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Study of electrolyte disturbances and renal parameters in asphyxiated newborn

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Abstract
The fluid and electrolyte shift can occur after birth asphyxia. Calcium is an important second messenger in our body and act as a cofactor for muscle function and several enzyme activities. Hypocalcemia triggers seizure activity and deleterious cardio-vascular sequences in asphyxiated newborns. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is a common problem in these neonates accounting for hyponatremia, hyperkalemia results from ischemic insult with eventual renal insufficiency. It is difficult to differentiate the seizure activity due to hypocalcemia and asphyxia.
The present study was done in SNCU of Department of Paediatrics, Darbhanga Medical College and Hospital, Laheriasarai, Darbhanga, Bihar. Total 30 cases were taken in the present study. The cases were divided as Group I: 15 cases of Asphyxiated Newborn and Group II: 15 control cases of newborn. The electrolytes (sodium, potassium and calcium) were measured from venous samples taken under aseptic precaution. The serum sodium and potassium levels were measured by 'electrolyte analyser' model ROCHE 8190. Serum sodium estimation was done by ion selective electrode method. The Serum calcium levels were measured by 'end point calorimetric method' using O cresolphethelin-complexone or OCPC.
The data generated from present study concludes that asphyxiated babies also develop hyperkalemia and hypokalemia but to reach to definite conclusion further studies are required. If inappropriate fluid and electrolytes are given, serious morbidity can result from fluid and electrolyte imbalance. So measurement of serum electrolyte is the best way to measure the baby's electrolyte status and the adequacy or excess of electrolyte intake. Hence early identification and time-based intervention of electrolyte abnormality in the early post asphyxiated period will significantly reduce the morbidity and mortality.

Keywords: Asphyxiated newborn, renal parameters, electrolyte disturbances, birth asphyxia, etc

Introduction
A birth injury is indicative of some kind of mistake that changed (what would have been) your normal delivery into a traumatic experience for your infant (and also for you). Birth injuries have many different facets to them. Sometimes a birth injury could seem like an isolated injury, or a birth injury could be related to a network of related injuries, all affecting your child in different ways. Perinatal asphyxia, or neonatal asphyxia, is one such birth injury: it is an initial birth injury that if gone untreated, could expose your child to a whole network of related birth injuries.
Perinatal asphyxia is the name for when your child doesn't breathe normally just before, during, or after birth. Asphyxia is a condition that describes a decreased or discontinued level of oxygen, and perinatal is the period that describes just before, during, and after delivery. Because the perinatal period is a brief window, a child with perinatal asphyxia is typically born quiet. Sometimes a paediatrician can be monitoring the vital signs of the baby, recognize an alarming decrease of oxygen and perinatal asphyxia, and result in a baby delivered via emergency C-section. Other times, a baby is born vaginally and is silent or limp with perinatal asphyxia. During both scenarios, the medical staff members work to get the child to breathe as quickly as possible.
There are a number of ways the baby could stop breathing. Sometimes it's related to a prolapsed umbilical cord (when the cord comes out before the baby does), or it's related to the umbilical cord being pinched somehow. Sometimes a baby stops breathing because of Meconium Aspiration Syndrome, a situation in which the baby is stressed, defecates meconium, and breathes it in either before, during, or just after vaginal delivery. Sometimes a child is born prematurely (before 37 weeks) and his or her lungs are under developed resulting in the inability to breathe his or herself. The cause is usually related to the extenuating circumstances, and the perinatal asphyxia describes the low level of oxygen the child is getting as a result of that. The symptoms of a child not breathing are pretty obvious. If the child is crying and breathing normally, he or she does not have it, but if a child is silent, limp, blue, or has trouble breathing (including rapid breathing), it's fairly obvious that the child has the condition.
If the child isn't breathing because of something like meconium aspiration syndrome, the medical staff needs to work to suction out the fluid so that the child can breathe normally. For other causes, the medical staff needs to respond to these obstacles as they come, though one of the many options may be to put the child on a respirator. If a child hasn't been breathing for a long period of time, the medical staff may choose to proactively attempt to reverse any brain damage by putting the infant in a hyperbaric oxygen tank, a therapy intended to expose the child to a 100% oxygen environment and to flood the body with as much restorative oxygen as possible.
When a child hasn't been breathing for any period of time,

you may experience even a mild risk of brain damage. Low levels of oxygen in the blood also create acidosis, a condition when too much acid in the blood builds up (another condition that could be treated with hyperbaric oxygen therapy). Any time a child stops breathing for close to five minutes, there is a very real risk of brain damage include intellectual disability, cerebral palsy, and other problems such as seizures. For this particular birth injury, the timing of the perinatal asphyxia will determine whether the child has other more severe birth injuries [1].

