STUDY ON ETIOLOGICAL AND CLINICAL PROFIILE OF ACUTE SYMPTOMATIC SEIZURES IN ADULTS IN A TERTIARY CARE HOSPITAL

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BRANCH - I
NEUROLOGY

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CERTIFICATE

This is to certify that this dissertation titled **"STUDY ON ETIOLOGICAL AND CLINICAL PROFIILE OF ACUTE SYMPTOMATIC SEIZURES IN ADULTS IN A TERTIARY CARE HOSPITAL "** is a bonafide work done by **Dr. E. AMAL RAJ IYADURAI**, Department of Neurology, Government Rajaji Hospital, Madurai Medical College, Madurai under my guidance and supervision in partial fulfillment of the regulations of The Tamilnadu Dr.M.G.R.Medical University for the award of DM**, Branch I (Neurology)** during the academic period of **August 2011 to August 2014.**

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DECLARATION

I, **Dr. E. AMAL RAJ IYADURAI** solemnly declare that the dissertation titled **"STUDY ON ETIOLOGICAL AND CLINICAL PROFIILE OF ACUTE SYMPTOMATIC SEIZURES IN ADULTS IN A TERTIARY CARE HOSPITAL"** has been prepared by me. This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of **D.M., Branch I (Neurology)** to be held in **August 2014.**

Place : Madurai **Dr. E. AMAL RAJ IYADURAI**

Date :

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CONTENTS

STUDY ON ETIOLOGICAL AND CLINICAL PROFIILE OF ACUTE SYMPTOMATIC SEIZURES IN ADULTS IN A TERTIARY CARE HOSPITAL

ABSTRACT:

SETTING: Department of Neurology**,** Government Rajaji Hospital, Madurai, Tamilnadu.

OBJECTIVES: 1. To study the etiological profile of acute symptomatic seizures in various age groups. 2. To assess the common seizure type in patients with acute symptomatic seizures of varied etiologies. 3. To study the Electro Encephalographic and Radiological profile of Acute symptomatic seizures.

DESIGN: Single Observational study**.** This study was conducted among 150 Acute symptomatic seizure patients who were admitted in our hospital with various etiologies since March 2013 to February 2014.

RESULTS: In the present study Acute symptomatic seizures were slightly more in males than in females. Acute symptomatic seizures were most common in 40 – 60 years of age group. Generalised seizures were the most common seizure type encountered in the study. Head ache, vomiting and altered sensorium were the most common non convulsive presenting symptoms. Diabetes and Hypertension were the co morbid systemic illness associated with cerebrovascular accidents. Metabolic abnormality was found as the cause in 13% of the patients and the predominant age group was above 60 years. Cerebrovascular diseases were the most frequent etiology in acute symptomatic seizures. Among the cerebrovascular diseases, cortical venous thrombosis and intra cerebral haemorrhage were commonly presented with seizure. Next common cause of acute symptomatic seizures in this study was CNS infections consisting of 19% of the patients. Among CNS infection, Neurocysticercosis was the

most common cause. In females eclampsia and cortical venous thrombosis were the common etiology for acute symptomatic seizures. Alcohol related seizures contributed to etiology in 12% of the patients. Hanging and post cardiac arrest were the common etiology the anoxic brain injury. Generalised seizures were common with cerebrovascular disease and alcohol related seizures. Partial seizures were common with Tuberculoma and Neurocysticercosis. Myoclonic seizures were common with anoxic brain injury. Cerebrovascular disease and Metabolic abnormality were common above 60 years where as eclampsia and alcohol related seizures were common in 20 – 60 years of age. Acute CNS infection was the predominant cause of seizures below 20 years of age.

Conclusion: Acute symptomatic seizures were more common in males than females and in 40 – 60 years of age. Cerebrovascular diseases were the most frequent cause of acute symptomatic seizures, followed by Acute CNS infections. Eclampsia and cortical venous thrombosis were the common etiology among females. Cerebrovascular diseases and metabolic abnormality were common above 60 years of age where as eclampsia and alcohol related seizures were common in 20-60 years of age. Acute CNS infections were the predominant cause of acute symptomatic seizures below 20 years of age. Generalised seizures were the most common seizure type encountered in this study. EEG and Radiological abnormalities were seen in nearly 60% of the patients.

Keywords: GTCS, CVT, SDH, SAH

INTRODUCTION

The foundation of our modern understanding of the derangement of brain function seen in seizures and epilepsy was laid in the 19th century with work of Hughlings Jackson.

The word seizure is derived from Latin word “Sacire” meaning ‘to take possession of’ indicating that the person having a seizure is possessed or atleast out of control.

Epilepsy can be broadly divided into idiopathic and symptomatic disorders. Idiopathic epilepsies are not associated with brain lesions or neurological abnormalities ie they are relatively benign. They tend to be self limited and respond well to antiepileptic therapy.

Symptomatic epilepsy is the one in which seizures are the consequence of an identifiable lesion or other specific etiology.

An acute symptomatic seizure was defined in a recent recommendation from the International League Against Epilepsy (ILAE) as a clinical seizure occurring in close temporal relationship with an acute central nervous system insult which may be metabolic, toxic, infectious or inflammatory.[1]

Cumulative observations of many clinical investigations along with adjuctive neurophysiological, imaging and genetic tools created a well accepted diversity in the etiologies of seizures in various age groups.

Efficacy of acute symptomatic seizure treatment is thought to depend on early determination of all reversible insults and their rapid correction. Patients with acute symptomatic seizure do not need to be treated with antiepileptic drugs on a long term basis, although such treatment may be warranted on a short term basis until the acute condition is resolved. [44]

As acute symptomatic seizures reflect at least partially, the severity of insult, it is understandable that their occurrence is generally associated with a poor outcome.

This study is to evaluate the clinical, etiological and radiological profile of acute symptomatic seizures in adults.

REVIEW OF LITERATURE

Acute symptomatic seizures are seizures closely related to neurological or systemic insults and represent about 40% of all first seizures.[1] It may be difficult to recognize the severity of insult needed to provoke epileptic seizures or in determining a clear temporal relationship. Appropriate therapeutic management, risk of developing epilepsy and mortality depend largely on the underlying disorder.

Severity of Insult Required to Provoke Seizures :

The severity of insult needed to provoke a seizure can be understood considering 4 conceptual models. [2]

1. **Acute disease model.**

   (eg) Progressive organic dysfunction in which multiple insults are needed to provoke a seizure.

2. **Chronic disease model**

   (eg) Stroke, Chronic renal failure in which seizures occur after a single insult in the context of chronic disease.

3. **Unique insult model**

   (eg) eclampsia in which seizures happen after a high magnitude insult.

4. **Genetic predisposition**

in which seizures occur after an insult of relatively low intensity.

CAUSES OF ACUTE SYMPTOMATIC SEIZURES[3]

- Acute stroke
- Traumatic Brain Injury
- Central Nervous System Infection
- Medication
- Alcohol
- Illicit drugs
- Electrolyte and Metabolic disorders
- Anoxic Encephalopathy
- Eclampsia
- Reversible Posterior Leukoencephlopathy
- Limbic Encephalitis

Delay Between Insult and Seizure :

With regard to the temporal relationship needed for an event to be considered an acute symptomatic seizure, it consists of the period until clinical stabilisation of disease which is a subjective concept in clinical practice. [1]

ILAE has conventionally considered

1. The period of 1 week in stroke, head trauma or anoxic encephalopathy.
2. The active phase in CNS infection or inflammatory disease based on persistent clinical, laboratorial or imaging findings.
3. Within 24 hours in documented severe selected metabolic derangements.
4. Within 7 to 48 hours of the last drink in alcohol withdrawal. [1]

Epidemiology of Acute Symptomatic Seizures

Population based studies showed that the cumulative risk for acute symptomatic seizures from birth to 80 yrs of age is 3.6%. In males the incidence is more which is related to underlying condition such as Head trauma rather than any biological phenomenon.

Acute symptomatic seizure commonly seen in age extremes during which period infection and vascular disorder are common. [3]

Acute symptomatic seizures caused by both neurological insults and diseases with systemic involvement.

NEUROLOGICAL INSULTS

Acute Stroke :

The Haemorrhagic stroke and cortical lesions are independent predictors of acute symptomatic seizures. Most seizures occur within 2 days and almost half within 24 hours after stroke. Seizures are mostly focal or focal with secondary generalization. The seizure onset zone corresponds to ischemic penumbra. The frequency of acute zone corresponds to ischaemic penumbra. The frequency of acute symptomatic seizures is almost twice in hemorrhagic stroke compared with the ischaemic stroke. [5]

Subarachnoid haemorrhage :

The seizures in subarachnoid haemorrhage is because of high blood volume in basal cisterns but not due to duration of loss of consciousness, presence of aneurysm or high blood pressure. Blood in cisterns that may have an irritant effect in the brain cortex[6] and the ischemic brain lesion caused by arterial vasospasm induce seizures. Most patients present generalized seizures, suggesting a large seizure onset zone. Prophylactic AED is necessary in the immediate post haemorrhagic period. [7]

Intracerebral haemorrhage :

Acute symptomatic seizures correlate with temporal or Parietal cortical involvement. Seizures possibly occur due to the presence of blood metabolism products namely hemosiderin that seems to have epileptogenic activity. AEDs are recommended during the acute phase and may be considered prophylactically also. [7] Risk of developing epilepsy after an acute symptomatic seizure is present commonly. [8]

Ischemic Stroke:

Severe infarcts and cortical involvement cause symptomatic seizures. In lacunar stroke MRI revealed ipsilateral posterofrontal or anterotemporal cortical ischemic lesion. Single photon emission CT showed hypoperfusion in the ipsilateral frontal area in all patients having lacunar stroke. [9]

Acute brain ischemia leads to a high concentration of extracellular glutamate that has been shown to have the ability to produce recurrent epileptiform discharges. [10] Acute symptomatic seizures are possibly harmful for ischemic penumbra due to additional metabolic stress involving that vulnerable zone. Repeated seizures in the context of brain ischemia increase infarct size and may harm functional recovery. [11]

Cerebral Venous Thrombosis :

In CVT 40% of patients show seizures at presentation of disease and a few percentage of patients have seizures with in 2 weeks of the insult. Those who are having focal neurological deficit more prone to develop seizures. The localization of CVT (superior sagittal sinus and cortical vein) and parenchymal lesions mainly if supratentorial are mostly associated with seizures. [12]

Acute symptomatic seizures may lead to neurological and systemic deterioration, status epilepticus and death .There is a moderate risk of developing epilepsy with in 1 year after acute CVT seizure.

Traumatic Brain Injury.

Acute Symptomatic seizures occur in 6% of patients who experience a traumatic brain injury. [13] It is common in children. Loss of consciousness or amnesia for >30 minutes, acute intracerebral hemorrhage or subdural hematomas are more prone to develop seizures. Prophylaxis with phenytoin is recommended for acute symptomatic seizure after severe injury.

Risk of epilepsy is more in adults than in children. [14] Mesial temporal lobe epilepsy may result from traumatic Brain Injury in adolescents and adults and not exclusively in children. Post Traumatic Epilepsy begins in

most patients within the first year although in severe injury it may happen upto 20 yrs later. There may be stepwise changes that occur in the neuronal network over days to weeks or even months and years, after an epileptogenic insult.

Later changes occurring over days to weeks include anatomic changes including axonal sprouting and dendritic modifications, such as the mossy fiber sprouting that is commonly observed as a hall mark of the chronic epileptic brain. [15]

CNS Infection or infestation :

Seizures are more common in encephalitis (14 times more than meningitis), In herpes simplex encephalitis, acute symptomatic seizures occur in 40 % to 60% of patients reflecting the virus tropism to the mesial temporal lobe. In neurocysticercosis the acute symptomatic seizures may occur during calcified cystic stage.

