



A study of serum electrolytes level in newborns with birth asphyxia

Dr. Joshita Gupta¹, Dr. Bhagyashri Bora²

¹ junior resident, pravara institute of Medical Sciences, loni, Maharashtra

² professor, pravara institute of Medical Sciences, loni, Maharashtra

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ABSTRACT:Background: Birth asphyxia is defined as the presence of hypoxia, hypercapnia, and acidosis which leads the newborn to systemic disturbances probably electrolyte disturbances. Information regarding these electrolyte disturbances is very valuable as it can be an important parameter which affects perinatal morbidity, mortality and ongoing management.

Methods: Birth asphyxia was diagnosed by APGAR score and hypoxic ischemic encephalopathy was diagnosed by SARNAT staging. All the newborn were evaluated for Serum Electrolytes from blood sample.

Result: The mean values of sodium for mild, moderate and severe asphyxia were 136.72, 130.7 and 127.15 meq/l respectively. The values of potassium for mild, moderate and severe asphyxia were 4.66, 5.83 and 6.75 meq/l respectively. Similarly, the mean values of ionized calcium for mild, moderate and severe asphyxia were 2.07, 1.12 and 0.99 mmol/l respectively. The values of sodium and potassium among different severity of asphyxia were significantly different (p -value < 0.001). Significant positive correlation was found between serum sodium and Apgar score at 5 min. significant negative correlation was present between serum potassium and Apgar score at 5 min.

Conclusion: The severity of birth asphyxia is directly proportional to the degree of hyponatremia and hyperkalemia. So these disturbances should always be kept in mind while managing cases of perinatal asphyxia and should be managed accordingly.

Keywords: Asphyxia, Hyponatremia, Hyperkalemia, Hypocalcaemia

I. INTRODUCTION

In basic term birth asphyxia is delay in establishing spontaneous respiration upon delivery of a newborn^[1]. More precisely, birth asphyxia is defined as the presence of hypoxia, hypercapnia, and acidosis leading to systemic disturbances in the newborn^[2]. As per AAP (American Academy of Pediatrics) and ACOG (American College of Obstetrics and Gynecology), all the following must

be present for designation of asphyxia such as, profound metabolic or mixed acidemia ($pH < 7$) in cord, Persistence of APGAR scores 0–3 for longer than 5 min, neonatal neurological sequel (eg: seizures, coma, hypotonia) and multiple organ involvement (kidney, lungs, liver, heart, intestine)^[3]. It is a common problem with the incidence varying from 0.5–2% of live births^[4]. Some report the incidence from 1 to 8 per 1000 live births^[5]

The National Neonatal Perinatal Database (NNPD) 2000 used a similar definition for perinatal asphyxia. It defined moderate asphyxia as slow gasping breathing or an Apgar score of 4–6 at 1 minute of age and severe asphyxia was defined as no breathing or an Apgar score of 0–3 at 1 minute of age.

Early recognition of renal injury is important for maintenance of fluid and electrolyte homeostasis. Renal failure has also been found to correlate with mortality and long term neurological outcome of asphyxiated babies^[5,6]

Normally hypernatremia is expected in the early neonatal period as there is contraction of extracellular fluid due to excretion of water through kidney and high insensible water loss whereas in neonates with perinatal asphyxia there might be hyponatremia as there is increased secretion of anti-diuretic hormone (ADH) in neonates with HIE which leads to increased water retention and hence dilutional hyponatremia^[7]. The other reason for hyponatremia is that the capacity of sodium reabsorption is limited and if the load of sodium reaching the Collecting Tubules (CT) increases significantly, reabsorption does not occur proportionately and the sodium load is excreted in the urine^[8]. Other contributing factors to hyponatremia are partial resistance to aldosterone^[9].

Hyperkalemia is present in early neonatal period due to shift of potassium from the intracellular to extracellular space. The magnitude of this shift roughly correlates with the degree of immaturity i.e. the more premature the baby the more chance of hyperkalemia^[10]. Serum potassium subsequently falls as this internal potassium “load” is excreted by the kidneys^[11]. The rise in level of



serum potassium can be explained from the fact that birth asphyxia is associated with acidosis, and in metabolic acidosis, more than one half of the excess hydrogen ions are buffered in the cells. In this scenario, electro neutrality is maintained in part by the movement of intracellular potassium into the extracellular fluid. It can be due to acute renal failure secondary to birth asphyxia which leads to decreased excretion of potassium and hence hyperkalemia.

