International Journal of Research in Medical Sciences
Patil B et al. Int J Res Med Sci. 2015 Mar;3(3):560-567
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012

DOI: 10.5455/2320-6012.ijrms20150306

Research Article

Clinicoradiological correlation in birth asphyxia

Basavaraj Patil*, Sandeep Harshangi, Bhagya Prabhu

Department of Paediatrics, M.R. Medical College, Gulbarga, Karnataka, India

Received: 14 December 2014
Revised: 22 January 2015
Accepted: 8 February 2015

***Correspondence:**
Dr. Basavaraj Patil,
E-mail: dr.basavarajpatil@gmail.com

ABSTRACT

Background: Hypoxic Ischemic Encephalopathy (HIE) is the most dreaded neurological disease of the new-born. Assessment of severity of HIE would help proper parent counseling and early institution of stimulation therapy for better development of the infant.
Methods: This study was conducted between December 2012 and May 2014. 37 term neonates with perinatal asphyxia were the subjects. The cranial ultrasound, EEG and MRI findings of these babies are analysed and correlated with each other and with clinical staging and the neurological condition of the babies at discharge.
Results: Among the 37 neonates, 21 were of HIE stage 2 and 16 were of stage 3. Sensitivity of EEG in detecting abnormality in the neurological condition according to our study is 76.9%, specificity 87.5%, positive predictive value 76.9%, negative predictive value 87.5%. Sensitivity of severe pattern of injury in MRI brain in detecting abnormality in neurological condition according to our study is 76.9%, specificity 91.6%, positive predictive value 83.3%, negative predictive value 88%. Involvement of both basal ganglia and cortex in MRI brain had statistically significant correlation with abnormal neurological condition at discharge in our study (P = 0.04).
Conclusion: An abnormal EEG and MRI brain in a term new-born with Hypoxic Ischemic Encephalopathy (HIE) is associated with poor neurological outcome. Involvement of basal ganglia/thalamus and cortex together in the MRI are predictors of abnormal outcome.

Keywords: Hypoxic ischemic encephalopathy, Electroencephalogram, Magnetic resonance imaging brain, Cranial ultrasound

INTRODUCTION

Hypoxic-Ischaemic Encephalopathy (HIE) in a full-term infant is a clinically defined syndrome of the disturbed neurologic function in the earliest days after birth in infancy, manifested by difficulty with initiating and maintaining respiration, the depression of the muscle tone and reflexes, the subnormal level of consciousness and often seizures.[1] HIE is the term used to describe the resultant condition due to a deficit in the oxygen supply to the brain.

The neonatal brain can have a lack of oxygen through two major pathogenetic mechanisms, hypoxemia, which is a diminished amount of oxygen in the blood supply, and ischemia, which is a diminished amount of blood perfusing the brain. During the perinatal period hypoxemia or ischemia or both occur as a result of asphyxia, an impairment in the exchange of respiratory gases, oxygen and carbon dioxide. The duration and severity of HIE depend of the severity of asphyxia. Neonatal asphyxial encephalopathy occurs in 1-2 per 1000 births in the high income countries,[2-4] in low-income countries, the incidence is much higher.[5,6]

Despite major advances in obstetric and neonatal care, and the introduction of therapeutic hypothermia, HIE is still a devastating occurrence that results in death or disability in 47% of infants with moderate or severe HIE.[7] As survivors of severe HIE have profound long-term neurologic disability like cerebral palsy, mental retardation and epilepsy[8,9] and a large majority of infants with moderate HIE have cognitive problems, sequelae of hypoxic-ischaemic brain injury require significant resources.[10] Published data show that 25-60% of the babies who survive, suffer from permanent neurodevelopmental handicaps including cerebral palsy, seizures, mental retardation, and learning disabilities. Assessment of severity of HIE would help proper parent counseling and early institution of stimulation therapy for better development of the infant.

In this study, an attempt has been made to associate the electroencephalogram (EEG), USG Cranium and MRI brain done in the newborn with the clinical staging and neurological condition at discharge in term asphyxiated newborn.

Failure to initiate or sustain respiration after birth has been defined as criteria for the diagnosis of perinatal asphyxia as per WHO. Perinatal asphyxia results in hypoxic injury to various organs including kidney, liver and lungs but the most serious effects seen on central nervous system.

