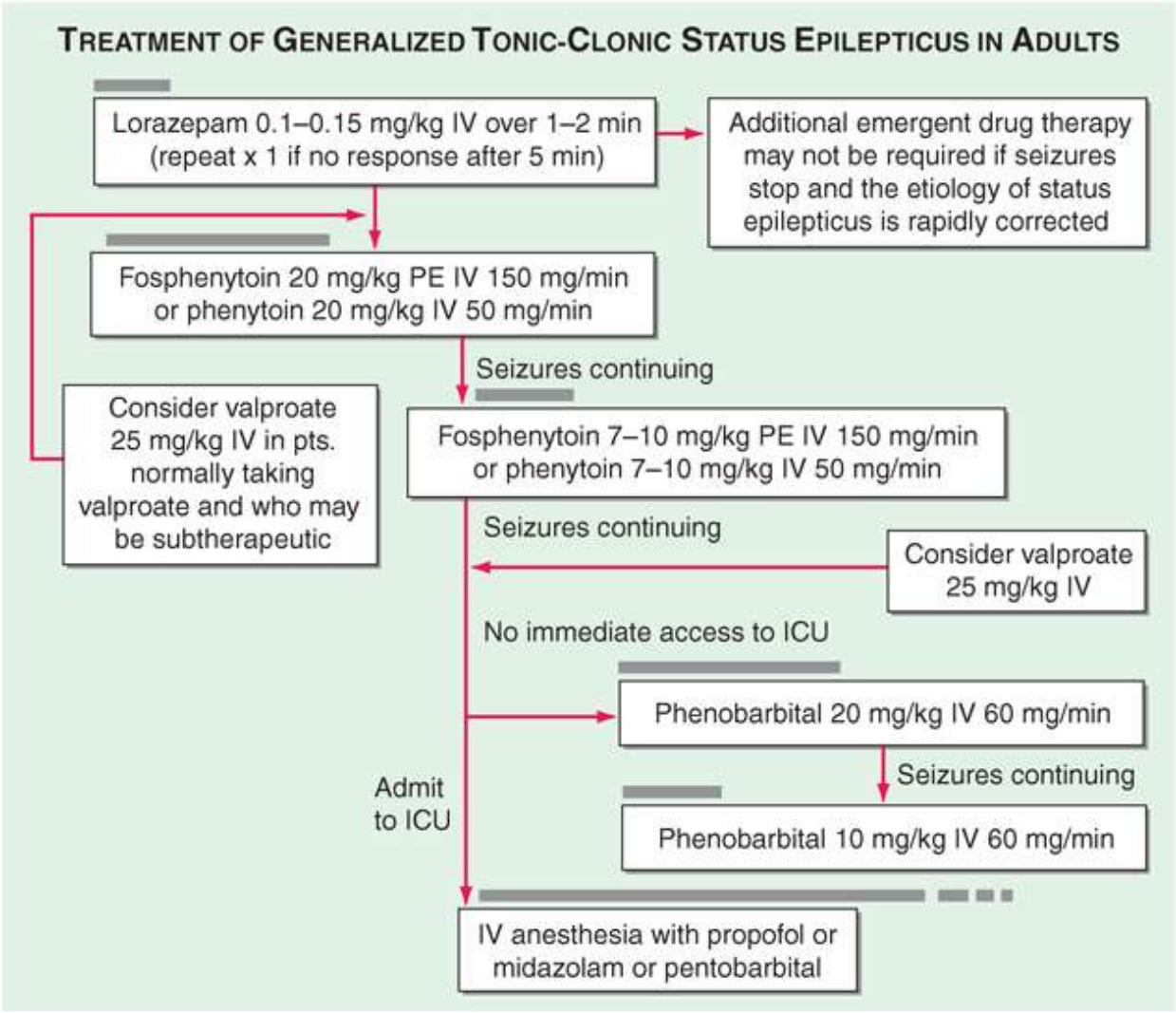
It refers to continuous seizures or repetitive, discrete seizures with impaired level of consciousness in the interictal period. It has numerous subtypes, including

generalized convulsive status epilepticus (GCSE) and nonconvulsive status epilepticus. To meet the definition of status epilepticus the duration of seizure activity has to be15–30 minutes. But, the duration of seizures prompts the acute use of anticonvulsant therapy in status epilepticus. For GCSE, this is typically when seizures last beyond 5 minutes.

Generalized convulsive status epilepticus *is an emergency and must be treated immediately*, since hyperthermia,cardiorespiratory dysfunction and metabolic derangements can occur as a consequence of prolonged seizures.These can lead to irreversible neuronal injury. Furthermore, CNS injury can also occur when the patient is paralyzed with neuromuscular blockade but continues to have electrographic seizures. CNS tumors, anticonvulsant withdrawal or noncompliance, metabolic disturbances, drug toxicity, refractory epilepsy, CNS infection, and head trauma are most common causes of GCSE.

GCSE is obvious when the patient is having overt convulsions. After 30–45 minutes of uninterrupted seizures, the signs become increasingly subtle. Sometimes patients may have , fine rapid movements of the eyes and mild clonic movements of only the fingers. There may be paroxysmal episodes of hypertension, tachycardia and pupillary dilation. In these cases, the EEG is the only method of establishing the diagnosis.If the patient stops having overt seizures,

yet remains comatose, an EEG must be performed to rule out ongoing status epilepticus.



AIM OF THE STUDY:

To analyse the clinical profile and to evaluate the patients with new onset seizures who presented to KMCH

MATERIALS AND METHODS

This is a Descriptive study conducted in Government Kilpauk medical college hospital, Department of medicine in collaboration with Neurology department, for a period of 9months. A total of 100 cases admitted in medical ward with new onset seizures were selected for the present study from March 2013 to November 2013.

Materials:

Detailed history, physical examination, ,RFT,Sr.Electrolytes ,EEG, CT Brain,MRI Brain, CSF, serology and other routine investigations.

METHODOLOGY: In patients with New onset seizures along with history and clinical examination, special investigations like CT BRAIN, MRI BRAIN,EEG, SEROLOGY, CSF ANALYSIS will be done to find out the etiology.

INCLUSION CRITERIA:

Patients admitted in medical wards of Govt. Kilpauk medical college hospital,

- Age more than 18 years.
- New onset seizures (Provoked and unprovoked)
- Status Epilepticus

EXCLUSION CRITERIA:

- Psychogenic seizures.
- Eclampsia

Data collection

The data of each patient was collected on a proforma specially designed for this study and included demographic details, detailed history, clinical features, past medical history. physical examination, RFT, Sr.Electrolytes, EEG, CT Brain ,MRI Brain and other routine investigations.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **statistics software.**

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATION AND ANALYSIS :

The results of the study are shown in tables as below. The baseline characteristics observed are as follows,

Number of cases of new onset seizures studied -100

Table 1: Age and sex distribution

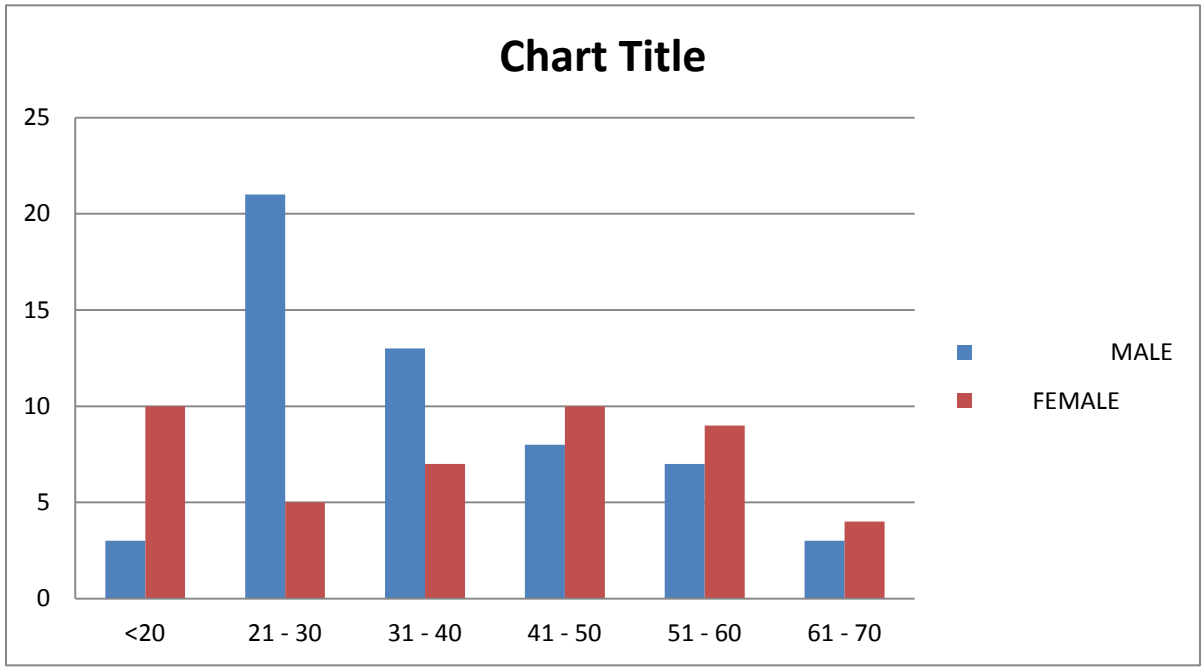
	AGE IN YEARS	MALE	FEMALE	TOTAL
1	<20	3	10	13
2	21 - 30	21	5	26
3	31 - 40	13	7	20
4	41 - 50	8	10	18
5	51 - 60	7	9	16
6	61 - 70	3	4	7
	TOTAL	55	45	100

In the present study patients age ranged from 18yrs to 80 years with the mean of

Majority of patients were in the age group of 21-30yrs(n=26) followed by 31to 40 yrs(n=20).

77% of patients were in 2nd – 5th decade. 6%of patients were in the age group of >60yrs.

FIGURE-1



Majority of males were in 3rd decade and females were in 5th decade.