In normal newborn total calcium concentration in cord plasma increases with increasing gestational age and is significantly higher than paired maternal values^[12, 13]. With the abrupt termination of calcium transport across the placenta at delivery, plasma calcium falls, reaching a nadir at age 24–48 h^[12].

In a case control study by Basu P et al. among asphyxiated newborn, hyponatremia and hypocalcaemia developed early and simultaneously and the decrease in their serum levels was directly proportional to each other and to the degree of asphyxia among cases^[14]. Similarly in a prospective study done by Shah G S et al. among asphyxiated neonates, hyponatremia and hypocalcaemia was noted respectively as 23.3 and 11.7%^[15]. But in a case control study by Varma V et al. among asphyxiated newborns, mean values of electrolytes showed no significant difference among cases and controls as well as in HIE stages^[16]. So there is limited information regarding electrolyte disturbance in asphyxiated newborn especially the correlation with severity of asphyxia. Hence based on above findings the present study was planned for evaluation of serum electrolytes level in newborns with birth asphyxia.

II. MATERIAL AND METHODS

This was a retrospective conducted in the Department of Pediatrics, Pravara Institute Of Medical Sciences, India. Total 160 Newborn out of which 80 were study group and 80 were control group were included for the study.

Inclusion Criteria

- Term (37-41 weeks), appropriate for gestational age (inborn and outborn)
- Outborn with history of birth asphyxia (delayed cry of 5-10 min) and inborn with apgar score at 1 min (less than7)

Exclusion Criteria

- Babies with congenital cardio pulmonary malformation
- Mother with diabetes mellitus and hypertension treated with diuretics,
- Any apparent major congenital abnormality
- Preterm babies

III. METHODOLOGY

This was a hospital based retrospective observational study. Apgar score at 1 and 5 min was taken after birth and cases were selected applying inclusion and exclusion criteria. Detailed antenatal, natal and postnatal history and clinical examination were taken and findings were recorded on predesigned proforma by retrieving informed assent from parents. Relevant investigation as per protocol of PIMS were collected example-Complete Blood Count (CBC), Hematocrit, electrolytes (sodium, potassium, calcium), urea, creatinine, septic screen Total Leucocyte Count (TLC), absolute neutrophil count (ANC), band cell ratio, Micro Erythrocyte Sedimentation Rate (micro ESR), C-Reactive Protein (CRP) which was sent from venous sampling within one hour of birth.

IV. RESULTS

This study interprets the association of electrolytes imbalance with severity of asphyxia Patients were classified according to Levene staging to grade the severity of HIE (Table 1).The serum sodium value was 134.5+3.42 mEq/L among the cases as compared to controls had 139.4+4.12 mEq/L.The mean serum potassium value was 5.5+0.84 mEq/L among the cases as compared to controls had 4.6+0.32 mEq/L. Serum potassium was found higher in study group as compared to control and the difference between both the groups was statistically significant. The mean serum calcium value among the cases was 7.7+0.81 mg/dl as compared to controls had 8.8+0.24 mg/dl. The serum calcium level was found lower among cases as compared to control population and the differences between the groups were statistically significant. (table 2)

**Table 1** A clinical grading system for hypoxic ischemic encephalopathy by LEVENE stage

Features	Mild	moderate	severe
Consciousness	Irritable	Lethargy	Comatose
Tone	Hypotonia	Marked hypotonia	Severe hypotonia
Seizures	No	Yes	Prolonged
Sucking/Respiration	Poor suck	Unable to suck	Unable to sustain spontaneous respiration

Table 2: Serum electrolytes level among cases and controls

	Group	Mean (mEq/L)	Std. Deviation	P value
Serum sodium	Study group	134.5	3.42	P<0.0001
	Controls	139.4	4.12	
Serum potassium	Study group	5.5	0.84	P<0.0001
	Controls	4.6	0.32	
Serum calcium	Study group	7.7	0.81	P<0.0001
	Controls	8.8	0.24	

V. DISCUSSION

Asphyxia can have serious impact on various organ systems. Perinatal hypoxia contributes significantly to neonatal mortality and morbidity. HIE is the major end result of perinatal asphyxia. In asphyxiated newborn most of the organ can be affected but the kidneys, myocardium, brain and bowels appear to be more sensitive to HIE. Kidneys are involved in 50%, brain involved in 28%, heart in 25% and lungs in 23% of cases.^[17]

This study interprets the association of electrolytes imbalance with severity of asphyxia. The treatment of hyponatremia in such conditions is by fluid restriction rather than increasing sodium load. So fluid should be restricted in cases of birth asphyxia till normalization of serum sodium with close monitoring of weight and serum sodium. Serum potassium and Electrocardiography (ECG) monitoring has been done to avoid the deadly complications of hyperkalemia. Apart from other treatment measures, correction of acidosis and usage of potassium free fluid are the most useful measures to correct hyperkalemia. For hypocalcaemia associated with birth asphyxia, regular supplementation and monitoring of serum calcium should be done.

