



## Correlation of Clinical Profile and Electroencephalographic Finding in Hypoxic-Ischemic Encephalopathy in a Tertiary Care Hospital: A Single-Center Experience from Bangladesh

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### Abstract

**Background:** Perinatal asphyxia and resultant hypoxic-ischemic encephalopathy (HIE) is not an uncommon phenomenon in a developing country, like Bangladesh. Electroencephalogram (EEG) is regarded as an effective prognostic tool. Correlation of clinical profiles and EEG findings of HIE patients are not commonly observed in Bangladesh. The aim of the study was to observe the clinical profile and EEG changes in different stages of hypoxic-ischemic encephalopathy and compare them in a tertiary care hospital. **Material & Methods:** This is a cross-sectional observational study conducted for a period of six months in Dhaka Medical College Hospital, Dhaka. Sarnat and Sarnat score was used to classify HIE. 20 asphyxiated neonates without HIE were selected as the control group (group II) while 30 asphyxiated neonates with HIE, were selected as the case group (group I) by purposive sampling. Clinical profiles, EEG findings, and immediate outcomes were observed and compared between the two groups. **Results:** 73.3% patients were delivered at term and 30% patients were delivered at home in group I. 70% patients' delivery were conducted by doctor in group I and 75% in group II. 63.3% patients had meconium stain in group I and 25% in group II, which was found significant. 46.7% had prolong labor in group I and 20% in group II, 40% had premature ruptured membrane (PROM) in group I and 40% in group II. Hypothermia, weak primitive reflexes, hypotonia, lethargy and seizure were significantly higher in group I. Changes in EEG correlated between the two groups and was found significant. Also, patients in group I, needed prolong hospital stay. **Conclusions:** The clinical profiles and EEG changes in patients with hypoxic ischemic encephalopathy was concluded that there is significant association with meconium stain, seizure, hypothermia, weak primitive reflexes, lethargic, miosis, hypotonia, poor APGAR score, burst suppression & SET findings in EEG and prolonged hospital stay in prenatal asphyxia with hypoxic ischemic encephalopathy.

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## INTRODUCTION

Neonatal death is a major barrier in improving survival of under-five mortalities in developing countries. It now accounts for more than two-thirds of all deaths under first year of life and for about half of under-five child mortality.<sup>[1,2]</sup> In rural Bangladesh, 12.5% of newborns do not breathe immediately after birth and 9-10% of urban newborns are born with moderate to severe birth asphyxia; more than 150,000 newborns are born asphyxiated every year.<sup>[3-6]</sup> Hypoxic Ischemic Encephalopathy (HIE) is the manifestation of damage to cells in the central nervous system (the brain and spinal cord) from inadequate oxygen.<sup>[7,8]</sup> More than a million children who survive birth asphyxia develop problems such as cerebral palsy, mental retardation, learning difficulties, and other disabilities.<sup>[9,10]</sup> For the past 30 years, term infants with HIE have been evaluated clinically with the Sarnat neurologic examination,<sup>[11,12]</sup> which correlates well with neurodevelopmental impairment in infancy and childhood.<sup>[13-15]</sup> The scoring systems adapted from the Sarnat scoring system, including the Thompson score and Miller score,<sup>[16,17]</sup> also have been shown to predict outcome. <sup>[18-20]</sup> Several studies have reported the use of the electroencephalogram (EEG) in HIE not only for the recognition of seizures but also as a reliable predictor of neurodevelopmental outcome.<sup>[21-23]</sup> In particular, it has been demonstrated that it is the background activity, which best predicts the outcome, whereas the significance of paroxysmal abnormalities is more controversial.<sup>[24-28]</sup> In this study, attempt was made to see the clinical profiles and EEG changes in the brain due to hypoxic ischemic encephalopathy in this regard that it might help

us to plan the management and predict the outcome of these vulnerable group.

## Objectives

This study is conducted to observe the clinical profiles of patient with all stages of HIE. To see the EEG changes in different stages of HIE. To compare the EEG changes in different stages of HIE.

## MATERIAL AND METHODS

This was a cross sectional observational study by using purposive sampling method. The study period was on Jan, 2012 - July, 2012. Sample size was 50. 30 neonates with perinatal asphyxia with HIE was enrolled in this study. The study was carried out in Department of Neonatology, Dhaka Medical College Hospital (DMCH), Dhaka, Bangladesh.

