"A STUDY OF CLINICOETIOLOGICAL PROFILE OF SEIZURES IN CHILDREN IN KOLAR"

By

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IN

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Under the guidance of

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ABSTRACT

BACKGROUND

Seizures are the most common pediatric neurological disorder, with 4% to 10% of children suffering at least one seizure in the first 16 years of life. The incidence is highest in children younger than 3 years of age, with a decreasing frequency in older children. Epidemiologic studies reveal that approximately 150,000 children will sustain a first-time, unprovoked seizure each year, and of those, 30,000 will develop epilepsy. In our hospital over the past one year among the cases admitted to pediatric wards, about 6.6 % were diagnosed to have new onset seizures and most of them were critically ill requiring ICU care. It is necessary to diagnose and differentiate epilepsy from other common conditions resembling it , as it involves long-term management and carries a lot of social and psychological stigma.

Thus, we intend to study the etiological profile, correlate the clinical presentation, physical findings and investigations of different seizure disorders . This will help to make diagnosis, classify different seizures and to select treatment plan as seizures may signal potentially serious underlying systemic or central nervous system (CNS) disorders that require thorough investigations and management

OBJECTIVE

The objective of present study is to evaluate the clinical profile of seizures in children and to determine the etiological factors of seizure disorders admitted to our hospital

METHOD

The study was conducted at R.L.Jallapa Hospital affiliated to Sri Devraj Urs Medical College, Kolar. All children between 1 month to 18 years admitted with new onset undiagnosed febrile or afebrile convulsive seizure disorder to our hospital from December 2013 to January 2015 were included. Minimum sample size of 150 was estimated after usage of appropriate sample size calculation methods and the samples were analyzed. Detailed history , from first hand witness, along with details of clinical profile ,investigations and treatment was noted in a predesigned proforma which included a consent form. An observational study was done to evaluate clinical profile of seizures and to determine the etiological factors of seizures.

RESULTS

Among 150 patients, mean age of onset of seizures was 5.19 ± 4.37 years. Female to male ratio is 1.05:1.Generalized tonic-clonic seizures were the most common type (56.6%). Febrile seizure(65.9%) was the most common cause of seizure below 5 years of age and Neurocysticercosis (50%) in the age group 6-18 years. Idiopathic generalized epilepsies (45%) were most common type of epilepsy. Simple febrile convulsions (79.3%) were more common than complex febrile convulsions (20.6%). Mean age for febrile seizure was 24.05 months. Mean temperature on admission to hospital in febrile seizure patients was 101.43^{0}F.The most common etiology of febrile seizure was upper respiratory tract infection followed by UTI.

CONCLUSION

Febrile seizures were the most common cause of seizures below 5 years and neurocysticercosis in older children. Neuroimaging should be advised in all afebrile children for diagnosis of neurocysticercosis. Children diagnosed with epilepsy require long term follow up studies including neuropsychologic studies.

ABBREVATIONS

AGE	Acute gastroenteritis
BCG	Bacillus calmette gurien
CAT	Computerized axial tomography
CBC	Complete blood count
CBZ	Carbamazipine
CFC	Complex febrile convulsion
CNS	Central Nervous System
CSF	Cerebrospinal fluid
CT	Computed Tomography
CVS	Cardio Vascular System
CXR	Chest X-ray
ECG	Electrocardiography
EEG	Electroencephalography
ELISA	Enzyme Linked Immuno Sorbent Assay
GABA	Gamma-amino butyric acid
GCAE	Global Campaign Against Epilepsy
GCS	Glasgow Coma Scale
GL	Gastric lavage
GTC	Generalised tonic-clonic
GT	Generalised tonic
ILAE	International League Against Epilepsy
LRE	Localisation related epilepsy

MAS	Meconium Aspiration Syndrome
MP	Malarial parasite
MSAF	Meconium Stained Amniotic Fluid
MT	Mantoux test
MRI	Magnetic resonance imaging
NCC	Neurocysticercosis
PS	Peripheral smear
RBS	Random blood sugar
RS	Respiratory System
SE	Status epilepticus
SFC	Simple febrile convulsion
SPECT	Single photon emission computed tomography
UMN	Upper motor neuron
URTI	Upper respiratory tract infection
USG	Ultra Sonography
WBC	White Blood Cell
WHO	World Health Organization

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INTRODUCTION

Seizures constitute the commonest neurological problem in children and the most common neurological emergency attended by paediatrician.

Due to the unpredictability of recurrence and varied clinical and subclinical manifestations, seizure disorder was always shrouded in mysticism and superstition. It was not until the 20th century when seizure disorders were separated from insanity and was not considered as a definite neurological illness which is potentially treatable.

Seizure is an excessive hyper synchronous electrical discharge from an aggregate of central nervous system neurons. Each burst of electrical activity is a called seizure. If a seizure arises from the motor cortex, leading to abnormal motor activity, it is called as convulsion. Epilepsy is defined as "two or more unprovoked seizures occurring at an interval more than 24 hours apart".[1]

The highest incidence of seizures is in early childhood and late adulthood. Seizures occur in 10% of children. Less than one third of seizures are caused by epilepsy.[1]Epilepsy incidence is 30-50 per 100000 population in high income countries and twice in low and middle income countries.[2] Annual prevalence is 0.5-0.8% globally.[1]The cumulative lifetime incidence of epilepsy is 3%; of which more than half of the cases begin in childhood. In childhood seizures, 10-20% are persistent seizures, refractory to drugs that pose a diagnostic and management challenge.[1]

A recent meta-analysis puts the overall prevalence rate of epilepsy in India at 5.59 per 1000 population. The worldwide prevalence of active epilepsy is 4-10 per 1000 population. [3]

Studies from different parts of India reveal that the prevalence of seizure disorders varies from 9/1000 in Bangalore, 5/1000 in Mumbai, 3/1000 near Calcutta to 4/1000 in New Delhi in 2001.[4]

At the global level, it is estimated that there are nearly 50 million patients suffering from seizure disorders of which three-fourths that is, 35 million, are in the developing countries. It accounts for 1% of the total global burden of disease. It is estimated that India alone has 10 million cases suffering from seizure disorders.[4] An estimated 2.4 million new cases of seizure occur each year globally. At least 50% of cases begin at childhood or adolescent age group. Of the total seizure patients, 70% to 80% could lead normal lives if properly diagnosed and treated. Still due to misbeliefs and unawareness, 60% cases of seizure disorders remain untreated in developing countries.[5]

To bring epilepsy "out of the shadows", a Global Campaign Against Epilepsy (GCAE) was launched in 1997 by World Health Organization (WHO). In 2005 report of GCAE, it was found that the mean number of cases with epilepsy per 1000 population was 8.93 from 105 responding countries. This data varies across the region. While it was 12.59 and 11.29 in the Americas and Africa, respectively, it was 9.97 in South-East Asia, 9.4 in the Eastern Mediterranean, 8.23 in Europe, and 3.66 in the Western Pacific. The mean number of cases with epilepsy per 1000 population ranged from 7.99 in the high-income countries to 9.50 in the low-income countries.[5]

Major advances in the understanding and treatment of epilepsy have occurred in the last century. Computerized axial tomography (CAT), magnetic resonance imaging (MRI), electroencephalography (EEG) and long-term EEG monitoring are available to health professionals. Therapeutic drug monitoring and neuropsychological services are also available, which lead to better understanding and research in pathophysiology, clinical presentation, etiological diagnosis, treatment plan and prognosis.

With advent of new investigation modalities and changing concepts due to research in seizure disorder, the ILAE had proposed new classification of seizure

disorders, which is used in this thesis work. These classification categories of patient are helpful to select Anti-epileptic Drugs (AEDs) and predict the prognosis.

Seizure disorder is a common and serious brain disorder. It is universal, with no age, sex, geographical, social class or racial boundaries. Since the diagnosis of epilepsy involves long-term management and carries a lot of social and psychological stigma, one needs to differentiate it from other common conditions resembling it. Misdiagnosis of seizures was found to be happening in 14% cases.[6] Thus this thesis work is taken up to study and correlate the clinical presentation of different seizures, clinical finding and investigations. This will help to make diagnosis, classify different seizures and to select treatment plan. As seizures may signal potentially serious underlying systemic or central nervous system (CNS) disorders that require thorough investigations and management.

AIMS AND OBJECTIVES

1. To evaluate the clinical profile of seizures in children admitted to R. L. Jalappa Hospital and Research Centre (RLJH & RC), Kolar.
2. To determine the etiological factors of seizure disorders admitted to the hospital.

REVIEW OF LITERATURE

HISTORICAL REVIEW:-

Seizure disorders are known as *Apasmara*in India and Sri Lanka, as "*Mirgee/Lata/Laran"* in northern India, "*Khichuni"* in Bangladesh, "*Ayan"* in Indonesia, while "*Rake Lom Ba Mu* or *Roke Lom Chak"* are the lay terms in Thailand.[4]

Basic concepts about seizure disorders in ancient Indian medicine were developed during the Vedic period of 4500-1500BC. In the Ayurvedic literature of Charaka Samhita dated 400BC, seizure is described as "apasmara" which means "loss of consciousness".[5] Epilepsy, mentioned in the *Nidanamsthanam*section of the *Charaka Samhita*, is described as a punishment for participation in the forbidden sacrifice.[4]

Charaka provided a definition of epilepsy almost confirming to the present concept: ‘‘Epilepsy is a disease characterized by derangement of the mind and memory. Therefore, victims of this disease experience disturbance in or loss of consciousness and undergo all kinds of ugly scenes.’’ Charaka described the prodromal symptoms of epilepsy as: ‘‘Epileptic seizure preceded by aura, a subjective phenomenon denoting the onset of an epileptic attack. During such episodes, a patient perceives some imaginary shapes or figures (visual aura), or hears certain peculiar sounds (auditory aura) before the onset of epileptic attack.’’[4]

Another ancient literature on seizure disorders is in a Babylonian textbook of medicine in the British Museum in London, comprising 40 topics dating as far back as 2000BC. This topic accurately records many of the different seizure types we recognize today.[4]

The Babylonian view was the forerunner of the Greek concept of "the sacred disease", as described in the famous treatise by Hippocrates (dated to the 5th Century

BC). Hippocrates, however, believed that epilepsy was not sacred, but a disorder of the brain.[4]

While both Hippocrates and the Charaka Samhita provided this less spiritualized understanding, the perception that epilepsy was a brain disorder did not begin to take root until the 18th and 19th Centuries AD. The intervening 2,000 years were dominated by more supernatural views.

A term "seizure" is derived from the *Latin* word *sacire*"to take possession of".[7]The first modern definition of seizure was given by **Hughling Jackson** in the second half of nineteenth century. He defined it as "the occasional, sudden, excessive, rapid and local discharge of grey matter of the brain". The current, operational definition of seizure is "the occurrence of transient paroxysms of excessive or uncontrolled discharges of neurons, which may be due to a number of different causes, leading to epileptic seizures".[4]

The word "epilepsy" is derived from a Greek term meaning "to possess", "to take hold", "to grab" or "to seize".[4] Working in Germany during the 1920s, **Hans Berger**, a psychiatrist, developed the human electroencephalograph (**EEG)**. Its important application from the 1930s onwards was in the field of seizure disorders. The EEG revealed the presence of electrical discharges in the brain. It also showed different patterns of brainwave discharges associated with different seizure types. The EEG also helped to locate the site of seizure discharges and expanded the possibilities of neurosurgical interventions.

During the first half of this century the main drugs for treatment were phenobarbitone (first used in 1912) and phenytoin (first used in 1938). Since the 1960s, there has been an accelerating process of drug discovery, based in part on a much greater understanding of the electrochemical activities of the brain, especially

the excitatory and inhibitory neurotransmitters. In developed countries in recent years, several new drugs have come into the market and seizures can now be controlled in 70% to 80% of newly diagnosed children and adults.[4]

Another recent stimulus towards the understanding and treatment of epilepsy in the last few decades has been the development of neuroimaging equipment like computed tomography (CT) scan, Magnetic resonance imaging (MRI), Ultrasonography (USG) and Single photon emission computed tomography (SPECT). Such technology has revealed many of the more subtle brain lesions responsible for epilepsy. Any type of brain disorders for example, congenital, developmental, infectious, vascular, neoplastic, degenerative etc. might lead to seizure disorders in some cases.

Of the 50 million people in the world with epilepsy, some 35 million have no access to appropriate treatment. This is either because services are non-existent or because seizure disorder is not viewed as a medical problem or a treatable brain disorder.[4]

The International League Against Epilepsy (ILAE) was founded in 1909 and is a professional organization with chapters in 60 countries. The International Bureau for Epilepsy (IBE) was founded in 1961 and is a lay organization with around 55 national chapters. In 1997 the ILAE and the IBE joined forces with the World Health Organization (WHO) to establish the Global Campaign Against Epilepsy (GCAE) to address these issues.

Mechanisms of Seizures:-

To initiate a seizure, there must be a group of neurons that are capable of generating a significant burst discharge and impairment of the gamma-amino butyric acid (GABA) inhibitory system. Seizure discharge transmission ultimately depends on

excitatory glutaminergic synapses. Evidence suggests that excitatory amino acid neurotransmitters (glutamate, aspartate) may have a role in producing neuronal excitation by acting on specific cell receptors. Seizures may arise from area of neuronal death, and these regions of the brain may promote development of novel hyper excitable synapses that can cause seizures. Lesions in the temporal lobe (including slow-growing gliomas, hamartoma, gliosis, hippocampal sclerosis, and arteriovenous malformations) cause seizures, and when the abnormal tissue is removed surgically, the seizures are likely to cease.

Two hypotheses have been suggested to explain the origin of seizures after brain injury. One suggests that inhibitory neurons are selectively damaged and remaining principal excitatory neurons becomes hyper excitable. The other hypothesis suggests that aberrant excitatory circuits formed as part of reorganization after injury. Convulsions may be produced in experimental animals by the phenomenon of kindling. In this model, repeated subconvulsive stimulation of the brain (amygdala) ultimately leads to a generalized convulsion by changes in synapses. This synaptic mechanism may also occur in humans.

Seizures are more common in infants. Certain seizures in the pediatric population are age specific (infantile spasms); this observation suggest that the underdeveloped brain is more susceptible to specific seizures than is the brain of an older child or adult. This is consistent with the basic science data indicating that the immature brain is more excitable than the mature brain, reflecting the greater influence of excitatory glutamate-containing circuits. The actions of GABA, the major inhibitory neurotransmitter, are often paradoxically excitatory in the immature brain. Enhanced excitatory activity may contribute to the developing brain's greater capacity for activity dependent plasticity.

Genetic factors account for at least 20% of all cases of epilepsy. Using linkage analyses, the chromosomal location of several familial epilepsies has been identified including benign neonatal convulsions (20q and 8q), juvenile myoclonic epilepsy (6p), and progressive myoclonic epilepsy (21q22.3). The genetic defect of benign familial neonatal convulsions has been characterized by the identification of submicroscopic deletions of chromosome 20q13.3. [1] The substantia nigra has an integral role in the development of generalized seizures. It has been proposed that functional immaturity of the substantia nigra may have a role in increased seizure susceptibility of the immature brain. The GABA-sensitive substantia nigra pars reticulate neurons play a part in preventing seizures. It is likely that substantia nigra outflow tracts modulate and regulate seizure dissemination but are not responsible for the onset of seizures.

TERMINOLOGY:-

Terminologies used to describe the seizure disorder are defined as below:-

1. **Seizure or convulsion** is a paroxysmal, time limited change in motor activity and / or behaviour that results from abnormal electrical activity in the brain.[1]
2. The **ILAE** Definition of epileptic seizures is ‘‘a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain.’’[8]
3. **Epilepsy** is defined as two or more unprovoked seizures occur at an interval greater than 24 hours apart.[1]
4. **Motor:** Involves musculature in any form that is, to increase or decrease the muscle contraction to produce movements. [9]

5. **Tonic:** A sustained increase in muscle contraction lasting for few seconds to minutes.[9]
6. **Clonic:** Myoclonus that is repetitive involves the same muscle groups, at a frequency of 2-3 c/s and is prolonged.[9]
7. **Tonic-clonic:** A sequence consisting of tonic followed by clonic phase. [9]
8. **Generalised tonic-clonic seizures:** Bilateral symmetric tonic contractions and then bilateral clonic contractions of somatic muscles usually associated with autonomic phenomena. [9]
9. **Myoclonic:** Sudden, brief (<100 ms) involuntary single or multiple contractions of muscles or muscle groups of variable topography (axial, proximal limb or distal). [9]
10. **Aura:** Subjective ictal phenomenon that, in a given patient, may precede an observable seizure; if alone constitute a sensory seizure. [9]
11. **Status epilepticus (SE):** It implies single seizure lasting more than 30 minutes or multiple episodes of seizures lasting more than 30 minutes without regaining consciousness in between.[10]
12. **Epilepsy Syndrome:** A complex of signs and symptoms that define a unique epilepsy condition with different aetiologies. This must involve more than just the seizure type; thus frontal lobe seizures per se, for instance, do not constitute a syndrome. [6]
13. **Benign epilepsy syndrome:** A syndrome characterized by epileptic seizures that are easily treated, or require no treatment, and remit without sequelae. [6]
14. **Idiopathic epilepsy syndrome:** A syndrome that is only epilepsy, with no underlying structural brain lesion or other neurological signs or symptoms. These are presumed to be genetic and are usually age-dependent. [6]

15. **Symptomatic epilepsy syndrome:** A syndrome in which the epileptic seizures are the result of one or more identifiable structural lesions of the brain. [6]
16. **Probably symptomatic epilepsy syndrome:** Synonymous with, but preferred to, the term cryptogenic, used to define syndromes that are believed to be symptomatic, but no etiology has been identified. [6]
17. **Active Epilepsy:** if the patient with epilepsy has at least one seizure in the preceding two years and is or has been on antiepileptic drugs for the same. [4]
18. **Acute symptomatic seizures**: Seizures occurring at the time of a systemic insult or in close temporal association with a documented brain insult. [11]
19. **Unprovoked seizures:** Seizures occurring in the absence of precipitating factors and may be caused by a static injury (remote symptomatic seizures) or a progressing injury (progressive symptomatic seizures). [11]
20. **Febrile Convulsions:** - seizures during fever occurring between 6 month to 5 years age in absence of infection of central nervous system in neurologically normal child.[12]
21. **Simple Febrile Convulsion (SFC) –** Features of SFC are -the convulsions occur within 24 hours of the onset of fever; convulsions last for duration less than 15 minutes; usually single attack of seizure per febrile episode; generalised tonic-clonic seizure type, only 4-18% are focal convulsions; no post-ictal focal neurological deficit and no family history of febrile convulsion in the siblings. [12]

Complex Febrile Convulsion (CFC) - Clinical features other than features of simple febrile convulsion. Features are - seizures lasted more than 15 minutes; repeated convulsions occur with in 24 hours or when focal activity or focal findings are present during post ictal period.[1]

In a study conducted by **Dura-Trave T et al** in children aged 1 month to 15 years, in Navarre, Spain from January to December 2005. International League Against Epilepsy criteria was used for diagnosis. A total of 365 children were recruited into the study. Mean age at diagnosis was 5.97 years, and time of follow-up was 4.6 years. Etiology was idiopathic in 166 (45.5%), cryptogenic in 106 (29.0%), and symptomatic in 93 (25.5%). Focal seizures were seen in 52.9% of the patients, generalized epilepsy in 43.5%, and 3.6% were not determined. In infants, West syndrome (34.1%) and focal symptomatic seizures (24.4%) were the most prevalent syndromes. In early childhood, the main syndromes were cryptogenic focal epilepsies (17.7%) and Doose syndrome (12.8%). In school-aged children, benign epilepsies (27.3%) and absences (24.5%) were prevalent. In adolescents, cryptogenic focal epilepsies (26.6%) and benign epilepsies were common (23.4%). [13]

Lin KL et al carried out a study on convulsive status epilepticus in children of Taiwan during year 1999 and 2006. They enrolled 141 patients with convulsive status epilepticus, aged 2 months to 18 years: 24.8% of first episodes developed convulsive status epilepticus, with a duration of over 60 minutes. First episodes of convulsive status epilepticus were most often evidenced in febrile status during acute central nerve system infections (48.2%) and in non-febrile status during acute non-central nervous system illness in previously epileptic children (28.4%). Before their first episode, 63.8% of children were neurologically healthy, and 12.2% exhibited a prolonged febrile seizure. The most common aetiology of mortality was acute central nervous system infection. Acute central nervous system infections appear to be markers for morbidity and mortality. [14]

A study carried out by **Sampaio LP et al** on seizure disorders among 101 cases during year 2005 to 2006 in Brazil. Partial seizures were the most frequent seizure type (62/101). Symptomatic focal epilepsy was the most common form, and hypoxic-ischemic encephalopathy the most common etiology, reflecting the socioeconomic conditions of this specific population. [15]

In a study carried out by **Dent W et al** on seizure disorders among 42 cases (21 males and 21 females) of active epilepsy during year 2005 in Austria. Thirty of them were generalized seizures, whereas 12 patients reported partial seizures. The peak prevalence was found in adolescent age group. Eleven patients (26.2%) were classified as "strongly suspected of symptomatic" epilepsy, the remaining 31 patients (73.8%) as possibly being idiopathic, symptomatic, or cryptogenic epilepsy cases. [16]

Richard et al studied seizure disorder among 900 cases aged 0-13 years in Kenya during year 2004-2006. They found that, over 80% of the seizures were associated with infections. Falciparum malaria 58% was the main infection associated with seizures in children. Falciparum malaria was also the main illness accounting to 57% associated with status epilepticus. Other illnesses associated with seizures included pyogenic meningitis, respiratory tract infections and gastroenteritis. Twenty-eight children (3.1%) with seizures died and 11 surviving children (1.3%) had gross neurological deficits on discharge. [17]

Singh RK et al carried out study on status epilepticus on 144 cases in year 2007, in Washington, USA. The average age was 3.4 years. The majority of seizures (72%) lasted between 21 and 60 minutes. The majority of patients had no significant

past medical history; one-fourth had a family history of epilepsy. Five (4%) patients with EEGs had electrographic seizures during the study. The most common etiology was febrile convulsion, followed by cryptogenic. The most common acute symptomatic cause was CNS infection; the most common remote symptomatic cause was cerebral dysgenesis. Combined CT and MRI provided a diagnosis in 30%. CT was helpful in identifying acute vascular lesions and acute edema, whereas MRI was superior in identifying subtle abnormalities and remote symptomatic aetiologies such as dysplasia and mesial temporal sclerosis. [18]

A study carried out by **Goel D et al** on total 1176 cases in year 2008 in India. The male cases were more than female cases. 58% cases had partial seizures, 28% had generalized seizures and 13% had undetermined seizures. When ILAE classification was applied, seizure typing was in 86.2%, 68.5% and 26.7% patients of partial, generalized and unclassified seizures respectively. Only, 146 patients (12.5%) found to have symptomatic cause for seizures. After utilizing the ILAE classification on 1030 patients (87.5%) of "unknown etiology" cases, almost 86.5% patients could be classified to a definite etiological class. [19]

Chen CY et al studied seizure disorders among 319 cases during year 2005-2007 in Changhua Children's Hospital, Taiwan. Among 319 patients, 218 (68%) presented with seizures and fever and 299 (94%) children were younger than 6 years of age. Generalized tonic-clonic seizures were the most common type (71.2%). Febrile seizures (62.1%) were the main etiology of the first seizure ($p < 0.001$). Seizures caused by severe electrolyte imbalance or hypoglycemia were noted in three patients.