Seizures may occur in acute CNS Infection setting mediated by proinflammatory cytokines such as IL-1 and tumoral necrosis factor inhibit gama aminobutyric acid (GABA) receptors and lower seizure threshold. [16]

Neurocysticercosis is the most common cause of epilepsy in developing countries where it accounts for up to 30% of all seizures. [17] Epilepsy usually begins within 5 years although it can occur 20 years after CNS Infection.

Refractory epilepsies developed after CNS infections are predominantly multifocal and temporal lobe epilepsies. Multifocal epilepsies usually occur after severe encephalitis with diffuse brain lesions. Temporal Lobe epilepsy may develop if CNS Infection occurs at young ages or if the etiologic agent involved presents tropism for this lobe.

Acute meningitis or encephalitis occurring before 4years of age are more commonly related to hippocampal sclerosis and when occurring after this age they are more frequently associated with extra hippocampal neocortical epilepsy. Recently the association between human herpesvirus-6 (HHV -6) infections, complex febrile seizures, and development of temporal lobe epilepsy has been under investigation. [18]

DISEASES WITH SYSTEMIC INVOLVEMENT

Medication

Acute symptomatic seizures may occur after use or withdrawal of medication.

Medication related to moderate risk of seizures are chlorpromazine, clozapine, clomipramine, bupropion, meperidine and flumazenil. A particularly high risk of seizures in seen in overdose of cyclic antidepressants (especially amoxapine and maprotiline), alkylating antineoplastic agents, isoniazid , theophylline and cyclosporine.

Penecillin prevents GABA from binding to the GABA A receptor. [19] Cephalosporins, imipenam and fluroquinolones antagonise GABA A receptors. Isoniazid competes with pyridoxine, which is usually transformed into pyridoxal phosphate, a cofactor for GABA synthesis thus leading to a decrease in GABA Levels. [20]

Seizures can be a feature of withdrawal from barbiturates, benzodiazepines. Short- acting barbiturates are most likely to produce seizures between day 1 and day 5 after discontinuation. Seizures during benzodiazepine withdrawal usually occur within 24 hours of stopping a short

acting agent and within several days of stopping a long acting agent. Withdrawal of any anticonvulsant and narcotic drugs can give rise to seizures.

Alcohol

Acute symptomatic seizures may occur after alcohol withdrawal or acute intoxication. Head injury and cerebrovascular accident may also be predisposed by alcohol intake.

Indication of alcohol withdrawal seizures include history of chronic alcohol abuse, history of recent reduction in consumption and generalized tonic clonic seizure with other symptoms of withdrawal such as tremors, sweats or tachycardia. The seizure must occur within 7 to 48 hours of the last drink. Less than 10% of patients may present as status epilepticus. Seizures are more commonly generalized and characteristically show normal interictal EEG. [21]

Acute alcohol ingestion has an inhibitory effect on N- Methyl –D-Aspartate (NMDA) receptors, reducing excitatory glutamatergic transmission and has an agonistic effect on GABA A receptors. In chronic alcohol abuse NMDA receptors are upregulated and GABA A receptors are downregulated leading to tolerance. The roles are reversed during abstinence with enchanced NMDA receptor function and reduced GABAergic

transmission leading to many of the symptoms and signs of acute withdrawal syndrome including seizures.[22] The seizure onset zone is located in the brainstem probably in the inferior colliculus and also involves the amygdala but not neocortex. Benzodiazepines are the drug of choice for alcohol withdrawal syndrome.

Illicit Drugs

Drug related seizures are mostly generalized but if focal may represent a cocaine related stroke. Cocaine related seizures common in women than men and within hours after drug abuse particularly if smoked and not necessarily with concomitant overdose signs. Amphetamine related seizures are often accompanied with other signs of over dose (fever, Hypertension, cardiac arrhythmias, delirium or coma).

Hallucinogens cause seizures only in very high doses. Heroin overdose with coma, pinpoint pupils and respiratory depression, is sometimes associated with seizures.

Electrolyte and Metabolic Disorders.

Acute symptomatic seizures are defined based on the blood sample obtained within 24 hours of the seizure, when it is logical to assume that the findings are similar to those at the time of seizure. The more rapid the disturbance develops the more likely it is to induce seizure. [23]

Seizures may occur in hyponatremia <115mg/ dl, especially when a rapid decrease of serum sodium occurs. Hypernatremia may cause seizure especially during rehydration and it is recommended that sodium convention is reduced at a rate below 0.5mmol /l- /h to present seizures. Hypomagnesemia < 0.8mg / dl and hypocalcemia < 5.0mg / dl predispose patients to seizures. Hypoglycemia <36 mg / dl cases seizures by mechanisms similar to those of cerebral hypoxia. Damage is greatest at the same levels of the cerebral cortex, hippocampus, striatum and cerebellum that are most vulnerable to hypoxia. Nonketotic hyperosmolar coma more commonly result in seizure than does diabetic ketoacidosis probably due to the anticonvulsant effect of ketosis. AED treatment is usually not needed. [24]

Anoxic Encephalopathy

Common after cardio respiratory arrest. Myoclonic and tonic clonic seizure are the predominate seizure types. [25] Myoclonic status with in 24 hours of the insult is recorded as a strong predictor of poor vital and functional outcome, including persistent vegetative state.

Preserved brain stem reactions somatosensory evoked potentials and EEG reactivity as favourable predictors for awakening beyond vegetative state in post anoxic status provided that the seizure was treated vigorously.

Continuous generalized periodic pattern PP is a common EEG endpoint of advanced coma, particularly after cerebral hypoxia. [26]

Eclampsia

Eclampsia is defined as the occurrence of seizures during gestational hypertension > 20 weeks with proteinuria (300mg/24h). Seizures may occur postpartum upto the sixth week. Seizures are usually preceeded by visual disturbances headache or epigastralgia.

Seizures are attributed to brain edema secondary to brain vasodilatation after autoregulation loss provoked by rapid increase in arterial hypertension. MRI typically shows vasogenic edema in parieto occipital areas. [27] It has been proposed recently that uteroplacental ischemia causes the release of neurokinin B, inflammatory cytokines, endothelins and tissue plasminogen activator that stimulate excitatory neuronal receptors independently of vascular effects. [28] Magnesium sulfate is effective both in preventing and treating eclampsia.

It acts through decreasing peripheral vascular resistance limits vasogenic edema and centrally inhibits NMDA receptors providing anticonvulsant activity by increasing the seizure threshold.

Eclampsia may be a risk factor for TLE-HS. The possible mechanism involved is vasoconstriction of posterior circulation with subsequent hippocampal ischemia after acute hypertension.

Reversible Posterior Leukoencephalopathy

Reversible Posterior leukoencephalopathy is characterized by imaging findings of subcortical vasogenic edema with preferential parietooccipital involvement and subsequent resolution. Occipital seizures are the predominant seizure type.[29] Rapidly increasing arterial tension beyond certain levels. (160/100 mm of Hg in normotensives or 220/110 mm of Hg in hypertension leads to loss of autoregulation with subsequent vasodilatation and edema. Preferential involvement of posterior circulation is due to scarce sympathetic innervation. Here acute symptomatic seizures do not progress to epilepsy. [30]

Limbic Encephalitis

Clinical features of LE include a recent onset of seizures, anterograde amnesia and affective disturbances. In addition one of additional features; neoplasm, LE associated autoantibodies, Temporomedial hyperintensity on MRI or a chronic lymphocytic microglial LE demonstrated on

histopathology. Seizures occur in 90% patients with LE with antibodies to cell membrane antigens either voltage gated potassium channel or other cell membrane antigens and in 50% of patients with paraneoplastic LE. Limbic encephalitis is the cause of TLE with HS of adult onset. HS after LE is more often bilateral than when related to other causes. [31]

History of the event

A description of the circumstances surrounding a paraoxysmal event can provide important diagnostic clues. Seizure semiology should be evolved thoroughly to identify the type of seizure. A witnessed few minutes episode that involved loss of consiousness, stiffening and jerking of the extremities followed by muscle soreness, head ache and the need to sleep for several hours afterwards strongly suggests a tonic clonic seizure.

Past Medical history

A review of the events leading up to the seizure may reveal factors that suggest it was provoked. Causes of provoked seizures include alcohol withdrawal, substance abuse, hypoxia, fever, electrolyte imbalance, hypoglycemia and sleep deprivation.

Drug history

Administration of the Drugs or withdrawal of the drugs may precipate seizures.

Theophylline, isoniazid, antipsychotic drugs, alkylating agents, B lactam antibiotics and quinolones are among the commonly implicated mediction in seizure.

Alcohol withdrawal and cocaine use provokes the seizures.

Physical examination

A thorough physical examination can help uncover possible causes of a seizure. Findings may include evidence of trauma, infection, malignancy, congenital anomalies and prior neurologic events ((eg) focal weakness, spasticity suggesting previous stroke).

During an emergency department evaluation of a patient immediately after a seizure, vital signs should be measured and a general medical examination performed. Guidelines for physical examination are as follows.

- Examine the patient for injuries from the seizures or fall.
- Check oxygen saturation and auscultate the chest for possible aspiration.

- Measure heart rhythm and rate, blood pressure and orthostatic changes for assessment of syncope.
- Auscultate for carotid murmurs or carotid bruits and sources of embolic stroke.

An electrocardiogram should be obtained to identify cardiac arrythmias, detect possible ischaemia and prolonged QT syndrome.

Neurologic examination

The purpose of the neurologic examination is to identify focal or diffuse cerebral dysfunction. This information is particularly helpful in localization related epilepsy. The presence of various features offers clues to the focus of a seizure. For example aphasia suggests a left frontal, temporal or parietal onset. Right or left hemiparesis suggests foci from the contralateral motor cortex.

In initial evaluation of a seizure patients should be observed for fluency of language, facial asymmetry, gaze preferences and pupillary asymmetry. The last presents in patients who have herniation from brain swelling caused by parenchymal or epidural bleeding and in those who have a rapidly growing brain tumor. An extensor plantar response may be noted for some time after a seizure and is not necessarily a pathologic finding,

Diagnostic Testing

Laboratory workup is an essential part of evaluation of seizure. Measurement of glucose, calcium, magnesium, thyroid hormone, and liver enzyme levels, as well as toxicology screening (including blood alcohol levels), may reveal common medical causes of seizures. A complete blood cell count may suggest infection, anemia or sickle cell disease.

In patients suspected to have had an infection or a fever or to have exhibited abnormal behavior just before the event, lumbar puncture should be performed after assessment of the possible risks of the procedure (eg, coagulopathy, mass lesion). Patients who are immunocompromised because of corticosteroid use, recent transplantation or HIV infection should undergo cerebrospinal fluid evaluation to detect possible fungal, bacterial or viral infection. In patients with a systemic malignant condition, cytologic evaluation of cerebrospibnal fluid can identify meningeal carcinoma.

Electroencephalogram.

- EEG should be performed within 24 hours of the seizure because it is significantly more sensitive when obtained during that period(King, 1998).If the routine EEG findings are normal , a sleep - deprived EEG should be performed.

- Standard EEG detects epileptiform discharges in 29 % of patients. Standard EEG combined with sleep – deprived EEG shows epileptiform discharges in 48% of patients (Van Donselarr, 1992).
- Schreiner and Pohlman – Eden studied the value of an EEG taken within 48 hours of the first seizure in an adult. They found that 38.0 % of patients without seizure recurrence and normal EEGs, while only 10.2% of patients with seizure recurrence had normal EEGs. Focal epiletiform activities were found significantly more frequently (26.5 % vs. 13.0%) in patients with seizure recurrence than in patients without seizure recurrence. [43]

Limitation of EEG:

An estimated 0.4 % of adults and 2.8 % of children who have never had a seizure may have inter ictal epileptiform discharges. Furthermore, a normal EEG does not refute the diagnosis of epilepsy. The initial EEG reveals epilepitiform activity in only 40 % of the patients with probable epilepsy.

