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Original Research Paper

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ROLE OF MRI IN EVALUATION OF HYPOXIC ISCHEMIC BRAIN INJURY IN PRETERM AND FULLTERM INFANTS

Sanjay Tyagi PG Resident, Department of Radiodiagnosis, Government Medical College, Haldwani, Nainital, Uttarakhand, India.

Pankaj Mahesh* Assistant Professor& HOD, Department of Radiodiagnosis, Government Medical College, Haldwani, Nainital, Uttarakhand, India. *Corresponding Author

ABSTRACT Hypoxic ischemic brain injury remains the common cause of infant morbidity. The long term sequele includes developmental delay, hearing and visual loss, seizures and cerebral palsy. MR imaging techniques remain the mainstay of infant neuroimaging now days. The purpose of this study was to evaluate the role of MRI in evaluation of hypoxic ischemic brain injury in preterm and fullterm infants and thus aid in early diagnosis and outcome prediction in young infants.

KEYWORDS : HII-Hypoxic Ischemic Injury, MRI-Magnetic Resonance Imaging, TIWI-T1 Weighted Imaging, T2WI-T2 Weighted Imaging, DWI-Diffusion Weighted Imaging.

INTRODUCTION

Hypoxia means decreased oxygen supply and ischemia is a resultant damage to the tissues .Hypoxic Ischemic Injury (HII) refers to a combination of hypoxia, hypercarbia and metabolic acidosis. Thus any cause which will lead to lack of oxygen supply to the brain tissues will give rise to a series of events that will lead to injury to the brain tissues. In infants different types of hypoxic-ischemic brain injury can occur [1]. Brain injury can be localized or diffuse. The infant may present with seizures, hypotonia, lethargy, or motor asymmetry or the infarction may be asymptomatic. Diffuse ischemic brain injury is the result of decreased brain perfusion or hypoxemia. This may result in ischemic injury in full-term infants and in germinal matrix hemorrhage and/or white matter injury in preterm infants [2]. In infants the clinical entity is referred to as hypoxic-ischemic brain injury. In general, infants with signs of hypoxic-ischemic brain injury show fetal distress prior to delivery, have abnormal Apgar scores, require resuscitation at birth, and have neurological abnormalities within the first days of life, such as feeding difficulties, irritability, abnormality of tone, seizures, and decreased level of consciousness .

MRI has the advantage of superbly displaying soft tissue contrast differentiation and moreover displaying the exact extent and site of brain injury better than cranial ultrasonography [3]. During the first six months of life the myelination process is best visualized on T1-weighted images, whereas after six months T2-weighted images better reflect this process. Because of the high water content of the immature brain, fluid-attenuated inversion recovery (FLAIR) imaging is of less use in the first year after birth than in older children and adults[4].Currently, the following more advanced MRI techniques are available for assessing hypoxic-ischemic brain injury and its sequelae, Diffusion-weighted imaging (DWI). DWI is sensitive in showing cytotoxic edema.

AIMS AND OBJECTIVE

The aims and objective of our study were to assess the role of magnetic resonance imaging in diagnosis and to describe the hypoxic ischemic brain lesions on MR image in full term and preterm infants and to evaluate the role of MRI in predicting clinical consequences of hypoxic-ischemic lesions and to correlate the severity of asphyxia and MR images and thus aid the clinician in establishing the diagnosis of Hypoxic Ischemic Brain Injury.

MATERIALS AND METHODS

A prospective cross sectional study was conducted on 50 patients in the department of Radiodiagnosis, Government Medical College and Dr.Susheela Tiwari Government Hospital, Haldwani, Nainital, Uttarakhand. The study included the infants (of age group birth to12 months of age) referred for MRI having suffered from perinatal hypoxia. Imaging was done with Siemens 1.5 Tesla Magnatom Avanto machine using head coil with appropiate sequence included axial, sagittal and coronal T1W and T2W, FLAIR, GRE sequences.

RESULTS

In our study of 50 cases, the brain lesions which were accurately diagnosed by MRI included germinal matrix hemorrhage, periventricular leukomalacia, cystic encephalomalacia, cerebral and cerebellar atrophy, corpus callosal thinning and agenesis and delayed myelination. Thus, in diagnosis of these conditions MRI was a definitive diagnostic modality MRI was able to diagnose these conditions in 72% of the patients. However, in rest of the 28 % patients, MRI was normal, even though the patient had a history of perinatal hypoxia or was symptomatic.In our study MRI of the brain was done in 50 patients out of whom 28 were males and 22 were females as seen in the chart below. Thus, males were more commonly affected than females.In our study 18 patients had a history of preterm delivery and 32 patients were term neonates.All patients were grouped in age of 0-3 months, 3-6 months, 6-9 months, and 9-12 months. Maximum patients were in the age group of 0-3 months (34.0 %),and then 9-12 months (26.0 %) 6-9 months(22%) ,3-6 months(18.0%), age group.

