

Can we predict the Occurrence of Hypsarrythmia in Term Newborns with HIE: A Prospective Observational Study.

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INTRODUCTION:

Hypoxic Ischemic Encephalopathy (HIE) is one of the commonest causes of epilepsy and Infantile Spasms Epilepsy Syndrome (ISES). Very few studies have described the dynamicity in which the EEG evolve across age at specific time points in infants with moderate to severe HIE and the likely EEG patterns (prehypsarhythmic) that might evolve into hypsarrythmia with clinical spasms. Thus this prospective study was performed to identify patterns that might predict development of hypsarrythmia in high risk infants.

OBJECTIVES:

1.Primary objective:

- To describe the evolution of EEG patterns in term newborns with moderate to severe HIE.

2. Secondary objectives:

- To study the occurrence of hypsarrythmia in infants with HIE.
- To describe the EEG abnormalities which might predict the occurrence of hypsarrythmia and its variants.

METHODOLOGY

All term newborns born from September 2022 to December 2023 fulfilling the inclusion criteria were enrolled in the study after obtaining written informed consent from the parents. EEG was performed at particular time periods - Within first 2 weeks(At discharge), 1 month, 3 months, 6 months of age along with developmental assessment done till 9 months.

EEG was performed as per standard 10-20 system and reported by 2 experienced pediatric neurologists individually, if any difference of opinion present, was reviewed together and if disagreement still persisted was reviewed by a third pediatric neurologist and considered final. Both background and abnormal patterns were reported

RESULTS

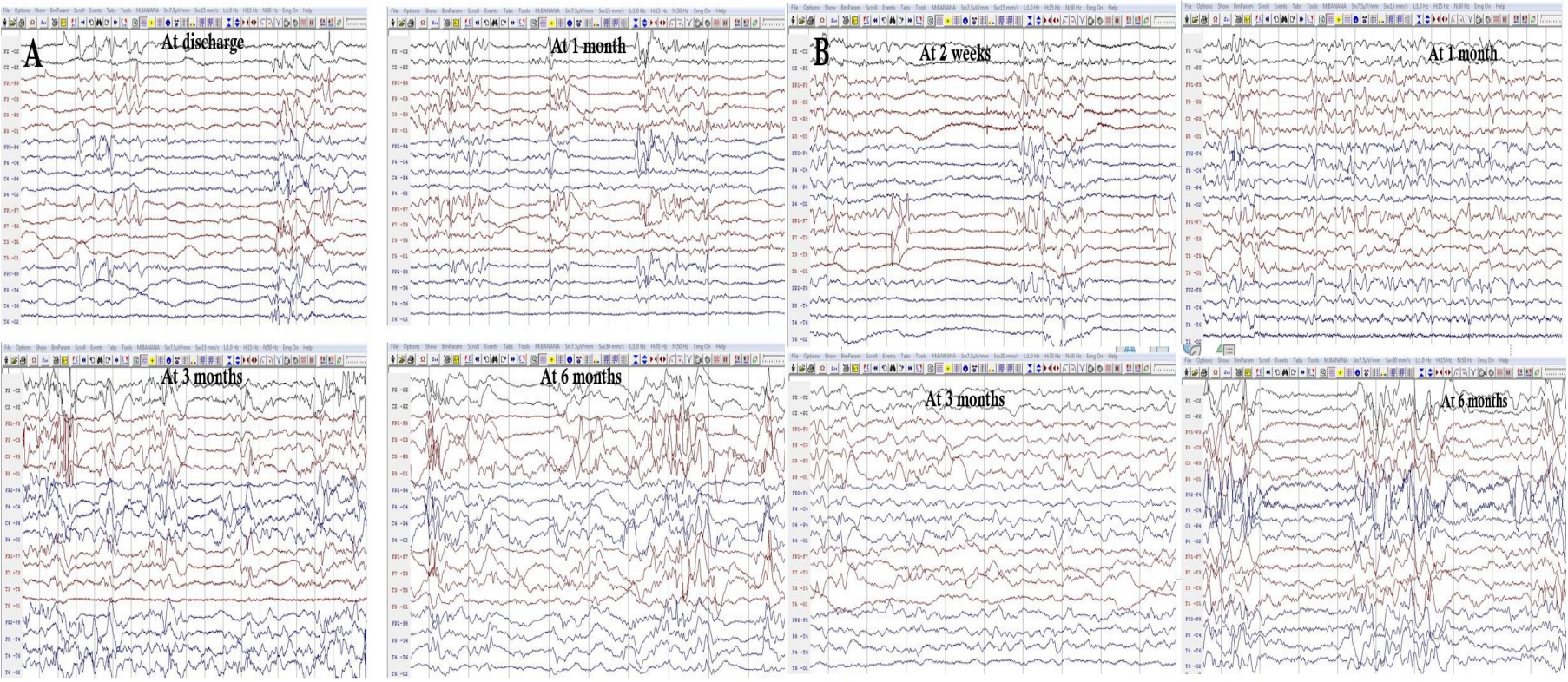
A total of 37 neonates were enrolled 25(67%) were males, 26(70%) NVD with birth weight of 2960± 420gms.

Abnormal EEG was seen in 54%, 43%, 40% and 35% at discharge, 1m , 3m and 6 months respectively.

RESULTS (Contd..)

2 neonates had a severe discontinuous background beyond 14 days of life and the discontinuity persisted until 1m, 3m and 6m and both eventually went on to develop prehypsarhythmic pattern by 3-5 months and clinical spasms by 6 months of age.

Sl. No	EEG Patterns	At Discharge, n(%)	At 1 month	At 3 months	At 6 months, n(%)
1.	Background Abnormalities:	17(46)	21	23	25(67)
1.	Normal	13(35)	05	02	03(08)
2.	Continuous Slow Background with sleep markers. (Mild asymmetry/voltage depression)	05(14)	08	10	07(19)
3.	Continuous slow background with absence of Sleep Markers (Discontinuous with IBI <10sec / asynchrony/asymmetry)	02(5)	02	02	02(05)
4.	Discontinuous Background (IBI 10-60secs/severe depression/no sleep wake cycles)				
2.	Epileptiform Abnormalities:	32(86)	35	32	30(81)
1.	Absent	03(08)	01	01	03(08)
2.	Focal Epileptiform Discharges	02(05)	01	03	02(05)
3.	Multifocal Epileptiform Discharges	00	00	01	02(05)
4.	Hypsarrythmia				



Evolution of EEG patterns in the 2 neonates (A and B) with severe discontinuous pattern abnormalities till 6 months of age and Hypsarrythmia at 6 months.

CONCLUSIONS

Severe discontinuous EEG abnormalities during neonatal period beyond 2 weeks can be a marker and a persistent discontinuous record by 1 – 3 months even in the absence of clinical spasm can be considered as a prehypsarhythmic pattern necessitating close follow up in such high risk infants during high risk follow up and performing EEG by 3-5 months can be warranted.

REFERENCES

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