

# Monitoring aEEG in a newborn with hyperbilirubinemia

## Patient characteristics

Male infant, 39 weeks gestation, born to a teenage mother 16 years old, normal vaginal delivery and graduated to 24 hours of life with no complications.

He was exclusively breast fed and his mother discussed having little milk production during the first 3 days. On the fifth day of life the infant was found to have a weak suck action and difficulty breathing. He was admitted to another hospital unit where they found the total bilirubin level to be 20mg / dL and was transferred to our hospital for further diagnosis and treatment accordingly.

## Initial examination and clinical impression

On admission the infant had good respiratory effort, but was hypotonic with a weak suck action. The blood analysis showed a total bilirubin level of 29.5mg / dL, blood group A +ve and Maternal blood group O +ve, a +ve Direct Coombs test and reticulocyte count + 3%.

On neurological examination he was found to be lethargic, with an absent suck and swallowing reflex, adduction of both thumbs, lower extremities with increased tone, these findings were suggestive of encephalopathy due to Hyperbilirubinemia.

Monitoring with aEEG was commenced detailing a pattern of Burst Suppression (Figure 1).

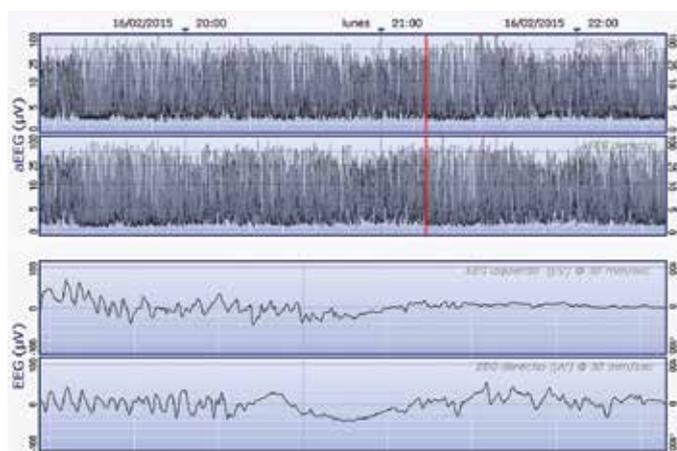


Figure 1. aEEG with Burst Suppression pattern (BS +) associated with elevated bilirubin. No sleep wake cycle was observed, no seizures were found.



Figure 2. Newborn infant requiring endotracheal intubation due to repetitive apnea and respiratory failure.

Management began with intensive phototherapy, an exchange transfusion and a dose of 1g / Kg intravenous immunoglobulin. Management with intensive phototherapy was continued.

On day 7, the infant developed recurrent apneas requiring mechanical ventilation (Figure 2). Laboratory results at this time showed a total bilirubin level 24.4mg / dL.

Further aEEG monitoring at this stage now showed a demise in the background pattern with Burst Suppression and repetitive seizures (Figure 3).

A neurological examination, showed only responses to noxious stimuli, with dyskinetic decorticate positions. Oral automatism were observed, these moves temporarily coincided with the electrical pattern of seizures.

A further exchange transfusion was performed and intravenous immunoglobulin was administered again. Initial management of seizures was with a phenytoin loading dose of 20 mg / kg / dose. Despite the seizure management with phenytoin the seizures showed tonic characteristics, hence Levetiracetam was added at a dose of 50mg / kg / day.

Seizures subsided and he was continued on maintenance Levetiracetam monotherapy. After 2 weeks of mechanical ventilation, the infants respiratory pattern improved, allowing extubation.

## Outcomes

After 60 days of hospitalization, the child continued to have several neurological disturbances (axial hypotonia with Rhizomelic spasticity), and in addition presented with major alterations in sucking and swallowing, resulting in a Gastrostomy and fundoplication to achieve enteral nutrition. The hearing screening performed with otoacoustic emissions showed no abnormalities. He continued with Levetiracetam management and was discharged with a diagnosis of Encephalopathy due to hyperbilirubinaemia.

## Discussion

Screening and effective treatment for newborns with jaundice are widely available in most areas today, hence most infants with severe hyperbilirubinemia recover and survive the Acute Bilirubin Encephalopathy phase. The aftermath of this pathology (Chronic Bilirubin Encephalopathy) have a broad clinical spectrum including dyskinetic cerebral palsy, abnormal movements and tone deafness. The incidence of chronic bilirubin encephalopathy is estimated at 1:50,000 to 1:100,000 live births. Recently it has been reported that the incidence is higher in countries like the UK and Canada<sup>1</sup>. In Mexico where this case is reported from the incidence of this disease is unknown, but currently in the facility reporting the case, which has about 6,000 births a year, there are approximately 5 documented cases of acute encephalopathy per year.

The role of aEEG as a prognostic factor in Neonatal Encephalopathy is well established, but in the case of bilirubin encephalopathy few studies detailing this exist. In 2013 Luo et al published a series of 10 cases which found that 8 (80%) of these children had electrical seizures, they also found that 85% of children with abnormal pattern background had a bad neurological outcome<sup>2</sup>.

In this documented case, the most important prognostic factor is the persistence of an abnormal pattern aEEG (Burst suppression) after 7 days of commencing hyperbilirubinemia management. This abnormal pattern background, combined with clinical seizures occurring with electrical correlation are helpful in making a prognostic indicator. The role of aEEG in Acute Bilirubin Encephalopathy demonstrates that therapeutic interventions, focused on normalizing bilirubin levels quickly, are effective in preventing neurotoxicity from this metabolite.

## Bibliography

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2. Luo F, Lin HJ, Bao Y, Chen Z, Ma XL, Shi LP, Du LZ. Amplitude-integrated electroencephalographic changes in neonates with acute bilirubin encephalopathy. Zhonghua Er Ke Za Zhi 2013 Mar;51(3):221-6.

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Figure 3. Background Pattern with repetitive BS + with rhythmic sharp waves.

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