Abnormal brain images were noted in 16 (26%) of 61 patients, most (12/16, 75%) of whom had abnormal history and physical or neurologic examinations. [20]

Kannoth S et al studied seizure disorder among 362 cases in year 2007 in Kerala, India. They concluded that family history of epilepsy, antecedent history of febrile seizures, birth by complicated delivery, and neonatal seizures emerged as strong independent predictors of epilepsy, followed in decreasing order by mental retardation, prematurity, perinatal distress, and incomplete immunization. There were more similarities than differences in the distribution of risk factors between generalized and localization-related epilepsy syndromes. [21]

A study carried out by **P Alizadeh Taheri et al** on81 students of seizure disorders during year 2008 in Iran. They categorized as having a history of seizure, 51.8% boys versus 48.2% girls. 11 (13.5%) were categorized as having epilepsy, 7 boys versus 4 girls. Forty eight (59.2%) reported febrile illness as the presumptive underlying cause of seizure (26 boys and 22 girls). The most common clinical type of seizure was the generalized form (either tonic, clonic, tonic clonic or atonic). Febrile convulsion was the most common etiology. A positive family history was detected in 29.6% of seizure cases. History of seizure was more prevalent in distant relatives than first degree relatives (21% versus 8.6%). Totally, 44 (54.3%) cases were either on anti-epileptic medication or had taken them in the past. The most common drug prescribed by the physicians was Phenobarbital, taken by 34(77%) of patients. Seven students had a history of taking Carbamazepine, 4 Phenytoin, 4 Sodium Valporate, 1 Primidone and 1 Lamictal. Of 11 cases of epilepsy, 4 were resistant to long term barbiturate usage and were taking Sodium Valporate instead. [22]

Nguefack S et al carried out a prospective study of 325 children in Yaounde city, France, from 15 January to 15 December 2008. The proportion of patients with febrile seizures among all admitted patients was 6.1%. The mean age was 24.6 months; the peak age of these patients was 12-17 months. The sex ratio was 1.5. The mean temperature on admission was 39.2 degrees C. There was a family history of febrile seizures in 36.4%. Simple febrile seizures were seen in 58.7% and the complex type in 41.3%. Malaria was the main etiology in 67.7%, followed by upper and lower respiratory tract infection in 14.1% and 9.8%, respectively. Lumbar puncture was performed in all patients. The commonest anticonvulsant used to stop seizures was rectal diazepam in 88%. Febrile seizure prophylaxis was administered to 43% of the patients; antipyretics were the most widely used, either alone or combined with rectal diazepam. [23]

Sinha S et al studied the convulsive refractory status epilepticus (RSE) on 98 patients in year 2009 at Karnataka in India. The precipitating factors included viral fever – 13 and AEDs stoppage - 7. EEG was abnormal in 81.5% of patients. CT and MRI were abnormal in 63.4% and 82.3% respectively. Thirty-four patients died. Seizures could still be controlled in two-thirds of patients with convulsive RSE. About 30% of patients achieved long-term seizure freedom. [24]

Shah P et al carried out on seizure disorders in 49 school-going children (6-18 years) during year 2009, in Kashmir valley. It included 55.1% males and 44.9% females. Age-specific prevalence was found to be 3.82/1000 (6-10 years), 3.44/1000 (11-14 years) and 2.33/1000 (15-18 years). Again, higher prevalence (3.38/1000) was observed in children from government run educational institutions. Generalised tonic clonic seizures (73.5%) were the commonest type of seizure observed. [25]

SudhirAdhikari et al studied a total of 551 patients admitted for seizures in a tertiary care hospital of Western Nepal. It included 338 (61.3%) males and 213 (38.7%) females. Among these patients, 295 (53.5%) presented with fever and 317 (57.5%) of children were less than 5 years of age. Generalized tonic-clonic seizures were the most common seizure type (69.9%). Seizure disorder (33.4%), febrile seizures (30.7%), CNS infections and neurocysticercosis were common etiologies. Abnormal brain images were noted in 111 (45.9%) of 242 patients and most common abnormality was neurocysticercosis 66 (59.5%).[26]

Prakash Poudel et al conducted a study in Nepal in 2013,It included 308 (age one month to 20 years) children with afebrile seizures.Median age at first seizure was 39 months. Status epilepticus was present in 26.0%. Cause of seizure was known in 44.2%. Seizure was generalized in 79.2%.Common causes of seizure were – birth asphyxia (12.3%), neurocysticercosis (8.8%), sequel of nervous system infection (6.5%) and structural brain abnormalities (7.1%).Neurological examination, electroencephalography and computed tomography (CT) were abnormal in 24.4%, 70.5% and 27.9% cases respectively.[27]

In a study conducted by **Sangeeta V B et al** in Karnataka in 2014,It included 505 children aged between 6 months to 24 months with first episode of febrile seizures .Simple febrile seizures accounted for 63% (n=203) out of which none of them had CSF findings suggestive of meningitis. Complex febrile seizures accounted for remaining 37% (n=119) out of which 5(4.2%) children had CSF findings suggestive of meningitis.The most common cause of febrile seizure was URTI followed by LRTI.[28]

MATERIALS AND METHODS

This was an one year observational hospital based study carried out at a tertiary health care centre. Sample size was estimated to be 150.

Sample size estimation:

Our hospital being a tertiary care center and total number of admissions for year 2012-13 were 3242 . Of which, 324 cases were admitted with seizures, among them 216 were new onset seizures. Based on this value, sample size was estimated at 6.66% expected proportion with 5% absolute error. At 95% confidence interval a minimum sample size of 96 was obtained. Considering 10% non response rate sample size required was 96+10=106.

In my study duration i.e from December 2013 to January 2015 total of 150 cases with new onset seizures were admitted and was included to my study.

Formula used:

$$\textbf{Sample size} = \frac{Z_{1-\alpha/2}^2 p(1-p)}{d^2}$$

Here

$Z_{1-\alpha/2}$ = Is standard normal variate (at 5% type 1 error ($p<0.05$) it is 1.96 and at 1% type 1 error ($p<0.01$) it is 2.58).As in majority of studies p values are considered significant below 0.05 hence 1.96 is used in formula.

p = Expected proportion in population based on previous studies or pilot studies.

d = Absolute error or precision. Has to be decided by researcher

p = 10% or 0.10

q = 90% or 0.9

d = 5% or 0.05

Data was entered in Microsoft Exel sheet and analysed using SPSS 11 version.

Descriptive statistics like mean ,standard deviation , frequency, proportion , were computed for quantitative and qualitative data respectively .

Study Period: The study was conducted over a period of one year from Dec 2013 to Jan 2015.

Place of Study: Study was conducted at Department of pediatrics, R. L. Jalappa Hospital, Kolar.

Source of Data: Children between 1 month to 18 years with seizures.

METHOD OF COLLECTION OF DATA:

Inclusion Criteria:

All children between 1 month to 18 years admitted with new onset undiagnosed febrile or afebrile convulsive seizure disorder to R.L. Jalappa Hospital and Research Centre , Kolar

Exclusion Criteria:

Patients presented with pseudo seizures.

Patients presented with head injury.

METHODOLOGY

Patient's detailed history was obtained from the informant who has witnessed the seizure episode. All details obtained from history, examination and investigations were recorded in a predefined case proforma.

In clinical history more emphasis was given on the points, which are helpful in classification and diagnosis of seizures. Age is one of the important characters, as different seizure types are presented in a specific age group. Age of patients was

divided in 3 groups as <1 year (infant), 1 to 5 years(toddlers and preschool children) and 6 to 18 years (school going and adolescents).Each patient's sex and consanguinity were also noted.

Details of the seizure episode were recorded as below-

1. Type of convulsion that is, generalised or focal,
2. Motor manifestation as tonic, clonic or tonic-clonic,
3. Autonomic components like vomiting or bowel-bladder incontinence,
4. Level of consciousness was measured by using AVPU scale[29]
5. Duration of convulsion,
6. Associated cyanosis or fever,
7. Post-ictal level of consciousness and focal neurological deficit,
8. Number of seizure episodes, that is 1,2 ,> or =3 (multiple)
9. Interval between repeated seizure episodes was within 24 hours or 24 hours apart.

Past history of seizures were excluded from study. Past history of measles, tuberculosis or tuberculosis contact were recorded. Birth history, immunisation status, nutrition and anthropometry were also noted in detail.

History of seizure disorders in any family member was enquired and details were noted as age of onset, diagnosis available if any, anticonvulsant if started any and natural course till now. Family history of any other neurological abnormality for example neurocutaneous syndrome, deafness, blindness etc, was recorded.

In general examination, patients' level of consciousness, febrile or afebrile, temperature at time of convulsion, vitals (pulse rate, blood pressure and respiratory

rate), BCG scar and neurocutaneous markers were recorded. Detailed systemic examination of central nervous system was done as higher function, cranial nerve deficit, motor system, sensory system, involuntary movements, signs of meningitis, cerebellar signs and fundus examination. Other systems were also examined and findings were noted.

Routine investigations were done in all patients, which include complete blood count (CBC), random blood sugar (RBS), serum electrolytes, serum creatinine, serum urea and chest X-ray.

Special investigations were done in selected cases or whenever necessary, in whom history and examination were suggestive of a specific etiology, for example malaria- peripheral smear for malaria parasite; rickets or hypocalcemic seizures- serum calcium, serum phosphorus and alkaline phosphatase level; urinary tract infection- urine microscopy and culture; enteric fever- Widal test and blood culture; child of seropositive parent or patient suspected to be seropositive- Enzyme Linked ImmunoSorbent Assay (ELISA) test for HIV; cases with Koch's contact were screened by Mantoux test (MT) given on volar aspect of left forearm or BCG test and Gastric lavage (GL) or sputum for acid fast bacilli (AFB). MT / BCG test readings were taken at 48 to 72 hours; heart disease- electrocardiogram (ECG) and 2-D ECHO; meningitis and encephalitis- cerebrospinal fluid (CSF) examination, blood culture, Ultrasonography (USG) scan of skull, neuroimaging and Electroencephalogram (EEG).

Lumbar puncture was done in any child who presents with a seizure and

- Fever and < 1 year
- Fever with meningeal signs and symptoms (neck stiffness, Kernig and/or Brudzinski signs) or History suggestive of neuroinfection or meningitis.[30]

Lumbar puncture was always done after fundus examination, to rule out increased intracranial pressure, under all aseptic precautions.

Neuroimaging

Emergency

- In a child of any age who exhibits a post ictal focal deficit not quickly resolving

Elective

- In any child with a significant cognitive or motor impairment of unknown etiology, unexplained abnormalities on neurologic examination
- A seizure of partial(focal) onset with or without secondary generalization.[30]

Electroencephalogram (EEG) will be done for

- All first unprovoked seizure.[30]

All EEGs were done on computerised "Neuropage plus" machine using 23 electrode system. All EEGs were done at least 24 hours after last episode of convulsion.[30] All EEGs were interpreted and reported by senior psychiatrist in the hospital.

Patients were classified according to the classification given below:-

International Classification of Epileptic Seizures [(1)]

PARTIAL SEIZURES

Simple partial (consciousness retained)

1. Motor
2. Sensory

3. Autonomic
4. Psychic

Complex partial (consciousness impaired)

1. Simple partial, followed by impaired consciousness
2. Consciousness impaired at onset

Partial seizures with secondary generalization

GENERALIZED SEIZURES

Absence

1. Typical
2. Atypical

Generalized tonic-clonic

Tonic

Clonic

Myoclonic

Atonic

Infantile spasms

UNCLASSIFIED SEIZURES

Classification of Epilepsies and Epileptic Syndromes[1]

LOCALIZATION RELATED (FOCAL, PARTIAL) EPILEPSIES

Idiopathic

1. Benign childhood epilepsy with centro-temporal spikes
2. Childhood epilepsy with occipital paroxysms

Symptomatic

The sub-classification determined by the anatomic location suggested by the clinical history, predominant seizure type, EEG, and imaging studies; thus SPS,CPS, or secondarily generalized seizures arising from frontal lobes, parietal, temporal, occipital, multiple lobes or an unknown focus

Localization related but uncertain symptomatic or idiopathic.

GENERALIZED EPILEPSIES

Idiopathic

1. Benign neonatal familial convulsions
2. Benign neonatal convulsions
3. Benign myoclonic epilepsy in infancy
4. Childhood absence epilepsy (pyknoepilepsy)
5. Juvenile absence epilepsy
6. Juvenile myoclonic epilepsy (impulsive petit mal)
7. Epilepsy with grand mal seizures upon awakening
8. Other generalized idiopathic epilepsies that do not conform exactly to the syndromes just described.

Cryptogenic or symptomatic generalized

1. West syndrome (infantile spasms)
2. Lennox- Gastaut syndrome
3. Epilepsy with myoclonic astatic seizures
4. Epilepsy with myoclonic absences
5. Symptomatic

6. Non-specific cause
7. Early myoclonic encephalopathy
8. Specific disease state manifesting

EPILEPSIES OR SYNDROMES UNDETERMINED AS FOCAL OR GENERALISED

1. With both generalized and focal seizures
2. Neonatal seizures
3. Severe myoclonic epilepsy in infancy
4. Epilepsy with continuous spike and wave pattern during slow wave sleep
5. Acquired epileptic aphasia (Landau-Kleffner syndrome)
6. Without unequivocal generalized or focal features
7. All cases with GTCS in which the EEG findings do not allow classification as generalized or localization-related.

SPECIAL SYNDROMES

Situation-related seizures

1. Febrile convulsions
2. Isolated seizures or isolated status epilepticus
3. Acute symptomatic seizures for example alcohol withdrawal seizures, eclampsia, uremia

Etiological diagnosis was done on the basis of diagnostic criteria for each condition.

Neurocysticercosis was defined by following criteria[31]

Absolute

- Histological demonstration of the parasite from biopsy of a brain or spinal cord lesion
- Evidence of cystic lesions showing the scolex on neuroimaging studies
- Direct visualization of sub retinal parasites by fundoscopic examination

Major

- Evidence of lesions highly suggestive of neurocysticercosis on neuroimaging studies(cystic lesions without showing the scolex, single or multiple ring or nodular enhancing lesions, and small parenchymal round calcifications)
- Positive serum immunoblot for the detection of anticysticercal antibodies
- Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel
- Spontaneous resolution of small single enhancing lesions

Minor

- Evidence of lesions compatible with neurocysticercosis on neuroimaging studies(Hydrocephalus and abnormal enhancement of the leptomeninges)
- Presence of clinical manifestations suggestive of neurocysticercosis
- Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens
- Evidence of cysticercosis outside the central nervous system

Epidemiological

- Individuals coming from or living in an area where cysticercosis is endemic
- History of travel to disease-endemic areas
- Evidence of a household contact with *T. solium* infection

Degrees of diagnostic certainty

Definitive

- Presence of one absolute criterion
- Presence of two major plus one minor and one epidemiological criteria

Probable

- Presence of one major plus two minor criteria
- Presence of one major plus one minor and one epidemiological criteria
- Presence of three minor plus one epidemiological criteria

Cerebral malaria was defined clinically as[32]

Coma at least 1 hour after termination of seizure or correction of hypoglycaemia ,with no other evident cause of coma.

Presence of p.falciparum in peripheral blood smear.

Encephalitis was defined as[33]

Major Criteria (required):

Patients presenting with altered mental status (defined as decreased or altered level of consciousness, lethargy or personality change) lasting ≥24 h with no alternative cause identified.

Minor Criteria (2 required for possible encephalitis; ≥3 required for probable or confirmed encephalitis):

- Documented fever ≥38° C (100.4°F) within the 72 h before or after presentation
- Generalized or partial seizures not fully attributable to a pre-existing seizure disorder

- New onset of focal neurologic findings
- CSF WBC count ≥5/cubic mm
- Abnormality of brain parenchyma on neuroimaging suggestive of encephalitis that is either new from prior studies or appears acute in onset
- Abnormality on electroencephalography that is consistent with encephalitis and not attributable to another cause.

Meningitis was defined as[34]

- Any child presenting with fever and seizures with signs of meningeal irritation and Lumbar puncture showing the following features

CONDITION	**LEUKOCYTES (mm^3)**	**PROTEIN (mg/dL)**	**GLUCOSE (mg/dL)**	
Acute bacterial meningitis	100-10,000 or more with neutrophilic predominance	100-500	Decreased,<40 (or <50% serum glucose)	Isolation of organism by gram stain and culture
Partially treated bacterial meningitis	5-10,000;usually neutrophils but lymphocytic predominance seen if pretreated for extended period of time	100-500	Normal or decreased	
Viral meningitis	<1,000 cells. Lymphocytic predominance	50-200	Normal; may be decreased to <40 in mumps	

Tubercular Meningitis diagnosed based on[35]

A.Clinical

Fever and headache lasting for more than 14 days (mandatory)

Vomiting or altered sensorium or focal deficits (optional)

B.Cerebrospinal fluid

Pleocytosis more than 20 cells predominantly lymphocytes(greater than 60%)

Protein greater than 100mg/dl

Sugars less than 60 mg/dl of corresponding blood sugars

Negative India ink and cytology for malignant cells (in relevant situations)

C.Radiological

CT studies showing 2 or more of the following

1. Exudates in basal cisterns or in the sylvian fissures
2. Hydrocephalus
3. Infarcts
4. Gyral enhancement

D.Extraneural Tuberculosis

Active tuberculosis of the lungs , gastrointestinal tract, urogenital tract, lymph nodes, skeletal system or skin as evidenced by appropriate radiological, microbiological tests or by presence of caseation necrosis by histological examination.

