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Study of Electoro Encephelography (EEG) and Magnetic Resonance Imaging (MRI) in full term Newborns with Hypoxic Ischemic Encephalopathy (HIE) in Comparison to full Term Normal Newborns

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Abstract:

Objectives: *To know the abnormalities in Electroencephalogram in full term newborns with hypoxic ischemic encephalopathy in comparison to full term normal newborns.*

To know he abnormalities in MRI of brain in full term newborns with hypoxic ischemic encephalopathy in comparison to full term normal newborns.

Material & Methods: *The Study population comprised of 100 term asphyxiated full term neonates and 100 fullterm normal neonates who were admited to the neonatal unit in Govt. General Hospital, Kakinada from Oct.2012 to Apr.'2014.*

The encephalopathy in term neonates was graded clinically into three stages based on Sarnat & Sarnat, Levene grading system EEG and MRI of brain were done in all new borns who were enrolled in the study.

Results: *In present study ot of 100 neonates HIE I-15, HIE II-80, HIE III-5 Cases EEG was normal in 94% and abnormal in 6% of HIE group abnormal EEG suggestive of burst suppreession pattern. In HIE Group MRI Brain was normal in 44% abnormal in 56% of cases, where as in controle group MRI Brain was normal 97% and abnormal in 3% neonates. In present study sensitivity and specificity of MRI Brain were found to be 94.91% and 74.04% respectively. MRI Brain is more sensitive and specific than EEG in detection of abnormalities in HIE.*

Conclusion: *MRI brain is more sensitive and specific in detection of abnormalities of HIE in neonates. Clinical staging of HIE reliability correlates with abnormal changes in MRI Brain than in EEG as structural abnormalities are seen in MRI Brain and functional intigrity known by EEG. MRI is better tool for early detection of the abnormalities of brain compared to EEG in HIE Cases.*

Key Words: *HIE (Hypoxic Ischemic Encephalopathy), EEG (Electro Encephalography), MRI (Magnetic Resonance Imaging),*

Introduction

Indian data as per NND the Incidence of both asphyxia is 14/1000 live births causing 30% neonatal and 50% Perinatal deaths and 30% still births. Manifestations of HIE were seen in approximately 1.5% of all babies. Asphyxia is defined as condition of impaired gas exchange that leads to 1) Hypoxemia. 2) Hypercapnea 3) Metabolic acidosis. Birth asphyxia-NNF (National Neonatology Forum) defined birth asphyxia as failure to establish respiration at the end of one minute with Apgar score of 0-6

AAP (American Academy of Pediatrics and ACOG (American College of Obstetricians & Gynecolotists) Defined perinatal asphyxia as:

A. Umbilical Cord arterial pH less than 7 with base deficit of > 10 meq/L.
B. Apgar score of 0-3 for longer than 5 min.
C. Neurological Manfestations suggestive of HIE.
D. Evidence of multisystem organ dysfunc-tion.

The factors indicating an increased risk for hypoxic-iscemic brain injury in infants are complex and multiple such as infecion, inflammation, extended labor or repeated asphyxia during birth. The risk factors involved could be related to

- Maternal conditions (eg.Primiparity, Chorioamnionitis, Infertility)
- Fetals condition as (Inta-Uterine Growth Restriction, Fetal Distress) and
- Neonatal conditions (Neonatal Resuscitation, Prematurity).

In term baby injury observed consists more of cortical gray matter commonly in the form of selective neuronal necrosis often seen as diffuse neuronal injury affecting the Cerebral Cortex, Basal Ganglia, Hippocampus, Brainstem, and Cerebellum. The other patterns of injury observed may be parasagittal cerebral injury or watershed infarcts reulsting in focal or Multi-Focal Ischemic injury.

Continuous EEG monitoring is possible with a Cereberal Function monitor which displays the amplitude of one or more channels of procesed EEG and modern equipment offers the opprotunity to obtained multiple channels of raw EEG and a digital video signal in addition to prepared and filtered amplitude integrated EEG. Abnoramal EEG activity such as severe amptitude depression on brust suppression, corelates well with later adverse outcome in both preterm and asphyxiated termbabies. EEG allows diagnosis of neonatal seizures and helps to determine prognosis of infants with HIE.

