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To Study the Correlation Between Perinatal Asphyxia and Electrolyte Imbalance

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ABSTRACT

Aim: To investigate electrolyte (sodium, potassium, and calcium) imbalances in babies who have suffered various degrees of hypoxia in the early neonatal period. To determine the relationship between sodium, potassium, and calcium levels and the various degrees of perinatal hypoxia.

Methodology: This prospective observational study was placed in a hospital. The sample was collected using the consecutive sampling method. Apgar scores were recorded at 1 and 5 minutes after birth, and patients were chosen using inclusion and exclusion criteria. After obtaining informed consent from the parents, a thorough antenatal, natal, and postnatal history and clinical examination were performed. Results were recorded on a predesigned form. Complete blood count (CBC), haemoglobin, electrolytes (sodium, potassium, calcium), urea, creatinine, septic screen total leukocyte count (TLC), absolute neutrophil count (ANC), band cell ratio, micro erythrocyte sedimentation rate (microESR), and C-Reactive Protein (CRP) were sent from venous sampling within an hour of birth as relevant investigations.

Result: There was a significant difference in the means of sodium, potassium, and ionised calcium when they were analysed using an ANOVA, with a p-value of 0.001. (Table4). Bivariate analysis was used to identify the relationship between the Apgar score and blood electrolyte level, and the Pearson test was used to calculate the correlation coefficient. A box plot was used to show the median and quartiles of the serum electrolyte in relation to the Apgar at 5 minutes. With a Pearson correlation coefficient of 0.448, serum sodium increased along with the Apgar score at 5 minutes, demonstrating a substantial relationship between the two variables (Fig.1).

The link between serum potassium and Apgar at 5 minutes was significantly negative with a Pearson correlation coefficient of 0.422. Serum urea and creatinine increased proportionately with an increase in the severity of HIE grade.

Conclusion: One of the main issues with perinatal hypoxia is electrolyte imbalance, which is assumed to be increasing death and morbidity. The most common anomaly associated with birth asphyxia is hyponatremia, which requires precise and meticulous therapy. Hyperkalemia is equally important and needs to be handled carefully. Similarly, serum urea and creatinine levels were significantly different between HIE I and HIE III. However, there was a modest linear negative correlation with hypocalcemia, which is not statistically significant.

Keywords: Perinatal hypoxia, electrolyte imbalance, hyponatremia, Hyperkalemia hypocalcemia

INTRODUCTION

In its most fundamental sense, birth asphyxia refers to a delay in the newborn's ability to begin breathing on their own [1]. Birth asphyxia is more specifically described as the combination of hypoxia,

hypercapnia, and acidosis that affects the baby systemically [2]. According to the American Academy of Pediatrics and the American College of Obstetrics and Gynecology, all of the following conditions must exist in order to diagnose asphyxia, including profound metabolic or mixed acidemia (pH 7) in the cord, the persistence of APGAR scores 0-3 for more than five minutes, and neonatal neurological sequelae (e.g.: seizures, coma, hypotonia) as well as numerous organ involvement (liver, heart, kidney, lungs, and gut) [3]. With an incidence ranging from 0.5% to 2% of live births, it is a widespread issue [4]. The incidence has been reported to range from 1 to 8 per 1000 live births [5]. Normal early neonatal hypernatremia is anticipated due to extracellular fluid contraction from water excretion through the kidney and high insensible water loss, whereas neonates with perinatal asphyxia may experience hyponatremia due to increased anti-diuretic hormone (ADH) secretion, which causes increased water retention and, ultimately, dilutional hyponatremia [6]. The ability of sodium reabsorption is also restricted, and if the amount of sodium reaching the collecting tubules (CT) increases sufficiently, reabsorption does not occur.