Hypoxic ischemic encephalopathy in a full term infant is clinically defined syndrome of the disturbed neurologic function in the earliest days after birth in infancy manifested by difficulty with initiating and maintaining respiration, depression of muscle tone and reflexes, the subnormal level of consciousness and often seizures.

Sarnat and sarnat classified HIE into 3 stages. HIE is of foremost concern in an asphyxiated neonate because of its potential to cause serious long term neuromotor sequelae among survivors.

Despite improvements in perinatal care in the developed countries asphyxia remains a major cause of mortality resulting upto 25% of perinatal mortality and morbidity and giving rise to 8-15% of all cases of cerebral palsy.

In particular, infants with severe (Sarnat stage 3)[11] encephalopathy are generally believed to have uniformly dire prognosis.[12] However, prognostication in infants with moderate encephalopathy is more difficult.[12-14] A variety of clinical, electrophysiological, and radiologic tools have been used to help prognosticate.[9] In particular, MRI has emerged as potentially one of the most useful tools for prognostication in HIE [15-17] and it has been recommended that all infants with HIE have an MRI performed between days 2 and 8 days.

Cranial ultrasound has advantage of being mobile and easily used on the neonatal unit. But it has low sensitivity in detecting brain abnormalities.

Sonography is often used for assessment of the neonate with suspected hypoxic-ischemic encephalopathy because the equipment is portable, it is inexpensive, and it does not subject the infant to a magnetic field or radiation. Findings include increased echogenicity in white matter and resultant increased gray matter-white matter differentiation. These findings, which can be focal or diffuse, are thought to reflect edema or necrosis and correspond to abnormalities on MRI. In more severely affected neonates, the cortex may become thickened and the distinction between gray matter and white matter less apparent. Abnormalities at this stage typically become more diffuse, are often asymmetric between hemispheres, and are associated with the loss of landmarks such as sulci and major fissures.[19]

Despite the advantages of sonography, limitations have been noted with regard to detection of lesions, as well as its dependence on the experience of the individual interpreting the study and its interobserver variability. In a study of 47 neonates, of whom two thirds were at term or near term, sonography was compared with MRI and CT for the detection of abnormalities and interobserver agreement.[20] CT and MRI had substantially greater sensitivity for the detection of cortical hypoxic-ischemic injury and markedly lower interobserver variability than sonography. Doppler sonography is not routinely used for assessment of neonatal hypoxia at many institutions. However, when used, this technique may identify a decrease in refractive indexes in arteries due to a relative increase in diastolic flow velocities.

A number of other limitations of sonography for the diagnosis of hypoxic-ischemic injury in the term neonate have been identified. These include that a substantial percentage of sonographic examinations are false-negative studies, its low sensitivity for detecting cortical lesions, and dependence on the skill of the operator performing the test.[21]

The most sensitive and specific imaging technique for examining infants with suspected hypoxic- ischemic brain injury is MR imaging.[21] Cranial USG and MRI show anatomical abnormalities in brain due to birth asphyxia whereas EEG can show early abnormal cerebral function.[22]

MRI - brain injury patterns are determined by nature of insult: chronic or acute. It is important to distinguish two forms of hypoxia: severe, total hypoxia and prolonged, partial hypoxia. The term “severe hypoxia” refers to sudden and total loss of oxygenation. A typical example would be that seen in abruptio placenta. The term “prolonged, partial hypoxia” refers to a more sustained, but incomplete, loss of oxygenation, such as that seen in prolonged difficult labor with prolonged decelerations,

repetitive late decelerations, and decreased heart rate. Although these two entities are discussed as distinct phenomena, the fact is that in some cases, overlap occurs between the two; an infant may be seen as having features of both types of hypoxic injury. Only a minority (10%) of HIE cases have a sentinel event.[23]

MRI Findings in the neonate with severe, total hypoxia - four major signs that occur in proximity to one another and which are collectively referred to as the "1-2-3-4 sign" as a means of facilitating the diagnosis of hypoxia in the term neonate.[19]

The four components of the 1-2-3-4 sign are

1. Increased signal intensity in the basal ganglia on T1-weighted images

2. Increased signal intensity in the thalamus on T1-weighted images,

3. Absent or decreased signal intensity in the posterior limb of the internal capsule on T1-weighted images (i.e., the "absent posterior limb sign"), and

4. Restricted water diffusion on diffusion-weighted images.

MRI findings in prolonged, partial hypoxia

Neonates with prolonged, partial hypoxia tend to have normal basal ganglia signal intensity on T1-weighted images but often exhibit more marked cortical signal changes, even though they have a less profound acute hypoxic injury.

