WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 15, 1784-1806. Research Article ISSN 2277–7105

A STUDY OF INCIDENCE, TYPES, ETIOLOGY AND MANAGEMENT OF SEIZURES IN CHILDREN OF AGE GROUP 5 MONTHS TO 6 YEARS IN PEDIATRICS I.C.U/WARD IN A TERTIARY CARE HOSPITAL

Ravi Prakash Degala[*1], G. Lakshmi Prasanna[2], A. Charan Kishore[3], B. Satya Lakshmi Bhavani[4], G. Raji[5], S. Sri Lakshmi[6] and M. Swathi[7]

[*1]Associate Professor and Head of the Department, Department of Pharmacy Practice, Pydah College of Pharmacy, Patavala, Kakinada, Andhra Pradesh, India.

[2,6]Research Scholar, Department of Pharmacy, Koringa College of Pharmacy, Korangi, Tallarevu, Kakinada, Andhra Pradesh, India.

[7]Assistant Professor, Department of Pharmacy Practice, Koringa College of Pharmacy, Korangi, Tallarevu, Kakinada, Andhra Pradesh, India.

Article Received on
21 June 2025,

Revised on 10 July 2025,
Accepted on 30 July 2025,

DOI: 10.20959/wjpr202515-37875

***Corresponding Author**
Ravi Prakash Degala
Associate Professor and Head of the Department, Department of Pharmacy Practice, Pydah College of Pharmacy, Patavala, Kakinada, Andhra Pradesh, India.

ABSTRACT

Background: Seizures are among the most common neurological emergencies in pediatric patients, particularly in children under 6 years of age. Early identification of seizure type, underlying etiology, and timely management are critical to improving outcomes and reducing complications. **Objective:** To evaluate the incidence, classify the types, identify the etiology, and assess the management strategies of seizures in children aged 5 months to 6 years admitted to the Pediatric ICU/ward of a tertiary care hospital. **Methods:** This cross-sectional observational study was conducted over a period of 12 months in the Pediatrics Department of a tertiary care hospital. A total of 109 children aged 5 months to 6 years presenting with seizures were included. Detailed clinical history, physical examination, laboratory investigations, neuroimaging, and EEG findings were recorded. Seizures were classified as febrile, afebrile, generalized, focal, and others. Management strategies, including pharmacological interventions and supportive care, were also documented and analyzed.

Results: Out of 150 pediatric admissions, 109 presented with seizures. Febrile seizures were the most common type followed by afebrile generalized seizures. The leading etiologies included febrile illness (30.2%), CNS infections (29.3%), and cerebral palsy (17.4%). Most patients responded well to first-line anticonvulsants such as phenytoin (27.5%) and Clobazam(21.1%). **Conclusion:** Seizures are a frequent presentation in the pediatric age group, with febrile seizures being the most prevalent. Timely recognition of seizure type and underlying etiology is vital for effective management and prognosis. Strengthening early diagnostic services and pediatric critical care can significantly reduce seizure-related morbidity.

KEYWORDS: Seizures, Pediatrics, Febrile Seizures, Etiology, Management, Pediatric ICU, Children 5 months–6 years.

INTRODUCTION

A seizure is an impermanent occurrence of signs or symptoms due to abnormal excessive or synchronous neuronal activity in the brain. Seizures in a newborn are one of the few neonatal neurological emergencies where prompt diagnostic and therapeutic plans are necessary; a delay in therapy results in poor neurological outcome. When the above is associated with motor component then they are known as convulsions. Epilepsy is a condition characterized by recurrent (two or more) unprovoked seizures occurring 24 hours apart.[1] Childhood epilepsy are a heterogeneous group of conditions that differ in their diagnostic criteria and management and have dramatically different outcomes.

Seizures in a newborn are one of the few neonatal neurological emergencies where prompt diagnostic and therapeutic plans are necessary; a delay in therapy results in poor neurological outcome.[2]

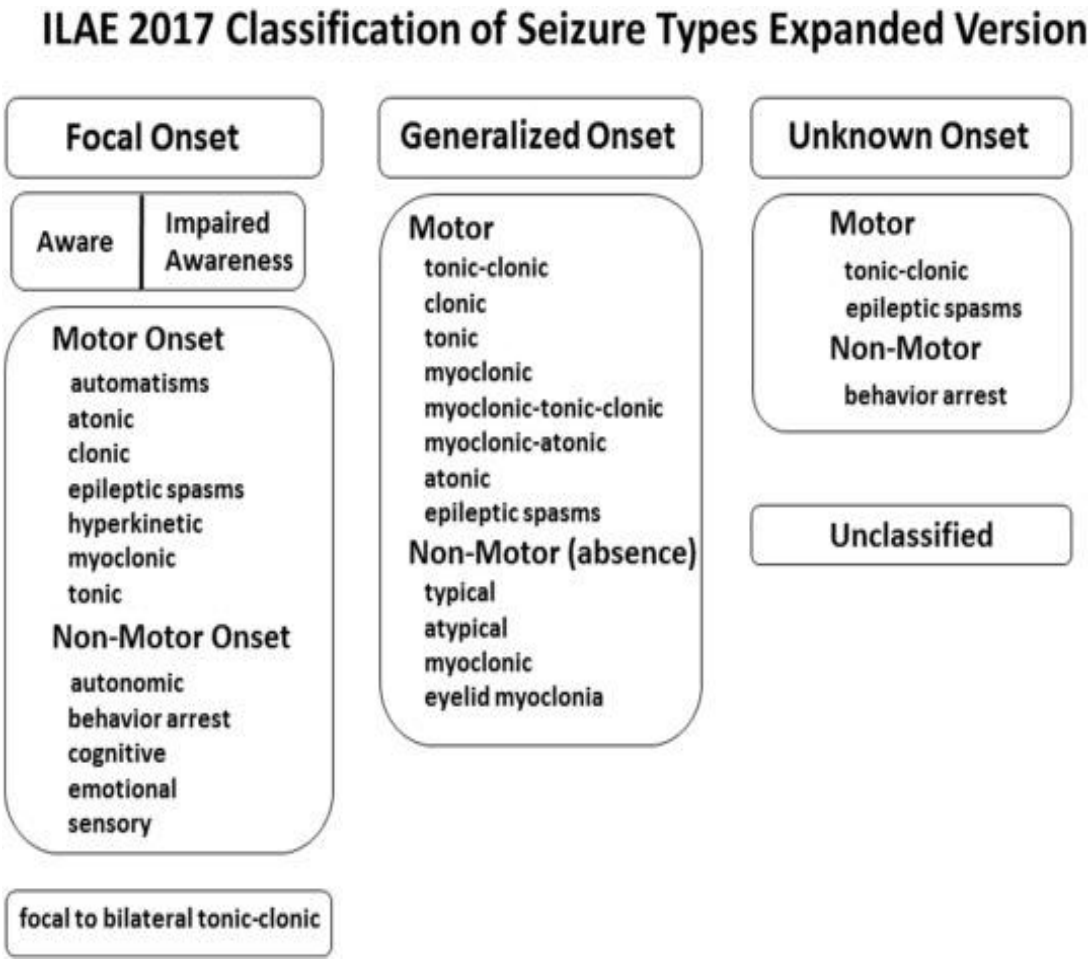
EPIDEMIOLOGY

Seizures are the most common and frightening pediatric neurological disorder, with 4-10% of children experiencing at least one seizure within first 16 years of life. Children below 3 years of age have the highest incidence of seizures than older children.[3] Seizures during the neonatal period are relatively common, occurring in approximately 1.8 to 3.5 per 1000 live births with greater frequency in premature or low birth weight babies as compared to term babies.[4] In the neonatal intensive care unit, the incidence goes as high as 10 to 25%, out of which about 15% will die and 35 to 40% will have major neurological sequel.[5] The

prevalence of seizures is >3 per 1000 in developed world as compared to 9 per 1000 in developing nations.[6] Christopher et al described that around 3% of all children below 15 years of age have a seizure, 50% of which are febrile seizures and epilepsy is the underlying cause in one of every hundred children with seizures.[7] Hamdy et al described the annual incidence of seizures was 153 per 100,000 for new onset seizures and it was significantly higher in south Asians.[8] Sillanpaa et al studied to show that children with epilepsy have 7% life time risk of sudden death at 40 years and 12% in another group of patients with epilepsy.[9] It affects 2% to 4% of all children in Europe and the United States by their fifth birthday.[10] The incidence in other areas varies from 0.35% in Hong Kong,[11] 1% in China to more than 8% in Japan and 14% in Guam.[10] It affects 2% to 4% of all children in Europe and the United States by their fifth birthday.[10] The incidence in other areas varies from 0.35% in Hong Kong,[11] 1% in China to more than 8% in Japan and 14% in Guam.[10]