1. Definitive TBM

i. Clinical criteria(A)

ii. Bacteriological isolation from CSF or diagnosis at autopsy

2. Highly probable TBM

i. Clinical criteria(A)

ii. All three of B and C

3. Probable TBM

i. Clinical criteria(A)

ii. Any two of B,C and D

4. Possible TBM

i. Clinical criteria(A)

ii. Any one of B,C and D

Hyponatremia is defined as serum sodium level<135meq/l[36]

Diagnostic Criteria for Syndrome of Inappropriate Antidiuretic Hormone[36]

Absence of: Renal, adrenal, or thyroid insufficiency Congestive heart failure, nephrotic syndrome, or cirrhosis Diuretic ingestion Dehydration
Urine osmolality > 100 (usually > plasma)
Serum osmolality < 280 and serum sodium < 135
Urine sodium > 25

Hypernatremia is defined as serum sodium level<145meq/l.[36]

Hypocalcemia defined as serum calcium<7mg/dl

Hypoglycemia defined as a whole blood glucose concentration of <50 mg/dL (10–15% higher for serum or plasma).[36]

Congenital anomalies of central nervous system are diagnosed based on clinical and radiological features.[37]

RESULTS

Statistical Methods:

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Chi-square was used as test of significance to find the association. Bar diagram and Pie diagrams were used to represent the data graphically. p value <0.05 was considered as statistically significant.

Table 1: Age distribution of patients studied (n=150)

		Cases	Percent
Age	Infants (< 1 year)	22	14.7
	1 to 5 years	66	44.0
	6 to 18 years	62	41.3
	Total	150	100.0

Majority of subjects studied were in the age group of 1 to 5 years 66(44.0%).

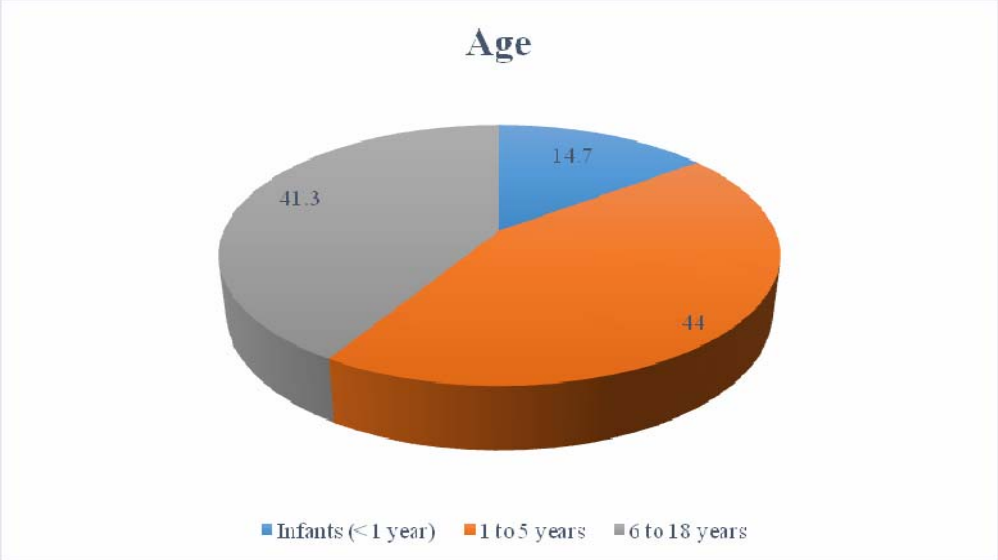

Figure 1: Age distribution of patients

Table 2: Gender distribution of patients

		Cases	Percent
Gender	Female	77	51.3
	Male	73	48.7
	Total	150	100.0

Majority of patients were Females 77 (51.3%).

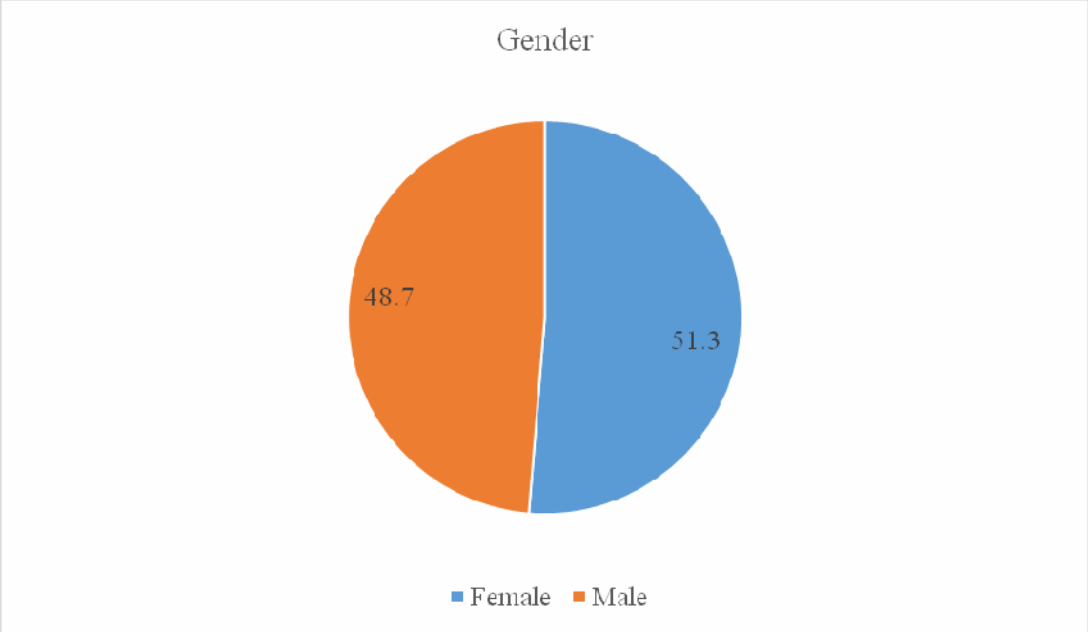

Figure 2: Gender distribution of patients

Table 3:Distribution of patients born to consanguineously married parents.

		Cases	Percent
Parents	Consanguineously married	16	10.7
	Non-Consanguineous	134	89.3
	Total	150	100.0

Majority of patients were born to non – consanguineous parents.

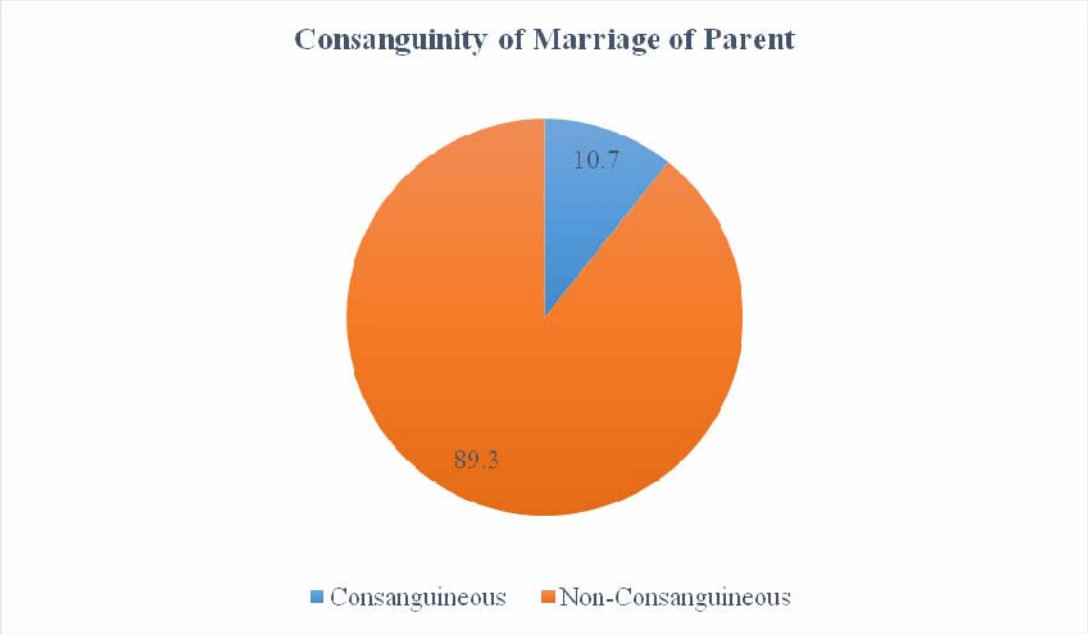

Figure 3: Patients born to Consanguineous parents

Table 4:Type of Seizures in the patients

		Cases	Percent
Type of Seizures	Partial	40	26.6
	Generalized	110	73.4
	Total	150	100.0

Majority of patients (73.4%) had generalized seizure .

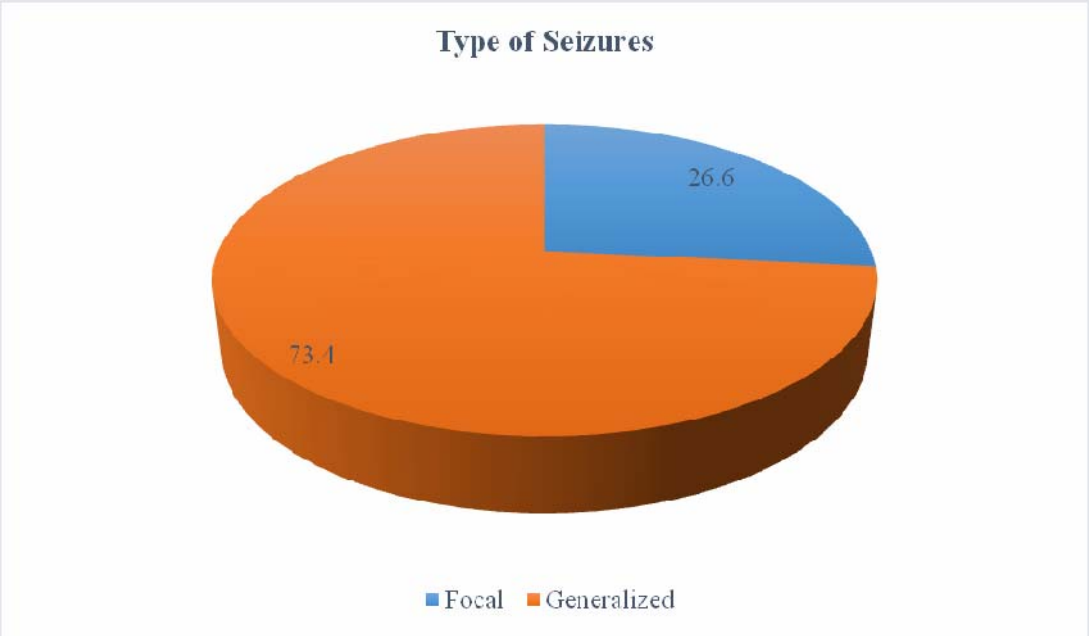

Figure 4: Type of Seizures

Table 5:Motor Manifestations of Seizures in the patients studied

		Number	Percent
Motor Manifestations of Seizures	Tonic-clonic	85	56.6
	Tonic	41	27.3
	Clonic	24	16
	Total	150	100.0

In this study, majority (56.6%) patients had Tonic clonic seizures.

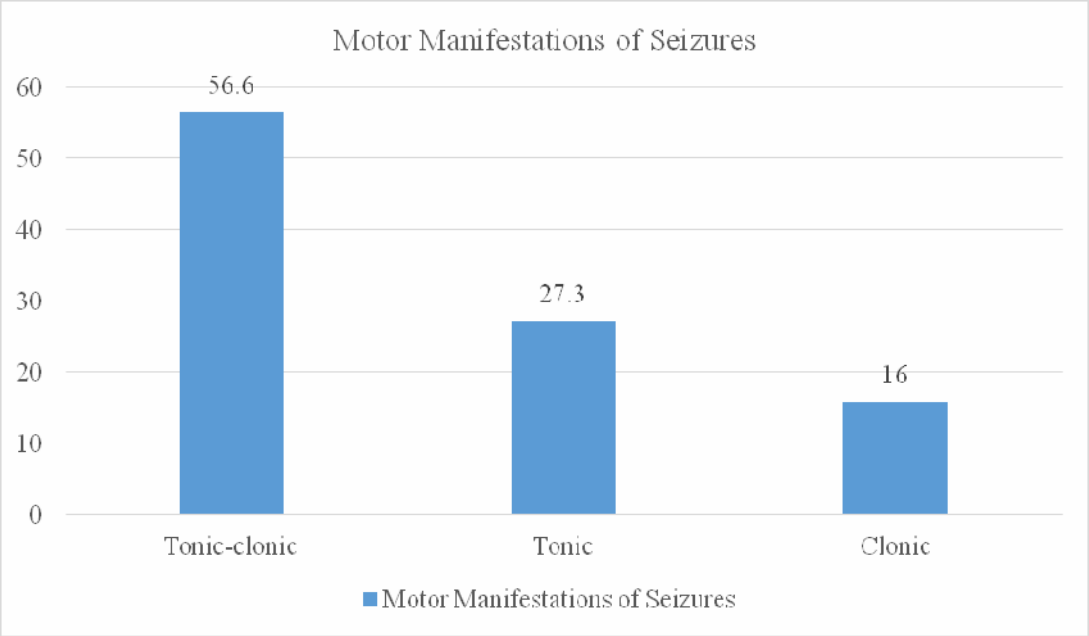

Figure 5: Motor Manifestations of Seizures

Table 6:Number of Seizure Episodes in patients

		Cases	Percent
Number of Seizure Episodes	One	67	44.7
	Two	52	34.7
	Multiple	31	20.7
	Total	150	100.0

Majority of patients (44.7%) had single episode of seizure.

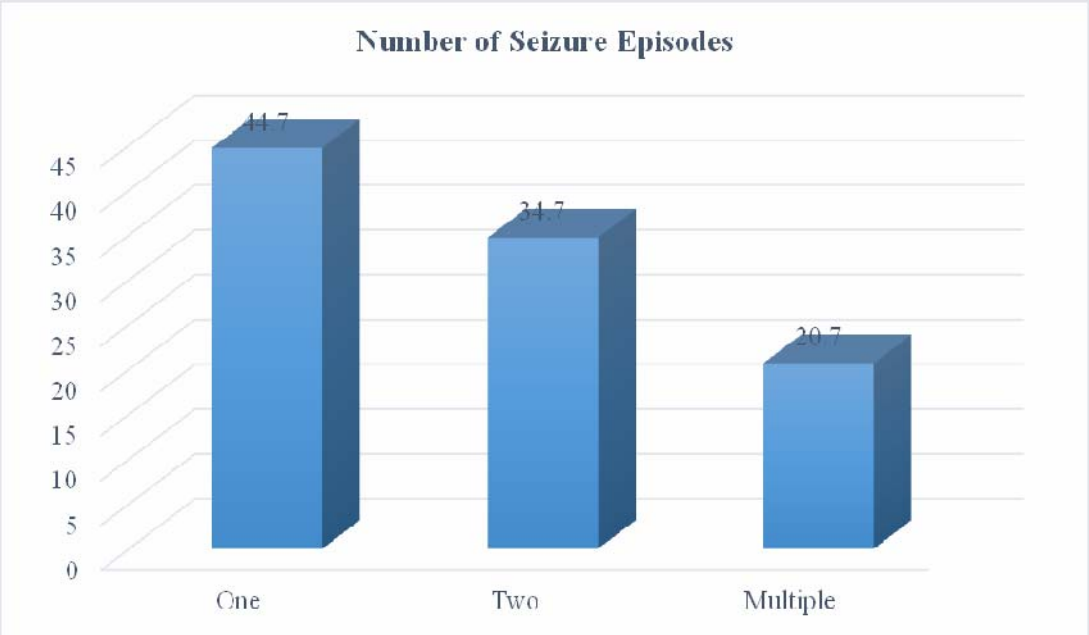

Figure 6: Number of Seizure Episodes

Table 7: Duration of each seizure episode in patients

		Cases	Percent
Duration of each seizure	<15 min	136	90.7
	16 to 30 min	9	6.0
	31 to 60 min	5	3.3
	Total	150	100.0

Majority of subjects (90.7%) had duration of seizure <15 min .

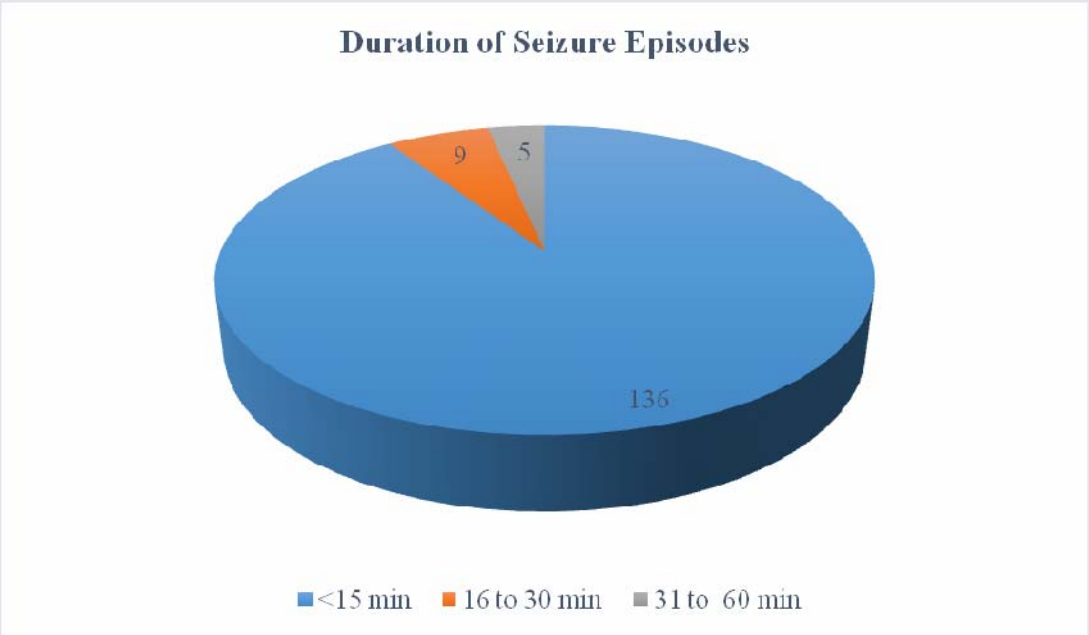

Figure 7: Duration of each Seizure episode in patients

Table 8: Mean and median duration of seizure in patients

Duration of Seizure Episode (min)						
N	Mean	Std. Deviation	Median	Minimum	Maximum	Range
150	8.19	9.692	5.00	1	60	59

Mean duration of seizure was 8.19 minutes.

Table 9: Showing number of patients who had epilepsy

		Cases	Percent
Epilepsy	Yes	20	13.3
	No	130	86.7
	Total	150	100.0

13.3% of cases had epilepsy.

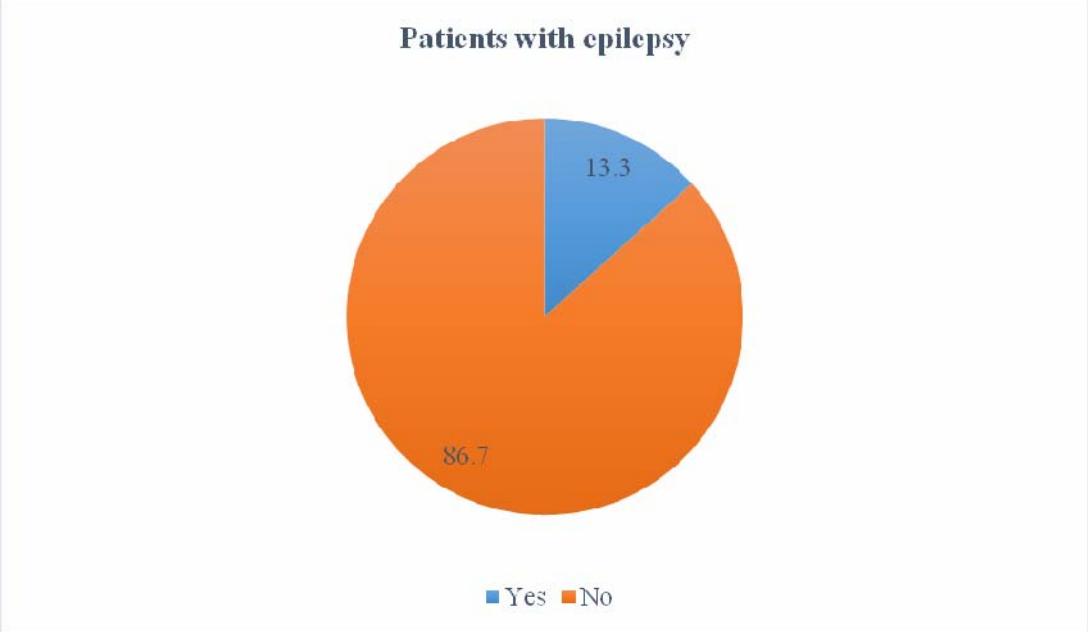

Figure 8: Number of patients with epilepsy

Table 10: Showing number of patients who had seizures associated with fever

		Cases	Percent
Fever	Present	87	58.0
	Absent	63	42.0
	Total	150	100.0

58% of patients studied had seizures associated with fever .

