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Radiodiagnosis

Utility of MRI in Neonatal Hypoglycemic Brain Injury (NHBI) – Case Series

Dr. Sanjay M. Khaladkar[1*], Dr. Aarushi Gupta[2], Dr. Rajesh S. Kuber[3], Dr. Akshay Waghmode[4], Dr. Rajshree Dhadve[5], Dr. Dwaram Jyoti Reddy[6]

[1]Professor, Department of Radiodiagnosis, Dr.D.Y.Patil Medical College and Research Center, Dr.D.Y.Patil Vidyapeeth, Pimpri, Pune India
[2]Post graduate resident, Department of Radiodiagnosis, Dr.D.Y.Patil Medical College and Research Center, Dr.D.Y.Patil Vidyapeeth, Pimpri, Pune India
[3]Professor, Department of Radiodiagnosis, Dr.D.Y.Patil Medical College and Research Center, Dr.D.Y.Patil Vidyapeeth, Pimpri, Pune India
[4]Post graduate resident, Department of Radiodiagnosis, Dr.D.Y.Patil Medical College and Research Center, Dr.D.Y.Patil Vidyapeeth, Pimpri, Pune India
[5]Assistant Professor, Department of Radiodiagnosis, Dr.D.Y.Patil Medical College and Research Center, Dr.D.Y.Patil Vidyapeeth, Pimpri, Pune India
[6]Post graduate resident, Department of Radiodiagnosis, Dr.D.Y.Patil Medical College and Research Center, Dr.D.Y.Patil Vidyapeeth, Pimpri, Pune India

Original Research Article

***Corresponding author**
Dr. Sanjay M. Khaladkar

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Abstract: To assess the role of MRI in neonatal hypoglycemia, a prospective study was done on 10 neonates in the Department of Radiodiagnosis in Dr. D. Y. Patil Medical College and Research Centre, Pimpri, Pune, who were clinically suspected to have hypoglycaemia. Out of the 10 cases examined : 4 patients showed restricted diffusion in parieto-occipital lobes involving both grey and white matter, 1 patient showed restricted diffusion in splenium of corpus callosum, 1 patient showed multiple small areas of hemorrhage in both cerebral hemispheres, 2 patients showed cortical laminar necrosis with restricted diffusion of splenium, 1 patient showed restricted diffusion in splenium and parieto-occipital lobes with hemorrhage in cerebral hemispheres, 1 patient showed restricted diffusion in splenium with hemorrhagic foci in cerebral hemispheres. 2 of these patients also showed unmyelinated posterior limb of internal capsule. Neonatal hypoglycemia can occur in preterm infants, small-for-gestational-age infants, and infants of diabetic mothers. Persistent or recurrent hypoglycemia can result in neonatal permanent brain injury like vision disturbance, occipital lobe epilepsy, cerebral palsy and cognitive impairment. Injury of splenium of corpus callosum along with involvement of bilateral parieto-occipital cortex and subcortical white matter are the most commonly observed injury patterns in neonatal hypoglycemia. MR imaging can be reliably used in the absence of laboratory values to suggest hypoglycemic brain injury. MRI, especially DWI with ADC mapping is extremely useful in detection of hypoglycemic insult in neonate. Restricted diffusion in the splenium of corpus callosum, parieto-occipital lobes and hemorrhagic foci in cerebral hemispheres are useful signs in detecting neonatal hypoglycemia.
Keywords: Neonatal hypoglycemia, parieto-occipital, splenium, diffusion restriction.

INTRODUCTION

Neonatal encephalopathy is a syndrome which results from disturbed neurological function in early neonatal life in the term infants. It can occur due to cerebral dysgenesis, congenital infections, hypoxia and hypoglycaemia. It occurs in 1 to 6 per 1000 live term births and can result in significant morbidity and mortality.

The first two can be prevented and are also treatable. Neonates with neonatal encephalopathy caused by hypoxic ischemic encephalopathy (HIE) are at increased risk of depleting their energy stores and developing concurrent hypoglycaemia. Neonatal hypoglycemia (<46 mg/dL) occurs in 5% to 15% of normal term neonates. It can cause visual impairment, epilepsy and cognitive deficits. To predict neurodevelopmental outcome, the aetiology and time of the brain injury need to be identified. MR imaging with diffusion weighted imaging can predict the extent of injury and predict outcome [1].

MATERIALS AND METHODS

Our study included a total of 10 term infants (36-42weeks) who were clinically suspected with hypoglycaemia and/ or recorded low blood sugar. Sequences taken were: Axial T1WI, T2WI, FLAIR, DWI with ADC, GRE; coronal T2WI and sagittal T1WI.