There are various risk factors for HII in a preterm or full term infants. Amongst all maternal and fetal factors no specific cause was found in around 26 % of the cases. It was followed by preeclampsia (20%) , anemia(14%), Caesarean section (14%),Placental factors(10%) Perinatal infection(8%) Assisted delivery(8%).Information of gravid status, antenatal examination, hospital or home delivery was obtained of all the mothers of 50 patients.The results were primigravida (54%), multigravida(46%) , no antenatal examination (44%), undergone antenatal examination(56%) ,home deliveries (28%), hospital deliveries(72%).

Thirty two cases of term neonates who suffered perinatal hypoxia underwent MRI, out of which 23 cases were abnormal (72%). Of them, eighteen showed white matter (cortical and/or periventricular) T2 hyperintensities(56%), which was the most common abnormality found. Other abnormalities were cerebral atrophy(50%), cystic encephalomalacia(37%), delayed myelination (31%), corpus callosum thinning(21%), cerebellar atrophy(15%), acute infarcts(15%). 9 cases were normal (28%).Eighteen cases of preterm neonates who suffered perinatal hypoxia were evaluated in our study by MRI, out of which 5 were normal (27.7 %) and 13 were abnormal (72.3 %). Of the abnormal studies, the most common were periventricular leucomalacia (33%) and cerebral atrophy (33%). Other pathologies which were observed were delayed myelination (22%), corpus callosum thinning(22%), cystic encephalomalacia (22%), acute infarcts(11%) and cerebellar atrophy(5%).

In this study (n=50), maximum cases were of developmental delay (32%). The other symptom complexes with which patients presented were seizures(28%), cerebral palsy(20%), hypotonia (14%)and drowsiness(6%)Out of 36 cases of HIE findings in perinatal hypoxia, maximum number of cases showed generalized cerebral

atrophy (61%), followed by T2WI hyperintensities(50%). The other findings in preterm as well as term neonates were cerebellar atrophy(17%), cystic encephalomalacia(44%), delayed myelination (39%), corpus callosum thinning(33%), acute infarcts(19%), periventricular leukomalacia(17%%), germinal matrix hemorrhage (0.3%). Out of 50 cases of perinatal hypoxia, 10 cases presented with cerebral palsy. Detailed analysis of MRI findings in these 10 cases is done as follows. 3 (30 %) cases were preterm infants and 7 (70 %) cases were term infants. Periventricular leukomalacia was the commonest finding in preterm infants, whereas, hyperintense lesions on T2WI images was the most common findings in term infants.

DISCUSSION

We carried out a MRI study on 50 patients of perinatal hypoxia presenting with various symptom complexes. Of them, 72% (36) were abnormal and 28% (14) were normal. Our study correlates with study carried out by M.A. Rutherford et al[5]. In our study of 50 patients; 56% (28) were males and 44% (22) were females with a mean age group of 6 month (age range= 0 to 12 months). Maximum patients were of age group 0 to 3 months. The male preponderance was similar to study carried out by Azhar Munir Qureshi et al[6]; 79.6% were males and 20.4% were females. In another study done by D.J.A. Connolly[7], the age group range taken into consideration was 1 to 24 years. Of them maximum patients were between the age group of 1 to 5 years.

In our study 18 (36 %) patients were preterms and 32 (64%) had term delivery. These findings are in accordance with study performed by R Yin[8]. He carried out the study on 42 patients in whom 12 were premature (28.5%) and 30 were full-term (71. 4%). The term preponderance correlates with overall pattern of distribution of deliveries. Various risk factors for HII have been proposed. In our study, preeclampsia was commonest risk factor 20 % (10) followed by anaemia 14 % (7). The other risk factors were placental factors 10% (5), perinatal infections 8% (4), and assisted delivery 8 %(4). No risk factors were identified in 26 % (13) cases. Caesarean section was done in 14 % (7) of the mothers. In the study carried out by Azhar Munir Qureshi et al [6]on 181 patients; the most common risk factor was PIH, observed in 27.7 % of the mothers, followed by anaemia seen in 16%. Placental causes were present in 18.3%. Assisted delivery was done for 7.2 % of the patients and in 15.5% no maternal cause was found. 34.3% were delivered by Caesarean section. Thus, findings are similar to other studies on risk factors. Inclusion of antenatal examination, home delivery, hospital delivery was taken into consideration in our study. We found that around 54 % (27) cases were primigravida and the remaining 46 %(23) multigravida, Of them, around 44% (22) patients had not undergone any antenatal examination. Rest 56% (28) had undergone antenatal examination. Home delivery was done in 28% (14) of mothers, whereas hospital deliveries were done in 72% (36) of mothers. In the study done by Azhar Munir Qureshi et al[6] 52.5% of the mothers were primigravida and of them 5% were managed at home. 47.5 % cases were multigravida and of them, 8 % were home deliveries. Thus my study is in accordance with this study.

