Neonatal hypoglycemic brain insult: MR findings

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Section: Paediatric radiology
Case Type: Clinical Cases
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Patient: 6 months, male

Clinical History:

A 6-month-old infant was referred to our department for MRI of the brain for evaluation of poorly controlled recurrent seizures.

Imaging Findings:

A 6-month-old infant was referred to our department for MRI of the brain for evaluation of poorly controlled recurrent seizures. The child was born out of a non-consanguineous marriage to a 24-years-old primigravida. It was a full-term normal vaginal delivery. Mother was healthy and non-diabetic and had an uncomplicated pregnancy. There was no history of birth asphyxia or complicated/prolonged labour. Child weighed 3kg at birth, had a normal APGAR score, was fed within 1st hour of delivery with no feeding difficulties during the first three days. On the 3rd post-delivery day, the mother noted that he was feeding poorly and had become increasingly sleepy. On the subsequent day he was brought to the hospital with seizures. His general examination and haematological examination was within normal limits except for low serum glucose of 20mg/dL. In spite of starting intravenous dextrose, the child convulsed repeatedly over the next 3 days. No underlying metabolic cause could be attributed for the hypoglycemia. CT scan of brain performed at that time was noncontributory and did no reveal any significant lesion. Subsequently, at 6 months of age he was brought to us for MR evaluation which revealed gliosis and cystic encephalomalacia in the bilateral parieto-occipital region. These changes were asymmetric, appearing more severe on the left side as compared to the contralateral side, and involved both the cortex and the underlying cerebral white matter.

Remaining brain parenchyma, infra-tentorial structures, basal ganglia and thalami appeared normal. MRI findings and relevant clinical history was consistent with neonatal hypoglycemic cerebral insult.

Discussion:

Hypoglycemia is the most common metabolic disorder encountered in the neonatal period. When compared to adults, the neonates have a proportionately higher demand for blood glucose owing to their higher brain-to-bodyweight ratio. Cerebral glucose uptake accounts for 90% of total glucose consumption in a neonate and the major source of brain glucose is the blood supply. Thus an adequate glucose supply is imperative for brain metabolism, without which the child may develop severe neonatal hypoglycemic encephalopathy. Hypoglycemia in the newborn may result in acute decompensation with or without permanent brain damage. Up till now, there is no discrete explanation for the parieto-occipital vulnerability to hypoglycemic brain insult. However, a variety of case reports pertaining to imaging in neonatal hypoglycemia have established the predominant damage to parieto-occipital lobes. The exact aetiology/pathogenesis is however still not well understood. A reduction in blood glucose is considered responsible for reduced neuronal electrical activity, membrane breakdown and altered amino acid metabolism in the brain, with resultant increased glutamate production. Glutamate is believed to play a major role in the pathophysiology of hypoglycemic brain injury. Hypoglycemia is associated with increased glutamate concentrations in the synaptic cleft. Excess activation of NMDA (N-methyl-D-aspartate) receptors by glutamate
leads to rigorous ionic imbalance and resultant selective neuronal necrosis. The probable basis for the parieto-occipital involvement may relate to high degree of axonal migration and synaptogenesis occurring within these areas during the neonatal period. Imaging studies are considered crucial in neonatal hypoglycemia to determine the characteristics and severity of cerebral lesions after hypoglycemia and predict the long term clinical outcome. On neuro-imaging, brain damage with predominantly posterior involvement is seen on CT or MR imaging. CT in the acute stage may show non-enhancing hypoattenuating area in the bilateral parieto-occipital region with loss of cortico-medullary differentiation. MR imaging demonstrates altered signal-intensity of the cortex as well as the subcortical white matter, especially posteriorly in the parieto-occipital region. This may demonstrate bright signal on diffusion-weighted (DW) imaging in the acute setting. Eventually glial injury results in atrophy, gliosis and encephalomalacia with resultant widening of adjacent sulci and subarachnoid CSF-spaces. Other sites which may be involved are white matter elsewhere, caudate-putamen, dentate gyrus and hippocampus. In contrast, hypoxic ischemic injury in preterm neonates typically manifest as periventricular white matter damage or periventricular leukomalacia and clinically manifest as cerebral palsy or spastic diplegia which is uncommon in neonates with hypoglycemic encephalopathy. The long term consequences of occipital brain injury include chronic occipital lobe epilepsy and visual impairment. Neonates with extensive cerebral insult may also develop cognitive impairment or global developmental delay.

**Differential Diagnosis List:** Neonatal hypoglycemic brain insult

**Final Diagnosis:** Neonatal hypoglycemic brain insult

**References:**


**Description:** Axial T2-w images show encephalomalacic changes in the bilateral parieto-occipital region (left>right). **Origin:**

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*Figure 1a*
Description: Axial T2-w images show encephalomalacic changes in the bilateral parieto-occipital region (left>right). Origin:
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Figure 2

Description: Axial T1-w images show cystic encephalomalacic changes in the bilateral parieto-occipital region (left-right). Origin:
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