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A Case – Control Study of Metabolic Changes in Newborns

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Abstract

Background: In neonates with birth asphyxia, serum calcium level may fall after birth due to cessation of placental transfer. The calcium plays multiple roles in body being the secondary messenger. Muscle movements are dependent on calcium. **Methods:** The serum calcium was analyzed usingo-cresolphthalein method in autoanalyzer Siemens Dimension series. The serum electrolytes was assessed using ion selective electrode (ISE) method in autoanalyzer. **Results:** In this study, 58 cases and 38 control were included. Among the 38 control 14 were female and 24 were male and from the 58 cases 18 were female and 40 were male. Serum calcium, potassium & sodium level calculated by mean and standard deviation. **Conclusion:** Development of hypocalcaemia in birth asphyxia may require medical attention. Early diagnosis and proper treatment of metabolic changes commonly occurring in neonates with birth asphyxia may improve their life specially neurodevelopmental aspect and also decrease the mortality rate.

Keywords: Newborns, Hypocalcaemia, Cases. Control, Autoanalyzer

INTRODUCTION

According to WHO, birth asphyxia is defined as "failure to initiate and sustain breathing at birth."[1] Birth asphyxia is the leading cause of morbidity and mortality (28.8%) among neonates in India.[2] Systemic hypoxia and or reduced cerebral blood flow are the main causes of birth asphyxia. Certain metabolic changes like hypocalcemia, hypoglycaemia, hyponatremia, hyperphosphatemia etc are frequently associated with this condition. Metabolic acidosis has also been reported to be associated with it.[3] In neonates with birth asphyxia, serum calcium level may fall after birth due to cessation of placental transfer. The calcium plays multiple roles in body being the secondary messenger. Muscle movements are dependent on calcium. It also acts as co-factor for several enzymes as well. Deficiency of calcium may lead to neuromuscular irritability, presenting as twitching, convulsion or jitteriness.[4,5] Endogenous breakdown of glycogen and tissue protein, which impairs the functions of parathyroid gland may lead to hypophosphatemia.[5] Encephalopathy and neurological injuries may occur due to hypoglycemia because glucose is an essential nutrient for the brain. In birth asphyxia, there is increased release of catecholamine's which may lead to development of hypoglycemia.[6]

Birth asphyxia is an important cause of metabolic derangements and acute neurologic injury in newborns .The term "asphyxia" is derived from a Greek word meaning "stopping of the pulse". Birth asphyxia is defined as metabolic or mixed acidemia (pH < 7.0) with persistence of an APGAR score of 0 to 3 at >5 min of birth along with hypotonia , seizures, coma or hypoxic ischemic encephalopathy and systemic impairment in immediate neonatal period.[7] Worldwide incidence of birth asphyxia is 2-9/1000 live full term births. In Pakistan this incidence is much higher due to poor availability and quality of health care.[8] It is estimated that around 23% neonatal deaths are due to birth asphyxia .It is also responsible for a large proportion of still births.[9] According to the World Health Organization (WHO), around four million newborns develop birth asphyxia annually, and out of these 1.2 million die and almost the same number develop severe sequelae such as developmental delay, cerebral palsy and epilepsy.[10] The grave morbidity associated with it, in the form of permanent neurological deficit like cerebral palsy and mental retardation, is up to 25%.[11] The severity of Hypoxic-Ischemic Encephalopathy (HIE) symptoms reflects the timing and duration of the insult.[12] Birth Asphyxia can be categorized into three stages of hypoxic ischemic encephalopathy according to Sarnat staging.[13-14] The Sarnat Grading Scale of HIE is a scoring system used to grade the severity of an HIE injury. Metabolic derangements have been seen in asphyxiated newborns along with multisystem involvement.[15-17] The electrolyte imbalance manifests in the form of hyponatremia and hypocalcaemia which have significant linear correlation with severity of birth asphyxia.[18-19]

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MATERIAL AND METHODS

Study population
58 newborns with birth asphyxia were included cases and 38 newborns without asphyxia were included control.

Study Area
The case control study was carried out in the Departments of Paediatrics in a tertiary care centre.

Study duration
Duration of study were three months.

Sampling technique & Data collection
The serum calcium was analyzed usingo-cresolphthalein method in autoanalyzer Siemens Dimension series.[1] The

serum electrolytes was assessed using ion selective electrode(ISE) method in autoanalyzer.[2]

Data Analysis

In this case control study, the unpaired student's t-test was used to compare both the groups.

RESULTS

In this study, 58 cases and 38 control were included. Among the 38 control 14 were female and 24 were male and from the 58 cases 18 were female and 40 were male. Serum calcium, potassium & sodium level calculated by mean and standard deviation.

Table 1: Gender wise distribution in both group

	Cases	Percentage	Control	Percentage
Male	40	68.9%	24	63.1%
Female	18	31.1%	14	36.9%
Total	58	100%	38	100%

Table 2: The mean serum calcium and electrolytes levels in both the groups

Parameters	mean		SD		P value	Significance
	Cases	Control	Cases	Control		
Serum calcium	7.96	9.5	.66	.64	<0.005	Highly significant
Serum potassium	5.33	134.97	5.6	5.44	0.129	Not significant
Serum sodium	136.76	5.27	.89	.99	.78	Not significant

DISCUSSION`

In the present study, the serum calcium levels were significantly lower ($p<0.005$) in birth asphyxia neonates than in control group. In study of Jadoo et al.[20] similar findings of significant low levels of serum calcium in birth asphyxia neonates were noticed. Jain et al,[5] reported significantly low total calcium (7.9±1.7mg/dl) in comparison to controls having total calcium(9.5±0.9mg/dl) at 48 hrs of life. Basu et al,[2] also revealed the low calcium levels in neonates with birth asphyxia (6.85±0.95 mg/dl) in comparison to controls (9.50±0.51mg/dl). Almost similar results were obtained by Rai et al,[1] in their study showing significantly low calcium level in birth asphyxia neonates (8.31±0.48 mg/dl) in comparison to controls(9.47±1.7mg/dl).

Rai et al,[1] also compared serum calcium levels among asphyxiated babies having different degree of asphyxia based on Apgar score. Mild and moderately asphyxiated babies do not have significant differences between calcium levels while severely asphyxiated babies were having significantly lower total calcium levels.

Basu et al,[2] also observed the serum sodium and potassium levels. Serum sodium levels were significantly lower (122.1±6.0 meq/L) in birth asphyxia neonates than that of control group (138.8±2.7 meq/L). Similarly, the serum potassium levels were lower (5.05±0.63meq/L) in comparison to controls (4.19±0.40meq/L). While, no statistical significant differences were found in present study in serum sodium and potassium levels between birth asphyxia neonates and controls.

CONCLUSION

Development of hypocalcaemia in birth asphyxia may require medical attention. Early diagnosis and proper treatment of metabolic changes commonly occurring in neonates with birth asphyxia may improve their life specially neurodevelopmental aspect and also decrease the mortality rate.

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