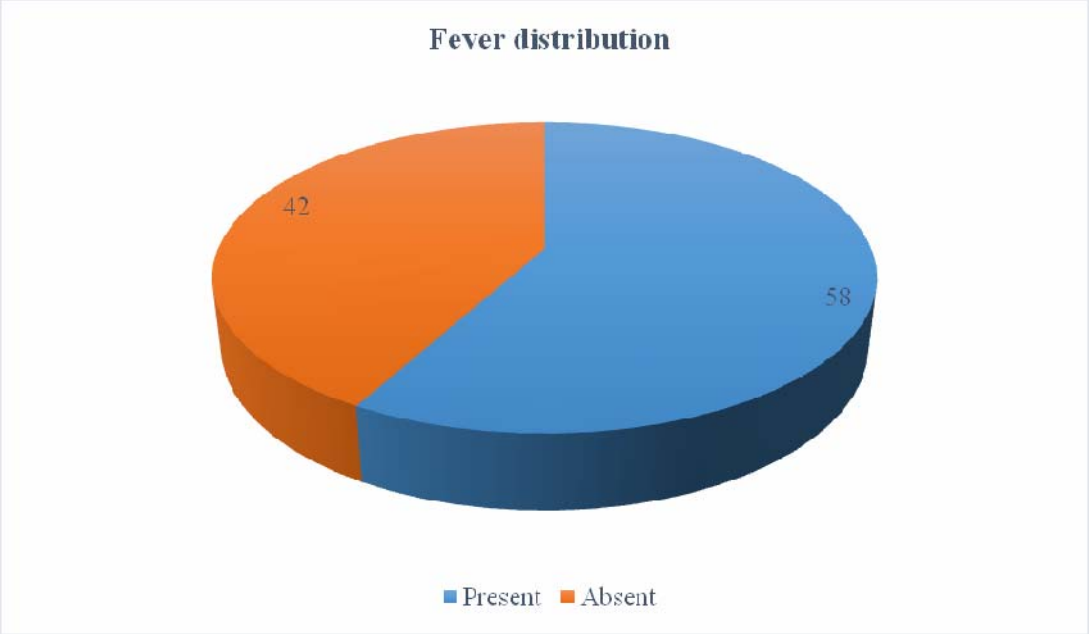

Figure 9: Number of patients who had seizure associated with fever

Table 11: Events during Seizure episode

		Cases	Percent
Loss of Consciousness during seizure episode	No (Conscious)	12	8.0%
	Yes (Unconscious)	138	92.0%

In the study 92.0% of patients had Loss of Consciousness during seizure episode.

Table 12: Post ictal phenomenon in seizure subjects

Level of Consciousness at admission	Awake	88	58.7%
	Response to verbal stimulus	50	33.3%
	Response to painful stimulus	6	4.0%
	Unresponsive	6	4.0%
Neurological deficits	Absent(No)	144	96.0%
	Present(Yes)	6	4.0%

58.7% were awake at admission to hospital and 4% had Neurological Deficits.

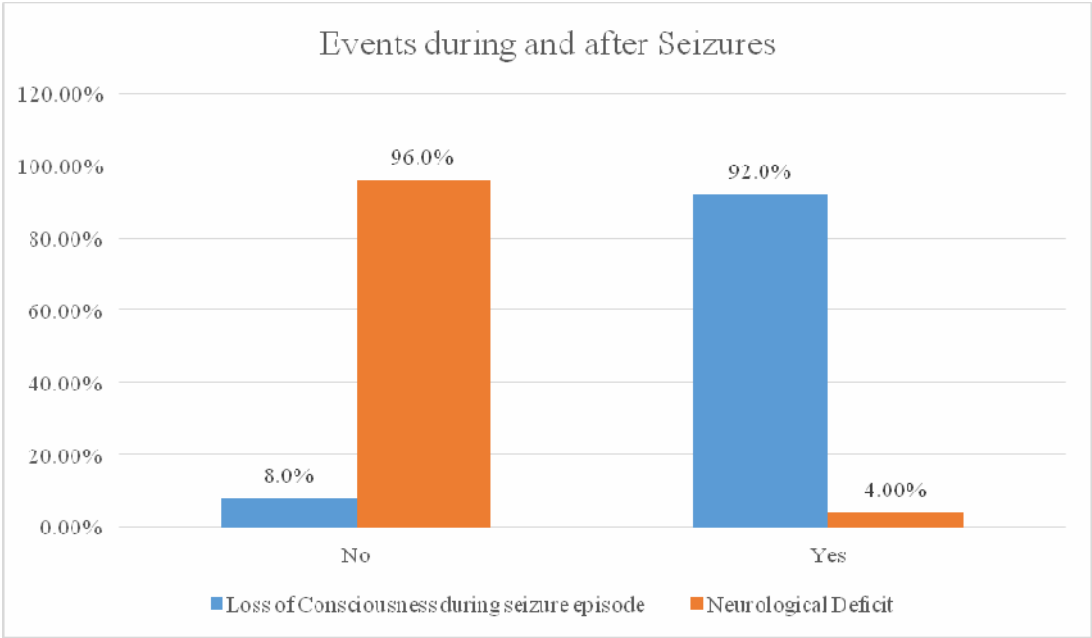

Figure 10: Events during and after Seizures

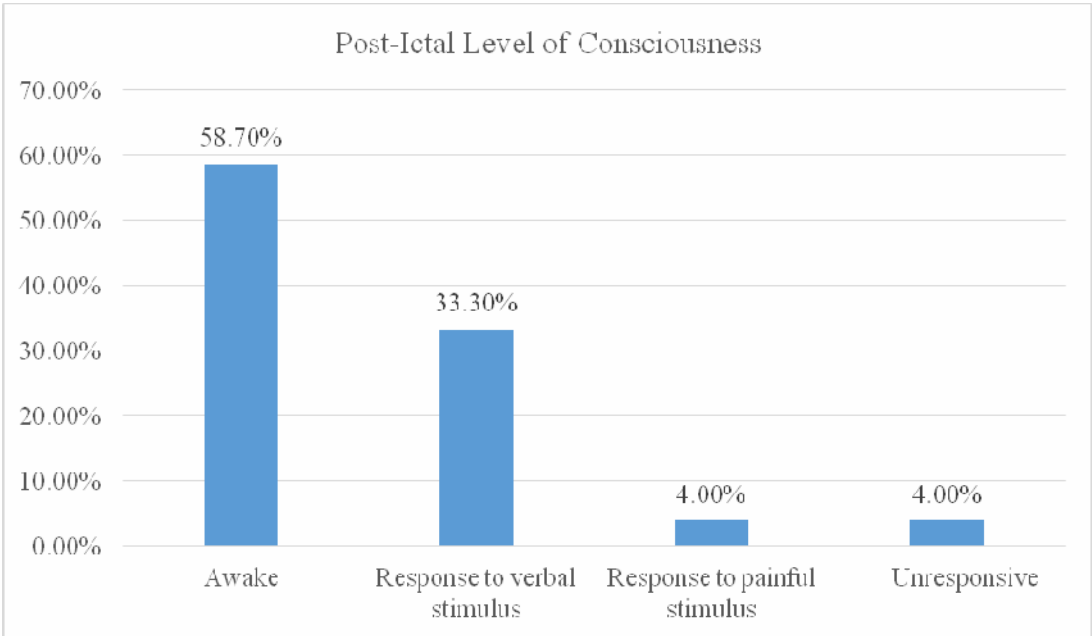

Figure 11: Post-Ictal Level of Consciousness

Table 13: Risk factors for seizures

		Cases	Percent
Family History of Seizures	No	138	92.0%
	Yes	12	8.0%
History of Birth Asphyxia	No	145	96.7%
	Yes	5	3.3%

8% had Family History of Seizures and 3.3% had History of Birth Asphyxia.

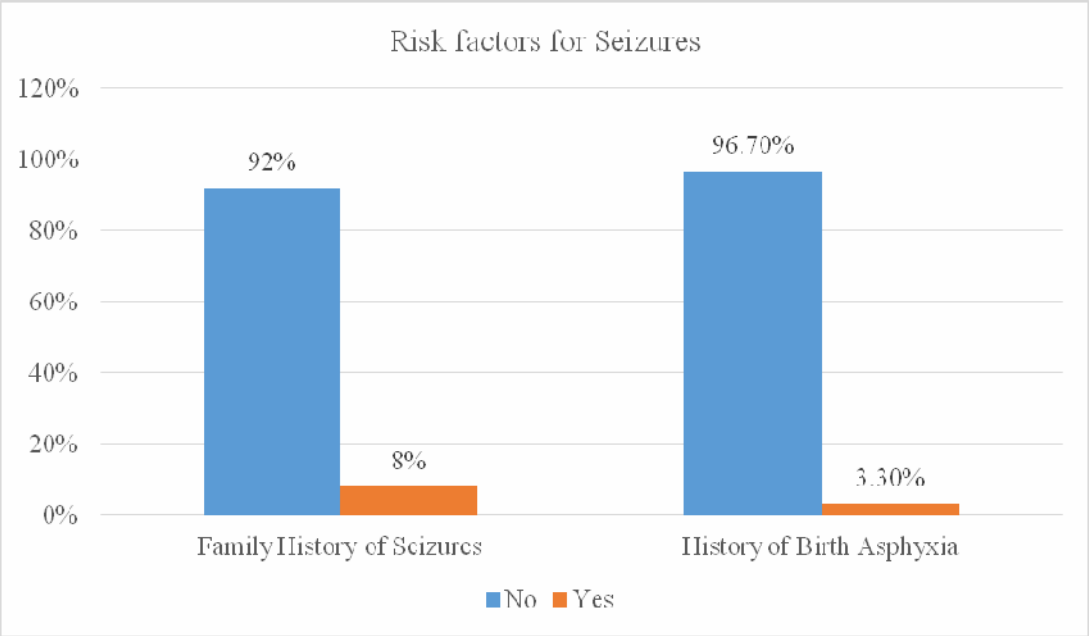

Figure 12: Risk factors for Seizures

Table 14: Neurological manifestations in patients

		Cases	Percent
Head Circumference	Microcephaly	6	4%
	Normal	144	96.0%
Signs of upper motor neuron type ofleison	Absent	144	96.0%
	Present	6	4.0%
Clinical signs of meningitis	Absent	143	94.0%
	Present	9	6.0%

4% had microcephaly, 4% had signs of UMN leison, 6.0% had signs of meningeal irritation.

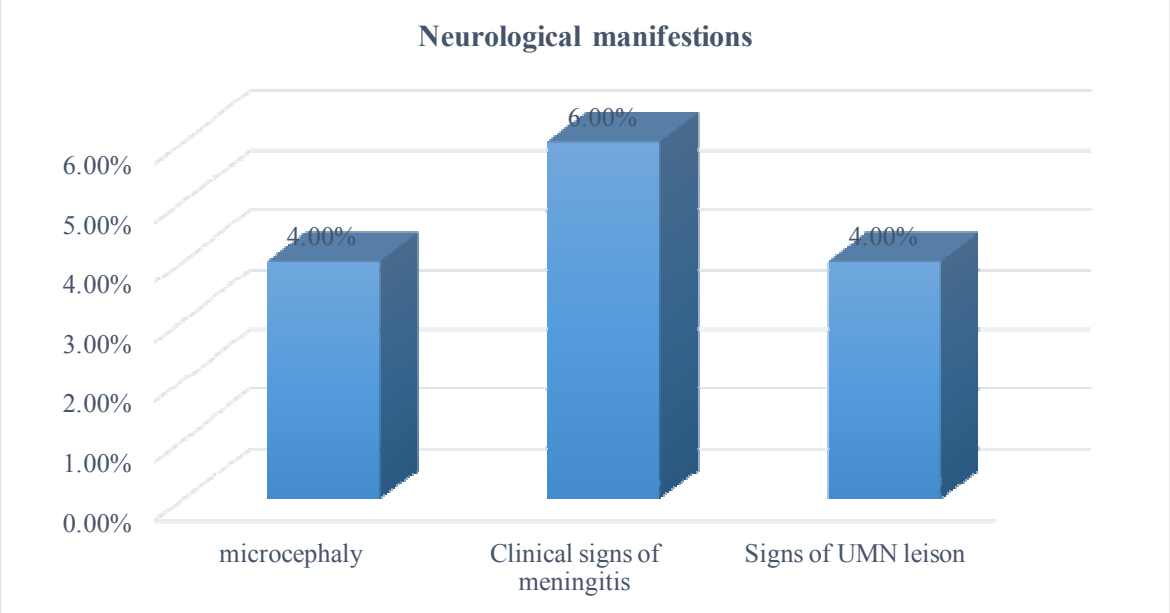

Figure 13: Neurological manifestations in patients

Table 15: Laboratory findings in patients

		Cases	Percent
Malaria Parasite in Peripheral Smear/Rapid diagnostic test	Negative	145	96.7%
	Positive	5	3.3%
Hyponatremia	Absent	149	99.3%
	Present	1	0.7%
Hypocalcemia	Absent	144	96.0%
	Present	6	4.0%
Widal Test Positive	No	148	98.7%
	Yes	2	1.3%
Abnormal CBC	No	127	84.6%
	Yes	33	15.4%
Urine culture Report	Candida	1	0.7%
	E.coli	7	4.7%
	Normal	142	94.7%
Tests for tuberculosis	Normal	146	97.3%
	Hilar nodes present in chest X ray	2	1.3%
	Mantoux Positive	3	2%
	Sputum for AFB	1	0.7%

In the study 3.3% showed malarial parasite in peripheral smear, 0.7% had hyponatremia, 4% had hypocalcemia.

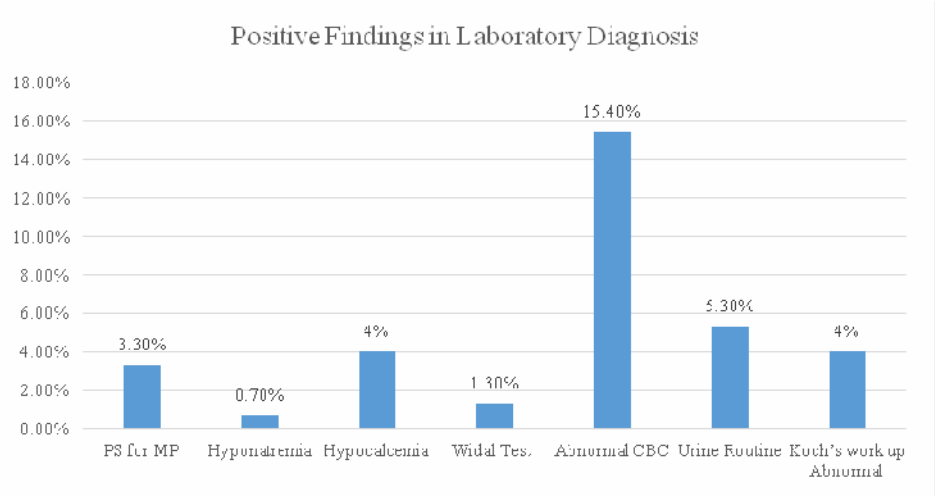

Figure 14: Positive Findings in Laboratory Diagnosis

Table 16: Mean and medium age for hypocalcemic seizures.

Age in months

Hypocalcemia	N	Mean	Std. Deviation	Median	Minimum	Maximum	Range
Present	6	10.32	4.84	10.18	4.07	18.23	14.17

Table 17: CT scan findings in patients with seizures(n= 82)

			Total	
			Cases	Percent
CT scan	Normal		46	56.1%
	Neurocysticercosis	Single lesion	14	17.1%
		Multiple NCC	7	8.5%
	Tuberculoma with perilesional odema		3	3.7%
	Leptomeningeal enhancement in basal cisterns		1	1.2%
	Cerebral edema		7	8.5%
	Cerebral atrophy		2	2.4%
	Bilateral Maxillary Sinusitis		2	2.4%
Total			82	

25.6% showed signs of Neurocysticercosis (Parietal NCC was common site).

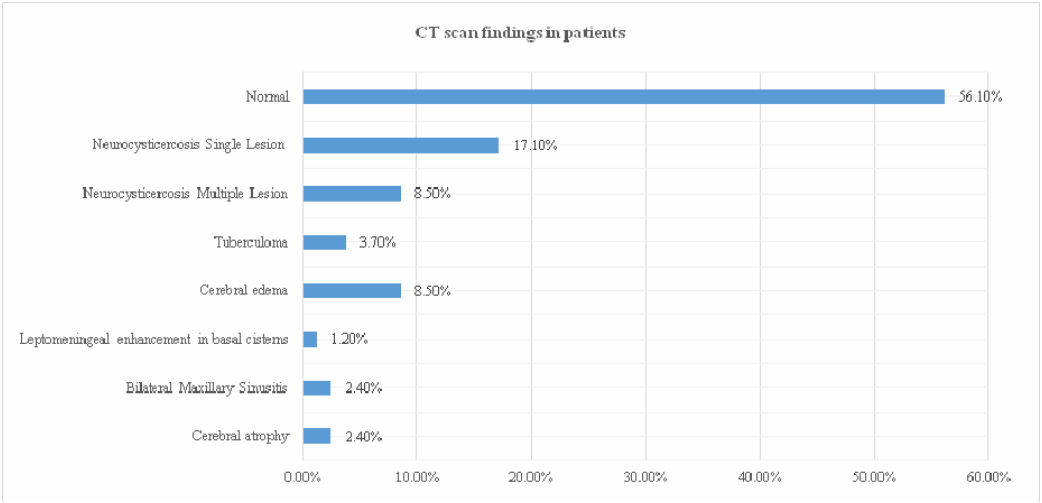

Figure 15: CT scan findings in patients

Table 18: MRI Diagnosis of patients with seizures(n= 25)

			Total	
			Cases	Percent
MRI	Normal		3	12%
	Neurocysticercosis	Multiple Lesion	2	8%
		Single Lesion	11	44%
	Cerebral edema		3	12.0%
	Leptomeningeal enhancement		2	8.0%
	Periventricular Leucomalasia		2	8.0%
	Porencephaly		1	4.0%
	Agenesis of corpus callosum		2	8.0%
Total			25	

52% showed signs of Neurocysticercosis

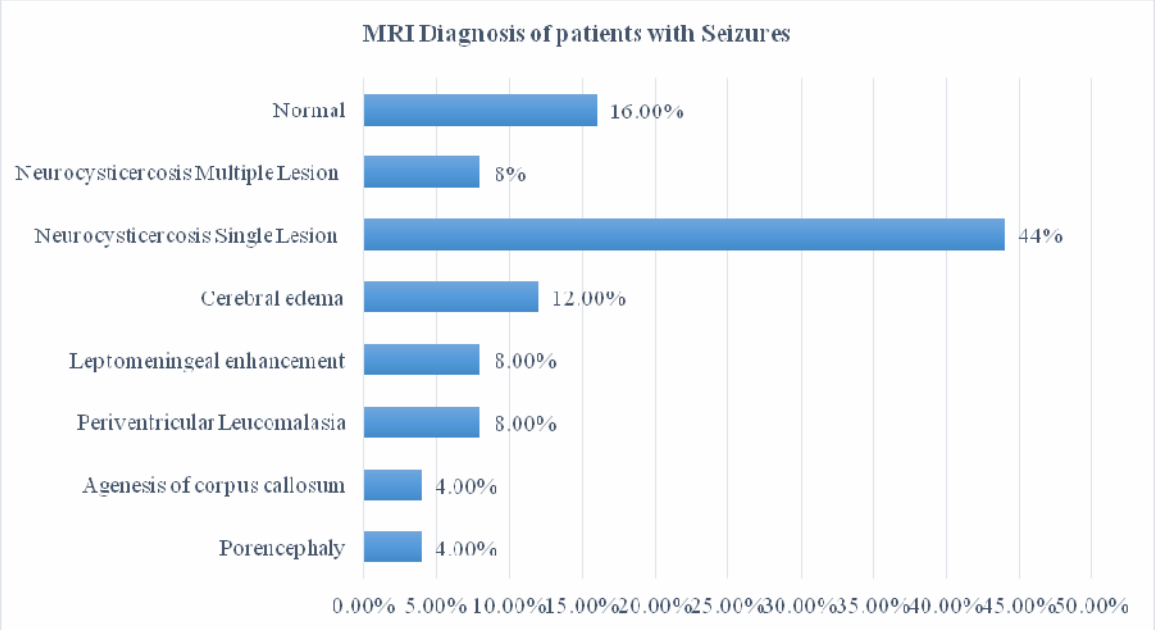

Figure 16: MRI Diagnosis of patients with Seizures

Table 19: EEG Findings in patients with seizures(n=72)

		Total	
		Cases	Percent
EEG	Normal	52	72.2%
	Generalized slowing	1	1.4%
	Generalized Epileptiform Activity	14	19.4%
	Centero-temporal lobe spike wave pattern	2	2.8%
	Epileptiform Activity In Left Fronto-Temporal Lobe	1	1.4%
	Epileptiform Discharges In Bilateral Temporal Lobe	1	1.4%
	Spike Waves with Occipital Paroxysm	1	1.4%
Total		72	

22.2% were abnormal pattern. Generalized epileptiform activity was the most common pattern observed in EEG.