Sodium load is eliminated in the urine when it does not occur proportionally [7]. Aldosterone partial resistance is one of the causes of hyponatremia [8]. Early neonatal hyperkalemia in infants is caused by potassium moving from the intracellular to extracellular space. The degree of immaturity and the size of this shift are roughly inversely correlated, meaning that the more preterm the newborn, the higher the risk of hyperkalemia [9]. Following the kidneys' excretion of this internal potassium "load," serum potassium decreases [10]. While metabolic acidosis is linked to acidosis, and in metabolic acidosis, more than half of the excess hydrogen ions are buffered in the cells, the rise in serum potassium level can be explained by the fact that birth asphyxia is connected with acidosis. In this situation, the flow of intracellular potassium into the extracellular fluid helps to preserve electrical neutrality. Acute renal failure leading to birth asphyxia, which results in reduced potassium excretion and, consequently, hyperkalemia, is another possible cause.

With increasing gestational age, the total calcium concentration in cord plasma of healthy newborns rises and is considerably higher than the paired maternal values. Plasma calcium

decreases after birth as calcium transport across the placenta abruptly stops, reaching a low in 24-48 hours [11]. After birth, there is a rise in serum parathyroid hormone (PTH) as a result of the decrease in plasma calcium levels. Even in the absence of exogenous calcium intake, this increase in PTH mobilises calcium from bone, causing a rise in plasma calcium concentration and subsequent stabilisation. In babies who have suffered suffocation, hypocalcaemia is clinically significant [12]. This is caused by a slow response in PTH secretion to the postnatal drop in plasma calcium concentration. A newborn that experiences asphyxia experiences a number of clinical and biochemical [13,14] changes that may have a negative impact on the prognosis [15]. Correcting the electrolyte disturbance is more beneficial than taking anticonvulsants to treat hyponatremic seizures [16]. Death and heart dysfunction are linked by hyperkalemia. Seizures, heart malfunction, and jitteriness are all linked to hypocalcaemia.

Furthermore, the severity of birth asphyxia may affect the degree of electrolyte imbalance. Hyponatremia and hypocalcaemia emerged early and simultaneously in an asphyxiated newborn population in a case-control research by Basu P et al., and the decline in their blood levels was directly proportional to both each other and the severity of asphyxia in the cases [17]. Similarly, hyponatremia and hypocalcaemia were found to be 23.3 and 11.7%, respectively, in a prospective investigation conducted by Shah G S et al. among asphyxiated neonates [18].

However, in a case-control research conducted by Varma V et al. on asphyxiated babies, mean electrolyte values did not significantly change between cases and controls or across phases of HIE [19]. This study was initiated in order to better understand electrolyte changes in asphyxiated babies of various severity throughout the early neonatal period and determine whether levels of sodium, potassium, calcium, urea and creatinine are correlated with the severity of prenatal asphyxia.

METHODS

A total of 88 cases of asphyxiated newborns born at this institute were included in this prospective observational study, which was carried out in a tertiary care hospital.

Inclusion Criteria

Birth asphyxia, defined by the World Health Organization as "failure to initiate and sustain breathing at birth," and based on Apgar score as an Apgar score of 7 at 5 minutes of life even after receiving resuscitation in accordance with Neonatal Resuscitation Program (NRP) guidelines were included in the study. Term newborns born and admitted at the tertiary care and appropriate for gestational age (those babies falling between 10th to 90th percentile of weight for their gestation) were included.

Exclusion Criteria

The study excluded children born preterm, those with IUGR (intrauterine growth retardation), gross congenital malformations, suspected metabolic diseases, cases receiving medications other than vitamin K prior to blood sample collection, children whose mothers had diabetes mellitus,
mothers who were taking antiepileptic drugs, and mothers who had suspected or confirmed electrolyte abnormalities. The study also eliminated children whose mothers had used diuretics, general anaesthesia, phenobarbital, pethidine, magnesium sulphate, antihypertensive medications, and other substances that might have affected their mothers' electrolyte balance or caused depression in the child.

Electrolyte estimation

Serum electrolytes (sodium, potassium, and calcium) were examined using an automated system and an ion-selective electrode.