Magnetic Resonance Imaging [12,3,4,5] of the infant brain has given an enormous insight into the naturational proecesses that take place after birth. The technique has made it possible to see in minute details changes in cortical folding, involution of the germinal layer, pre myelination changes within white matter, myelination, iron deposition, and the growth of different regions of the brain that is no possible with computed tomography or ultrasound.

MRI Findings in the Neonate with severe, Hypoxia[6]

A. Increased Signal Intensity of Basal ganglia on T1-Weighted Images.

These are deep gray matter structure (Basal Ganglia and Thalamus) are the most metabolically active in the brain and these are more vulnerable to oxidative stress and show the effects of hyproxia.

B. Increased Signal Intensity in the Thalamus on T1-Weighted Images.

C. The "Absenent Posterior Limb Sign" This sign is due to loss of the normal increased signal intensity that is associated with myelination.

D. Finding on Diffusion The 4th imoprtant sign of severe total hypoxia is that of restricted diffusion in the basal ganglia, the posterior limb of the internal capsule, or the thalamus, manifested by bright signal on diffusion-weighted images and reduced apparent diffusion, coefficient values on apparent diffusion coefficient maps. [7,8]

MR Spectroscopy findings in full-term infants with hypoxic-ischemic injury include elevation of choline relative to creatine, decreased N-acetylaspartate (NAA), and the presence of a lactate peak [9]

Ultrasound scanning is an easily applicable bedside tool but has low sensitivity in term babies HIE. Because of higher sensitivity and specifity to maturation changes such as visualization of myelination and changes in cerebral structures MRI has had been enormous impact on neurological imaging.[10,11]

Materials and Methods

The study population comprised of 100 term asphyxiated full term neonates and 100 full term normal neonates who were admitted to the neonatal Unit in Govt. General Hospital, Kakinada. from Oct.'20012 to Apr.'2014 Moderat aspyxia as slow gaspring breathing or an Apgar score of 4-6 at minute of age. Severe asphyxia as no breathing or an Apgar score of 0-3 at 1 minute of age.[4]

Detailed history and cinical examination were done in all the included babies at admission, gestational age was assessed by New Ballard score. Those babies with congenital malforma-tions, suspected intrauterine infections and trauma were excluded. The babies were monitered round the clock for signs ans sysmptoms of encephalopathy and also for multiorgan dysfunction. The encephalopathy in term neonates was graded clinically into 3 stages based on the Sarnat & Sarnat, Levene stagng system, electro encephalogram and magnetic resonance Imaging of brain were done for all newborns who were enrolled in the study. Morbidity pattern was analysed during hospital stay.

Observations and Results

In present study, out of 100 neonates of HIE group males accounts for 57%, Females accounts for 43%. Out of 100 neonates of control group males accounts for 62% and Females accounts for 38%.

Out of 100 HIE – 15 Cases HIE – I, 80 cases HIE – II, and 5 cases were HIE – III

Destribution of New Born with HIE Based on Stage & Risk Factors

ASSOCIATED RISK FACTORS	HIE – I	HIE – II	HIE – III	TOTAL
Mecoonium Stained Liquor	2	17	2	21
Prolonged Labour	2	7	0	9
PROM	1	7	0	8
PIH	1	4	0	5
Fetal Distress	1	2	1	4
`Cord Around the Neck	0	1	0	1
Abruptio Placenta	0	1	0	1
Total	7	39	3	49

In our study, in HIE group associated risk factors noted in 49% of neonates.

Morbidity Pattern According To HIS Stages

Feature	HIE – I	HIE – II	HIE – III	Total
Meconium Aspiration Syndrome	2	17	2	21
Early Onset Sepis	3	8	1	12
Neonatal Sepsis	2	8	2	12
Hypoglycemia	1	6	1	8
Shock	0	2	0	2
Total	8	41	6	55

In present study, morbidities such as Meconium Aspiration Syndrome (MAS), Early Onset Sepsis (EOS), Neonatal Jaundice (NNJ), Hypoglycemia, Shock were more seen in HE Stage – II.