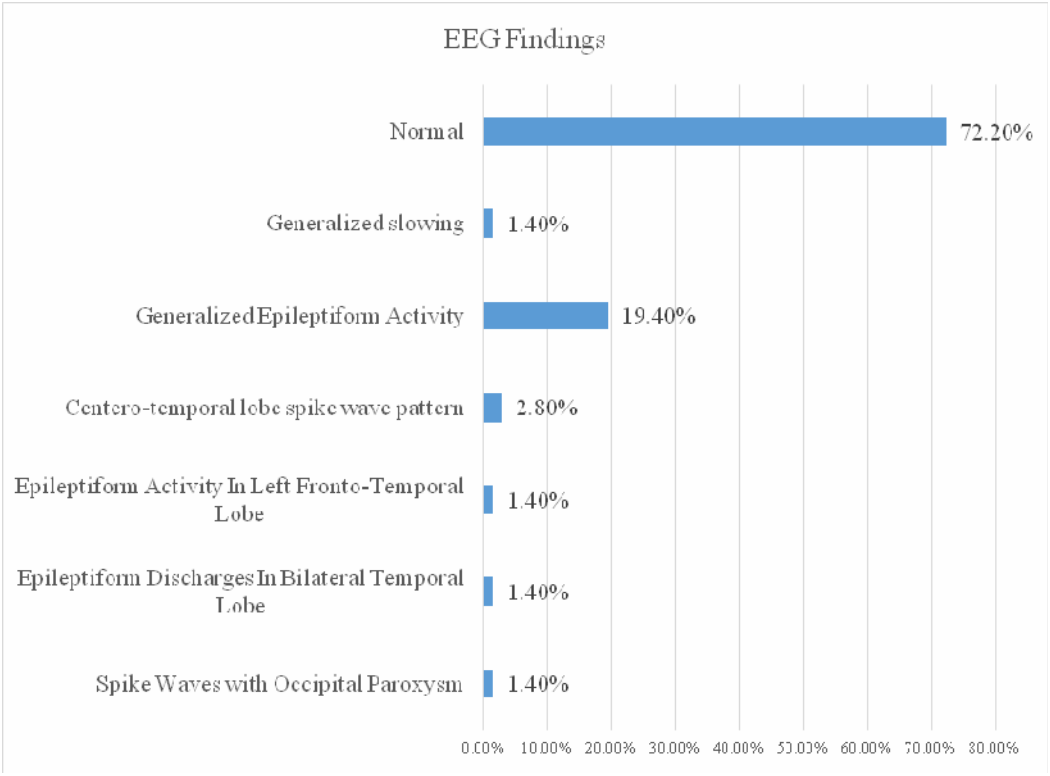

Figure 17: EEG Findings in patients with Seizures

Table 20: Gender distribution of febrile seizure patients(n= 58)

		Sex				Total
		Female	%	Male	%	
Febrile seizures	Simple Febrile Seizures	21	45.7%	25	54.3%	46
	Complex Febrile Seizures	6	50.0%	6	50.0%	12
Total		27	46.6%	31	53.4%	58

Majority (79.3%) patients had simple febrile seizures with male predominance.

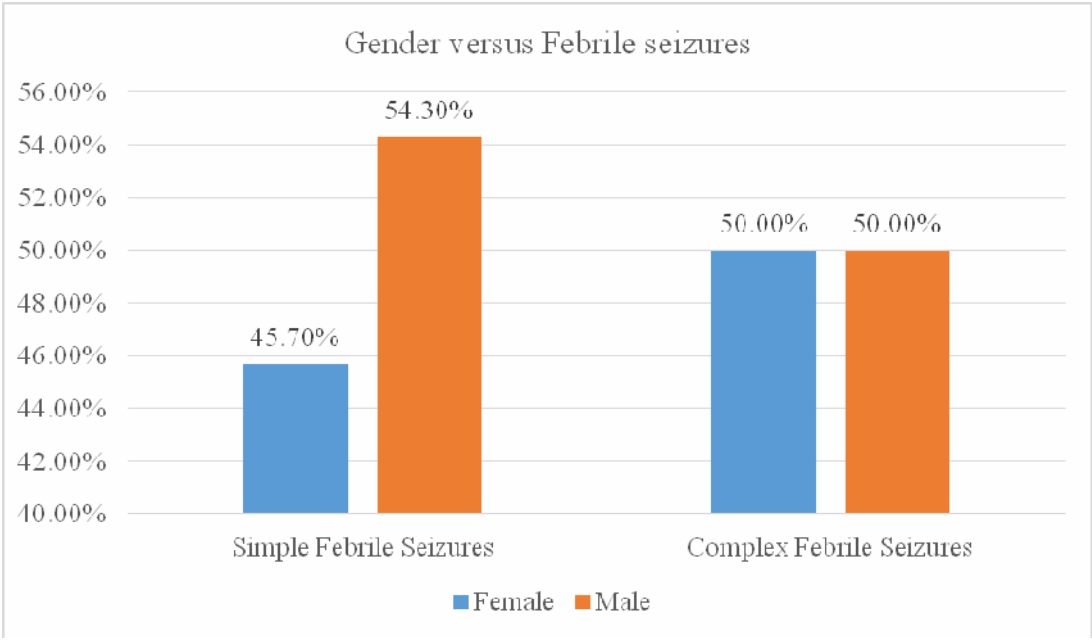

Figure 18: Gender distribution of febrile seizure patients

Table 21:Age distribution of febrile seizure patients

Age in Months						
Febrile seizures	N	Mean	SD	Min	Max	Range
Simple Febrile Seizures	46	25.17	15.53	6.10	59.1	53.00
Complex Febrile Seizures	12	19.77	10.01	8.13	36.50	28.37
Total	58	24.05	14.65	6.10	60.83	54.73

Mean age for febrile seizure was 24.05 months

Table 22:Temperature at admission in febrile seizure patient

Temperature in degree of Fahrenheit							
Febrile seizures	N	Mean	SD	Min	Max	Range	Quartile Range
Simple Febrile Seizures	46	101.65	1.015	100.00	105.00	5.00	1
Complex Febrile Seizures	12	100.58	0.514	100.00	101.00	1.00	1
Total	58	101.43	1.027	100.00	105.00	5.00	1

Mean temperature on admission to hospital in febrile seizure patients was 101.43^0F.

Table 23: Etiological Diagnosis of patients with Seizures

			Total	
			Cases	Percent
Etiological Diagnosis	Febrile Seizures	Simple febrile seizure	46	30.7%
		Complex febrile seizure	12	8.0%
	Infectious Causes	Neurocysticercosis	34	22.7%
		Encephalitis	19	12.7%
		Pyogenic meningitis	7	4.7%
		Tuberculoma	2	1.3%
		Tuberculous meningitis	2	1.3%
		Cerebral Malaria	1	0.7%
	Metabolic Causes	Hypocalemic seizure	6	4.0%
		Hypoglycemic seizures	1	0.7%
		Hyponatremic seizures	1	0.7%
	Cerebral palsy		4	2.6%
	Congenital anomalies		2	1.3%
	Not found		13	8.7%
Total			150	

Infectious causes were the most common causes for seizures in the study (42.7%), followed by Febrile seizures (38.7%) and other causes (Metabolic and CP). In 8.7% Etiological diagnosis was not found.

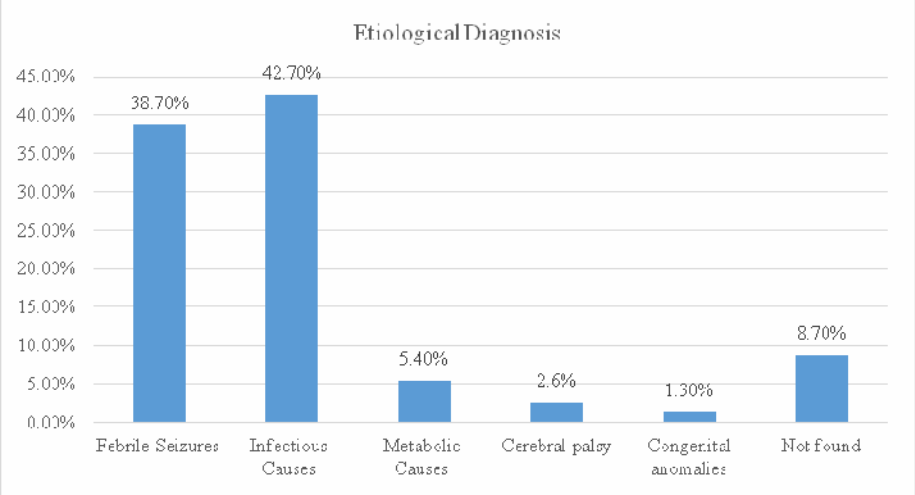

Figure 19: Etiological Diagnosis of patients with Seizures

Table 24: ILAE Classification of seizure types in patients

		Cases	Percent
IIAE Classification of seizure types	Generalized Tonic-clonic	85	56.6%
	Generalized Tonic	25	16.6%
	Simple Partial Seizure	12	8.0%
	Complex Partial Seizure	28	18.6%

Majority of patients (56.6%) had as Generalized Tonic-clonic seizures.

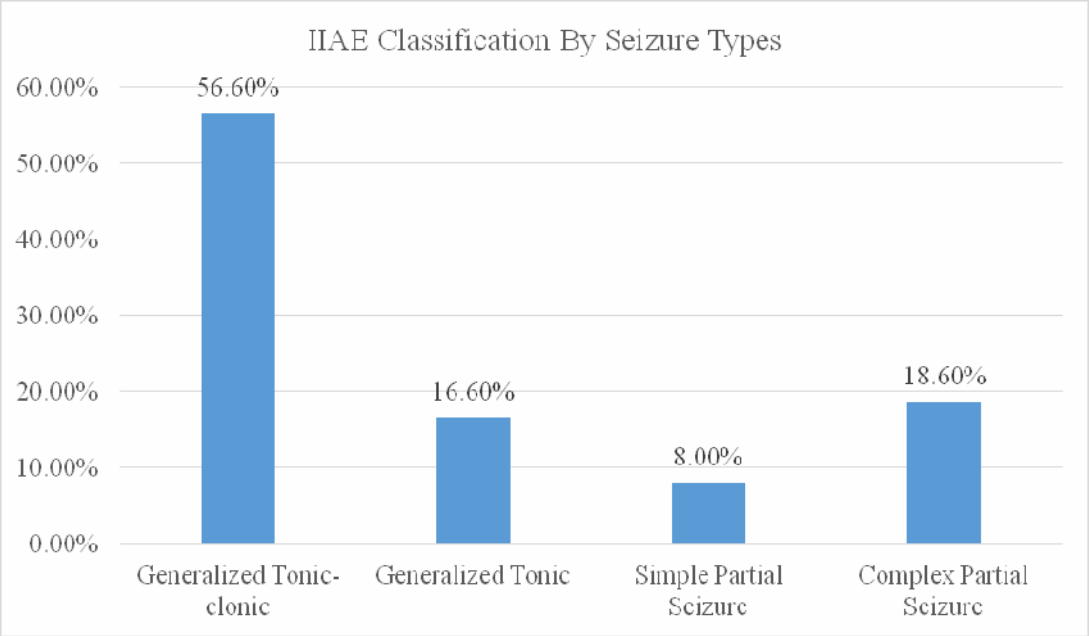

Figure 20: IIAE Classification by Seizure Types

Table 25: Showing ILAE Classification of epilepsies and epileptic syndromes

Epilepsies and Epileptic syndromes	Cases	%	Total
Situation-related seizures			
1. Febrile seizures	58	44.6	
2. Acute symptomatic seizures	67	51.6	130(86.6%)
3. Status epilepticus	05	3.8	
Generalised epilepsy			
1. Idiopathic	09	60	
2. Symptomatic	06	40	15(10%)
3. Cryptogenic	00		
Localisation related epilepsy (LRE)			
1. Idiopathic	03	60	5(3.4%)
2. Symptomatic	02	40	
Total	**150**		**150**

Situation related seizures were the most common (86.6%) in the present study.

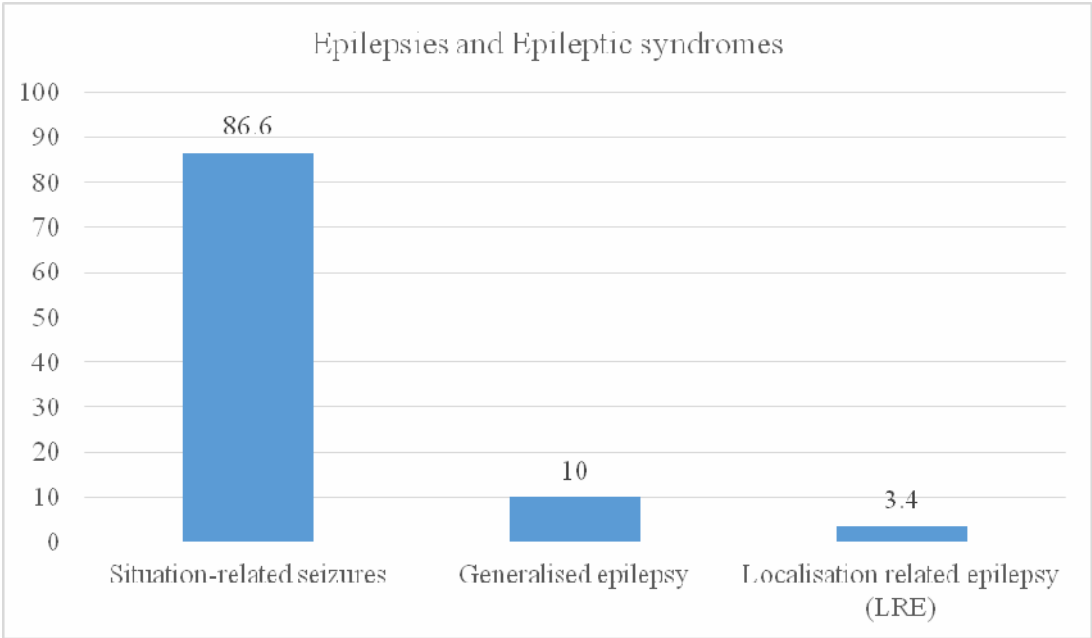

Figure 21: Epilepsies and Epileptic syndromes

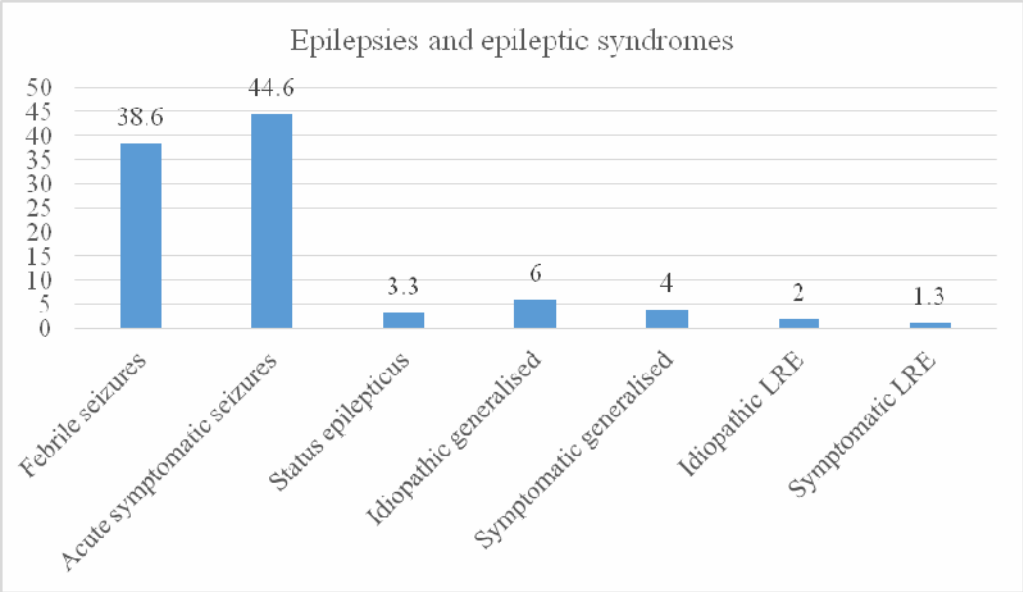

Figure 22: Sub group of ILAE Epilepsies and epileptic syndromes

Table 26: Association between Etiological Diagnosis and Types of seizures

			Types of Seizure		Total
			Partial	Generalized	
Etiological Diagnosis	Febrile Seizures	Simple febrile seizure	0	46	46
		Complex febrile seizure	0	12	12
	Infectious Causes	Neurocysticercosis	34	0	34
		Encephalitis	0	19	19
		Pyogenic meningitis	0	7	7
		Tuberculoma	2	0	2
		Tuberculous meningitis	0	2	2
		Cerebral Malaria	0	1	1
	Metabolic Causes	Hypocalemic seizure	0	6	6
		Hypoglycemic seizures	0	1	1
		Hyponatremic seizures	0	1	1
	Cerebral palsy		0	4	4
	Congenital anomalies		0	2	2
	Not found		4	9	13
Total			40	110	150

$\chi 2 = 51.72$, df = 3 , p <0.001*

Neurocysticercosis was the most common cause for focal seizures and Simple febrile seizure was the most common cause for generalized seizure. This observation was statistically significant.

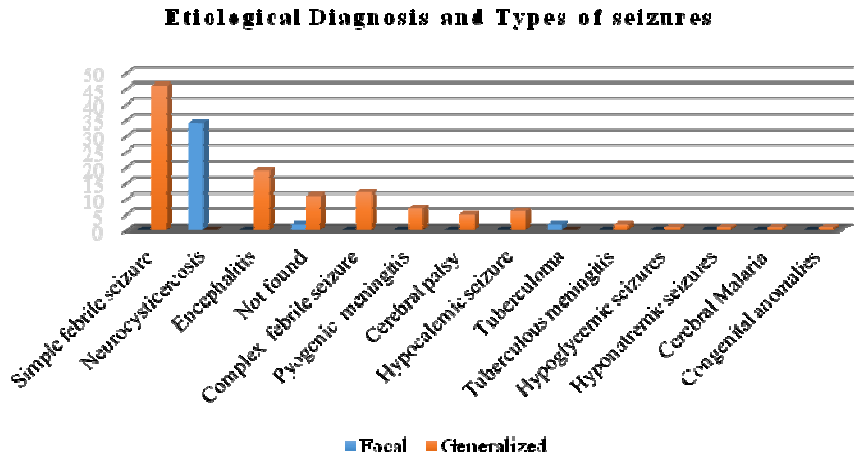

Figure 23: Association between Etiological Diagnosis and Types of seizures

Table 27: Age wise distribution of etiology of seizures

Etiological Diagnosis	Age					
	Infants (< 1 year)		1 to 5 years		6 to 18 years	
	Count	%	Count	%	Count	%
Cerebral Malaria	0	0.0%	1	1.5%	0	0.0%
Cerebral palsy	1	4.5%	3	6.2%	0	0.0%
Complex febrile seizure	2	9.0%	10	15.4%	0	0.0%
Encephalitis	0	0.0%	3	4.6%	16	26.6%
Hypocalemic seizure	4	18.2%	2	3.1%	0	0.0%
Hypoglycemic seizures	0	0.0%	1	1.5%	0	0.0%
Hyponatremic seizures	0	0.0%	0	0.0%	1	1.6%
Neurocysticercosis	0	0.0%	4	6.2%	30	50.0%
Not found	0	0.0%	4	6.2%	9	15.0%
Pyogenic meningitis	4	18.2%	1	1.5%	2	3.3%
Simple febrile seizure	10	45.4%	36	54.5%	0	0.0%
Tuberculoma	0	0.0%	0	0.0%	2	3.3%
Congenital anomalies	1	4.5%	1	1.6%	0	0.0%
Tuberculous meningitis	0	0.0%	0	0.0%	2	3.2%

Pearson Chi-square value = 132.2, df = 26, p = <0.00001

There was significant association between etiological diagnosis and age. I.e. in < 1 year infants & 1 to 5 years most common etiology was Simple febrile seizures 45.4% & 54.5% respectively. In 6 to 18 years age group most common etiology was Neurocysticercosis (50.0%).