Out of 100 patients 55 were males and 45 were females with male to female ratio of 1.22 : 1.

Table 2:etiologies and sex distribution

ETIOLOGY	MALE (n=55)	FEMALE (n=45)	P value
ALCOHOL WITHDRAWAL(n=34)	31	3	0.001
IDIOPATHIC (n=29)	13	16	
NEURO INFECTION (n=16)	6	10	
CVA (n=12)	4	8	
METABOLIC (n=9)	1	8	

Among etiologies and sex distribution the P vaue is <0.001. This shows there is significant correlation between etiologies and sex distribution in this study.

FIGURE-2

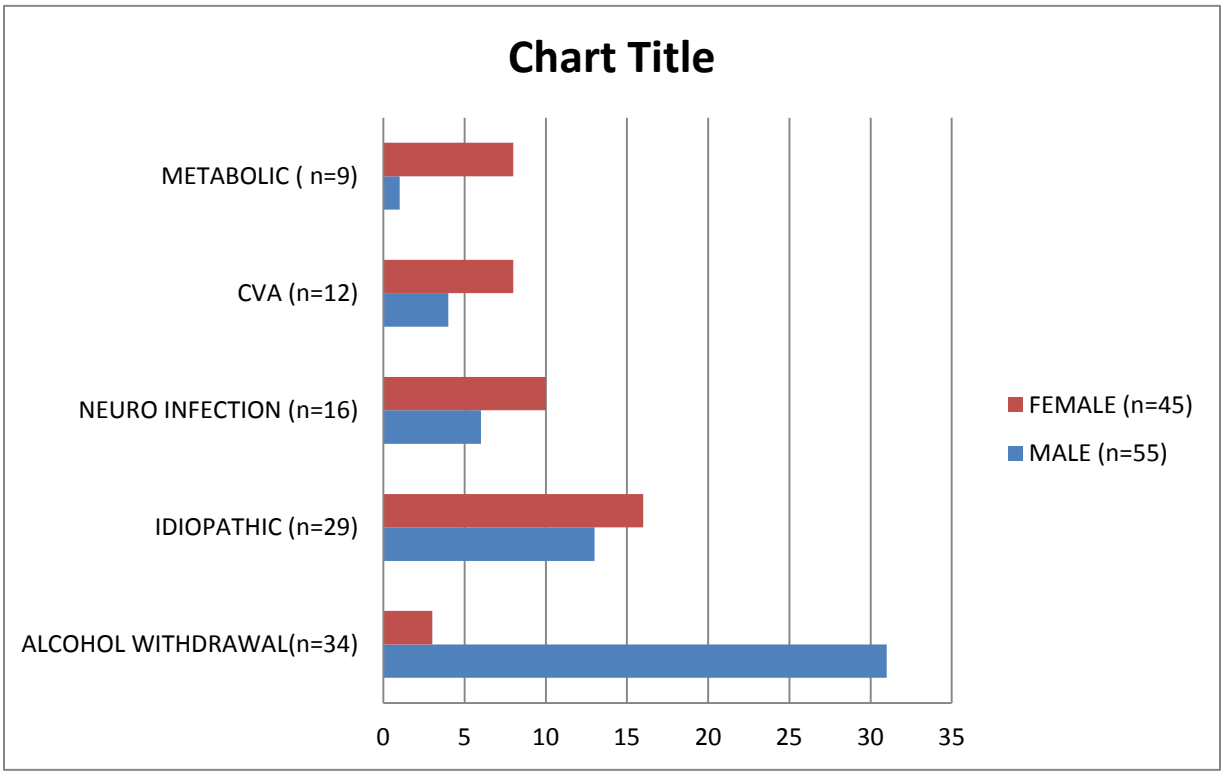


Table 3: distribution of etiologies in patients with seizures

	ETIOLOGIES	NUMBER (n=100)	P value
1	ALCOHOL WITHDRAWL	34	
2	IDIOPATHIC	29	
3	NEURO INFECTION	16	
4	CVA	12	<0.001
5	METABOLIC	9	

Among distribution of various etiologies in seizures shows the P value of <0.001. This shows there is significant relation among distribution of various etiologies in seizures.

Alcohol withdrawal is leading cause of seizure,which accounted for 34% followed by Idiopathic 29%, Neuro infection 16%,CVA 12% and metabolic 9%

FIGURE-3

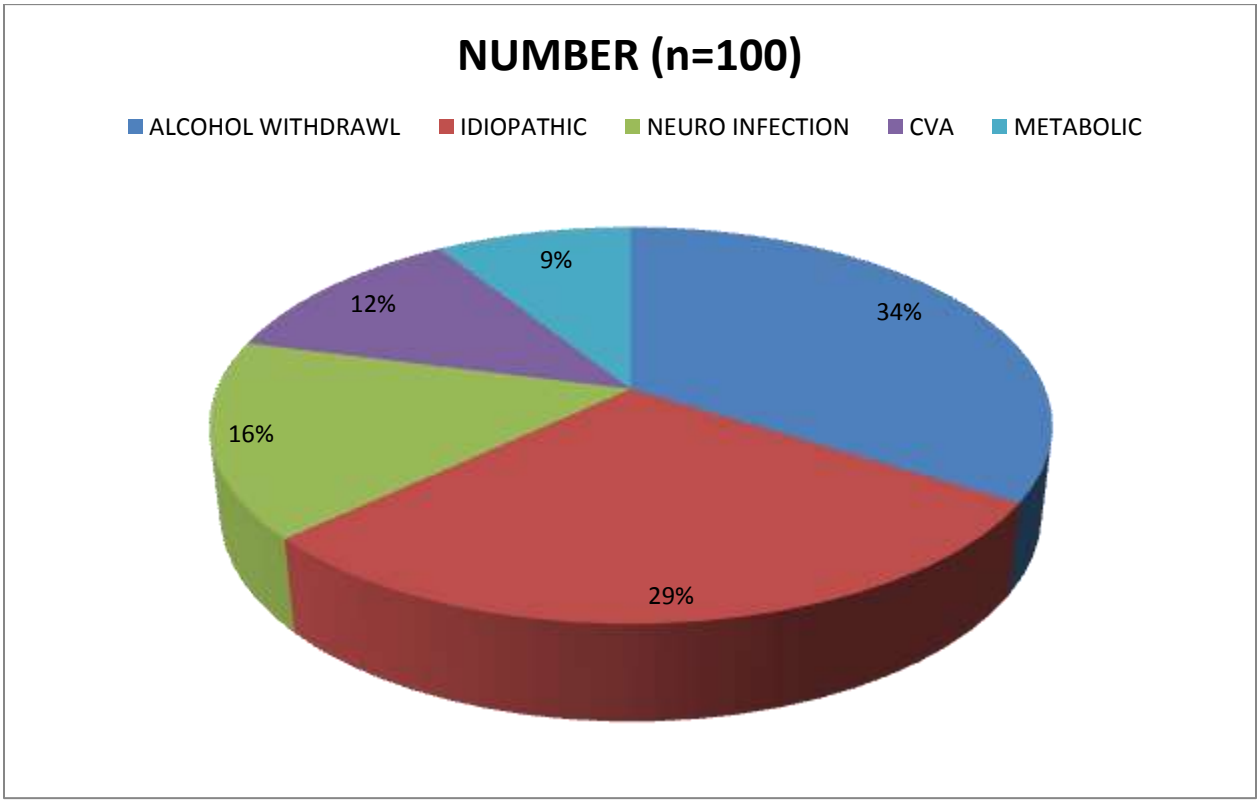


Table -4:correlation of etiologies with age group

	ETIOLOGIES	AGE IN YEARS						
		18-20	21- 30	31-40	41-50	51-60	61- 70	P value
1	ALCOHOL WITHDRAWAL	0	11	8	8	5	2	<0.001
2	IDIOPATHIC	9	9	7	2	1	1	
3	NEURO INFECTION	4	6	3	0	1	2	
4	CVA	0	0	2	5	5	0	
5	METABOLIC	0	0	2	2	2	2	

Alcohol withdraeal seizures is more common in 21-30 years.whereas CVA and metabolic seizures are common in older people**.**

In 18-20 yrs 69%is due to idiopathic,31%is due to neuro infection.

In 21-30 yrs most common etiology is 42%alcohol withdrawal followed by 35% idiopathic and 23% neuroinfection.

In 31-40 yrs most common etilogy is alcohol withdrawal 36% followed by idiopathic 31%, neuroinfection 14%, CVA9%, metabolic 9%

In 41-50 yrs most common etiology is alcohol withdrawal 47% followed by CVA 29%,idiopathic 12% and metabolic 12%

In50-60yrs most common etiology is alcohol withdrawal36% and CVA 36% followed by metabolic 19%, idiopathic%, neuroinfection 7%

In age >60 yrs most commo etiology is neuroinfection and metabolic seizures 29% followed by 28%alcohol withdrawal and14% idiopathic.

The P value of etiologies and age distribution is <0.001.This shows there is significant correlation between etiologies and age distribution of this study.

