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Case Report

NEONATAL HYPOGLYCEMIA BRAIN INJURY – A CASE REPORT

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ABSTRACT
We report a case of a 2 day old full term male newborn with symptomatic hypoglycemia and sequelae of neonatal hypoglycemia brain injury (NHBI) justified at day 7 of post natal life. NHBI has not yet been well understood by some clinician no diagnostic criteria have been available due to lack of specific clinical manifestation, although brain imaging studies are now an important diagnostic and prognostic tool, Rozance *et al.,* (2006). Early detection of the imaging evidence of NHBI is important for the early judgment of the disease and assessment of prognosis.

Keywords: *Hypoglycemia, NHBI*

INTRODUCTION
Hypoglycemia is the commonest metabolic disorder in newborn. The overall incidence of hypoglycemia in neonates varies from 0.2% to 11.4%. However, in the presence of certain risk factor i.e. small for date, large for date, infants of diabetic mother, prematurity etc, the probability of hypoglycemia increases many fold, Cornblath *et al.,* (1996) and Dutta *et al.,* (2000). Hypoglycemia can be symptomatic with features of jitteriness, convulsion, refusal to feed, hypotonia, cyanosis, high pitched cry, hypothermia or may be asymptomatic. Hypoglycemia is known to be associated with brain dysfunction and neuromotor developmental retardation in both symptomatic and asymptomatic cases, Cornblath *et al.,* (1996) and Williams (1997). It leads to brain cell softening, swelling, necrosis, gyri atrophy, or white matter demyelination, Tam *et al.,* (2012). This form of brain injury due to hypoglycemia is termed as neonatal hypoglycemic encephalopathy or neonatal hypoglycemic brain injury, Lou *et al.,* (2010).

CASES
Clinical Presentation: A day 3 male babies was brought in the OPD by the father with complaints of one episode of stiffness of upper and lower limbs lasting for few seconds associated with upward rolling of eyes. Baby was advised admission however the father refused for the same. In the evening the baby was again brought in the emergency with similar episode of abnormal movement and letharginess.
History of Present Illness: The baby was noted to have symptoms of lethargy and an episode of abnormal movement at day 2 at home. The baby was being fed with very little amount of artificial feeds in addition to breast milk.
Past History: The baby was a full term male baby born by normal vaginal delivery. There was no significant history of any natal or post natal events and the baby was discharged on day 2 of life on breast feed.
***Clinical Examination Findings*:** On admission, the baby had following vital parameters:-
Heart Rate- 160/min, respiratory Rate -56/ min, CFT < 3sec, spo2 -92 %, temperature 98.2^0F and evidence of jitteriness +ve.
Investigations and Treatment Outline: Random blood sugar (rbs) by strip showed a low value. Hence, baby was given i.v bolus of 10 % dextrose 22ml/kg and samples for sepsis screen, serum electrolytes and serum calcium was send. I.V antibiotics were started and glucose was infused @ 6mg/kg/min with regular rbs monitoring. In order to abort the seizures, midazolam stat dose and inj. phenobarbitone was given.
The maximum glucose infusion rate went up to 10mg/kg/min on day 3 of admission. Gradually feed was added and fluid tapered off accordingly. By day 7 the baby was put on breast feeds and neuroimaging as MRI brain was planned. The cause of neonatal seizures in this case after investigation was found to be hypoglycemia as the sepsis screen did not reveal any thing significant and the serum calcium and serum

electrolyte were in the normal range. Since the baby was full term /AGA with LBW of 2.066 kg, ineffective feeding was supposed to be the cause of neonatal hypoglycemic seizures. The MRI of the brain revealed features suggestive of the same. There were extensive areas of diffusion abnormality involving both grey and white matter in bilateral parieto-occipital regions, posterior part of body and splenium of corpus callosum, bilateral pulvinar nuclei and posterior limb of bilateral internal capsules.

DISCUSSION

In 1959, Cornblath *et al.*, reported 8 cases of symptomatic neonatal hypoglycemia. Data have shown that if neonatal hypoglycemia is not timely treated, the infant may develop permanent brain injury and neonatal hypoglycemic encephalopathy, Lou *et al.*, (2010). However, no prospective, randomized controlled trial (RCT) has confirmed the long term sequelae of hypoglycemia and no experiment has confirmed the exact extent and duration of hypoglycemia that causes brain injury. Boluyt *et al.*, (2006) found that by far no clinical research of high quality on the exact relationship between blood glucose level and neurological outcome had presented convincing results. The gestation that is more vulnerable is still debatable. In studies of Dhananjay *et al.*, (2011) and Singhal *et al.*, (1992), most of the infants with birth asphyxia and hypoglycemia were of term birth i.e. 61.11% and 71.5% respectively; suggesting that the glucose released in stress is rapidly utilized in term infants, while it is poorly utilized in pre term infants.

Energy metabolic disorders during hypoglycemia may lead to brain cell softening, swelling, necrosis gyrus atrophy or white matter demyelination, Tam *et al.*, (2012). Su *et al.*, (2007) have found that hypoglycemia and hypoxemia can induce similar changes in brain function but different intracerebral pathological changes. Takeuchi *et al.*, (2005) found that capsule interna, splenium of corpus callosum and corona radiate could also be involved during injury associated with hypoglycemia similar to imaging findings in our study.

Take Home Message

1. Neonatal hypoglycemia is a common metabolic alteration found in both preterm and term babies due to various factors.
2. Morbidity in the form of long term neurodevelopment sequelae can occur following an episode of symptomatic hypoglycemia. Hence, early recognition and treatment for hypoglycemia in neonates, especially high risk cases, is necessary.
3. Brain imaging should be routinely carried out in neonate with symptomatic hypoglycemia in order to screen for the underlying hypoglycemic changes or damage and accordingly define further regular follow up of babies to screen for any early or late neurological deficiencies.

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