

Our study had limitations of a small sample size. We also could not test for *SCC mecA* gene and PVL toxins. Rational antibiotic policy to prevent the rise in resistant staphylococci is the need of the hour.

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## Etiology and Short-term Outcome of First Seizure in Hospitalized Infants

We enrolled 75 consecutive infants presenting with history of first seizure at a tertiary-care hospital in New Delhi, India. Clinical and biochemical work-up for etiology, and electroencephalography were performed in all infants. Developmental assessment was done 3-month after discharge. 72% had generalized seizures, and fever was the commonest co-morbidity (57.3%). 68% had provoked seizures, mainly due to hypocalcemia (34.3%) or neuro-infections (29.3%). Seven (9.3%) infants died during hospital stay; mostly those with neuro-infections. 13 (20.3%) infants had developmental delay.

**Keywords:** *Child; Cause; Epilepsy; Prognosis.*

A seizure is one of the commonest childhood neurological illnesses and the risk is the highest in the first year of life [1]. Even after four decades of the initial studies on etiology and outcome of first seizure in infants [1], not much information is available on this aspect from India [2,3].

This descriptive study was conducted from April 2012 to March 2013 in the Pediatrics department of a public hospital to describe the clinico-etiological profile and short-term outcome of first seizure in infants.

After Institutional Ethical board's clearance, consecutive infants (aged 4-52 weeks) presenting with seizures (on three pre-specified days per week) were admitted in the department and evaluated for inclusion after initial management and stabilization. A written informed consent was obtained from parents. Inclusion criteria were: history of first episode of seizure, or history of more than one seizure (within last 7 days) but not evaluated. Infants who had received any medication (other than anti-convulsants) prior to coming to the hospital, and infants with no documentation of treatment received for the seizure, were excluded. Prospective enrolment continued till a pre-decided sample size of convenience of 75 infants was achieved.

Detailed neurological history including details of each episode of seizure was obtained from the parents, primary caregiver or any additional person who had observed the seizure. Based on the history, seizure semiology was

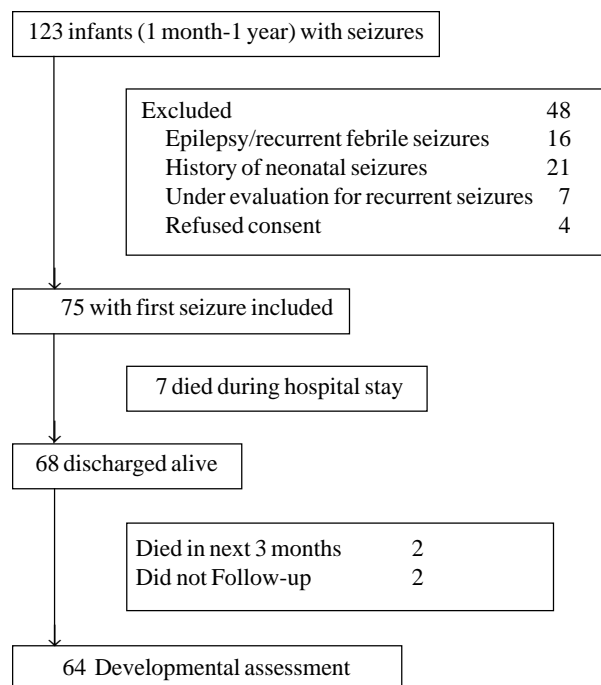
classified for those not having any seizure observed by a health worker. If the child had further seizures during the hospital stay, the attending doctor classified the seizure semiology, as per ILAE classification, 1981 [4]. All infants enrolled in the study were treated using standard treatment protocol of the unit. In brief, the protocol included essential investigations at time of admission for all infants: blood glucose, serum calcium and electrolytes, Serum urea/creatinine, and complete blood count. Additional investigations were also done as guided by history and physical examination and as per standard recommendations [5]. EEG was done at least two weeks after discharge with a 32 channel digital video EEG using International 10-20 system of electrode placement. Neuroimaging, and work-up for inborn errors of metabolism were done as required.

The presumed etiology of seizure was ascribed based on the history, clinical findings and investigation results, as Provoked seizures (occurring in close temporal relationship with an acute CNS insult, which may be metabolic, toxic, structural, infectious, or due to inflammation), Unprovoked seizures (occurring in the absence of a potentially responsible clinical condition), and Febrile seizures, using standard criteria [4,6,7].

All patients were kept in contact through monthly OPD visits or telephonically, and neurodevelopmental assessment was done by Developmental Assessment Scale for Indian Infants (DASII) three months ( $\pm 1$  week) after discharge, by a single examiner. Data were analyzed using SPSS 17.0 software. Comparisons between groups were done using Chi-square test for discrete variables and Student's t-test for continuous variables.

