

**CHANGES IN SERUM CALCIUM AND SERUM GLUCOSE LEVELS IN ASPHYXIATED SGA**Seema Rai<sup>1</sup>, Sharanjit Kaur<sup>2</sup>, Abdul Hamid<sup>3</sup>**HOW TO CITE THIS ARTICLE:**

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**ABSTRACT: OBJECTIVE:** To study effect of birth asphyxia on serum calcium and serum glucose in serum samples of asphyxia small for gestational age (SGA) of different severity and compare with controls. **METHODS:** Serum calcium and glucose levels were estimated in the serum samples of asphyxiated SGA newborns of different grades and non-asphyxiated control group at 24 hours of age. **RESULTS:** Mean serum calcium in SGA of study group ( $8.31 \pm 0.48$  mg/dl) was lower than control SGA ( $9.47 \pm 0.49$  mg/dl)  $p < 0.01$ . Mean serum glucose in SGA of study group ( $54.4 \pm 10.91$  mg/dl) was lower than control SGA ( $76 \pm 15.5$  mg/dl)  $p < 0.01$ . The present study showed that there was significant negative correlation of serum calcium and glucose with severity of asphyxia ( $p < 0.01$ ). **CONCLUSION:** Among cases, hypocalcaemia and hypoglycaemia developed early and decrease in their serum levels was directly proportional to degree of asphyxia.

**KEYWORDS:** Hypocalcemia, Hypoglycemia, Apgar score, Perinatal Asphyxia, SGA.

**INTRODUCTION:** Small for gestational age is defined as less than 10% of predicted birth weight for that gestational age which can lead to significant mortality and morbidity if not properly managed.<sup>[1]</sup> A fetus affected by IUGR forms a subset of cases of Small for Gestational Age (SGA) infants.<sup>[2]</sup> In SGA, the estimated weight of the fetus is below the 10th percentile for its gestational age and abdominal circumference (AC) is below the 2.5th percentile.<sup>[3]</sup> In accurately dated pregnancies, approximately 80-85% of fetuses identified as being IUGR are constitutionally small but healthy, 10-15% are 'true' IUGR cases, and the remaining 5-10% of fetuses are affected by chromosomal/structural anomalies or chronic intrauterine infections.<sup>[4]</sup> IUGR can complicate 10% to 15% of all physiologic pregnancies.<sup>[5]</sup> However, it must be remembered that the incidence of such cases varies depending on the population, geographic location being scrutinized and the standard growth curves used as reference.<sup>[3]</sup>

Birth asphyxia is a common neonatal problem and contributes significantly to neonatal morbidity and mortality. Globally, hypoxia of the newborn (Birth asphyxia) or the fetus ("Fresh stillbirth") is estimated to account for 23% of the 4 million neonatal deaths and 26% of the 3.2 million stillbirths each year.<sup>1</sup> Data from National Neonatal Perinatal database (NNPD) suggests that perinatal asphyxia contributes to almost 20% of neonatal deaths in India.<sup>2</sup> Neonatal hypocalcemia and hypoglycaemia are predominant metabolic causes of seizures especially in IUGR infants.<sup>[6]</sup>

Risk of hypocalcemia and hypoglycaemia are further increased in asphyxiated infants Birth asphyxia is associated frequently with metabolic changes like hypoglycaemia, hypocalcaemia, hyponatremia, hyperphosphatemia and metabolic acidosis. Calcium is an important second messenger in our body and also helps out muscle function and acts as a co-factor for several enzymatic activities.<sup>[7]</sup>

## ORIGINAL ARTICLE

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After birth due to abrupt cessation of placental transfer of calcium hence levels starts falling to 8-9 mg/dl and ionized calcium to 4.4-5.4 mg/dl at 24 hours of age. Serum calcium then starts rising to reach levels comparable to older children and adults by two weeks of age.<sup>[8]</sup>

Glucose is an essential nutrient for the brain. Abnormally low level can cause encephalopathy and have the potential to produce long term neurological injury. Serum glucose levels decline after birth until 1-3 hours of age, when levels spontaneously increase in normal infants. In healthy term infants, serum glucose values are rarely less than 35 mg/dl between 1 and 3 hours of life, less than 40 mg/dl from 3-24 hours and less than 45 mg/dl after 24 hours of life.<sup>[9]</sup> In birth asphyxia, hypoglycaemia is due to glycogen depletion secondary to catecholamine release and to an unexplained hyperinsulinemic state. An initial phase of hyperglycemia and hypoinsulinemia (5-10 minutes following an acute event due to a catecholamine surge which inhibits insulin release and stimulates glucagon release) may be followed within 2-3 hours by profound hypoglycaemia.<sup>[10]</sup>

This study was undertaken to detect incidence of hypocalcemia and hypoglycaemia in asphyxiated SGA babies as to prevent the adverse effects of these biochemical abnormalities in the newborns.