Imaging appearances are influenced by

a. Sequences used.

b. Time of imaging from injury.

After imaging, the second most important role of a pediatric radiologist in the work up of infants with HIE is to predict, if possible, the clinical outcome based on imaging findings at an early stage. This is a crucial but difficult situation.

In infants with HIE, characteristic increase in the echogenicity of thalamus and basal ganglia in USG cranium been reported, usually in the second week after the episode of asphyxia. Visualisation of this sign predicts poor prognosis and has been reported to be associated with mortality in 1% and long term morbidity in 56%.

Delayed myelination in a term neonate in the first few days of life seen in MRI brain suggests significant perinatal asphyxia and hence poor prognosis.

Abnormal signal intensity within PLIC has been found to be an excellent predictor of abnormal motor outcome. Asymmetrical involvement predicts hemiplegia and bilateral symmetrical involvement correlates with subsequent development of spastic diplegia or quadriplegia.

The severity of basal ganglia and thalamic lesions detected on MRI also has good correlate with severity and nature of cerebral palsy. The involvement of white matter exacerbates cognitive impairment and poor head growth.

METHODS

This prospective cohort study has been done in the department of pediatrics, M.R. medical college, Gulbarga.

37 term neonates admitted in the NICU of Basaveshwar Teaching and general hospital and Sangameshwar hospital with history of birth asphyxia and clinical findings suggestive of birth asphyxia HIE stage 2 and 3 are included in this study.

Inclusion criteria

- Term babies
- HIE stage 2 and 3

Exclusion criteria

- Associated surgical anomalies
- Associated metabolic disorders
- Preterm babies
- Associated hyperbilirubinemia leading to kernicterus.

Baseline characteristics like sex, gestational age, birth weight, maternal history with risk factors, mode of delivery, place of delivery are noted. Detailed history has been taken and detailed examination was done. The neonates are graded into HIE stages using sarnat and sarnat staging.

Cranial USG was done after 7 days or once the baby is stable unless there is an emergency to rule out any intracranial hemorrhage where bedside ultrasonography is done.

MRI Brain was performed in all babies between 14 days and 30 days of postnatal life.

Magnetic Resonance Imaging (MRI) was done in all the babies by using Siemens Avantom 1.5 Tesla machine using multichannel head coils under sedation. The MR sequences that were employed were flair, T2, T1, diffusion, susceptibility weighted, and inversion recording imaging. The MRI findings were classified according to basal ganglia/watershed pattern as described in a study by Barkovich et al.[24] Normal was classified as score 0, abnormal signal in basal ganglia/thalamus as score 1, abnormal signal in cortex as score 2, abnormal signal in areas of cortex and basal nuclei as score 3, and abnormal signal in entire cortex and basal nuclei as score 4.

EEG was done on 14 ± 2 days. The EEG were reported as either normal or had epileptiform activity.

The cranial ultrasound, EEG and MRI findings of these babies are analysed and correlated with each other and with clinical staging and the neurological condition of the babies at discharge.

The neurological condition of the babies at discharge was considered as abnormal if there was abnormality in mental status/difficulty in sucking/abnormality in tone.

Descriptive statistical analysis was carried out. Results on categorical measurements are presented in number (%). Significance is assessed at 5% level of significance. Chi-square/Fisher exact test was used to find the significance of study parameters on categorical scale between two or more groups. Diagnostic statistics viz. sensitivity, specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV), were computed to find the correlation of EEG, USG cranium and MRI brain with abnormal neurological condition at discharge.

