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Original Article

Paediatrics Section

Value of Cranial Ultrasonography and Resistive Index of Cerebral Arteries in Predicting Neuromotor Outcomes in Newborns with Hypoxic Ischaemic Encephalopathy

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ABSTRACT

Introduction: Perinatal asphyxia is one of the leading cause of neonatal mortality and childhood morbidity. Use of cranial Ultrasonography (USG) and cranial arterial doppler indices has been evaluated for predicting risk of neuromotor impairment.

Aim: Study was aimed to evaluate the role of cranial USG and Resistive Index (RI) of Anterior and Middle Cerebral Arteries (ACA, MCA) in predicting neuromotor outcomes at six months of age in term neonates with perinatal asphyxia and Hypoxic Ischaemic Encephalopathy (HIE).

Materials and Methods: This prospective study was carried out at a tertiary care teaching hospital. Subjects with perinatal asphyxia and HIE were grouped in different Sarnat and Sarnat stages. Cranial USG and RI measurement of ACA, MCA was done within five days of birth. They were followed up at six months of age for neuromotor outcome. Outcome was correlated with Sarnat stages, USG finding and RI of ACA, MCA. It was carried out using R statistical environment.

Results: A total of 43 neonates with perinatal asphyxia and HIE were evaluated for neuromotor outcomes. Both ACA and MCA, RI were also significantly associated with the clinical stages ($p<0.0001$). Out of these 19 cases presented with neuromotor abnormality. USG was abnormal in 14 cases. RI of MCA and ACA in subjects with abnormal neuromotor outcome were 0.57 and 0.53 respectively. There was significant association between abnormal USG and decreasing RI with increasing neuromotor scale. Both ACA RI and MCA, RI were significant predictors for neuromotor impairment by univariate and multivariate logistic regression and USG was significant in univariate logistic regression.

Conclusion: Abnormality detected by cranial USG and RI of cerebral vessels within few days of life in cases of perinatal asphyxia and HIE were significantly associated with poor neuromotor outcome. The strength of association in predicting neuromotor outcome was more marked with RI than USG.

Keywords: Neuromotor impairment, Perinatal asphyxia, Resistive index

INTRODUCTION

Perinatal asphyxia is one of the leading causes of neonatal mortality and childhood morbidity worldwide. According to national neonatal perinatal database, it accounts for 28.8% of deaths in neonates [1]. Perinatal asphyxia may lead to HIE, which contributes to chronic sequelae such as global developmental delay, deafness, loss of vision and even death.

Transfontanellar sonography with spectral analysis of the cerebral blood flow allowing calculation of the RI is a safe, bed-side, and cost-efficient method to measure cerebral haemodynamics following HIE [2]. During the post asphyxiated period there is cerebral vasodilatation resulting in fall of vascular resistance. This hyperemic phase is responsible for secondary brain injury. Abnormal cranial USG findings at birth have been found to be associated with long-term neuromotor outcomes [3,4]. Doppler indices calculated from the ACA and MCA reflect haemodynamic changes in HIE and serves as an early predictor for neuromotor outcome [5]. Studies have found decreased cerebral vessels RI to reasonably predict the risk of subsequent neurodevelopmental impairment [6-10]. There is paucity of data on the prognostic role of USG findings and doppler indices in perinatal asphyxia

and HIE from India [11]. Hence, this study was undertaken to evaluate the role of cranial USG and RI of ACA and MCA in predicting neuromotor outcomes at six months of age in term neonates with perinatal asphyxia and HIE.

MATERIALS AND METHODS

This prospective study was carried out from April 2015 to September 2016 in the Department of Radiodiagnosis and Paediatrics at Saroini Naidu Medical College Agra after obtaining approval from the Institutional Ethical Committee. Total 107 term neonates (>37 weeks of gestation) admitted (both inborn and outborn) in the Neonatal Intensive Care Unit (NICU) of tertiary care hospital during the study period with perinatal asphyxia and HIE were included in the study after taking consent from their guardian/caregiver. Perinatal asphyxia was defined as per WHO criteria as failure to initiate and sustain breathing at birth [12]. Neonates with perinatal asphyxia and features of HIE according to Sarnat and Sarnat staging in first 24 hours of birth were evaluated for the study [13]. Neonates with congenital anomalies and sepsis were excluded from the study. A detailed examination of the neonates was done and they were classified according to modified Sarnat and Sarnat staging system into Stage 1, 2 and 3 [13]. These neonates underwent cranial USG and doppler evaluation within five days of birth.