FIGURE-4

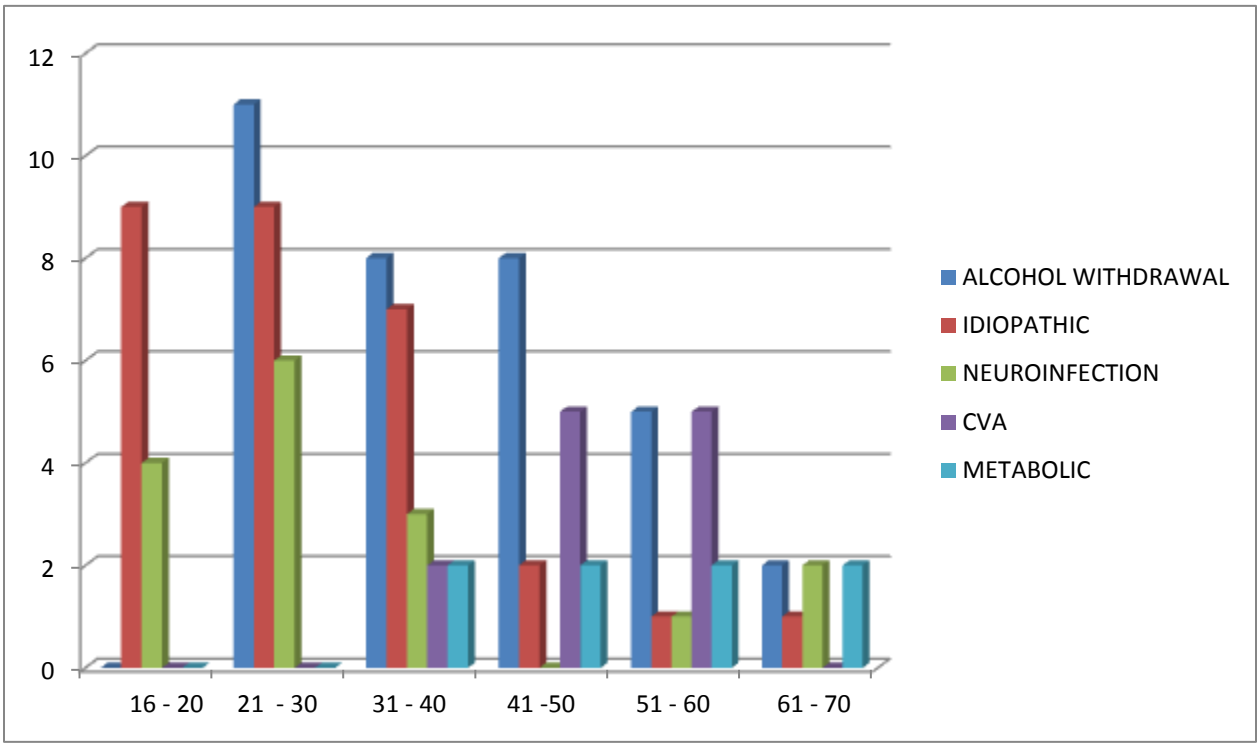


Table -5: various types of neuro infection

	NEURO INFECTION	NUMBER(n=16)	%AMONG NEURO INFECTION
1	TUBERCULOMA	6	37.5
2	NEUROCYSTICERCOSIS	4	25
3	MENINGITIS	2	12.5
4	MENINGOENCEPHALITIS	3	18.75
5	CEREBRAL MALARIA	1	6.25

Among neuro infection Tuberculoma accounts for 37.5%(6),neurocysticercosis4(25%),meningitis 2(12.5%),meningoencephalitis 3(18.75%),cerebral malaria 1(6.25).

FIGURE-5

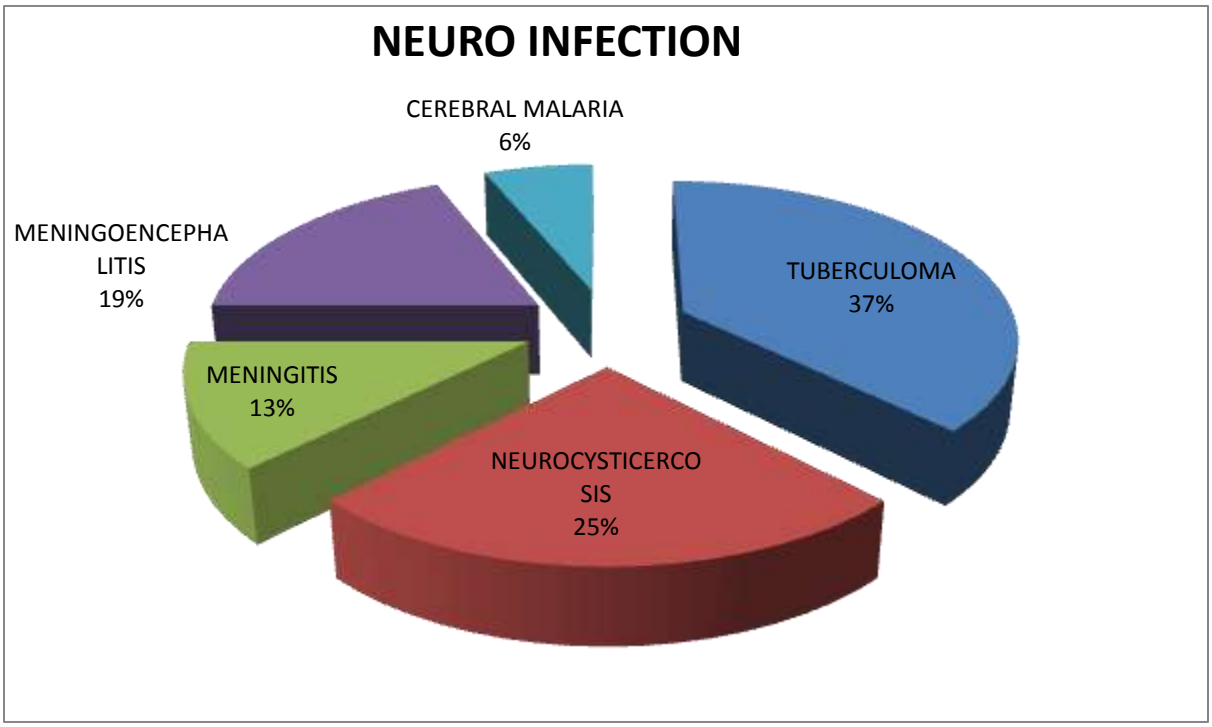


Table 6:Various types of CVA

	CVA	NUMBER(n=12)	% AMONG CVA
1	INFARCT	5	41.67
2	HAEMORRHAGE	3	25
3	CVT	1	8.33
4	SAH	2	16.67
5	SDH	1	8.33

Among CVA infarct 5(42%),haemorrhage3(25%),CVT1(8%),SAH2(17%) and SDH1(8%)

FIGURE-6

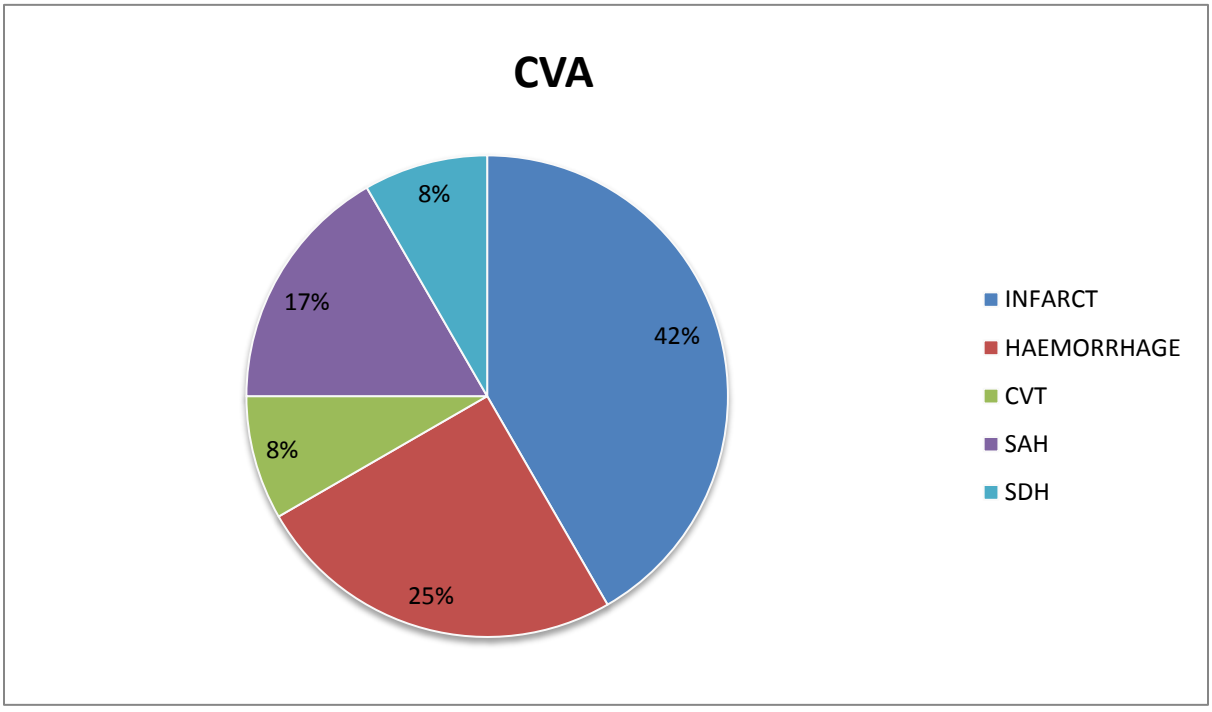


Table 7: various metabolic causes

	METABOLIC CAUSE	NUMBER (n=9)	%AMONG METABOLIC CAUSE
1	HYPOGLYCAEMIA	5	55.56
2	HYPERGLYCAEMIA	1	11.11
3	HYPOCALCAEMIA	1	11.11
4	HYPONATRAEMIA	2	22.22

Among metabolic causes 56% is due to hypoglycaemia followed by hyponatremia 22%,hyperglycemia and hypocalcemia 11%