Distribution of EEG Changes in HIE and Normal Newborns

Group	Abnormal EEG	Normal EEG	Total	P Value
HIE	6	94	100	0.013
Control	0	100	100	
Total	6	194	200	
Sensitivity – 94.91%, Specificity – 74.05%				

Distribution of MRI Changes in HIE and Normal Newborns

Group	Abnormal EEG	Normal EEG	Total	P Value
HIE	56	44	100	0.000
Control	3	97	100	
Total	59	141	200	
Sensitivity – 100%, Specificity – 51.54%				

Distribution of MRI Brain Changes According to HIE Stages

Stage	Normal MRI	Abnormal MRI	Total	P Value
HIE – I	14	1	15	0.000
HIE – II	30	50	80	
HIE – III	0	5	5	
Total	44	56	100	

Distribution of Abnormal MIR Brain Findins According to HIE Stages

Feature	HIE – I	HIE – II	HIE – III	Total
White Matter	0	23 (41%)	1 (1.7%)	24
Bg & Thalamus	0	7 (12.5%)	2 (3.5%)	9
Cortical Infacts	0	6 (10.7%)	1 (1.7%)	7
Internal Capsule	0	4 (7.1%)	0	4
Periventricular Perirolandic	0	4 (7.1%)	0	4
Hemorrhage	0	3 (5.3%)	1 (1.7%)	4
Theombosis	0	3 (5.3%)	0	3
Partial Coampus Callosal Agenesis	1	0	0	1
Total	1	0	5	56

Comparision of MRI Brain & Eeg in HIE

MRI Findings	Normal EEG	Abnormal EEG	Total
Normal	42	2	44
White Matter	22	2	24
Bg & Thalamus	9	0	9
Cortical Infarct	6	1	7
Internal Capsule	3	1	4
Periventricular Perirolandic Area	4	0	4
Hemorrhage	4	0	4
Thrombosis	3	0	3
Partial Corpus Callosal Agenesis	1	0	1
Total	94	6	100

Discussion

In present study, out of 100 HIE neonats, Males accounts for 57%, Females accounts for 43%, Similar to Tanzania study in which males and females were 62% and 38% respectively. In Egypt and Tamilnadu studies Males accounts for 72% and 77%, Females accounts for 28% and 23% respectively.

In our study, 15% neonates belongs to HIE stage - I 80% to HIE stage – II and 5% to HIE stage – III, simlar to Egypt study in which 12% were in HIE stage – I 76% in HIE stage – II and 12% were in HIE stage – III.

In Tamilnadu study 29% were in Stage – I - 42% were in stage – II and 29% were in HIE stage III. In Tanzania study, HIE – I neonates were more i.e. 70% where as 18% and 12% were in HIE stage – II and III respectively.

In present study, 94% neonates showed normal EEG in HIE groups and 6% of HIE neonoates showed EEG abormality, which is different from Auoty and Jose studies probably because of not including the discontinuity patterns. Ternsient discharges in EEG as abnormalities as aken in Auty and Jose studies.

In Auoty study, 28% of neonates with HIE has normal EEG and 2% had abnormal EEG. In jose study EEG was normal in 20% and abnormal in 80% of cases.

In present study, MRI brain abnomalities were noted in 56% of neonates with HIE, where as in Auoty study 88% neonates showed abnormalities in MRI brain, in Jose study MRI ws abnormal in 39% neonates and in Miller study MRI brain was abnormal in 70% of cases.

In our study MRI Brain was normal in 44% of the cases. 61% and 12% of the cases showed normal MRI brain in Jose and Auoty studies respectively. In Miller study MRI brain was normal in 30% of cases.

In our study 42% of HIE neonates showed abnormality in white matter of brain, smimilar of Mller study in which white matter abnormalities were noted in 45% of the cases. In Auoty study 36% of HIE neonates showed abnormality in white matter. In Jose study 23.1% showed abnomality in white matter.