RESULTS

Among the 37 cases included in the study, 22 were males, 15 were females. Risk factors were found during pregnancy in 29 cases. 9 babies were delivered by LSCS, 25 were delivered by NVD, and 3 by assisted vaginal delivery. 13 cases were intramural and 24 were extramural. 21 cases were of HIE stage 2 and 16 cases were of stage 3. Convulsions were seen in all except 3 cases. Mechanical ventilation was required in 16 cases (Table 1).

Cranial USG detected abnormalities in only 4 cases. EEG showed seizure activity in 13 cases (Table 2).

MRI brain showed changes in white matter in 19 cases, basal ganglia in 10 cases, thalamus in 2 cases, internal capsule in 6 cases, brain stem in 2 cases and grey matter in 7 cases. Pattern of severe brain injury was found in MRI in 12 cases, less severe changes found in 13 cases and MRI brain did not show any changes in 12 cases (Table 3).

Table 1: Baseline variables.

	Number	Percentage
Males	22	59%
Females	15	41%
Risk factors present	29	78%
NVD	25	68%
LSCS	9	24%
Assisted vaginal delivery	3	8%
Intramural	13	35%
Extramural	24	65%
HIE stage 2	21	57%
HIE stage 3	16	43%
Convulsions	34	92%
Mechanical ventilation	16	43%

Table 2: Percentage of brain abnormalities detected by cranial USG, EEG and MRI Brain in HIE 2 and 3.

	HIE 2 (N=21)	HIE 3 (N=16)
Abnormalities in USG	1 (4.7%)	3 (18.7%)
Seizure activity in EEG	5 (23.8%)	8 (50%)
Abnormalities in MRI	11(52.3%)	14 (87.5%)
Severe pattern in MRI	3 (14.2%)	9 (56.2%)

Table 3: Different areas of brain affected in MRI brain.

Areas of brain involved	Number N=37	Percentage
White matter	19	51%
Basal ganglia	10	27%
Thalamus	2	5%
Internal capsule	6	16%
Brain stem	2	5%
Grey matter	7	19%

Sensitivity of MRI brain in detecting abnormality in the neurological condition according to our study is 92.3%, specificity 45.8%, positive predictive value 48%, negative predictive value 91.6% (Table 4).

Table 4: Correlation of MRI brain with neurological condition at discharge.

Neurological condition at discharge	Abnormality in MRI present	Abnormality in MRI absent	Total
Abnormal	12	1	13
Normal	13	11	24
Total	25	12	37

Sensitivity of severe pattern of injury in MRI brain in detecting abnormality in neurological condition according to our study is 76.9%, specificity 91.6%, positive predictive value 83.3%, negative predictive value 88% (Table 5).

Table 5: Correlation of severe pattern of injury in MRI brain with neurological condition at discharge.

Neurological Condition at discharge	Severe pattern of injury in MRI	Less severe pattern of injury or normal in MRI	Total
Abnormal	10	3	13
Normal	2	22	24
Total	12	25	37

Sensitivity of Cranial USG in detecting abnormality in the neurological condition according to our study is 15.3%, specificity 91.6%, positive predictive value 50%, negative predictive value 66.6% (Table 6).

Table 6: Correlation of cranial USG with neurological condition at discharge.

Neurological Condition at discharge	Abnormalities in cranial USG +	Abnormalities in cranial USG -	Total
Abnormal	2	11	13
Normal	2	22	24
Total	4	33	37

Table 7: Correlation of EEG with neurological condition at discharge.

Neurological condition at discharge	Seizure activity present	Seizure activity absent	Total
Abnormal	10	3	13
Normal	3	21	24
Total	13	24	37

Table 8: Sensitivity, specificity, positive predictive value and negative predictive values of different modalities of imaging and EEG.

	MRI brain	EEG + MRI	Severe changes in MRI	Cranial USG	EEG
Sensitivity	92.3%	69.2%	76.9%	10%	76.9%
Specificity	45.8%	91.6%	91.6%	88%	87.5%
PPV	48%	81.8%	83.3%	25%	76.9%
NPV	91.6%	84.6%	88%	72%	87.5%

Sensitivity of EEG in detecting abnormality in the neurological condition according to our study is 76.9%, specificity 87.5%, positive predictive value 76.9%, negative predictive value 87.5% (Table 7).

Table 9: Correlation of both MRI brain and EEG with Neurological condition at discharge.