Imaging Studies.

The role of imaging studies depends on the stage of evaluation. Immediately after a seizure, computed tomography can detect the presence of bleeding or gross structural lesions. However, magnetic resonance imaging is the study of choice because it is more sensitive and specific for evaluating structural lesions and brain parenchyma. Particular attention should be directed to the hippocampus for evaluation of lesions (eg, mesial temporal sclerosis) and to the cortical architecture for detection of abnormalities (eg,dysplasia).

Drug Treatment

If drug treatment is considered after first seizure the chosen antiepileptic drug should have high efficacy, good tolerability and low interaction potential and allow a good quality of life especially since half of all patients would never have another seizure without treatment. The starting dose should be in the lower range.

Drugs Recommended[45]

	GTCS	**Partial**	**Myoclonic**
First Line	Valproic acid Lamotrigine Topiramate	Carbamazepine Phenytoin	Valproic acid Lamotrigine Topiramate
Alternatives	Phenytoin Carbamazepine Oxcarbazepine Phenobarbital Primidone	Levetiracetam Topiramate Tiagabine Gabapentin Felbamate	Clonazepam Felbamate

In acute symptomatic seizure, it is crucial to rapidly identify all insults possibly involved , treat underlying diseases reverse correctable factors and in the case of CNS involvement use AEDS during the acute period. [47] Some refractory epilepsies in adults, mostly epilepsy due to HS, may be preceded

by acute symptomatic seizures related to severe insults including CNS infection, LE, head trauma and eclampsia.

Summary

The occurrence of a first seizure in an adult requires a detailed evaluation aimed mainly at identifying provoking factors that may be involved and to evaluate the risk of developing epilepsy. The patient's history as well as physical and neurological examination may disclose a probable cause of seizure. Routine testing for glucose, Serum electrolytes calcium and full blood count has been recommended .Toxicology screening may be necessary in some specific clinical circumstances. Neuro imaging and routine EEG should be considered as part of the Neurodiagnostic evaluation. A lumbar puncture is essential if the clinical presentation is suggestive of an acute brain infection.

Efficacy of acute symptomatic seizure treatment is thought to depend on early determination of all reversible insults and their rapid correction. Patients with acute symptomatic seizure do not need to be treated with antiepileptic drugs on a long term basis although such treatment may be warranted on a short term basis until the acute condition is resolved.

3. AIM OF THE STUDY

- To study the etiological profile of acute symptomatic seizures in various age groups.
- To assess the common seizure type in patients with acute symptomatic seizures of varied etiologies.
- To study the Electro Encephalographic and Radiological profile of Acute symptomatic seizures.

4. MATERIALS AND METHODS

The study was done in the setting of the department of Neurology and Neuro surgery, Govt.Rajaji Hospital, Madurai. The study had collaborations with the Department of Medicine, Trauma, Toxicology, Obstetrics and also with the departments of Biochemistry, Pathology, Radiology and Microbiology.

The study was designed to analyse all adult patients who are more than 12 years of age in whom the specific cause for seizure could be identified. The sample size was 150 and the study period was one year.

Inclusion criteria

All adult patients who presented with acute seizures in whom the specific cause for seizure could be identified.

Exclusion Criteria

Age less than 13 years

Known epileptic patients

Patients with family history of seizure

I have obtained written consent from the patient or relative after explaining about the study in detail. I got the permission from ethical committee for this study from Madurai Medical college, Madurai. The patients admitted in Neurology, Neurosurgery, Trauma, Toxicology, Obstetric and Medical wards in the Teritiary care Hospital, Govt Rajaji Hospital, Madurai during a one year period were taken up for study.

The clinical details were obtained from the patient or relative with the help of the prepared proforma.

In depth probes in the history for provocation factors and features suggesting organicity were attempted. Significant past medical history if any were noted.

A thorough clinical examination was performed at the time of admission and relevant findings recorded. A routine metabolic screening which included blood sugar, Urea, serum creatinine, electrolytes and Liver function tests were done at the time of admission.

Lumbar puncture and CSF analysis was done if infective etiologies were suspected.

Earliest possible EEG was attempted and was performed using 32 channel digital.

EEG recorder.

CT Brain plain study in all patients and contrast studies when necessary were done in all patients in the study group.

MRI Brain with MRA / MRV done when indicated.

Limitation were encountered inaffordability of patients for MRI scanning.

Early EEG could not be performed due to delay in referral of the patients to this institution and because of the time taken for stabilizing the patient.

Calculation of sample size and Analysis of statistics.

Around 150 patients who presented with Acute seizures in whom the specific cause identified were followed up over a one year period, who attended the epilepsy outpatient clinic. P.value of <0.05 was considered significant statistical value to correct for the multiple comparisons.

SPSS 13.0 is used for statistical analysis.

5. OBSERVATIONS

Seizures in 150 adult patients aged more than 12 years were studied; of which 86 are males and 64 are females.

Table 1 Sex distribution of patients

Sex	Number	Percentage
Male	86	57.3 %
Female	64	42.7%

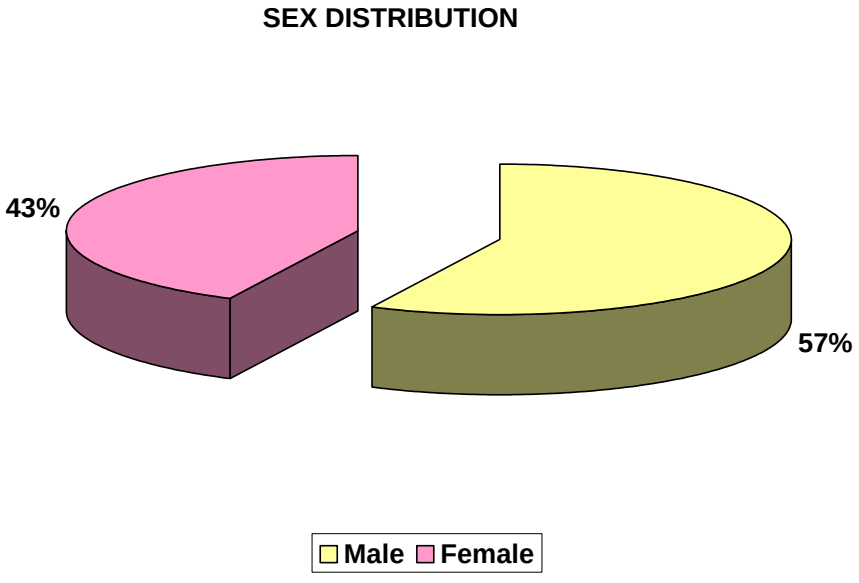
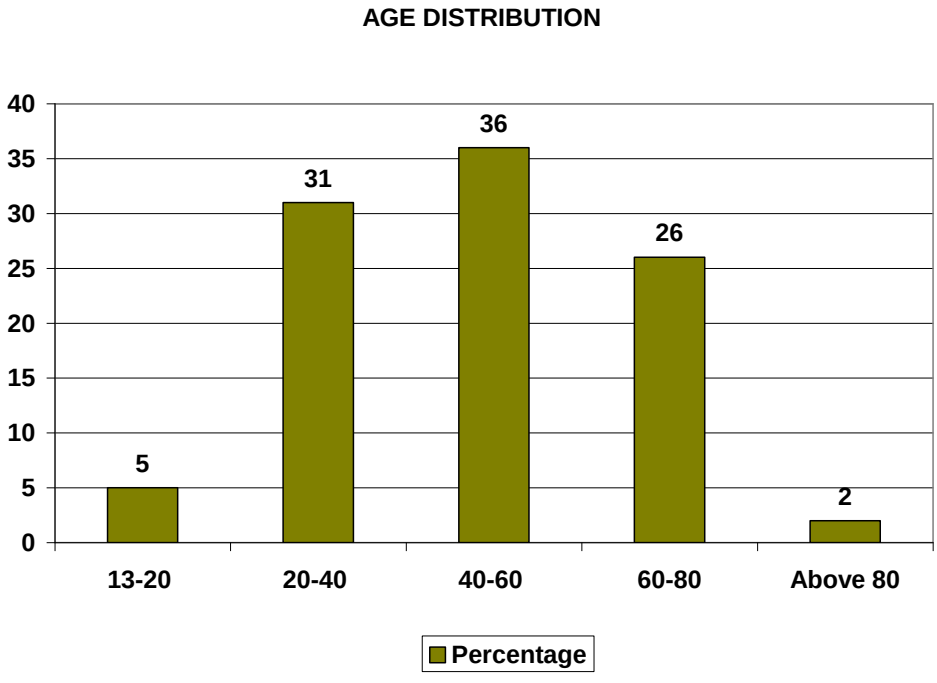


Table 2 shows the distribution of various age group

Age Group	No	Percentage
13 – 20	8	5%
20 – 40	47	31%
40 – 60	54	36%
60 – 80	37	26%
Above 80	4	2%



The seizures are grouped into various types during the presentation and analysed.

Table 3 Seizure types found in the study

Seizure Type	No of patients	Percentage
GTCS	97	65 %
Partial	34	23 %
Status epilepticus	11	7 %
Epileptia Partialis continua	5	3%
Myoclonic seizure	3	2%

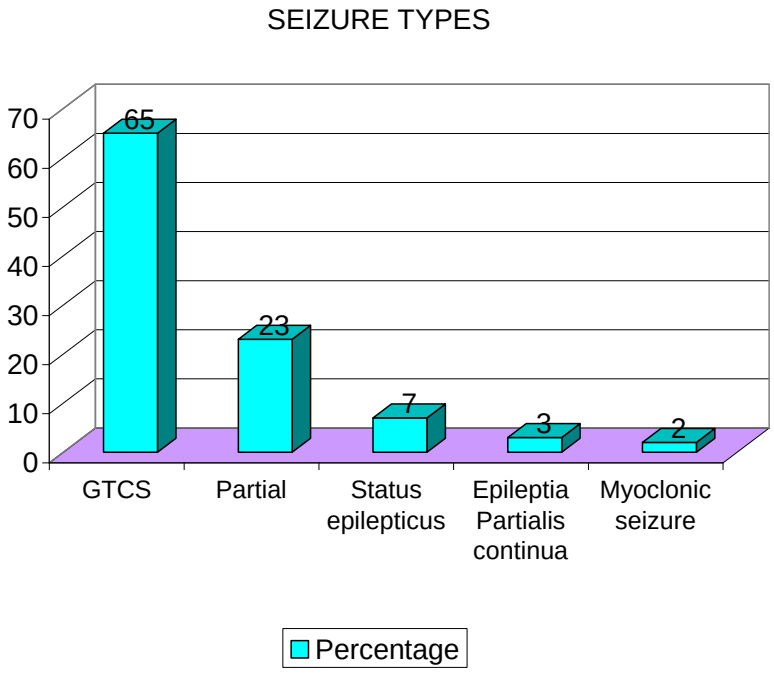


Table 4 Co existent non convulsive symptoms

Symptoms	Frequency
Head ache	68%
Altered sensorium	43%
Vomiting	37%
Fever	25%
Visual disturbance	21%
Limb weakness	18%