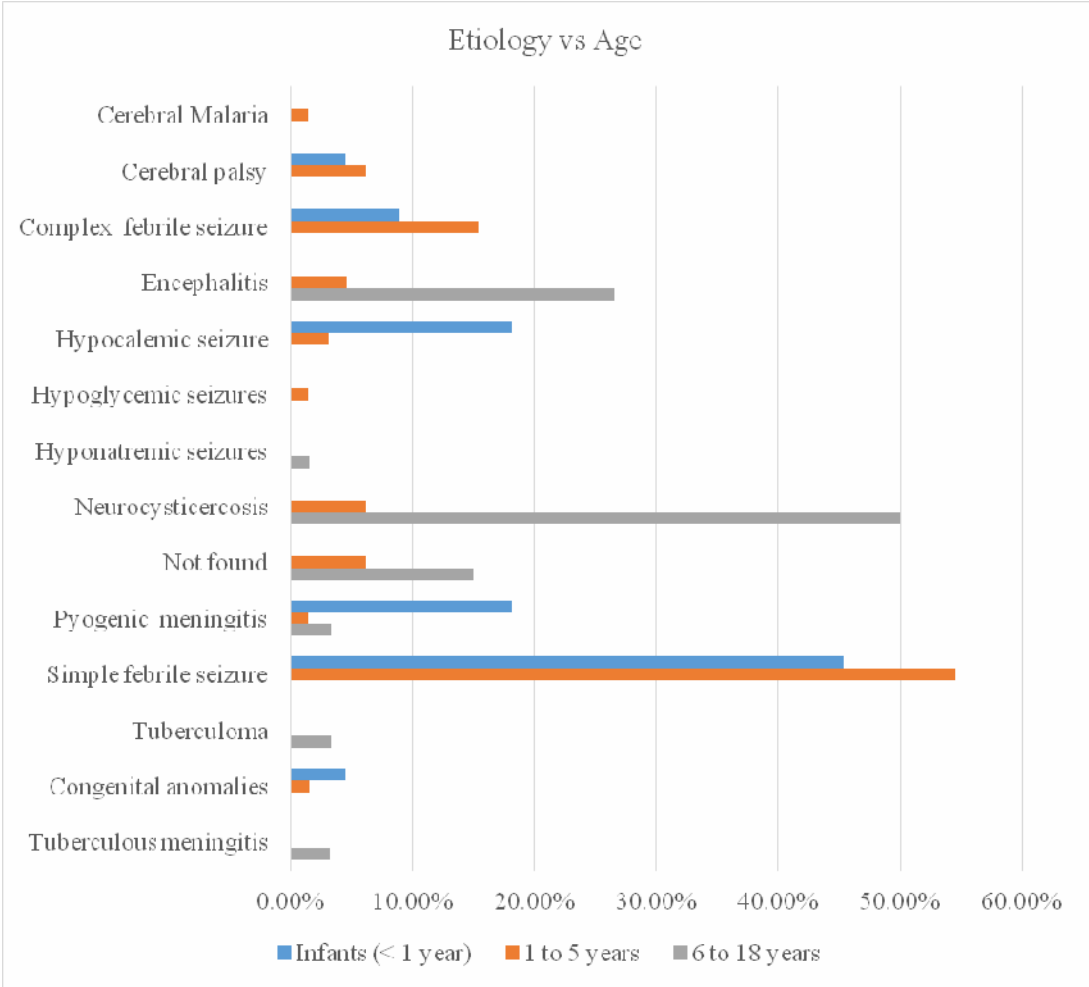

Figure 24: Age wise distribution of etiology of seizures

Table 28:Age wise distribution of type of seizures

IIAE Classification By Seizure Types	Age						P value
	Infants (< 1 year)		1 to 5 years		6 to 18 years		
	Count	%	Count	%	Count	%	
Complex Partial Seizure	1	4.5%	4	6.2%	23	37.3%	<0.001*
Generalized tonic	6	27.3%	13	19.6%	6	9.6%	
Generalized Tonic-clonic	15	68.2%	47	71.2%	23	37.0%	
Simple Partial Seizure	0	0.0%	2	3.0%	10	16.1%	

Pearson Chi-square value = 39, df = 6, p = 0.001

There was significant association between ILAE classification and Age. I.e. In all age groups most common seizure was generalized Tonic-clonic.

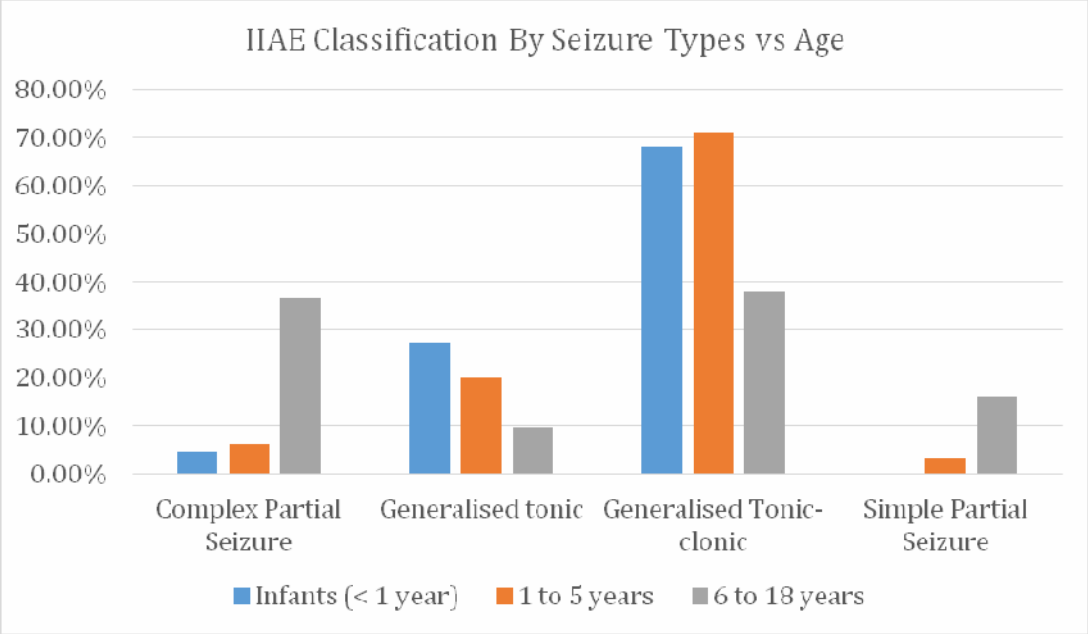

Figure 25: Wise distribution of type of seizures

Table 29: Age wise distribution of epilepsies and epileptic syndromes .

Syndromes	Age						P value
	Infants (< 1 year)		1 to 5 years		6 to 18 years		
	Count	%	Count	%	Count	%	
Acute Symptomatic Seizure	8	36.4%	10	15.2%	49	79.1%	
Complex Febrile Seizure	2	9.1%	10	15.2%	0	0.0%	
Idiopathic Generalized Epilepsy	0	0.0%	2	3.0%	7	11.3%	
Idiopathic LRE	0	0.0%	1	1.5%	2	3.2%	
Simple Febrile Seizure	10	45.5%	36	54.6%	0	0.0%	<0.001*
Symptomatic Generalized Epilepsy	2	9.0%	4	6.0%	0	0.0%	
Symptomatic LRE	0	0.0%	1	1.5%	1	1.6%	
Symptomatic Status Epilepticus	0	0.0%	2	3.0%	3	4.8%	

Pearson Chi-square value = 83.96, df = 14, p = <0.001

There was significant association between IIAE Classification of Epileptic Syndromes and Age. I.e. in infants simple febrile seizures were most common and >1 years age Acute symptomatic seizures were common.

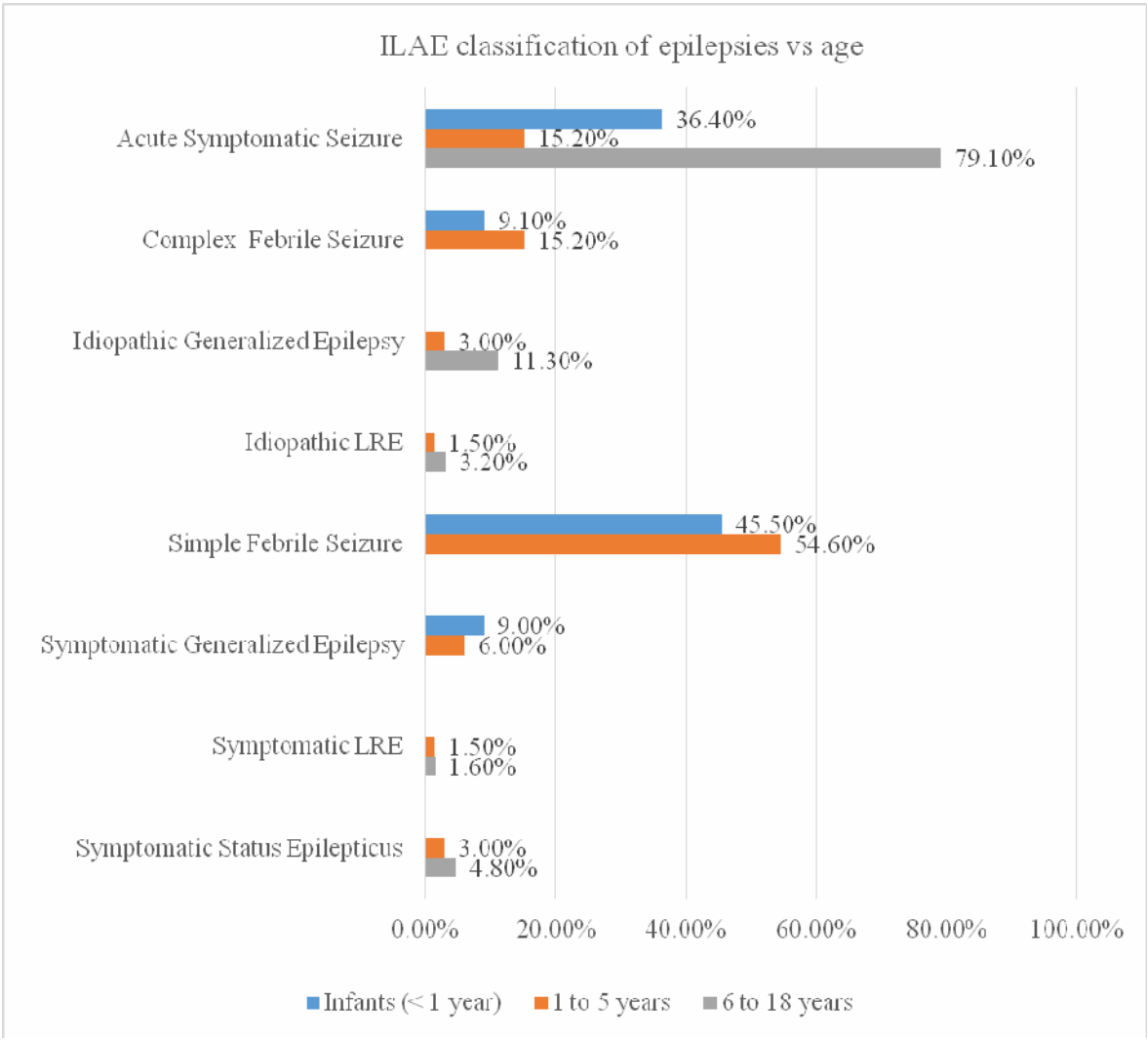

Figure 26: Age wise distribution of epilepsies and epileptic syndromes

Table 30:Anticonvulsants used in treatment of patients with Seizures

			Total	
			Cases	Percent
Anti – Convulsant used	None		5	3.3%
	Calcium		6	4.0%
	Clobazam prophylaxis		58	38.6%
	Monotherapy	Carbamazepine	33	22.0%
		Valproate	22	14.7%
		Levitiracetam	1	0.7%
		Phenytoin	12	8.0%
		Phenobarbitone	9	6.0%
	Polytherapy	Phenytoin and phenobarbitone	3	2%
		Multiple anticonvulsants (phenytoin,phenobarbitone and valproate)	1	0.7%
Total			150	

Clobazam prophylaxis was used in all patients with febrile seizures

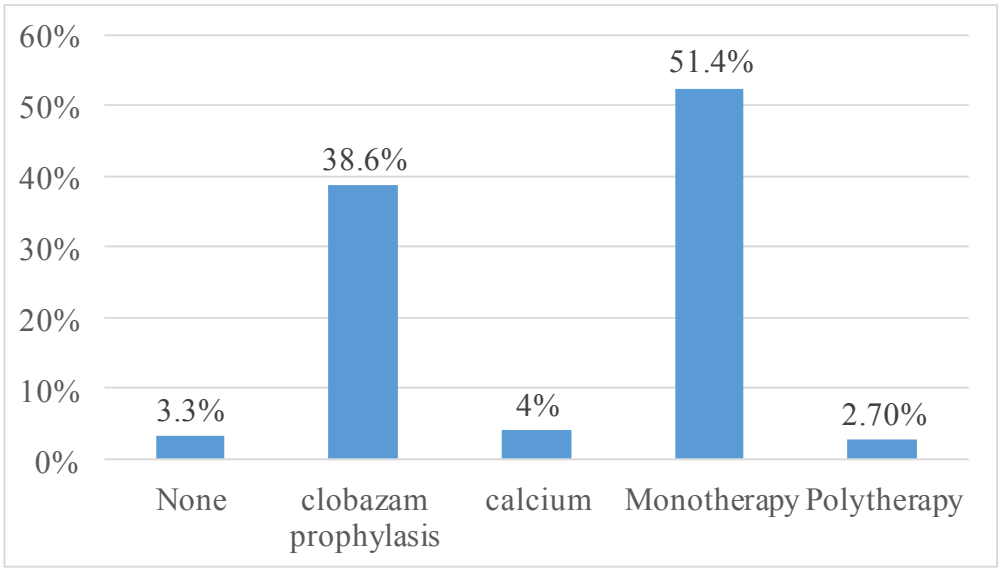

Figure 27: Anticonvulsant therapy in patients with seizures

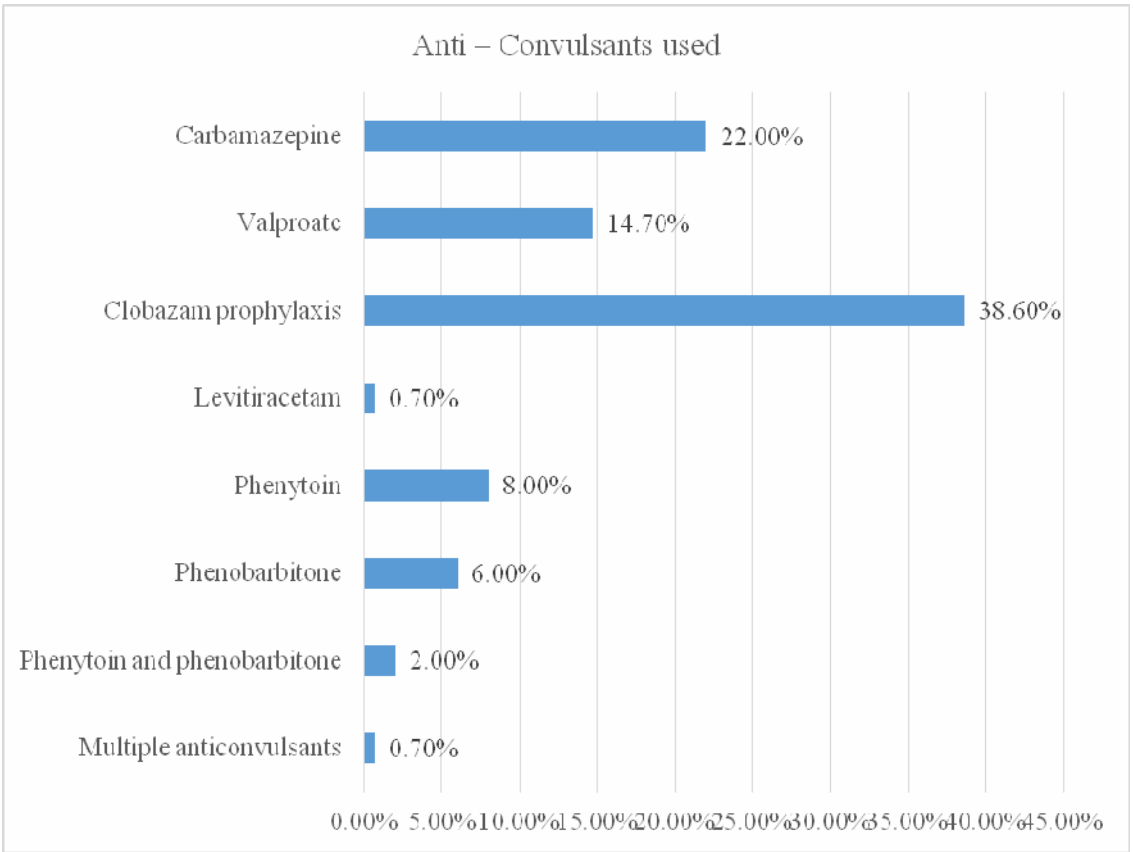

Figure 28: Anti – Convulsants used in patients with seizures

DISCUSSION

During the 1 year study period, total of 150 patients were analyzed. Distribution of cases according to age, was done into 3 groups - <1 year(infants),1-5 years(toddlers and preschool children),6-18 years(school going and adolescents).Out of 150 patients, 22 (14.7%) patients were between age group 1 month to <1 years age ,66 (44%) patients between 1 to 5 years and 62 (41.3%) patients were from age group 6 year to 18 years.Mean age of onset of seizure was 5.19 ± 4.37 years , Median age of onset of seizure was 4 years. It suggests that onset of seizures is more in between 1-5 years of life. These findings were consistent with the study conducted by **Chen C Y et al**[20], they found onset of seizures is maximum between 1-6 years.In a study done by **Metsarnata P et al** in 2004, mean age of onset of seizure was 4.5 ± 3.10 years.[38]

Female patients were 77 (51.3%) and males were 73 (48.7%) out of 150 patients studied. Females were more in number than male patients, which suggest that seizure disorders were more common in females than males (female:male= 1.05: 1). These findings were consistent with studies conducted by **Metsarnata P et al**[38] where female to male ratio was 1.08:1,**Sidenvall et al** [39]in year 1993 where male: female ratio was1.1:4, **Mathai et al**[40] in 1969 (female: male 1.4:1) and **Koul R et al**[41] in Kashmir in 1988.

Out of 20 cases with epilepsy, 4 (20%) cases were born to consanguineously married couple. This indicates that consanguinity has a role in occurrence of epilepsy. Family history of seizures was found in 12 (8%) out of 150 cases. History of seizure was present in siblings in 4 (33%) cases and in cousins in 8 (67%) cases out of 12.These findings correlate with study conducted by **Ramasundrum et al** in year 2004 in Indian origin patients in Malaysia who found that 29.5% of cases of epilepsy had a parental consanguineous marriage. [42]

Duration of seizure episode in 136(90.7%) lasting less than 14 minutes, in which maximum were febrile convulsions occurring for short duration, 9 (6%) cases had duration of seizures from 15 to 29 minutes, 5(3.3%) cases had seizure duration >30 minutes.

In our study mean duration of seizure episode was 8.5 min with minimum 1 minute and maximum 60 minutes. This significantly correlates with the etiology, that was shortest in febrile convulsion (1 minute) and longest duration in acute symptomatic seizures and status epilepticus (SE) (60 minutes). These findings were consistent with the study carried out by **Metsarnata Pet al** in 2004 found that mean duration of seizure episode was 16 minutes with maximum duration 88 minutes in symptomatic group. [38]

Majority of children 67(44.7%) had single episode of convulsion and 31(20.7%) had multiple convulsions. In post-ictal state, consciousness was intact in 88 (58.7%) cases and 6(4%) were unresponsive.

87(58%) patients had fever in the present study population. These findings were consistent with study done by **SudhirAdhikari et al** in Nepal, who found that 53.3% of cases with seizures had fever in their study population .[26]

Out of 150 cases studied, history of birth asphyxia was present in 5 (3.3%) and low developmental quotient was found in 6 (4.0%), which includes 4 (66.6%) females and 2 (33.3%) males. Out of 6 cases with low DQ , 4cases had cerebral palsy, 1 had simple febrile seizure, 1 had agenesis of corpus callosum and 1 had porencephaly. Microcephaly was found in 6 (4.0%) cases, which include 4 cases of cerebral palsy and 1cases with agenesis of corpus callosum and 1 case of porencephaly.

Neurocutaneous markers were found in 2 cases and dysmorphic facial features were found in 2 (1.3%) cases out of 150, which include low set ears and ear tag in both

patients. In all of these cases no significant syndromic association was found on evaluation.

On central nervous system examination of 150 cases studied, signs of UMN type of lesion were found in 6 (4%) , clinical signs of meningitis in 9 (6.0%) (5 females and 4 males), scissoring sign in 2 (1.3%) and decerebrate posturing in 3 (2%) cases. All cases with meningitis were associated with high grade fever. Fundus examination was abnormal in 4 (2.6%) cases of 150 cases, which includes bilateral papiloedema in all 4 cases.

Abnormal blood reports were found in 33 (22%) cases of 150 patients. These cases include 9(27.2%) cases of meningitis, 8 (24.2%) cases of UTI, 5 (15.1%) cases of malaria, 2 (6%) cases (Widal test positive) of enteric fever.