Cranial USG and Doppler was performed on Medison Sono Ace X8 Ultrasound machine using C2-5EL convex and HL5-12ED linear phased array transducer in the Department of Radiodiagnosis by experienced radiologists. Brain structures were visualised through the fontanelles as acoustic windows as well as transtemporal in the coronal and sagittal planes. Six coronal images and five sagittal images including a midline sagittal were obtained. We evaluated anatomical brain structure for ventricular sizes and lumen, grey and white matter, grey white demarcation, thalamus and basal ganglia outline and echogenicity, cerebrospinal fluid spaces, and posterior fossa contents. Sonographic findings were grouped in to brain oedema with or without loss of grey white demarcation, involvement of thalamus and basal ganglia, posterior fossa structural involvement and haemorrhage (intraparenchymal/intraventricular). Doppler evaluation of both side ACA and MCA was done simultaneously in all subjects to obtain a spectral tracing and RI. All efforts were taken to keep the angle of insonation less than 15 degree.

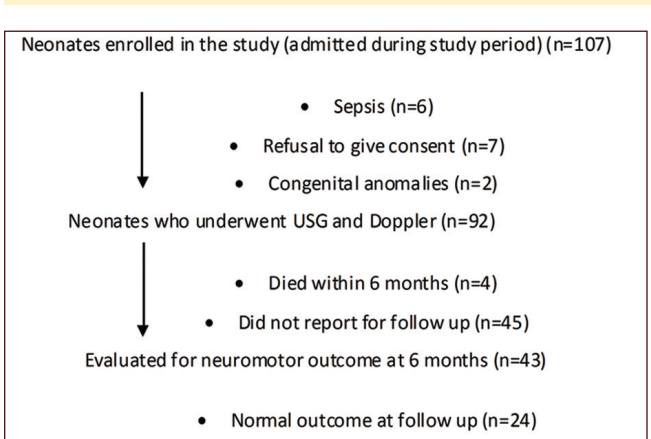
These infants were followed up at six months of age for the assessment of neuromotor status. Those neonates who did not report for follow-up were excluded from statistical analysis. Neuromotor status was evaluated in the neonatal unit by applying standard neuromotor evaluation scale [14]. It included assessment of child's posture, gait, muscle tone, reflexes and cranial nerve function. Based on this evaluation subjects were distributed into 6 groups: normal neuromotor status, slight change in muscle tone or reflexes; slight change in muscle tone and reflexes; changes in muscle tone or reflexes or both and reduced power in body and extremities; any associated cranial nerve damage; and spastic quadriparesis. Presence of neuromotor impairment was taken as poor outcome whereas normal neuromotor status was taken as good outcome. These subjects with good outcome served as control.

STATISTICAL ANALYSIS

Anterior cerebral arteries-RI, MCA-RI and USG findings were the predictors in the study. Association of these variables (ACA-RI, MCA-RI and USG) with the Sarnat and Sarnat Stage, the presence/absence of neuromotor impairment and the six neuromotor groups was tested. The significance of the association of Sarnat and Sarnat Stage with ACA-RI and MCA-RI was tested by Jonckheere-Terpstrata trend test and with USG by the Fisher's exact test. For the purpose of this analysis, the USG findings were broadly categorised into two groups, namely, Normal (in which no abnormality could be detected) and abnormal (in which there was presence of atleast one abnormality). The association of the USG findings to neuromotor impairment was tested by the Fisher's exact test. The Odds ratio (OR), sensitivity, specificity, Positive Predictive Value (PPV) and Negative Predictive Value (NPV) of USG to detect any neuromotor impairment was also estimated. Additionally, USG was studied for predictive accuracy by univariate and multivariate (using the Sarnat and Sarnat stages as a covariate) Logistic regression. The ACA-RI and MCA-RI of the different neuromotor outcome groups were compared and tested for differences by the Kruskall-Wallis test followed by Jonckheere-Terpstrata trend test and Kendall Correlation coefficient. The significance of the association of USG with the neuromotor scale was estimated by the chi-square test for trend. The accuracy of ACA-RI and MCA-RI to predict neuromotor impairment were measured by means of ROC curve analysis in which the Area Under Curve (AUC) was estimated. The sensitivity and specificity and Youden index of MCA-RI and ACA-RI were estimated at different cut-off points of the ROC curve to estimate an "optimal" diagnostic cut-off. The independent significance of the predictive strength of ACA-RI and MCA-RI was also tested by multivariate Logistic regression using the Sarnat and Sarnat stages as a covariate. The analysis was carried out using R statistical environment (version 3.1.2) aided by the R packages "pROC" and "PMCMR".

