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Original Research Article

Clinical Profile, Aetiological Spectrum and Short-Term Outcome of Seizures in Children Aged One Month to Twelve Years Admitted to a Tertiary Care Centre: A Prospective Observational Study

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Abstract

Background: Seizures are among the commonest neurological emergencies in paediatric practice, and the aetiological spectrum differs substantially between developed and developing countries and between age strata within childhood. Accurate characterisation of the local clinical and aetiological profile guides rational investigation and early treatment. This study was undertaken to describe the clinical presentation, seizure semiology, aetiology and short-term outcome of seizures in children aged one month to twelve years admitted to a tertiary care centre.

Methods: A prospective hospital-based observational study enrolled 150 consecutive children aged one month to twelve years admitted with seizures over an eighteen-month period. Demographic details, seizure characteristics, examination findings, laboratory results, cerebrospinal fluid analysis, electroencephalography and neuroimaging were recorded on a structured proforma. Seizures were classified according to the 2017 International League Against Epilepsy operational classification. Categorical associations were tested by the chi-square test and the Fisher exact test.

Results: The male to female ratio was 1.46 to 1 and 47.3 percent of children were aged one to five years. Generalised seizures predominated (72.0 percent), of which generalised tonic-clonic seizures accounted for 54.7 percent of the total. Febrile seizures were the commonest aetiology (37.3 percent), followed by central nervous system infection (18.0 percent), idiopathic epilepsy (16.0 percent) and metabolic causes (12.0 percent). Status epilepticus occurred in 14.0 percent. Complete recovery was achieved in 84.0 percent, neurological deficit persisted in 8.7 percent and mortality was 3.3 percent. Adverse outcome was strongly associated with aetiology ($p < 0.001$) and with status epilepticus ($p = 0.002$).

Conclusion: Febrile seizures and central nervous system infection together accounted for more than half of all paediatric seizure admissions, and aetiology rather than seizure semiology determined outcome. Early identification of the underlying cause, prompt exclusion of treatable metabolic derangement and infection, and aggressive management of status epilepticus are the principal determinants of a favourable short-term outcome.

Keywords: Seizures; Child; Epilepsy; Seizures, febrile; Status epilepticus; Central nervous system infections.

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Introduction

A seizure is the transient occurrence of signs or symptoms arising from abnormal excessive or synchronous neuronal activity in the brain, and it constitutes one of the most frequent reasons for acute paediatric neurological consultation and hospital admission. The immature brain is inherently more susceptible to seizure generation than the mature brain, a susceptibility attributable to the delayed maturation of inhibitory gamma-aminobutyric acid mediated neurotransmission relative to excitatory glutamatergic transmission, to the depolarising rather than hyperpolarising action of gamma-aminobutyric acid in early life, and to incomplete myelination. Consequently the incidence of seizures is highest in the first years of life and declines progressively through later childhood.The classification of seizures and of the epilepsies was substantially revised by the International League Against Epilepsy in 2017. The operational classification of seizure types replaced the term partial with focal, introduced awareness as a classifier of focal seizures, eliminated the terms simple partial, complex partial, dyscognitive and secondarily generalised, and permitted classification where the onset was unobserved, a provision of particular value in paediatric practice

where the onset of a seizure is frequently unwitnessed.[1] The companion classification of the epilepsies established a three-level framework proceeding from seizure type to epilepsy type to epilepsy syndrome, and required that aetiology be considered at every level because of its immediate therapeutic implications.[2] These documents built upon the practical clinical definition of epilepsy published in 2014 and upon the revised terminology of 2010.[3,4] The definition and classification of status epilepticus was similarly revised, with operational time points of five minutes for the initiation of treatment and thirty minutes for the risk of long-term consequences in the case of convulsive status epilepticus.[5]

The aetiological spectrum underlying childhood seizures differs markedly between settings, and this variation is the principal reason why locally derived data retain their value despite the abundance of international literature. In high-income countries, febrile seizures and genetic generalised epilepsies predominate, and the American Academy of Pediatrics guidelines on the neurodiagnostic evaluation and the long-term management of simple febrile seizures reflect a context in which central nervous system infection is uncommon and in which routine lumbar puncture, electroencephalography and neuroimaging are explicitly discouraged after an uncomplicated simple febrile seizure.[6,7] In low-income and middle-income settings the calculus is different. Central nervous system infections, including bacterial meningitis, viral encephalitis, cerebral malaria, tuberculous meningitis and neurocysticercosis, contribute a far larger share of the burden, and metabolic derangements arising from malnutrition, diarrhoeal dehydration and rickets remain important and eminently reversible causes. The Kenyan cohort reported by Sadarangani and colleagues illustrates this divergence starkly, with an incidence of convulsive status epilepticus and an associated mortality far exceeding those observed in the United Kingdom population-based study of Chin and colleagues.[8,9] Neurocysticercosis in particular has been documented as a leading cause of afebrile seizures in several Indian series, with implications for imaging strategy that do not apply in Western practice.[10]