Out of 46 CSF samples analyzed, abnormal CSF findings were found in 21 (14%), which includes 2 (9.5%) cases of tuberculous meningitis, 7 (33.3%) cases of pyogenic meningitis and 12(57.1%) cases of encephalitis. Thus, CSF analysis was helpful in diagnosing the above conditions.

Cranial CT scan was done in 82 cases, out of which 36 (43.9%) were abnormal.These findings were consistent with **Murthy et al** ,who found that brain lesions were detected in 50% cases of total CT brain scans done, in their study.[43]The most common brain lesion detected on CT scan was NCC (21 in 36 cases;58.3%).

MRI brain was done in 25 (16.6%) cases out of 150. It was abnormal in 22 (88%) cases. The most common brain lesions detected were neurocysticercosis (NCC) in 13 (59.0%) cases.

In our study 58 (38.6%) cases out of 150 had abnormal brain images. These findings were consistent with study by **Sudhir Adhikari et al**, in which abnormal brain images were noted in 111 (45.9%) of 242 patients and most common

abnormality was neurocysticercosis in 66 (59.5%) cases.[26]Thus, neuroimaging was helpful in diagnosing NCC.

EEG was done in 72 cases, out of which 20 (27.7%) were abnormal. Most common EEG finding was generalised epileptiform activity, seen in 15 (75%) cases. Epileptiform foci localised to one or more hemisphere of brain was reported in 5 (25%) cases.

Out of 150 cases, etiological diagnosis was possible in 137 cases only and in 13 cases etiology was not found. Overall, the most common etiology was infectious cause, which was found in 123 (89.7%) cases. Other etiological causes were metabolic causes in 8 (5.8%) cases, perinatal insult 4 (2.6%) cases . These finding were consistent with **Feyzullah et al,** who found that the most common etiology was infectious followed by hypocalcemia and perinatal brain damage.[44]

Febrile seizures were seen in 58 (38.6%) of 150 cases, of which 27 (46.6%) were female and31 (53.4%) were male patients. Maximum cases of febrile seizures were in second year of life. Minimum age was 6 months and maximum age was 5 years with mean age 24.05 months. These findings were consistent with the studies done by **Abolfazl et al** in year 2007 [45] and **Nguefack S et al**[23]. They found that maximum cases of febrile seizures were from second year of life.

In our study, the mean temperature at admission to hospital in febrile convulsion patients was 101.43°F ± 1.027°F. The minimum temperature noted was 100°F and maximum temperature noted was 105 °F. Maximum number of patients had temperature between 101 to 102°F which includes 42(72.4%) cases out of 58. These findings were consistent with the study done by **Nguefack S et al**, who found that mean temperature for febrile convulsion at admission was 39.2°C which approximately equals to 102°F. [23]**Abolfazl et al** found that the mean temperature, at

admission for febrile convulsions, was 38.9°C ± .37°C which approximately equals to 101 to 102°F.[45]

Out of 58 cases of febrile convulsion, 46(79.3%) cases were simple febrile convulsions and 12 (20.6%) cases were complex febrile convulsions. This indicates that simple febrile convulsions were the most common type in present study. These findings were consistent with the study conducted by **Freedman SB et al** in year 2003, who found that simple febrile convulsions were present in 80% cases of study group. [46]

Among febrile seizure patients, 45(77.5%) had upper respiratory tract infection (URTI),8 (13.7%) cases had urinary tract infection (UTI) , 4 cases (6.8%) had malaria and 2(3.4%) had enteric fever. UTI was more common in female children. These findings were consistent with study done by **Abolfazl et al** in year 2007, who found that main etiology of febrile seizures was upper respiratory tract infection in 53.8% cases, acute gastroenteritis (AGE) in 24.4% cases and UTI in 6.4% cases of study group. [45]**Nguefack S et al** found that main leading etiology was malaria in 67.7% cases and upper respiratory tract infection in 24% cases of febrile seizures. [23]

Out of 137 cases in which etiology was identified, upper respiratory tract infection (URTI) was most common infection present in 45 (32.8%) cases. In URTI, there was sudden rise in temperature causing febrile seizure mostly simple febrile seizures.

Second most common infection was neurocysticercosis (NCC) which was found in 34 (24.8%) cases. Now a days, NCC is a leading cause of convulsion in India and in all developing countries, as it is a disease of poverty and underdevelopment and diagnosis by neuroimaging and serological assessment has greatly improved over the

past decade. **Kumar Garg R et al** found that NCC is a leading cause of convulsion in India.[47]

Encephalitis was present in 19(13.8%) cases and malaria in 5(3.6%). This includes 4 (80%) cases of P. Vivax malaria which lead to febrile seizures and 1 (20%) case of P. Falciparum malaria. P.falciparum lead to cerebral malaria.**Nguefack et al** found that malarial cases were leading to febrile convulsions in 50% cases of study group.[23]

Pyogenic meningitis was one of the infectious causes, which was present in 7 (5.1%) cases. These findings were consistent with study by **Joffe et al ,**who found that pyogenic meningitis was present in 4.56% of study group. [48]

Among the Metabolic causes, hypocalcemia was the leading cause seen in 6(4.3%) cases. Hypocalcemia lead to acute symptomatic seizures, which includes 4 cases from age less than 1 year and 2 cases from 1-5 years of age. Mean age of patients with hypocalcemic seizures was 10.32 months ± 4.84 months (minimum age 4.07 months and maximum age 18.23 months).These findings were consistent with the study conducted by **Feyzullahet al** found that hypocalcemic convulsions were occurring with mean age 6.3 months ± 5.9 months. [44]

Cerebral palsy (CP) was seen in 2.6%. In MRI brain, 2 (33.3%) patients had periventricular leucomalacia (PVL).All cases of CP had symptomatic generalized epilepsy and mental retardation. These findings were consistent with the study done by **Fukuda K et al** found that cases of cerebral palsy had recurrent symptomatic seizures and also, associated with mental retardation. [49]

In the present study, patients were classified according to ILAE classification, as seizure types and epilepsy and epileptic syndromes. [(8)]

ILAE Classification by seizure type was done in 150 cases, most common type was generalized which includes generalized tonic clonic (GTC) in 85 (56.6%) cases (39 females and 46 males), generalized tonic (GT) in 25 (16.6%) cases. These findings were consistent with study conducted by **P Alizadeh et al** in 2008 who found that most common seizure type in study group was generalized seizure type in 78% cases. They also found that GTC occurred in 36 % cases and GT seizure type in 16% cases.[22] Similar results were also seen in study conducted by **Shakya KN et al**. [50]

Partial seizures were seen in 40(26.6%) of 150 cases, out of which 12 (30%) cases were simple partial seizures and 28 (70%) were complex partial seizure (CPS).In a study conducted by **Sudhir Adhikari et al**, they found that partial seizures occurred in 19.7% cases.[26] In a study done by **P Alizadeh et al** in 2008, partial seizure type was found in 22% cases.[22]

The ILAE classification of Epilepsy and Epileptic syndromes includes three categories that are 1) Localisation related epilepsies 2) Generalized epilepsies and 3) Situation related seizures.

The most common category was situation related seizures, present in 130 (86.6%) cases, followed by generalised epilepsy in 15 (10%) cases and localisation related epilepsy in 5 (3.4%) cases out of 150.Acute symptomatic seizures were the most common seizure type in situation related seizures in present study.

Of 130 cases of situation related seizures, febrile seizures were present in 58 (44.6%), acute symptomatic seizures in 67 (51.6%) and status epilepticus in 5 (3.8%) . Status epilepticus (SE) includes all 5 cases of symptomatic SE . Of the total 150 cases, 6 (4%) cases were symptomatic generalized epilepsy, 2(1.3%) cases were symptomatic localization related epilepsy (LRE), 9 (6%) cases were idiopathic generalized epilepsy, 3 (2%) cases were idiopathic LRE. In a study done by **Feyzullah**

et al in year 2004, situation related seizures were present in 75.6% cases; symptomatic generalized epilepsy were present in 18.6% and cryptogenic epilepsy were present in 5% patients.[44] In another study conducted by **P Alizadeh et al** found that febrile seizures were most common seizures found in 59.25% cases of study population. They also found that situation related seizures were present in 45% cases, symptomatic epilepsy in 36% cases, idiopathic seizures in 10% cases and cryptogenic epilepsy in 9% cases of study group.[22]

Of 20 cases with epilepsy, generalized epilepsies were found in 15 (75%) cases.9 (45%) cases were idiopathic generalized epilepsies, 6 (30%) cases were symptomatic generalized epilepsies. This seizure type was more common in age group>1 years. This is a statistically significant finding. (p=0.001). In a study conducted by **Dura Trave et al** in 2005, symptomatic generalized epilepsy present in 25% cases, cryptogenic epilepsy in 29% cases and idiopathic generalized epilepsy in 44.5% cases of study group.[13]**Oka et al** found that, generalized epilepsy was present in 44% patients of study group.[51]**Murthy et al** found that, symptomatic generalized epilepsy present in 48% cases, idiopathic generalized epilepsy present 3% cases and cryptogenic generalized epilepsy present 49% cases in study group.[43]**Sidenvall et al** found that, symptomatic generalized epilepsy in 42% cases, idiopathic generalized epilepsy in 28% cases and cryptogenic generalized epilepsy in 30% cases of study group.[38]

In our study ,localisation related epilepsies (LREs) were found in 5 (25%) cases out of 20 cases of epilepsy. Of 5 cases, symptomatic LREs were present in 2 (40%) cases and idiopathic LREs in 3 (60%) cases. All these cases were from age >1year. This is a statistically significant finding(p=0.001). In previous studies done on seizure disorder suggest that LRE were leading cause in the study group. These

studies were, **Yukiyoshi et al** found that, LREs present in 55% patients including 41% cases of symptomatic LREs and 59% cases of idiopathic LREs.[52]**Murthy et al** found that, LREs present in 48% patients in study group.[43]**Oka et al** in year 2006 found that LREs present in 76% patients in study group.[51]

Anticonvulsant monotherapy was used in 77 (51.4%) cases and 4 (2.7%) required polytherapy to control seizures. Clobazam was used as a prophylaxis for febrile seizures in 58(38.6%) cases. Carbamazepine was used in 33(22%) cases and valproate in 22 (14.7%) cases out of 150. Intravenous Calcium Gluconate was given in 6 (4%) cases.

CONCLUSION

Seizure disorders were more common in female children than male children. Onset of seizures was maximum in between 1-5 years of age. The most common clinical presentation of seizures was generalized tonic-clonic type. According to ILAE classification, the most common group found was situation related seizures in which, acute symptomatic seizures were the most common type in present study.The most common epilepsy was idiopathic generalized epilepsy. The most common aetiology was infectious. Febrile seizure was the most common etiology below 5 years and Neurocysticercosis in the age group 6-18 years. Among the metabolic causes, hypocalcemia was the most common etiology, majority of cases occurring in infancy.

The current study was conducted over short time period in a single hospital setup. But the studies on large scale over longer duration should be carried out, to put forth the diagnostic guidelines and management plans on the basis of clinical presentation and etiological causes in a specific age group. This will not only be helpful to decrease the percentage of misdiagnosis of seizures but also will aid in giving appropriate treatment, which now a days, a major pitfall of seizure control in our country.

SUMMARY

The prospective observational thesis work was conducted to study the clinical and etiological aspects of seizure disorders in 150 cases during December 2013 to January 2015 in a tertiary care hospital, in a district .

- In current study, female patients outnumbered male patients (female: male 1.05:1). Thus, females were more susceptible to seizure disorders than males. The mean age of onset of seizure in study group was 5.19 ± 4.37 years.The age group at which maximum onset of seizures found was between 1-5 years of life.
- The most common clinical presentation of seizures was generalized tonic-clonic type (56.6%).
- The most common aetiology was infectious (66.1%), in which the most common was upper respiratory tract infection followed by neurocysticercosis .Among metabolic causes, hypocalcemia was most common cause.
- Febrile seizure (65.9%) was the most common cause of seizure below 5 years of age and Neurocysticercosis (50%) in the age group 6-18 years.
- According to the International League Against Epilepsy (ILAE) classification, the most common group found was situation related seizures (86.6%).
- Idiopathic generalized epilepsies (45%) were most common type of epilepsy
- Febrile convulsions accounted for 38.7% cases in present study. Simple febrile convulsions (79.3%) were more common than complex febrile convulsions (20.6%).Mean temperature on admission to hospital in febrile seizure patients was 101.43^{0}F.Mean age for febrile seizure was 24.05 months.The most common etiology for febrile seizure was upper respiratory tract infection followed by UTI.

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ANNEXURES

PROFORMA

TITLE :TO STUDY THE CLINICO ETIOLOGICAL PROFILE OF SEIZURES IN KOLAR

Serial No.

Name

Age Sex Religion

Address

Informant

Date of admission

Complaining of

1. Convulsion
2. Fever

H/O present illness :

Convulsion

Aura: Sensory Motor Autonomic

Time of Day

During sleep Playing

Generalized / Focal

Tonic-clonic Tonic Clonic

Uprolling of eyeballs Y/N Frothing from mouth Y / N

Vomiting Y/N Bowel/bladder incontinence Y /N

Duration of convulsion minutes

Loss of consciousness Y/N Duration

Associated cyanosis Y/N

Postictal- Unconsciousness/ irritability/ Drowsiness/Todd's palsy

Focal neurological deficits: Y/N

Fever : High / moderate / mild. Continuous / intermittent with chills / rash

History of ear discharge/ head injury/recent vaccination

PAST HISTORY

Tuberculosis / TB contact

Hypertension / Diabetes / Psychiatric illness

BIRH HISTORY

Preterm /Near term/Term Normal Delivery / Assisted Delivery/ Caesarian section

IInd stage of labor - Normal/Prolonged.

Baby cried immediately after birth Y/N Required resuscitation

Birth Weight NICU admission

If yes, Diagnosis: Sepsis / MSAF / MAS / RD / Seizure / Hyperbilirubenemia / exchange transfusion / hypoglycemia

Mother - ANC Complications: PIH / Seizure / anticonvulsants

IMMUNIZATION Complete / incomplete

DIET Adequate / Inadequate

DEVELOPMETAL HISTORY

Before onset of seizure After onset of seizure

Gross motor

Fine motor

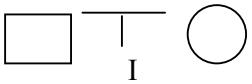
Language

Social

Bladder / bowel control

Appropriate for age-Y/N. School going-Y/N. Behavioral problems-Y/N

FAMILY HISTORY

Consanguinity Y/N  I II III

Family H/O - Epilepsy/Febrile seizure/ CP / MR / Ataxia/ Neurocut.Syn

Family H/O - Similar complaints Y/ N starting age Frequency of Episodes

Diagnosis Any anticonvulsant/medication

GENERAL EXAMINATION

Conscious Y/N

Febrile / afebrile Temperature

Pulse rate Respiratory rate

BP

Pallor Icterus Cyanosis Clubbing Edema

Lymphadenopathy

BCG Scar

Throat

Spine

Skull shape

Neurocutaneous markers :

ANTHROPOMENTRY

Wt.

Ht.

Head Circumference Birth Present

SYSTEMIC EXAMINATION

CNS-Higher functions

Level of consciousness GCS

Handedness Rt/Lt

Gait normal / spastic / cerebellar / waddling / high stepping

Language Memory

Cranial nerves

Olfactory

Optic : pupils - field - acuity of vision - color vision

III, IV, VI : ocular movements nystagmus ptosis diplopia

V : sensory motor jawjerk

VII : sensory motor palsy - Rt / Lt UMN / LMN

VIII : vestibular

IX, X, XI : uvula movement gag reflex palatal movement.

XII :

Motor system

Limb position

Bulk

Power (Proximal/distal)

Tone

REFLEXES

Superficial reflexes	Rt	Lt
Corneal		
Conjunctival		
Anterior Abdominal Wall		
Cremastric		
Plantar		

Deep tendon reflexes

	RUL	**LUL**	**RLL**	**LLL**
Biceps		Knee		
Triceps		Ankle		
Supinator				

Clonus

sensory examination:

Involuntary movements:

Primitive reflexes:

Cerebellar signs – Dysmetria/dysdiadokokinesia / intentional tremers / Ataxia

Autonomic dysfunction

Signs of meningeal irritation

Cranio spinal axis

Abdominal Examination

Liver Spleen

Respiratory system

Air entry

Crepts

Rhonchi

CVS

S1 S2

Murmur

Endocrine

Obesity Y/N

Other

Clinical diagnosis

INVESTIGATION CHART

DATE				
1.CBC- Hb				
WBC				
N/E/L/M				
Platelets				
2.PSMP				
3.Widal T.				
4.RBS				
5.Urea/Creatinine. Na/ K/ Cl				
6.Sr.Ca.Ion/Tot Serum Phosphate. Serum Alkaline phosphatase.				
7.LFT-BiIi- T/D SGOT/PT Protein- A/G				
8.HIV				
9.Kochs W/Up Chest X-Ray MTx/BCG GL/Sputum-AFB				
10.CSF/Protein Sugar WBC- N / L Gram/Zn Stain CSF – C/S				
11.Urine- R/M Urine- C/S				

12.Blood C/S				
13.USG Skull USG Abdomen.				
14. CT Scan CT-Brain CT-Thorax				
15.M R I Brain				
16. EEG				
17.Other				

TREATMENT / ANTI-EPILEPTIC DRUGS (AEDs):-

DIAGNOSIS :-

1) **Etiological Diagnosis:-**

2) **ILAE Classification By Seizure Types:-**

3) **ILAE Classification as Epilepsies and Epileptic Syndromes:-**

CONSENT FORM

I/we have been explained in our own language about our child being enrolled in a research study about "A study of clinicoetiological profile of seizures in children in kolar" conducted by Dr. Suvarna Rekha Puvvada, Post Graduate in department of Pediatrics in Sri Devraj Urs Medical College, Kolar under the guidance of Dr. Krishnappa J, Associate Professor, department of Pediatrics,Sri Devraj Urs Medical College, Kolar.

I/we have been explained about the details of the study, purpose of the study and have also been explained that the management of the patient will not be affected irrespective of the enrollment in the study. Once decided to participate I/We have been explained that we are free to withdraw from the study without affecting any of the management.

I/We would not be given any financial incentive and confidentiality would be maintained.

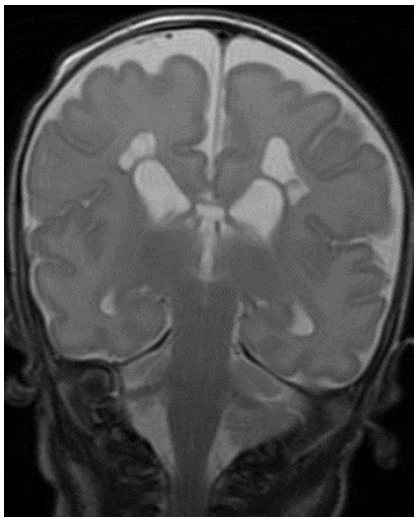
I/We after understanding the above mentioned give consent for the same.