Serum potassium levels also depend on blood pH levels because pH affects the distribution of potassium between ICF and ECF compartments. A low pH level shifts K+ out of the cell, whereas alkalosis drives K+ into the cell. Therefore, acidosis increases the potassium concentration in the blood or serum, whereas alkalosis lowers the potassium concentration. A handy rule is that 0.1 U of pH change results in a 0.3-0.6mEq/L change in the serum potassium level. Hypokalemia is defined as a serum potassium level of less than 3.5mEq/L. Unless the patient is receiving digoxin therapy, hypokalemia is rarely a cause for concern until the serum potassium level is less than 3.0mEq/L. Hypokalemia often results from chronic diuretic use and unreplaced electrolyte loss from NG drainage. Electrocardiographic manifestations of hypokalemia include a flattened T wave, prolongation of the QT interval, or the appearance of U waves. Severe hypokalemia can produce cardiac arrhythmias, ileus, and lethargy. When significant, this condition is treated by slowly replacing potassium either intravenously or orally. Rapid administration of potassium chloride is not recommended, because it is associated with life-threatening cardiac dysfunction.

Hyperkalemia is defined as a serum potassium level of greater than 6mEq/L measured in a non-hemolyzed specimen. Hyperkalemia is of far more concern than hypokalemia, especially when serum potassium levels exceed 6.5 mEq/L or if electrocardiographic changes have developed. Electrocardiographic manifestations of hyperkalemia are a progression from peaked T waves, as the earliest sign, to a widened QRS configuration, bradycardia, tachycardia, supraventricular tachycardia (SVT), ventricular tachycardia, and ventricular fibrillation.

Hyponatremia is defined as a serum sodium level of less than 135mEq/L. usually, this is not a cause for concern until the serum sodium has dropped to less than 125mEq/L. Remember that hyponatremia usually results from excessive free water intake relative to insensible and sensible water loss. However, inadequate sodium intake can contribute to the development of hyponatremia, especially in the extremely premature infant with increased sodium loss.

Hypernatremia is defined as a serum sodium level greater than 150mEq/L. Usually, this is not a cause for concern until the serum sodium level has risen to greater than 155mEq/L. Hypernatremia is commonly seen in the first few days of life in ELBW preterm infants and most often occurs when free-water intake is inadequate to compensate for very high IWL. Very rarely, hypernatremia is the result of excessive administration of sodium in either the diet or IVFs. A common cause of excessive administration of sodium is associated with the administration of sodium bicarbonate to infants with pulmonary hypertension or metabolic acidosis in an effort to increase blood pH levels. Remember that most of the potassium in the body is contained in the intracellular compartment; therefore, serum potassium levels often do not accurately indicate total-body potassium stores.

Total serum calcium levels in term infants decline from values of 10-11mg/dL at birth to 7.5-8.5mg/dL over the first 2-3 days of life. Approximately 50% of the total calcium is in the ionized form and is the only biologically available form of calcium. Ionized calcium values, rather than total values, correlate better with calcium functions, such as cardiac contractility. Therefore, many centers rely exclusively on measurements of ionized calcium. Calcium concentrations can be reported either in milligrams per deciliter (mg/dL) or in millimolar units (mmol/L). Conversion between the 2 methods is accomplished by dividing by 4 (eg, 4mg/dL of ionized calcium equal 1mmol/L). Hypercalcemia is rarely observed in neonates; it is defined as a total serum calcium concentration of higher than 11mg/dL or an ionized calcium concentration of higher than 5mg/dL (1.25mmol/L). Hypocalcemia is more common and is defined as a total serum calcium concentration of less than 7mg/dL or an ionized calcium concentration of less than 4mg/dL (1mmol/L).

