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Research Article

Prognostic Utility of Magnetic Resonance Imaging of Hypoxic Ischemic Encephalopathy at Tertiary Care Teaching Hospital

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Abstract: ***Background:*** Hypoxic-ischaemic encephalopathy in adults and older children (i.e. not neonates), also known as global hypoxic-ischaemic injury, is seen in many settings and often has devastating neurological sequelae. Magnetic Resonance imaging—has the potential to play a significant role in diagnosis and early intervention in cases of HII. In addition, imaging studies performed in the subacute stages of injury provide information on the severity and extent of injury and can be helpful in predicting long-term outcome. ***Material and Methods:*** This prospective study was conducted in a Department of Radiology, Shadan Institute of Medical Sciences, Teaching Hospital & Research Centre, Hyderabad over a period of 1 year. A total of 70 patients with history of birth asphyxia were included in the study who underwent MRI of brain and were followed up clinically at the end of one year to assess the neurological outcome. ***Result:*** Of the 65 babies, 43 were males and 22 females, which correspond to 66.1% of male and the rest female babies. Maximum patients, i.e., 53.8% (n = 35) were having Apgar score of 4-6 followed by ≤3 score was 29.2% and least were > 7 score were 16.9%. In HIE 2 cases, 32.3% had involvement of corpus callosam. 24.6% had PVL, 16.9% had basal ganglia or thalamus lesion. There was no MRI evidence of HIE in 26.1% ***Conclusion:*** HIE is an important cause of morbidity and mortality in the neonatal period. MRI show characteristic pattern of brain injury and help to exclude other causes of encephalopathy. Imaging plays an important role in early diagnosis and timely intervention, thereby reducing the severity of neonatal brain injury.

Keywords: Magnetic Resonance imaging, Hypoxic ischemic encephalopathy, Neurological impairment.

INTRODUCTION

Hypoxic-ischemic encephalopathy (HIE) in term and near-term newborns is an important cause of morbidity and mortality. [1] Therapeutic hypothermia has been a major advance in the management of neonatal HIE. Meta-analyses of several large multicenter trials have concluded that hypothermia treatment is associated with a reduction in death and neurological impairment in early childhood. [2] These results were confirmed in the recently reported Infant Cooling Evaluation (ICE) randomized controlled trial, in which whole-body hypothermia treatment was shown to reduce death or major sensorineural disability at 2 years of age compared with normothermia. [3]

Magnetic resonance imaging (MRI) assists in defining the nature and extent of perinatal brain injury. Because hypoxic-ischemic (HI) cerebral injury is a dynamic process, the diagnostic and prognostic utility of MRI needs to be interpreted in the context of the timing of the MRI. Patterns of brain injury on conventional T1- and T2-weighted MRI at 1 week after birth have been shown to predict abnormal neuromotor outcome in early childhood. [4] Diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) changes with HI injury are most prominent from days 2 through 5 and can be detected earlier than abnormalities detected on the conventional T1- and T2-weighted MRI. [5] In the Total Body Hypothermia for Neonatal Encephalopathy (TOBY) trial, MRI studies performed a median of 8 days after birth reported a reduction in the incidence of cerebral injury compared with normothermia but consistent prognostic value from MRI irrespective of treatment. [6-8] However, we need to determine whether this prognostic utility is similar in a different cohort with MRI performed at a different median age.

MATERIAL AND METHODS

This prospective study was conducted in a Department of Radiology, Shadan Institute of Medical Sciences, Teaching Hospital & Research Centre, Hyderabad over a period of 1 year. A total of 70 patients with history of birth asphyxia were included in

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the study who underwent MRI of brain and were followed up clinically at the end of one year to assess the neurological outcome.

Inclusion criteria

Full term (>37 weeks of gestation),
Pre-term (<37 weeks of gestation)
Neonates born with birth asphyxia and APGAR score at 5 minutes after birth

Exclusion criteria

Term or preterm neonates with infection and suspected Metabolic disease.
Patients with prosthesis, heart valve prosthesis, artificial/prosthetic limb, surgical staples, clips or metallic sutures and claustrophobia.

A 1.5 T MR scanner was used, with a gradient system that can reach a maximum gradient strength of 21 m T/m in each main direction. The imaging protocol consisted of a spin-echo T1-weighted series (568/18 TR/TE), a turbo spin-echo T2- weighted series (4381/120), and an inversion-recovery series. (3436/18/400 TR/TE/IR). Standard 8-channel birdcage (volume) coil was used. Anaesthesiologists using intravenous Propofol according to weight, after pre-anaesthesia check, sedated the neonates.

Images obtained were analysed on the workstation. Images were assessed for the presence of ischemic damage by the investigators. The extent of ischemic damage in each subject was determined according to MRI findings. Three plane anisotropic diffusion weighted images were examined for signal changes not accounted for by normal white matter anisotropy. Conventional MR studies were examined for the findings of HIE, blinded of the diffusion-weighted imaging appearances. The location and extent of involvement (by volume) of ischemic damage on each sequence were noted.

Statistical analysis

Statistical analysis was done using SPSS version 15 and sensitivity, specificity, positive and negative predictive value of MRI in comparison to clinical follow up at the end of one year was assessed. A grading system was devised for both MRI and clinical follow up for statistical purpose.