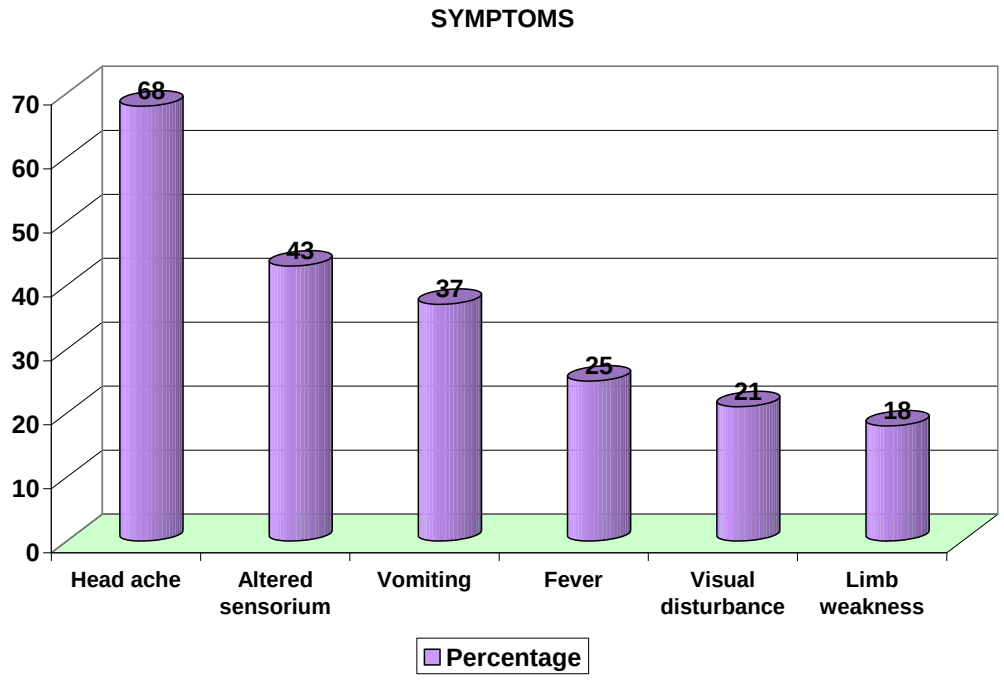
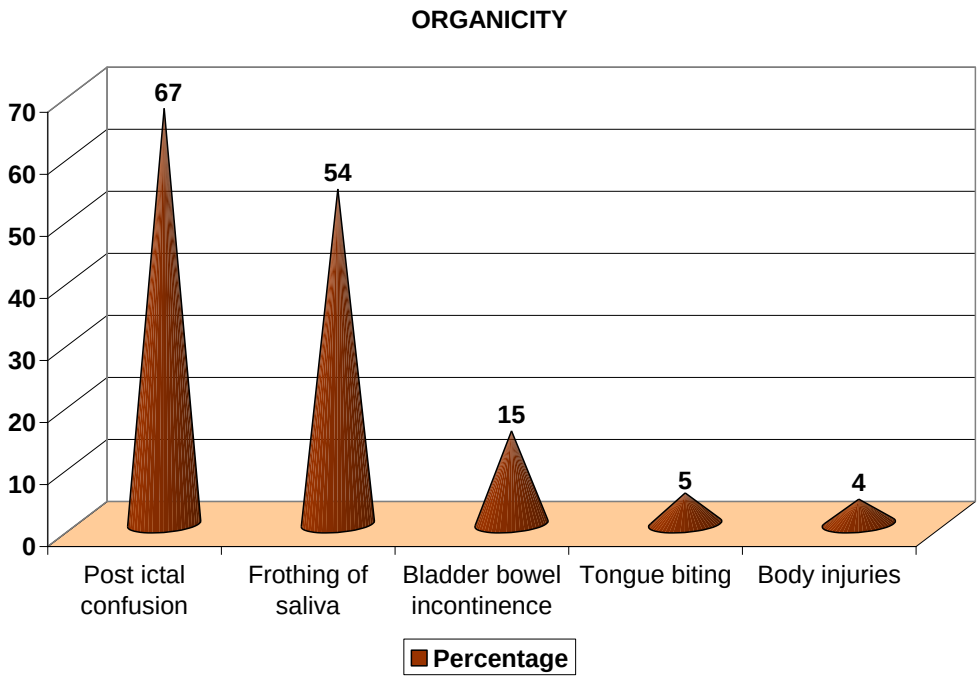


Table 5 History suggestive of organicity

Organicity	Percentage
Post ictal confusion	67%
Frothing of saliva	54%
Bladder bowel incontinence	15%
Tongue biting	5%
Body injuries	4%



Associated systemic illness was notified and significant past medical history was enumerated.

Table 6 Profile of significant past medical history

Past medical history	No of patients
Diabetes	7
Hypertension	12
Both diabetes & Hypertension	4
Renal Failure	6
Liver disease	1
Pulmonary Tuberculosis	4
Heart disease	3

Metabolic abnormalities at the time of admission were investigated as they are among the most readily treatable causes of seizures. The abnormalities in metabolic parameters were noted in 13% of patients in this study.

Table 7 Metabolic abnormality

Metabolic abnormality	No of patients
Hyperglycemia	8
Hypoglycemia	7
Hyponatremia	3
Hypocalcemia	2

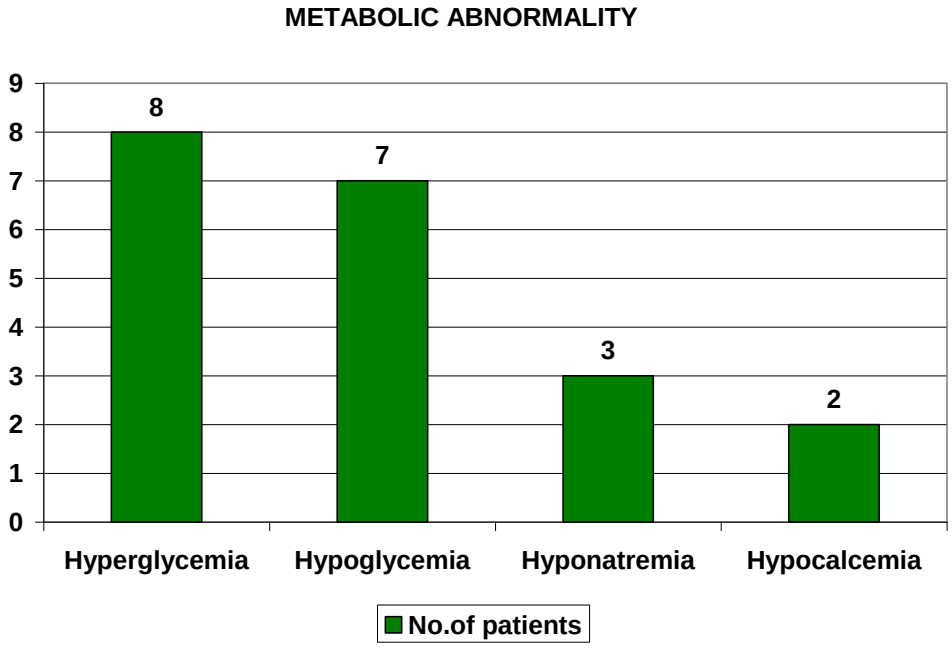


Table 8 Types of Cerebrovascular Disease

Cerebrovascular Disease	No of patients
Cortical Venous Thrombosis	**12**
Intracerebral Haemorrhage	14
Subarachnoid haemorrhage	6
Ischaemic stroke	4

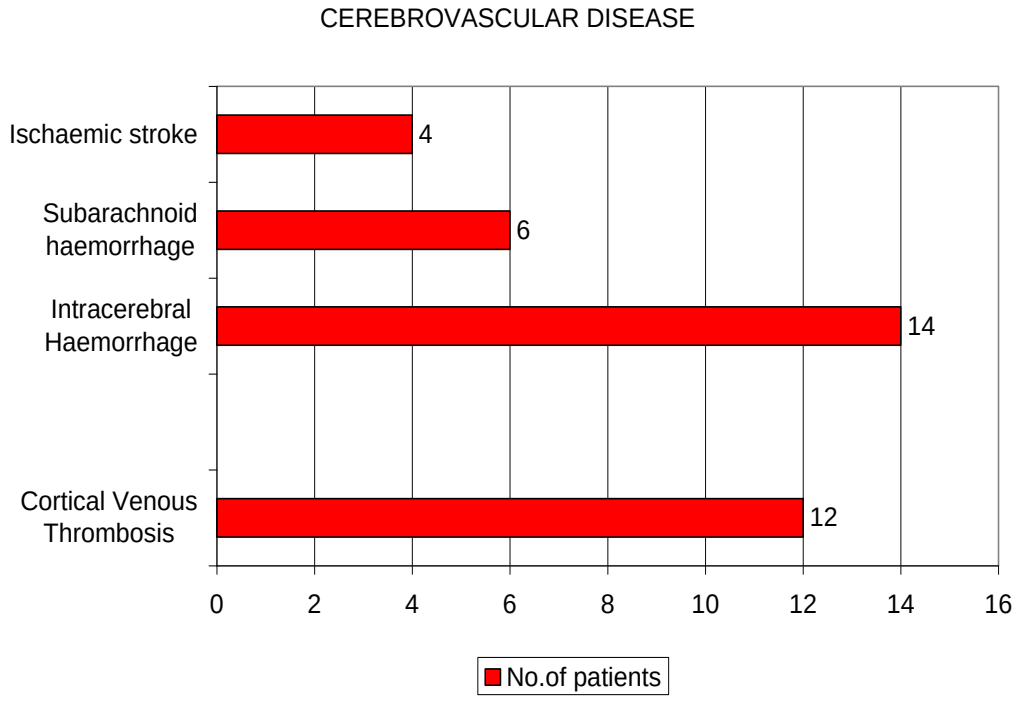
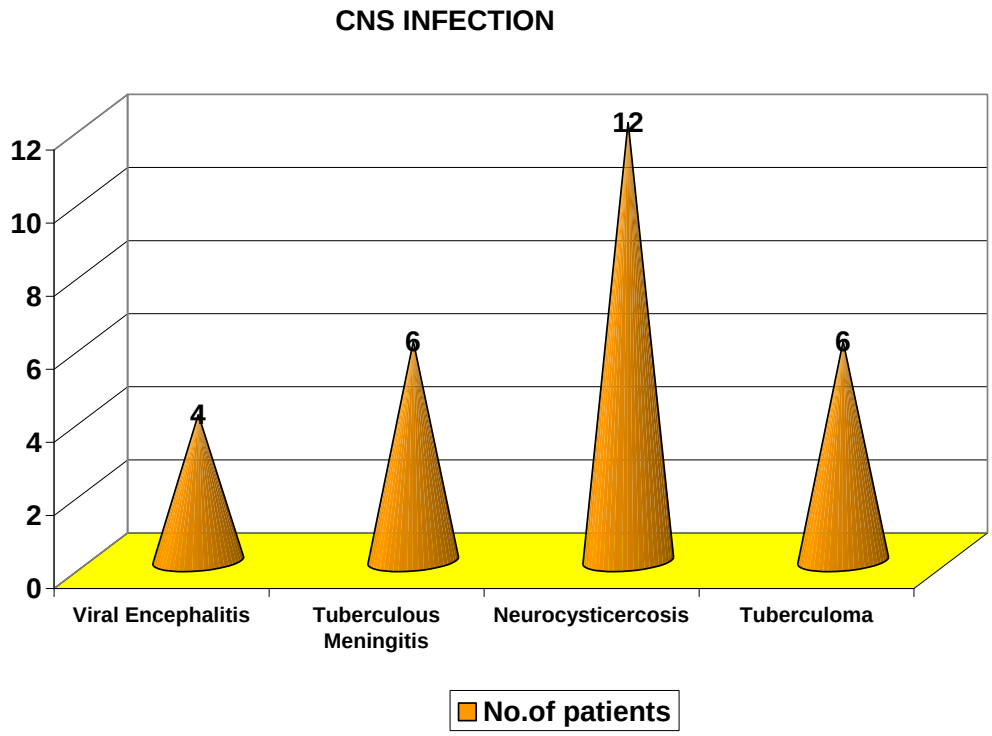