Neurological condition at discharge	MRI + EEG abnormal	MRI/EEG/both normal	Total
Abnormal	9	4	13
Normal	2	22	24
Total	11	26	37

Sensitivity of EEG and MRI together in detecting abnormality in the neurological condition according to our study is 69.2%, specificity 91.6%, positive predictive value 81.8%, negative predictive value 84.6% (Table 9).

Involvement of both basal ganglia and cortex in MRI brain had statistically significant correlation with abnormal neurological condition at discharge in our study (Table 10c).

Table 10a: Correlation of involvement of basal ganglia/thalamus in MRI brain with Neurological condition at discharge.

	Neurological condition at discharge		Total
	Abnormal	Normal	
Only basal ganglia / thalamus involved	6	4	10
Basal ganglia / thalamus not involved	7	20	27
Total	13	24	37

P = 0.1178 (not significant) Fisher exact test

Table 10b: Correlation of involvement of cortex in MRI brain with Neurological condition at discharge.

	Neurological condition at discharge		Total
	Abnormal	Normal	
Cortex involved	10	10	20
Cortex not involved	3	14	17
Total	13	24	37

P = 0.0823 (not significant) Fisher exact test

Table 10c: Correlation of involvement of both basal ganglia and cortex in MRI brain with Neurological condition at discharge.

	Neurological condition at discharge		Total
	Abnormal	Normal	
Basal ganglia + cortex involved	4	1	5
Basal ganglia + cortex not involved	9	23	32
Total	13	24	37

P = 0.04 (statistically significant) Fisher exact test

DISCUSSION

This study attempted to associate EEG and neuroimaging like USG cranium and MRI with the clinical staging in term babies with HIE. Out of the 37 cases, 24 had normal EEG pattern and 21 of them had a normal neurological condition at discharge (87.5%). This is similar to the finding by El-Ayouty et al., who reported that normal EEG background activity was associated with normal neurological outcome at 12 months of age.[25]

In detecting an abnormal neurological state, EEG has a sensitivity of 76.9%, specificity of 87.5%, PPV of 76.9%, NPV of 87.5%, and P value of 0.0002. Ong et al., has reported that in asphyxiated newborns, EEG has a PPV of 100%, NPV of 80.6%, and sensitivity of 53%, and specificity of 100%.[26] El-Ayouty et al., have reported that for the prediction of poor outcomes, abnormal EEG background activity had a sensitivity, specificity, PPV, and NPV of 100%.[27] Early EEG (within 7 days of life) done on 77 asphyxiated infants in a study done by Caravale et al., showed that out of the 52 who had normal EEG, 83% had a normal outcome at 1 year, 17% had mild abnormalities and none of them had any severe abnormalities.[28] Presslan et al., have documented the usefulness of serial EEG in the newborn period in predicting the neurological outcome.[29] The NPV of a normal EEG was emphasized by Murray et al., who concluded that a normal or mildly abnormal EEG within 6 h of life was associated with normal neurodevelopmental outcome at 24 months.[30] Our study has shown a 87.5% NPV for a normal EEG.

Imaging studies are usually done in all neonates with HIE. MRI is difficult to perform during the acute stage, since it takes almost an hour and needs deep sedation which is risky in asphyxiated babies. USG is easier to perform and helps in practical management by detecting intracranial hemorrhage.

In our study, abnormal sonographic findings were detected in only 4 patients. The sensitivity of USG is 15.3%, specificity is 91.6%, PPV is 50% and NPV is 66.6% in our study. There is no significant association found between ultrasonographic changes and abnormal neurological condition at discharge.This is similar to the study done by Ezgu et al.[31] who found that ultrasonographic findings did not seem to predict the grade of encephalopathy or the outcome. Cranial ultrasound is one of the first used methods that show the ischemic areas as hyperechoic; but, this finding is not visible within the first 24 h of life. The sensitivity of this method was calculated as 30.5%.