CLASSIFICATION

There are many types of seizures in children which include focal, generalized, unknown seizures and electro clinical syndromes according to ILAE classification.[12]



Focal seizures are often preceded by certain experiences, known as an aura.[12] These may include: sensory, visual, psychic, autonomic, olfactory or motor phenomena.[14]

In a **complex partial seizure** a person may appear confused or dazed and cannot respond to questions or direction. Focal seizure may become generalized.[14]

Jerking activity may start in a specific muscle group and spread to surrounding muscle groups—known as a Jacksonian march.[15] Unusual activities that are not consciously created may occur.[15] These are known as automatisms and include simple activities like smacking of the lips or more complex activities such as attempts to pick something up.[15]

Generalized seizures, There are six main types of generalized seizures: tonic-clonic, tonic, clonic, myoclonic, absence, and atonic seizures.[16] They all involve a loss of consciousness and typically happen without warning.[17]

- Tonic-clonic seizures present with a contraction of the limbs followed by their extension, along with arching of the back for 10–30 seconds.[17] A cry may be heard due to contraction of the chest muscles.[17] The limbs then begin to shake in unison.[17] After the shaking has stopped it may take 10–30 minutes for the person to return to normal.[17]
- Tonic seizures produce constant contractions of the muscles.[17] The person may turn blue if breathing is impaired.[17]
- Clonic seizures involve shaking of the limbs in unison.[17]
- Myoclonic seizures involve spasms of muscles in either a few areas or generalized through the body.[17]
- Absence seizures can be subtle, with only a slight turn of the head or eye blinking.[16] The person often does not fall over and may return to normal right after the seizure ends, though there may also be a period of post-ictal disorientation.[16]
- Atonic seizures involve the loss of muscle activity for greater than one second.[15] This typically occurs bilaterally (on both sides of the body).[15]

ETIOLOGY

Better understanding of seizures in terms of clinical presentation and etiology is required not only for abortion of acute attack but also for long term control of epilepsy. In the newborn, seizures are always due to an underlying cerebral or biochemical abnormality. Childhood seizures have many different causes which include prenatal or genetic causes (65%), perinatal causes (8%) and complications of prematurity (13%) or acquired causes (7%). Epilepsy is

common in some families like (36%) had first- or second-degree relatives affected and mostly it is idiopathic (54% vs 30%) even without neurological sequalae (57% vs 26%).[18] Seizures not only cause physical concerns, behavioural issues and cognitive impairment but also are responsible for persistent psychosocial stress for the children and their parents.[19] The seizures in children lead to regular follow up visits to concerned specialists, affecting their school performance, and their inability to take part in sports and other extracurricular activities.[20] Burton et al described that seizures cause long term cognitive impairment (64%), behaviour disorder (61%), motor difficulties (26%), burns and other previous injuries (26%) and poor school attendance (50%).[21] There are many possible etiologies of a first attack of seizure in children, including infection, neurologic/developmental causes, traumatic head injury and metabolic disturbances.[22] The common etiologies of seizures are determined by the geographical variations. Febrile seizures (FS) are reported in many studies to be the most common type of seizures seen in the pediatric population, and majority being less than five years of age.[10] Infections are the most associated causes and have a good outcome.[23]

FEBRILE SEIZURES is defined as seizure during fever, between 6 months to 6 years of age in absence of intracranial infection or previous unprovoked seizure.[24,25] Another definition from the International League Against Epilepsy (ILAE) is "a seizure occurring in childhood after 1 month of age up to 6 years, associated with a febrile illness not caused by an infection of the Central Nervous System (CNS), without previous neonatal seizures or a previous unprovoked seizure, and not meeting the criteria for other acute symptomatic seizures".[26,27] Febrile seizures are divided into 2 types: simple febrile seizures (which are generalized, last <15 min and do not recur within 24 h) and complex febrile seizures (which are prolonged, recur more than once in 24 h, or are focal). Complex febrile seizures are prolonged (greater than 15 min), focal or multiple (recurrent within same febrile illness over 24 h period).[27,28]

However there are studies, which have shown febrile seizure as a major cause of seizure in children. There are limited studies in developing countries that look at the causes and outcome of seizures in children. Most of the studies are focused on afebrile seizures.

Most febrile seizures are triggered by fevers from viral upper respiratory infections, ear infections, or roseola. However, meningitis, if bacterial in etiology, can also present with fever and a single, self-limited seizure. Therefore it is a priority to diagnose or exclude bacterial meningitis.[29] According to AAP guidelines in a child who presents with a febrile

seizure, a lumbar puncture should be performed in a child who has obvious meningeal signs and symptoms (such as neck stiffness, Kernig or Brudzinski signs) or other clinical signs that are indicative of meningitis or intracranial infection. Additionally lumbar puncture should be considered in any infant between 6 and 12 months of age who is not up to date with Hib or Streptococcus pneumoniae immunizations, or whose immunization status cannot be determined, especially because symptoms and signs of meningeal irritation can be absent in this age group. And the last indication for performing lumbar puncture is in any infant between 6 and 12 months of age who has received systemic antibiotic therapy by any route in the days preceding the seizure, because of a risk that signs of meningitis are masked.[29]

However, it seems that current guidelines are not followed strictly and in practical medicine. Recent studies showed variable predicting factors of bacterial meningitis in children presenting with febrile seizure.

Children who present to the Emergency Department with new onset of non-febrile seizure are often evaluated using cranial computed tomography (CT); however, according to recent investigations, brain CT scans are not routinely arranged for these patients.[30] Primary care physicians in the Emergency Department have to face the challenge of initial management of patients with seizures and further evaluation of the indications for brain imaging studies, especially in patients who present with a first attack of seizure. After stabilizing the patient in the Emergency Department, the primary challenge for the Emergency Department physician is to decide whether to admit or discharge the patient. Misdiagnosis carries the potential risk of legal problems, can cause family anxiety, lead to excessive hospital stay, and possibly result in life-threatening events.

