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ORIGINAL ARTICLE

STUDY OF ELECTROLYTE STATUS, GLUCOSE AND URIC ACID LEVELS IN PERINATAL ASPHYXIA

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ABSTRACT: OBJECTIVE: This is a hospital based, prospective clinical study on the electrolyte status, glucose & uric acid levels in asphyxiated newborns in the umbilical cord arterial blood. **METHODS:** The study was a prospective study conducted on asphyxiated and non-asphyxiated term neonates recruited from a Neonatal Intensive Care Unit. Cases and Controls comprised of asphyxiated and non-asphyxiated neonates, respectively. The cord blood arterial samples from the 50 neonates comprising the cases and 50 neonates comprising the controls constituted the material for the study. Maternal and neonatal risk factors were noted and data analyzed by statistical package SPSS-15. **RESULTS:** A significant correlation was detected between the levels of hyponatremia and the severity of HIE in the asphyxiated neonates. A significant correlation was also detected between the levels of hypocalcaemia and the severity of HIE in the asphyxiated neonate. Although serum values of potassium, uric acid and creatinine were found to be higher in asphyxiated neonates as compared to normal neonates their values were found to be in the higher range of normal. Serum glucose values did not have a significant correlation with mild to moderate severity of asphyxia. **CONCLUSION:** The umbilical cord arterial values of serum sodium and serum calcium was found to be a good, early, simple and reliable screening test for the early diagnosis and assessment of severity perinatal asphyxia and their values correlate well with the severity of HIE.

KEYWORDS: Perinatal asphyxia.

INTRODUCTION: Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. Globally, hypoxia of the newborn (birth asphyxia) or the fetus ("fresh stillbirth") is estimated to account for 23% of the 4 million neonatal deaths and 26% of the 3.2 million stillbirths each year[1] .Data from National Neonatal Perinatal database (NNPD) suggests that perinatal asphyxia contributes to almost 20% of neonatal deaths in India.[2]

In India, 8.4% of inborn babies have a one minute Apgar score less than 7 and 1.4% suffer from hypoxic ischemic encephalopathy (HIE).[2]

Only a third of deliveries in India are institutional[3] and many asphyxiated babies are brought late to hospitals. The signs of asphyxial injury are nonspecific and overlap with other illnesses. In the absence of perinatal records, it is difficult to retrospectively diagnose perinatal asphyxia

A variety of markers have been examined to identify perinatal hypoxia. Though there are more and more studies for understanding mechanisms leading to birth asphyxia, studies for early determination of tissue damages due to birth asphyxia are still lacking.

Hence this study was conducted to find out any correlation between the electrolyte status, calcium, glucose and uric acid levels in cord blood with the severity of asphyxia, so that problems could be anticipated early and appropriate measures are taken, so neonatal morbidity and mortality could be reduced.

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MATERIALS AND METHODS: The study was a prospective study conducted on asphyxiated and non-asphyxiated term neonates recruited from Neonatal Intensive Care Unit (NICU) Kempegowda Institute of Medical Sciences, Bangalore from December 2010 to May 2012. Cases and Controls comprised of asphyxiated and non-asphyxiated neonates, respectively. The cord blood arterial samples from the 50 neonates comprising the cases and 50 neonates comprising the controls constituted the material for the study.

The case group included 50 neonates fulfilling the following criteria Intrapartum signs of fetal distress, as indicated by non-reassuring NST on continuous electronic fetal monitoring and/ or by thick Meconium staining of the amniotic fluid, Apgar score of <7 at one minute of life, Resuscitation with >1 minute of positive pressure ventilation before stable spontaneous respiration, Profound metabolic or mixed acidemia (pH<7.00) in an umbilical artery blood sample.

The following babies were excluded from the study babies with congenital malformations, history of maternal drug addiction, Neonates born to mothers who would have received magnesium sulphate within 4 hours prior to delivery or opioids (pharmacological depression). Hemolytic disease of the newborn, Neonates born to mothers consuming alcohol, are smokers and on anti-epileptic drugs.

The control group included 50 term apparently healthy neonates appropriate for gestational age without signs of perinatal asphyxia as evidenced by normal fetal heart rate patterns, clear liquor and one minute Apgar score ≥7.