In present study, basal ganglia and thalamus abnormalities were noted in 16% of the cases of HIE, which is different from other studies as 25% of neonates which HIE in Miller study 44% in Auoty study and 7.7% in Jose study showed abnormalities in basal ganglia and thalamus.

Conclusion

MRI Brain is more sensitive and specific in detection of abnormalities of HIE in neonates.

Clinical staging of HIE reliabley correlates wih abnormal changes in MRI brain than in EEG as structural abnormalities are seen in MRI brain and functional integrity known by EEG.

MRI is a better tool for early detection of the abnormaliies of brain compared to EEG in HIE cases.

In new borns with HIE where clinical screening and EEG are normal, MRI brain will give clue to actively follow such abnormal cass fo developmenal screeing.

In 2 New Borns who were clinically normal and MRI brain was abnormal there was bad obstetric history.

White matter abnormalities on MRI brain were more common in term newborns with HIE.

In our study we have found periventricular abnormalities in 7% of the term neonates with HIE.

Stage – II Hypoxic Ischemic Encephalopathy was more commonly seen in male newborns.

Follow up studies in the newborns with abnormal findings on MRI brain may reveal the neurodevelopmental outcome, so that we can associate certain abnormal MRI brain findings with outcome.

References

1. Jyoti R, O'Neil R, Hurrion E, Pediicting outcome in term neonates with hypoxic Ischemic encephalapathy using simplified MR Criteria, Pediatr Radiol 06: 36: 38-42.
2. Rutherford MA, Pennock JM, Counsell SJ et al. Abnormal Manetic Responnance Signal in the Internal Capsule predicts poor neurodevelopment outcome in infancts with hypoxic ischemic encphalopaty Pediatrics 1998: 102:323-8.
3. Liauw L, van der Ground J, van dem Berg – Huysmans AA, Pal Menders IH, van buchem MA, van Wezel-Mijier G, Hypoxic Ischemic Encephalopathy: Diagnostic vale of conventional MR Imaging pulse sequences in termbour neonates. Radiology 2008: 247: 204-12.
4. Chau V, Poskitt KJ, Sargent MA, et al. Pediatrics 2009: 123 : 319-26.
5. Barkovich AJ, Hajnal BL, V igneron D et al. AJNR 1998: 19: 143-9.
6. Liaw L, van der Ground J, Van den Berg-Huysmans AA, Laan LA, van Buchem MA, van Wezel-Meijler G. AJR 2008: 29 : 1789-94.
7. Magnetic Resonance Imaging of brain in normal infant brain, Francis M cowan.
8. American Joural of Roentgenology 2009: 192: 41-47, 10.2214/AJR.08.1321.
9. Robertson Rl, Liat B, Barnes PT, et al, MR linescan diffusion-weigted imaging of term neonates with perinatal brain ishemia, Am J Neuroradiol 1999: 20 : 1658-1670.
10. Provenzale JM, Sorenson AG, Diffusion-Weighted MR in acute stroke: Theoretic considerations and clinical applications, AJR 1999: 173: 1459-1467.
11. Barkovich AJ, Miller SP, Bartha A, et al. MR imaging , MR spectroscopy, and diffusion tensor imaging of sequential studies i neonates with encephalopathy. Am J Neuroradiol 2006: 27: 533-547.
12. Keeney 5, Adcock EW, Mc Ardle CB. Prospective observations of 100 high-risk neonates by high field (1-5 Testa).
13. Martin E, Boesch C, Zuerrer M, Kikinis R, Molinari L, Kaelin P, et al. MR Imaging of brain maturation in noraml ad developmentally handicapped childern. Jf Comput Assist Tmogr 1990 14:685-92.
14. Natinal Perinatal Neonatal Database. Available for URL, www.newbornwhocc.org/pdf/npd_report_ 2002-03. PDF: Accessed Feb.' 23/2014.
15. Birth asphyxia in developing countries: Current status and public health implications Haider a Bhutta, dol: 10.1016/ J. Cppeds. 2005:11.002.