CEREBRAL PLASY is caused by abnormal development or damage to the parts of the brain that control movement, balance and posture. Most often the problems occur during pregnancy; however, they may also occur during childbirth, or shortly after birth. Often the cause is unknown. Risk factors include preterm birth, being a twin, certain infections during pregnancy such as toxoplasmosis or rubella, exposure to methyl mercury during pregnancy, a difficult delivery, and head trauma during the first few years of life, among others.[31] Epilepsy is known to have a higher association with cerebral palsy; 15–60% of children with cerebral palsy have been reported to have epilepsy.[32] It has been observed that seizures in these children tend to have an earlier onset, necessitating the use of more than one antiepileptic drug (AED) with the risk of seizure relapse after AED discontinuation.[33,34] It is

important to know the risk factors for recurrence following a first unprovoked seizure in children despite, nowadays most experts think for most children no indication exists to start therapy with antiepileptic drugs after a first seizure as treatment after a first seizure does not influence the prognosis of the epilepsy for those children who will have recurrence.[35] There is a controversy on labelling children as epileptics after a single unprovoked seizure especially in many third world communities including Jordan since epilepsy is still regarded as a stigma.[36,38] The reported risks of recurrence following a first unprovoked seizure in previous studies that included adults and/or children vary from 27 to 71%.[39,54] Most children will remain seizure-free after drug withdrawal since risk of recurrence after discontinuation of AED is generally 20 -40% in children.[55,59] Age at onset of seizures, idiopathic etiology without a neurological disease, absence of interictal abnormalities on first electroencephalogram (EEG) and EEG before withdrawal, duration of AED withdrawal and seizure-free period before withdrawal are associated with a low risk of recurrence; however, there is still no general agreement with respect to the criteria to predict safe discontinuation.[55,61]

Status epilepticus (SE) is a serious neurological problem in children. Both convulsive and nonconvulsive SE affects people of all ages, though it is more common and causes greater morbidity and mortality in infants.[61,66] Age, etiology, and the duration of seizure activity correlate with mortality.[67] Convulsive SE defined as a seizure lasting more than 30 min or recurrent seizures lasting more than 30 min from which the patient does not regain consciousness (ILAE, 1981). However the current definition of 30 min is not universally accepted, and several clinical studies have been published using duration of 10 or 20 min.[67,68] It has been reported that the mortality is nearly 10-fold higher for seizure lasting 30 min or longer than for those lasting 10–29 min.[68] The optimal management of children with SE remains unclear, and large, controlled studies comparing the various agents are lacking. All patients with SE must be managed with aggressive monitoring of their hemodynamic and respiratory status and continuous EEG monitoring. There are multiple regimens for treating SE in children.[69,71] the choice of initial agent may depend on individual patient characteristics, prior antiepileptic drug therapy, and physician preference. However more information is needed to develop a structured treatment regimen based on an operational definition for SE.

MANAGEMENT

The conventional antiepileptics are still the main modality of epilepsy treatment in Asian children even after the availability as well as safety of newer antiepileptics, like Valproate (about 40%). Valproic acid was advised mostly followed by carbamazepine and benzodiazepines. The newer antiepileptics are increasingly being prescribed 26.9% nowadays.[72] Monotherapy is recommended because of fewer adverse drug effects, absence of drug-drug interactions, better compliance, and lower cost compared to therapy with multiple AEDs.[73,76] Studies in developed countries with adequate resources for treatment have however shown that 17-40 % of children do not respond to the first drug used and may require multiple AEDs.[77,78]

It has been suggested that the patients' clinical characteristics such as frequent, focal and long duration of seizures, symptomatic or syndromic epilepsy, history of status epilepticus, and the presence of neurological deficits, is the primary reason for failure of the first AED, rather than drug related factors such as efficacy and adverse effects. The answers to these questions are important, because inadequate response to initial treatment with the first AED and subsequent treatment with multiple AEDs is believed, in itself, to be a poor prognostic factor in epilepsy.[79,81]

There are four phases for CSE management: prehospital; first-line treatment in the accident and emergency department; second-line treatment after the failure or absence of benzodiazepine first-line therapy; and general anaesthesia. However, there is a paucity of data on the benefit ts of prehospital treatment and the choice and route of administration of antiepileptic drugs (AEDs) in hospital.[82,83] Furthermore, the predictors of respiratory depression, which is an important complication of the treatment of CSE, are inadequately researched.[84,85]

Neither of the current UK treatment guidelines—the Advanced Paediatric Life Support (APLS) guidelines and the National Institute for Health and Clinical Excellence (NICE) guidelines—cover the prehospital setting, despite most episodes of CSE starting in the community.[86,87] Both guidelines recommend similar hospital treatments, despite the absence of good evidence for treatments for CSE.[82]

National Institute for Health and Clinical Excellence (NICE) guidelines for pediatrc seizures

Guidelines for treating convulsive status epilepticus in children (published in 2011) The original guidelines for the treatment of convulsive status epilepticus (CSE) were published in 2000. They were subsequently adopted by the Advanced Life Support Group (ALSG) and taught in their courses across the UK and Europe. They represent the basis for much of the management of CSE by junior doctors although they are not intended to cover all situations. They are hospital guidelines and take no account of pre-hospital treatment. They do not include infants, those born very prematurely and/or less than 28days of age. Also, they do not cover children who have frequent episodes of CSE for whom an individually tailored guideline is the best option as their seizures may respond better to specific treatments than others. Generalised convulsive (tonic–clonic) status epilepticus is defined as a generalised convulsion lasting 30minutes or longer or repeated tonic–clonic convulsions occurring over a 30minutesperiod without recovery of consciousness between each convulsion. However, the guideline stated that 'for practical purposes, the approach to the child who presents with a tonic–clonic convulsion lasting more than 5 minutes should be the same as the child who is in " established" status – to stop the seizure and to prevent the development of status epilepticus'. The consensus guideline can be seen in the table below.

Table: Treating convulsive status epilepticus.

Time 0 mins (1st step)	Seizure starts Check ABC, high flow O_2 if available Check blood glucose	Confirm clinically that it is an epileptic seizure
5 mins (2ndstep)	Midazolam 0.5mg/kg buccally or Lorazepam 0.1mg/kg if intra venous access established	Midazolam may be given by parents, carers or ambulance crew in non-hospital setting
15mins (3rdstep)	Lorazepam.1mg/kg intravenously	This step should be in hospital Call for senior help Start to prepare phenytoin for 4thstep Re-confirm it is an epileptic seizure
25mins (4thstep)	Phenytoin 20mg/kg by intravenous infusion over 20mins or (if on regular phenytoin) Phenobarbital 20mg/kg intravenously over 5mins	Paraldehyde 0.8ml/kg of mixture may be given after start of phenytoin infusion as directed by senior staff Inform intensive care unit and/or senior anaesthetist
45mins (5thstep)	Rapid sequence induction of anaesthesia using thiopental sodium 4mg/kg intravenously	Transfer to paediatric intensive care unit

2004 Guideline Development Group

This is less common than tonic–clonic status epilepticus. Treatment for non-convulsive status epilepticus is less urgent than for convulsive status epilepticus. Treatment should be considered as follows:

- Maintenance or reinstatement of usual oral AED therapy.
- Use of intra venous benzodiazepines under EEG control, particularly if the diagnosis is not established.
- Referral for specialist advice and/or EEG monitoring[88]

Electrographic Seizures or Electrographic Status Epilepticus occur in 7–47% of critically ill children who undergo cEEG.[89,100] and several studies have reported an association between ES and ESE and worse short term outcome.[99,102] When surveyed, most physicians reported that they initiated antiepileptic drugs (AEDs) in response to Electrographic Seizures or Electrographic Status Epilepticus, but there was substantial variability in the specific AEDs they reported administering.[103] Further, survey responses may not reflect true practice. Data regarding AED usage patterns will help guide clinical management and develop feasible prospective AED effectiveness studies.

INDIAN GUIDELINESS OF MANAGEMENT

Principles of Treatment

Treatment should be started with a single conventional AED (monotherapy). The formulation or brand of AED should preferably not be changed (variations in bioavailability or different pharmacokinetic profiles may increase the potential for reduced effect or excessive side effects). The dose should be slowly built up until seizure control is achieved or side effects occur. If the initial treatment is ineffective or poorly tolerated then monotherapy using another AED can be tried. The dose of the second drug is slowly increased until adequate or maximum tolerated dose is reached. The first drug is then tapered off slowly. Combination therapy (polytherapy or adjunctive or “add on” therapy) can be considered when two attempts at monotherapy with AEDs have not resulted in seizure freedom. Modified release formulations are convenient but expensive. Once-daily administration should be used with caution in pregnancy.