RESULTS

One hundred and seven term neonates were admitted with perinatal asphyxia and hypoxic ischemic encephalopathy during the study period. Sixty-four were excluded as per exclusion criteria shown in [Table/Fig-1]. Remaining 43 cases were evaluated for neuromotor development and data were analysed.



[Table/Fig-1]: Study flow chart.

Out of the 43 newborns with perinatal asphyxia and HIE, 18 (41.9%) were in Sarnat Stage 1 followed by 14 (32.5%) in Stage 2 and 11 (25.6%) were in Sarnat stage 3. Abnormal USG was found in 9 (64.3%) cases of Stage 2 and 9 (81.3%) cases of Stage 3. Median RI in Stage 2 was 0.53 (ACA) and 0.54 (MCA) as against 0.70 (ACA) and 0.71 (MCA) in Stage 1 respectively. The association between ACA-RI, MCA-RI, USG with Sarnat and Sarnat stage is given in [Table/Fig-2]. There was significant association between USG and Sarnat Stage (p-value=0.0006). Both ACA-RI and MCA-RI were also significantly associated with the Sarnat and Sarnat Stage (p-value ACA-RI and MCA-RI <0.0001).

[Table/Fig-3] shows correlation of USG, MCA-RI and ACA-RI with neuromotor outcomes at six months of age. About 19 (44.2%) subjects showed abnormal neuromotor outcomes. Out of these 19 cases, 14 had abnormal USG findings. Diffuse parenchymal oedema with loss of grey white demarcation [Table/Fig-4] was the most common USG finding seen in 11 (78.5 %) cases. Of these nine showed abnormality in tone and/or reflexes and two had in addition reduced power. Involvement of thalamus and basal ganglia was seen in two subjects; both of them presented with spastic quadriparesis at follow-up. There was significant association of USG with the different neuromotor outcome groups [Table/Fig-3].

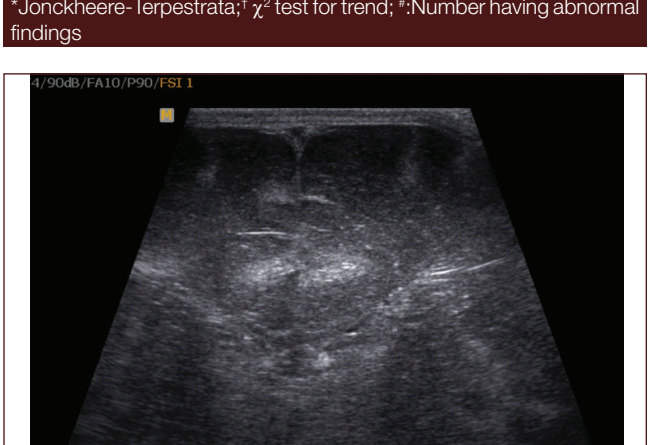
MCA-RI [Table/Fig-5] and ACA-RI in subjects with abnormal neuromotoroutcome was 0.57 and 0.53 respectively. On testing for differences in ACA-RI and MCA-RI among the different outcome groups, significant difference (p<0.0001 for both ACA-RI and MCA-RI). The Jonckheere-Terpstrata Trend test showed a significant association between decreasing Resistive indices and increasing neuromotor scale [Table/Fig-3]. The Kendall correlation coefficient between ACA. RI and the neuromotor scale was -0.71(bootstrap 95% CI: -0.78 to -0.59), while the Kendall correlation coefficient between MCA-RI and the neuromotor scale was -0.64 (bootstrap 95% CI: -0.7309 to -0.4297).

Sarnat and Sarnat Stage	ACA-RI: Median (Range)	MCA-RI: Median (Range)	USG#
1 (n=18)	0.70 (0.56 to 0.79)	0.71 (0.41 to 0.82)	5
2 (n=14)	0.70 (0.51 to 0.82)	0.69 (0.47 to 0.84)	9
3 (n=11)	0.53 (0.39 to 0.59)	0.54 (0.45 to 0.6)	9
p-value	<0.0001*	<0.0001*	0.0006†