## Inclusion Criteria

All neonates with history of failure or delayed onset of spontaneous respiration after birth with or without any of the followings-

Those babies requiring resuscitation at birth. Abnormal APGAR scores at 5 min (<7). Abnormal neurologic feature or clinical seizure, due to birth asphyxia.

## Study Procedure

30 patients asphyxiated neonates who fulfilled the inclusion criteria along with neurological signs of HIE were selected as cases (group I) and 20 patients with only history of delayed cry or perinatal asphyxia without any neurological sign of HIE were selected as control group (group II) by purposive sampling. Gestation age, birth weight, relevant perinatal history,

examination findings were recorded. The post asphyxiated neonates were managed according to neonatal resuscitation protocol. Assessment of the neurologic status was done by Sarnat & Sarnat staging for HIE. EEG of brain was done from DMCH, by modified International 10-20 electrode placement for neonates, preferably within 2 weeks, to see any correlation with hospital outcome. Total hospital stays and outcome was addressed appropriately.

### Ethical Issues

A written informed consent was taken from parent/legal guardian. Ethical clearance was taken from institute ethical review committee.

### Statistical analysis

Data were entered into computer with the help of software SPSS for windows programmed version 26.0. Results on continuous measurements are presented on Mean  $\pm$ SD (Min-Max) and results on categorical measurements are presented in number (%). Chi-square has been used to find the significance of the study parameters on categorical scale between two variables.

## RESULTS

[Table 1] showed patient's condition at birth. Almost three fourth (73.3%) patients were term in group I and 17 (85%) in group II. Two third (66.7%) patients were male and 10 (33.3%) were female in group I. 10 (33.3%) patients were found to have low birth weight in group I and 4(20%) in group II. The difference was statistically not significant ( $p>0.05$ ) between the two groups.

[Table 2] showed labor and delivery history of the study patients. 30% patients delivered at

home in group I and 25% in group II. Almost three fourth (70%) patients' delivery conducted by doctor in group I and 75% in group II. 53.3% patients were born through vaginal delivery in group I and 30% in group II. The difference was statistically not significant ( $p>0.05$ ) between the two groups.

[Table 3] showed maternal health information of the study patients. Almost three fourth (73.3%) had regular ANC in group I and 65% in group II. Half (50%) of the patients came from lower income family in group I. On antenatal health of mother, 20% had HTN in group I. The differences were statistically not significant between the two groups.

[Table 4] showed risk factors of the study patients. It was observed that almost two third patients were meconium stained in group I and 25% in group II. Almost half (46.7%) had born after prolong labor in group I and 20% in group II. Other findings are depicted in the table.

[Table 5] showed clinical feature of the study patients. Only 13.3% patients had no respiration in group I and no such findings in group II. More than three fourth had seizure in group I and no such findings in group II. The seizure difference was statistically significant ( $p<0.05$ ) between the two groups.

[Table 6] showed physical examination of the study patients. It was observed that half of the patients had anemia in group I and 35% in group II, 40% had cyanosis in group I and 25% in group II. Respiratory difficulty was found in 70% in group I and 50% in group II. Grunting was found 40% in group I and 25% in group II. Hypothermia was found in 43.3% in group I and 25% in group II. Weak primitive reflexes were found 70% in group I and 35% in group II. 70% patients in group I, were found lethargic, while

20% in group II. Miotic condition of pupil was observed in 70% in group I. Hypotonia was seen in 66.7% in group I. 63.3% patients had poor perfusion in group I and 15% in group II. Almost three fourth (70%) patients had seizure in group I and no such findings in group II. Temperature, primitive reflexes, level of consciousness, condition of pupil, muscle tone, poor perfusion and seizure difference were statistically significant ( $p < 0.05$ ) between the two groups.

[Table 7] showed APGAR score of the study patients. It was observed that majority (95.8%) of the patients belonged to  $<7$  in group I and 9 (60%) in group II. The difference was statistically not significant between the two groups.

[Table 8] showed EEG findings of the study patients. 66.6% patients had SET findings in

group I. Other findings are depicted in the table. The difference was statistically significant ( $p < 0.05$ ) between two groups.

[Table 9] showed hospital outcome of the study patients. Two third of the patients had prolonged hospital stay (8-14 days) in group I and 30% in group II. 66.7% discharged patients had no apparent sequel in group I and 33.3% had apparent neurological sequel, while 100% in group II had no neurological deficit apparently. The hospital stay difference was statistically significant ( $p < 0.05$ ) between the two groups.