Outcome is likewise heterogeneous and is determined principally by aetiology rather than by seizure semiology. Population-based and hospital-based data consistently show that the child with a simple febrile seizure faces an excellent prognosis, whereas the child whose seizure is symptomatic of acute central nervous system infection or of an underlying structural lesion carries a substantial risk of neurological sequelae or death. Status epilepticus superimposes an additional and independent hazard, with reported case fatality varying by an order of magnitude between settings.[11,12] These observations argue that the initial diagnostic effort in a child presenting with seizures should be directed towards the rapid identification of aetiology, and that the intensity of investigation should be calibrated to the local prevalence of treatable and dangerous causes rather than transplanted uncritically from guidelines written for a different epidemiological context.

Published Indian data on the clinical and aetiological profile of childhood seizures remain fragmented, drawn largely from single-centre series of varying methodology, using classification systems that predate the 2017 revision, and often restricted either to status epilepticus or to first seizures rather than encompassing the whole spectrum of seizure admissions. Data from the semi-urban and rural catchment served by medical colleges in southern Karnataka are particularly sparse. The present study was therefore undertaken to describe systematically the demographic profile, clinical presentation, seizure semiology classified according to current criteria, aetiological spectrum, investigative yield and short-term outcome of children aged one month to twelve years admitted with seizures to a tertiary care centre, and to examine which of these variables were associated with an unfavourable outcome.

Aims and Objectives

The study aimed to characterise comprehensively the presentation and course of seizures in children admitted to a tertiary care paediatric service, so that the local pattern of disease could inform investigation and management. The primary objective was to describe the aetiological spectrum of seizures in children aged one month to twelve years admitted with seizures, and to determine the relative contribution of febrile seizures, central nervous system infection, idiopathic epilepsy, metabolic derangement, structural and perinatal causes, and trauma. The secondary objectives were to describe the demographic profile of the affected children including age distribution, sex distribution and relevant antenatal, natal and developmental history, to classify the observed seizures according to the 2017 International League Against Epilepsy operational classification of seizure types, and to document the clinical features accompanying the seizure including the presence of fever, seizure duration, number of episodes before admission, postictal state and findings on neurological examination.

A further objective was to determine the diagnostic yield of the investigations employed, comprising haematological and biochemical tests, cerebrospinal fluid analysis, electroencephalography and neuroimaging, and to

record the short-term outcome at discharge in terms of complete recovery, residual neurological deficit, referral to a higher centre or death. The final objective was to examine the association between outcome and the aetiological category, the occurrence of status epilepticus, and the age of the child.

Materials and Methods

Study Design and Setting: This prospective, hospital-based, observational descriptive study was conducted in the Department of Paediatrics of the Adichunchanagiri Institute of Medical Sciences, B. G. Nagara, Mandya District, Karnataka, over an eighteen-month period from January 2024 to June 2025. The institution is a tertiary care teaching hospital serving a predominantly rural and semi-urban catchment population, with a paediatric ward, a paediatric intensive care unit and facilities for electroencephalography, computed tomography and magnetic resonance imaging. Approval of the Institutional Ethics Committee was obtained before the commencement of enrolment. Written informed consent was obtained from a parent or legal guardian of every child before inclusion, and assent was obtained from children of sufficient age and understanding. The study was conducted in accordance with the principles of the Declaration of Helsinki and with the National Ethical Guidelines for Biomedical and Health Research Involving Children issued by the Indian Council of Medical Research. No investigation was performed solely for the purposes of the study, and all tests undertaken formed part of standard clinical care as determined by the treating team.

Inclusion Criteria: All children aged one month to twelve years admitted to the paediatric ward or the paediatric intensive care unit with one or more seizures, whether the seizure occurred before or after admission, and whose parent or legal guardian consented to participation, were eligible for inclusion. Children with a previously established diagnosis of epilepsy who were admitted with a breakthrough seizure were included, and the pre-existing diagnosis was recorded.

Exclusion Criteria: Neonates aged below one month and children aged above twelve years were excluded. Children whose paroxysmal event was determined on evaluation to be a non-epileptic phenomenon, including breath-holding spells, syncope, benign paroxysmal vertigo, night terrors, shuddering attacks, tics and psychogenic non-epileptic events, were excluded. Further exclusions comprised children whose parent or guardian declined consent, children who left against medical advice before completion of the basic evaluation, and children in whom the record of the seizure event was insufficient to permit classification.

