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Original article:

Study of etiopathogenesis of first seizer in hospitalized infants in tertiary care Hospital

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Abstract:

Introduction: Neonatal seizures or neonatal convulsions are epileptic fits occurring from birth to the end of the neonatal period.The neonatal period is the most vulnerable of all periods of life for developing seizures, particularly in the first 1–2 days to the first week from birth

Methodology: The present observational study was carried out in our Department for last six months. This was observational study. We included 50 consecutive infants presenting with history of first seizure at a tertiary care hospital . Clinical and biochemical work-up for etiology, and electroencephalography were performed in all infants. Developmental assessment was done 3-month after discharge. The sample size was estimated with help of expert. We included the infants who had first seizer in hospitalized cases. History of prior cases were excluded from present study.

Results: In our present study there were 32 (64 %) were male babies while 18 (36%) were female babies. In our present study, most common etiological factor was found neuroinfection cases (22%) and hypocalcaemia (28%)

Conclusion: 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.

Introduction:

Neonatal seizures or neonatal convulsions are epileptic fits occurring from birth to the end of the neonatal period. [1]The neonatal period is the most vulnerable of all periods of life for developing seizures, particularly in the first 1–2 days to the first week from birth. They may be short-lived events lasting for a few days only. However, they often signify serious malfunction of or damage to the immature brain and constitute a neurological emergency demanding urgent diagnosis and management.[2] Current therapeutic strategies for epilepsy include anti-epileptic drugs and surgical treatments that are mainly focused on the suppression of existing seizures rather than the occurrence of the first spontaneous seizure. These symptomatic treatments help a certain proportion of patients, but these strategies are not intended to clarify the cellular and molecular mechanisms underlying the primary process of epilepsy development, i.e., epileptogenesis. Epileptogenic changes include reorganization of neural and glial circuits, resulting in the formation of an epileptogenic focus.[3]

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Methodology:

The present study was carried out in our Department for last six months. This was observational study.

We included 50 consecutive infants presenting with history of first seizure at a tertiary care hospital . Clinical and biochemical work-up for etiology, and electroencephalography were performed in all infants. Developmental assessment was done 3-month after discharge.

The sample size was estimated with help of expert.

We included the infants who had first seizer in hospitalized cases. History of prior cases were excluded from present study.

Data was tabulated in MS Excel sheet and analyzed.

Results:

Table 1) Age group distribution in present study

Age of infants	Number of infants	Percentage
Less than one month	26	52
One month – six months	18	36
More than six months	6	12

In our present study there were 32 (64 %) were male babies while 18 (36%) were female babies.

Table 2) Type of seizers observed in infants

S.NO.	Type of seizers	Number of infants	Percentage	Associate Co morbidity
1	Generalized seizers	39	78%	Fever
2	Provoked seizers	31	62%	Neuro infection
3	Clonic seizers	6	12%	Fever
4	Myoclonic seizers	3	6%	--

78% had generalized seizures, and fever was the commonest co-morbidity (61 %). 62% had provoked seizures, mainly due to hypocalcemia (28%) or neuro-infections (22%).

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Table 2) Etiological factors assessment

S.NO.	Etiological factors	Number of infants	Percentage
1	Neuroinfection	11	22
2	Hypocalcimia	14	28
3	Meningitis cases	3	6
4	Other causes	22	44

In our present study, most common etiological factor was found neuroinfection cases (22%) and hypocalcaemia (28%)

Discussion:

We included 50 consecutive infants presenting with history of first seizure at a tertiary care hospital . Clinical and biochemical work-up for etiology, and electroencephalography were performed in all infants. Developmental assessment was done 3-month after discharge. In our present study , maximum infants were of less than one month. In our present study there were 32 (64 %) were male babies while 18 (36%) were female babies. In our present study, most common etiological factor was found neuroinfection cases (22%) and hypocalcaemia (28%)

To achieve the goal of developing "anti-epileptogenic" drugs, we need to clarify the step-by-step mechanisms underlying epileptogenesis for patients whose seizures are not controllable with existing "anti-epileptic" drugs. Epileptogenesis has been studied using animal models of neonatal seizures because such models are useful for studying the latent period before the occurrence of spontaneous seizures and the lowering of the seizure threshold. Further, neonatal seizure models are generally easy to handle and can be applied for *in vitro* studies because cells in the neonatal brain are suitable for culture. Here, we review two animal models of neonatal seizures for studying epileptogenesis and discuss their features, specifically focusing on hypoxia-ischemia (HI)-induced seizures and febrile seizures (FSs). Studying these models will contribute to identifying the potential therapeutic targets and biomarkers of epileptogenesis.[4,5]

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 [6]. 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]. 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% [7]. A relatively high death rate during follow-up observed in our study, has also been reported by few other studies [1,3,6].

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.

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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.

Conclusion:

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.

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