Table 9 CNS Infections noted in the study

CNS Infection	No of patients
Viral Encephalitis	4
Tuberculous Meningitis	6
Neurocysticercosis	12
Tuberculoma	6



In obstetric ward, eclampsia patients having convulsions in both Antenatal and Posterior natal were studied

Table 10 Eclampsia notified in this study

Eclampsia	No of patients
AP Eclampsia	12
PP Eclampsia	7
PRES	5

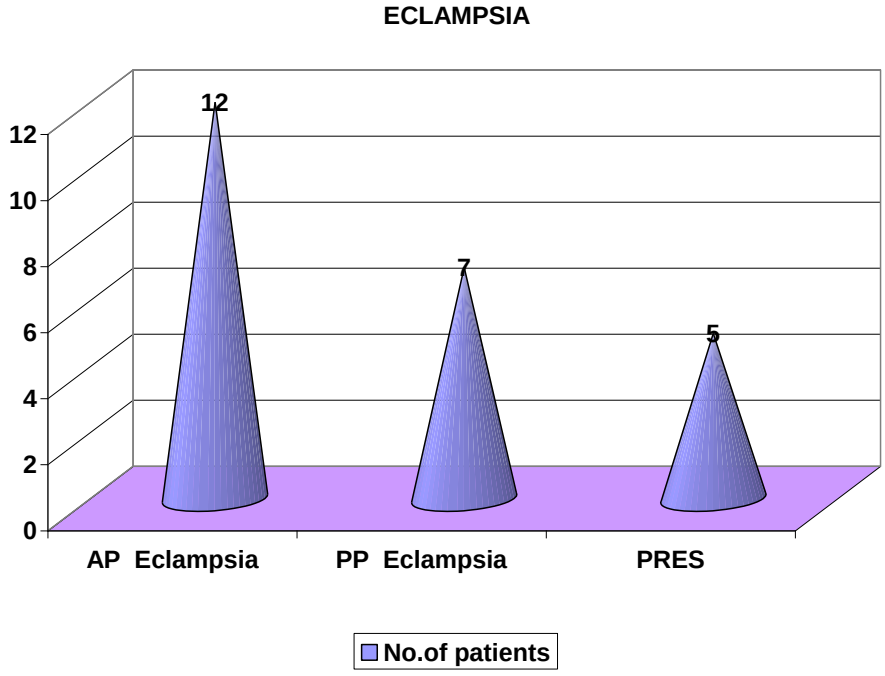


Table 11 Anoxic patients studied

Hanging	6
Post cardiac arrest	2

Head injury patients those developed Acute symptomatic seizure were studied.

Table 12 Traumatic Brain Injury

Type of Head Injury	**No of patients**
Haemorrhagic Contussion	5
Diffuse Axonal Injury	2
Traumatic SAH	3
Post Traumatic Meningitis	2

Table 13 Various Tumours noted in the study

Tumour	No of patients
Astrocytoma	1
Meningioma	2
Glioblastoma multiforme	1

Lumbar Puncture and CSF analysis done in 31 patients suspected of meningitis or encephalitis revealed abnormality in 28 cases.

EEG was taken after stabilizing the patient and all were taken in the interictal period. EEG was done in 114 of the 150 patients in this study. Abnormalities were found in 92 patients.

The most common observed pattern in EEG was sharp & spike waves, polyspike pattern during the interictal period.

Table 14 EEG in this study

Total patients	Number of patients EEG was done	Number of abnormal record
150	114	92

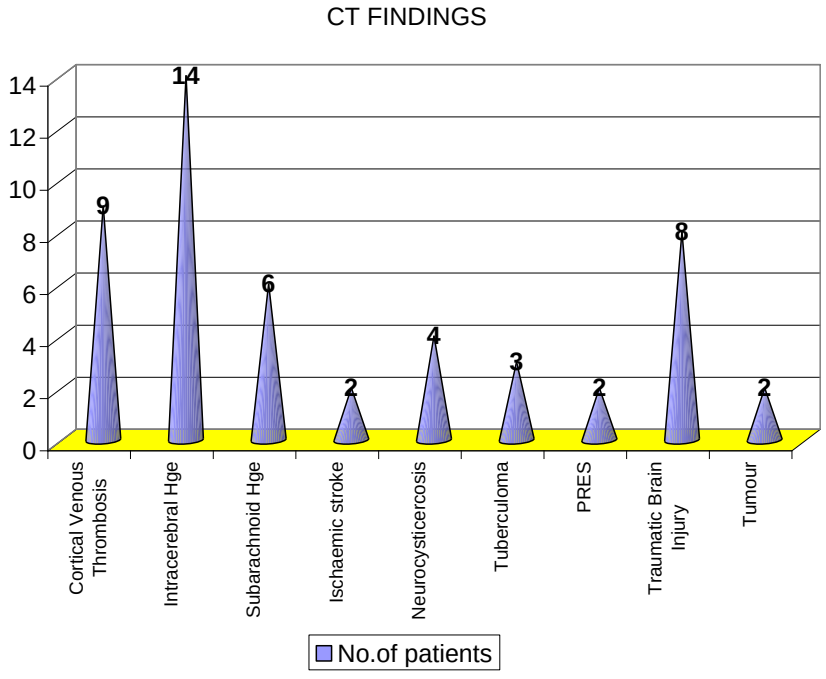
Table 15 Type of abnormality

EEG Finding	Generalised	Focal
Sharp & spike waves	36	17
Slow waves	18	21

CT Brain was done in all the patients in the study group. Radiologist opinion was obtained for all images.

Table 16 CT Findings

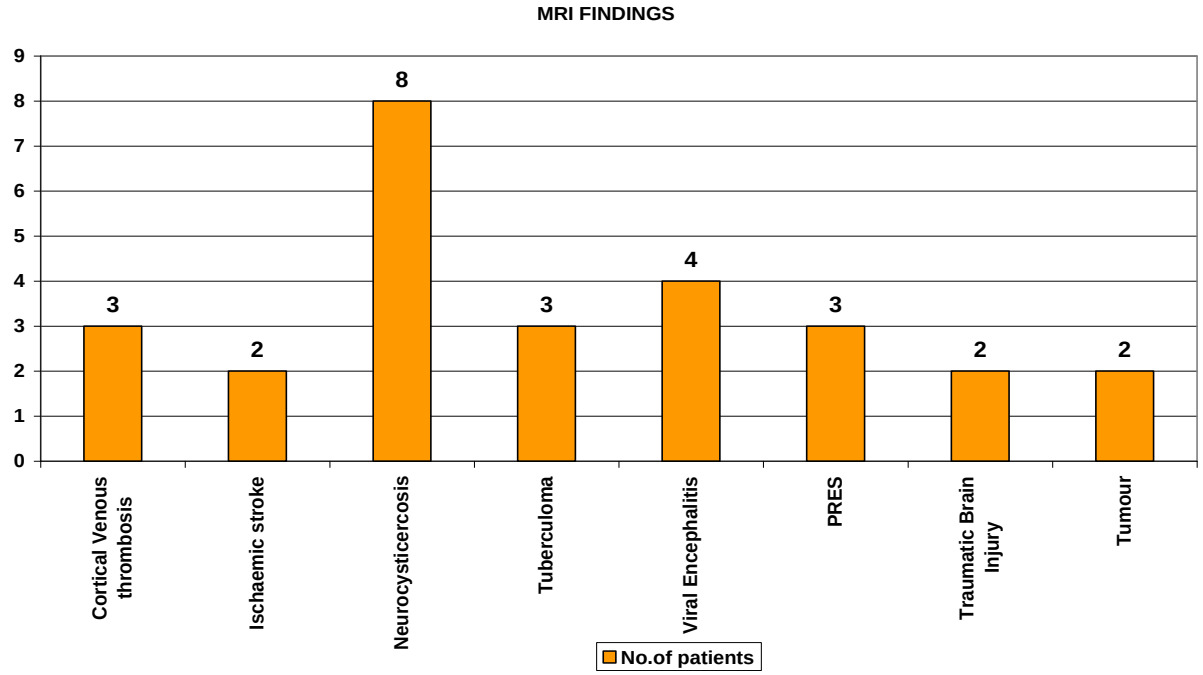
CT findings	Number
Cortical Venous Thrombosis	9
Intracerebral Hge	14
Subarachnoid Hge	6
Ischaemic stroke	2
Neurocysticercosis	4
Tuberculoma	3
PRES	2
Traumatic Brain Injury	8
Tumour	2



MRI brain was done in patients for more evaluation. MRI was helpful in uncovering lesions missed in CT.

Table 17 MRI findings

MRI findings	No
Cortical Venous thrombosis	3
Ischaemic stroke	2
Neurocysticercosis	8
Tuberculoma	3
Viral Encephalitis	4
PRES	3
Traumatic Brain Injury	2
Tumour	2



For analyzing the type of seizures in various etiology in this study, the type of seizure and the percentage was enumerated and tabulated.

Table 18 Types of seizures in various etiology

Etiology	GTCS		PARTIAL		EPC		Status Epilepticus		Myoclonus	
	No	%	No	%	No	%	No	%	No	%
CerebroVascular Disease	19	53%	14	39%	-	-	3	8%	-	-
CNS Infection	10	35%	15	54%	-	-	3	11%	-	-
Eclampsia	21	88%	1	4%	-	-	2	8%	-	-
Alcohol	18	100%	-	-	-	-	-	-	-	-
Metabolic abnormality	14	70%	-	-	4	20%	1	5%	1	5%
Anoxic	5	63%	-	-	-	-	1	12%	2	25%
Traumatic	9	76%	1	8%	1	8%	1	8%	-	-
Tumour	1	25%	3	75%	-	-	-	-	-	-

For analyzing the age group with the etiology, the age group divided into three category and observations were tabulated.

