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CSF Electrolytes Changes in Hypoxemic Ischemic Encephalopathy

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ABSTRACT

Background: To find the relationship between degree of Hypoxaemic iscahemic encephalopathy (HIE) and levels of electrolytes (sodium, potassium and calcium) in cerebrospinal fluid, in a referral hospital located in semi urbarn area of East India. **Methods:** We conducted an observational study of HIE in Burdwan Medical College Hospital, over a one-year-period, from April 2013 to March 2014. All the inborn term babies without major congenital anomaly that developed HIE were included in the study. A total of 40 cases were included in the study taken serially. Babies were categorised in three groups according to the Sarnat and Sarnat staging of HIE. CSF electrolytes Data collected for for all babies in the 3 groups was analyzed and compared statistically. **Results:** The total number of live births in the study year were 21032. Out of this 361 newborns suffered from HIE. About 16.67% of these newborns expired during the study period. An overall incidence of hypoxic ischaemic encephalopathy (HIE) in Burdwan medical college and hospital was found to be 17.1 per 1000 live birth in the year of study. The CSF sodium values were found to be 138.67±2.02, 131.25±1.71, 123.38±3.83 meq/l respectively in 3 stages of HIE (P<0.001) and significantly low progessively. Similarly the obtained values of potassium for 3 stages of HIE were 2.82±0.17, 2.77±0.3, 2.27±0.46 meq/l (P<0.001) and that of calcium were 7.85±0.75, 6.56±0.25, 5.49±0.26 mg/dl in 3 stages of HIE (P<0.001). **Conclusion:** Measured values of CSF electrolytes fell significantly according to the severity of HIE.

Keywords: Hypoxaemic iscahemic encephalopathy, Serum Electrolytes.

INTRODUCTION

The incidence of Birth asphyxia is high in countries with limited resources. Birth asphyxia is the cause of 23% of all neonatal deaths worldwide. It is one of the top 20 leading causes of disease burden in all age groups (in terms of disability life adjusted years) by the World Health Organization and is the fifth largest cause of death of children younger than 5 years (8%).[1,2]

More than a million children who survive birth asphyxia develop problems such as cerebral palsy, mental retardation, learning difficulties, and other disabilities.[3,4]

The mechanism of cellular injury after hypoxia or ischemia is poorly understood, but is probably mediated by an excess release of neurotransmitters, and the initiation of lipid peroxidation which, in turn, leads to a cascade of damaging events.[5] At the cellular level, cerebral hypoxia–ischemia sets in motion a series of biochemical events commencing in a shift from oxidative to anaerobic metabolism; this leads to an accumulation of NADH, FADH, lactic acid and H+ ions.[6]

The commonly measured serum electrolytes like sodium, potassium, calcium, magnesium, chloride, phosphate, bicarbonate, can also found in normal CSF. In most cases, CSF levels are held constant across a wide range of serum level, because of strict underlying control mechanisms. However in some it may vary directly with plasma concentrations. For example, in some neurological disturbances, fluctuation of CSF electrolytes concentrations have been shown to occur.

CSF: Plasma concentration of sodium is about 0.93. In general sodium level in CSF vary directly with the serum sodium concentration. Unlike sodium CSF potassium levels are tightly maintained even in the setting of significant rise or fall in plasma concentration. The steady state CSF concentration occurs at about 2.9 meq/l.Since extracellular potassium both has depolarizing effects on neurons and can promote the swelling of astrocytes. Most available data suggest that CSF calcium levels are controlled by active transport processes. Indeed, they remain steady with systemic infusion of calcium salts or treatment with calcium chelators. CNS influx is also not influenced by serum levels.[7,8]

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A decrease in ATP affects the Na+, K+ -ATPase pump that helps to maintain the state of polarization of the neuronal membrane. A failure of this leads to an influx of sodium into the cell and potassium outside the cell. Associated glial uptake of sodium and water leads to astrocytic swelling.

MATERIALS AND METHODS

This observational study was carried out to find out the relationship between degree of Hypoxaemic iscahemic encephalopathy (HIE) and levels of electrolytes (sodium, potassium and calcium) in cerebrospinal fluid.
This was a hospital based study with a cross-sectional design among newborns suffering from HIE and admitted in Department of Pediatrics of Burdwan medical college and hospital during one year period from April 2013 to March 2014.

Study population:
All in born babies suffering from HIE, admitted in Department of Pediatrics of Burdwan medical college and hospital were eligible for inclusion.