Sample Size Calculation: The sample size was calculated for the estimation of the proportion of children in whom febrile seizure was the underlying aetiology, this being the commonest single category reported in comparable paediatric series. The formula for a single proportion with specified absolute precision was applied:

$$n = Z^2_{1-\alpha/2} \times p\,(1 - p) / d^2$$

where $Z_{1-\alpha/2}$ was 1.96 corresponding to a 95 percent confidence level, p was the anticipated proportion of febrile seizures taken as 40 percent on the basis of previously published Indian data, and d was the absolute precision, set at 8 percent.

$$n = (1.96)^2 \times 0.40 \times 0.60 / (0.08)^2$$

$$n = 3.8416 \times 0.24 / 0.0064$$

$$n = 0.9220 / 0.0064 = 144.1$$

The minimum required sample size was therefore 145 children. This was rounded upward to 150 to allow for incomplete records and to provide adequate cell frequencies for the planned subgroup analyses by age stratum. Consecutive sampling was employed until the target was reached.

Study Procedure and Data Collection: Every eligible child was evaluated by the investigator using a pre-designed and pre-tested structured proforma. A detailed history was obtained from the parent or the accompanying caregiver and, where available, from an eyewitness to the seizure. The history recorded the age at onset, the description and duration of the seizure, the number of episodes preceding admission, the presence and duration of fever, the postictal state, any history of preceding illness, trauma, drug ingestion or toxin exposure, the antenatal and perinatal history including birth asphyxia and neonatal intensive care admission, the developmental history assessed against standard milestones, the immunisation status, the dietary and nutritional history, the family history of seizures or epilepsy, and the presence of consanguinity between the parents.

A complete general physical examination was performed, recording anthropometry plotted against Indian Academy of Pediatrics growth charts, vital parameters, the presence of pallor, features of malnutrition or rickets, cutaneous stigmata of neurocutaneous syndromes, and dysmorphic features. A detailed neurological examination assessed the level of consciousness, cranial nerves, motor and sensory systems, deep tendon reflexes, plantar responses, meningeal signs and the fundus. Anterior fontanelle status was recorded in infants. Systemic examination of the cardiovascular, respiratory and abdominal systems was performed in all children. Investigations were performed as clinically indicated by the treating team. Complete blood count, random blood glucose, serum

electrolytes including sodium, potassium, calcium and magnesium, renal function tests and, where fever was present, blood culture and peripheral smear for malarial parasite were obtained in all children. Cerebrospinal fluid analysis was performed where central nervous system infection was suspected and no contraindication existed. Electroencephalography was performed in children with afebrile seizures, recurrent seizures, focal seizures or abnormal neurological findings. Neuroimaging by computed tomography or magnetic resonance imaging was performed in children with focal seizures, focal neurological deficit, raised intracranial pressure, refractory seizures, developmental regression or suspected structural or infective aetiology.

Definitions and Classification: Seizures were classified according to the 2017 International League Against Epilepsy operational classification of seizure types into focal onset, generalised onset and unknown onset categories, with focal seizures further characterised by the preservation or impairment of awareness, and the instruction manual accompanying the classification was used to resolve ambiguous events.[13] A febrile seizure was defined as a seizure occurring in a child between six months and sixty months of age in association with a fever of 38 degrees Celsius or above, in the absence of central nervous system infection, metabolic derangement or a history of afebrile seizures. A simple febrile seizure was one that was generalised, lasted less than fifteen minutes and did not recur within twenty-four hours, and a febrile seizure not meeting all three criteria was classified as complex. Convulsive status epilepticus was defined as a continuous convulsive seizure lasting five minutes or longer, or two or more discrete seizures between which there was incomplete recovery of consciousness.[5]

Epilepsy was diagnosed in accordance with the 2014 practical clinical definition.[3] Central nervous system infection was diagnosed on the basis of clinical features supported by cerebrospinal fluid analysis, culture, serology or characteristic neuroimaging appearances as appropriate. A metabolic aetiology was assigned where a biochemical derangement of sufficient magnitude to account for the seizure was documented and the seizure did not recur following its correction.

Outcome at discharge was categorised as complete recovery, defined as resolution of the seizure with a normal neurological examination at discharge; recovery with neurological deficit, defined as the persistence of any motor, sensory, cognitive or visual deficit attributable to the illness; referral to a higher centre for care not available at the institution; or death during the admission.

Statistical Analysis: Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics version 26.0. Continuous variables were tested for normality by the Shapiro-Wilk test, and were expressed as mean with standard deviation where normally distributed and as median with interquartile range where not. Categorical variables were expressed as frequencies with percentages. Associations between categorical variables were tested by the Pearson chi-square test, with the Fisher exact test substituted where any expected cell frequency was below five, and the chi-square test for linear trend was used where an ordered exposure was examined. A two-tailed p value below 0.05 was considered statistically significant.

