International Journal of Medical and Health Research
ISSN: 2454-9142; Impact Factor: RJIF 5.54
Received: 28-10-2019; Accepted: 29-11-2019
www.medicalsciencejournal.com
Volume 5; Issue 11; November 2019; Page No. 229-233

Correlation the levels of serum electrolyte levels and renal parameters in cases of birth asphyxia

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Abstract
Perinatal hypoxia is one of the most common primary cause of neonatal mortality and morbidity in India. 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. In cases of birth asphyxia most of the organ like brain, kidneys, heart, lungs can be affected but Kidneys are more sensitive to oxygen deprivation so they leads to renal insufficiency approximately within 24 hours of hypoxic ischemic injury. Hence based on above findings the present study was planned for correlation the levels of serum electrolyte levels and renal parameters in cases of birth asphyxia.
The present study was planned in Department of Paediatrics, Government Medical College, Bettiah, West Champaran, Bihar. Total 40 cases of the newborns were selected in the present study. The 20 cases were of suffered from birth asphyxia and 20 cases are control cases. Cases were selected based on inclusion criteria and detailed clinical examination was done and physical findings were recorded on pre designed proforma after informed consent obtained from parents sample was collected and following investigations were done.
Perinatal asphyxia is an important cause of neonatal renal failure. Monitoring of blood levels of urea, serum creatinine, serum calcium and urine output helps in the early diagnosis and management of renal failure in birth asphyxia. The biochemical parameters in both blood and urine should be monitored. The renal indices should be calculated, as fractional excretion of sodium is preferred to classify the renal failure into pre renal or intrinsic renal failure as management differs for both entity.

Keywords: serum electrolyte, renal parameters, birth asphyxia, neonates, etc

Introduction
Birth asphyxia, defined as the failure to establish breathing at birth, accounts for an estimated 900 000 deaths each year and is one of the primary causes of early neonatal mortality. Guidelines for neonatal resuscitation, such as those endorsed by WHO and the American Academy of Pediatrics, represent a standard practice set that improves outcomes in asphyxiated newborns. These algorithms stress the importance of drying, stimulating and warming babies in distress, as well as clearing their airways. In the face of persistent apnoea or bradycardia, ventilation with the use of bag-and-mask or equivalent device is indicated, and is felt by many to constitute the critical step in managing asphyxiated infants. Newborns have a remarkable ability to withstand hypoxia and many improve rapidly with timely implementation of these techniques.
Perinatal asphyxia (also known as neonatal asphyxia or birth asphyxia) is the medical condition resulting from deprivation of oxygen to a newborn infant that lasts long enough during the birth process to cause physical harm, usually to the brain. It is also the inability to establish and sustain adequate or spontaneous respiration upon delivery of the newborn. It remains a serious condition which causes significant mortality and morbidity. It is an emergency condition and requires adequate and quick resuscitation measures.
Perinatal asphyxia is also an oxygen deficit from the 28th week of gestation to the first seven days following delivery. It is also an insult to the fetus or newborn due to lack of oxygen or lack of perfusion to various organs and may be associated with a lack of ventilation. In accordance with WHO, perinatal asphyxia is characterised by- Profound metabolic acidosis, with a PH <7.20 on umbilical cord arterial blood sample, Persistence of an APGAR score of 3 at the 5th minute, Clinical neurologic sequelae in the immediate neonatal period, Evidence of multiorgan system dysfunction in the immediate neonatal period.
Hypoxic damage can occur to most of the infant's organs (heart, lungs, liver, gut, kidneys), but brain damage is of most concern and perhaps the least likely to quickly or completely heal. In more pronounced cases, an infant will survive, but with damage to the brain manifested as either mental, such as developmental delay or intellectual disability, or physical, such as spasticity.It results most commonly from antepartum causes like a drop in maternal blood pressure or some other substantial interference with blood flow to the infant's brain during delivery. This can occur due to inadequate circulation or perfusion, impaired respiratory effort, or inadequate ventilation. Perinatal asphyxia happens in 2 to 10 per 1000 newborns that are born at term, and more for those that are born prematurely [1]. WHO estimates that 4 million neonatal deaths occur yearly due to birth asphyxia, representing 38% of deaths of children under 5 years of age [2].
Perinatal asphyxia can be the cause of hypoxic ischemic encephalopathy or intraventricular hemorrhage, especially in preterm births. An infant suffering severe perinatal asphyxia usually has poor color (cyanosis), perfusion, responsiveness, muscle tone, and respiratory effort, as reflected in a low 5 minute Apgar score. Extreme degrees of