Early onset hypocalcemia may occur within the first 3 days of life in premature infants born to mothers with poorly controlled diabetes or in infants who experienced perinatal asphyxia. If the infant is asymptomatic and has a total serum calcium level of more than 6.5mg/dL or an ionized calcium level of more than 0.8-0.9mmol/L, close observation alone is appropriate. Calcium supplementation should be provided if the total serum calcium level is less than 6.5mg/dL or if the ionized level is less than 0.8-0.9mmol/L. Late-onset hypocalcemia develops after the first week of life and is usually associated with conditions with high serum phosphate levels, including hypoparathyroidism, maternal anticonvulsant use, and vitamin D deficiency. Vitamin D deficiency usually resolves with reduction of the renal phosphate load or with vitamin D supplementation [2].

Syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is a common problem in these neonates accounting for hyponatremia, hyperkalaemia results from is chaemic insult with eventual renal insufficiency. The fluid and electrolyte shift can occur after birth asphyxia. Calcium is an important second messenger in our body and act as a cofactor for muscle function and several enzyme activities. Hypocalcemia triggers seizure activity and deleterious cardio vascular sequences in asphyxiated new-borns. It is difficult to differentiate the seizure activity due to hypocalcimia and asphyxia.

Methodology

The present study was done in SNCU of Department of Paediatrics, Darbhanga Medical College and Hospital, Laheriasarai, Darbhanga, Bihar. Total 30 cases were evaluated in the present study. The cases were divided as Group I: 15 cases of Asphyxiated Newborn and Group II: 15 control cases of newborn. The electrolytes (sodium, potassium and calcium) were measured from venous samples taken under aseptic precaution. The serum sodium and potassium levels were measured by 'electrolyte analyser' model ROCHE 8190. Serum sodium estimation was done by ion selective electrode method. The Serum calcium levels were measured by 'end point calorimetric method' using O cresolphethelin-complexone or OCPC.

Inclusion criteria: All full term asphyxiated newborns (both intramural and extramural) admitted in SNCU on day 1 of

life.

Exclusion criteria: Cases born to mothers with abnormal electrolyte values, hypertension, diabetes, fever within 2 weeks, on antiepileptics, received general anaesthesia, preterm and congenital malformations and suspected metabolic disease.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Results and Discussion

Perinatal asphyxia is a dreadful neonatal problem and contributes significantly to neonatal mortality and morbidity. Hypoxic ischemic encephalopathy is the major consequence of perinatal asphyxia. The important three (potassium, sodium and calcium) abnormalities will be the major risk factor for brain injury in an already asphyxiated one. Careful correction of the above electrolyte abnormalities will surely improve the outcome of newborns. This study interprets the association of electrolyte abnormalities with the different severity of asphyxia.

In newborns there is hyperkalemia 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 [3]. Serum potassium subsequently falls as this internal potassium "load" is excreted by the kidneys [4]. Whereas 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 setting, electro neutrality is maintained in part by the movement of intracellular potassium into the extracellular fluid. It can also 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. With the abrupt termination of calcium transport across the placenta at delivery, plasma calcium falls, reaching a nadir at age 24–48 h [5]. Serum parathyroid hormone (PTH) increases postnatally in response to this fall in plasma calcium concentration. This increase in PTH mobilizes calcium from bone, and plasma calcium concentration rises and subsequently stabilizes even in the absence of exogenous calcium intake. Clinically significant hypocalcaemia occurs in asphyxiated newborns [6]. The etiology behind this is a sluggish response in PTH secretion to the postnatal fall in plasma calcium concentration.