From all neonates included in the study the following data was collected: Detailed maternal history, assessment of intrauterine fetal well-being by continuous electronic fetal monitoring, meconium staining of amniotic fluid, birth events, Apgar score, sex of the baby and weight of the baby were recorded on the precoded proforma. Thorough clinical and neurological examination was done for all the neonates included in the study.

The asphyxiated neonates (case group) were monitored for seizures, hypotonia and HIE in the immediate neonatal period in the NICU. Grading system used to grade the severity of HIE was levene staging. The cases were also observed for other systemic effects of asphyxia. Cord blood sample were drawn in the labor room and sent for analysis of ABG, serum electrolytes, serum calcium, serum glucose, serum uric acid levels &serum creatinine levels.

STATISTICAL ANALYSIS: Descriptive and inferential statistical analysis has been carried out in the present study. Analysis of variance (ANOVA) has been used to find the significance of study parameters between three or more groups of patients. Chi-square/Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups

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RESULTS:

Bio chemical parameters	HIE				P value
	Stage 0	Stage 1	Stage 2	Stage 3	
Sodium (Na) MEQ/L	135.30±5.06	134.04±2.96	131.42±3.7	123.33±0.58	<0.001**
Potassium (k) MEQ/L	4.99±0.91	4.98±0.97	4.44±1.02	4.63±0.38	0.394
Chloride MEQ/L	102.40±4.58	100.32±4.46	100.42±4.44	98.67±2.08	0.507
Calcium mg/dl	9.31±0.64	9.48±0.90	8.76±0.41	8.33±0.32	0.012*
Uric acid mg/dl	5.49±0.93	5.57±1.04	5.37±1.19	6.03±0.12	0.787
Glucose mg/dl	64.80±11.81	91.92±55.31	51.92±17.32	44.00±1.73	0.025*
Creatinine mg/dl	0.72±0.15	0.75±0.22	0.7±0.14	0.70±0.10	0.891

Table 1: Comparison of bio chemical parameters in three stages of HIE of patients studied

DISCUSSION: Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. Birth asphyxia is a common and important cause of preventable cerebral injury occurring in the neonatal period .The value of present biochemical markers for diagnosing asphyxia is inadequate and controversial.[4]

There are only a few studies that compare the umbilical arterial blood values of sodium, potassium, glucose, uric acid, calcium creatinine with different severity of asphyxia and different stages of HIE.

A few studies conducted who have followed up the infants over a period of time have found out that long term neurological outcome of these infants correlate well with the severity of asphyxia and with the severity of various biochemical alterations like hyponatremia, hypoglycemia, hypocalcemia, hyperuricemia and elevated creatinine values.

In the present study an attempt has been made to evaluate the incidence of various biochemical alterations in birth asphyxia and to find out any correlation between the severity of asphyxia and the severity of various biochemical alterations, so that with early detection of these changes in the umbilical cord blood interventions could be instituted at the earliest so as to prevent early neonatal brain injury so that the child can have a normal neurological outcome and can lead a productive life useful to the society.

Although several studies on birth asphyxia have been conducted most of them are on early neonatal blood and not on cord arterial blood, this has provided us with a unique opportunity to compare the results of our study with studies on early neonatal blood and to corroborate the findings.

The results of the present study were in concordance with those of Pallab Basu and Herendranath das[5] who reported that serum sodium levels were significantly lower in asphyxiated neonates compared to non-asphyxiate neonates and serum levels of potassium was slightly higher in asphyxiated infants compared to non-asphyxiated infants, also serum calcium levels were significantly lower in asphyxiated neonates as compared to non-asphyxiated neonates.

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Our results are in agreement with Gupta[6] and colleagues who found that serum creatinine values were higher in asphyxiated infants compared to non-asphyxiated infants As per W. W. Payne and P.T.Acharya[7] in their study the effect of abnormal birth on blood chemistry during the first 48 hours of life, the serum potassium shows no change in the first 12 hours, it rises in the later period (18-48 hours).

In contrast the serum sodium in the stressfully born infants is lower. Sugar, after the first 3 hours, falls to lower levels but by the end of 48 hours there is no material difference in stressed and nonstressed groups. This finding is in concordance with our study regarding the serum sodium values, but we also found higher levels of potassium in cord blood though it was within normal range in asphyxiated neonates as compared to non-asphyxiated neonates.