asphyxia can cause cardiac arrest and death. If resuscitation is successful, the infant is usually transferred to a neonatal intensive care unit.

There has long been a scientific debate over whether newborn infants with asphyxia should be resuscitated with 100% oxygen or normal air [3]. It has been demonstrated that high concentrations of oxygen lead to generation of oxygen free radicals, which have a role in reperfusion injury after asphyxia [4]. Research by Ola Didrik Saugstad and others led to new international guidelines on newborn resuscitation in 2010, recommending the use of normal air instead of 100% oxygen [5, 6]. There is considerable controversy over the diagnosis of birth asphyxia due to medicolegal reasons [7]. Because of its lack of precision, the term is eschewed in modern obstetrics.

Despite major advances in monitoring technology and knowledge of fetal and neonatal pathologies, hypoxic-ischemic encephalopathy (HIE) remains a serious condition that causes significant mortality and long-term morbidity. HIE is characterized by clinical and laboratory evidence of acute or subacute brain injury due to asphyxia (ie, hypoxia, acidosis). Most often, the exact timing and underlying cause remain unknown. The American Academy of Pediatrics (AAP) and American College of Obstetrics and Gynecology (ACOG) published guidelines to assist in the diagnosis of severe hypoxic-ischemic encephalopathy [8, 9].

Brain hypoxia and ischemia due to systemic hypoxemia, reduced cerebral blood flow (CBF), or both are the primary physiologic processes that lead to hypoxic-ischemic encephalopathy (HIE). The initial compensatory adjustment to an asphyxial event is an increase in CBF due to hypoxia and hypercapnia. This is accompanied by a redistribution of cardiac output to essential organs, including the brain, heart, and adrenal glands. A blood pressure (BP) increase due to increased release of epinephrine further enhances this compensatory response.

Limited data in the human fetus and the newborn infant suggest that CBF is stable over much narrower range of BPs [10, 11]. Some experts have postulated that, in the healthy term newborn, the BP range at which the CBF autoregulation is maintained may be only between 10-20 mm Hg (compared with the 40 mm Hg range in adults noted above). In addition, the autoregulatory zone may also be set at a lower level, about the midpoint of the normal BP range for the fetus and newborn. However, the precise upper and lower limits of the BP values above and below which the CBF auto regulation is lost remain unknown for the human newborn.

In the fetus and newborn suffering from acute asphyxia, after the early compensatory adjustments fail, the CBF can become pressure-passive, at which time brain perfusion depends on systemic BP. As BP falls, CBF falls below critical levels, and the brain injury secondary to diminished blood supply and a lack of sufficient oxygen occurs. This leads to intracellular energy failure. During the early phases of brain injury, brain temperature drops, and local release of neurotransmitters, such as gamma-aminobutyric acid transaminase (GABA), increase. These changes reduce cerebral oxygen demand, transiently minimizing the impact of asphyxia.

At the cellular level, neuronal injury in HIE is an evolving process. The magnitude of the final neuronal damage depends on the duration and severity of the initial insult, combined with the effects of reperfusion injury, and apoptosis. At the biochemical level, a large cascade of events follow hypoxic-ischemic injury.

