

aEEG monitoring on infant with HIE

Patient characteristics

Baby Boy W is the product of a 39 week EGA born to a 19YO G2 P1 mother by repeat C-section. The baby was born limp with minimal movements. He required vigorous resuscitation which included bag mask ventilation, CPR, a normal saline bolus, O negative blood and 4 doses of epinephrine. Despite successful intubation and CO₂ obtained on the capnograph, the patient was re-intubated due to poor saturations. His APGAR scores were 1, 1, at 1 and 5 minutes respectively.

Initial exam and clinical impression

The chest x-ray revealed an undiagnosed left congenital diaphragmatic hernia. The initial arterial blood gas was 6.81/81/66/-21. The baby's initial neurologic exam was consistent with a Sarnat II. The baby was passively cooled after discussion with the tertiary care center. An OG was placed to decompress the bowel, sedation was given and a dose of sodium bicarbonate was administered. Upon arrival to the referring facility, the neonatal transport team began active hypothermia.

Upon arrival to the tertiary care facility, the baby was continued on cooling and the initial exam was consistent with a Sarnat 2. The aEEG was placed on the baby and the initial pattern was consistent with a burst suppression but evolved to a low voltage pattern (Figure 1).¹ Active hypothermia was continued and the baby went on VA ECMO on DOL 2 for worsening respiratory failure. Active hypothermia was continued during VA ECMO. The baby received phenobarbital on DOL 2 due to concerns of seizure activity on the aEEG (Figure 2).¹ At that time the background pattern was noted to evolve to a discontinuous activity pattern. The baby was rewarmed after 72 hours of hypothermia and underwent ECMO for 15 days. His background pattern continued to evolve after rewarming on DOL 5 (Figure 3).¹ He had his left congenital diaphragmatic hernia repaired on DOL 16.

Outcome

An MRI obtained on DOL 30 of life revealed punctate areas of increased T1 signal in the right periventricular white matter/trigonal white matter which could represent hypoxic-ischemic injury. The remainder of the brain demonstrated normal formation and myelination without evidence of hypoxic-ischemic injury. The MRS did not demonstrate injury of the basal ganglia.

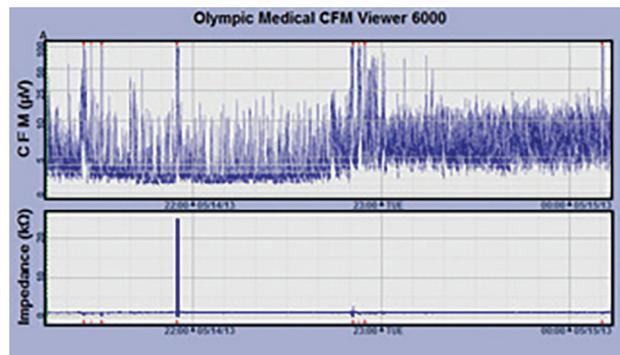


Fig. 1: The initial aEEG trace is consistent with a burst suppression pattern (minimum without fluctuations and bursts of higher voltage). The pattern mid-trace evolves to a low voltage pattern with lower margin less than 5micV and the upper below 10 micV.

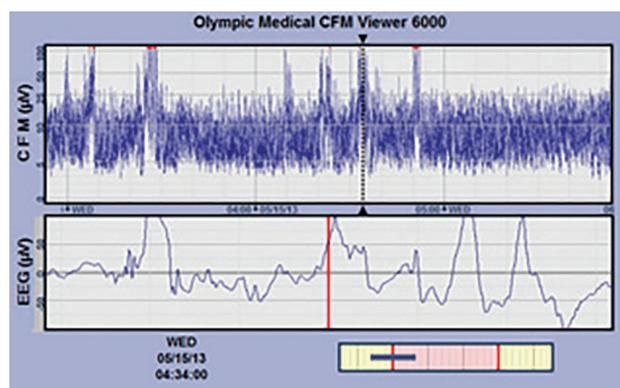


Fig. 2: An abrupt increase was noted in the maximum amplitude. Several of these increases were seen over a span of 2 hours. The raw EEG is shown in the lower panel during one such increase. Also note the evolution of the background pattern to a discontinuous activity background (minimum amplitude below 5 micV and the maximum above 10 micV).

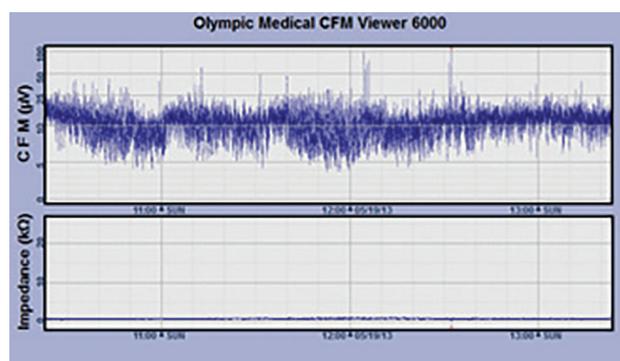


Fig. 3: At DOL 6, the aEEG demonstrates a continuous activity pattern (Minimum amplitude above 5 micV and the maximum above 10-50 micV) with sleep wake cycling (smooth sinusoidal variations with broader and narrower bandwidth).

Discussion

aEEG at less than 6 hours is the best single outcome predictor in neonates with HIE at normothermia.² However, the predictive value is lessened in the hypothermic patient and there may be a delay in normalized background patterns in these patients which highlights the need for continuous monitoring during hypothermia.² The presented case illustrates this evolution of the background trace with a delay in the occurrence of sleep wake cycling and a normal background pattern.

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References

- 1 Hellstrom-Westas, L. and I. Rosen. Continuous brain-function monitoring: state of the art in clinical practice. *Semin Fetal Neonatal Med*, 2006. 11(6): p. 503-11.
- 2 Thoresen, M., et al. Effect of hypothermia on amplitude-integrated electroencephalogram in infants with asphyxia. *Pediatrics*. 126(1): p. e131-9.

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