Result

A total of 65 patients who fulfilled the selection criteria during the study were enrolled. The data were analysed, and the final observations were tabulated as below.

Table 1: Distribution of Gender

Sex	No. of patients	Percentage
Male	**43**	**66.1**
Female	**22**	**33.8**
Total	**65**	**100**

Of the 65 babies, 43 were males and 22 females, which correspond to 66.1% of male and the rest female babies in table 1.

Table 2: Distribution of the number of children according to age group

Age group	No. of patients	Percentage
< 1 month	**31**	**47.6**
2-12 months	**23**	**35.8**
> 1 year	**11**	**16.9**
Total	**65**	**100**

In table 2, the maximum number of patients were in the age group of <1 year which were 47.6% (n =31) of total followed by age group 2–12 months having 35.8% (n = 23) in this group and 16.9% were more than 1 year.

Table 3: Clinical profile distribution among study population according to Apgar score

Apgar score	No. of patients	Percentage
Score > 7 Generally Normal	**11**	**16.9**
Score of 4-6; fairly low	**35**	**53.8**
Scores ≤3; critically low, needs intervention	**19**	**29.2**
Total	**65**	**100**

In our study, maximum patients, i.e., 53.8% (n = 35) were having Apgar score of 4-6 followed by ≤3 score were 29.2% and least were > 7 score were 16.9% in table 3.

Table 4: Distribution of MRI changes in study population with stage2 HIE

Site of Lesion	Frequency	Percentage

Corpus Callosam	**21**	**32.3**
BG/thalamus	**11**	**16.9**
No Change	**17**	**26.1**
Periventricular leucomalacia	**16**	**24.6**

In HIE 2 cases, 32.3% had involvement of corpus callosam. 24.6% had PVL, 16.9% had basal ganglia or thalamus lesion. There was no MRI evidence of HIE in 26.1%.

Table 5: Distribution of MRI changes in study population with stage 3 HIE

Site of Lesion	Frequency	Percentage
Bilateral BG	**36**	**55.3**
Bilateral thalami	**23**	**35.3**
Subcortical white matter	**6**	**9.2**

Out of 6 babies with stage 3 HIE, 55.3% had involvement of bilateral basal ganglia. 35.3% had bilateral thalami lesion and the rest showed subcortical white matter lesion.

DISCUSSION

HIE occurs when the oxygen and blood supply to a baby's brain is cut off or severely limited. This deprivation causes cells in the brain to break down, eventually leading to cell death if deprivation continues. When cells in the brain die, brain damage results. This damage can often be identified with an MRI. [9]

During an MRI of the brain, images are taken from the top of the baby's skull down to the base, from the front of the skull to the back, and across the skull from side to side. [10] When the results come back, each image represents a unique slice of the brain, ensuring that all areas of the baby's brain are imaged. Often, when a baby has brain damage, it will show up in one or more of these images in areas with increased signal intensity. [11]

In our study, out of the 65 patients who were enrolled in the study, our study shows male preponderance. According to a study by Flodmark O et al, there was no gender predilection. [12] Male gender being a risk factor for HIE has also been reported by other studies. [13] Our study shows, that term babies are more affected by MRI than preterms it may be because neonatal brain injury is difficult to diagnose in premature infants because either obvious signs are absent or if present, are attributed to developmental immaturity. [14] Preterm infants can also suffer from hypoxic ischemic encephalopathy, but, most often the change is not recognized early. For preterms findings will be obvious when MRI is done at corrected gestational age. Significantly higher numbers of primi gravida mothers in the affected babies are seen. It may be because the first delivery is more difficult than the subsequent ones. This points to the importance of intrapartum factors in the causation of HIE. [15]

In our study, 86% of term babies had changes in basal ganglia and/or thalamus. Other authors have also observed this finding. [16-21] This is because basal ganglia and thalami are metabolically very active in the immature brain. Occasionally severe basal ganglia lesions are seen with less obvious precipitating events. This may reflect failure to recognize the severity of asphyxia or due to individual susceptibility to damage because of previous hypoxic ischemic events or underlying metabolic or thrombotic disorders.

Term infants who develop HIE following a well-defined acute hypoxic injury typically sustain bilateral lesions within the basal ganglia and thalami. In this study, out of the six babies with clinical stage 3 HIE 52.8% of them had bilateral basal ganglia involvement and 34.2% had bilateral thalami involvement. In stage 2 HIE no stage specific change in MRI could be found. Preterm brain is highly susceptible to injury including periventricular leucomalacia, intraventricular hemorrhage/ germinal layer hemorrhage and parenchymal hemorrhagic infarction. In this study 27.1% of preterm babies had periventricular leucomalacia.

CONCLUSION

HIE is an important cause of morbidity and mortality in the neonatal period. MRI show characteristic pattern of brain injury and help to exclude other causes of encephalopathy. Imaging plays an important role in early diagnosis and timely intervention, thereby reducing the severity of neonatal brain injury. We can predict the infant development based on the MRI pattern of hypoxic-ischemic lesions, however, we cannot forget about the amazing malleability/flexibility of the child brain that can surprise both radiologists and clinicians.

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