VI. CONCLUSION

Hyponatremia, hypocalcaemia and hyperkalemia occur in neonates with birth asphyxia which may cause increased morbidity and mortality. Severe hyponatremia should be suspected if there is GRADE 3 birth asphyxia and vice versa. Hence its level should be regularly monitored to prevent the problems associated with it. Severe hyperkalemia is associated with increasing severity of birth asphyxia and vice versa; so regular potassium monitoring and ECG monitoring is required to detect cardiac changes associated with it so that prompt management can be instituted. This study finds out that monitoring of serum electrolytes helps in the early diagnosis and management of birth asphyxia related complications.

REFERENCES

- [1]. Airede AI, Weerasinghe HD. Birth asphyxia: a review. East Afr Med J. 1995; 72(4):252–7.
- [2]. Fernández-Carrocera LA, Flores-Tamez E, Salinas-Ramírez V, Bravo-Cabrera Z, Venta-Sobero JA, Udaeta-Mora E, Ugartechea JC, Lozano-González CH. The Apgar score as a predictor of neurologic



- sequellae. *Bol Med Hosp Infantil Mex.* 1989;46(8):554–8.
- [3]. Perlman JM, Risser R. Can asphyxiated infants at risk for neonatal seizures be rapidly identified by current high-risk markers? *Pediatrics.* 1996;97(4):456–62.
- [4]. Rowe RD, Hoffman T. Transient myocardial ischemia of the newborn infant: a form of severe cardiorespiratory distress in full-term infants. *J Pediatr*
- [5]. Perlman JM, Tack ED. Renal injury in the asphyxiated newborn infant: Relationship to neurologic outcome. *J Pediatr* 1988; 113: 875– 79.
- [6]. Misra PK, Kumar A, Natu SM, Kapoor RK, Srivastava KL, Das K. Renal failure in symptomatic perinatal asphyxia. *Indian Pediatr* 1991; 28: 1147–51. 11
- [7]. Bauer K, Versmold H. Postnatal weight loss in preterm neonates < 1500 g is due to isotonic dehydration of the extracellular volume. *Acta Paediatr.* 1989; 78(s360):37–42
- [8]. Wu PY, Hodgman JE. Insensible water loss in preterm infants: changes with postnatal development and non-ionizing radiant energy. *Pediatrics.* 1974; 54(6):704–12.
- [9]. Shaffer SG, Meade VM. Sodium balance and extracellular volume regulation in very low birth weight infants. *J Pediatr.* 1989;115(2):285–90.
- [10]. Sato K, Kondo T, Iwao H, Honda S, Ueda K. Internal potassium shift in premature infants: cause of nonoliguric hyperkalemia. *J Pediatr.* 1995;126(1): 109–13.
- [11]. Lorenz JM, Kleinman LI, Kotagal UR, Reller MD. Water balance in very low-birth-weight infants: relationship to water and sodium intake and effect on outcome. *J Pediatr.* 1982;101(3):423–32.
- [12]. Tsang RC, Chen IW, Friedman MA, Chen I. Neonatal parathyroid function: role of gestational age and postnatal age. *J Pediatr.* 1973;83(5):728–38.
- [13]. Tsang RC, Kleinman LI, Sutherland JM, Light IJ. Hypocalcemia in infants of diabetic mothers: studies in calcium, phosphorus, and magnesium
- [14]. Basu P, Das H, Choudhuri N. Electrolyte status in birth asphyxia. *Indian J Pediatr.* 2010;77(3):259–62.
- [15]. Shah GS, Agrawal J, Mishra OP, Chalise S. Clinico-biochemical profile of neonates with birth asphyxia in eastern Nepal. *J Nepal Paediatric Soc.* 2013; 32(3):206–9.
- [16]. Vandana V, Amit V, Meena V, Anuradha B, Vivek B, Deepak V, Salone MR. Study of basic biochemical and haematological parameters in perinatal asphyxia and its correlation with hypoxic ischemic encephalopathy staging. *J Adv Res Biol Sci.* 2011; 3(2):79–85.
- [17]. Roberto Antonucci, Annalisa Porcella, Maria Dolores Pilloni. Perinatal asphyxia in the term newborn. *J Pediatric Neonatal Individualized Med* 2014; 3(2):e030269.