CASE STUDY

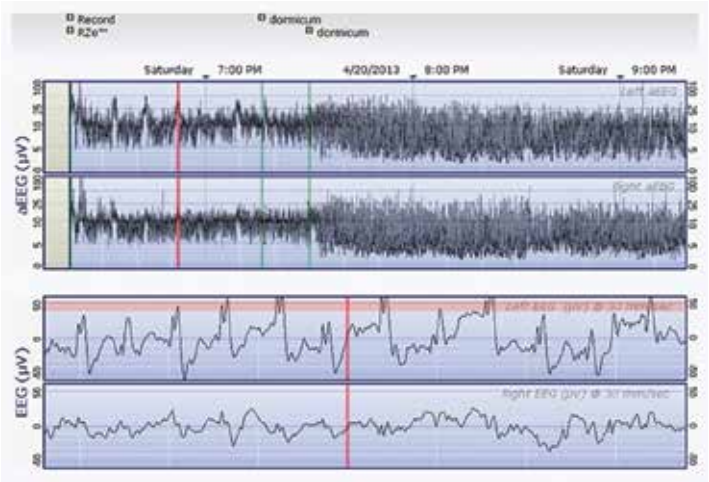
aEEG monitoring on infant with perinatal stroke

Patient characteristics

A female infant born at 38+4 weeks gestation with a birth weight of 3170 grams. The mother was G1P0. She was admitted for induction at 38+4 weeks gestation because of pregnancy induced hypertension. On rupture of the membranes there was blood loss and on CTG a fetal tachycardia was seen. An emergency caesarean section was performed and during the resuscitation the infant had profound bradycardia with no spontaneous breathing effort. After 5 inflation breaths the heart rate increased and after 10 minutes she was breathing spontaneously but with an irregular breathing pattern. Apgar scores were 2, 5 and 7 at 1, 5 and 10 minutes respectively. She was admitted to the neonatal unit in an outlying hospital where she stabilized quickly. Her first glucose level was 3.6 mmol/L and she had a capillary sample lactate of 11.2 mmol/L. Around 12 hours after birth she had an incident of bradycardia, followed by a drop of oxygen saturation and thereafter clinical seizure like activity involving both arms and legs. A loading dose of phenobarbitone (20 mg/kg) was given. Ten hours later she developed clinical seizures again with movements of her right arm and a drop in oxygen saturation. Another dose of phenobarbitone was given and she was transported to our NICU for further cerebral monitoring and investigation.

Initial exam and clinical impression

Olympic Brainz Monitor



On admission she was clinically stable. The initial physical exam was normal and biochemical examination showed no abnormalities. Her aEEG was recorded immediately on admission at 24 hours after birth and showed repetitive seizure discharges on a normal background pattern, without clinical seizures. More seizures were seen on the left side. A loading dose of midazolam (0.05 mg/kg) was given followed by continuous infusion of 0.05 mg/kg/hour. Thereafter no clinical and subclinical seizures were seen. The background pattern was initially discontinuous, probably due to the midazolam, but returned to normal voltage within one day. Her cranial ultrasound (cUS) showed increased subcortical echogenicity on the left side. An MRI performed on day 2 after admission showed a MCA infarct on the left side. Involvement of the PLIC was seen and the peduncle seemed not to be involved. Normal flow was seen in the MCA. After permission of the parents EPO was started (for 3 days), because of the possible neurogenerative effect.[1] Because of the stroke, additional thrombophilia screening was performed, showing no homo- or heterozygosity for MTHFR and no mutation of the FVL or the prothrombin gene.

Outcome

At the age of 3 months the MRI was repeated. A minimal asymmetry was seen in the PLIC, but the peduncles were symmetric. At the age of 9 months she was seen in the follow-up clinic and she had a normal development with no asymmetry in movements.

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Discussion

One of the causes of perinatal arterial ischemic stroke is hypoxia, as seen in this case. This stresses the importance of good clinical observation after resuscitation. The infant in this case showed in the first instance general seizures, but later on hemi convulsions. This was helpful in making the diagnosis. Also on the aEEG more seizures were seen on the affected side. In the first hours after the stroke the ultrasound can be normal, but in a few days the abnormalities will be apparent. If there is a possibility to perform a MRI shortly after the event the DWI will be helpful in making the diagnosis. EPO was given because of the possible neurogenerative effect and it appears safe in neonates.

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References

1 Benders MJ, van der Aa NE, Roks M et al. Feasibility and safety of erythropoietin for neuroprotection after perinatal arterial ischemic stroke. *J Pediatr.* 2014 Mar;164(3):481-6.e1-2.