[Table 10] showed poor perfusion had 6.000 (95% CI 1.118 to 64.491) times increase in odds having hypoxic ischemic encephalopathy. Poor perfusion was significantly associated with hypoxic ischemic encephalopathy.

**Table 1:** Distribution of the study patients by baby's condition at birth (N=50).

Baby's condition at birth	Group-I (n=30)		Group-II (n=20)		P value
	n	(%)	n	(%)	
Gestational age (weeks)					
Preterm ( $\leq 36+$ weeks)	8	26.7	3	15.0	0.329 <sup>ns</sup>
Term (37-42 weeks)	22	73.3	17	85.0	
Sex					
Male	20	66.7	10	50.0	0.239 <sup>ns</sup>
Female	10	33.3	10	50.0	
Age of admission (hours)					
$\leq 12$	24	80.0	15	75.0	0.676 <sup>ns</sup>
$> 12$	6	20.0	5	25.0	
Birth weight (gm)					
1600-2499	10	33.3	4	20.0	0.304 <sup>ns</sup>
2500-4000	20	66.7	16	80.0	

**Table 2:** Distribution of the study patients by labor and delivery history (N = 50).

Labor and delivery	Group-I (n=30)		Group-II (n=20)		P value
	n	(%)	n	(%)	
Place of delivery					
Home	9	30.0	5	25.0	0.129 <sup>ns</sup>

DMCH	7	23.3	10	50.0	
Other hospitals	14	46.7	5	25.0	
Delivery conducted person					
Doctor	21	70.0	15	75.0	0.812 <sup>ns</sup>
Nurse	3	10.0	1	5.0	
Dai	6	20.0	4	20.0	
Mode of delivery					
Vaginal	16	53.3	6	30.0	0.104 <sup>ns</sup>
UCS	14	46.7	14	70.0	

**Table 3:** Distribution of the study patients by maternal health information (N=50)

Maternal health information	Group-I (n=30)		Group-II (n=20)		P value
	n	(%)	n	(%)	
Maternal age (years)					
≤20	8	26.7	3	15.0	0.283 <sup>ns</sup>
21-30	20	66.6	13	65.0	
>30	2	6.7	4	20.0	
ANC					
Regular	22	73.3	13	65.0	0.529 <sup>ns</sup>
Irregular	8	26.7	7	35.0	
Parity					
Primiparous	13	43.3	7	35.0	0.556 <sup>ns</sup>
Multiparous	17	56.7	13	65.0	
Socioeconomic status					
Low	7	23.3	6	30.0	0.365 <sup>ns</sup>
Lower middle	8	26.7	8	40.0	
Middle	15	50.0	6	30.0	
Antenatal health of mother					
HTN	6	20.0	0	0.0	0.102 <sup>ns</sup>
GDM/DM	4	13.3	3	15.0	

**Table 4:** Distribution of the study patients by risk factors (N=50)

Risk factors	Group-I (n=30)		Group-II (n=20)		P value
	N	(%)	n	(%)	
Obstructed labor	2	6.7	0	0.0	0.239 <sup>ns</sup>
Prolong labor	14	46.7	4	20.0	0.054 <sup>ns</sup>
Meconium stain	19	63.3	5	25.0	0.008 <sup>s</sup>
APH	5	16.7	3	15.0	0.875 <sup>ns</sup>
PET	4	13.3	2	10.0	0.722 <sup>ns</sup>
Assisted breech	2	6.7	1	5.0	0.808 <sup>ns</sup>
PROM	12	40.0	8	40.0	

**Table 5:** Distribution of the study patients by clinical feature (N=50)

Clinical feature	Group-I (n=30)		Group-II (n=20)		P value
	n	(%)	n	(%)	
Ist Cry					
No cry	2	6.7	0	0.0	0.239 <sup>ns</sup>
Delayed cry	28	93.3	20	100.0	
No respiration					
Yes	4	13.3	0	0.0	0.089 <sup>ns</sup>
No	26	86.7	20	100.0	
Seizure					
Yes	23	76.7	0	0.0	0.001 <sup>s</sup>
No	7	23.3	20	100.0	