HIE staging

Patients were categorised based on Leven staging to assess the severity of HIE (Table1). Due to the lack of such a facility at our institution, no cases were treated with therapeutic hypothermia; instead, cases were managed in accordance with the institutional protocol. Ionized sodium, potassium, and calcium levels considered normal were 130-145 meq/l, 3.7-5.9 meq/l, and 1–1.5 mmol/l, respectively [20].

Table 1: A clinical grading system for HIE by LEVENE stage

Feature	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

STATISTICAL ANALYSIS

Data visualisation was done using descriptive statistics. The statistical data analysis was carried out using SPSS version 20 (Statistical Package for the Social Sciences, SPSS Inc.). ANOVA test for parametric data was used to statistically compare the means of various electrolytes with varying degrees of foetal asphyxia, and significant results were further investigated using post hoc test. Bivariate analysis was done to determine the correlation between APGAR score at 5 mins and serum electrolytes. Pearson test was used to calculate the correlation coefficient. A boxplot was utilised to display the median

and quartile levels of serum electrolytes in relation to the five-minute Apgar score.

RESULTS

Out of the 88 cases who were registered, 60 (68%) were male and 28 (32%) were female, with a mean weight of 2975.45 g. (Tables2 and 3). When the means of sodium, potassium, and ionised calcium were compared using an ANOVA, there was a significant difference between them with a p-value of 0.001. (Table4). The association between the Apgar score and serum electrolyte level was discovered using bivariate analysis.

Table 2: Electrolyte status according to different stages of HIE

Levene	No.	Hyponatremia	Hyperkalemia	Hypocalcaemia
Consciousness	Irritable	Lethargy	Comatose	7 (28%)
Tone	Hypotonia	Marked Hypotonia	Severe Hypotonia	7 (23%)
Seizures	No	Yes	Prolonged	15 (45.4%)

Table 3: Electrolyte status according to different stages of HIE-1

Electrolyte	LEVENE	No.	Mean	Std. Deviation
Sodium	Mild	25	132.52	4.51
	Moderate	30	130.7	2.58
	Severe	33	127.15	2.26
	Total	88	130.73	4.60
Potassium	Mild	25	4.96	0.73
	Moderate	30	5.93	0.55
	Severe	33	6.78	0.87
	Total	88	5.98	1.03
Calcium	Mild	25	1.07	0.14
	Moderate	30	1.12	0.13
	Severe	33	0.99	0.12
	Total	88	1.05	0.14

Table 4: Mean values of biochemical parameters in differentstages of hypoxic-ischemic encephalopathy

Parameters	HIE-I	HIE-II	HIE III	HIE I vs HIE II P-value	HIE I vs HIE III P-value	HIE II vs HIE II P-value
Serum Na^+ (mmol/l) ± SD	137.5±3.8	132.7±6.8	124.4±4.4	< 0.01	< 0.01	< 0.01
Serum K^+ (mmol/l) ± SD	5.00±0.79	5.55±0.77	6.17±0.89	< 0.05	< 0.01	< 0.01
Serum Ca^{+2} (mmol/l) ± SD	1.06+ 0.17	0.86 + 0.15	0.83+ 0.09	<0.01	<0.001	>0.05
Serum urea (mg/dl) ± SD	26.75± 8.8	47.98± 24.7	89.38± 28.6	<0.01	<0.001	< 0.01
Serum creatinine (mg/dl) ± SD	0.70±0.16	0.94±0.54	2.41±0.89	>0.05 (NS)	< 0.01	< 0.01

The median and quartiles of the serum electrolyte with respect to the Apgar at 5 minutes were displayed using a box plot. As the Apgar score at 5 minutes climbed, serum sodium likewise increased with a Pearson correlation coefficient of 0.448, indicating a significant association between the two variables (Fig.1). With a Pearson correlation coefficient of 0.422, there was a significant negative connection between serum potassium and Apgar at 5 minutes with a p-value of 0.001. (Fig.2). The change in the value of the Apgar score at 5 minutes had no correlation with the change in serum ionised calcium level, and the Pearson correlation coefficient was 0.479.