Results

Table 1: Demographic and background characteristics of the study population (N = 150)

Variable	Number	Percentage
Age group		
1 month to 1 year	34	22.7
>1 year to 5 years	71	47.3
>5 years to 12 years	45	30.0
Mean age, years (mean ± SD)	3.8 ± 3.2	—
Sex		
Male	89	59.3
Female	61	40.7
Male to female ratio	1.46 : 1	—
Background history		
Family history of seizures or epilepsy	26	17.3
Parental consanguinity	22	14.7
History of birth asphyxia	19	12.7
Neonatal intensive care admission	16	10.7
Developmental delay	24	16.0
Moderate or severe undernutrition	31	20.7
Incomplete immunisation for age	18	12.0
Known epilepsy on antiseizure medication	21	14.0

SD, standard deviation. Percentages are calculated on the total study population of 150 children.

Table 2: Clinical presentation and seizure characteristics (N = 150)

Clinical feature	Number	Percentage
Fever at presentation	92	61.3
Seizure duration exceeding 5 minutes	48	32.0
More than one episode before admission	61	40.7
Convulsive status epilepticus	21	14.0
Postictal drowsiness exceeding 30 minutes	98	65.3
Postictal focal weakness	11	7.3
Altered sensorium on admission	38	25.3
Meningeal signs	24	16.0
Focal neurological deficit	17	11.3
Papilloedema	6	4.0
Bulging anterior fontanelle (infants, n = 34)	7	20.6
Vomiting	52	34.7
Headache (children able to report, n = 61)	19	31.1
Seizure onset to hospital arrival, min, median (IQR)	65 (40–130)	—

IQR, interquartile range. Denominators differ where indicated; all other percentages are calculated on 150 children.

Table 3: Seizure semiology classified according to the 2017 ILAE operational classification (N = 150)

Seizure type	Number	Percentage
Generalised onset	108	72.0
Generalised tonic-clonic	82	54.7
Tonic	11	7.3
Clonic	6	4.0
Myoclonic	5	3.3
Atonic	2	1.3
Absence	2	1.3
Focal onset	32	21.3
Focal, awareness preserved	14	9.3
Focal, awareness impaired	11	7.3
Focal to bilateral tonic-clonic	7	4.7
Unknown onset	10	6.7
Epileptic spasms	4	2.7
Unclassified (onset unwitnessed)	6	4.0
Total	150	100.0

ILAE, International League Against Epilepsy. Distribution by sex: $\chi^2 = 1.24$, $p = 0.538$. Distribution by age stratum: $\chi^2 = 14.82$, $p = 0.005$.

Table 4: Aetiological spectrum stratified by age group (N = 150)

Aetiology	1 mo–1 yr (n = 34)	>1–5 yr (n = 71)	>5–12 yr (n = 45)	Total	%
Febrile seizure	8	41	7	56	37.3
Simple febrile	5	29	5	39	26.0
Complex febrile	3	12	2	17	11.3
CNS infection	9	12	6	27	18.0
Pyogenic meningitis	6	5	1	12	8.0
Meningoencephalitis	3	5	1	9	6.0
Neurocysticercosis	0	1	3	4	2.7
Tuberculoma	0	1	1	2	1.3
Idiopathic epilepsy	3	8	13	24	16.0
Metabolic	9	5	4	18	12.0
Hypoglycaemia	4	1	1	6	4.0
Hypocalcaemia	3	1	1	5	3.3
Hyponatraemia	1	2	2	5	3.3
Hypernatraemia	1	1	0	2	1.3
Structural or perinatal	4	4	7	15	10.0
Head trauma	0	1	4	5	3.3
Others or undetermined	1	0	4	5	3.3
Total	34	71	45	150	100.0

CNS, central nervous system. Association between aetiological category and age stratum: $\chi^2 = 51.34$, degrees of freedom 12, $p < 0.001$. Subcategory rows are components of the preceding major category and are not counted separately in the totals.

Table 5: Laboratory, electroencephalographic and neuroimaging findings (N = 150)

Investigation	Performed, n	Abnormal, n	Yield (%)
Haemoglobin below 11 g/dL	150	68	45.3
Total leucocyte count above 15,000/mm³	150	41	27.3
Random blood glucose below 45 mg/dL	150	6	4.0
Serum calcium below 8 mg/dL	150	5	3.3
Serum sodium outside reference range	150	7	4.7
Blood culture positive	92	11	12.0
Cerebrospinal fluid analysis abnormal	48	21	43.8
Electroencephalography abnormal	74	39	52.7
Generalised epileptiform discharges	—	22	29.7
Focal epileptiform discharges	—	13	17.6
Non-specific slowing	—	4	5.4
Neuroimaging abnormal	62	24	38.7
Ring-enhancing lesion	—	6	9.7
Cerebral atrophy	—	5	8.1
Infarction	—	4	6.5
Malformation of cortical development	—	4	6.5
Hydrocephalus	—	3	4.8
Intracranial haemorrhage	—	2	3.2

Yield is expressed as a percentage of the number in whom that investigation was performed. Neuroimaging yield by seizure type: focal seizures 16 of 29 (55.2%) versus generalised seizures 7 of 28 (25.0%), $\chi^2 = 5.42$, $p = 0.020$.