Choosing the Appropriate Antiepileptic Drug

Phenytoin (PHT), phenobarbitone (PB), carbamazepine (CBZ), oxcarbazepine (OXC) and valproate (VPA) are usually called "conventional" or "first-line drugs". The other AEDs are called "new" or "second-line drugs". It is preferable to use a conventional AED as the initial drug since those are less expensive and the side effects with long-term use are well-known (Flow chart 1). The newer AEDs can also be used when:

- There are contraindications to the first-line drugs due to coexisting illnesses
- The first-line drugs interact with other drugs; the person is taking (notably oral contraceptives, anticoagulants, antiretrovirals or immunosuppressants).

Indications for Monitoring AED Blood Levels

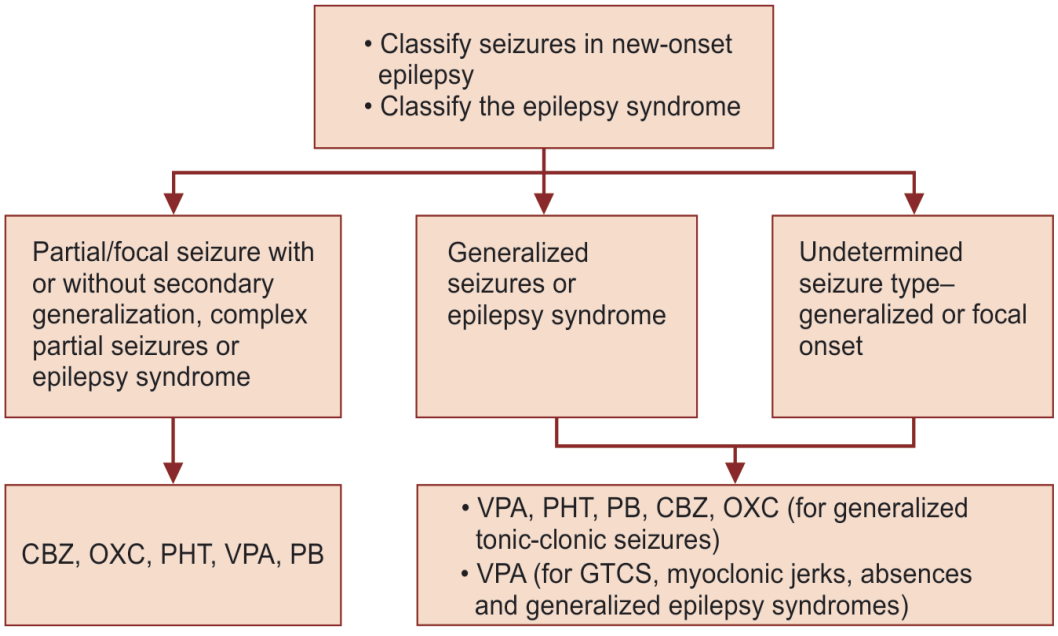
- Detection of AED noncompliance in case of uncontrolled seizures
- Documenting suspected AED toxicity
- Adjustment of AED dose while managing drug interactions
- Specific clinical conditions (e.g. SE, liver or renal disease and pregnancy).

Monitoring the Antiepileptic Drug Therapy

The following tests may be carried out as necessary:

- Complete blood count, liver enzymes and renal functions before starting AED.

Flow chart 1: Algorithm for choice of antiepileptic drug (AED) among new-onset epilepsy patients



Abbreviations: CBZ, Carbamazepine; OXC, Oxcarbazepine; PHT, Phenytoin; VPA, Valproate; PB, Phenobarbitone; GTCS, Generalized tonic-clonic seizure.

Flow chart 2: Algorithm for strategies in case of failure of initial treatment

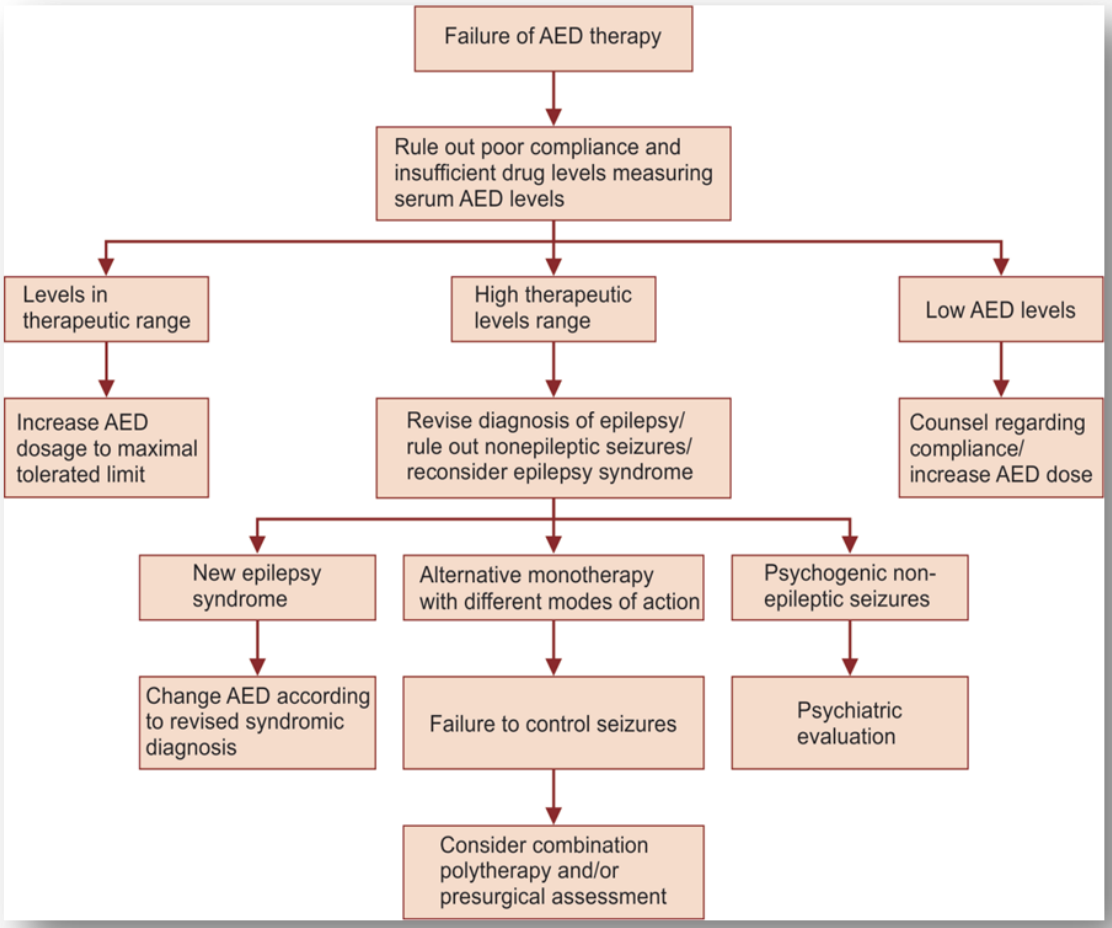


TABLE Initial and maintenance daily doses and important side effects of commonly used antiepileptic drugs (AEDs)

Antiepileptic drugs (AEDs)	Starting dose in average adults	Maintenance dose in average adults (mg/day)	Important side effects
Carbamazepine (CBZ)	100 mg BID	400–1000	Sedation, dizziness, ataxia, skin rash (occasionally Stevens- Johnson syndrome), hyponatremia, weight gain, seizure worsening in some epilepsy syndromes
Clobazam (CLB)	10 mg OD (HS)	10–30	Sedation, ataxia, somnolence, irritability, depression,weight gain,tolerance (reduced antiepileptic effect)
Lamotrigine	25 mg OD	100–300	Sedation, ataxia, dizziness, skin rash