Among the 37 cases 12 (32.4%) had normal MRI, 10 (27%) showed abnormal signal in the basal ganglia/thalamus, and 20 (54%) showed abnormal signal in the cortex. 5 (13.5%) cases showed abnormal signal in the cortex and the basal ganglia. Of the 12 infants with normal MRI, 11 had a normal neurological condition at discharge. El-Ayouty et al., in their study of 25 newborns have reported that all infants with a normal MRI in the first 4 weeks were neurologically normal at 12 months of age.[25] Among 5 cases which had shown abnormal signals in the cortex and thalamus 4 had an abnormal outcome (P = 0.04). MRI brain has a sensitivity of 92.3%, specificity of 45.8%, PPV of 48%, NPV of 91.6%, in detecting abnormal neurological condition at discharge (P = 0.02). If only severe pattern of injury in MRI brain is taken into consideration sensitivity of 76.9%, specificity of 91.6%, PPV of 83.3% and NPV of 88% is found in our study. P = 0.0001, extremely significant association is found in cases with severe pattern of injury in MRI brain with abnormal neurological condition at discharge. El-Ayouty et al., have reported that abnormal MRI scans had a sensitivity of 100%, specificity of 43%, PPV of 82%, and NPV of 100% in predicting an abnormal outcome at 18 months.[27]

Many studies have found the basal ganglia watershed score to be an excellent predictor of the neurological outcome.[24] Studies based on the topographic pattern of neuronal injury have shown that term infants with predominant injury to basal ganglia and thalamus have an unfavorable neurological outcome.[32] Rutherford et al., compared MRI and cranial ultrasonographic findings with the outcome at 1 year of age and found a poor outcome if both of the investigations showed a basal ganglia or thalamic lesion.[32] Mercuri et al., have reported that discrete lesions in basal ganglia were associated with normal motor outcome at 1 year of age in 57% cases, but Barnett et al., followed up seven cases of asphyxia with such lesion till school age and reported that only one out of seven had a completely normal motor outcome.[33,34] Kaufman et al., have shown that more basal ganglia involvement in MRI correlates with more severe encephalopathy.[35] In our study, it was seen that infants with lesions in both the cortex and basal ganglia and/or thalamus were significantly associated with an abnormal neurological condition at discharge (P = 0.04), which is consistent with the findings of other studies.

An attempt was made to see if EEG in addition to an imaging study improves the predictive ability. EEG along with MRI (P = 0.0002) detects abnormal neurological

state with the highest statistical significance. Biagioni et al., have reported very good correlation between EEG and MRI findings in neonatal encephalopathy and affirmed their value in predicting the neurological outcome.[36]

Our study shows that in a term newborn with HIE, a normal EEG and/or MRI of brain during the neonatal period is associated with normal neurological condition. Involvement of the basal ganglia/thalamus and cortex in the MRI also detected abnormal neurological state. EEG combined with an MRI once the patient is stable is most useful in detecting the neurological abnormality at discharge. Similar studies using larger numbers of patients and follow-up for longer duration are needed to confirm the findings of our study.

Limitations of the study are small sample size, neurodevelopmental follow up not being included in the study. There is no blinding done with respect to pediatrician who is examining the baby or radiologist who has reported the USG cranium or MRI brain or neurologist who reported the EEG. EEG is reported only based on the presence or absence of the epileptiform activity. Specific patterns of EEG in HIE are not commented in the study due to the lack of facilities.