Table 6: Short-term outcome at discharge and its association with aetiology and status epilepticus (N = 150)

Category	Complete recovery, n (%)	Adverse outcome, n (%)	Deaths, n	Test statistic	p
Overall outcome	126 (84.0)	24 (16.0)	5	—	—
Recovered completely	126 (84.0)	—	—		
Neurological deficit	—	13 (8.7)	—		
Referred to higher centre	—	6 (4.0)	—		
Died	—	5 (3.3)	5		
Outcome by aetiology				$\chi^2 = 34.82$	<0.001
Febrile seizure (n = 56)	55 (98.2)	1 (1.8)	0		
CNS infection (n = 27)	15 (55.6)	12 (44.4)	3		
Idiopathic epilepsy (n = 24)	22 (91.7)	2 (8.3)	0		
Metabolic (n = 18)	16 (88.9)	2 (11.1)	0		
Structural or perinatal (n = 15)	9 (60.0)	6 (40.0)	2		
Head trauma (n = 5)	4 (80.0)	1 (20.0)	0		
Others (n = 5)	5 (100.0)	0 (0.0)	0		
Outcome by status epilepticus				$\chi^2 = 24.61$	<0.001
Status epilepticus (n = 21)	10 (47.6)	11 (52.4)	4		
No status epilepticus (n = 129)	116 (89.9)	13 (10.1)	1		
Outcome by age stratum				$\chi^2 = 9.13$	0.010
1 month to 1 year (n = 34)	23 (67.6)	11 (32.4)	3		
>1 to 5 years (n = 71)	64 (90.1)	7 (9.9)	1		
>5 to 12 years (n = 45)	39 (86.7)	6 (13.3)	1		
Course of admission					
PICU admission	28 (18.7)	—	—		
Invasive mechanical ventilation	9 (6.0)	—	—		
Hospital stay, days (mean ± SD)	5.4 ± 3.1	—	—		

CNS, central nervous system; PICU, paediatric intensive care unit; SD, standard deviation. Adverse outcome is the composite of persistent neurological deficit, referral to a higher centre and death. Mortality in status epilepticus versus no status epilepticus: 4 of 21 (19.0%) versus 1 of 129 (0.8%), Fisher exact test p = 0.002.

Demographic Profile: One hundred and sixty-three children admitted with seizures were screened during the study period. Thirteen were excluded, seven because the paroxysmal event was subsequently determined to be non-epileptic, four because consent was declined, and two because the description of the event was inadequate for classification. One hundred and fifty children constituted the study population. The mean age was 3.8 ± 3.2 years. Thirty-four children (22.7 percent) were aged between one month and one year, 71 children (47.3 percent) between one and five years, and 45 children (30.0 percent) between five and twelve years. Eighty-nine children (59.3 percent) were male and 61 (40.7 percent) female, giving a male to female ratio of 1.46 to 1. A family history of seizures or epilepsy was elicited in 26 children (17.3 percent) and parental consanguinity in 22 (14.7 percent). A history of birth asphyxia was present in 19 children (12.7 percent) and developmental delay was documented in 24 (16.0 percent). Moderate or severe undernutrition was present in 31 children (20.7 percent). Demographic and background characteristics are presented in Table 1.

Clinical Presentation: Fever accompanied the seizure in 92 children (61.3 percent). The seizure had lasted more than five minutes in 48 children (32.0 percent), and 61 children (40.7 percent) had experienced more than one episode before reaching hospital. Convulsive status epilepticus was documented in 21 children (14.0 percent). Postictal drowsiness lasting more than thirty minutes was observed in 98 children (65.3 percent), and postictal focal weakness in 11 children (7.3 percent). Altered sensorium at the time of admission was present in 38 children (25.3 percent), meningeal signs in 24 (16.0 percent) and focal neurological deficit in 17 (11.3 percent). Papilloedema was detected in 6 children (4.0 percent). The median interval from the onset of the seizure to arrival at hospital was 65 minutes with an interquartile range of 40 to 130 minutes. Clinical presentation data are set out in Table 2.

Seizure Semiology: Generalised onset seizures accounted for 108 children (72.0 percent), focal onset seizures for 32 children (21.3 percent), and seizures of unknown onset, comprising epileptic spasms and unwitnessed events not otherwise classifiable, for 10 children (6.7 percent). Among generalised seizures, the generalised tonic-clonic type was overwhelmingly predominant, occurring in 82 children and representing 54.7 percent of the entire cohort, followed by tonic seizures in 11 children (7.3 percent), clonic in 6 (4.0 percent), myoclonic in 5 (3.3 percent), and atonic and absence seizures in 2 children each (1.3 percent each). Among focal seizures, 14 children (9.3 percent) had focal seizures without impairment of awareness, 11 (7.3 percent) had focal seizures with impaired awareness, and 7 (4.7 percent) had focal to bilateral tonic-clonic seizures. The distribution of seizure semiology did not differ significantly between the sexes ($\chi^2 = 1.24$, $p = 0.538$) but did differ significantly across age strata ($\chi^2 = 14.82$, $p = 0.005$), with focal seizures being proportionately commoner in children above five years. Seizure semiology is detailed in Table 3.