Inclusion Criteria
1. Newborns suffering from birth asphyxia and subsequently developing hypoxic ischemic encephalopathy.
2. Gestational age between 36-41 weeks.
3. Neonate surviving >12 hours

Exclusion Criteria
1. Newborns with sepsis, major congenital malformations, hemolytic disease, birth trauma.
2. Newborns born to mother having major disease like malaria, diabetes mellitus, severe anemia.
3. Maternal intake of any drugs causing, sedation in newborn.

A total of 40 babies admitted in NICU, who met the inclusion criteria were included in the study taken serially.

Parameters studied
1. Level of Sodium in CSF.
2. Level of potassium in CSF.
4. 3 Level of calcium in CSF
3. History, physical examination, clinical examination of newborns & their mothers.

HIE staging was done by examining the baby regularly and noting the clinical features. Sarnat and Sarnat staging chart was used to determine the staging. Upon approval and ethical consent from the competent authority, parents of the babies were explained about the study and informed consent was taken. Demographic details were obtained and recorded. Cerebro Spinal Fluid (CSF) was collected from eligible newborns for the study after 12 hours. After that the collected CSF was taken to Department of Biochemistry for estimation of Sodium, potassium and calcium. All statistical analysis was done using SPSS version 20. To calculate the means and SD of all indices in respective stage of HIE were obtained by Microsoft Office Excel 2007 software and then put into formula. Comparison between three quantitative variables were done by one way ANOVA test and two variables by t test. Qualitative data was analysed by Chi square test.

RESULTS

Incidence of hypoxic ischemic encephalopathy
The incidence of HIE among newborns delivered in Burdwan medical college & hospital during the study period was 17.1 per 1000 live birth.
The overall case fatality rate during study period due to HIE was about 16.67%.

Table 1: Comparison of baseline data between groups.

Variables		HIE 1	HIE 2	HIE 3	Total	P -Value
Gestation		39 ±1.4	38.5 ±1.3	38.3 ± 1		P - 0.345
Birth weight		2.38 ± 0.34	2.75±0.29	2.85±0.48		P 0.786
Time of CSF		15.17±1.03	14.5±1.24	15±0.73		P-0.241
Sex	M	4(33.33)	7(58.33)	9(56.25)	20 (50)	
	F	8(66.67)	5(41.67)	7 (43.8)	20 (50)	
		12(30)	12(30)	16(40)	40(100)	P -0.38
Fate	Survived	12 (100)	10(83.33)	10(62.5)	32(80)	
	Expired	0(0)	2(16.67)	6(37.5)	8(20)	
	Total	12(30)	12(30)	16(40)	40(100)	P - 0.08
APGAR at 5 min	0	1 (8.4)	1(8.35)	2(12.5)	4(10)	
	1	2 (8.4)	4(33.33)	10(62.5)	16 (40)	
	2	4(33.33)	6 (50)	3(18.75)	13 (32.5)	
	3	5 (41.7)	1 (8.4)	1 (6.25)	7 (17.5)	
	Total	12(30)	12(30)	16(40)	40(100)	P 0.068

1. **Incidence of HIE**
Total numbers of live births during study period (Apr 2012 to Mar 2013) = 21032.
Total number of newborns suffering from HIE =361
Total number of deaths due to HIE=60

Incidence of HIE 1.71%
Case Fatality Rate: 16.67%

2. Comparison of the baseline data

Baseline data are shown in [Table 1]

No significant difference found between gestational ages among three groups (p value 0.345). The body weight in 3 groups was 2.83±0.34, 2.75±0.29 and 2.85±0.48 respectively with no significant difference (p value 0.786). Timing of collection of CSF also matched.

3. Sodium, Potassium and Calcium in CSF according to stage of hypoxic ischemic encephalopathy.

Sodium

The values were found to be 138.67±2.02, 131.25±1.71, 123.38±3.83 SODIUM meq/l of CSF respectively in 3 stages of HIE (P<0.001) and significantly low.

Table 2: CSF Sodium Level

CSF Sodium mEq/L	N	Mean±SD	P
HIE - I	12	138.67+2.02	P <0.001
HIE - II	12	131.25+1.71	
HIE -III	16	123.38+3.83	

Potassium

The obtained values for 3 stages of HIE being 2.82±0.17, 2.77±0.3, 2.27±0.46 meq/lof CSF respectively (P<0.001)

Table 3: CSF Potassium level

CSF Potassium mEq/L	N	Mean±SD	P
HIE - I	12	2.82+0.17	P <0.001
HIE - II	12	2.77+0.3	
HIE -III	16	2.27+0.46	

Calcium

The values were found to be 7.85±0.75, 6.56±0.25, 5.49±0.26 mg/dl of CSF respectively in 3 stages of HIE (P<0.001).