(LTG)	(HS); lower dose VPA		(occasionally Stevens-Johnson syndrome)
Levetiracetam (LEV)	250 mg BID	1,000–3000	Somnolence, dizziness, cognitive slowing, psychosis
Oxcarbazepine (OXC)	150 mg BID	600–1800	Sedation, dizziness, ataxia, headache, hyponatremia, skin rash
Phenobarbitone (PB)	60–90 mg OD (HS)	60–180	Sedation, ataxia, depression, memory problems, skin rash, hyperactivity in children
Phenytoin (PHT)	200–300 mg OD (HS)	200–400	Ataxia, sedation, gum hyperplasia, coarsening of facial features, hirsutism, memory problems, osteomalacia and bone loss, skin rash
Topiramate (TPM)	25 mg OD	100–400	Sedation, somnolence, cognitive problems, weight loss, word-finding difficulty, renal stones, seizure worsening
Valproate (VPA)	200 mg BID	500–2000	Anorexia, weight gain, nausea, vomiting, tremors, hair loss, polycystic ovarian syndrome, thrombocytopenia
Zonisamide (ZNS)	50 mg OD (HS)	200–500	Sedation, anorexia, renal stones, forgetfulness,
Abbreviations: OD, Once daily; BID, Twice daily; HS, At night			

Abbreviation: AED, Antiepileptic drug

- Certain AEDs (PHT, PB, CBZ and OXC) induce hepatic enzymes and enhance the metabolism of lipid-soluble drugs. These interact with other AEDs, oral contraceptive pill (OCP) and oral anticoagulants.
- Valproate inhibits hepatic enzymes and slows down the metabolism of concomitant AEDs and other drugs having hepatic metabolism causing toxicity and requiring dose adjustments.
- Drug interactions become important while using AEDs with theophylline group erythromycin, ciprofloxacin or ofloxacin; antitubercular drugs (like isoniazid and rifampicin are enzyme inducers and also hepatotoxic), antiretroviral drugs and mefloquine (Flow chart 2).[104]

METHODOLOGY

Study site: Inpatient department of Pediatrics, Government General Hospital, Kakinada.

Study Duration: 6 Months.

Study Design: Descriptive cross sectional study.

Sample Size: The sample size *n* and margin of error *E* are given by

$x = Z(^{C}/_{100})^2 r(100-r)$

$n = {}^{Nx}/((N-1)E^2 + x)$

$E = \text{Sqrt}[{}^{(N-n)x}/_{n(N-1)}]$

Where N is the population size N = 150,

r is the fraction of responses that you are interested in r = 50%,

Z(C/100) is the critical value for the confidence level c, Z(C/100) = 95%

By applying the above formula the Sample size obtained is 109.

Source of data

Patients, Patient caretakers, case sheets.

A data collection form which suits our present study was framed. It contains demographic data like name, gender, child's age, location, and age of first onset of seizure, symptoms, duration and type of seizures, cause of seizure, final diagnosis, clinical data and treatment.

The questions were asked to the patient or their care taker regarding their past history, family history, symptoms regarding the seizures.

Sampling Technique

Based on the nature and aim of the study, a **purposive sampling technique** which involved using a predefined group of study subjects was used. This sampling technique would enable to obtain specific and relevant information about group of pediatric population with seizures. The selection process can be described as purposive, judgmental based on strict selection criteria for the participants.

Ethical committee approval

Preceding the study, approval for the study was obtained from the pediatric department, Government General Hospital, Kakinada.

Participants

Inclusion criteria

1. Pediatric patients, aged between 5 Months to 6 years and of either sex who presented with seizures with any etiology.
2. Those who are willing to participate in the study.

Exclusion criteria

1. Children who are having diseases other than seizures.
2. Children with age less than 5 Months and more than 6 Years.
3. Seizure cases who expired immediately after hospitalization (before diagnosis).
4. Not willing to give consent.

Data collection and analysis

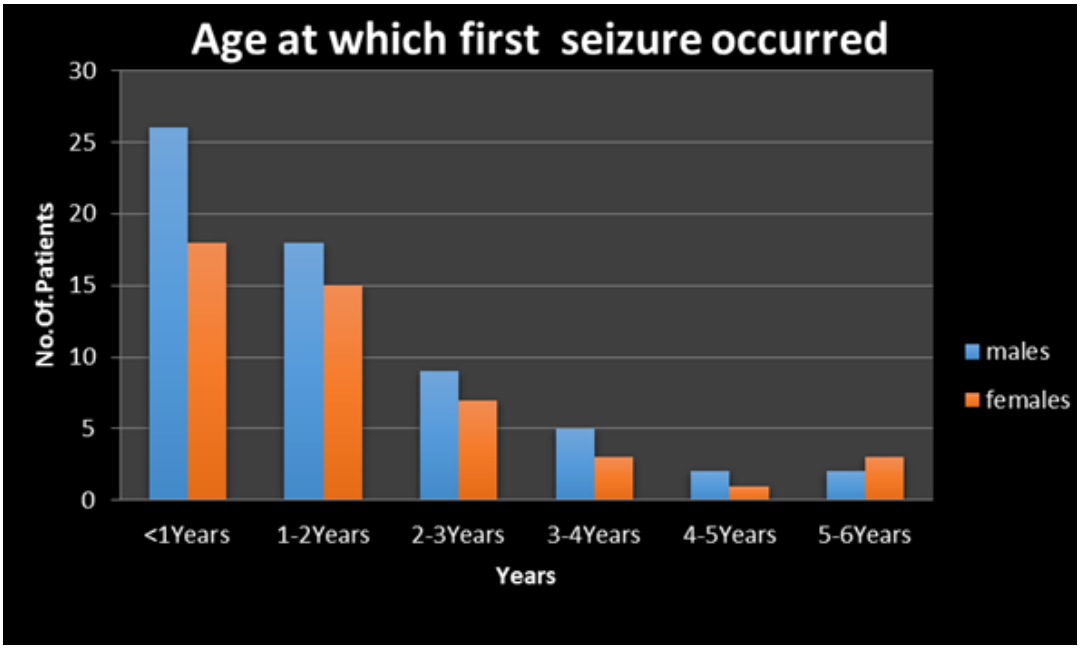
Children of age group 5 Month to 6 Years were selected. The aim and objectives of the study were explained clearly. Upon their will, data was collected from them using already prepared data collection form. The collected data was tabulated periodically till the end of the study period. At the end, the data was analyzed statistically to find the Age at which first Seizure occurred, Type of seizures diagnosed in children, etiology and Type of therapy given for the management of seizures in children of 5 Months to 6 Years. Data is analyses by quantitatively and qualitatively. The data comes under quantitative analysis is Age at which first seizure occurred, which is analysis by frequency and percentage of responses, mode, median, mean, standard deviation and is plotted by using histogram and line graph. The data comes under quantitative analysis is Type of seizures diagnosed in children, Etiology, Type of therapy given for the management of seizures, which is analysis by frequency and percentage of responses and is plotted by using bar chart and pie chart.