Aetiological Spectrum: Febrile seizure was the commonest single aetiology, accounting for 56 children (37.3 percent), of whom 39 (26.0 percent of the cohort) had simple and 17 (11.3 percent) complex febrile seizures. Central nervous system infection was the second commonest cause, identified in 27 children (18.0 percent), comprising pyogenic meningitis in 12, meningoencephalitis in 9, neurocysticercosis in 4 and tuberculoma in 2. Idiopathic epilepsy accounted for 24 children (16.0 percent) and metabolic derangement for 18 children (12.0 percent), the latter comprising hypoglycaemia in 6, hypocalcaemia in 5, hyponatraemia in 5 and hypernatraemia in 2. Structural and perinatal causes, encompassing cerebral palsy with symptomatic epilepsy, sequelae of hypoxic ischaemic encephalopathy and congenital malformations of cortical development, accounted for 15 children (10.0 percent). Head trauma accounted for 5 children (3.3 percent) and miscellaneous causes including hypertensive encephalopathy, drug and toxin exposure and undetermined aetiology for a further 5 children (3.3 percent).

The distribution of aetiology varied markedly and significantly across age strata ($\chi^2 = 51.34$, degrees of freedom 12, $p < 0.001$). Febrile seizures were concentrated in the one to five year stratum, in which 41 of 71 children (57.7 percent) had this aetiology, and were uncommon above five years, accounting for only 7 of 45 children (15.6 percent). Metabolic causes were disproportionately represented in infancy, accounting for 9 of 34 infants (26.5 percent) compared with 5 of 71 children (7.0 percent) aged one to five years and 4 of 45 children (8.9 percent) above five years. Idiopathic epilepsy showed the reverse gradient, accounting for 13 of 45 children (28.9 percent) above five years compared with 3 of 34 infants (8.8 percent). Central nervous system infection was distributed relatively evenly across the three strata. The aetiological distribution stratified by age is presented in Table 4.

Investigative Findings: Anaemia, defined as a haemoglobin concentration below 11 g/dL, was present in 68 children (45.3 percent). Random blood glucose was below 45 mg/dL in 6 children (4.0 percent), serum calcium was below 8 mg/dL in 5 children (3.3 percent), and serum sodium was outside the reference range in 7 children (4.7 percent). Cerebrospinal fluid analysis was performed in 48 children (32.0 percent) and yielded abnormal findings in 21 (43.8 percent of those tested), the commonest abnormality being pleocytosis with raised protein. Electroencephalography was performed in 74 children (49.3 percent) and was abnormal in 39 (52.7 percent of those tested), with generalised epileptiform discharges in 22, focal epileptiform discharges in 13 and non-specific slowing in 4.

Neuroimaging by computed tomography or magnetic resonance imaging was performed in 62 children (41.3 percent) and demonstrated an abnormality in 24 (38.7 percent of those imaged), comprising ring-enhancing lesions in 6, cerebral atrophy in 5, infarction in 4, malformation of cortical development in 4, hydrocephalus in 3 and intracranial haemorrhage in 2.

The yield of neuroimaging was substantially higher among children with focal seizures, in whom 16 of 29 scans performed (55.2 percent) were abnormal, than among those with generalised seizures, in whom 7 of 28 scans (25.0 percent) were abnormal ($\chi^2 = 5.42$, $p = 0.020$). Investigative findings appear in Table 5.

Outcome and its Determinants: Twenty-eight children (18.7 percent) required admission to the paediatric intensive care unit and 9 children (6.0 percent) required invasive mechanical ventilation. The mean duration of hospital stay was 5.4 ± 3.1 days. At discharge, 126 children (84.0 percent) had recovered completely, 13 children (8.7 percent) had a persistent neurological deficit, 6 children (4.0 percent) were referred to a higher centre for further care, and 5 children (3.3 percent) died during the admission.

Outcome was strongly and significantly associated with aetiology ($\chi^2 = 34.82$, degrees of freedom 6, $p < 0.001$). An adverse outcome, defined as the composite of persistent neurological deficit, referral or death, occurred in 12 of 27 children with central nervous system infection (44.4 percent) and in 6 of 15 children with structural or perinatal causes (40.0 percent), compared with only 1 of 56 children with febrile seizures (1.8 percent). Adverse outcome occurred in 2 of 24 children with idiopathic epilepsy (8.3 percent), 2 of 18 with metabolic causes (11.1 percent) and 1 of 5 with head trauma (20.0 percent).

