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Case Report
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Neonatal Seizures – Interesting Case Series

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Abstract

We present three neonatal cases that presented with abnormal tone at birth and developed early seizures due to hyponatraemia secondary to low maternal sodium levels

Case Report 1

A male infant was born at 37+6 weeks by emergency c-section after mum presented in labour following an episode of gastroenteritis. She developed seizures (thought to be due to low sodium) and was rushed to the theatres. Baby was born in poor condition with a slow heart rate and no respiratory effort. After the initial ventilation breaths, he was stabilized and transferred to the neonatal unit on CPAP. Cord gases showed pH art 7.06 / ven 7.14, BE – 9.5, and lactate 6.4. Baby was noted to be hyponatraemic on the initial blood gas (Na 115) and had clinical and electrical seizures at 1 and 4 hours of age. The background brain wave activity on CFM was noted to be suppressed. Baby was treated with two doses of phenobarbitone for each of the seizure episodes, and hyponatraemia was corrected with hypertonic (3%) saline boluses, followed by an infusion for over 48 hours, as per the local hyponatraemia treatment protocol. Once sodium was corrected, no further seizures were reported, and the baby's CFM monitoring showed a rapid improvement in the background activity to normal. Baby received 48 hours of 1st line antibiotics and infection markers were negative. His brain MRI and pre-discharge examination including neurology was normal.

Case Report 2

A male infant was born at 40+3 weeks by ventouse assisted vaginal delivery following failure to progress and pathological CTG. The delivery was complicated by shoulder dystocia. Baby was born in poor condition and needed ventilation due to poor respiratory effort. Cord gases revealed pH art 7.12 / ven 7.22, BE -9, Lactate 6.6 with sodium 118. Due to the need for resuscitation and abnormal tone, baby was cooled for 72 hours. CFM showed burst suppression pattern. He had two episodes of seizure in the first 12 hours of admission and received phenobarbitone for the same. His admission sodium was 114, which gradually improved with sodium supplementation with intravenous fluids.

The baby's mother had a tonic-clonic seizure following the delivery that was thought to due to hyponatraemia. She had been drinking large amounts of water at home and had consumed approximately 6 litres over the preceding 14 hours, some of which she vomited out. In addition, she had received IV fluids on admission.

Case Report 3

A male infant was born at 40+4 weeks gestation by emergency c-section because of a pathological CTG and failure to progress. He was born with poor respiratory effort and required intubation at birth. Cord gases were acceptable (pH art 7.26 / ven 7.34, BE -6, lactate 5.5), but in view of hypotonia and on-going need for resuscitation at 10 minutes, he was passively cooled pending further assessment. On arrival to NICU he was noted to have abnormal tone with jerking movements of all four limbs that correlated with electrical seizures on CFM. He was given a dose of phenobarbitone with good response. Therapeutic hypothermia was commenced. His admission blood gas showed a metabolic acidosis and sodium of 110. He was started on a sodium bicarbonate infusion for the acidosis. Baby's repeat blood gases also showed hyponatremia with a trough of 112. As this correlated with maternal hyponatremia and he had seizures within the first hour of life, it was felt that this could be the cause of his presentation. He was therefore treated with hypertonic (3%) saline and his sodium rose above 125 after 17 hours. It had normalised by 48 hours. Baby was cooled for 72 hours. Baby received 48 hours of 1st line antibiotics and infection markers were negative. An MRI on day five of life and his pre-discharge examination including neurology was normal.

Baby's mother was noted to have had a change in behaviour over the preceding 12 hours, becoming more withdrawn and

less verbal. This was initially put down to a change in mental health on a background of having depression, anxiety and panic attacks. On post-operative review in recovery, however, she had a reduced GCS of 10 and was posturing with her sodium on the blood gas being 113. She was admitted to adult ITU where it was ascertained that she most likely had a chronic mild hyponatremia from fluoxetine, which had been exacerbated by excessive oral fluid intake during a heat wave in the few days preceding delivery, 5 litres of IV fluids during delivery, and the vasopressin action of her oxytocin infusion.

Discussion

Hyponatremia is a well-known cause of neonatal seizures accounting for about 12% of cases [1-9]. According to a study, the risk factors for mortality among the babies with seizures were clinical seizures in the first 24 hours of life, birth asphyxia co-existing with hyponatremia and presence of cerebral oedema [2]. There have been case reports of new born babies who suffered severe hyponatremia and early seizures, associated with maternal fluid overload due to high oral intake [4] or with electrolyte free solutions and high doses of oxytocin for labour augmentation [3,7] or excessive fluid administration in the new-borns [6]. Treatment of hyponatremic seizures with routine anticonvulsants may be ineffective. Administering 0.9% saline solution intravenously [6] or hypertonic (3%) saline [8] until the plasma sodium level reaches normal ranges is safe and efficacious in managing acute symptomatic hyponatremia [8]. The increased body water in pregnant women and the birth-related activation of water-sparing systems contribute to a high risk of perinatal water intoxication if the mother drinks too much water during labour. Awareness of this diagnosis in the delivery unit is very important, because the clinical picture may mimic that of pre-eclampsia or dehydration [9]. These three case reports illustrate the importance of educating women in labour on appropriate volumes and the type of fluid intake (avoiding hypotonic drinks) leading up to delivery to avoid such complications.

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