Excitatory amino acid (EAA) receptor overactivation plays a critical role in the pathogenesis of neonatal hypoxia-ischemia. During cerebral hypoxia-ischemia, the uptake of glutamate the major excitatory neurotransmitter of the mammalian brain is impaired. This results in high synaptic levels of glutamate and EAA receptor overactivation, including N-methyl-D-aspartate (NMDA), amino-3-hydroxy-5-methyl-4 isoxazole propionate (AMPA), and kainate receptors. NMDA receptors are permeable to Ca++ and Na+, whereas AMPA and kainate receptors are permeable to Na+. Accumulation of Na+ coupled with the failure of energy dependent enzymes such as Na+/ K+ - ATPase leads to rapid cytotoxic edema and necrotic cell death. Activation of NMDA receptor leads to intracellular Ca++ accumulation and further pathologic cascades activation.

EAAs accumulation also contributes to increasing the pace and extent of programmed cell death through secondary Ca++ intake into the nucleus. The pattern of injury seen after hypoxia-ischemia demonstrate regional susceptibility that can be largely explained by the excitatory circuity at this age (putamen, thalamus, perirolandic cerebral cortex). Finally, developing oligodendroglia is uniquely susceptible to hypoxia-ischemia, specifically excitotoxicity and free radical damage. This white matter injury may be the basis for the disruption of long-term learning and memory faculties in infants with hypoxic-ischemic encephalopathy.

Intracellular Ca++ concentration increases following hypoxia-ischemia as a result of (1) NMDA receptor activation, (2) release of Ca++ from intracellular stores (mitochondria and endoplasmic reticulum [ER]), and (3) failure of Ca++ efflux mechanisms. Consequences of increases intracellular Ca++ concentration include activation of phospholipases, endonucleases, proteases, and, in select neurons, nitric oxide synthase (NOS). Activation of phospholipase A2 leads to release of Ca++ from the ER via activation of phospholipase C. Activation of proteases and endonucleases results in cytoskeletal and DNA damage.

During the reperfusion period, free radical production increases due to activation of enzymes such as cyclooxygenase, xanthine oxidase, and lipoxygenase. Free radical damage is further exacerbated in the neonate because of immature antioxidant defenses. Free radicals can lead to lipid peroxidation as well as DNA and protein damage and can trigger apoptosis. Finally, free radicals can combine with nitric oxide (NO) to form peroxynitrite a highly toxic oxidant.

NMDA receptor activation results in activation of neuronal NOS vias PSD-95 and results in the early and transient rise in NO concentration observed in the initial phase of hypoxia. Inducible NOS is expressed in response to the marked inflammation secondary to cerebral ischemia and results in a second wave of NO overproduction that can be prolonged for up to 4-7 days after the insult.

This excessive NO production plays an important role in the pathophysiology of perinatal hypoxic-ischemic brain injury. NO neurotoxicity depends in large part on rapid reaction with superoxide to form peroxynitrite [12]. This, in turn, leads to peroxynitrite-induced neurotoxicity, including lipid peroxidation, protein nitration and oxidation, mitochondrial damage and remodeling, depletion of antioxidant reserve, and DNA damage.

Inflammatory mediators (cytokines and chemokines) have been implicated in the pathogenesis of hypoxic-ischemic encephalopathy and may represent a final common pathway of brain injury. Animal studies suggest that cytokines, particularly interleukin (IL)-1b contributes to hypoxic-ischemic damage. The exact mechanisms and which inflammatory mediators are involved in this process remains unclear.

Following the initial phase of energy failure from the asphyxial injury, cerebral metabolism may recover following reperfusion, only to deteriorate in a secondary energy failure phase. This new phase of neuronal damage, starting at about 6-24 hours after the initial injury, is characterized by mitochondrial dysfunction, and initiation of the apoptotic cascade. This phase has been called the "delayed phase of neuronal injury."

The duration of the delayed phase is not precisely known in the human fetus and newborn but appears to increase over the first 24-48 hours and then start to resolve thereafter. In the human infant, the duration of this phase is correlated with adverse neurodevelopmental outcomes at 1 year and 4 years after insult [13].

Perinatal hypoxia is one of the most common primary cause of neonatal mortality and morbidity in India. 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 [14-16].