.FIGURE-7

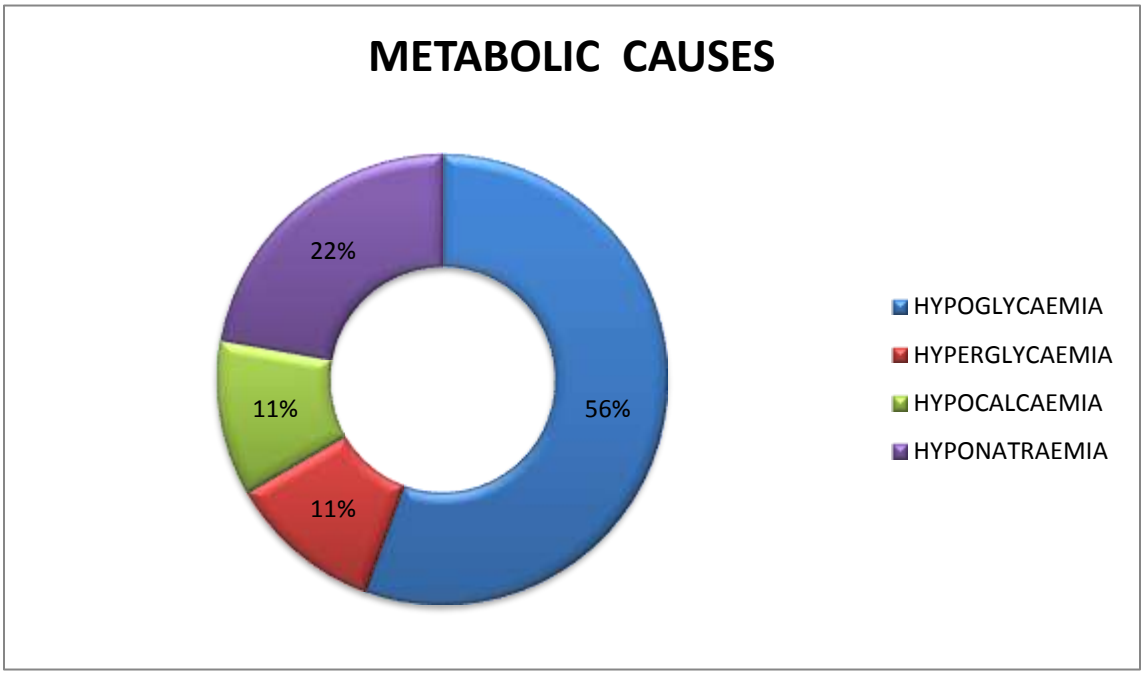


Table 8:Association for etiology and type of seizures

	ETIOLOGY	TYPE OF SEIZURE		P value
		GTCS	FOCAL	
1	ALCOHOL WITHDRAWAL	28	6	<0.001
2	IDIOPATHIC	21	8	
3	NEURO INFECTION	8	8	
4	CVA	9	3	
5	METABOLIC	4	5	
	TOTAL	70	30	

The P value of <0.001 shows there is significant relation between etiology and type of seizures in this study.

Seizures are commmonly present as GTCS.Most of the Alcohol withdrawal seizures 28(82%) are present as GTCS.

FIGURE-8.

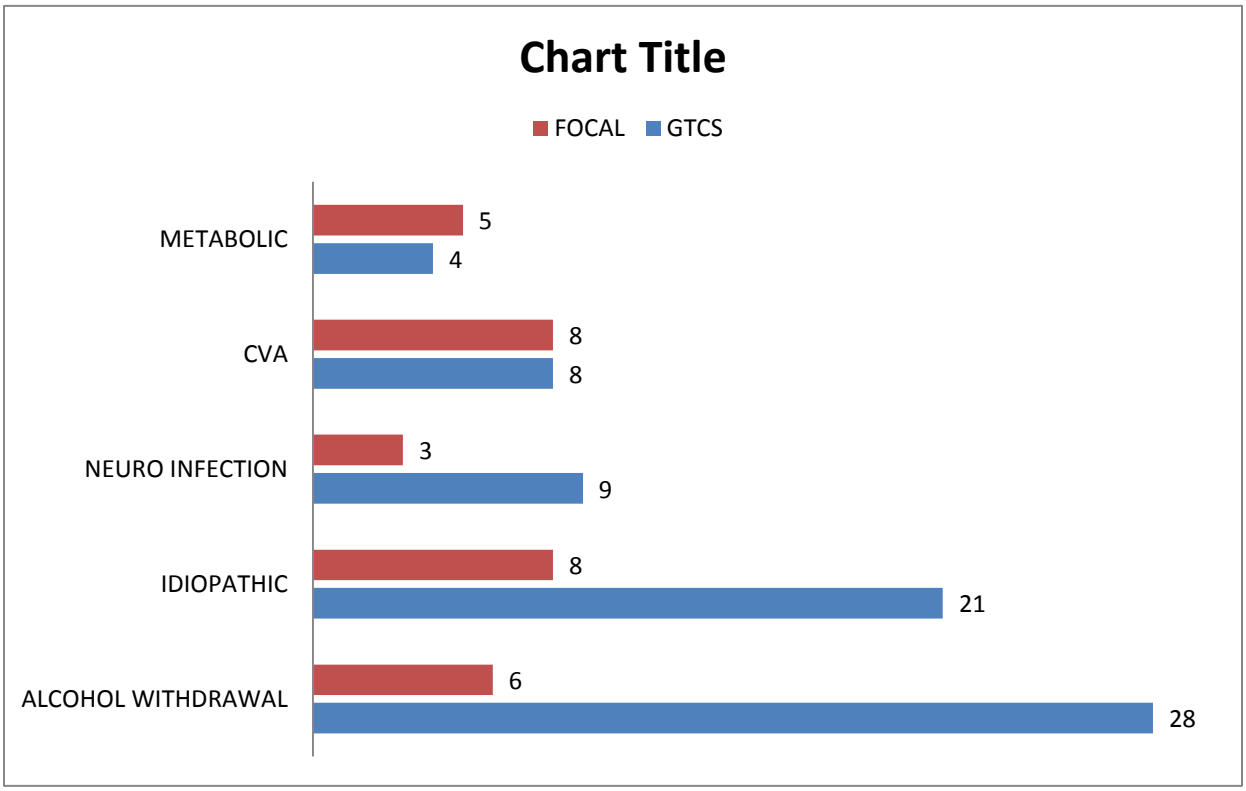


Table 9: GTCS distribution

ETIOLOGY	NUMBER OF CASES (n=70)
ALCOHOL WITHDRAWAL	28
IDIOPATHIC	21
CVA	9
NEURO INFECTION	8
METABOLIC	4

GTCS common in alcohol withdrawal seizures 40% folloed by idiopathic seizures 30%.

FIGURE-9

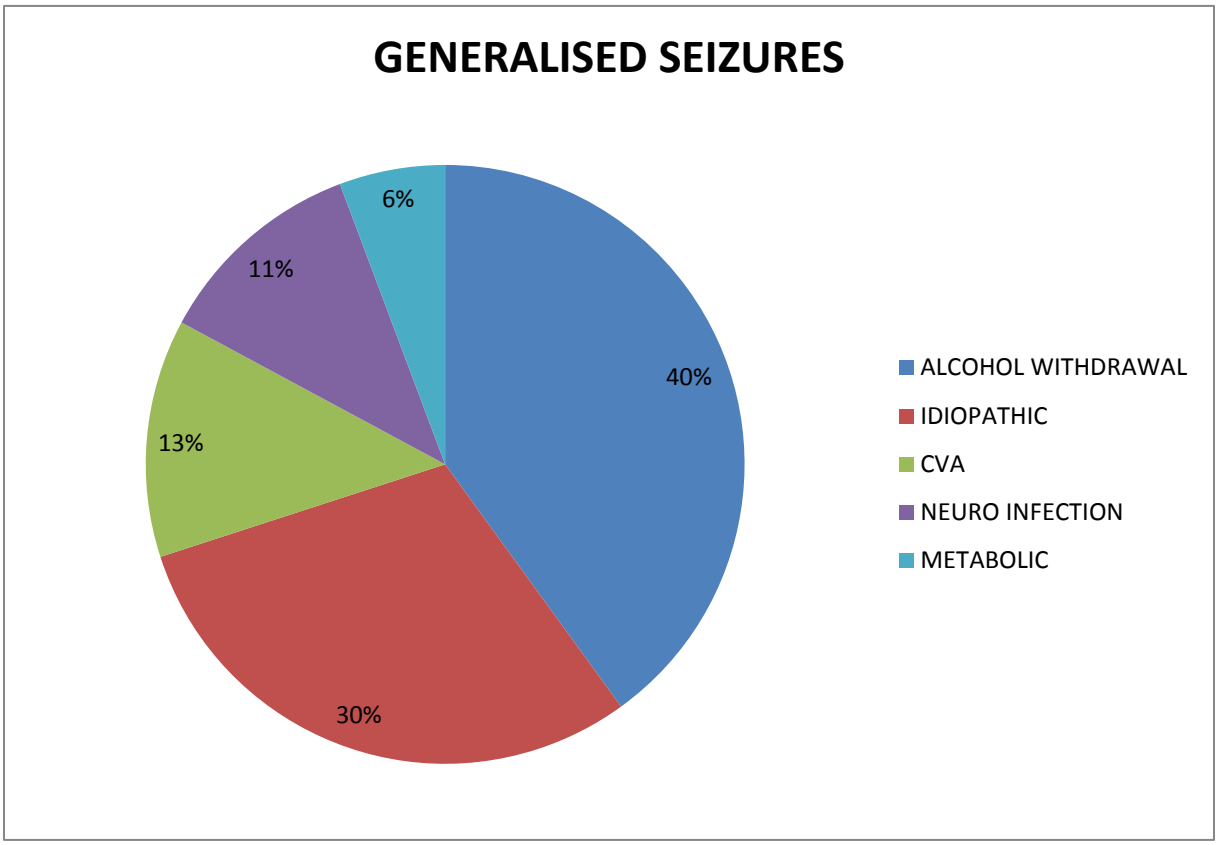
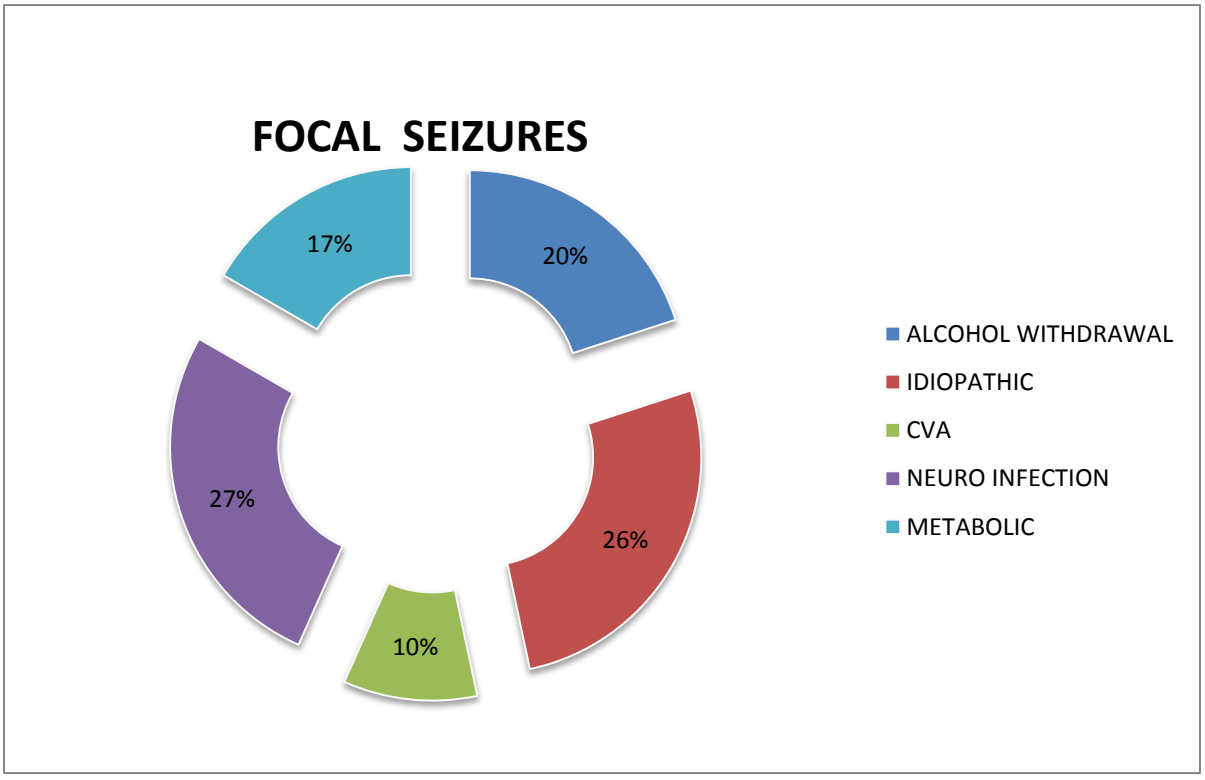