CONCLUSION

MR imaging and EEG has significant advantages over sonography for the diagnosis of abnormal neurological state in new born with HIE. Nevertheless, early screening for infants at risk for future neurodevelopmental delays for more effective intervention remains problematic. New neuroimaging technologies that examine both physiologic and biochemical abnormalities may provide more insight into the complexity of the pathophysiology of HII and may allow for earlier diagnosis and therapeutic intervention in neonates suffering from HII. Prompt recognition of these imaging findings can help exclude other causes of encephalopathy affect prognosis and facilitate earlier (although mostly supportive) treatment. Early radiological detection of brain injuries very important in detecting the neurological abnormalities in babies with HIE.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Nelson KB, Leviton A. How much of neonatal encephalopathy is due to birth asphyxia? Am J Dis Child. 1991;145:132-31.
2. Marlow N, Budge H. Prevalence, causes, and outcome at 2 years of age of new-born encephalopathy. Arch Dis Child Fetal Neonatal Ed. 2005;90:F193-4.
3. Pierrat V, Haouari N, Liska A, Thomas D, Subtil D, Truffert P. Prevalence, causes, and outcome at 2 years of age of new-born encephalopathy: population based study. Arch Dis Child Fetal Neonatal Ed. 2005;90:F257-61.
4. Barkovich AJ. Brain and spine injuries in infancy and childhood. In: Barkovich AJ, eds. Pediatric Neuroimaging: 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2005: 190-290.
5. Ellis M, Manandhar DS, Wyatt J, Bolam AJ, Costello AM. Stillbirths and neonatal encephalopathy in Kathmandu, Nepal: an estimate of the contribution of birth asphyxia to perinatal mortality in a low-income urban population. Paediatr Perinat Epidemiol. 2000;14:39-52.
6. Black RE, Cousens S, Johnson HL, Lawn JE, Rudan I, Bassani DG, et al. Global, regional, and national causes of child mortality in 2008: a systematic analysis. Lancet. 2010;375:1969-87.
7. Edwards AD, Brocklehurst P, Gunn AJ, Halliday H, Juszczak E, Levene M, et al. Neurological outcomes at 18 months of age after moderate hypothermia for perinatal hypoxic-ischaemic encephalopathy: synthesis and meta-analysis of trial data. BMJ. 2010;340:c363.
8. Finer NN, Robertson CM, Richards RT, Pinnell LE, Peters KL. Hypoxic-ischemic encephalopathy in term neonates: perinatal factors and outcome. J Pediatr. 1992;98:112-7.
9. Perlman JM. Summary proceedings from the neurology group on hypoxic-ischemic encephalopathy. Pediatrics. 2006;117:S28-33.
10. Lindström K, Hallberg B, Blennow M, Wolff K, Fernell E, Westgren M. Moderate neonatal encephalopathy: pre- and perinatal risk factors and long-term outcome. Acta Obstet Gynecol Scand. 2008;87:503-9.
11. Sarnat H, Sarnat M. Neonatal encephalopathy following fetal distress: a clinical and electroencephalographic study. Arch Neurol. 1976;33(10):696-705.
12. Shevell MI, Majnemer A, Miller SP. Neonatal neurologic prognostication: the asphyxiated term newborn. Pediatr Neurol. 1999;21(5):776-84.
13. Thayyil S, Chandrasekaran M, Taylor A, Bainbridge A, Ernest B. Cady, W. K. Kling Chong, et al. Cerebral magnetic resonance biomarkers for predicting neurodevelopmental outcome following neonatal encephalopathy: a meta-analysis. Pediatrics. 2010;125(2):e382-95.
14. Liauw L, van der Grond J, van den Berg- Huysmans AA, Laan LA, van Buchem MA, van Wezel-Meijler G. Is there a way to predict outcome in (near) term neonates with hypoxic-ischemic encephalopathy based on MR imaging? AJNR Am J Neuroradiol. 2008;29(9):1789-94.
15. Robertson NJ, Wyatt JS. The magnetic resonance revolution in brain imaging: impact on neonatal intensive care. Arch Dis Child Fetal Neonatal Ed. 2004;89(3):F193-7.