Therefore, there was no significant correlation between the two variables (Fig.3 and Fig.4).

Fig. 1 Box pot showing median and quartile of serum sodium at 5 min Apgar.

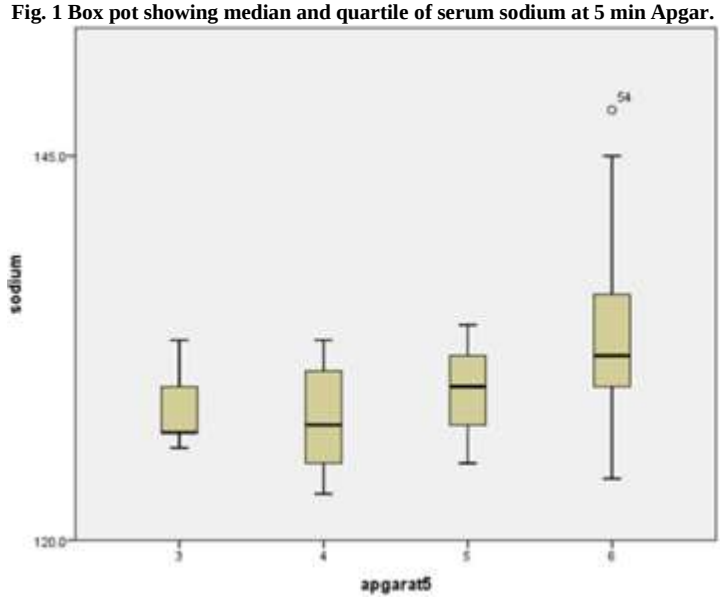


Fig. 2 Box plot showing median and quartiles of serum potassium at 5 min Apgar

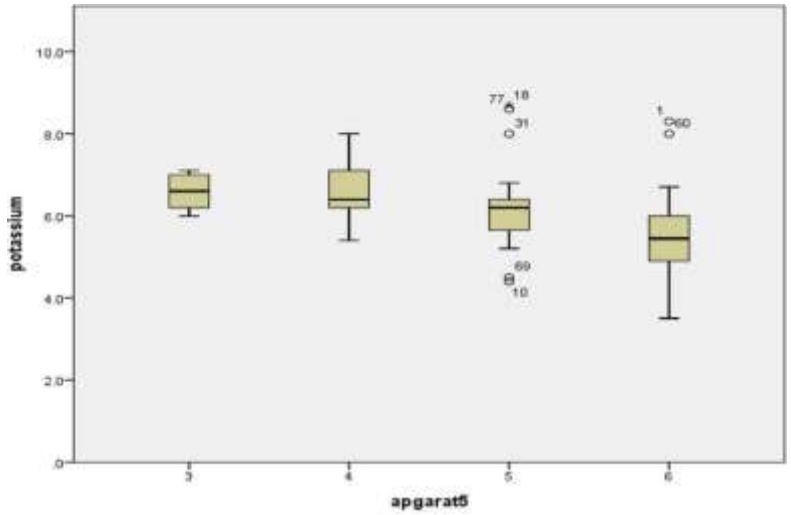


Fig. 3 Box plot showing median and quartiles of serum ionised calcium at 5 min Apgar