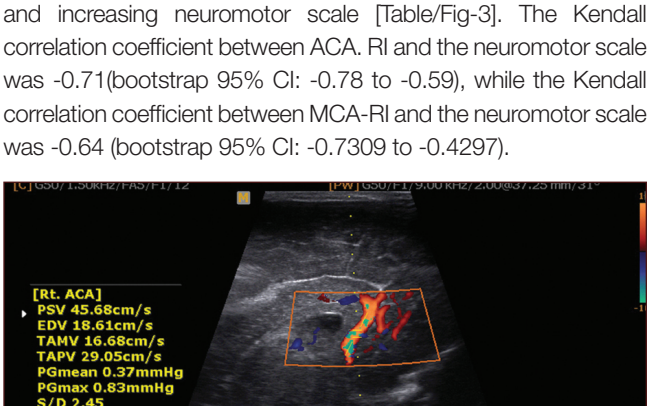
[Table/Fig-2]: Association of ACA-RI, MCA-RI and USG with Sarnat and Sarnat Stage.
*Jonckheere-Terpe strata test;† Fisher-exact test; #: Number having abnormal findings

Neuromotor Scale Groups	ACA-RI Median (Range)	MCA-RI Median (Range)	USG-GS#
Group 1 (n=24)	0.72 (0.59 to 0.82)	0.72 (0.41 to 0.84)	7
Group 2 (n=8)	0.58 (0.53 to 0.72)	0.59 (0.54 to 0.73)	5
Group 3 (n=3)	0.54 (0.39 to 0.57)	0.54 (0.47 to 0.6)	2
Group 4 (n=6)	0.51 (0.41 to 0.56)	0.465 (0.45 to 0.59)	5
Group 6 (n=2)	0.515 (0.51 to 0.52)	0.535 (0.53 to 0.54)	2
p-value	<0.0001*	<0.0001*	0.003†

[Table/Fig-3]: Association of ACA-RI, MCA-RI and USG with groups defined by Neuromotor scale.
*Jonckheere-Terpestrata;† χ^2 test for trend; #:Number having abnormal findings



[Table/Fig-4]: Coronal scan showing diffuse parenchymal oedema with chinked lateral ventricles and loss of grey white demarcation.



[Table/Fig-5]: Right anterior cerebral artery spectral waveform of same subject showing low RI (0.59).

Covariates included in Multivariate Analysis		Univariate Analysis	Multivariate Model 1	Multivariate Model 2	Multivariate Model 3
			ACA-RI , Sarnat and Sarnat Stage	MCA-RI , Sarnat and Sarnat Stage	USG-GS , Sarnat and Sarnat Stage
ACA-RI	Coefficient	-39.70	-33.40	NA	NA
	Standard error	11.85	11.31	NA	NA
	p-value	0.0008	0.0031	NA	NA
MCA-RI	Coefficient	-22.20	NA	-15.85	NA
	Standard error	6.27	NA	5.73	NA
	p-value	0.0004	NA	0.0057	NA
USG-GS	Coefficient	2.14	NA	NA	0.86
	Standard error	0.72	NA	NA	0.96
	p-value	0.0031	NA	NA	0.37
Sarnat and Sarnat Stage	Coefficient	2.63	2.03	2.40	2.35
	Standard error	0.7465	1.40	1.006	0.77
	p-value	0.0004	0.14	0.017	0.002

[Table/Fig-6]: Univariate and Multivariate Logistic regression models testing the predictive significance of ACA-RI, MCA-RI and USG.

USG was found to be a significant predictor for any abnormal neuromotor status on follow-up with OR of 8.003 (95% CI: 1.737 to 46.207), sensitivity of 0.778 (95% CI: 0.524 to 0.936), specificity of 0.708 (95% CI: 0.489 to 0.874), PPV of 0.667 (95% CI: 0.430 to 0.854) and NPV of 0.810 (95% CI: 0.581 to 0.946).

Both ACA-RI and MCA-RI had high ability to predict for neuromotor outcome with AUC for ACA-RI being 0.9705 (bootstrap 95% CI: 0.9046 to1) and the AUC for MCA-RI being 0.9265 (bootstrap 95% CI: 0.8224 to 1). The present analysis showed that a cut-off value of 0.63 showed the highest Youden index for ACA-RI with a sensitivity of 0.947 and specificity of 0.958. Similarly, a value of 0.64 showed the highest Youden index for MCA-RI with a sensitivity of 0.875 and specificity of 0.947.

Both ACA-RI and MCA-RI were significant predictors for neuromotor outcome by univariate and multivariate Logistic Regression [Table/Fig-6], suggesting that both ACA-RI and MCA-RI gave additional predictive information for the neuromotor status compared to the Sarnat and Sarnat stage. However, USG attained statistical significance only in univariate Logistic regression, but not on multivariate analysis [Table/Fig-6].