Table 19 Etiology in various age group

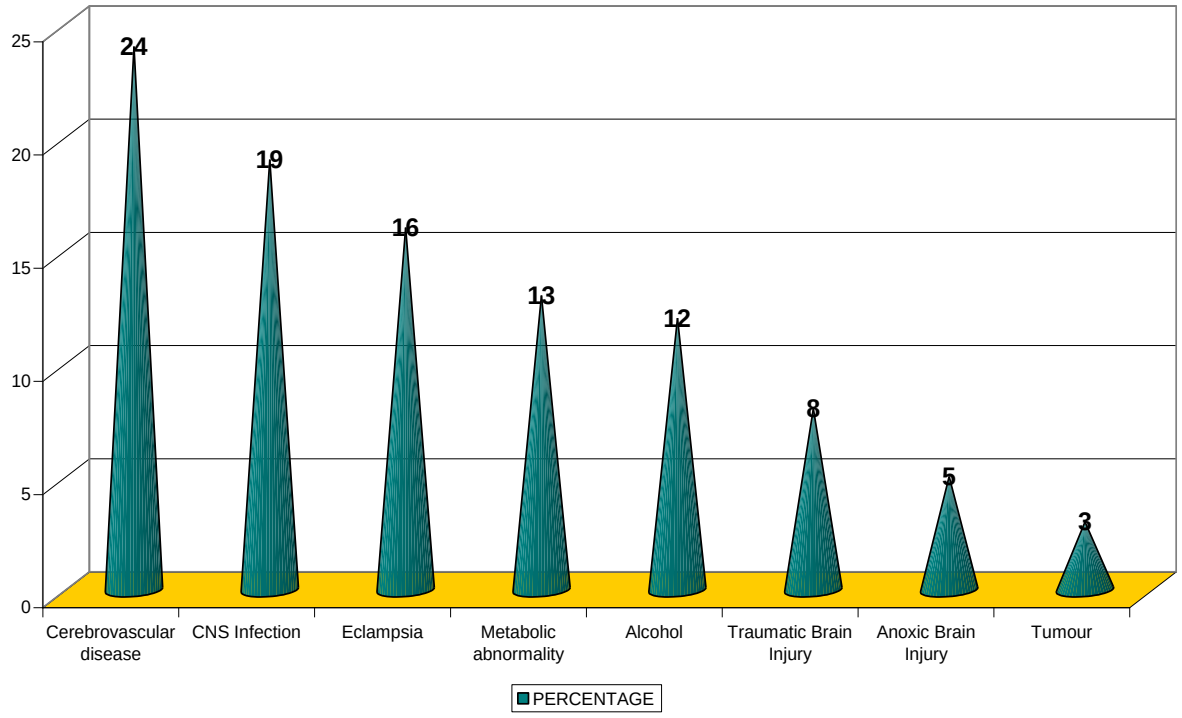
Etiology	< 20 (8)		20 - 60 (101)		> 60 (41)	
	No	%	No	%	No	%
Cerebrovascular Disease	-	-	20	19.8%	16	39%
CNS Infection	5	63%	19	18.8%	4	9.7%
Eclampsia	-	-	24	23.7%	-	-
Alcohol	-	-	18	17.8%	-	-
Metabolic abnormality	-	-	7	6.9%	13	31.7%
Anoxic	1	12%	7	6.9%	-	-
Traumatic	2	25%	6	5.9%	4	9.7%
Tumour	-	-	-	-	4	9.7%

Among 150 patients the various etiologies and their percentage were tabulated in the order of frequency.

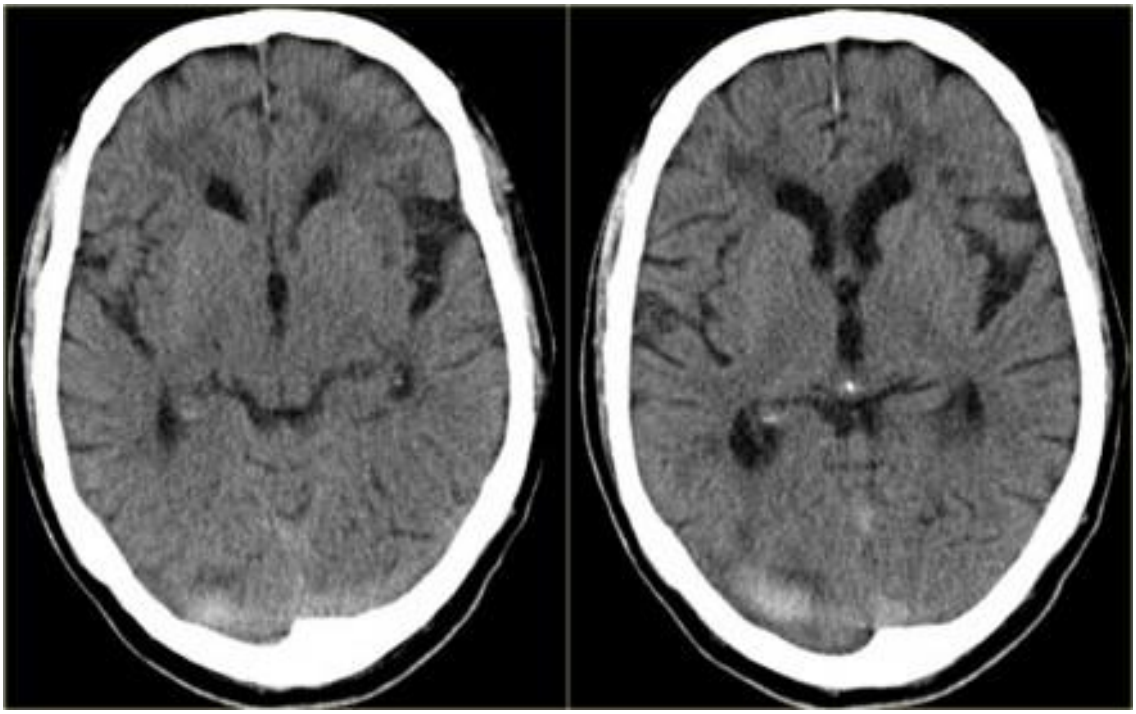
Table 20 Etiology and its percentage

Etiology	No	%
Cerebrovascular disease	36	24%
CNS Infection	28	19%
Eclampsia	24	16%
Metabolic abnormality	20	13%
Alcohol	18	12%
Traumatic Brain Injury	12	8%
Anoxic Brain Injury	8	5%
Tumour	4	3%

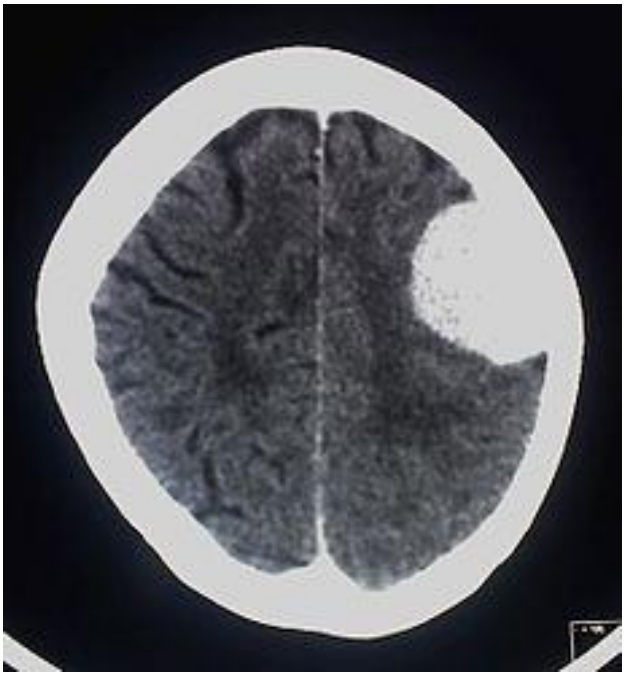
ETIOLOGY AND ITS PERCENTAGE



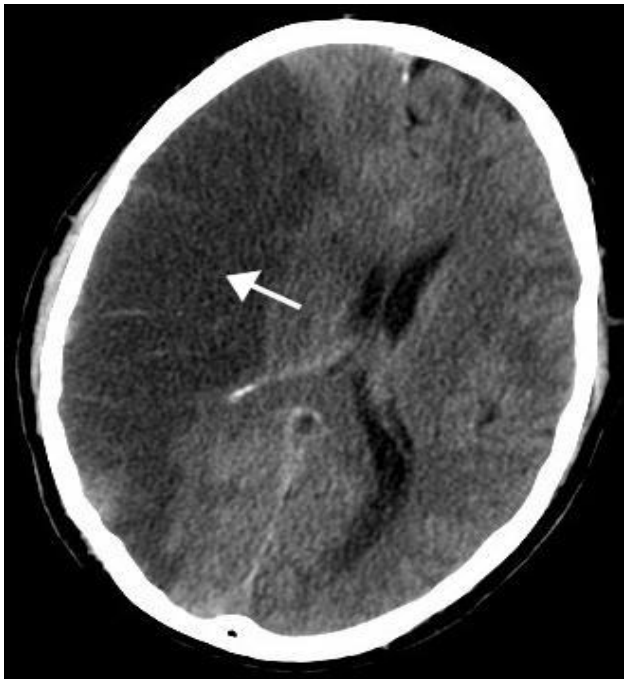
CORTICAL VENOUS THROMBOSIS



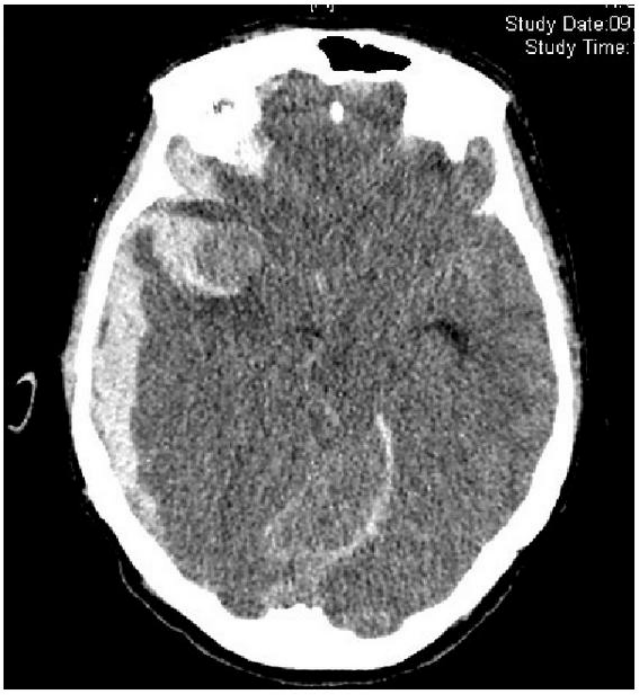
MENINGIOMA



RIGHT MCA INFARCT



SUBDURAL HEMATOMA



RESULTS

- In the present study Acute symptomatic seizures were slightly more in males than in females.
- Acute symptomatic seizures were most common in 40 – 60 years of age group.
- Generalised seizures were the most common seizure type encountered in the study.
- Head ache, vomiting and altered sensorium were the most common non convulsive presenting symptoms.
- Diabetes and Hypertension were the co morbid systemic illness associated with cerebrovascular accidents.
- Metabolic abnormality was found as the cause in 13% of the patients and the predominant age group was above 60 years.
- Cerebrovascular diseases were the most frequent etiology in acute symptomatic seizures.
- Among the cerebrovascular diseases, cortical venous thrombosis and intra cerebral haemorrhage were commonly presented with seizure.

- Next common cause of acute symptomatic seizures in this study was CNS infections consisting of 19% of the patients.
- Among CNS infection, Neurocysticercosis was the most common cause.
- In females eclampsia and cortical venous thrombosis were the common etiology for acute symptomatic seizures.
- Alcohol related seizures contributed to etiology in 12% of the patients.
- Hanging and post cardiac arrest were the common etiology the anoxic brain injury.
- EEG which was done in 76% of the patients in this study and abnormalities recorded in 81%.
- Vascular lesions and space occupying lesions were evaluated by CT brain and MRI brain.
- In 27 patients MRI uncovered the lesion which was missed by CT.
- Generalised seizures were common with cerebrovascular disease and alcohol related seizures. Partial seizures were common with Tuberculoma and Neurocysticercosis.
- Myoclonic seizures were common with anoxic brain injury.

- Cerebrovascular disease and Metabolic abnormality were common above 60 years where as eclampsia and alcohol related seizures were common in 20 – 60 years of age.
- Acute CNS infection was the predominant cause of seizures below 20 years of age.

DISCUSSION

In this study 150 patients of Acute symptomatic seizures were evaluated clinically and with EEG and CT scan.

The study group comprised of 86 males and 64 females. Most authors report a small to moderate preponderance of men in their studies of Acute symptomatic seizures in adults (Van Donselaar, 1992; Musicco, 1997; Hopkins, 1988; King 1998).A male to female ratio of 1.3 : 1 is observed in this study, a trend noted in other studies.

In this study there was an over all male preponderance. Male constituted 57.3% and females 42.7%.

A study made by Bryniarska D etal shared a similar male preponderance 58% were male and 42% were female.

In Delengre T et al study it was demonstrated that alcoholism in young adults and vascular pathology in the elderly play an important role in triggering the seizures which accounts for the male predominance in acute symptomatic seizures in adult.