Ilves p in his study, studied serum total magnesium and ionized calcium concentrations in asphyxiated term newborn infants with hypoxic ischemic encephalopathy and concluded that there was significant hypermagnesemia, hypocalcaemia and hyponatremia in asphyxiated newborns as compared to nonasphyxiated newborns.[8]

Deepak jajoo[9] and colleagues came to a conclusion that serum calcium levels were significantly lower in term appropriate for gestational age infants who had history of birth asphyxia, they were of the opinion that hypoxia impairs the functions of parathyroid gland resulting in lower calcium levels.

Alphonsus in his study showed that serum calcium levels at 12 hrs. were significantly lower in asphyxiated neonates as compared to non-asphyxiated neonates, this conclusion is in concordance with our study where the serum calcium levels were found to be significantly lower in cord arterial blood in asphyxiated neonates when compared to non- asphyxiated neonates.[10]

CONCLUSION: Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. The signs of asphyxial injury are nonspecific and overlap with other illnesses. In the absence of perinatal records, it is difficult to retrospectively diagnose perinatal asphyxia.

Infants with asphyxia have lower sodium concentration in the umbilical cord arterial blood, and lower calcium concentration, also umbilical cord arterial potassium though found to be normal range but their values are in the higher range of normalcy, serum uric acid and creatinine is found to be in the higher in asphyxiated neonates but the finding was not statistically significant, glucose levels were found to low in severely asphyxiated neonates but were within normal range in mild and moderately asphyxiated neonates .umbilical cord arterial values of sodium and calcium might be used as an indicator for assessment of severity of birth asphyxia and severity of HIE in neonates.

There is a need to identify neonates who will be at high risk for HIE and early neonatal death as a consequence of perinatal hypoxia. estimation of umbilical cord arterial serum sodium and serum calcium concentration is an easy and affordable test and at the same time early biochemical marker of birth asphyxia which biochemically supports the clinical diagnosis and severity grading of asphyxia by Apgar score and correlates well with the severity of HIE.

The predictive factors identified in this study should be examined for their ability in a larger sample population before these markers can be applied on routine basis in a clinical scenario in infants with perinatal asphyxia.

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REFERENCES:

1. Lawn JE, Cousens S, Zupan J. Lancet Neonatal Survival Steering Team. 4 million neonatal deaths: When? Where? Why? Lancet 2005; 365 (9462):891-900.
2. NNPD network. National Neonatal Perinatal Database–report for the year 2002-2003. NNF NNPD network. New Delhi: 2005.
3. Indian Institute of Population Studies. National Family Health Survey (NFHS-2) 1998-99. Mumbai: 2000.
4. Tekgul H, Yalaz M, Kutukculer N et al. Value of biochemical markers for outcome in term infants with asphyxia. Pediatr Neurol. 2004; 31 (5): 326-32
5. Pallab Basu, Sabbasachi Som, Nabendu Choudhuri, Harcndranath Das. Contribution of the blood glucose level in perinatal asphyxia. Eur J Pediatr (2009) 168:833-838
6. B.D. Gupta, Pramod Sharma, Jyoti Bagla, Manish Parakh, J.P. Soni. Renal failure in asphyxiated neonates. Indian Pediatrics 2005; 42:928-934
7. W. W. Payne, P. T. Acharya. The effect of abnormal birth on blood Chemistry during the first 48 hours of life. Arch Dis Childh, 1965, 40, 436.
8. Ilves P, Kiisk M et al. Serum total magnesium and ionized um concentration in asphyxiated term newborn infants with hypoxic ischemic encephalopathy. Acta Pediatr 2000;89: 680-685
9. Deepak Jajoo, Ashok Kumar, R. Shankar, V. Bhargava. Effect of Birth Asphyxia on Serum Calcium Levels in Neonates. Indian J Pediatr 1995; 62: 455-459
10. Alphonsus N. Onyiriuka. Prevalence of neonatal hypocalcaemia among full-term infants with severe birth asphyxia. Pacific Journal of Medical Sciences: Vol. 8, No. 1, April 2011

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