Inclusion criteria were term infants, clinical history of hypoglycaemia, recorded low blood sugar levels on Day 1 - <30mg/dl, recorded low blood sugar levels after 24 hours - <45mg/dl and severe hypoglycaemia < 27mg/dl

Exclusion criteria were preterm infants and clinically suspected HIE. 7 infants had low birth weight, 2 infants had history of maternal diabetes and 1 infant was large for date (Table 1). 6 infants presented with convulsions, 1 infant had lethargy with hypotonia, 1 infant was irritable with poor feeding while 2 were asymptomatic (Table 2). Restricted diffusion in parieto-occipital lobes involving both grey and white matter was observed in 4 infants, restricted diffusion was observed in splenium of corpus callosum in 2 infants, multiple small areas of haemorrhage were observed in both cerebral hemispheres in 1 infant, cortical laminar necrosis with restricted diffusion of splenium was seen in 2 infants, restricted diffusion in splenium and parieto-occipital lobes with multiple areas of haemorrhage in both cerebral hemispheres was observed in 1 infant (Table 3).

OBSERVATIONS AND TABLES

Table-1: Risk factors

Low birth weight	7
Maternal diabetes	2
Large for date	1

Table-2: Presenting symptoms

Asymptomatic	2
Convulsions	6
Lethargy with hypotonia	1
Irritability, poor feeding	1

Table-3: MRI findings

Restricted diffusion in parieto-occipital lobes involving both grey and white matter	4
Restricted diffusion in splenium of corpus callosum	2
Multiple small areas of haemorrhage in both cerebral hemispheres	1
Cortical laminar necrosis with restricted diffusion of splenium	2
Restricted diffusion in splenium and parieto-occipital lobes with haemorrhage in cerebral hemispheres	1

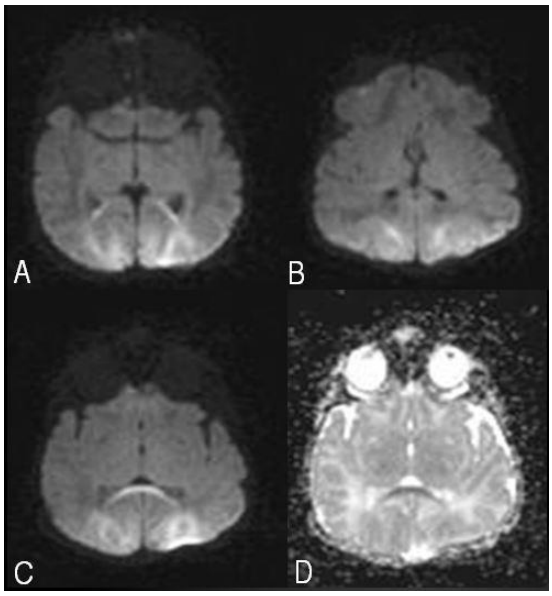

Fig-1: DWI (A-C) and ADC (D) showing diffusion restriction in parieto-ocipital white matter and splenium with low ADC values

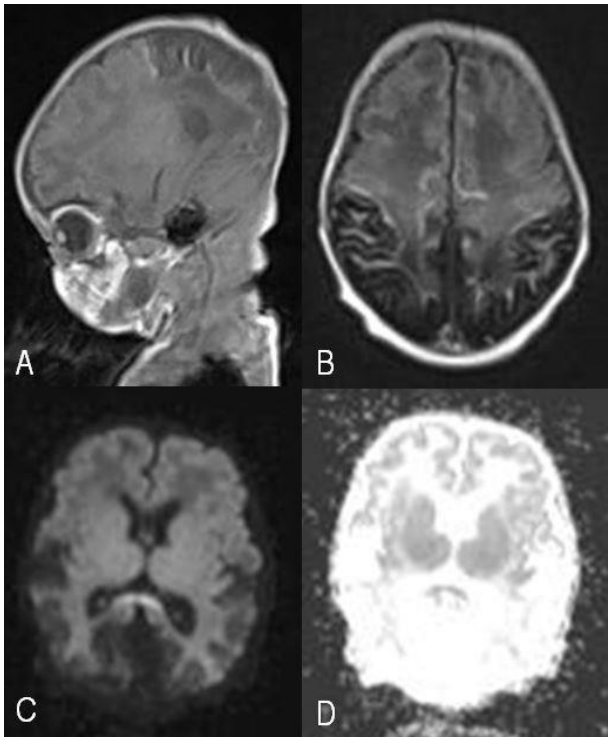

Fig-2: T1WI – parasagittal (A), axial (B), DWI (C) and ADC (D) showing cortical laminar necrosis with restricted diffusion in splenium with low ADC values. Cortical laminar necrosis shows gyriform hyperintensity on T1WI