75 infants (61.3% males) with mean (SD) age of 5.8 (3.4) months were finally enrolled (**Fig. 1**). Seizure was the only complaint in 42.7%, and fever (57.3%) was the commonest co-morbidity. Solitary seizure was the presentation in 57 (76%) infants, and 12 (16%) had more than one seizure in previous 24 hours; seizure recurrence during hospital stay occurred in 7 (9.3%) infants. and (93.3%) had a short-lasting seizure (<15 min). Seizure semiology was determined based on eye-witness account in 77.3% and observation of seizures by a pediatrician in the rest. Majority (72%) had generalized seizure (tonic in most), though 7 (9.3%) had unclassified seizures. 68% of the infants had provoked seizures, mainly due to hypocalcemia and neuro-infections. All patients with hypocalcemia had nutritional rickets. Of the febrile seizures, a quarter presented with febrile status epilepticus (**Table I**). Thirteen (20.3%) infants had developmental delay, with majority having moderate delay.

Nine (12%) infants died during the course of the study,



**FIG.1** Flow of participants in the study.

7 of whom died during hospital stay. Maximum seizure deaths were noticed in the neuro-infection group (**Web Table I**). The median time from admission to death in those dying in hospital was 24 hours (range, 96 hours). The only infant in the unprovoked group dying in the hospital died 4 days after admission, with septic shock. Two infants, discharged in healthy state, died after discharge. One 2-

**TABLE I** DISEASE CHARACTERISTICS OF THE STUDY POPULATION (N=75)

Characteristic	No (%)
<i>Presumed etiology</i>	
Provoked	51 (68.0)
Metabolic derangement	27 (36)
Hypocalcemia	26 (34.3)
Neuroinfections	22 (29.3)
Pyomeningitis	16 (21.3)
Others	2 (2.7)
Unprovoked <sup>#</sup>	16 (21.3)
First febrile seizure	8 (10.7)
<i>Co-morbidities</i>	
Rickets	16 (21.3)
Developmental delay (n=64)	13 (20.3)
Death during hospital stay	7 (9.3)

<sup>#</sup>Benign infantile convulsions in 5.

month-old girl with acyanotic congenital heart disease, died one month after discharge during an episode of bronchopneumonia. An 11-month-old girl, diagnosed as benign infantile convulsion, was brought dead to the hospital with a history of high-grade fever of three days duration, two month after discharge.

Most studies on first non-febrile seizure in children have shown very few abnormal results on laboratory studies [5]. In two studies of both febrile and non-febrile seizures, results of laboratory studies did not contribute to diagnosis or management [8,9]. However, in another study of 65 children with new onset afebrile seizures, around 10% had either hyponatremia or hypocalcemia, mostly in those younger than six months [5,10]. Previous results from developing countries also suggest hypocalcemia to be a common cause of seizures in infants [3,7]. Our observation of developmental delay in 20% is similar to previous reports of 15-27% [11,12]. A relatively high death rate during follow-up observed in our study, has also been reported by few other studies [13,14].

Limitations of the current study include a convenience sample, absence of objective pre-morbid developmental status, lack of video-EEG confirmation of seizure semiology, and a short duration of follow-up, especially for seizure-recurrence and developmental delay.

The major finding of the present study was that hypocalcemia (due to rickets in majority) was responsible for more than a third of the infants with the first seizure. Guidelines for evaluation of first seizure in children from developed countries do not recommend evaluation for metabolic derangements in a child with first seizure [5]. Our results favor evaluation for hypocalcemia in all infants presenting with the first seizure. The presence of developmental delay in nearly a fifth of the infants suggests that this group of infants may be considered as a high-risk group for assessment and screening for developmental delay.

*Contributors:* DM: conceived the study, provided overall supervision, prepared the manuscript, and will be the guarantor; NKN: identified, enrolled and managed the patients, searched literature, analyzed the data, and helped in manuscript preparation; MJ, BT: Provided important intellectual inputs during the planning and conduct of the study and preparation of the manuscript; BT: reported on the EEG findings. All authors approved the final manuscript for publication.

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**WE**TABLE I SHORT-TERM MORTALITY IN THE STUDY POPULATION

<i>Presumed etiology</i>	<i>No. (%)</i>	<i>During hospital stay</i>	<i>After discharge</i>	<i>Probable cause of death</i>	
				<i>Seizure-related</i>	<i>Unrelated to seizure</i>
<i>Provoked (n=51)</i>	7 (9.3)	6	1	2	5
Neuroinfections ( <i>n=21</i> )	6 (8.0)	5	1	2	4
Metabolic ( <i>n=28</i> )	0	0	0	0	0
Others ( <i>n=2</i> )	1 (1.3)	1	0	0	1
<i>Unprovoked (n=16)</i>	2 (2.7)	1	1	0	2
<i>Febrile seizures (n=8)</i>	0	0	0	0	0
Total	9 (12.0)	7	2	2	7