Signature of subject: **Date:**

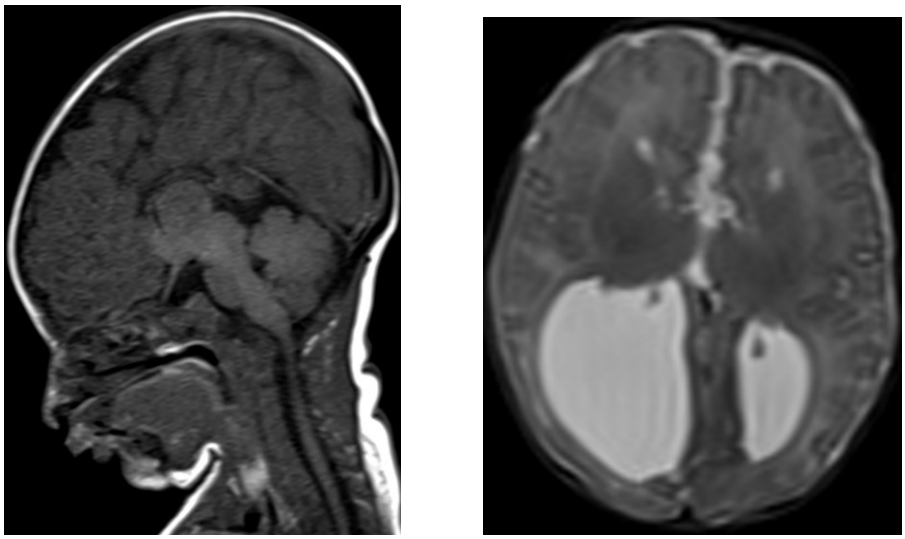
Name of the subject:

Signature of witness: **Date:**

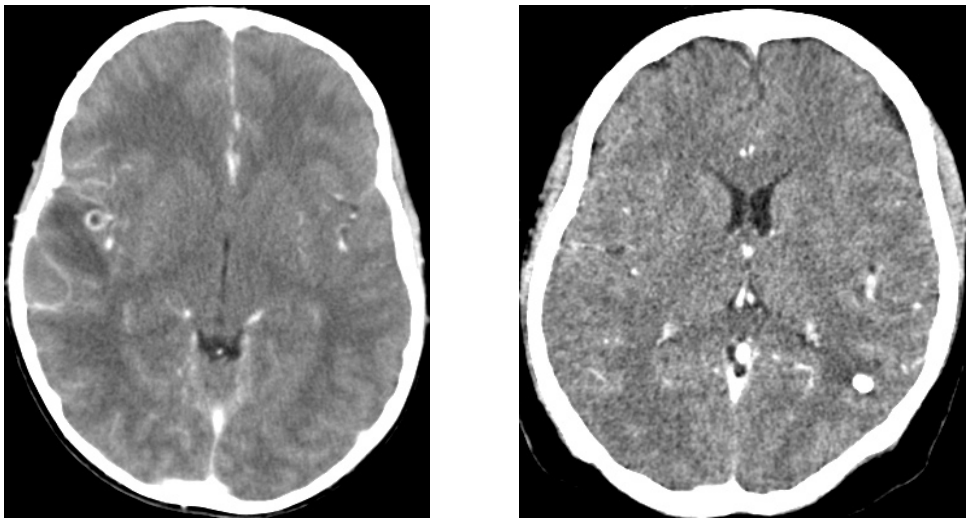
Name of the witness:



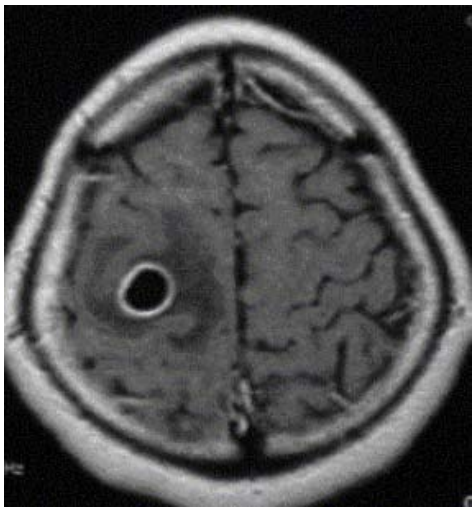
Coronal T2 weighted MR image of an infant showing bilateral symmetric periventricular white matter hyperintense signal intensity suggestive of periventricular leukomalacia



Sagittal and axial T2 weighted MRI images of a 4 month old showing absence of corpus callosum, elevation of the third ventricle, widely and parallel lateral ventricles with colpocephaly (right>left)



CT contrast in a 8 year old child showing ring enhancing lesion in the right temporal lobe with moderate perilesional edema – suggestive of neurocysticercosis in colloid vesicular stage. CSF density lesion in left fronto-parietal region – suggestive of neurocysticercosis in granular nodular stage.



Axial post-contrast T1 weighted MRI image in a 9 year old in a shows a ring enhancing lesion in the right frontal lobe grey-white matter junction with mild perilesional white matter edema, suggestive of granulomatous lesion - Neurocysticercosis

MASTER CHART

s. no	name	age	hosp no	route	gravida	period of gestation	lcb	contraception knowledge	usage of mtp pill knowledge	indication for mtp	failure of contraception	fever	chills	nausea	vomiting	diarrhoea	duration of bleeding	induction expulsion time	amount of blood loss	blood transfusion	acceptance of route	failed induction	surgical evacuation
1	sumi	24	89464	oral	G4P1L1A2	7w 1d	1y 6m	yes	yes	missed abortion		no	no	no	no	yes	24-48hrs	8h 30min	<1g	no	yes	no	no
2	bharathi	18	48578	oral	primi	9w		yes	yes	unwanted		no	no	no	no	no	<24hrs	1h 30min	<1g	no	yes	no	no
3	thahera khanum	25	26804	oral	G3P2L2	7w 3d	2y	yes	no	unwanted		no	yes	yes	no	no	24-48hrs	12h 30min	>2g	no	yes	no	no
4	shilpa	25	74284	oral	G4P3L3	6w	1y 3m	yes	no	failed contraception	failed sterilzation	no	no	no	no	yes	24-48hrs	7h 15min	<1g	no	yes	no	no
5	rehana	26	60092	oral	G2P1L1	6w 5d	2y 2m	yes	no	blighted ovum		no	yes	yes	yes	no	48-72hrs	12h 15min	<1g	no	yes	no	no
6	suguna	33	60462	oral	G3P2L2	7w 1d	1y	yes	yes	unwanted		no	no	yes	no	no	24-48hrs	8h 25min	<1g	no	yes	no	no
7	anjali	20	60243	oral	primi	6w 5d		yes	yes	failed contraception	barrier	yes	no	no	yes	no	<24hrs	3h 30min	<1g	no	yes	no	no
8	salma sulthana	26	64860	oral	primi	6w 5d		yes	yes	missed abortion		no	yes	no	no	no	24-48hrs	5h 45min	<1g	no	yes	no	no
9	shobana	26	95547	oral	primi	5w 2d		yes	yes	failed contraception	barrier	no	yes	no	no	no	24-48hrs	10h 15min	<1g	no	yes	no	no
10	sharada	26	99340	oral	G2P1L1	5w	2y 5m	yes	yes	unwanted		no	yes	no	no	no	<24hrs	4h 15min	<1g	no	yes	no	no
11	mallikanthamma	32	34146	oral	G5P4L4	6w 3d	1y	no	no	failed contraception	barrier	no	no	yes	no	yes	48-72hrs	11h 20min	<1g	no	yes	no	no
12	lakshmi bai	28	123066	oral	G4P2L2A1	7w6d	1y	yes	yes	unwanted		yes	yes	yes	yes	no	>72hrs		1-2g	no	no	yes	yes
13	vasantha kumari	26	45146	oral	primi	7w 4d		yes	yes	missed abortion		no	no	no	no	no	<24hrs	4h 30min	<1g	no	yes	no	no
14	sudharani	26	64116	oral	G3P2L2	6w 2d	1y	yes	yes	unwanted		no	no	yes	yes	no	24-48hrs	8h	<1g	no	yes	no	no
15	sowjanya	21	74804	oral	G2P1L1	5w 6d	7m	yes	no	missed abortion		no	no	yes	no	no	48-72hrs	12 45min	1-2g	no	yes	no	no
16	aruna	23	76094	oral	G2P1L1	4w 5d	2y	yes	yes	blighted ovum		no	no	yes	no	no	<24hrs	4h 45min	<1g	no	yes	no	no
17	bhavani	26	50834	oral	primi	5w 4d		yes	yes	unwanted		no	yes	yes	no	no	24-48hrs	6h 40min	<1g	no	yes	no	no
18	roja	23	81804	oral	G4P3D3	6w 6d	1y	yes	yes	missed abortion		no	no	yes	no	yes	24-48hrs	10 45min	1-2g	no	yes	no	no
19	ashwini	24	122905	oral	G3P2L2	6w 3d	1y 6m	yes	yes	blighted ovum		no	no	no	yes	no	<24hrs	5h 50min	<1g	no	yes	no	no
20	kanchana	32	64154	oral	G5P3L3A1	7w 4d	7m	yes	yes	unwanted		no	no	no	no	yes	24-48hrs	7h 10min	<1g	no	yes	no	no
21	senthamazhi selvi	19	114761	oral	primi	4w 6d		yes	yes	failed contraception	barrier	yes	yes	yes	yes	no	24-48hrs	10h 15min	>2g	no	yes	no	no
22	savitha	25	79992	oral	primi	6w 6d		yes	yes	missed abortion		no	no	yes	no	yes	24-48hrs	10 45min	1-2g	no	yes	no	no
23	naveena	22	80029	oral	G2P1L1	7w 4d	7m	yes	yes	unwanted		no	no	no	no	yes	24-48hrs	7h 10min	<1g	no	yes	no	no
24	sujatha	24	79601	oral	G2P1L1	4w 6d	1y	yes	yes	unwanted		yes	yes	yes	yes	no	48-72hrs	10h 15min	>2g	no	yes	no	no

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25	manjula	22	63994	oral	G5P3L1D2	7w 1d	1y	yes	yes	missed abortion		no	no	yes	no	no	24-48hrs	8h 25min	<1g	no	yes	no	no
26	sonia	24	94609	oral	G2P1L1	6w 3d	1y	no	no	unwanted		no	no	yes	no	yes	48-72hrs	11h 20min	<1g	no	yes	no	no
27	shabeena	24	97885	oral	G6P5L3D2	7w 4d	7m	yes	yes	missed abortion		yes	no	no	no	no	>72hrs		1-2g	no	no	yes	yes
28	mala	24	100318	oral	G3P1L1A1	7w 1d	1y 6m	yes	yes	unwanted		no	no	no	no	yes	24-48hrs	8h 30min	<1g	no	yes	no	no
29	susheelamma	24	118631	oral	primi	6w		yes	no	failed contraception	failed sterilzation	no	no	no	no	yes	>72hrs		>2g	no	no	yes	yes
30	indira	26	118180	oral	G2P1L1	7w 3d	2y	yes	no	unwanted		no	yes	yes	no	no	48-72hrs	12h 30min	>2g	no	yes	no	no
31	suvarna	22	86935	buccal	primi	8w		yes	no	blighted ovum		no	no	yes	yes	no	<24hrs	2h 15min	<1g	no	yes	no	no
32	pallavi	19	86998	buccal	primi	5w 5d		yes	yes	unwanted		no	yes	yes	yes	no	<24hrs	1h 45min	<1g	no	yes	no	no
33	umera	22	6456	buccal	G3P1L1D1	5w 1d	2y 2m	yes	yes	active ptb		no	yes	no	no	no	24-48hrs	6h 30min	<1g	no	yes	no	no
34	nethravathi	23	132027	buccal	primi	6w 6d		yes	yes	unwanted		no	no	yes	no	no	24-48hrs	5h 50min	<1g	no	yes	no	no
35	manjula	18	134344	buccal	primi	8w6d		yes	no	missed abortion		no	no	no	no	no	24-48hrs	7h 30min	<1g	no	yes	no	no
36	suma	20	136997	buccal	primi	8w		yes	yes	unwanted		no	yes	no	no	no	24-48hrs	6h 15min	<1g	no	yes	no	no
37	jameesha taj	33	137908	buccal	G3P2L2	7w 6d	2y	yes	yes	failed contraception	barrier	no	no	no	no	no	<24hrs	3h 15min	<1g	no	yes	no	no
38	divya	21	154183	buccal	G3P2L2	6w 5d	1y 2m	yes	yes	failed contraception	barrier	no	yes	yes	no	no	24-48hrs	5h 20min	<1g	no	yes	no	no
39	salma taj	25	166597	buccal	G2P1L1	5w	10m	yes	yes	unwanted		no	no	no	no	no	48-72hrs	10h 15min	<1g	no	yes	no	no
40	veena	28	131234	buccal	G3P2L1D1	8w 5d	8m	yes	yes	unwanted		no	no	no	no	no	24-48hrs	6h 45min	<1g	no	yes	no	no
41	reshma	20	166742	buccal	G2P1L1	6w 5d	1y	yes	yes	unwanted		no	no	yes	yes	no	24-48hrs	5h 45min	<1g	no	yes	no	no
42	shar taj	25	174692	buccal	G2P1L1	7w	1y 8m	yes	no	blighted ovum		no	no	no	no	no	<24hrs	4h 15min	<1g	no	yes	no	no
43	sumithra	27	173560	buccal	G4P1L1A2	4w 5d	2y	yes	yes	blighted ovum		no	no	yes	no	no	<24hrs	4h 45min	<1g	no	yes	no	no
44	ashwini shree	24	180507	buccal	primi	6w 5d		yes	yes	unwanted		no	no	yes	yes	no	24-48hrs	5h 45min	<1g	no	yes	no	no
45	venkatalakshmamm	28	3310	buccal	G2P1L1	7w	1y 8m	yes	no	blighted ovum		no	no	no	no	no	<24hrs	4h 15min	<1g	no	yes	no	no
46	asifa khanum	30	14699	buccal	G2P1L1	5w 4d	4y	yes	yes	failed contraception	barrier	no	yes	yes	no	no	24-48hrs	6h 40min	<1g	no	yes	no	no
47	gayathri	20	33229	buccal	G2A1	6w 3d	1y 6m	yes	yes	blighted ovum		no	no	no	yes	no	24-48hrs	5h 50min	<1g	no	yes	no	no
48	mallika	26	62739	buccal	G4P2L2A1	5w 2d	1y 7m	yes	yes	unwanted		no	yes	no	no	no	48-72hrs	10h 15min	<1g	no	yes	no	no
49	shwetha	22	52784	buccal	primi	5w		yes	yes	failed contraception	barrier	no	yes	no	no	no	<24hrs	4h 15min	<1g	no	yes	no	no
50	bhavya	26	72995	buccal	G3P2L1D1	7w6d	1y	yes	yes	unwanted		yes	yes	yes	yes	no	>72hrs		1-2g	no	no	yes	yes
51	chandrakala	25	1376	buccal	G3P1L1E1	6w 2d	1y	yes	yes	unwanted		no	no	yes	yes	no	48-72hrs	8h	<1g	no	yes	no	no
52	varalakshmi	25	76664	buccal	G4P1L1A2	5w	10m	yes	yes	unwanted		no	no	no	no	no	48-72hrs	10h 15min	<1g	no	yes	no	no
53	sarala	24	107929	buccal	G4P1L1A2	5w 6d	1y 6m	yes	no	missed abortion		no	no	yes	no	no	48-72hrs	12h 45min	1-2g	no	yes	no	no

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54	kavya	19	110396	buccal	primi	8w		yes	no	blighted ovum		no	no	yes	yes	no	<24hrs	2h 15min	<1g	no	yes	no	no
55	reshma banu	26	133486	buccal	G3P2L2	5w 5d	3y	yes	yes	unwanted		no	yes	yes	yes	no	<24hrs	1h 45min	<1g	no	yes	no	no
56	savithri	21	137122	buccal	primi	5w 1d		yes	yes	failed contraception	barrier	no	yes	no	no	no	48-72hrs	6h 30min	<1g	no	yes	no	no
57	pavithra	26	148597	buccal	G3P1L1A1	9w	1y 2m	yes	yes	unwanted		no	no	no	no	no	>72hrs		1-2g	no	no	yes	yes
58	hema	37	157694	buccal	G3P2L2	6w 5d	2y 6m	yes	yes	missed abortion		no	no	yes	no	no	24-48hrs	5h 25min	<1g	no	yes	no	no
59	veena	36	170867	buccal	G4P3L3	6w 5d	4y	yes	no	blighted ovum		no	yes	yes	yes	no	48-72hrs	12h 15min	<1g	no	yes	no	no
60	shaziya khanum	19	156015	buccal	primi	6w 5d		yes	yes	unwanted		yes	no	no	yes	no	<24hrs	3h 30min	<1g	no	yes	no	no
61	amaravathi	24	116221	vaginal	G3P2L2	7w 3d	1y 6m	yes	no	unwanted		no	no	yes	yes	no	<24hrs	4h	<1g	no	yes	no	no
62	vanitha	23	114576	vaginal	G3P1L1A1	6w 1d	2y	yes	yes	missed abortion		no	no	yes	no	no	>72hrs		1-2g	no	yes	yes	yes
63	mounika	21	118096	vaginal	G2P1L1	8w 2d	1y	yes	no	unwanted		no	no	no	no	no	<24hrs	4h 30min	<1g	no	yes	no	no
64	apoorva	26	107246	vaginal	primi	6w 3d		yes	yes	blighted ovum		no	no	yes	no	no	24-48hrs	5h 15min	<1g	no	yes	no	no
65	kavitha	23	15510	vaginal	G2P1L1	8w 1d	8m	yes	yes	missed abortion		no	no	no	no	no	48-72hrs	11h 20min	<1g	no	yes	no	no
66	nethravathi	21	45447	vaginal	primi	7w 3d		yes	yes	unwanted		no	no	yes	yes	no	24-48hrs	6h 50min	<1g	no	yes	no	no
67	naveeda	20	16415	vaginal	primi	5w 5d		yes	no	failed contraception	barrier	no	no	no	no	no	<24hrs	3h 40min	<1g	no	yes	no	no
68	thabasum kouser	22	16436	vaginal	G2A1	6w 1d	4y	yes	yes	unwanted		yes	yes	yes	no	no	>72hrs		>2g	no	no	yes	yes
69	shabana begum	34	107359	vaginal	G3P2L2	6w	3y	yes	yes	failed contraception	barrier	yes	no	yes	no	no	48-72hrs	8h 30min	<1g	no	yes	no	no
70	sumana	29	93982	vaginal	G2P1L1	6w	1y 8m	yes	yes	unwanted		no	no	no	no	no	<24hrs	2h 45min	<1g	no	yes	no	no
71	asha rani	24	123347	vaginal	G2P1L1	8w 3d	5y	yes	yes	blighted ovum		no	yes	no	no	no	<24hrs	4h 45min	<1g	no	yes	no	no
72	aruna	20	18246	vaginal	primi	8w 5d		yes	yes	unwanted		no	no	no	no	no	>72hrs		1-2g	no	no	yes	yes
73	shobarani	23	132515	vaginal	primi	9w		yes	no	missed abortion		no	no	yes	no	no	48-72hrs	7h 30min	<1g	no	yes	no	no
74	saritha	32	135342	vaginal	G2P1L1	6w 2d	2y 6m	yes	yes	unwanted		no	no	no	no	no	24-48hrs	5h 45min	<1g	no	yes	no	no
75	anitha	20	131097	vaginal	G3P2L1D1	7w 3d	9y	yes	yes	blighted ovum		yes	yes	no	no	no	48-72hrs	9h 15min	<1g	no	yes	no	no
76	manjula	26	19272	vaginal	G2P1L1	6w 3d	4m	yes	yes	failed contraception	barrier	no	no	no	no	no	24-48hrs	5h 45min	<1g	no	yes	no	no
77	roopa	25	157805	vaginal	G2P1L1	8w 5d	10m	yes	yes	unwanted		no	yes	no	no	no	48-72hrs	8h	<1g	no	yes	no	no
78	ramya	20	104729	vaginal	primi	7w 5d		yes	no	missed abortion		yes	no	no	no	no	48-72hrs	8h 20min	<1g	no	yes	no	no
79	anitha	28	22337	vaginal	G3P1L1A1	7w 3d	9y	yes	yes	blighted ovum		yes	yes	no	no	no	48-72hrs	9h 15min	<1g	no	yes	no	no
80	saroja	23	3691	vaginal	G2P1L1	6w 3d	4m	yes	yes	unwanted		no	no	no	no	no	24-48hrs	5h 45min	<1g	no	yes	no	no
81	shilpa	25	14857	vaginal	primi	8w 5d		yes	yes	unwanted		no	yes	no	no	no	48-72hrs	8h	<1g	no	yes	no	no
82	ammalamma	35	24593	vaginal	G7P6L6	7w 5d	10m	yes	no	missed abortion		yes	no	no	no	no	48-72hrs	8h 20min	<1g	no	yes	no	no

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83	fayeema	26	25106	vaginal	G4P3L3	6w	3y	yes	yes	failed contraception	barrier	yes	no	yes	no	no	48-72hrs	8h 30min	<1g	no	yes	no	no
84	shobamma	25	25047	vaginal	G3P2L2	6w	1y 8m	yes	yes	unwanted		no	no	no	no	no	<24hrs	2h 45min	<1g	no	yes	no	no
85	hamsaveni	20	26888	vaginal	G2P1L1	8w 3d	5y	yes	yes	blighted ovum		no	yes	no	no	no	<24hrs	4h 45min	<1g	no	yes	no	no
86	navitha	28	30652	vaginal	G3P2L2	8w 5d	4y	yes	yes	unwanted		no	no	no	no	no	24-48hrs	6h 25min	<1g	no	yes	no	no
87	nageen taj	23	32859	vaginal	G3P2L2	6w 1d	2y	yes	yes	missed abortion		no	no	yes	no	no	>72hrs		1-2g	no	yes	yes	yes
88	munilakshmi	26	34181	vaginal	G3P2L2	8w 2d	1y	yes	no	unwanted		no	no	no	no	no	<24hrs	4h 30min	<1g	no	yes	no	no
89	jyothi	22	35406	vaginal	G2P1L1	6w 3d	11m	yes	yes	blighted ovum		no	no	yes	no	no	24-48hrs	5h 15min	<1g	no	yes	no	no
90	madhavi	22	39763	vaginal	G2P1D1	7w 3d	3y	yes	yes	unwanted		no	no	yes	yes	no	48-72hrs	6h 50min	<1g	no	yes	no	no