Status epilepticus was likewise strongly associated with outcome. Four of the 21 children presenting in status epilepticus died (19.0 percent), compared with 1 of the 129 children who did not present in status epilepticus (0.8 percent), a highly significant difference (Fisher exact test, $p = 0.002$). An adverse composite outcome occurred in 11 of 21 children with status epilepticus (52.4 percent) compared with 13 of 129 without (10.1 percent) ($\chi^2 = 24.61$, $p < 0.001$). Adverse outcome was also commoner among infants, occurring in 11 of 34 children below one year (32.4 percent) compared with 7 of 71 children aged one to five years (9.9 percent) and 6 of 45 children above five years (13.3 percent) ($\chi^2 = 9.13$, $p = 0.010$). All five deaths occurred in children with central nervous system infection or structural pathology, and none occurred in a child with a febrile or metabolic seizure. Outcome data and their determinants are presented in Table 6.

Discussion

This prospective study of 150 children aged one month to twelve years admitted with seizures found a male preponderance, a concentration of cases in the one to five year age group, a clear predominance of generalised tonic-clonic semiology, and an aetiological spectrum in which febrile seizures and central nervous system infection together accounted for more than half of all admissions. Outcome was determined principally by aetiology and by the occurrence of status epilepticus rather than by seizure type, and mortality was confined entirely to children with central nervous system infection or structural pathology.

The male preponderance of 1.46 to 1 observed here is consistent with the great majority of published paediatric seizure series and is generally attributed to a combination of biological susceptibility and, in South Asian settings, differential health-seeking behaviour favouring male children. The concentration of cases in the one to five year stratum, which contained 47.3 percent of the cohort, reflects the age-specific incidence of febrile seizures and the heightened seizure susceptibility of the immature brain, and accords with the reported age distribution in comparable series from India and Nepal.[14]

The predominance of generalised seizures, at 72.0 percent, with generalised tonic-clonic events accounting for 54.7 percent of the whole cohort, is a consistent finding across the paediatric literature. It is nevertheless likely to represent an overestimate of the true proportion of generalised onset events. Focal onset seizures with rapid bilateral spread are readily misclassified as generalised where the onset is unwitnessed or where the caregiver history is imprecise, a limitation explicitly acknowledged in the 2017 operational classification, which introduced the unknown onset category partly to

accommodate this difficulty.[1,13] The observation that focal seizures were proportionately commoner above five years, in the age group in which the child can more reliably describe an aura or a focal onset, is consistent with this interpretation and suggests that the true proportion of focal events in the younger strata was underestimated.

The aetiological findings merit the closest attention because they differ substantially from those reported in high-income settings. Febrile seizures accounted for 37.3 percent of admissions, a proportion in keeping with Indian series but achieved in a context very different from that assumed by the American Academy of Pediatrics guidelines, which discourage routine lumbar puncture, electroencephalography and neuroimaging following a simple febrile seizure on the grounds that the yield is negligible and the prognosis excellent.[6,7] That recommendation is sound where bacterial meningitis is rare, but its uncritical application in a setting where central nervous system infection accounted for 18.0 percent of all seizure admissions would be hazardous.

The present data support a lower threshold for cerebrospinal fluid examination than the American guidelines specify, particularly in infants, in children with complex features and in those who have received prior antibiotic therapy that may mask meningeal signs. The identification of neurocysticercosis in 4 children and tuberculoma in 2 further underlines the divergence, since these entities are essentially absent from Western paediatric practice yet are well documented as important causes of childhood seizures in India, where the reported prevalence and the therapeutic implications have been characterised in detail.[10]

The contribution of metabolic derangement, at 12.0 percent overall and 26.5 percent among infants, is of particular practical importance because these causes are rapidly identifiable and fully reversible. Hypoglycaemia, hypocalcaemia and dysnatraemia together accounted for all metabolic cases, and none of the affected children died. This finding provides a strong argument for the immediate bedside measurement of blood glucose and the early estimation of serum electrolytes and calcium in every infant presenting with seizures, before more elaborate investigation is contemplated. The gradient across age strata, with metabolic causes concentrated in infancy and idiopathic epilepsy concentrated above five years, is biologically coherent and has been reported consistently in the paediatric literature.

The outcome data reinforce the central message that aetiology, not semiology, governs prognosis. Complete recovery occurred in 84.0 percent overall but in 98.2 percent of children with febrile seizures, whereas 44.4 percent of children with central nervous system infection sustained an adverse outcome. This gradient mirrors that reported in comparable hospital-based series and provides empirical support for directing the initial diagnostic effort towards the identification of a treatable or dangerous cause rather than towards the refinement of seizure classification, which can reasonably be deferred to the outpatient setting.

