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WAVEFORM WINDOW #38

EEG Stages of Neonatal Hypoxic Ischemic Encephalopathy: From Background Suppression to Resolution of Neonatal Seizures

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EEG testing is frequently ordered when there is a clinical suspicion of seizures or when a patient is placed on a hypothermia protocol following anoxic brain injury. Hypoxic ischemic encephalopathy (HIE) is a common indication for neonatal EEG recording in the neonatal intensive care unit, as seizures have been reported in as many as 26% of neonates with HIE (Shellhaas et al. 2011; Tsuchida et al. 2013; Massaro et al. 2015). Overall seizure burden has been shown to be significantly reduced in neonates receiving therapeutic hypothermia for HIE (Boylan et al. 2015); therefore, EEG is very important during the cooling protocol and is more critical in neonates as they are often sedated or on paralytic medication, which may increase the risk for undetected subclinical seizure activity. At the same time, there is a particularly high risk of overdiagnosis in critically ill neonates, with some benign movements mislabeled as seizures (Orivoli et al. 2015). For these reasons, accurate diagnosis generally requires the use of continuous EEG monitoring (Boylan et al. 2015).

Neonatal seizures related to HIE have a mean onset around 13–24 hours postpartum, with a maximum seizure burden at around 19 hours postpartum (Lynch et al. 2015). Therefore, it is of utmost importance that continuous EEG monitoring be started as soon as possible. However, in the very early stages of HIE, neonatal seizures are often missed for various reasons, including the lack of uniform availability of EEG technologists to perform the recording. This report describes a neonate with HIE in which we recorded all of the stages of seizure development during the initial week of neonatal HIE.

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This term patient was born to a mother of two who had no pregnancy complications during an unplanned delivery at home, where no Apgar scores were recorded. At birth, the umbilical cord was wrapped around the infant's neck and the mother reported that the baby's breathing was shallow. Within the first hour of life, a transport team was called and the infant was transferred to the hospital. Upon arrival, the infant was unresponsive with bilateral pupils fixed and dilated. Initial venous gas was of pH 6.68 and PaCO₂ 113 mm Hg compared to the normal neonatal venous gas scores of pH 7.35–7.45 and PaCO₂ 35–45 mm Hg with reactive pupils. Shortly after, repeated venous gas improved to a pH of 7.01, which clinically coincided with sluggish pupillary constriction.

The infant was placed on a hypothermia protocol from about 5 hours postpartum and EEG monitoring was started at 9 hours (Table 1). The first few hours of EEG showed profound background suppression, which is noted in the first stage of HIE (Figure 1). The EEG was completely suppressed, under 5 μ V of amplitude. There were no faster frequencies and the EEG was unreactive to any stimulation to the infant. This stage lasted for the first 3 hours of EEG recording (between 9 and 12 hours postpartum). In comparison, a normal EEG at this age should have state changes between awake, quiet sleep, and active sleep, and should be symmetric and synchronous in each state. A symmetric continuous mixed frequency of delta-alpha with amplitude between 25 μ V and 50 μ V is seen in normal neonatal EEG during awake and active sleep, with interburst intervals less than 4 seconds and 25 μ V or higher in amplitude in quiet sleep (Shellhass et al. 2008).

At about 12 hours postpartum, post-anoxic myoclonus was noted in upper and lower extremity, but the EEG showed only diffuse muscle artifacts without any clear cortical activity (Figure 2). As recording progressed, the EEG began to show bursts of brain activity with multiregional epileptic interictal discharges from 12 hours postpartum

Hours postpartum	Clinical	EEG
1	Unresponsive, bilateral pupils fixed, dilated	
5	Hypothermia protocol began	
9-12	EEG recording began	Background suppression, $< 5 \mu$ V, unreactive.
12–15	Myoclonus, upper & lower extremities	Muscle artifacts, with no clear changes in cortical activity.
12	No clinical signs	Bursts of brain activity with multiregional epileptic interictal discharges.
14	No clinical signs	Multifocal subclinical EEG seizures with slow $(1-2 \text{ Hz})$, rhythmic sharply contoured, and phase reversing delta noted in the left and right hemisphere.
15–16	Single limbs, clonic jerking of the upper and lower extremities or the hemibody	Electroclinical seizures with clonic jerking of the upper and lower extremities, contralateral to the seizure focus.

Table 1. Timeline of electroclinical findings.