In cases of birth asphyxia most of the organ like brain, kidneys, heart, lungs can be affected but Kidneys are more sensitive to oxygen deprivation so they leads to renal insufficiency approximately within 24 hours of hypoxic ischemic injury. Hence based on above findings the present study was planned for correlation the levels of serum electrolyte levels and renal parameters in cases of birth asphyxia.

Methodology

The present study was planned in Department of Paediatrics, Government Medical College, Bettiah, West Champaran, Bihar. Total 40 cases of the newborns were selected in the present study. The 20 cases were of suffered from birth asphyxia and 20 cases are control cases.Cases were selected based on inclusion criteria and detailed clinical examination was done and physical findings were recorded on pre designed proforma after informed consent obtained from parents sample was collected and following investigations were done.

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.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: 1. Term (34-41 weeks), appropriate for gestational age (inborn & outborn) ; 2. Outborn with history of birth asphyxia (delayed cry of 5-10 min) and inborn with apgar score at 1 min (less than 7) .

Exclusion Criteria: Babies with congenital cardio pulmonary malformation; Those born mother with diabetes mellitus and hypertension treated with diuretics, general anaesthesia, phenobarbitone, pethidine, magnesium sulphate and other drugs likely to cause depression.

Results & Discussion

The postnatal development of renal functions, especially in early life, has a great bearing on the successful transition from fetal to extrauterine life. Asphyxia can have serious impact on various organ systems. Impairment of renal functions can jeopardize the success of this transition. Perinatal hypoxia contributes significantly to neonatal mortality and morbidity. HIE is the major consequence of perinatal asphyxia. In asphyxiated newborn most of the organ can be affected but the brain, myocardium, kidneys 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. In cases of birth asphyxia Kidneys are more sensitive to oxygen deprivation which leads to renal insufficiency approximately within 24 hours of hypoxic ischemic injury.

Table 1: Basic Characteristic

Cases of	Birth Asphyxia	Control Cases
No. of Cases	**20**	**20**
Gestation weeks	34 – 39	35 – 38
Weight gm	2100 – 2950	2240 – 3105
Caesarean Delivery	10	12
Respiratory Distress	8	9

Table 2: Sex of New Borns

Renal Failure	Birth Asphyxia		
	Mild	Moderate	Severe
Renal Failure	2	11	2
No Renal Failure	1	4	0

Table 3: Serum Electrolytes& Renal Parameters

Cases of	Birth Asphyxia	Control Cases
No. of Cases	**20**	**20**
Serum Sodium (mEq/L)	124.5 – 135.6	134.5 – 142.9
Serum Potassium (mEq/L)	4.4 – 6.2	4.2 – 4.7
Serum Calcium (mEq/L)	7.4 – 8.5	8.8 – 9.4
Serum Creatinine	1.31 – 1.82	0.91 – 1.35
Urine Sodium	28.5 – 43.5	13.6 – 18.3
Urine Creatinine	19.3 – 35.4	14.1 – 23.7

Palab basu *et al* [17] studied the correlation of serum electrolytes and calcium levels in asphyxiated babies of different severity, he found serum sodium and calcium levels were significantly lower and serum potassium was higher in cases than controls. Hence he concluded hyponatremia and hypocalcaemia developed early and simultaneously and decrease in their serum level was directly proportional to each other and to the degree of asphyxia.

Shah GS *et al* [18] did hospital based observational study from feb 2010 to jan 2011and concluded neonates having birth asphyxia had metabolic derangements like hyponatremia , hypocalcaemia and hypoglycemia. He also noted other complications of acute renal failure moderate and severe asphyxia

Gupta *et al* [19] showed lower serum sodium levels among asphyxiated babies as compared to the control group and no statistically difference in the serum potassium levels between cases and controls.

Varma vandana *et al* [20] carried out a study to determine status of basic biochemical and hematological parameters in asphyxiated babies and their relation with APGAR score and HIE staging and development of ARF. Electrolyte status

showed no significant variation in cases and controls. Highly significant rise was seen in blood urea and serum creatinine values.

Alphonsus .N.onyiriuka [21] studied serum calcium levels in asphyxiated newborns found overall prevalence of early onset neonatal hypocalcaemia among infants with severe birth asphyxia. The mean total serum calcium levels in bicarbonate treated infants were higher than their counterpart without bicarbonate therapy.