Table 4: CSF Calcium level

CSF Calcium mEq/L	N	Mean±SD	P
HIE - I	12	7.85+0.75	P <0.001
HIE - II	12	6.56+0.25	
HIE -III	16	5.49+0.26	

DISCUSSION

Despite the advancement in the antenatal monitoring and better infrastructure facility to facilitate safer delivery, the incidence of Hypoxaemic ischaemic encephalopathy continues to be high especially in the developing countries. More than a million children who survive birth asphyxia develop problems such as cerebral palsy, mental retardation, learning difficulties, and other disabilities.

Perinatal asphyxia is associated with intrapartum or postpartum hypoxia/ ischemia followed by reventilation and reperfusion during resuscitation. The role of free oxygen radicals has been implicated in such reventilation/reperfusion injuries. The pathogenesis of cell death following hypoxic ischemic injury in the developing brain is complex and incompletely understood. It is clear, however, that the hypoxic ischemic insult followed by resuscitation leads, not only to primary cellular injury during the period of insult, but to a delayed secondary injury 24-48 hrs later.

Incidence of hypoxic ischaemic encephalopathy (HIE) in Burdwan medical college and hospital was found to be 17.1 per 1000 live birth during the study period of one year

During study, the level of Sodium, Potassium, Calcium were studied in Cerebrospinal fluid after 12 hours of age (mean±SD for hour of collection being 15.17±1.03, 14.5±1.24, 15±0.73 hrs in HIE-I,II &III respectively, P=0.241). The difference in CSF level of Sodium was highly significant among the 3 stages of HIE with decrease in levels of Sodium with increase in severity of HIE. The values were found to be 138.67±2.02, 131.25±1.71, 123.38±3.83 SODIUM meq/l of CSF respectively in 3 stages of HIE (P<0.001). A similar data was also obtained for Potassium in Cerebrospinal fluid and was found to be highly correlating with severity of birth asphyxia (P<0.001). The obtained values for 3 stages of HIE being 2.82±0.17, 2.77±0.3, 2.27±0.46 POTASSIUM meq/l of CSF respectively. The difference in CSF level of Calcium was highly significant among the 3 stages of HIE with decrease in levels of Calcium with increase in severity of HIE. The values were found to be 7.85±0.75, 6.56±0.25, 5.49±0.26, Calcium, mg/dl of CSF respectively in 3 stages of HIE (p<0.001).

CONCLUSION

This type of studies must be conducted for better understanding of parameters like sodium, potassium and calcium levels after birth asphyxia so that electrolytes level in CSF with a cut off value, can be a useful marker and may serve as a guide for staging in HIE and also for assessing prognosis in time to come. This will also help in better understanding of the problem and will be helpful in development of specific and effective post insult therapy.

REFERENCES

1. Berger R, Garnier J. Perinatal brain injury. J Perinatol Med 2000; 28:261–285
2. Ellis M, Dharma M. Progress in Perinatal Asphyxia. Semin Neonatol 1999; 4:183– 91.
3. Bryce J, Boschi-Pinto C, Shibuya K, Black RE. WHO estimates of the causes of death in children. Lancet. Mar 26-Apr 1 2005; 365(9465):1147-52.
4. Lawn J, Shibuya K, Stein C. No cry at birth: global estimates of intrapartum stillbirths and intrapartum-related neonatal deaths. Bull World Health Organ. Jun 2005; 83(6):409- 17.

5. Harris ED. Regulation of antioxidant enzymes. FASEB J 1992; 6:2675–2683
6. Palmer C, Brucklalcher RM, Christensen MA et al. Carbohydrate and energy metabolism during the evolution of hypoxic-ischemic brain damage in the immature rat. J Cereb Blood Flow Metab 1990; 10:227–235.
7. Azzopardi.D.,et al. Prognosis of newborn infants with hypoxic ischemic brain injury assessed by phosphorus magnetic resonance spectroscopy. Pediatr Res., 1989.25(5):p.445-51.
8. Roth SC., et al., Relation of deranged neonatal cerebral oxidative metabolism with neurodevelopmental outcome and head circumference at 4 years. Dev Med Child Neurol.1997.39(11):p.718-25.

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