**Table 6:** Distribution of the study patients by physical examination (N=50)

Physical examination	Group-I (n=30)	Group-II (n=20)		P value	
	N	(%)	n		(%)
Anemia					
Yes	15	50.0	7	35.0	0.295 <sup>ns</sup>
No	15	50.0	13	65.0	
Cyanosis					
Yes	12	40.0	5	25.0	0.273 <sup>ns</sup>
No	18	60.0	15	75.0	
Respiratory difficult					
Yes	21	70.0	10	50.0	0.154 <sup>ns</sup>
No	9	30.0	10	50.0	
Grunting					
Yes	12	40.0	5	25.0	0.273 <sup>ns</sup>
No	18	60.0	15	75.0	
Temperature					
Normal	17	56.7	9	45.0	0.005 <sup>s</sup>
Hyperthermia	0	0.0	6	30.0	
Hypothermia	13	43.3	5	25.0	
Primitive reflexes					
Strong	3	10.0	13	65.0	0.001 <sup>s</sup>
Weak	21	70.0	7	35.0	
Absent	6	20.0	0	0.0	
Consciousness					
Alert	3	10.0	16	80.0	0.001 <sup>s</sup>
Lethargic	21	70.0	4	20.0	
Coma	6	20.0	0	0.0	

Condition of pupil					
Normal	0	0.0	20	100.0	0.001 <sup>s</sup>
Mydriasis	3	10.0	0	0.0	
Miosis	21	70.0	0	0.0	
Unequal	6	20.0	0	0.0	
Muscle tone					
Normal	4	13.3	20	100.0	0.001 <sup>s</sup>
Hypotonic	20	66.7	0	0.0	
Flaccid	6	20.0	0	0.0	
Poor perfusion					
Yes	19	63.3	3	15.0	0.001 <sup>s</sup>
No	11	36.7	17	85.0	
Seizure					
Yes	21	70.0	0	0.0	0.001 <sup>s</sup>
No	9	30.0	20	100.0	

**Table 7:** Distribution of the study patients by APGAR Score (N=39)

	Group-I (n=24)		Group-II (n=15)		P value
	n	%	n	%	
APGAR Score					
<7	23	95.8	9	60.0	0.005 <sup>s</sup>
≥7	1	4.2	6	40.0	
Mean ±SD	4.3±1.3		5.6±1.4		

**Table 8:** Distribution of the study patients by EEG findings (N=50)

EEG findings	Group-I (n=30)		Group II (n=20)		P value
	n	%	n	%	
Normal	6	20.0	20	100.0	0.001 <sup>s</sup>
SET	20	66.6	0	0.0	
Burst suppression	2	6.7	0	0.0	
Others	2	6.7	0	0.0	

**Table 9:** Distribution of the study patients by hospital outcome (N=50)

Hospital outcome	Group-I (n=30)		Group II (n=20)		P value
	n	%	n	%	
Hospital stays (in days)					
≤7	8	26.7	14	70.0	0.008 <sup>s</sup>
>7	22	73.3	6	30.0	
Mean ±SD	9.9±3.6		6.4±3.88		
Discharge					
Without apparent sequel	20	66.7	20	100.0	0.004 <sup>s</sup>

With neurologic sequel	10	33.3	0	0.0	
Out come					
Alive	24	80.0	18	90.0	0.452 <sup>ns</sup>
DORB	4	13.3	2	10.0	
Dead	2	6.7	0	0.0	

**Table 10:** Factors associated with hypoxic ischemic encephalopathy in multivariable regression model

	B	P value	OR	95.0% C.I. for EXP(B)	
				Lower	Upper
Meconium stain	34.42	0.048	2.94	3.71	32.66
Seizure	87.49	0.033	3.05	3.26	8.89
Temperature	33.51	0.038	2.71	4.75	42.29
Primitive	-17.55	0.031	2.74	3.16	10.64
Level of consciousness	16.05	0.044	2.34	5.89	18.8
Condition of pupil	-74.17	0.048	2.99	4.75	31.28
Muscle tone	5.8	0.049	1.53	4.96	39.04
Poor perfusion	3.29	0.033	4.38	6.85	35.92
Seizure	118.82	0.036	1.49	3.37	20.23
EEG	18.03	0.033	1.31	4.25	41.77
Constant	-398.38	0.992	0		

## DISCUSSION

Perinatal asphyxia or hypoxic ischemic encephalopathy (HIE) is a condition of impaired blood gas exchange during intra partum period that, if it persists, leads to progressive hypoxemia and hypercapnia with a metabolic acidosis.<sup>[6-10]</sup> A total of 50 patients with perinatal asphyxia admitted in the Special Care Baby Unit (SCBU), in of Dhaka Medical College & Hospital, Dhaka, during January 2017 to June 2017, were included in this study. Among them 30 asphyxiated patients with HIE were considered as group I (case) and 20 asphyxiated patients without HIE were considered as group II (control). Those with congenital anomalies and birth weight <1000 gm was excluded from the study. In this present study, it was observed that 73.3% patients were term (37-42 weeks) in group I and 85% in group