DISCUSSION

In this study, we evaluated the role of cranial USG, ACA-RI and MCA-RI during the first few days of birth in neonates with perinatal asphyxia and HIE and correlated to neuromotor outcome at 6 months of age.

Cranial USG at birth showed significant association with neuromotor outcome at six months of age. Himpens E et al., have observed that any brain injury detected by USG increases probability of cerebral palsy by 7-fold [15,16]. According to another study 33% full term newborns with neuromotor and mental abnormalities during second year of life had abnormal USG scans related to HIE at birth [3]. Whereas, in present study abnormal cranial USG scans were found in larger number of cases with abnormal neuromotor outcomes. In present study subjects with brain oedema were associated with mild impairment, this was in contrast with Boo NY et al., reported severe neuromotor impairment at the age of one year in subjects with brain oedema [4]. In present study subjects showing involvement of thalamus and basal ganglion on USG scan presented with severe neuromotor impairments such as spastic quadriparesis. Similar observations have been reported where presence of brain swelling involving thalamus, basal ganglia severe neuromotor and mental development impairment was detected in 33% to 92.3% of the subjects [17-20]. In another study it has been observed that injury to thalamus and basal ganglia detected by USG increased the probability of spastic cerebral palsy by 31-fold ($p<0.001$) [15,16].

This study showed that subjects with lower RI values in MCA and ACA demonstrated poor neuromotor outcome. Similar observations have been made in other studies as well [21-23]. In subjects with perinatal asphyxia and HIE, ACA/MCA RI may be reduced due to increase in cerebral circulation with reduced resistance [24]. There was significant difference of RI in cranial arteries in subjects with abnormal neuromotor outcome as observed with those with normal outcome, similar observation has also been made by Kumar AS et al., [11]. Subsequent to the demonstration of the predictive ability of ACA-RI and MCA-RI in predicting presence/absence of neuromotor disability in neonates with perinatal asphyxia and HIE, the present study demonstrated strong association of both ACA-RI and MCA-RI with the neuromotor scale, as evidenced by the large negative Kendall correlation coefficients. This suggests that the resistive indices potentially may predict the severity of the neuromotor impairment; however, the robustness of this finding is limited by the low number of children having severe neurological impairment in the present study.

The most striking finding in the present study is the demonstration of the ability of ACA-RI and MCA-RI to predict neuromotor outcome in neonates with perinatal asphyxia. Both ACA-RI and MCA-RI show

high AUCs in differentiating children with poor neuromotor outcome from those with good outcome. Additionally, a cut-off of 0.63 for ACA-RI and 0.64 for MCA-RI appear to be optimal in predicting neuromotor abnormality on follow up. In study by Aurele K et al., reported RI less than 0.56 to have specificity of 0.95 sensitivity of 0.33. At a similar cut-off in present study, the specificity of ACA-RI and MCA-RI are 1.0 and 0.96 respectively [9]. The corresponding figures for sensitivity are 0.63 and 0.47 for ACA-RI and MCA-RI. Thus, the cut-off 0f 0.56 has high specificity but low sensitivity; authors believe that a higher cut-off of about 0.63 as found in present study will increase the sensitivity for predicting neuromotor abnormality markedly while still keeping a high specificity. However, these cut offs should be validated by a larger study with support of Decision theory.

Multivariate logistic regression shows independent significance and added predictive value of both Resistive indices even when information given by the Sarnat and Sarnat Stage is taken in account. Though the study showed association of USG, ACA-RI and MCA-RI in predicting the outcome, strength of association is more in latter. This can be attributed to the fact that measurement of RI value of vessels is angle independent and thus objective and intra and inter observer variation is low.

LIMITATION

Small number of subjects and large percentage of lost to follow-up are some of the limitations of the study.

CONCLUSION

The results of present study suggested that abnormality detected by cranial USG, lower RI of ACA and MCA within few days of life in cases of perinatal asphyxia and HIE were significantly associated with poor neuromotor outcome. Strength of association in predicting neuromotor outcome was more marked with RI than USG abnormality. With a cut-off value of RI of MCA and ACA at 0.63 showed high sensitivity and specificity for neuromotor outcome. These findings may be important for devising the follow-up and rehabilitation plan for such neonates during the postnatal period and later.

AUTHOR'S CONTRIBUTION

SN, VV, DA and PKS drafted the study. PKS, DA, RB collected the data. VV and SN performed USG and doppler scan. NC analysed the data. SN, DA, NC drafted the manuscript. All authors approved the study.

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