RESULTS

Table 1

Age at which first seizure occurred

Age at which first seizure occurred	Males	Females	Total	Percentage
<1 Year	26	18	44	40.3%
1-2 Years	18	15	33	30.3%
2-3 Years	9	7	16	14.6%
3-4 Years	5	3	8	7.3%
4-5 Years	2	1	3	2.7%
5-6 Years	2	3	5	4.5%
Total	62	47	109	100%



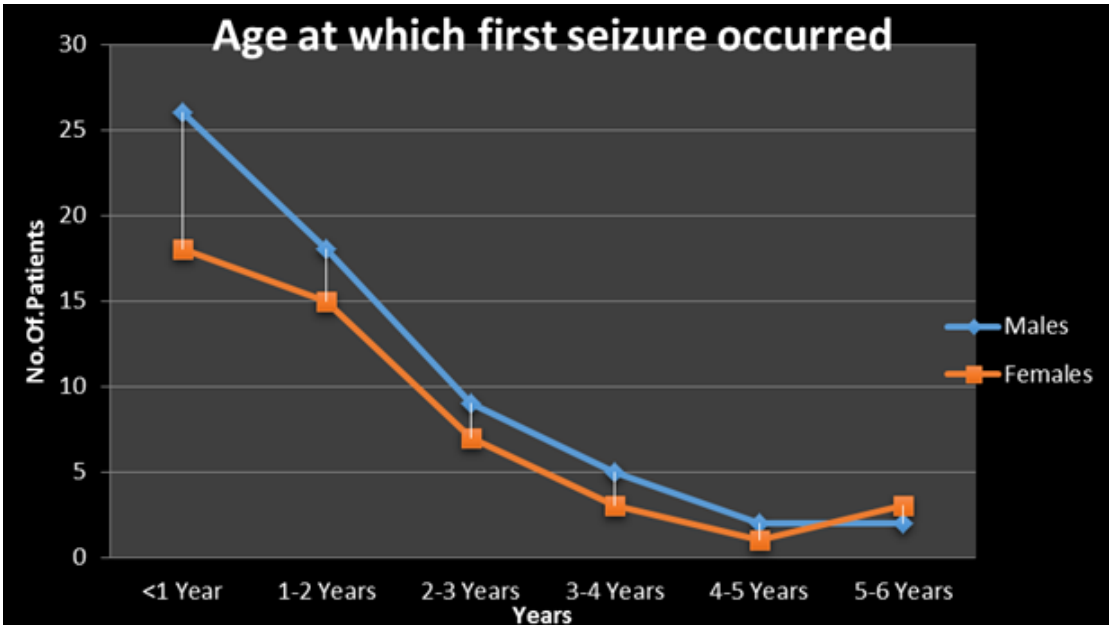


Table 2

Type of seizures diagnosed in children

Type of seizures	Males	Females	Total	Percentage
GTCS	39	29	68	62.3%
Tonic	14	12	26	23.8%
Clonic	2	2	4	3.6%
Partial	5	4	9	8.2%
Absence	1	0	1	0.9%
Myoclonic	0	1	1	0.9%
Total	61	48	109	100%

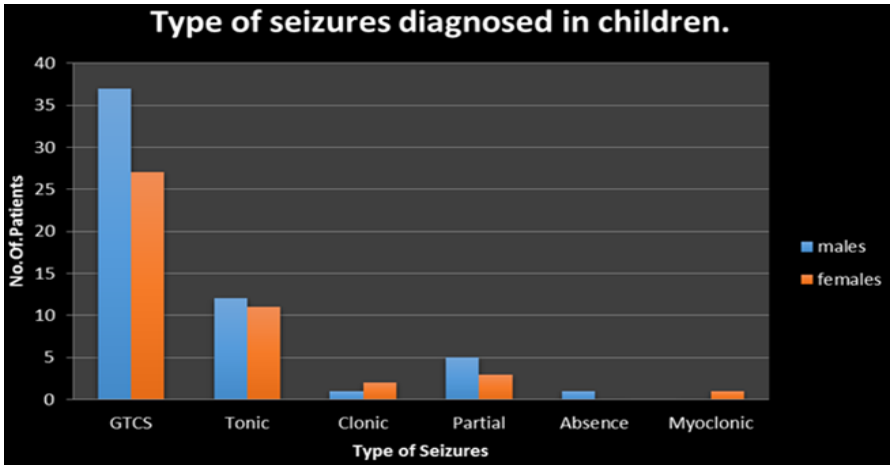


Table 3

Etiology

Etiology	Males	Females	Total	Percentage
Febrile seizures	19	14	33	30.2%
CNS Infections	19	13	32	29.3%
Cerebral palsy	10	9	19	17.4%
Neurodegenerative disorders	5	1	6	5.5%
Metabolic disorders	2	2	4	3.6%
Intracranial hemorrhage	0	2	2	1.8%
Drug withdrawal seizures	0	2	2	1.8%
Tumors	2	0	2	1.8%
Idiopathic	2	3	5	4.5%
Others	3	1	4	3.6%
Total	62	47	109	100%

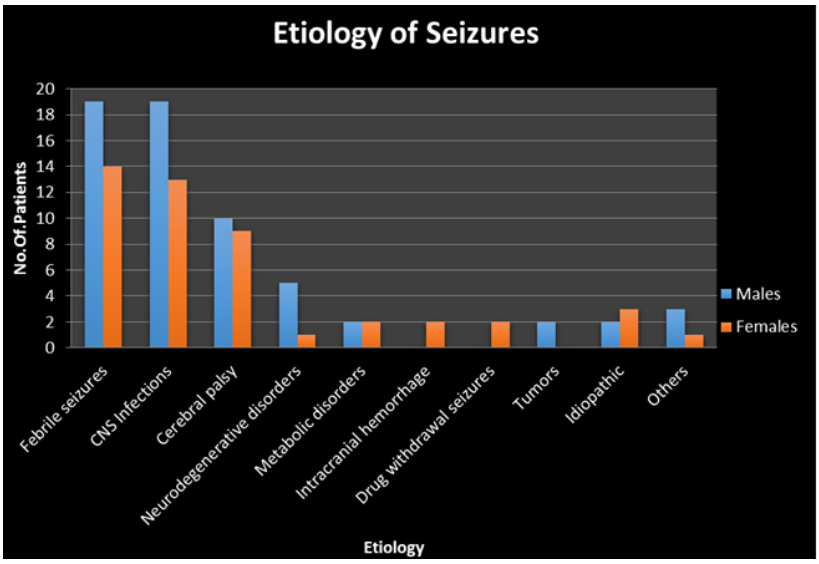
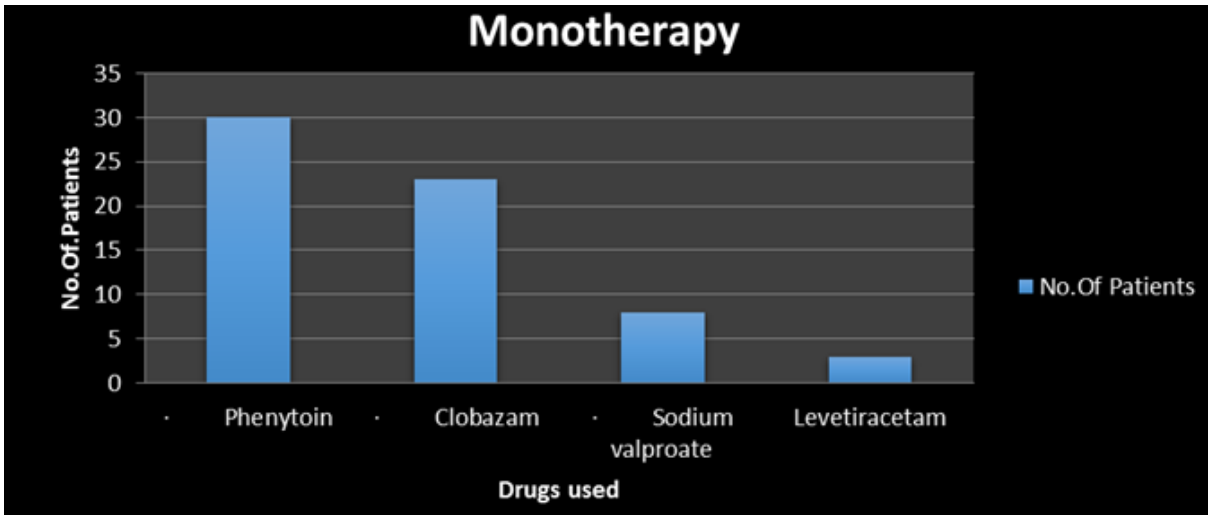
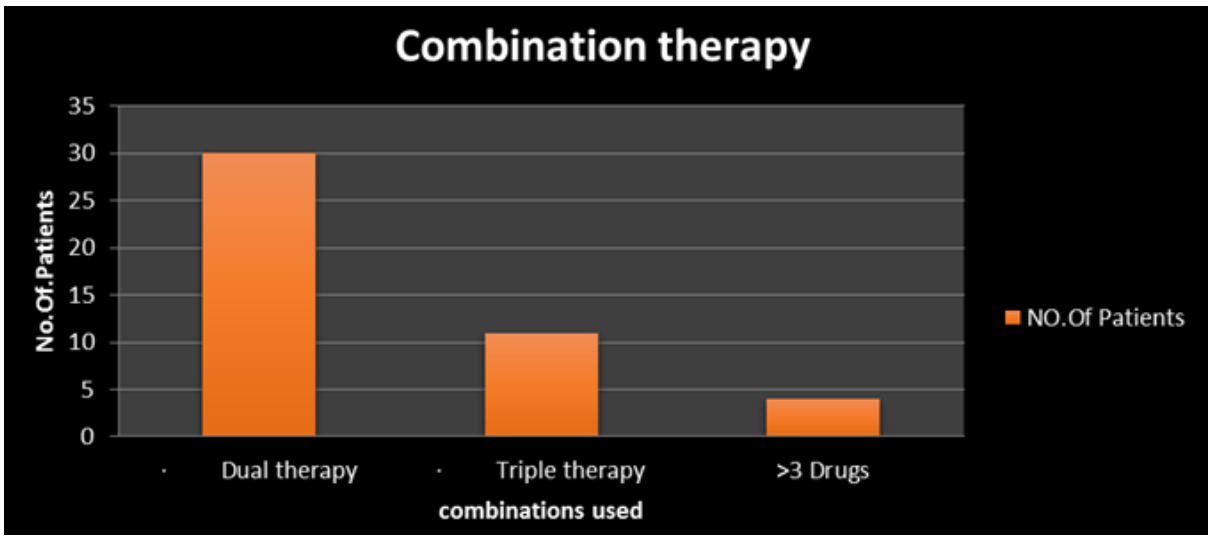