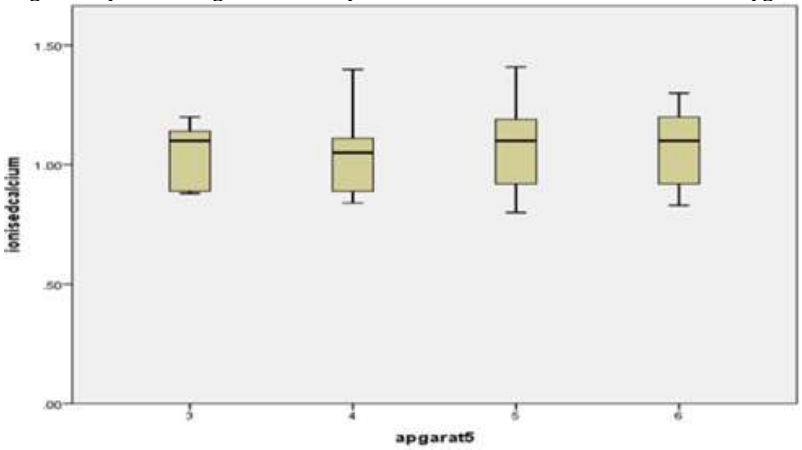
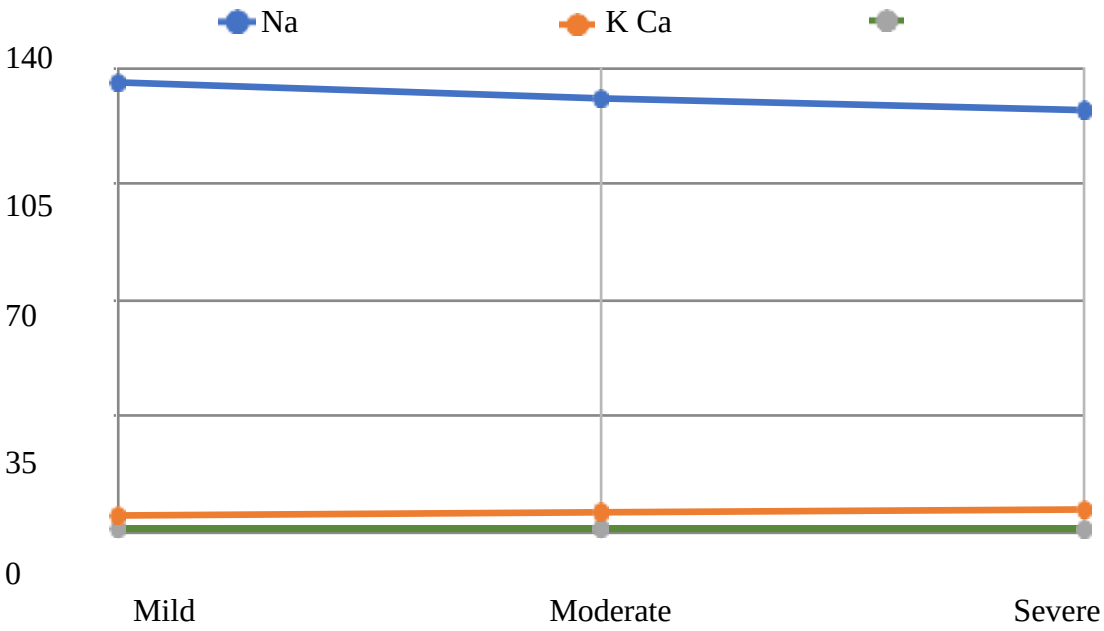
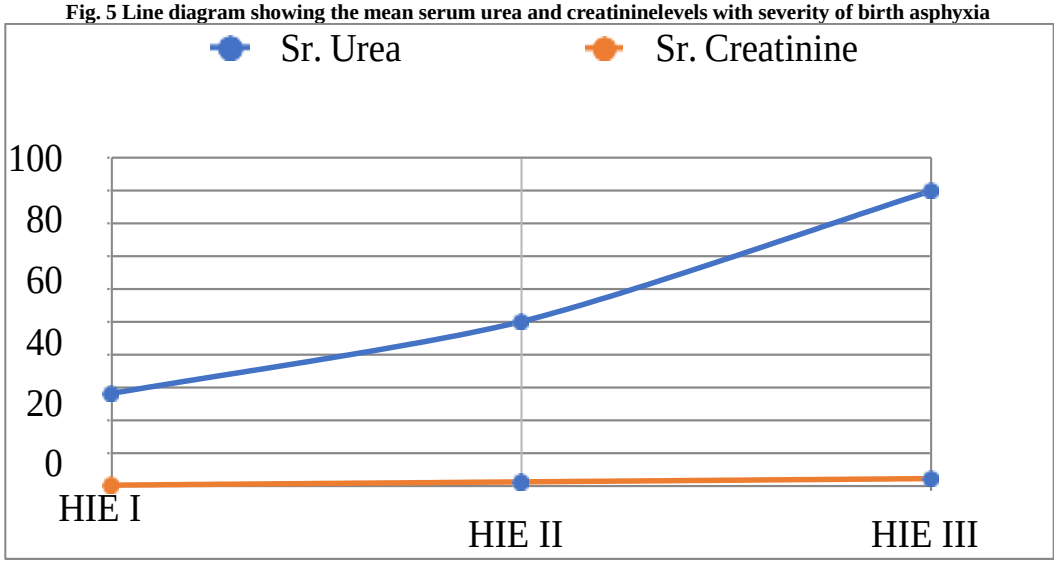