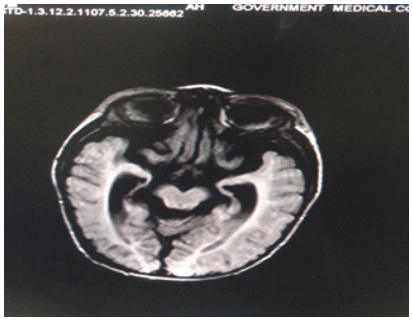
We evaluated 32 (64 %) patients with a history of term delivery. Out of which MRI pattern was normal in 28 % (9) and abnormal in 72 % (23) . Out of the abnormal cases cortical and subcortical T2 hyperintensities were noted in 56% (18) cases. Cerebral atrophy was also common and found in 50 % (16) cases. In patients presenting with acute symptoms, 15 % (5) cases showed acute infarcts. Delayed myelination was seen in 31 % (10) of the patients. Mary Rutherford[5] found cortical and subcortical T2 hyperintensities in 50 % of the patients and cerebral atrophy in 50 % of the cases. Basal ganglia infarction was observed in 18 % cases and 6 % cases showed insular infarcts. The difference in the results of our study and other studies can be because of difference in the sample size and different age distribution of patients.

We studied 18 (36%) patients who were born with the history of preterm delivery. Of these, MRI findings in 13 (72.3%) cases were found to be abnormal. 33% (6) cases showed periventricular leukomalacia and 33 % (6) patients showed cerebral atrophy as the commonest findings. Delayed myelination and corpus callosum thinning was observed in 22 % (4) cases. 27 % cases did not show any significant abnormality on MRI. Acute infarcts were seen in 11 % (2) cases. Cerebellar atrophy was present in 5 % (1) cases. Germinal matrix hemorrhage was diagnosed in 5 % (1) cases. In a study carried out by Gul Serdaroglu et al[9] ,89 children with PVL were evaluated. The aim of this study was to find out neurodevelopmental delay in children with periventricular leukomalacia (PVL). Thinning of the corpus callosum and cortical atrophy was identified respectively in 73% and 47.2% of the patients. Delayed myelination was noted in 14.3% cases. MRI was normal in 18 % of the infants. In a similar study done by Pavithra Logitharajah[10], the major sites of injury were basal ganglia (BG, 75%), white matter (89%), Cortex was involved (58%) followed by brain stem in 44% cases No abnormality was found in 32%. Significant central gray matter and brainstem injury was found in many preterm infants with HIE. Neonatal MRI findings allowed accurate prediction of neurodevelopmental outcome on follow up studies. The difference in the results of our study and other studies can be because of different age selection criteria of our study compared to other studies.

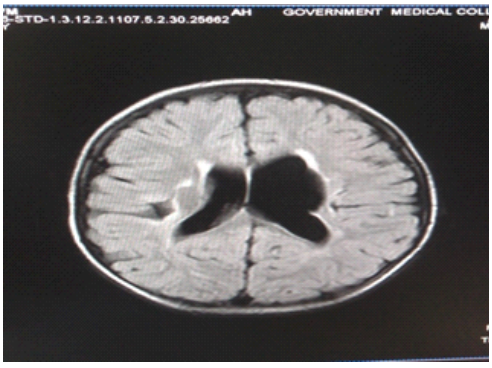
We came across various symptom complexes with which patients presented. Of them, maximum patients presented with developmental delay 32% (16). This was followed by epilepsy seen in 28 % (14) patients. Cerebral palsy was seen in 20 % (10) cases. Patients presenting with hypotonia were 14 % (7) whereas 6 % (3) cases had an acute presentation in the form of drowsiness or altered sensorium. Thus, our findings are in concordance with the study by Gul Serdaroglu et al[9]where maximum cases of developmental delay were noted followed by epilepsy 33.7% and cerebral palsy in 30.8% of the patients. Approximately 28 % patients had diplegia. Our findings were similar to those studied by R.Yin[8]. He performed a study of MRI findings in 42 patients, of which 8 were premature (38 %), 13 were full-terms (62%). Periventricular leukomalacia was seen in 66.6%. Cerebral atrophy was seen in 33.3% cases and 33.3% children did not demonstrate PVL. Charles L. Truwit[11] did a study on 40 patients with cerebral palsy. Of these, 11 were premature and 29 were term infants. Of the 11 patients born prematurely, MR revealed deep white matter loss, especially in the peritrigonal regions. 81 % of the scans demonstrated thinning of the corpus callosum.Thus, genetic or functional, rather than gross structural lesions, may underlie the pathophysiology of CP. Overall our findings corroborate with the studies.