Table 10: Focal seizures distribution

ETIOLOGY	NUMBER OF CASES(n=30)
ALCOHOL WITHDRAWAL	6
IDIOPATHIC	8
CVA	3
NEURO INFECTION	8
METABOLIC	5

Focal seizures are common in neuroinfection and iddiopathic seizures.

FIGURE-10



DISCUSSION

In this study, a total of 100 patients with new onset epilepsy were included.

Sex ratio:

There was a slight male preponderance (M: F=1.22:1) in this study as quoted by other studies on epilepsy in United States and Europe (Granieri et al,1983).

Age distribution:

Maximum number of patients were in age group of 18- 40 years, the youngest being 18 years.Alcohol withdrawal seizures and adult onset idiopathic seizures are more common in this age group. Epilepsy due to cerebral infections like neurocysticercosis, tuberculoma and brain abscess are common in middle age.

Type of seizures: generalized tonic clonic seizures 70% are more common than focal seizures 30% in this study.

Etiology of epilepsy

In our study Alcohol withdrawal seizures (34%) was the commonest cause, followed by idiopathic epilepsy (29%), Neuro infection (16%),CVA(12%) and Metabolic seizures(9%).It was very difficult to differentiate between tuberculoma and NCC based on CT findings. We did chest X ray PA view and TB ELISA for patients suspected of tuberculoma. There was a history of chronic cough in one patient. Another patient who was diagnosed to have pulmonary tuberculosis 1 year ago had taken anti tubercular drugs for about 3 months and had presented with generalized tonic clonic convulsions. The CT scan showed 3 large ring enhancing lesions which were more than 20 mm in size.

It should be emphasized that despite careful investigations, a sizable proportion of patients (34%) were diagnosed as alcohol withdrawal seizures.

Etiologies observed in various stidies

1)SANDER et al(1990) UK

VASCULAR	**15%**
TUMOUR	**6%**
INFECTION	**2%**
ALCOHOL RELATED	**9%**

2)HAUSER et al (1995)USA

CVA	**18%**
NEURO INFECTION	**15%**
TRAMATIC BRAIN INJURY	**13%**
TUMOUR	**13%**
ALCOHOL WITHDRAWAL	**11%**
METABOLIC	**10%**

3) MURTHY JMK and RAVI (1999)Hyderabad

NEUROINFECTION	**77%**
VASCULAR	**14%**
METABOLIC	**3%**
TUMOUR	**7%**

4)NARAYANAN JT and JMK (2007)

NEUROINFECTION	**32%**
VASCULAR	**21%**
METABOLIC	**32%**
ALCOHOL	**9%**
OTHERS	**15%**

In present study:

	ETIOLOGIES	NUMBER (n=100)
1	ALCOHOL WITHDRAWL	34
2	IDIOPATHIC	29
3	NEURO INFECTION	16
4	CVA	12
5	METABOLIC	9

	GTCS	FOCAL SEIZURES
1)SANDER et al	39%	61%
2)MURTHY JMK and RAVI Y	22%	78%
3)NARAYANAN JT	55%	45%
4)OUR STUDY	70%	30%

In our study:

Most of Alcohol withdrawal seizure patients presented with GTCS(82%)

72% Of idiopathic seizure patients presented with GTCS followed by focal 28%

50% Of neuro infection patients presented with GTCS and focal seizures 50%

75% Of CVA patients presented with GTCS followed by focal seizures25%

54%Of metabolic seizure patients presented with focal seizures followed by GTCS.

CONCLUSION

From the present study “CLINICAL PROFILE AND EVALUATION OF NEW ONSET SEIZURES”the following conclusions were made.

1.Underlying etiologies were made in acute symptomatic seizures which contributed to 79%.

2.Majority of seizures occurred in patients <50 yrs of age.

3. Etiological spectrum were varied and included alcohol withdrawal,neuro infection,CVA,metabolic.

4. Alcohol withdrawal accounted for significant number of seizures in all the age groups.

5.Tuberculoma is most common cause of seizures in neuroinfection.

6.Infarct is most common cause of seizures in CVA patients.

7. Hypoglycemia is an important cause of seizures in metabolic seizures.

SUMMARY

This Descriptive study was done in the KMC hospital to know the various etiologies.100 cases of new onset seizures who fulfilled the criteria as mentioned in materials and methods were included in the study.

Out of 100 patients 55% were males,45%were females with male to female ratio of 1.2:1.

Majority of males were in 2^{nd} decade and females were in 4^{th} decade.patients age ranged from 18 yrs to 80 yrs,with the mean of 40.11yearswith 77% of the patients were in the below 50 yrs.

Alcohol withdrawal was the leading xcause of seizureswhich account for 34% followed by idiopathic seizures (29%),neuro infection (16%),CVA 12% and metabolic (9%).

Among neuro infection tuberculoma acconted for followed by neurocysticrcosis 25%,meningitis13%,meningoencephalitis 19% and cerebral malaria 6%.

Among CVA infarct accounted for 42% followed by haemorrhage 25%,SAH 17%,SDH 8% and CVT 8%.

Among metabolic causes hypoglycemia accounted for 56% followed by hyponatraemia 22% ,hyperglycemia 11% and hypocalcemia 11%.

GTCS was the most common seizure 70%.Most common cause of GTCS was alcohol withdrawal 40%followed by idiopathic 30%,CVA 13%,neuro infection11%,metabolic 6%.

APPENDIX

BIBLIOGRAPHY

1. Orrin Devinsky MD. The falling sickness. Owsei Temkin MD: A guide to understanding and living with epilepsy, the history of epileptic therapy. DF Scott.
2. Bharucho NE. Epidemiology of epilepsy in India. Epilespia 2003; 44(1):9-11
3. Sridheran R, Murthy BN. Prevalence and pattern of epilepsy in India Epilepsia 1999; 40 (5): 631- 6
4. Radhakrishnan K, Pandian JD. Prevalence, knowledge, attitude and practice of epilepsy in Kerala, South India. Epilespia 2000; 41(8): 1027-35
5. Ray BK, Bhattacharya S. Epidemiology of epilepsy – Indian perspective. J Indian Med Assoc 2002; 100(5): 322- 26.
6. Sander JW. The epidemiology of epilepsy revisited. Curr Opin Neurol 2003; 16(2): 165-70
7. Oguni H. Diagnosis and treatment of epilepsy. Epilepsia 2004; 45(Suppl.8): 13-16
8. Dam A, Fuglsang – Freder, Ksen A, Svaree – Olsen V, et al. Late onset epilepsy: etiologies, types of seizures, and value of clinical investigation, EEG, and computerized tomography scan. Epilepsia 1985; 26: 227- 31
9. Luef G, Schauer RS, Baues G. Idiopathic generalized epilepsy of late onset: A new epileptic syndrome? Epilepsia 1996; 37(4): 4
10. Cutting S, Lauchheimer A, Barr W, et al. Adult onset idiopathic generalized epilepsy: Clinical and behavioral features. Epilepsia 2001; 42: 1395- 8.
11. Fittipaldi F, Curra A, Fusco L, et al. EEG discharges on awakening: a marker of idiopathic generalized epileps y. Neurology 2001; 56: 123- 6.