The common age group in this study was 40 – 60 years.

In Bryniarska D et al study the average age of the patient getting symptomatic seizure was 57.5.

In Narayanan JT et al study, for the new onset acute symptomatic seizures the mean age was 49.07 + 20.20 years.

In Neundorfer B et al study seizures was more prevalent in males and the manifestations occurred mainly between 30 & 40 years of age.

In Pradeep PV et al study the mean age of the seizures was 41 years.

In this study also the common age group was found to be 40 – 60 years even with varied etiology.

In some studies it was shown the seizure incidence going on increasing with age. But in this present study it was observed that next common age group was 20 – 40. As alcohol abuse, eclampsia and cortical venous thrombosis which were occurred in earlier age group covered the significant number of the study group. 20 – 40 was the second common age group.

The seizure type classified in this study revealed generalized seizures in 65% and partial seizures in 23%.

Neundorfer B studied the seizures in adults and noted most seizures were primarily of the generalized type 68% and the partial seizures were 24%.

Z hu PG studied new onset seizures in the ages between 20 and 80 revealed generalized seizures in 64% and partial in 30%.

Retrospective study of Perez et al in 250 patients with symptomatic seizures revealed 59% generalized and 41% partial in nature.

The Observation of seizures types in this study is almost similar to the above mentioned studies.

Comparison of seizure Type encountered in this study with various studies

Seizure Type	This study	Neundorfer	Z hu PG	Perez etal
Generalised	65%	68%	64%	59%
Partial	23%	24%	30%	41%

In this study Status epilepticus was noted in 11 patients. 30% of them are due to cerebrovascular disease. In the study of Sung CY et al, cerebrovascular disease was the leading cause of Status, contributing 35%.

Head ache, vomiting and altered sensorium were the most common non convulsive symptoms observed in this study. Head ache presented in 68% and next to it altered sensorium was presented in 43%.

In Kim et al study it was observed that in Acute CNS infection the common presentation was impairment of consciousness with GCS < 12.

In Bruns and Hauser study, in Traumatic Brain Injury altered sensorium and amnesia more than 30 minutes were common with acute intra cerebral hematomas or subdural haemorrhages.

Vomiting presented in 37% and the fever presented in 25% in this study.

Previous history of Diabetes was present in 14 patients and Hypertension present in 24 patients. 11 patients of Renal failure developed acute symptomatic seizure both due to cerebrovascular disease and metabolic abnormality.

Systemic illnesses were found to be the triggering factors for the development of cerebrovascular accidents.

Alcohol related seizures observed in this study was 12%. The seizures were precipitated both by withdrawal and intoxication.

In Bryniarska D et al study about the Etiological spectrum of symptomatic epilepsy in adults, 205 cases were included in the study.

Vascular lesions of the brain were the most frequent cause of symptomatic seizure 46.4% and the alcohol abuse 13.6%.

In this study also the cerebrovascular diseases were the common etiology and the alcohol abuse comprised 12%.

Next to cerebrovascular diseases the common etiology for Acute symptomatic seizures was acute CNS infection in this study. 19% of the patients presented with CNS infection. Out of 28 cases of infective etiology 12 were Neurocysticerosis and presented with focal seizures.

In the Manipal Study neurocysticercosis accounted for 17.5% of the symptomatic seizures and in the Brazilian study by Valenca MM etal in the group between 15 and 45 years, the major cause of seizure was neurocysticercosis.

In females eclampsia was the predominant cause of symptomatic seizure occurred in 16% of the patients and it was the third common etiology next to CNS infections.

Posterior reversible encephalopathy syndrome were found in 5 patients of pregnancy induced hypertension and they were revealed by imaging.

Metabolic abnormalities contributed to etiology in 13% of patients and most of them were readily treatable. The common abnormalities were Hyperglycemia and Hypoglycemia. The hyponatremia noted in 3 patients and hypocalcemia in 2 patients.

In Boggs Whang et al study and Gao et al study it was observed that acute symptomatic seizures are encountered in the following metabolic abnormalities in the following metabolic abnormalities. Na < 115 mg/dl , Mg <0.8 mg/dl ,Ca <5mg/dl,Glucose <36 mg/dl and >450 mg/dl.

In this study also patients having keto acidosis Blood sugar >450 exhibited high incidence of seizures and the sodium was found to be below 115 in hyponatremic patients.

EEG was done in 114 (76%) patients in the study. Abnormalities were found in 92 (81%) of the EEGs done. The average period from the onset of seizures to the record of EEG was five days owing to the late referral of patients to this institution and to the time taken to stabilize the patient before shifting to EEG room.

The yield of abnormalities in the EEG in this study could have been better if it were done more early or special methods such as continuous EEGs.

The most common abnormality in EEG was sharp & spike waves and poly spike activity. Anti convulsant drugs show the normal background rhythm in EEG and almost 90% of the patients in the study group were under the anticonvulsant drugs when EEG was performed which explains the slow waves pattern in the EEG.

Focal findings in the EEG originating from the temporal lobes were recorded in two patients which helped in the diagnosis of encephalitis.

CT scan was done in all patients in the study group. When CT was inconclusive in revealing the lesion MRI was done. Imaging abnormalities contributed to the etiologies in 52% of patients.

Among cerebrovascular diseases CT revealed abnormalities in 31 out of 36 and the rest of the 5 cases. 3 cases of Cortical venous thrombosis and 2 cases of Ischaemic stroke were revealed by MRI. Also in Neurocysticercosis inconclusive findings in the CT were revealed by MRI in 8 patients. 4 cases of Encephalitis were found out by MRI and confirmed by CSF analysis.

Etiological profiles revealed CVA, CNS infections, Eclampsia, Alcohol abuse, Metabolic abnormality, Anoxic Brain Injury, Traumatic Brain Injury and Tumour as the causes of Acute symptomatic seizures.

Most of the studies showed the cerebrovascular disease as the common etiology in the elderly and CNS Infections in the young adults.

In Pradeep PV study the results were as like this present study.

Etiology	In This Study	Pradeep PV
Cerebrovascular Disease	24 %	20 %
CNS Infections	19 %	18 %
Traumatic Injury	8 %	6 %
Tumour	3 %	4 %

In Jimenez Jimenez FJ study conducted in an area of rural health care Neurocysticercosis contributed to 6.3 % of the seizure etiology.

Etiology	In This Study	Jimenez FJ
Cerebrovascular Disease	24 %	20 %
Alcohol	12%	10 %
Neurocysticercosis	8 %	6.3 %
Tumour	3 %	6.3 %

The study, Etiological spectrum of symptomatic Seizures in adults conducted in 205 cases over 20 years by Bryniarska enumerated the various etiologies and it was compared with this present study.

Etiology	In this study	Bryniarska
Cerebrovascular Disease	24 %	46.4 %
Alcohol Abuse	12 %	13.6 %
Tumour	3 %	10.2 %
Traumatic Brain Injury	8 %	8.8 %

The etiological profile in this study is almost similar to the above mentioned study except the prevalence of tumour.

In Sung CY etal study and Roberts Ma study the Metabolic abnormality contributed to 10 % and 11 % respectively.

In this study the Metabolic abnormality was the etiology of Acute symptomatic Seizures in 13 % and found commonly in patients more than 60 years of age.

Comparison of Etiologies with other studies.

Etiology	This Study	Sung CY etal	Robert Ma
CVD	24 %	35 %	44%
Metabolic	13 %	10 %	11 %
Brain Tumour	3 %	8 %	12 %

As all adult patient above 12 years were included in this study, the etiology was varied in various age group and varied related to sex.

As like other studies the Cerebrovascular diseases were the leading cause of symptomatic seizures. EEG and Imaging took significant role in identifying the etiological factors.

Cerebrovascular diseases and Metabolic abnormalities were the etiology in older patients, alcohol abuse in middle age and the Acute CNS infections in the young adults and the results of this study was similar to various other studies.

CONCLUSIONS

1. Acute symptomatic seizures were more common in males than females and in 40 – 60 years of age.
2. Cerebrovascular diseases were the most frequent cause of acute symptomatic seizures, followed by Acute CNS infections.
3. Eclampsia and cortical venous thrombosis were the common etiology among females.
4. Cerebrovascular diseases and metabolic abnormality were common above 60 years of age where as eclampsia and alcohol related seizures were common in 20-60 years of age.
5. Acute CNS infections were the predominant cause of acute symptomatic seizures below 20 years of age.
6. Generalised seizures were the most common seizure type encountered in this study.
7. EEG and Radiological abnormalities were seen in nearly 60% of the patients.

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PROFORMA FOR EVALUATION

Name Age Sex

Address

Occupation Contact no

Medical unit prof op/ip no ward DOA DOD

Neuro unit prof EEG no

Referred from

Elaboration of seizure

Witness of seizure Reliability

Before the episode

- Recent illness (headache/fever)
- Unusual stress
- Medications
- Last alcohol intake
- Last meal
- Sleep deprivation
- Activity just before seizure

During the episode

- Time of day
- Aura
- Duration
- Ability to talk & comprehend
- Ability to recall events
- Movements of eyes face arms legs
- Tongue bite frothing

Bowel / bladder incontinence

Bodily injuries sustained

After event

Confusion duration

Focal neurological deficits

Headache

Any other significant symptoms

SIGNIFICANT PAST HISTORY

Diabetic: yes / no duration & treatment

Hypertension CAD CKD

tuberculosis

any others

alcohol intake y / n duration freq quantity

last intake

smoking

Family h/o seizures

<u>Clinical Examination</u>

General exam

Neurocutaneous markers

Vitals: BP Pulse RR Temp

CNS :

at presentation Time after seizure

signs of meningial irritation

higher functions

motor system

sensory system

cranial nerves

cerebellum

CVS : **RS:** **P/A:**

COURSE DURING HOSPITAL STAY

INVESTIGATIONS

Hematology **TC: DC: P L E B HB: ESR:**

Biochemistry sugar urea creatinine Na k Ca

CSF analysis

Others:

ECG:

Cxr :

ECHO:

CT BRAIN:

MRI BRAIN:

EEG:

Treatment

MASTER CHART

S. No	Name	Age	Sex	Symptoms	Seizures	Etiology	EEG	CT	MRI
1.	Thangavel	44	M	H	G	I A	A	N	A
2.	Latha	20	F	H	G	III A	A	N	N
3.	Sulochana	41	F	F	G	II B	A	N	N
4.	Madan	47	M	V	G	IV	A	N	-
5.	Neethiarasu	54	M	V	G	V D	A	N	-
6.	Thuvarammal	81	F	H	P	VIII B	A	A	A
7.	Duraipandi	66	M	H	G	VII B	A	N	A
8.	Navajothi	49	F	A	G	VI A	A	N	-
9.	Rajeswari	22	F	H	G	I A	N	A	A
10.	Gangadevi	24	F	V	G	III B	A	N	-
11.	Muthupandi	32	M	V	G	VII C	A	A	-
12.	Muthumari	58	F	A	G	V C	-	N	-
13.	Sigappi	64	F	L	P	VIII B	-	A	A
14.	Perumayee	46	F	H	G	II C	A	A	A
15.	Deivakani	21	F	H	S	III A	A	N	-
16.	Alagarsamy	68	M	H	G	VIII A	A	N	A
17.	Veeranan	65	M	V	P	I B	A	-	-
18.	Dinesh	42	M	H	G	IV	N	N	-
19.	Vellai	45	M	H	G	VII A	A	A	-
20.	Thenmozhi	22	F	H	G	III B	A	N	N
21.	Mohan	28	M	A	M	VI A	N	N	-
22.	Ramuthai	22	F	H	G	III A	N	N	-
23.	Selvam	36	M	V	P	II C	A	N	A