CONCLUSION

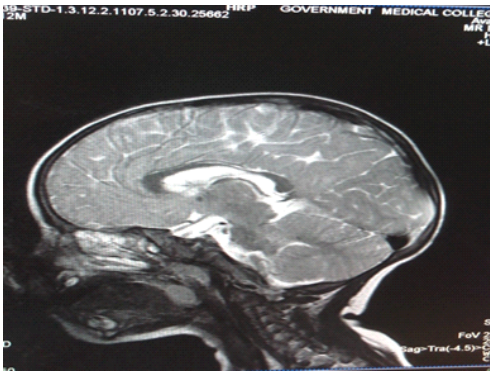
Thus the conclusion of our study is that the MRI was able to differentiate between patterns of brain injury, according to the brain maturity, severity and length of the ischemic insult. In our study, patients presenting late with HII were more than that of those presenting with acute presentation. MRI brain was normal in few patients who had suffered with perinatal hypoxia. Also, follow up MRI studies for those children who have suffered with perinatal hypoxia is extremely important to know the prognosis of the condition and thus aid clinician for better outcome.



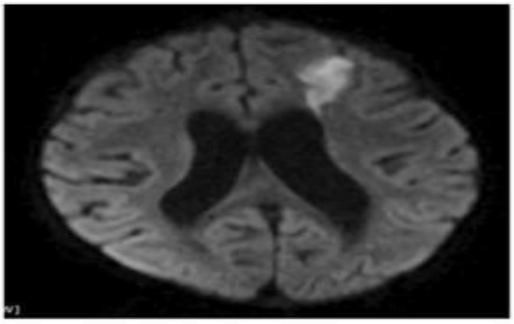
Axial FLAIR MR images showing prominence of ventricular system and cortical sulci suggestive of generalized cerebral atrophy



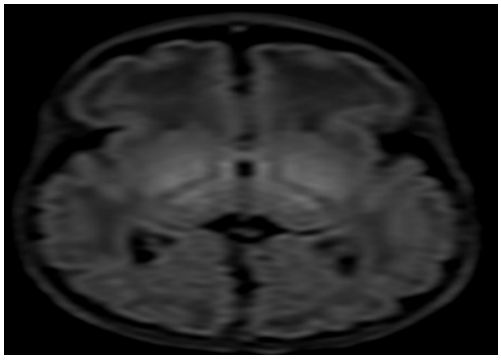
FLAIR MR image showing loss of peritrigonal whgite matter, multiple gliotic &hyperintense areas in posterior thalmi,bilateral periventricular deep & subcortical white matter causing exvacuo prominence of left lateral ventricle.



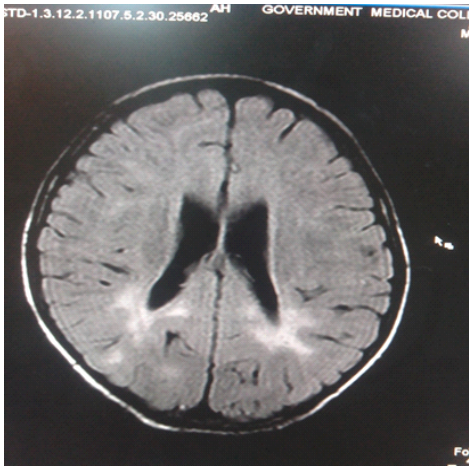
T2 Saggital image showing asymmetrical periventricular white matter hyperintensities with thinning of corpus callosum in body region.



Axial DW MR Image at the level of lateral ventricles showing focus of restricted diffusion in the left frontal periventricular white matter. This is suggestive of periventricular type of pattern of acute infarct



Preterm infant with history of perinatal asphyxia.Bilateral symmetrical areas of signal abnormality in the basal ganglia and thalami appearing hyperintense on T1W images Similar signal abnormality was also seen in the dorsal brainstem



Axial FLAIR image showing white matter hyperintensites in bilateral parieto-occipital regions.

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