II. The difference was statistically not significant. Bhunia and Saharia,<sup>[29]</sup> showed 99% were term in case group and 94% in control group. Similar observations were also seen in several studies.<sup>[30,31]</sup> In this current study, it was observed that 66.7% patients were male and 33.3% were female in group I. The difference was statistically not significant between the two groups. Bhunia and Saharia,<sup>[29]</sup> also found male predominant in their study, where male to female ratio was 2.35:1. Similarly Jose et al.<sup>[32]</sup> also observed that HIE is more frequent in male subject. In this study, 80% patients found getting admitted within 12 hours after birth in group I and 75% in group II. The difference was statistically not significant. Asphyxiated babies needed earlier admission for resuscitation or had other different serious complications. In this study, it was observed that 33.3% patients in group I were low birth weighted. The



difference was statistically not significant, though it is one of the major culprits for causing birth asphyxia.<sup>[33]</sup> A potential confounder for this could be the fact that mother of low-birth-weight babies often related to complications such as maternal hypertension and diabetes.<sup>[33]</sup> Similarly, Shireen et al.,<sup>[31]</sup> found the mean birth weight of the asphyxiated babies was 2.4 kg with a range of 1.0- 4.1 kg. Similar finding were also observed.<sup>[29]</sup> In this series, it was observed that 30% patients were delivered at home in group I and 25% in group II. 70% patients delivery were conducted by doctor in group I and 75% in group II. 53.3% patients had vaginal delivery in group I and 30% in group II. The difference was statistically not significant. Babies delivered by Spontaneous or assisted vaginal delivery and babies who required prolonged resuscitation in the form of bag & mask or bag & tube ventilation developed HIE more often. When the antepartum and intrapartum risk factors were distributed in the three grades of HIE (HIE I, II, III) statistically significant risk factors were in spontaneous vaginal delivery/assisted vaginal delivery. Several studies,<sup>[29,30]</sup> found that normal vaginal delivery was frequent in asphyxiated patients. Incidence of caesarian section delivery in Aslam et al.,<sup>[30]</sup> was 51% among the asphyxiated group mothers and 63.3% in the control group mother. Timely intervention with caesarian section could have saved many of these unfortunate babies from being asphyxiated at birth. In this current study, it was seen that 73.3% of the patients' mother had regular ANC in group I compared to 65% in group II. The difference was insignificant. Multiparity were found 56.7% in group I and 65% in group II. Bhunia and Saharia,<sup>[29]</sup> showed primiparous had 69% in case group and 93% in control group, while,

multiparity were more common in case group. Shireen et al.,<sup>[31]</sup> obtained in their study that parity was found to be an important factor. Prolonged labor, prematurity and low birth weight, which are common in primiparous, might be the possible explanation. On demographic profile, in this study, it was observed that half of the patients came from lower income family in group I and 30% in group II. The difference was insignificant. HIE occurs at a rate of approx.1-2 /1000 full term live birth in high income countries, while, in low-income countries the incidence is much higher.<sup>[34]</sup> In this present study, 20% patients' mothers had HTN in group I. The difference was statistically insignificant between the two groups. Aslam et al.,<sup>[30]</sup> mentioned in their study that maternal hypertension and diabetes mellitus were not related to an increase risk of birth asphyxia. Similar observation also reported.<sup>[31]</sup> In this series, it was observed that 63.3% patients were meconium stained in group I and 25% in group II. 46.7% had prolong labor in group I and 20% in group II, 40% had PROM in both group I and II. Risk factors, like, obstructed labor, APH, PET, assisted breech and PROM were almost alike between the two groups. Bhunia and Saharia,<sup>[29]</sup> showed 2.3% mothers had history of obstructed labor in group I and 4.2% in group II. 21.8% had prolong labor in group I and 2.8% in group II. 32% had meconium-stained liquor in group I and 34% in group II. Prolong labor had 24.4% in group I and 9.4% in group II observed by Aslam et al.<sup>[30]</sup> The investigators also observed that 19.5% asphyxiated patients were meconium stained in case group . 33.3% mothers had prolonged ruptured membrane (PROM) in group I and 5.1% in group II. In another study,<sup>[31]</sup> reported that, 33% of case group had history of PROM