Table 4

Type of therapy

Type of Therapy	No. of patients	Percentage
Monotherapy	64	58.7%
• Phenytoin	30	27.5%
• Clobazam	23	21.1%
• Sodium valproate	8	7.3%
• Levetiracetam	3	2.7%
Combination therapy	45	41.2%
• Dual therapy	30	27.5%
• Triple therapy	11	10.1%
• >3 Drugs	4	3.6%





DISCUSSION

The present study aimed to evaluate the incidence, types, underlying causes, and management approaches of seizures in children aged 5 months to 6 years admitted to the pediatric

I.C.U/ward of a tertiary care hospital. Seizures are a common neurological emergency in children, with febrile seizures being the most frequent in this age group. Our findings revealed that febrile seizures accounted for the majority of seizure presentations, followed by idiopathic epilepsy and structural/metabolic causes such as hypoxic-ischemic encephalopathy, CNS infections, and electrolyte imbalances.

Male children were found to be more frequently affected than females, which aligns with other regional and global studies, possibly due to genetic, hormonal, or sociocultural factors influencing healthcare access. The peak incidence was observed in children between 12 to 24 months, consistent with the natural history of febrile seizure vulnerability.

Etiological analysis indicated that fever-related illnesses such as viral respiratory tract infections and gastroenteritis were major contributors. Cases with hypoglycemia, hypocalcemia, and CNS infections such as meningitis and encephalitis highlighted the importance of early biochemical and radiological evaluation.

Management protocols followed standard guidelines, including the use of benzodiazepines for acute control and antiepileptics like phenytoin and levetiracetam for recurrent or prolonged seizures. Supportive care, antipyretics, and treatment of underlying infections were crucial components of therapy. The majority of patients responded well to treatment, with favorable short-term outcomes, while a small proportion required long-term neurologic follow-up.

Early diagnosis and prompt initiation of treatment significantly improved prognosis and reduced complications. Parental education on recognizing seizure activity, temperature control, and adherence to antiepileptic therapy were emphasized as part of discharge planning.

CONCLUSION

This study concludes that **febrile seizures are the most common type of seizure** in children aged 5 months to 6 years, with **fever-related illnesses being the leading cause**. Male children and those aged 1–2 years are more frequently affected. Prompt identification of the seizure type and underlying etiology is critical for effective management.

Early intervention with anticonvulsants and supportive treatment yields favorable outcomes in the majority of cases. A multidisciplinary approach involving pediatricians, neurologists, and intensive care teams is essential for optimal care. Public health strategies focusing on

early treatment of febrile illnesses, **nutrition**, **infection control**, and **caregiver education** can play a pivotal role in reducing the incidence and complications of seizures in this vulnerable age group.

REFERENCES

1. Verma A, Kunju PAM, Kanhare S, Maheshwari N. IAP Textbook of Pediatric Neurology. New Delhi: Jaypee Brothers Medical Publishers (p) Ltd, 2014; 112.
2. Scher MS. Controversies regarding the neonatal Seizures recognition and epileptic disorders. J Neonat, 2002; 4(2): 139-58.
3. Adhikari S, Sathian B, Koirala DP, Rao KS. Profile of children admitted with seizures in a tertiary care hospital of Western Nepal. BMC Pediatr, 2013; 13: 43. PubMed | Google Scholar
4. Eli M. Neonatal Seizures and neonatal epileptic syndromes. Neurol Clin Epilep, 2001; 19(2): 427-56.
5. David P, De Vivo DC. The nervous system. In: Rudolph Colin D. Abraham D. Rudolph, Margaret K. Hostellter, 21st edi. New York: McGraw Hills, 2002: 2267.
6. Banerjee PN, Filippi D, Allen Hauser W. The descriptive epidemiology of epilepsy-a review. Epilepsy Res, 2009; 85(1): 31- 45.
7. Christopher FL, Westermeyer RR. Seizures in children. emedicinehealth.com
8. Hamdy NA, Ginby D, Feltbower R, Ferrie CD. Ethnic differences in the incidence of seizure disorders in children from Bradford, United Kingdom. Epilepsia, 2007; 48(5); 913–916.
9. Sillanpää M, Shinnar S. Long-term mortality in childhood-onset epilepsy. N Engl J Med., 2010; 23; 363(26): 2522-9.
10. Hauser WA. The prevalence and incidence of convulsive disorders in children. Epilepsia, 1994; 35(2): 1-6. PubMed | Google Scholar
11. Chung B, Wat LC, Wong V. Febrile seizures in southern Chinese children: incidence and recurrence. Pediatr Neurol, 2006 Feb; 34(2): 121-6. PubMed | Google Scholar
12. Panayiotopoulos CP. The new ILAE report on terminology and concepts for the organization of epilepsies: critical review and contribution. Epilepsia, 2012; 53(3): 399-404.
13. Shearer, Peter. "Seizures and Status Epilepticus: Diagnosis and Management in the Emergency Department". Emergency Medicine Practice.