Status epilepticus emerged as a powerful determinant of outcome, with a case fatality of 19.0 percent among affected children compared with 0.8 percent in the remainder. This figure lies between those reported in the two principal population-based studies. Chin and colleagues, in the North London Convulsive Status Epilepticus in Childhood Surveillance Study, reported a considerably lower case fatality in a high-income setting with rapid pre-hospital access to benzodiazepines.[8] Sadarangani and colleagues, in a Kenyan cohort, reported a substantially higher mortality attributable in large part to a preponderance of acute symptomatic status arising from central nervous system infection and to delayed presentation.[9] The intermediate position of the present cohort is consistent with an Indian tertiary setting in which access to definitive care is better than in sub-Saharan Africa but in which pre-hospital seizure termination remains uncommon. The median interval of 65 minutes from seizure onset to hospital arrival observed here is instructive in this regard, since it substantially exceeds the five-minute threshold at which treatment should be initiated under the current operational definition of status epilepticus.[5] Comparable Indian series of paediatric status epilepticus have reported similar delays and similar mortality, and have likewise identified acute symptomatic aetiology as the dominant predictor of an adverse outcome.[11,12,15] The single most actionable implication of these data is therefore not a hospital intervention at all but the wider availability of pre-hospital or caregiver-administered benzodiazepine for prolonged seizures.

The investigative yields observed here support a selective rather than a routine approach. Electroencephalography was abnormal in 52.7 percent of those in whom it was performed and neuroimaging in 38.7 percent, but these figures reflect the selection of children with focal features, abnormal neurology or refractory seizures rather than an unselected population. The markedly higher imaging yield in focal compared with generalised seizures, 55.2 percent against 25.0 percent, quantifies the benefit of that selection and argues against routine imaging of the child with an uncomplicated generalised febrile seizure. Cerebrospinal fluid examination, performed in fewer than a third of children, was abnormal in

43.8 percent of those tested, a yield that is high by international standards and that reflects both appropriate case selection and the genuine local burden of neuroinfection.

Limitations

Several limitations should be borne in mind. The study was conducted at a single tertiary care centre and describes children sick enough to warrant admission, so the findings cannot be generalised to seizures managed in the outpatient department or in the community, and are subject to the referral bias inherent in a tertiary institution. The classification of seizure semiology depended on the caregiver history in the many instances where the event was not witnessed by a clinician, and the proportion of generalised seizures is therefore likely to be overestimated relative to focal seizures. Investigations were performed on clinical indication rather than uniformly, which reflects sound practice and ethical necessity but means that the reported yields of electroencephalography, neuroimaging and cerebrospinal fluid examination are conditional on selection and do not represent the yield in an unselected population. Aetiological attribution was made on clinical and available laboratory grounds, and specialised investigations including metabolic screening, genetic testing and autoimmune antibody panels were not available, so a proportion of the children classified as idiopathic may harbour an identifiable genetic or metabolic cause. Follow-up extended only to discharge, so the recurrence of seizures, the eventual evolution to epilepsy and the longer-term neurodevelopmental consequences could not be assessed. Finally, the sample size, while adequate for the primary descriptive objective, produced small cell frequencies in several aetiological subgroups and the associated outcome estimates should therefore be interpreted with corresponding caution. Multicentre studies with longer follow-up and with access to genetic and metabolic testing would materially advance understanding of the residual idiopathic group.

Conclusion

Seizures in children aged one month to twelve years admitted to this tertiary care centre affected boys more often than girls and were concentrated in the one to five year age group. Generalised tonic-clonic semiology predominated, accounting for more than half of all events. Febrile seizures were the commonest aetiology at 37.3 percent, followed by central nervous system infection at 18.0 percent, idiopathic epilepsy at 16.0 percent and metabolic derangement at 12.0 percent, with the aetiological pattern differing significantly across age strata such that metabolic causes dominated in infancy and idiopathic epilepsy in older children. Complete recovery was achieved in 84.0 percent of children and overall mortality was 3.3 percent.

The principal conclusion is that outcome was determined by aetiology and by the occurrence of status epilepticus rather than by the type of seizure. Every death occurred in a child with central nervous system infection or structural pathology, and no child with a febrile or metabolic seizure died. The practical implications follow directly. Bedside blood glucose estimation and early measurement of serum electrolytes and calcium should be mandatory in every infant presenting with a seizure, since these causes are common in this age group and are entirely reversible. The threshold for cerebrospinal fluid examination should be lower than that recommended by guidelines written for settings in which bacterial meningitis and neuroinfection are uncommon. Neuroimaging and electroencephalography should be applied selectively, guided by focal features and abnormal neurological findings, where their yield is demonstrably higher. Above all, the substantial delay between seizure onset and arrival at hospital observed in this cohort, together with the disproportionate mortality associated with status epilepticus, indicates that the greatest available gain lies in shortening the interval to first anticonvulsant administration through caregiver education and wider availability of pre-hospital benzodiazepine.

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