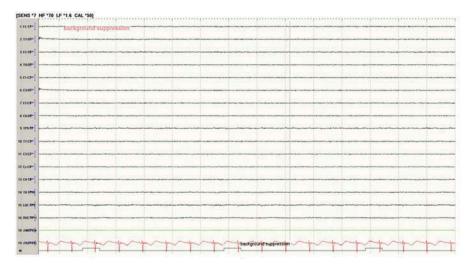


FIG. 1. Profound background suppression at 9 hours postpartum.

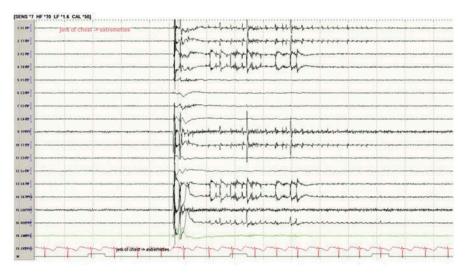


FIG. 2. Myoclonic jerks with no ictal EEG changes at 12 hours postpartum.

(Figure 3), which evolved into multiregional seizures. Multifocal subclinical EEG seizures with slow (1-2 Hz), rhythmic, sharply contoured, and phase-reversing delta were noted, shifting from the left or right hemisphere independently (Figures 4 and 5).

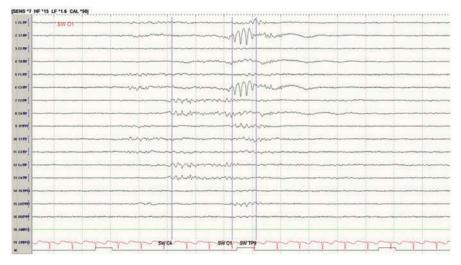


FIG. 3. Interictal discharges in O1, C4, and centrotemporal theta at 12 hours postpartum.



FIG. 4. EEG seizure in the left temporal region (T7) seen at 1–1.5 Hz frequency at 14 hours postpartum. No clinical signs were noted during this seizure.

Later, at about 15 hours postpartum, electroclinical seizures with clonic jerking of the upper and lower extremities in each side of the body began. These single limb or hemiclonic clonic seizures were contralateral to the seizure focus (Figure 6).



FIG. 5. EEG seizure in the right central region (C4) seen with 1 Hz sharp wave frequency at 14 hours postpartum. No clinical signs were noted during this seizure.

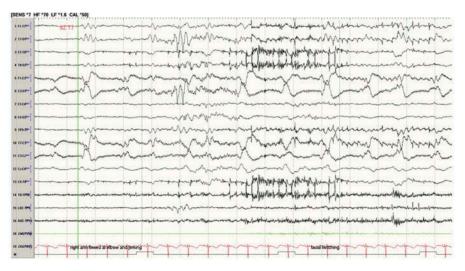


FIG. 6. At about 15 hours postpartum, clinical seizure began with contralateral EEG seizure focus. EEG seizure in the left central region (C3) seen with a run of sharp waves at 0.5–1.0 Hz frequency with right arm clonic and facial twitching.



FIG. 7. Posterior sharp waves at 5 days postpartum.

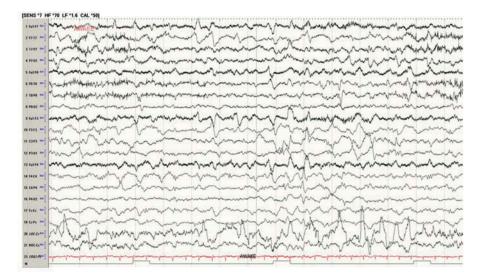


FIG. 8. Twenty-eight days postpartum. The EEG shows frequent multiregional sharp wave discharges with generalized diffuse baseline slowing.

With medical treatment (phenobarbital, levetiracetam, fosphenytoin, and midazolam), seizures were controlled on day 3 postpartum, but frequent multiregional interictal epileptiform discharges persisted (Figures 7 and 8).

On follow-up at 6 months of age, the infant had overt, developmental delay. He was alert and active with no apparent distress, cooperative, smiling, and consolable. He was beginning to roll and grab for objects. He feeds via a feeding tube. He remains seizure-free.

In conclusion, our case documents that the early neonatal HIE stages have similarities with the postanoxic stages described in older children and adults, in which there is a predominance of background suppression and postanoxic myoclonus before the appearance of the seizures.

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