Table 1: Sex of New Borns

Groups	Asphyxiated Newborn	Control Cases
Males	9	11
Females	6	4
Total	15	15

Table 2: Renal Parameters

Groups	Asphyxiated Newborn	Control Cases
BUN	21.5 – 32.9	18.5 – 26.2
Serum Creatinine	1.35 – 1.86	0.79 – 1.29
Urine Sodium	24.5 – 42.9	13.5 – 19.6
Urine Creatinine	19.2 – 32.7	11.3 – 22.4

Table 3: Serum Electrolytes

Groups	Asphyxiated Newborn	Control Cases
Serum Sodium (mEq/L)	125.9 – 137.1	133.2 – 141.8
Serum Potassium (mEq/L)	4.1 – 5.9	3.9 – 4.6
Serum Calcium (mEq/L)	7.4 – 8.7	8.6 - 9.4

Most of the control infants, particularly those with the respiratory distress syndrome, were hypoxic and hypercapnoeic during life, pointing to cerebral ischaemia as the main pathophysiological difference between the test and control groups and suggesting that brain water and electrolyte abnormalities in asphyxiated infants without cerebral cortical necrosis represent pre-necrotic ischaemic brain damage. Experiments designed to examine the effects of hypoxia and hypercapnoea without cerebral ischaemia have demonstrated that in adult cast brain water and electrolyte concentrations remain unchanged if the blood pressure is maintained [7]. Arterial blood pressure is not usually recorded in newborn infants, but infants with a low Apgar score have bradycardia and may even have episodes of cardiac arrest at the time of birth. Furthermore, experiments with fetal lambs and monkeys have shown that asphyxia causes a fall in heart rate and blood pressure [8]. The importance of ischaemia in the causation of brain damage at the time of birth is also illustrated by the neuropathological findings in spastic and mentally deficient survivors of abnormal births, many of whom show lesions which are undoubtedly the result of prolonged hypotension [9, 10].

Basu P *et al.* had studied assessed the electrolyte abnormalities in 50 controls as well as in 50 asphyxiated babies of variable severity. The results were same as in the present study. This study showed significant hypocalcaemia in the case group [11]. Jajoo *et al.*, studied the calcium levels in 35 asphyxiated new-borns at birth, 6, 24 and 5 th day of life. They observed significant low calcium levels at birth and other periods also [12].

Gupta *et al.* had studied the relation between the electrolyte abnormality and acute renal failure in asphyxiated new-borns. They had included preterm babies also in the account. This study also concluded that increased electrolyte derangement with increased severity of HIE stages. The subjects with hyponatremia were very much prone for ARF. But one variation from the present study is they measured ionized calcium levels which were significant [13]. Lackmann *et al.*, measured potassium levels in 98 asphyxiated new-borns and none of them showed significant hyperkalaemia in the initial 144 hours of life [14].

In a study by Anu Aggarwal *et al.* [15]. in 2005 it was found that serum urea and creatinine values were significantly higher on day 4 but not on day 2. In our study significant positive correlation found on day 3 urea, creatinine values with increase severity of birth asphyxia. G Jayashree *et al.*

[16] found 43% of asphyxiated babies had acute renal failure. But they did not find any significant correlation between Apgar score and development of renal failure, rather a significant relationship seen between hypoxic ischaemic encephalopathy and acute renal failure. In a study by B D Gupta *et al.* [17] found 33% of asphyxiated babies developed renal failure. In their study biochemical derangements correlated well with Hypoxic ischaemic stage and Apgar score.

The treatment of hyponatremia in such condition is by fluid restriction rather increasing sodium load for reasons mentioned in background section. 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 should be done to avoid the deadly complications of hyperkalemia. Apart from other treatment measures, correction of acidosis and use of potassium free fluid are the most useful measures to correct hyperkalemia. Our study however did not find significant hypocalcaemia with increasing severity of HIE but there was hypocalcaemia associated with birth asphyxia, so regular supplementation and monitoring of serum calcium should be done.

Conclusion

From present study we concludes that asphyxiated babies also develop hyperkalemia and hypokalemia but to reach to definite conclusion further studies are required. If inappropriate fluid and electrolytes are given, serious morbidity can result from fluid and electrolyte imbalance. So measurement of serum electrolyte is the best way to measure the baby's electrolyte status and the adequacy or excess of electrolyte intake. Hence early identification and time-based intervention of electrolyte abnormality in the early post asphyxiated period will significantly reduce the morbidity and mortality.

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