12. Zarrelli MM, Behgi E, Rocca WA, et al. Incidence of epileptic syndromes in Rochester Minesote 1980- 1984. Epilepsia 1999; 40: 1708- 14.
13. Cutting S, Lauchheimer A, Barr W, et al. Adult onset idiopathic generalized epilepsy: clinical and behavioral features. Epilepsia 2001; 42: 1395- 98.
14. Sitajayalakshmi S, Mani J, Borgohoin, Mohondas S. Post stroke epilepsy. Neurol India 2002; 50(Suppl.1) S78- S84.,
15. Dhonuka AK, Misra UK, Kalita J. Seizures after stroke: a prospective clinical study. Neurol India 2001; 49(1): 33- 36.
16. Shinton RA, Gill JS, Zezuulka AV, et al. The freque ncy of epilepsy preceding stroke; case control study in 230 patients. Lacnet 1987; 1: 11- 13.
17. Kilpatrick CJ, Davis SM, Tress BM, et al. Epileptic seizures in acute stroke. Arch Neurol 1990; 47: 157- 160.
18. Blodin CF, Alexandrow AV, Bellavance et al. Seizures after stroke: a prospective multicenter study. Arch Neurol 2000; 57(11): 1617- 22.
19. Arboix A, Garcia – Eroles L, Marsons JB, et al. Predictive factors of early seizures after acute cerebrovascular disease. Stroke 1997; 28: 1590- 1594.
20. Lancman ME, Golimstok A, Norscini J, et al. Risk factors for developing seizures after a stroke. Epilepsia 1993; 34(1): 141- 143.
21. Gupta SR, Naheedy MH, Elias D, et al. Post infarction seizures: a clinical study. Stroke 1988; 19: 1477- 81
22. Gupta RK, Jena A, Sharma A. MR imaging of intracranial tuberculomas.J Compt Assist Tomogra 1988; 12: 280.

23. Paolucci S, Silvestri G, Lubich S, et al. Post stroke late seizures another role in rehabilitation of in patients. Epilepsia 1997; 38(3): 266-270

24. Horner S, Xiu- Shi N, Duft M, et al. EEG, CT and neuro sonographi cfindings in patients with postischemic seizures. J Neurol Sci 1995; 132:57-60.

25. Kishore D, Misra S. Short course of oral prednisolone on disappearance of lesion and seizure recurrence in patients of so litary cysticercol granuloma with single small enhancing CT lesion: An open Label randomized prospective study. JAPI 2007; 55: 419- 424.

26. Wadia RS, Makhole CN, Kelkar AV, Grant KB. Focal epilepsy in India with special reference to lesions showing ring or dis c like enhancement on contrast computed tomography. J Neurol Neurosurg Psychiatry 1987; 50: 1298- 301.

27. Sethi PK, Kumar BR, Madon VS, Mohan V. Appearing and disappearing CT scan abnormalities and seizures. J Neurol Neurosurg Psychiatry1985; 48: 866-9.72

28. Rajshekar V. Etiology and management of single small CT lesions in patients with seizures: understanding a controversy. Acta Neurol Scand 1991; 84: 465-70.

29. Rajshekar V. Solitary cerebral cysticercus granuloma. Epilepsia 2003; 44(Suppl.1):25- 28.

30. Del Brutto OH, Sotelo J. Neurocysticercosis: An update. Rev Infect Dis 1988; 10:1075- 87.

31. Escobar A. In: Polocio E, Rodriguez- Carabazal J, Taveras JM (Ed). The pathology of neurocysticercosis. Cysticercosis of the central nervous system. Charles C.Thomas (Illinosis) 1983 .p.27- 54.

32. Singh G. Neurocysticercosis in south- central America and the Indian subcontinent: A comparative evaluation. Arq Neuro Psiquiatr 1997; 55: 349-55.

33. Shah GV. Central nervous system tuberculosis. Neuroimaging. Clin North Am 2000; 10: 355- 74.

34. Garg RK. Diagnostic criteria for neurocysticercosis: Some modifications are needed for Indian patients. Neuro l India 2004; 52: 171-77

35. Singh G, Kausal V, Ram S, et al. Cysticercosis immunoblot assay in patients with single small enhancing CT lesions and multilesional neurocysticercosis. JAPI. 1999; 47:476-9.

36. Ramamurthy B, Varadarajan MG. Diagnosis of tuberculomas of the brain: Clinical and radiological correlation. J Neurosurg 1961; 8:1.

37. Garg RK. Diagnosis of intracranial tuberculoma. IndJ Tub 1996; 43:35- 39

38. Gree GT, Bazan III C, Jinkings JR. Miliary tuberculosis involving the brain MR findings. ARJ 1992; 159: 1075.

39. JinkinsJR.Computed tomography of intracranial tuberculosis.Neuroradiology 1991; 33: 186.

40. Gupta RK, Jena A, Sharma A. MR imaging of intracranial tuberculomas.J Compt Assist Tomogra 1988; 12: 280.

41. DanielTM. New approaches to the radio – diagnosis of TB meningitis.J Infect Disc 1987; 155: 599.

42. Gerberding JL, Saude AM. Acquired immunodeficiency syndrome. In Stein HJ, et al Internal Medicine, 5th edn. Mosby, 1998 .p.1478.

43. Fauli S, Lane HC. Human immunodeficiency virus disease, AIDs and related disorders in Kasper LD, Braunwald E, Fauci SA, Hauser LS, Longo LD, Jameson LJ. Harrison's principles of internal disease. 16th edn.McGraw Hill, 2005 .p.1115- 9.

44. Bisburg B, Sundervam G, Rerchman LB, Kapila R. CNS tuberculosis with AIDS. Ann Intern Med 1986: 210- 3.

45. Chandha DS, Hand a A, Sharma SK, Varadarajulu P, Singh AP. Seizures in patients with human immunodeficiency virus infection. J Assoc Physician India 2000; 48: 573- 6.

46. Baulac S, Baulac M: Advances on the genetics of mendelian idiopathic epilepsies. Neurol Clin 27:1041, 2009[PMID: 19853223]

47. Bonnett LJ et al: Risk of recurrence after a first seizure and implications for driving: Further analysis of the Multicentre study of early Epilepsy and Single Seizures. BMJ 341:6477, 201

48. Brodie MJ et al: Epilepsy in later life. Lancet Neurol 8:1019, 2009[PMID: 19800848]

49. French JA, Pedley TA: Clinical practice. Initial management of epilepsy. N Engl J Med 359:166, 2008.[PMID: 18614784]

50. ——— et al: Efficacy and tolerability of the new antiepileptic drugs I: Treatment of new onset epilepsy: Report of the Therapeutics and Technology Assessment Subcommittee and Quality Standards Subcommittee of the American Academy of Neurology and the American Epilepsy Society. Neurology 62:1252, 2004

51. ———: Efficacy and tolerability of the new antiepileptic drugs II: Treatment of refractory epilepsy: Report of the Therapeutics and Technology Assessment Subcommittee and Quality Standards Subcommittee of the American Academy of Neurology and the American Epilepsy Society. Neurology 62:1261, 2004

52. Millikan D et al: Emergency treatment of status epilepticus: Current thinking. Emerg Med Clin North Am 27:101, 2009[PMID: 19218022]

53. Pitkänen A, Lukasiuk K: Molecular and cellular basis of epileptogenesis in symptomatic epilepsy. Epilepsy Behav 14:16, 2009[PMID: 17023142]

54. Rakhade SN, Jensen FE. Epileptogenesis in the immature brain: emerging mechanisms. Nat Rev Neurol 5:380, 2009[PMID: 19578345]

55. Schuele SU, Lüders HO: Intractable epilepsy: management and therapeutic alternatives. Lancet Neurol 7:514, 2008[PMID: 18485315]

56. Tomson T et al: Sudden unexpected death in epilepsy: current knowledge and future directions. Lancet Neurol 7:1021, 2008[PMID: 18805738]

57. Walker SP et al: The management of epilepsy in pregnancy. BJOG 116:758, 2009[PMID: 19432564]

58. Ramamurthy B, Varadarajan MG . Diagnosis of tuberculomas of the brain: Clinical and radiological correlation. J Neurosurg 1961; 8:1

59. Garg RK. Diagnosis of intracranial tuberculoma. Ind J Tub 1996; 43: 35- 39

60. Gree GT, Bazan III C, Jinkings JR. Miliary tuberculosis involving the brain MR findings. ARJ 1992; 159: 1075.

61. Jinkins JR Computed tomography of intracranial tuberculosis Neuroradiology 1991; 33: 186.

LIST OF ABBREVATIONS USED

AED	Antiepileptic Drug
CT	Computerized Tomography
EEG	Electroencephalo Gram
EITBA	Electroimmuno Transfer Blot Assay
ELISA	Enzyme Linked Immunosorbent Assay
FNAC	Fine Needle Aspiration Cytology
GABA	Gamma Amino Butyric Acid
GTCS	Generalized Tonic Clonic Seizures
HIV	Human Immunodeficiency Virus
MRI	Magnetic Resonance Imaging
NCC	Neurocysticercosis
PCDD	Primary Cerebral Degenerative Disorder
PCR	Polymerase Chain Reaction
RBS	Random Blood Sugar
SCCG	Solitory Cerebral Cysticercal Granuloma
SSECL	Single Small Enhancing CT Lesion
TIA	Transient Ischemic Attack

PROFORMA

NAME : DIAGNOSIS:

AGE/SEX:

OCCUPATION:

ADDRESS:

DETAILS OF PRESENT ILLNESS:

SEIZURES:

- Time of onset
- Duration
- Aura
- Any precipitating factors
- Partial (face/ hand/ upper limb/ lower limb/ foot)
- Generalised (LOC, tongue bite, bladder/bowel incontinence, post ictal confusion, residual neurological deficit)
- Number of episodes

ASSOCIATED COMPLAINTS:

- Fever
- Headache
- Vomiting
- Focal neurological deficit
- Head injury

PAST HISTORY: DM-2 / SHT /TB / CVA / Meningitis / Encephalitis / RHD

FAMILY HISTORY: DM-2/SHT/Seizure disorder/Mental retardation

PERSONAL HISTORY: Sleep,Diet, Smoking, Alcoholism,Drug abuse,

Tobacco chewing

GENERAL EXAMINATION:

- Built & nourishment:
- Height: weight: BMI:
- P/I/CY/CL/LN/PE
- VITALS- PR.BP,RR,TEMPERATURE
- Cutaneous stigmata of neuroepidermal syndromes

  -adenoma sebaceum,

  -café-au-lait spots,

  -neurofibromatosis,

  -ash leaf macules,

  -shagreen patch

SYSTEMIC EXAMINATION

❖ Central nervous system

- Higher functions :

- Cranial nerves :
- Motor system :
- Sensory system :
- Reflexes :
- Autonomic nervous system :
- Skull & spine :
- Meningeal signs :
  - ❖ Cardiovascular system
  - ❖ Respiratory system
  - ❖ Abdominal system

INVESTIGATIONS:

- ❖ CBC – TC,DC, ESR, Hb,Platelet count.
- ❖ BIOCHEMISTRY:-
  - RBS,FBS,PPBS
  - B.Urea, S.Creatinine.,
  - Serum electrolytes(Na, K, Ca)
- ❖ URINE ROUTINE EXAMINATION---sugar,albumin, deposits
- ❖ CHEST X-RAY

- HIV - ELISA
- CSF ANALYSIS- appearance, pressure,sugar, protein, cell count, gram stain, cytology, culture
- EEG
- CT- BRAIN
- MRI- BRAIN

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.1223/ME-1/Ethics/2013 Dt:07.03.2013.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study on clinical profile and evaluation of new onset seizure in adults" for Project work submitted by Dr. M.Dhanasekar MD (GM), IInd year PG Student, Kilpauk Medical College, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

19/4/13

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

GOVT. KILPAUK MEDICAL COLLEGE
9 APR 2013
Kilpauk, Chennai-10.

S. NO	NAME	AGE	SEX	SEIZURE TYPE	NO OF EPISODES	FEVER	HEADACHE	VOMITING	PAST HISTORY	PERSONAL HISTORY	GPE	CNS EXAMINATION	BLOOD SUGAR mg%	BLOOD UREA mg%	Sr.CREATINE mg%	Sr.SODIUM mEq/L	Sr.POTASSIUM mEq/L	Sr.CALCIUM mg%	CSF	CXR	EEG	CT BRAIN	ETIOLOGY
1	RAJU	30	M	GTCS	4	NO	NO	NO	NOT REL	SMOKER	NORMA	NORMAL	126	26	1.3	142	4.2	8	-	NORMAL	-	ACTIVE GRANULOMA IN Rt HIGH PARIE	TUBERCULOMA
2	VINAYAGAMOORTHY	29	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	108	36	0.7	138	3.8	8.1	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
3	MASIATH	61	M	GTCS	1	YES	YES	NO	NOT REL	SMOKER	NORMA	NECK STIFFN	106	42	1.2	140	4.1	8.1	BACTEI	NORMAL	-	NORMAL STUDY	BACTERIAL MENINGITIS
4	SUGANTHI	20	F	GTCS	1	NO	YES	NO	NOT REL	-	NORMA	NORMAL	135	20	0.9	138	4	7.8	-	NORMAL	-	MODERATELY ENHANCING MASS IN ANT	TUBERCULOMA
5	SAGAYAMARY	43	F	FOCAL	5	NO	NO	NO	DM	-	UNCON	UNCONSCIOI	40	33	1.3	144	5	8	-	NORMAL	NORMAL	NORMAL STUDY	HYPOGLYCAEMIA
6	KUPPAN	56	M	FOCAL	3	NO	NO	NO	NOT REL	ALCOHOL	UNCON	UNCONSCIOI	90	22	1.3	142	3.4	9	-	NORMAL	NORMAL	MILD CEREBRAL EDEMA	ALCOHOL WITHDRAWAL
7	VENKATACHALAM	22	M	GTCS	4	NO	NO	YES	NOT REL	ALCOHOL	NORMA	NORMAL	113	25	0.8	135	4.5	8.7	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
8	MARIYAMMAL	56	F	GTCS	3	NO	NO	NO	NOT REL	-	UNCON	UNCONSCIOI	132	35	0.8	143	4	8.6	-	NORMAL	-	Lt MCA TERRITORY INFARCT	CVA - INFARCT
9	POVARASAN	40	M	FOCAL	2	NO	NO	NO	NOT REL	SMOKER	UNCON	UNCONSCIOI	122	25	1.3	144	5	8	-	NORMAL	-	MASSIVE INFARCT IN Rt PARIETAL LOBE	CVA - INFARCT
10	SHREE	18	M	GTCS	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	132	33	1	134	4	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
11	MARI	21	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	156	30	1	136	4.6	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
12	MUNIYAN	28	M	GTCS	1	YES	YES	YES	NOT REL	ALCOHOL	IRRITAE	NECK STIFFN	112	32	1.4	154	4.7	8.3	BACTEI	NORMAL	-	NORMAL STUDY	BACTERIAL MENINGOENCEPHALITIS
13	KANMANI	33	F	FOCAL	1	NO	YES	YES	NOT REL	-	IRRITAE	IRRITABLE	135	33	1	145	4	8	-	NORMAL	-	SSS THROMBOSIS	CORTICAL VENOUS THROMBOSIS
14	SENTHIL	19	M	FOCAL	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	98	32	1.1	147	4	8.2	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
15	RAMESH	26	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	134	27	1.2	137	5	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
16	SARASU	56	F	FOCAL	2	NO	YES	YES	SHT	-	UNCON	UNCONSCIOI	122	32	0.9	143	5.1	8.5	-	NORMAL	-	INFARCT IN LtPARIETO OCCIPITAL REGI	CVA - INFARCT
17	RAKESH	18	M	FOCAL	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	111	25	1	145	4	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
18	ANANTHI	24	F	GTCS	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	157	40	1	138	4.9	8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
19	SELVI	20	F	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	187	28	1	143	3.6	8.9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
20	PALANI	40	M	GTCS	1	YES	NO	NO	SHT	ALCOHOL	IRRITAE	NECK STIFFN	112	33	0.7	153	4.3	8	TUBER(	NORMAL	-	NORMAL STUDY	TB MENINGITIS
21	MANESH	23	M	FOCAL	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	89	24	1	137	4	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
22	SURESH	42	M	FOCAL	3	NO	NO	NO	NOT REL	ALCOHOL	UNCON	UNCONSCIOI	87	34	1.2	139	3.7	9.8	-	NORMAL	NORMAL	MILD CEREBRAL EDEMA	ALCOHOL WITHDRAWAL
23	PAVAYEE	57	F	FOCAL	1	NO	NO	NO	NOT REL	-	UNCON	UNCONSCIOI	122	22	1	140	5	8	-	NORMAL	-	Rt MCA TERRITORY INFARCT	CVA - INFARCT
24	PARVATHY	55	F	FOCAL	1	NO	NO	NO	DM	-	UNCON	UNCONSCIOI	40	23	1.1	144	4	9	-	NORMAL	NORMAL	NORMAL STUDY	HYPOGLYCAEMIA
25	JANCI	19	F	FOCAL	3	YES	YES	YES	NOT REL	-	NORMA	NORMAL	112	33	1	136	5	8	-	NORMAL	-	RING ENHANCING LESION IN PARIETAL L	TUBERCULOMA
26	VASANTH	25	M	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	90	30	1	1145	4	8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
27	ANTONY	34	M	FOCAL	3	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	189	33	1.3	133	3.7	8.8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
28	BABU	29	M	GTCS	2	NO	NO	YES	NOT REL	ALCOHOL	NORMA	NORMAL	122	24	0.9	142	4.6	9	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
29	PAVALAM	55	F	GTCS	4	NO	NO	NO	SHT	-	UNCON	UNCONSCIOI	165	34	1.4	112	4	8	-	NORMAL	NORMAL	NORMAL STUDY	HYPONATRAEMIA
30	SHANMUGAM	27	M	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	122	33	1	144	3.6	7.8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
31	RATHINAM	50	F	GTCS	1	NO	YES	YES	SHT	-	UNCON	UNCONSCIOI	123	32	0.9	152	4	8.9	-	NORMAL	-	ACUTE INFARCT IN Rt MCA TERRITORY	CVA - INFARCT
32	VISHNU	42	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	UNCON	UNCONSCIOI	113	26	1	145	3.8	8.6	-	NORMAL	NORMAL	MILD CEREBRAL EDEMA	ALCOHOL WITHDRAWAL
33	NISHANTH	25	M	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	99	22	1	154	4	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
34	SELVAM	36	M	GTCS	1	YES	YES	YES	NOT REL	ALCOHOL	IRRITAE	NECK STIFFN	166	32	1.3	137	3.9	9.5	BACTEI	NORMAL	-	NORMAL STUDY	BACTERIAL MENINGO ENCEPHALITIS
35	MASILAMANI	58	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	123	27	1	144	3.8	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
36	ROHID	26	M	GTCS	2	NO	NO	NO	NOT REL	-	NORMA	NORMAL	89	44	2	154	5.3	7.8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
37	MUKESH	42	M	GTCS	2	NO	YES	YES	SHT	ALCOHOL	UNCON	UNCONSCIOI	112	23	1	143	5	9	-	NORMAL	-	INTRACEREBRAL HAEMORRHAGE	CVA -HAEMORRHAGE
38	MAHESH	36	M	FOCAL	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	143	31	1	135	3.8	9	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
39	MARIYAPPAN	58	M	GTCS	6	NO	NO	NO	DM	ALCOHOL	UNCON	UNCONSCIOI	TOO LO	34	0.9	145	4.5	8.7	-	NORMAL	NORMAL	NORMAL STUDY	HYPOGLYCAEMIA
40	SUNDAR	27	M	FOCAL	2	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	122	20	0.8	146	4.8	9	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
41	GOWRI	24	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	109	37	0.7	132	4.9	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
42	MALARVIZHI	22	F	FOCAL	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	102	34	1	144	4.4	9.4	-	NORMAL	-	SINGLE RING ENHANCING LESION IN FRC	NEUROCYSTICERCOSIS
43	SIVARAJ	22	M	GTCS	4	NO	NO	NO	NOT REL	-	NORMA	NORMAL	100	28	1	154	5	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
44	BASHA	35	M	GTCS	3	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	108	21	1.1	147	4	9.4	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
45	DAVID	33	M	GTCS	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	133	34	1.4	140	4	8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
46	MOHAMMAD	58	M	GTCS	1	NO	YES	YES	SHT	ALCOHOL	UNCON	UNCONSCIOI	143	32	1	145	5	9	-	NORMAL	-	INTRACEREBRAL HAEMORRHAGE	CVA -HAEMORRHAGE
47	DEVENDRAN	53	M	GTCS	5	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	192	24	1.2	149	3.8	9.5	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
48	BALAN	26	M	GTCS	3	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	99	22	0.8	146	3.5	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
49	KATHIR	34	M	FOCAL	2	NO	NO	NO	NOT REL	-	NORMA	NORMAL	101	1.1	21	141	4.1	9.1	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
50	KANNAN	24	M	GTCS	4	NO	NO	YES	NOT REL	ALCOHOL	NORMA	NORMAL	91	26	0.7	134	4.6	9	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL

51	APPURAJ	34	M	GTCS	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	96	33	1	138	4	9.1	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
52	SURESH	25	M	FOCAL	5	YES	YES	YES	NOT REL	ALCOHOL	NORMA	NORMAL	154	22	0.8	144	4.3	8.7	-	NORMAL	-	FOCAL ENHANCING LESION IN FRONTAL	TUBERCULOMA
53	ISMAAIL	31	M	GTCS	4	NO	NO	YES	NOT REL	ALCOHOL	NORMA	NORMAL	165	32	0.9	144	5.1	8	-	NORMAL	NORMAL	MILD CEREBRAL EDEMA	ALCOHOL WITHDRAWAL
54	MURUGAMMAL	57	F	GTCS	4	NO	NO	NO	DM	-	UNCON	UNCONSCIOI	44	22	0.8	145	4.6	9	-	NORMAL	NORMAL	NORMAL STUDY	HYPOGLYCAEMIA
55	RAMESH	45	M	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	146	34	1.8	141	4.7	9.8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
56	VIJAYAKUMAR	47	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	176	41	1	143	4	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
57	RAGHUPATHY	51	M	GTCS	2	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	143	22	0.9	145	3.8	8.6	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
58	SRIDHARAN	36	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	UNCON	UNCONSCIOI	132	26	1	143	3.6	8.3	-	NORMAL	NORMAL	MILD CEREBRAL EDEMA	ALCOHOL WITHDRAWAL
59	ANITHA	20	F	FOCAL	1	YES	NO	NO	NOT REL	-	NORMA	NORMAL	109	20	1	153	4.8	8.5	-	NORMAL	-	MULTIPLE CALCIFIED LESION IN CEREBF	NEUROCYSTICERCOSIS
60	RUKMANI	56	F	GTCS	2	NO	NO	NO	SHT	-	NORMA	NORMAL	176	35	1.4	145	4.6	4.2	-	NORMAL	NORMAL	NORMAL STUDY	HYPOCALCAEMIA
61	SANTHI	52	F	GTCS	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	179	36	1	145	4.9	7.9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
62	KARUPPAYEE	66	F	FOCAL	4	NO	NO	NO	SHT	-	UNCON	UNCONSCIOI	122	32	1	114	5	9	-	NORMAL	NORMAL	NORMAL STUDY	HYPONATRAEMIA
63	REVATHY	56	F	GTCS	1	NO	YES	YES	SHT	-	UNCON	UNCONSCIOI	167	24	1.6	153	4	8	-	NORMAL	-	INTRACEREBRAL HAEMORRHAGE	CVA -HAEMORRHAGE
64	NAGARAJAN	44	M	GTCS	5	NO	NO	YES	NOT REL	ALCOHOL	UNCON	UNCONSCIOI	154	30	1.1	145	3.6	8.6	-	NORMAL	NORMAL	MILD CEREBRAL EDEMA	ALCOHOL WITHDRAWAL
65	PARTHASARATHY	62	M	GTCS	2	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	156	32	0.8	144	3.7	8.6	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
66	STELLA	68	F	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	100	1	20	140	4	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
67	PRABA	22	F	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	139	33	0.9	136	4	8.8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
68	RUKKU	43	F	FOCAL	2	NO	NO	NO	NOT REL	-	NORMA	NORMAL	132	23	1.2	154	4	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
69	KRISHNAVENI	28	F	FOCAL	1	YES	NO	NO	NOT REL	-	NORMA	NORMAL	144	41	1.3	143	4.6	8.4	-	NORMAL	-	MULTIPLE HEALED CALCIFIED GRANULC	TUBERCULOMA
70	PAARU	33	F	GTCS	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	199	20	1	152	4.6	8.3	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
71	ARASI	42	F	GTCS	4	NO	YES	YES	SHT	-	UNCON	UNCONSCIOI	189	32	1.6	155	4	8	-	NORMAL	-	SAH	CVA - SAH
72	KANNAMMA	45	F	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	121	22	1	137	4.4	9	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
73	RAJAN	37	M	GTCS	2	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	104	25	0.7	146	3.7	9.8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
74	FATHIMA	34	F	GTCS	4	NO	NO	NO	NOT REL	-	NORMA	NORMAL	123	28	1	146	4.2	8.7	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
75	PANJALI	48	F	FOCAL	6	NO	NO	NO	DM	-	NORMA	NORMAL	512	34	1.6	146	4.8	9	-	NORMAL	NORMAL	NORMAL STUDY	HYPERGLYCAEMIAs
76	ESWARI	35	F	GTCS	4	NO	NO	NO	NOT REL	-	NORMA	NORMAL	111	22	1	144	4	8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
77	HARI	43	M	GTCS	4	NO	NO	YES	NOT REL	ALCOHOL	NORMA	NORMAL	97	33	1	147	3.5	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
78	ASWINI	38	F	FOCAL	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	156	45	1.6	154	4	9.4	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
79	KAMARAJ	38	M	GTCS	2	NO	NO	YES	NOT REL	ALCOHOL	NORMA	NORMAL	85	22	0.7	145	4.3	9	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
80	BANUMATHY	45	F	GTCS	3	NO	YES	YES	SHT	-	UNCON	UNCONSCIOI	165	32	0.8	145	4.7	8.4	-	NORMAL	-	SAH	CVA - SAH
81	KUPPAMMAL	18	F	FOCAL	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	113	33	0.8	150	5	9	-	NORMAL	NORMAL	SINGLE RING ENHANCING LESION IN FRC	NEUROCYSTICERCOSIS
82	MOHAN	28	M	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	85	27	1	143	4	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
83	MUNIYAMMAL	38	F	GTCS	1	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	158	35	0.9	147	3.8	8.7	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
84	RAJESHWARI	34	F	FOCAL	2	YES	NO	NO	NOT REL	-	NORMA	NORMAL	122	24	0.8	136	4.5	7.8	-	PTB	-	3 RING ENHANCING LESION IN PARIETAL	TUBERCULOMA
85	KAMATCHI	65	F	GTCS	2	YES	YES	NO	SHT	-	IRRITAE	IRRITABLE	134	34	1.2	145	4.4	8.6	NAD	NORMAL	-	NORMAL STUDY	CEREBRAL MALARIA
86	ABI	37	F	GTCS	4	NO	NO	NO	NOT REL	-	NORMA	NORMAL	98	43	1	134	5	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
87	ROHINI	29	F	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	122	31	0.8	146	4	7.8	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
88	ESWARAMOORTHY	55	M	GTCS	3	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	154	26	0.7	147	3.7	8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
89	KASTHURI	60	F	GTCS	1	YES	YES	YES	NOT REL	-	IRRITAE	NECK STIFFI	106	34	1.2	156	4.8	9.2	VIRAL	NORMAL	-	NORMAL STUDY	VIRAL MENINGOENCEPHALITIS
90	SUGANYA	18	F	GTCS	4	NO	NO	NO	NOT REL	-	NORMA	NORMAL	157	34	1	135	5	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
91	JANAKI	41	F	GTCS	3	NO	NO	NO	NOT REL	ALCOHOL	UNCON	UNCONSCIOI	108	25	0.9	146	4	7.8	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
92	ARTHY	19	F	GTCS	3	NO	NO	NO	NOT REL	-	NORMA	NORMAL	122	34	1	145	5	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
93	BALU	58	M	GTCS	5	NO	YES	YES	SHT	ALCOHOL	UNCON	UNCONSCIOI	176	33	1	134	5	8	-	NORMAL	-	SDH	CVA - SDH
94	MURUGESAN	47	M	GTCS	4	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	144	32	1.1	144	4.5	8.4	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
95	PRABHAKAR	21	M	FOCAL	4	YES	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	122	34	1	135	4.8	8	-	NORMAL	-	MULTIPLE RING ENHANCING LESIONS IN	NEUROCYSTICERCOSIS
96	KANNIYAMMAL	68	F	FOCAL	6	NO	NO	NO	DM	-	UNCON	UNCONSCIOI	52	35	0.9	148	4.8	8.6	-	NORMAL	NORMAL	NORMAL STUDY	HYPOGLYCAEMIA
97	SOORYA	23	M	GTCS	4	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	130	21	1.3	144	4	8.6	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
98	BANUMATHY	20	F	FOCAL	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	176	24	1	154	5	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC
99	GANAPATHY	65	M	GTCS	4	NO	NO	NO	NOT REL	ALCOHOL	NORMA	NORMAL	135	27	1.4	135	4.1	8.6	-	NORMAL	NORMAL	NORMAL STUDY	ALCOHOL WITHDRAWAL
100	JAYANTHI	20	F	GTCS	1	NO	NO	NO	NOT REL	-	NORMA	NORMAL	133	24	1	143	4	9	-	NORMAL	NORMAL	NORMAL STUDY	IDIOPATHIC