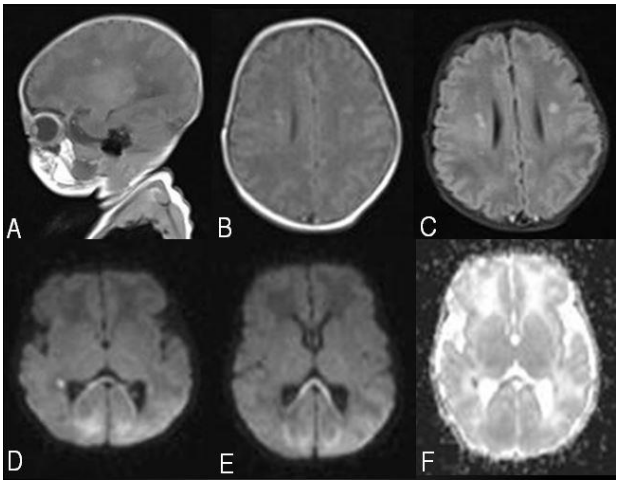

Fig-3: TIWI – parasagittal (A), axial(B), FLAIR(C), DWI(D, E) and ADC (F) – showing restricted diffusion in splenium with low ADC values. Punctate haemorrhage in bilateral periventricular white matter appear hyperintense on T1WI and FLAIR with restricted diffusion on DWI

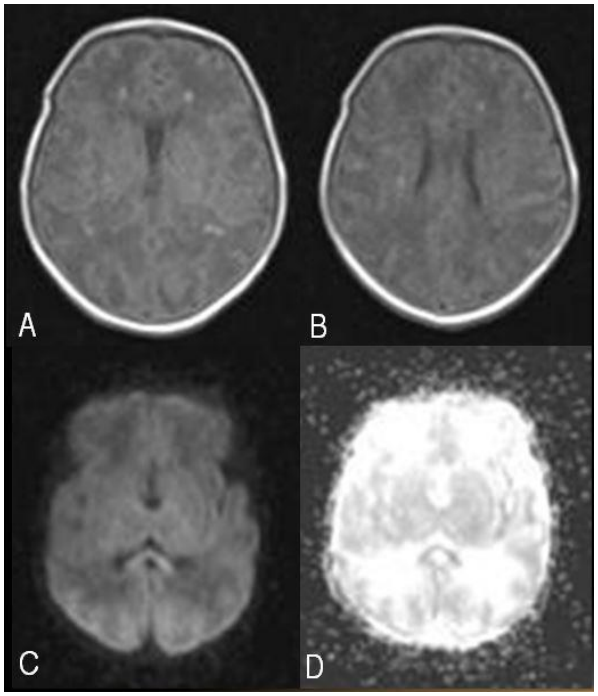

Fig-4: Axial T1WI (A, B), DWI (C) and ADC(D) showing multiple punctate foci of haemorrhage in bilateral cerebral white matter appearing hyperintense on T1WI and restricted diffusion in splenium with low ADC values

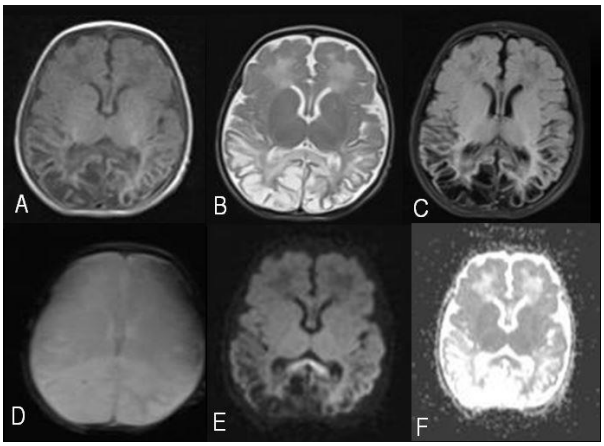

Fig-5: T1WI (A), T2WI (B), FLAIR(C), GRE (D), DWI (E), ADC (F) showing cystic encephalomalacia with restricted diffusion in splenium. Cystic encephalomalacia appears hypointense on T1WI and FLAIR and hyperintense on T2WI. Restricted diffusion in splenium appears bright on DWI with low ADC values

DISCUSSION

Neonatal hypoglycaemia can occur in preterm infants, small-for-gestational-age infants, infants of diabetic mothers. Persistent or recurrent hypoglycaemia can result in neonatal permanent brain injury like vision disturbance, occipital lobe epilepsy, cerebral palsy and cognitive impairment [2].