16. Rutherford M, Srinivasan L, Dyet L, Ward P, Allsop J, Counsell S, et al. Magnetic resonance imaging in perinatal brain injury: clinical presentation, lesions and outcome. Pediatr Radiol. 2006;36(7):582-92.
17. Barkovich AJ, Miller SP, Bartha A, Newton N, Hamrick SE, Mukherjee P, et al. MR imaging, MR spectroscopy, and diffusion tensor imaging of sequential studies in neonates with encephalopathy. AJNR Am J Neuroradiol. 2006;27(3):533-47.
18. F. Ralph Heinz, James M. Provenzale. A study by Imaging Findings in neonatal hypoxia; a practical review. AJR Am J Roentgenol. 2009;192:41-7.
19. Daneman A, Epelman M, Blaser S, Jarrin JR. Imaging of the brain in full-term neonates: does sonography still play a role? Pediatr Radiol. 2006;36:636-46.
20. Blankenberg FG, Loh NN, Bracci P, D'Arceuil HE, Rhine WD, Norbash AM, et al. Sonography CT, and MR imaging: a prospective comparison of neonates with suspected intracranial ischemia and hemorrhage. AJNR Am J Neuroradiol. 2000;21:213-8.
21. Barkovich AJ. The encephalopathic neonate: choosing the proper imaging technique. AJNR Am J Neuroradiol. 1997;18:1816-20.
22. Chen Y, Wang ZP, Zhang ZF, Shen ZY. Prognostic value of EEG background in full term neonates with asphyxia. Zhongguo Dang Dai Er Ke Za Zhi. 2007 Oct;9(5):425-8.
23. Akudo Okereafor, Joanna Allsop, Serena J. Counsell, Julie Fitzpatrick, Denis Azzopardi, Mary A. Rutherford, et al. Patterns of brain injury in neonates exposed to perinatal sentinel events. Pediatrics. 2008;121;906-14.
24. Barkovich AJ, Hajnal BL, Vigneron D, Sola A, Partridge JC, Allen F, et al. Prediction of neuromotor outcome in perinatal asphyxia: Evaluation of MR scoring systems. AJNR Am J Neuroradiol. 1998;19:143-9.
25. El-Ayouty M, Abdel-Hady H, El-Mogy S, Zaghlol-H, El-Beltagy M. Prognosis of term infants with Hypoxic ischemic encephalopathy: a clinical, EEG and MRI study. Int J Child Neuropsychiatr. 2005;2:56-73.
26. Ong LC, Kanaheswari Y, Chandran V, Rohana J, Yong SC, Boo NY. The usefulness of early ultrasonography, electroencephalography, and clinical parameters in predicting adverse outcome in asphyxiated term infants. Singapore Med J. 2009;50:705-9.
27. El-Ayouty M, Abdel-Hady H, El-Mogy S, Zaghlol H, El-Beltagy M, Aly H. Relationship between electroencephaology and magnetic resonance imaging findings after hypoxic-ischemic encephalopathy at term. Am J Perinataol. 2007;24:467-73.
28. Caravale B, Allemand F, Libenson MH. Factors predictive of seizures and neurologic outcome in perinatal depression. Pediatr Neurol. 2003;29:18-25.
29. Presslan RM, Boylan GB, Morton M, Binnie CD, Rennie JM. Early serial EEG in hypoxic ischemic encephalopathy. Clin Neurophysiol. 2001;112:31-7.
30. Murray DM, Boylan GB, Ryan CA, Connolly S. Early EEG findings in hypoxic-ischemic encephalopathy predict outcomes at 2 years. Pediatrics. 2009;124:e459-67.
31. Ezgu FS, Atalay Y, Gucuyener K, Tunc S. Neuron specific enolase levels and Neuroimaging in asphyxiated term new-borns. J Child Neurol. 2002;17:824-9.
32. Rutherford MA, Pennock JM, Dubowitz LM. Cranial ultrasound and magnetic resonance imaging in hypoxic ischaemic encephalopathy: a comparison with outcome. Dev Med Child Neurol. 1994;36:813-25.
33. Mercuri E, Ricci D, Cowan FM, Lessing D, Frisone MF, Haataja L, et al. Head growth in infants with hypoxic-ischemic encephalopathy: correlation with neonatal magnetic resonance imaging. Pediatrics. 2000;106(2 Pt 1):235-43.
34. Barnett A, Mercuri E, Rutherford M, Haataja L, Frisone MF, Henderson S, et al. Neurological and perceptual-motor outcome at 5-6 years of age in children with neonatal encephalopathy: Relationship with neonatal brain MRI. Neuropediatrics. 2002;33:242-8.
35. Kaufman AS, Miller PS, Ferriero DM, Glidden DH, Barkovich AJ, Partridge JC. Encephalopathy as a predictor of magnetic resonance imaging abnormalities in asphyxiated new-borns. Pediatr Neurol. 2003;28:342-6.
36. Biagioni E, Mercuri E, Rutherford M, Cowan F, Azzopardi D, Frisone MF, et al. Combined use of electroencephalogram and magnetic resonance imaging in full-term neonates with acute encephalopathy. Pediatrics. 2001;107:461-8.

DOI: 10.5455/2320-6012.ijrms20150306
Cite this article as: Patil B, Harshangi S, Prabhu B. Clinicoradiological correlation in birth asphyxia. Int J Res Med Sci 2015;3:560-7.