Neonatal Seizures

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Abstract- ***A low-birth-weight term neonate born at 38 weeks 5days to non-diabetic primigravida mother by normal vaginal delivery with uneventful perinatal events presented with complaints of recurrent seizures on day two of life. Antenatal ultrasound scans showed features of early intrauterine growth restriction. The baby had jitteriness, poor activity and poor feeding. The mother gives a history of inadequate lactation. Routine new born blood screening showed reduced blood sugar levels with multiple readings in the range of 30-50mg/dl despite corrections. Metabolic investigations didn't show any cause for hypoglycemia. EEG was normal.***

I. IMAGING FINDINGS

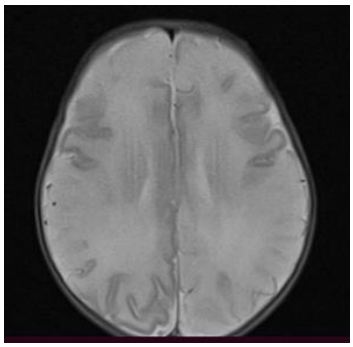

Fig 1 – T2 axial images of the brain shows increased signal in the white matter of bilateral frontal, parieto-occipital lobes with thinned out cortex and ill-defined grey and white matter differentiation.

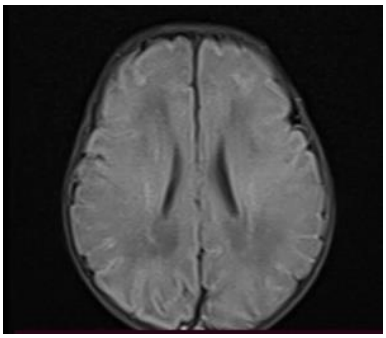

Fig 2- T2 FLAIR images show hyper intensities in the cortical grey matter of all lobes of both cerebral hemispheres.

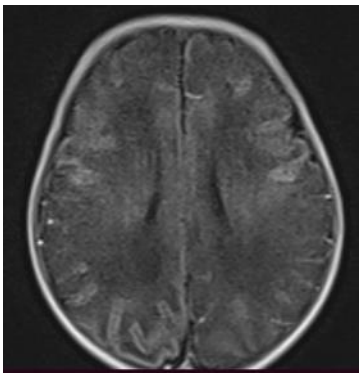

Fig 3 – T1 FLAIR images show hyperintensities in cortical grey matter and basal ganglia of all lobes of both cerebral hemispheres.

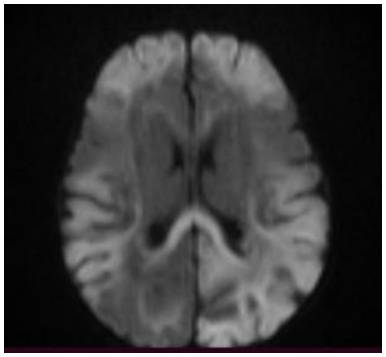

Fig 4 – DWI images show extensive areas of diffusion restriction involving all lobes of both cerebral hemisphere, corpus callosum, posterior limb of bilateral internal capsule.

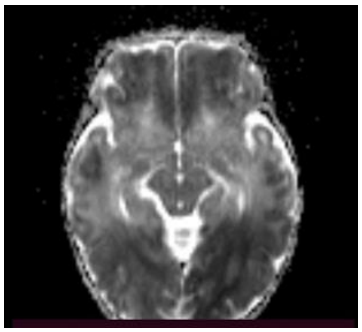

Fig 5 - ADC images show areas of low ADC involving all lobes of bilateral cerebral hemisphere, corpus callosum and posterior limb of bilateral internal capsule.

T2 axial images of MRI- brain showed abnormal high signal changes in the white matter of all lobes of bilateral cerebral hemispheres with loss of grey and white matter differentiation. Abnormal gyral hyper-intensities are seen in T1WI, T2FLAIR images with generalized thinned-out cortex. Extensive areas of diffusion restriction with low ADC values were seen in bilateral frontal lobes, bilateral parieto-occipital, and temporal lobes, splenium and genu of the corpus callosum, and posterior limb of bilateral internal capsule. All the imaging features were in the favor of hypoglycemic insult. Most of the published cases related to imaging findings of central nervous system in neonatal hypoglycemia show changes predominantly in occipital and parietal lobes but in our case, it was diffuse involvement in all lobes of both the cerebral hemispheres and the corpus callosum.

II. DISCUSSION

Brain needs continuous supply of oxygen and nutrients for normal functioning. Any significant deprivation of these nutrients especially glucose can result in significant brain damage with permanent sequelae. Neonate born prematurely and with low birth weight are more susceptible for insults from hypoglycemia, hypoxia and hypothermia. Glucose monitoring is commonly ignored in care of new born in developing countries. Several factors may interfere with glucose homeostasis like limited metabolic stores commonly seen in low-birth-weight preterm babies.

Hypoglycemic insults on brain can have both acute and long-term abnormalities in infants and children. Acute manifestations of hypoglycemia that may precede abnormal neurologic development include jitteriness, seizures, and vomiting. More delayed neurologic sequelae may include seizures, mental retardation, neurodeficits, spasticity, and microcephaly. [1]

Imaging studies of brain in multiple studies of neonatal hypoglycemia showed significant parenchymal loss most prominent in the occipital region. T2/FLAIR hyper-intensities in subcortical white matter and cortical thinning with ill-defined grey and white mater interface commonly seen in the parieto-occipital cortex in acute phase with profound volume loss, encephalomalacia and gliotic changes of these regions with neurological sequelae on long term follow up [3]. The findings presented seem to be relatively specific for neonatal hypoglycemia and correlate well with previously published reports. [1] In our case with severe hypoglycemia there is involvement of both the frontal and temporal lobes, the corpus callosum, posterior limb of the internal capsule with significant diffusion restriction and low ADC values which is a rare entity.

Although the effect of mild transient asymptomatic hypoglycemia on brain development remains unclear, a correlation between severe and prolonged hypoglycemia and cerebral damage has been proven. On long-term follow up there will be cortical atrophy with white matter volume loss and associated delayed neurological sequelae. Hence it is important to initiate breastfeeding as soon as possible after birth, monitor poor feeding babies born to mothers with reduced milk output for possible hypoglycemia, and initiate early and prompt treatment to prevent brain injury. This is a very much preventable and easily treatable condition.

III. FINAL DIAGNOSIS

Neonatal hypoglycemia

REFERENCES

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