14. Hammer, edited by Stephen J. McPhee, Gary D. (2010). "7". Pathophysiology of disease : an introduction to clinical medicine (6th ed.). New York: McGraw-Hill Medical. ISBN 978-0-07-162167-0
15. Bradley, Walter G. (2012). "67". Bradley's neurology in clinical practice. (6th ed.). Philadelphia, PA: Elsevier/Saunders. ISBN 978-1-4377-0434-1.
16. National Institute for Health and Clinical Excellence (January 2012). "Chapter 9: Classification of seizures and epilepsy syndromes". The Epilepsies: The diagnosis and management of the epilepsies in adults and children in primary and secondary care(PDF). National Clinical Guideline Centre. 119–129.
17. Simon, David A. Greenberg, Michael J. Aminoff, Roger P. (2012). "12". Clinical neurology (8th ed.). New York: McGraw-Hill Medical. ISBN 978-0-07-175905-2
18. Camfield C, Camfield P. Preventable and unpreventable causes of childhood-onset epilepsy plus mental retardation. Pediatrics, 2007; 120(1): e52-5.
19. Shore CP, Buelow JM, Austin JK, Johnson CS. Continuing psychosocial care needs in children with new-onset epilepsy and their parents. J Neurosci Nurs., 2009; 41(5): 244-50.
20. Aguiar BV, Guerreiro MM, McBrian D, Montenegro MA. Seizure impact on the school attendance in children with epilepsy. Seizure, 2007; 16(8): 698-702.
21. Burton K, Rogathe J, Whittaker RG, Mankad K. Co-morbidity of epilepsy in Tanzanian children: a community-based case-control study. Seizure, 2012; 21(3): 169-74.
22. Friedman MJ, Sharieff GQ. Seizures in children. Pediatr Clin North Am., 2006; 53: 257–77.
23. Verity CM, Greenwood R, Golding J. Long-term intellectual and behavioral outcomes of children with febrile convulsions. N Engl J Med., 1998; 338(24): 1723-8. PubMed | Google Scholar
24. Nelson KB, Ellenberg JH. Predictors of epilepsy in children who have experienced febrile seizures. New Engl J Med., 1976; 295: 1029-33.
25. Sadier LG, Scheffer IE. Febrile Seizure. BMJ, 2007; 334: 307-11.
26. Loddenkemper T, Kellinghaus C, Wyllie E, Najm IM, Gupta A, Rosenow F. A proposal for a five-dimensional patient-oriented epilepsy classification. Epileptic Disord., 2005 Dec; 7(4): 308-16.
27. Luders H, Acharya J, Baumgartner C, Benbadis S, Bleasel A, Burgess R, et al. Semiological seizure classification. Epilepsia, 1998 Sep; 39(9): 1006-13.
28. Hauser WA. The prevalence and incidence of convulsive disorder in children. Eplepsia. 1994; 35(Suppl 2): S1-6.

29. American Academy of Pediatrics, Subcommittee on Febrile Seizures. Clinical Practice Guideline – Febrile Seizures: Guideline for the Neurodiagnostic Evaluation of the Child with a Simple Febrile Seizure. Pediatrics, 2011; 127: 389-94.
30. Maytal J, Krauss JM, Novak G, Nagelberg J, Patel M. The role of brain computed tomography in evaluating children with new onset of seizures in the emergency department. Epilepsia, 2000; 41: 950−4.
31. https://en.wikipedia.org/wiki/Cerebral_palsy.
32. Kwong, K., Wong, S. N. and So, K. T. Epilepsy in children with cerebral palsy. Pediatric Neurology, 1998; 19: 313–316.
33. Aksu, F. Nature and prognosis of seizures in patients with cerebral palsy. Developmental Medicine and Child Neurology, 1990; 32: 661–668.
34. Delgado, M. R. Discontinuation of antiepileptic drug treatment after two seizure-free years in children with cerebral palsy. Pediatrics, 1996; 97 (2): 192–197.
35. Report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society on the treatment of a first seizure, Practice Parameter: treatment of the child with a first seizure. Neurology, 2003; 60: 166–175.
36. First Seizure Trial Group. Randomized clinical trial on the efficacy of antiepileptic drugs in reducing the risk of relapse after a first unprovoked tonic–clonic seizure. Neurology, 1993; 43: 478–483.
37. Vickrey, B. G., Gifford, D. R., Belin, T. R. et al. Practice styles of US compared to UK neurologists. Neurology, 1998; 50: 1661–1669.
38. Freeman, J. M., Tibbles, J., Camfield, C. and Camfield, P. Benign epilepsy of childhood: a speculation and its ramifications. Pediatrics, 1987; 79: 864–868.
39. Shinnar, S., Berg, A. T., Moshe, S. L. et al. The risk of recurrence following a first unprovoked seizure in childhood: an expanded follow up. Pediatrics, 1996; 98: 216–225.
40. Shinnar, S., Berg, A. T., Moshe, S. L. et al. The risk of recurrence following a first unprovoked seizure in childhood: a prospective study. Pediatrics, 1990; 85: 1076–1085.
41. Berg, A. and Shinnar, S. The risk of seizure recurrence following a first unprovoked seizure: a quantitative review. Neurology, 1991; 41: 965–972.
42. Lizana, J. R., Garcia, E. C., Carrasco Marina, L. L. et al. Seizure recurrence after a first unprovoked seizure in childhood: a prospective study. Epilepsia, 2000; 41: 1005–1013.

43. Johnson, L. C., DeBolt, W. L., Long, M. T. et al. Diagnostic factors in adult males following initial seizure: a three years follow up. Archives of Neurology, 1972; 27: 193–197.
44. Hauser, W. A., Rich, S. S., Annegers, J. F. and Anderson, V. E. Seizure recurrence after a first unprovoked seizure: an extended follow up. Neurology, 1990; 40: 1163–1170.
45. Hauser, W. A., Anderson, V. E., Loewnson, R. B. and McRoberts, S. M. Seizure recurrence after a first unprovoked seizure. The New England Journal of Medicine, 1982; 307: 522–528.
46. Elwes, R. D. C., Chesterman, P. and Reynolds, E. H. Prognosis after a first untreated tonic–clonic seizure. Lancet, 1985; 2: 752–753.
47. Hopkins, A., Garman, A. and Clarke, C. The first seizure in adult life: value of clinical features, electroencephalography, and computerized tomographic scanning in prediction of seizure recurrence. Lancet, 1988; 1: 721–726.
48. Blom, S., Heijbel, J. and Bergfors, P. G. Incidence of epilepsy in children: a follow up study three years after the first seizure. Epilepsia, 1978; 19: 343–350.
49. Hirtz, D. G., Ellinberg, J. H. and Nelson, K. B. The risk of recurrence of nonfebrile seizures in children. Neurology, 1984; 34: 637–641.
50. Camfield, P. R., Camfield, C. S., Dooly, J. M. et al. Epilepsy after a first unprovoked seizure in childhood. Neurology, 1985; 35: 1657–1660.
51. Annegers, J. F., Shirts, S. B., Hauser, W. A. and Kurland, L. T. Risk of recurrence after an initial unprovoked seizure. Epilepsia, 1986; 27: 43-50.
52. Boulloche, I., Leloup, P., Mallet, E., Parain, D. and Torn, P. Risk of recurrence after a single unprovoked generalized tonic–clonic seizure. Developmental Medicine and Child Neurology, 1989; 31: 626–632.
53. Hui, A. C. F., Tang, A., Wong, K. S., Mok, V. and Kay, R. Recurrence after a first untreated seizure in Hong Kong Chinese population. Epilepsia, 2001; 42: 94–97.
54. Report of the Quality Standard Subcommittee of the American Academy of Neurology, the Child Neurology Society, and the American Epilepsy Society on the evaluation of a first seizure in childhood. Neurology, 2000; 55: 616–623.
55. Altunbas¸ ak S, Artar O, Burgut R, Yildiztas¸ D. Relapse risk analysis after drug withdrawal in epileptic children with uncomplicated seizures. Seizurem, 1999; 8: 384–9.