Jajoo *et al* [22] measured serum calcium and phosperous in neonates with perinatal asphyxia and he found significant low calcium levels in asphyxiated infants than controls.

The mean blood urea levels were higher among cases as compared to controls which were statistically significant but there was no significant difference among groups with severity of asphyxia. The results of our study are comparable to other studies. In our study we found that serum creatinine levels were higher in cases as compared to controls and the difference between the groups was statistically significant both at 48 and 72 hrs. But however the serum creatinine value was higher in cases with severe asphyxia, the difference among the groups was not statistically significant. The observations in our study are comparable with other studies.

In our study the mean serum sodium concentration was lower among the cases as compared to controls which were comparable with other studies which was statistically significant. The serum sodium levels were lower in neonates with severe birth asphyxia; the mean serum sodium is higher in our study as compared to Misra *et al*., [23] study in which the study population is small with most neonates belonging to either stage II or stage III which indicates either moderate or severe degree of asphyxia. Pallab Basu and colleagues + study does not mention the distribution of cases in with different severity of asphyxia. The mean serum potassium level was higher in cases than the controls which is comparable with other studies. In Misra *et al*., [23] study the mean serum potassium level was higher than the other studies in which only 7 neonates have been studied and majority had severe birth asphyxia.

The mean serum calcium level in our study was lower as compared to controls, which was noticed after 24-48 hrs, as only few patients belong to severe birth asphyxia. The mean serum calcium level is not reduced to as low as in Pallab Basu [29] study in which they have not considered regarding the distribution of cases. As most neonates were on calcium supplementation, hypocalcaemia of < 7 was not seen in any neonates.

Prerenal failure may result in intrinsic kidney failure if not treated promptly. The kidneys of neonates are particularly susceptible to hypoperfusion, because of the physiologic characteristics of neonatal kidneys; high renal vascular resistance, high plasma renin activity, low glomerular filtration rate, decreased intercortical perfusion, and decreased reabsorption of sodium in the proximal tubules are the susceptibilities of the kidneys in the first days of a neonate. Thus, newborn infants are vulnerable to acute tubular necrosis or cortical necrosis [25]

. Acute renal failure (ARF) is defi ned as the rapid elevation in the concentration of blood urea nitrogen (BUN), creatinine, and other cellular waste products in the blood resulting from diminished glomerular fi ltration rate (GFR) in the kidney [26]. In term babies, the concentration of serum creatinine normally rises somewhat in the first 24 to 36 hours after birth, subsequently decreasing and stabilizing at about 0.4 mg/dL (35.4 mcmol/L) by 5 days of age [27]. A clearly elevated value beyond the normal range indicates decreased glomerular function. Urine output is another key indicator of renal function. Commonly, ARF is suspected when oliguria is present, defined as a period during which urine output is less than 0.5 mL/kg per hour [28]. Renal involvement is frequent in neonates with perinatal asphyxia, which correlates with the severity of neurological damage. Renal failure presents as oliguria and, during recovery, as highoutput tubular failure, leading to signifi cant water and electrolyte imbalances. In one study it has been shown 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. Though, mean potassium level was within the normal limit, the value was higher among cases than controls and directly proportional to asphyxia [29].

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. The diagnosis of renal dysfunction in neonates is however difficult because the routine clinical and biochemical parameters are affected by many non-renal factors and maternal parameters. Calculated renal indices may also be affected by the difficulty in collecting urine samples and interference by interventions like saline bolus, diuretics or aminophylline [30].

Conclusion

Perinatal asphyxia is an important cause of neonatal renal failure. Monitoring of blood levels of urea, serum creatinine, serum calcium and urine output helps in the early diagnosis and management of renal failure in birth asphyxia. The biochemical parameters in both blood and urine should be monitored. The renal indices should be calculated, as fractional excretion of sodium is preferred to classify the renal failure into pre renal or intrinsic renal failure as management differs for both entity.

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