6.7% in group II. Prolonged 2<sup>nd</sup> stage of labor had 34% in group I and 3.3% in group II. antepartum hemorrhage (APH) were seen in only 9% in group I, which all are comparable with the current study. In this study, it was observed that 93.3% baby had delayed cry in group I and 100% in group II. 13.3% patients no respiration in group I and no such findings in group II. 76.7% had seizure in group I and no such in group II. The seizure difference was statistically significant ( $p < 0.05$ ) between two groups, which is similar to other studies.<sup>[29-35]</sup> However, subtle seizures were most common type of neonatal seizures in some paper.<sup>[29]</sup> In this study, it was observed that 50% patients had anemia in group I and 35% in group II, 40% had cyanosis in group I and 25% in group II. Bhunia and Saharia,<sup>[29]</sup> observed anemia commonly occurred in case group. Similarly, in another study <sup>[30]</sup> showed anemia 48% and 37.6% in group I and group II respectively, which are comparable with this study. Respiratory difficult was found in 70% in group I and 50% in group II in this study. In another study,<sup>[31]</sup> found respiratory distress was only 10% in their study patients, which differs with the present study. It was observed in this study, that 40% patients in group I had grunting and 25% in group II. 43.3% patients were found hypothermic in group I and 25% in group II. Weak primitive reflexes were found in 70% in group I and 35% in group II. Lethargy was present in 70% in group I and only 20% in group II. Pupil were constricted in 70% in group I. Hypotonia were found 66.7% in group I. 63.3% patients in group I found to have poor perfusion, while only 15% in group II. 70% patients had seizure in group I and no such findings in group II. Hypothermia, weak primitive reflexes, level of consciousness,

lethargy, constricted pupil, hypotonia, poor perfusion and seizure were significantly ( $p < 0.05$ ) higher in group I. EEG evidence of diffuse cerebral injury like burst suppression, multifocal sharp waves, dysmaturity, attenuation of background were commonly associated with HIE,<sup>[36-38]</sup> but the exact incidence of these findings has not been well established. In this study, it was observed that 66.6% patients had SET findings in group I. Other findings are depicted in the table. The difference was statistically significant ( $p < 0.05$ ). Jose et al.,<sup>[32]</sup> found 92.9% babies with burst suppression pattern were abnormal ( $p < 0.05$ ). In another study,<sup>[36]</sup> it was showed that out of the 52 who had normal EEG, 83% had a normal outcome at 1 year, 17% had mild abnormalities. In this current study, it was observed that 66.6% patients had longer hospital stay (8-14 days), in group I and 30% in group II. Discharged, 33.3% patients had abnormal apparent neurological sequelae in group I, while in group II, 100% discharged without any apparent neurological deficit. 80% patients discharged alive in group I. The hospital stay difference was statistically significant ( $p < 0.05$ ) between the two groups. Shireen et al.,<sup>[31]</sup> showed mortality among the asphyxiated babies was 16% during hospital stay, whereas no fatality was recorded among the control. Neurological sequelae were observed in 28% of asphyxiated babies but absent in controls. Average hospital stay was almost similar in both groups. 28% of the asphyxiated babies developed neurological sequelae and mortality was 16%. No neurological sequelae or mortality was seen in control group. In this study, it was observed that a subject with poor perfusion had 6.0 (95% CI 1.118 to 64.491) times increase in odds for having hypoxic ischemic encephalopathy. Poor



perfusion was significantly associated with hypoxic ischemic encephalopathy.

## CONCLUSIONS

This study was undertaken to observe the clinical profiles and EEG changes in patients with hypoxic ischemic encephalopathy and concluded that there is significant association with meconium stain, seizure, hypothermia, weak primitive reflexes, lethargy, miosis, hypotonic, poor APGAR score, burst suppression & SET findings in EEG and prolonged hospital stay in prenatal asphyxia with HIE. No significant change in others demographic and clinical profiles in prenatal asphyxia patients with HIE. A normal EEG in a

term newborn with HIE is associated with good neurological outcome. Burst suppression pattern in EEG is the predictors of abnormal outcome.

## Limitations of the Study

The study population was selected from one selected hospital in Dhaka city, so that the results of the study may not reflect the exact picture of the country. The present study was conducted at a very short period of time. Small sample size was also a limitation of the present study. Therefore, in future further study may be under taken with large sample size to overcome the limitation.

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