SERUM IONISED CALCIUM IN BIRTH ASPHYXIA

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ABSTRACT

Serum total and ionised calcium levels were measured at birth and at 48 hours in 25 term neonates with birth asphyxia (one minute APGAR score of 6 or less) and in 25 normal term neonates (one minute APGAR score of 7 or more). Infants were categorised into two groups TAGA (term appropriate for gestational age) and TSGA (term small for gestational age). Asphyxiated infants had significantly lower serum total and ionised calcium values at birth as well as at 48 hours. Abnormal clinical features were observed in 48% of asphyxiated infants. Low ionised calcium was detected in symptomatic babies, who had otherwise normal total calcium values. Due to hypocalcemia especially ionised calcium in asphyxiated infants and high frequency of functional derangement associated with this hypocalcemia, serial monitoring of serum ionised calcium levels is necessary.

KEY WORDS: Birth asphyxia, Serum ionised calcium.

INTRODUCTION

Birth asphyxia is one of the leading causes of perinatal mortality and morbidity in our country. It accentuates the physiological fall in serum calcium levels in neonates leading to hypocalcemia (1-4). The cause of this hypocalcemia in birth asphyxia is still debated. Hyperphosphatemia due to endogenous breakdown of glycogen and tissue proteins in birth asphyxia (5, 6) which impairs the functional capabilities of parathyroid glands has been postulated as possible etiopathological factors. Hypocalcemia may result in neuromuscular irritability which presents in the form of convulsions, jitteriness, twitchings and/or irritability. Nonspecific symptoms like poor feeding, lethargy and vomiting may also result.

Studies conducted so far have measured serum total calcium only in asphyxiated infants. Ionised calcium is the biologically active fraction which has

not been measured in asphyxia. In this study, in addition to measuring serum total calcium, ionised calcium levels have been estimated. Babies were observed for clinical manifestations of hypocalcemia in asphyxia.

MATERIALS AND METHODS

The study was conducted on 50 neonates in Dayanand Medical College and Hospital, Ludhiana. The 25 term neonates with birth asphyxia one minute APGAR (7) score ≤ 6 were further divided into two groups, TAGA (term appropriate for gestational age) and TSGA (term small for gestational age). Babies with conditions known to be associated with hypocalcemia viz, infants of diabetic mothers, premature infants, infants with hemolytic disease requiring exchange transfusion, bicarbonate therapy, septicemia and congenital malformations were excluded. Besides, 25 term nonasphyxiated neonates with one minute APGAR score of 7 or more served as control group.

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Serum total calcium estimation of all neonates was estimated using Multichannel Autoanalyser, Hitachi-911 and ionised calcium levels (directly from Nova Blood Gas Analyser) at birth and at 48 hours. Serum magnesium and phosphorus were determined at 48 hours of life using autoanalyser Hitachi-911. Hypocalcemia was defined as serum total calcium of 7.0 mg/dl and less, and ionised calcium values of 4.0 mg/dl and less. Statistical analysis was done by Student's t test. The babies were watched for clinical signs and symptoms of hypocalcemia, i.e. irritability, lethargy, jitteriness, poor feeding and convulsions.

RESULTS

There was significant decrease ($P<0.01$) in total

calcium levels in asphyxiated infants at 48 hours (7.9 ± 1.7 mg/dl) as compared to control values at birth (9.5 ± 0.9 mg/dl) (Table 1). Asphyxiated infants showed significantly low ($P<0.01$) serum ionised calcium levels at birth (3.8 ± 1.5 mg/dl) as well as at 48 hrs. (3.4 ± 1.2 mg/dl) (Table 2). Asphyxiated infants did not show significant decrease in serum magnesium and serum phosphorus levels at 48 hours when compared to controls. TSGA infants when compared with TAGA infants, showed comparable serum total as well as ionised calcium levels. Functional derangement's were observed in 48% of asphyxiated babies (Table 3). Out of these, maximum babies had jitteriness (58.3%) followed by lethargy (16.7%), poor feeding (8.3%), irritability (8.3%) and seizures (8.3%). The

Table 1. Mean serum total calcium levels at birth and at 48 hours.

	N	At birth (mg/dl)	At 48 hours (mg/dl)	t value
Study group	25	9.5 ± 0.9	7.9 ± 1.7	4.4*
Controls	25	9.6 ± 0.7	9.4 ± 0.9	1.1NS
t. Value		0.3 NS	3.9*	

* Significant, $P<0.01$, NS-Non significant.

Table 2. Mean serum ionised calcium levels at birth and at 48 hours.

	N	At birth (mg/dl)	At 48 hours (mg/dl)	t-value
Study group	25	3.9 ± 1.5	3.4 ± 1.23	1.1 NS
Controls	25	5.4 ± 0.6	5.1 ± 0.52	1.7 NS
t- Value		4.76 *	6.08*	

* Significant, $P<0.01$, NS-Non significant.

Table 3. Distribution of hypocalcemic babies in study group in relation to signs and symptoms.

Sign/symptom	Number	Percentage
Jitteriness	7	58.3%
Lethargy	2	16.7%
Poor feeding	1	8.3%
Irritability	1	8.3%
Seizures	1	8.3%

possibility of asphyxia causing convulsion was ruled out by giving intravenous calcium.

DISCUSSION

In the present study, the asphyxiated infants showed lower serum ionised calcium values at birth as well as at 48 hours. Hypocalcemia in birth asphyxia is thought to be due to several factors including low calcium intake, functional hypoparathyroidism due to hypoxia, excessive bicarbonate therapy and hyperphosphatemia due to endogenous break down of glycogen and tissue proteins (5, 6). In this study bicarbonate therapy was not given to any infants. Hyperphosphatemia as a possible etiological factor contributing to hypocalcemia in birth asphyxia does not seem to hold good as in this study, phosphorous levels were comparable to non-asphyxiated infants. Thus hypoxia has played a role in causing hypocalcemia in asphyxiated infants.

Compared to total calcium levels, serial ionised calcium estimation was found to be more helpful in identifying hypocalcemic asphyxiated infants who were symptomatic otherwise and showed normal calcium levels. This observation supports the fact that

ionised calcium is the biologically active fraction. Decrease in this fraction accounts for the functional derangement like jitteriness, etc. in asphyxiated infants.

Among asphyxiated infants no significant difference was found in total as well as ionised calcium values, when TAGA infants were compared to TSGA infants (8, 9). Serum phosphorus and magnesium levels were also comparable in both these groups (10). This observation is at variance from various studies suggesting hyperphosphatemia in TSGA infants with birth asphyxia as a possible etiological factor to cause hypocalcemia.

To conclude, significantly lower serum ionised calcium levels were observed quite frequently in asphyxiated infants and this hypocalcemia causes functional derangement in many babies. Therefore, it is necessary to monitor serum calcium levels, especially ionised fraction in asphyxiated infants.

Role of prophylactic calcium supplementation in asphyxiated infants to prevent functional derangements requires further evaluation.

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