Diagnostic criteria for neonatal hypoglycemic brain injury (NHBI) - Mao Jian *et al*. have presented the diagnostic basis of NHBI:

Obvious hypoglycaemia-related clinical manifestations or history of severe hypoglycaemia (< 1.7 mmol/L) at admission; whole blood glucose ≤2.0 mmol/L; manifestations of nervous system dysfunction during hypoglycaemia and for a period of time after the correction of blood glucose; brain injury changes under magnetic resonance imaging ; after ruling out brain injuries caused by abnormal brain development, intracranial infection, intracranial haemorrhage, sepsis, congenital metabolic disorders and endocrine diseases[3].

Pathological changes for NHBI-Hypoglycemia causes brain cell softening, swelling, necrosis, grey matter atrophy and white matter demyelination. Though hypoxia and hypoglycaemia induce similar changes in brain function, their pathological changes differ. NHBI affects bilateral posterior parieto-occipital lobes, do not match vascular territory.Brain stem and cerebellum are often not

involved and usually not accompanied by cerebral haemorrhage, while hypoxic injury has specific pattern in pre term and term infants [2].

Hypoglycemia involves Layer 2 and 3 of cerebral cortex. Ischemia affects pyramidal cells in Layers 3, 5, 6[4]. Imaging manifestations of neonatal NHBI- MRI is a sensitive and a specific screening method for diagnosing NHBI. DWI and ADC images can detect early brain injury within 24 hours, while conventional MRI sequences take 5 – 6 days to detect them. Magnetic resonance diffusion tensor imaging (DTI) can detect abnormal myelination during mild brain white matter injury. Magnetic resonance spectroscopy (MRS) can be used for detection of other metabolites, including lactic acid and kreatine. DWI can detect hypoglycaemic injury in early stages (within 24 hours) when conventional MRI sequences are normal [2].

Patterns of cerebral injury in neonatal hypoglycaemia are as follows

Injury of the splenium of corpus callosum along with involvement of bilateral parieto-occipital cortex and subcortical white matter are the most commonly observed injury pattern in neonatal hypoglycemia [2]. (Figure 1).

Other patterns are cortical laminar necrosis (Figure 2), absence or abnormal myelination in the posterior limb of internal capsule (PLIC), haemorrhage or abnormal signal intensity in cerebellum and/or brainstem, extracerebral or intraventricular haemorrhage and focal haemorrhage in white matter (Figure 3, 4), MCA infarct or widespread infarct[5].

Neonates with HIE are at increased risk of concurrent hypoglycaemia. It is difficult to differentiate hypoglycaemic brain injury from HIE on imaging alone unless there is parieto-occipital distribution. Patterns of HIE in full terms infants on MRI are as follows: peripheral or watershed pattern in mild to moderate HIE; basal ganglia, thalamus pattern – severe HIE, (acute <10mins); total brain injury – severe HIE (prolonged 15-25 minutes)[1].

HIE with superimposed hypoglycemia is often seen (Figure 5). Injury to posterior white matter and pulvinar in cases of hypoglycemia is diagnostic of hypoglycemic injury superimposed on HIE. Watershed pattern of HIE is frequently associated with hypoglycaemia [1].

Brain injury pattern in NHBI are posterior white matter abnormalities, cortical injury; white matter haemorrhage; injury of basal ganglia, thalami and PLIC; restricted diffusion in the occipital lobes, parieto-occipital grey and white matter, corpus callosum and optic radiation are the other patterns of NHBI injury. Focal or multiple areas of punctuate haemorrhage may be seen in the white matter in NHBI. Though no true histopathological evidence of haemorrhage has been documented, these foci may be due to ischemia with secondary haemorrhage and may be arterial or venous. Another less accepted theory is hypoglycaemia with concomitant polycythemia with resultant CVST. Parieto –occipital lobes are vulnerable to hypoglycaemic injury. The exact mechanism is not known. There is regional increase in blood flow during hypoglycaemia with reduction in glucose extraction. This causes cellular injury (increased mitochondrial free radical generation and initiation of apoptosis, increased activity of excitatory neurotoxins at N-methyl-D-aspartate receptors and altered cerebral energetic characteristics). White matter injury occurs in NHBI as an exaggerated pathophysiological response, compared with other cerebral tissue types. Another theory is less mature white matter in such neonates [5].

CONCLUSION

Neonatal hypoglycaemia can occur in preterm infants, small-for-gestational-age infants and infants of diabetic mothers. Posterior white matter abnormalities, cortical injury, white matter haemorrhage, injury of the basal ganglia and thalami and PLIC are commonly observed in NHBI. Restricted diffusion in the occipital lobes, parieto-occipital grey and white matter, corpus callosum and optic radiation are the other patterns of NHBI injury. MRI is a sensitive and a specific screening method for diagnosing NHBI. DWI and ADC images can detect early brain injury, within 24 hours, while conventional MRI sequences take 5–6 days to detect them.

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