24.	Petchiammal	20	F	H	G	III A	A	N	N
25.	Unnamalai	66	F	A	G	V C	-	N	-
26.	Ravi	44	M	F	G	II A	A	N	A
27.	Chelladurai	64	M	H	G	VII A	-	A	-
28.	Rengammal	44	F	F	G	II B	A	N	N
29.	Karthiga	20	F	V	G	III C	-	A	A
30.	Sudha	20	F	H	G	III A	N	N	-
31.	Regina beevi	23	F	H	G	I A	N	N	A
32.	Yogesh	15	M	F	S	II A	A	N	A
33.	Saravanan	51	M	V	M	V B	-	N	-
34.	Shivani	21	F	V	G	III C	A	A	A
35.	Dhanasekaran	62	M	A	G	V C	A	N	-
36.	Saradha	21	F	V	G	III C	A	N	A
37.	Poovatha	65	F	V	G	V B	A	N	-
38.	Sangam	45	M	H	G	I A	A	A	A
39.	Aswin	42	M	F	G	II A	-	N	A
40.	Lakshmi	20	F	H	S	III A	A	N	-
41.	Navarathinam	45	F	H	G	II D	-	A	A
42.	Ulagammal	22	F	V	G	III B	A	N	N
43.	Vengaiah	28	M	V	G	IV	A	N	-
44.	Madurai veeran	48	M	A	S	V A	A	N	-
45.	Murugeswari	20	F	V	G	III A	-	N	N
46.	Senthil	65	M	H	P	II B	A	N	A
47.	Subramani	32	M	V	G	IV	-	N	-
48.	Manikandan	49	M	A	G	V C	A	N	-
49.	Karthick	15	M	A	S	VI A	A	N	-
50.	Muthu	42	M	H	G	I A	N	A	A
51.	Chellapandi	28	M	V	P	II C	A	N	A

52.	Jothilakshmi	26	F	V	G	III A	A	N	-
53.	Bharathiraja	46	M	F	G	II B	A	N	N
54.	Manimegalai	61	F	V	P	I B	A	A	-
55.	Ramya	23	F	V	G	III B	A	N	N
56.	Sarkarai	46	M	L	G	I D	A	A	-
57.	Malathi	52	F	V	G	V D	A	N	-
58.	Nithya	20	F	H	G	III A	A	N	N
59.	Ganesan	64	M	F	G	II B	A	N	N
60.	Gangadaran	54	M	A	G	IV	A	N	-
61.	Murugeswari	32	F	A	G	VI A	A	N	-
62.	Muniammal	70	F	H	G	VII D	-	N	N
63.	Karuppayee	65	F	H	S	I B	-	A	-
64.	Velu	38	M	H	P	II C	A	N	A
65.	Kokila	21	F	H	G	III A	-	N	N
66.	Annadurai	43	M	H	P	II C	N	N	A
67.	Jothilakshmi	21	F	H	G	I A	A	A	A
68.	Janakiraman	43	M	A	G	I V	N	N	-
69.	Karuthapandi	26	M	A	M	VI A	A	N	-
70.	Murugan	48	M	V	P	I B	A	A	-
71.	Parameswari	19	F	A	S	II A	-	N	A
72.	Ayeeshabanu	26	F	H	G	III C	A	N	A
73.	Nallakannu	69	M	H	G	VII C	A	A	-
74.	Ochathevar	58	M	V	E	V B	A	N	-
75.	Vallarasu	42	M	A	P	VII A	N	A	-
76.	Masilamani	63	M	A	G	I C	A	A	-
77.	Gowtham	14	M	H	P	II C	-	N	A
78.	Velammal	20	F	H	G	III A	A	N	-
79.	Surulipandi	69	M	A	G	V C	N	N	-

80.	Sonaiammal	62	F	V	G	V B	N	N	-
81.	Sithan	34	M	H	G	IV	A	N	-
82.	Gangammal	65	F	H	P	I B	A	A	-
83.	Anusiya	25	F	H	G	III B	A	N	-
84.	Rajathi	56	F	A	G	VI B	N	N	-
85.	Kannupillai	67	M	A	E	V B	A	N	-
86.	Ramachandran	47	M	H	P	II C	A	N	A
87.	Dhanalakshmi	23	F	H	G	III A	A	N	N
88.	Kalilappan	41	M	A	G	IV	-	N	-
89.	Govindammal	61	F	A	G	V A	N	N	-
90.	Suganya	48	F	V	S	I B	-	A	-
91.	Sithika banu	26	F	V	G	III B	A	N	N
92.	Thangaiah	50	M	H	G	IV	A	N	-
93.	Sangili	36	M	H	G	II B	A	N	A
94.	Virumandi	71	M	V	P	III C	A	N	A
95.	Pandi	44	M	H	P	II D	-	N	A
96.	Prema	23	F	V	P	III C	-	N	A
97.	Seva Thaiya	71	M	V	E	V B	-	N	-
98.	Hariharan	19	M	V	E	VII D	-	N	N
99.	Radhakrishnan	55	M	H	G	I A	A	N	A
00.	Suresh	18	M	H	G	II D	N	N	A
101.	Rajagopal	17	M	A	S	VII A	A	A	-
102.	Sakthivel	58	M	V	P	I B	N	A	-
103.	Malarvizhi	54	F	H	P	I B	A	A	-
104.	Parvathi	20	F	V	G	III B	-	N	-
105.	Mohamed	49	M	V	G	IV	A	N	-
106.	Navaneetha Krishnan	41	M	H	G	VII A	-	A	-

107.	Sekar	45	M	V	P	II C	-	N	A
108.	Subbaiah	66	M	A	G	V A	N	N	-
109.	Manohara Moorthi	44	M	H	G	VII C	A	A	-
110.	Mookkan	44	M	A	G	V	-	N	-
111.	Naveen kumar	41	M	F	S	II B	A	N	N
112.	Subban	51	M	A	G	IV	N	N	-
113.	Ramasamy	65	M	V	P	I B	A	A	A
114.	Marutha nayagam	45	M	H	P	II D	A	A	A
115.	Angayarkanni	28	F	H	P	II C	-	A	A
116.	Muthuselvi	24	F	H	G	I A	A	A	A
117.	Lakshmipriya	28	F	A	G	VI A	-	N	-
118.	Muniyandi	66	M	V	P	I B	-	A	-
119.	Veerasamy	61	M	F	G	II B	N	N	N
120.	Lakshmanan	51	M	A	G	IV	N	N	-
121.	Sankarapandi	48	M	A	G	VI B	-	N	-
122.	Periyasamy	66	M	H	G	I C	N	A	-
123.	Nallu	46	M	A	G	IV	-	N	-
124.	Umadevi	25	F	H	G	I A	A	A	A
125.	Shanmugam	66	M	H	P	II D	N	A	A
126.	Adaikalammal	63	F	V	G	V B	A	N	-
127.	Seethaiammal	64	F	L	P	I D	A	A	-
128.	Rani	32	F	H	P	II C	A	A	A
129.	Varadharajan	57	M	H	G	I C	A	A	-
130	Muthusamy	70	M	V	P	I B	A	A	-
131.	Rakkammal	26	F	V	G	I A	A	A	A
132.	Vanitha	16	F	H	P	II C	A	N	A
133.	Rajalingam	52	M	H	G	IV	-	N	-
134.	Veerakalai	68	M	V	P	I B	A	A	-

135.	Sivakumar	48	M	A	G	IV	A	N	-
136.	Chinnasamy	81	M	L	P	I D	A	N	A
137.	Poovatha	23	F	H	G	I A	-	A	A
138.	Soundara pandian	45	M	H	G	IV	A	N	-
139.	Thangamuthu	63	M	L	P	I D	-	N	A
140.	Venkata chalam	46	M	A	G	IV	A	N	-
141.	Vivekanandan	54	M	V	S	I B	-	A	-
142.	Arumugam	61	M	V	G	I C	A	A	-
143.	Ayyavu	82	M	H	P	I B	-	A	-
144.	Karumpan	66	M	H	G	I C	N	A	-
145.	Panchavarnam	24	F	V	G	I A	A	A	A
146.	Avudaiammal	64	F	A	G	V C	A	N	-
147.	Petchiammal	82	F	A	G	V C	A	N	-
148.	Anusiya	36	F	V	P	II C	A	A	A
149.	Sundara moorthy	63	M	A	E	V B	A	N	-
150.	Parasakthi	68	F	V	G	I C	-	A	-

KEY TO MASTER CHART

Symp	--	Predominant Symptoms
Seiz	--	Seizure Type
Etio	--	Etiology
M	--	Male
F	--	Female
H	--	Headache
V	--	Vomiting
A	--	Altered Sensorium
F	--	Fever
L	--	Limb Weakness
G	--	Generalised
P	--	Partial
S	--	Status Epilepticus
E	--	Epileptia Partialis continua
M	--	Myoclonic Seizure
N	--	Normal
A	--	Abnormal
I A	--	Cortical Venous Thrombosis
I B	--	Intracerebral haemorrhage
I C	--	Subarachnoid haemorrhage
I D	--	Ischemic stroke
II A	--	Viral Encephalitis
II B	--	Tuberculous Meningitis
II C	--	Neurocysticercosis
II D	--	Tuberculoma

III A	--	Antepartum Eclampsia
III B	--	Postpartum Eclampsia
III C	--	PRES
IV	--	Alcohol
V A	--	Hyponatremia
V B	--	Hyperglycemia
V C	--	Hypoglycemia
V D	--	Hypocalcemia
VI A	--	Hanging
VI B	--	Postcardiac Arrest
VII A	--	Haemorrhagic Contusion
VII B	--	Diffuse Axonal Injury
VII C	--	Traumatic SAH
VII D	--	Post traumatic Meningitis
VIII A	--	Astrocytoma
VIII B	--	Meningioma
VIII C	--	Glioblastoma Multiforme

Ref. No. 20735/E4/2/2013

Govt. Rajaji Hospital,
Madurai.20. Dated: 20.12.2013

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for November 2013
Approved list -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 18.11.2013, Monday at 10.00 am to 12.00.noon at the Anaethesia Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr. V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology)	Professor & H.O.D of Surgical Oncology(Retired) D.No.72, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr. I. Jeyaraj, M.S., (Anatomy) Cell.No 9566211947	Director & Professor Institute of Anatomy /V.P Madurai Medical College	Member
4. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056	Director of Pharmacology Madurai Medical College	Member
5. Dr.S. Vadivel Murugan, MD., (Gen.Medicine) Cell.No 9566543048	Professor of Medicine Madurai Medical College	Member
6. Dr.S. Meenakshi Sundaram, MS (Gen.Surgery) Cell.No 9842138031	Professor & H.O.D of Surgery i/c Madurai Medical College	Member
7. Mrs. Mercy Immaculate Rubalatha, M.A., Med., Cell. No. 9367792650	50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20	Member
8. Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
9. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20	Member

The following Project was approved by the committee

..2..

Name of P.G.	Course	Name of the Project	Remarks
Dr. E. Amalraj Iyadurai	PG in DM (Neurology), Madurai Medical College and Government Rajaji Hospital, Madurai.	Etiological & Clinical profile of Acute symptomatic Seizures in Adults in a Tertiary Care Hospital.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman**
Ethical Committee

DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

20.12.13

To
The above Applicants
-thro. Head of the Department concerned

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STUDY ON ETIOLOGICAL AND CLINICAL PROFIILE OF ACUTE SYMPTOMATIC SEIZURI IN ADULTS IN A TERTIARY CARE HOSPITAI

Dissertation submitted
In partial fulfillment of the regulation for
the final examination of

DOCTOR OF MEDICINE
BRANCH - I
NEUROLOGY