Fig. 4 Line diagram showing the mean serum sodium (Na+) and potassium (K+) levels withthe severity of birth asphyxia



The kidney is the organ that experiences HIE the most frequently in the first 24 hours of life, and if hypoxia is not treated later, cortical necrosis may become irreversible. In accordance with the stages of birth asphyxia, our investigation discovered proportionally rising serum urea and creatinine concentrations (Fig. 5).

Fig. 5 Line diagram showing the mean serum urea and creatininelevels with severity of birth asphyxia



DISCUSSION

As we drew blood as soon as humanly feasible no later than one hour after life, there was little chance of electrolyte adjustment by the body's internal milieu, where we discovered higher severity of newborn hypoxia together with increased severity of hyponatremia, hyperkalemia, and hypocalcaemia. The hyponatremia and hyperkalemia patterns matched those in our study. Similar to this, a case-control research by Jajoo, Rai, and Schedewie revealed that asphyxiated babies had lower serum calcium left than their controls. For the reasons listed in the background section, fluid restriction rather than increasing salt intake is used to treat hyponatremia in this illness. Therefore, fluid should be limited in cases of birth asphyxia until serum sodium returns to normal while weight and serum

sodium are closely monitored. To prevent the fatal consequences, serum potassium and electrocardiography (ECG) monitoring should be used.

The most effective ways to treat hyperkalemia are to address acidosis and utilise potassium-free fluid in addition to other treatments. Although there was hypocalcaemia linked to birth asphyxia in our study, there was no significant association between hypocalcaemia and HIE severity.

Acute tubular necrosis, renal vein thrombosis, and nephron destruction are all results of perinatal hypoxia. The direct Coombs test's (DCT) sodium reabsorption capacity as well as increased sodium loss in the urine as a result lead to hyponatremia. Additionally, birth hypoxia results in partial aldosterone resistance and the syndrome of inadequate antidiuretic hormone secretion (SIADH), which both result in hyponatremia. Hyponatremia leads to hypovolemia and further compromise of renal function leading to a rise in the serum urea.

CONCLUSION

One of the main issues with perinatal hypoxia is electrolyte imbalance, which is assumed to be increasing death and morbidity. The most common anomaly associated with birth asphyxia is hyponatremia, which requires precise and meticulous therapy. Hyperkalemia is equally important and needs to be handled carefully. Neonates with birth asphyxia often have hyponatremia, hyperkalemia, hyperuricemia and hypocalcemia, which can increase morbidity and death. If there is significant birth asphyxia, more severe hyponatremia should be suspected, and vice versa. Its level should therefore be checked more frequently to stop the issues it could cause. Regular potassium monitoring and ECG monitoring are necessary to detect the cardiac abnormalities associated with severe hyperkalemia and severe birth asphyxia. Therefore, for better outcomes for these neonates, early diagnosis and appropriate hydration and electrolyte management are necessary.

Declaration by Authors

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