**MATERIAL AND METHODS:** This was a hospital based case control study following simple random sampling with equal number of cases and controls, a convenient number of 74 SGA newborns were selected, 36 were asphyxiated and 38 were non-asphyxiated. This study was approved by the Institutional Ethical Committee, and informed consent was obtained from the parents of each subject. In this study, 100 asphyxiated neonates (Apgar score at one minute 7 or less) were taken as cases of study. 35 normal neonates (Apgar score at 1 minute more than 7) were taken as control. Total serum calcium and serum glucose levels were determined at 24 hours of life in all the newborns. Serum calcium was estimated by O- Cresolphthalein complexone (O-CPC) end point (kit) method (Connerty and Briggs, 1966).<sup>[6]</sup> Blood glucose estimation was done by Asatoor and King Method (Varley, 2004).<sup>[7]</sup> Babies with congenital malformations, serum creatinine levels more than 1.5mg/dl, suspected metabolic disease, treated with diuretics and those born to mothers having hypertension, diabetes mellitus, toxemia of pregnancy were excluded from the study.

**RESULTS:** In present study 74 SGA cases were selected and divided into study and control basis of asphyxia, 36 asphyxiated were study group and 38 were control group. Statistical comparison of measured values between the study and control group were done by unpaired 't' test of means. It was found that the means of serum calcium and glucose, (8.31±0.48) and (54.4±10.91) significantly ( $p<0.001$ ) lower in cases than controls (9.47±0.49) and (76±15.5) as shown in table-1. In asphyxiated SGA patients, significant positive correlation was found between serum calcium and glucose with grades of asphyxia as shown in table -2. With increase in severity of asphyxia there was significant ( $p<0.05$ ) fall in calcium except in, mildly asphyxiated newborns where it was not significant ( $p>0.05$ ) as shown in figure-1. With increase in severity of asphyxia there was significant ( $p<0.001$ ) fall in glucose as shown in figure-2. The statistical data was analysed by SPSS software of windows, version 10 and is shown in table 3 and 4.

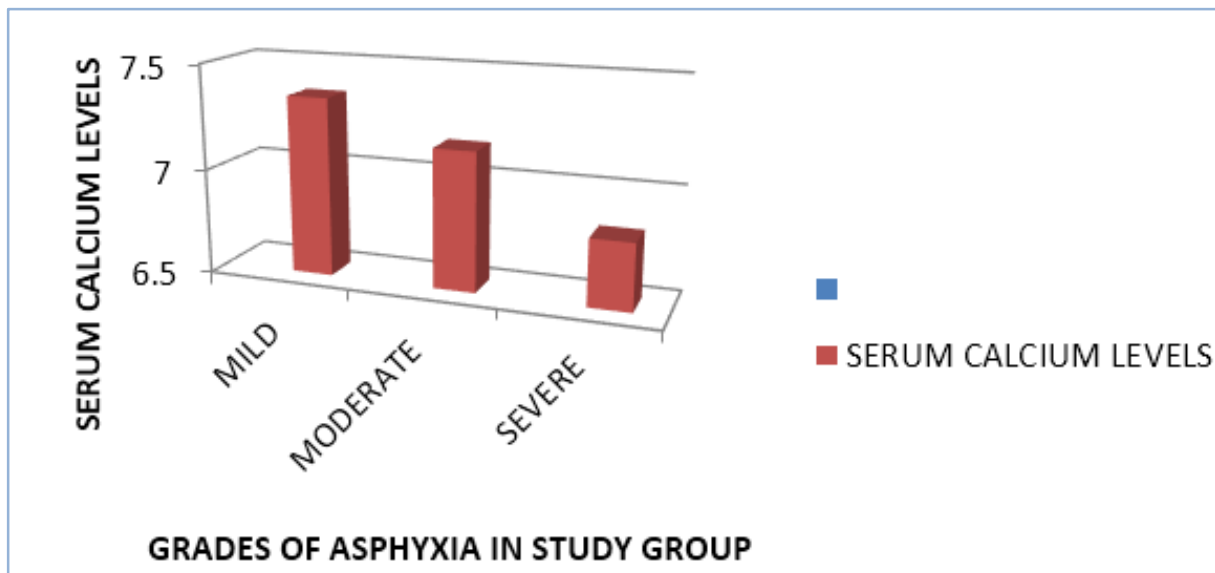
## ORIGINAL ARTICLE

	ASPHYXIATED IUGR	NON ASPHYXIATED IUGR	P value
No.	36	38	
Serum calcium levels	8.31 ± 0.48	9.47 ± 0.49	t- 12.2 <0.001
Serum glucose levels	54.4±10.91	76 ± 15.5	t- 8.97 <0.001

**Table 1: Showing serum glucose and serum calcium in asphyxiated and non- asphyxiated newborns**

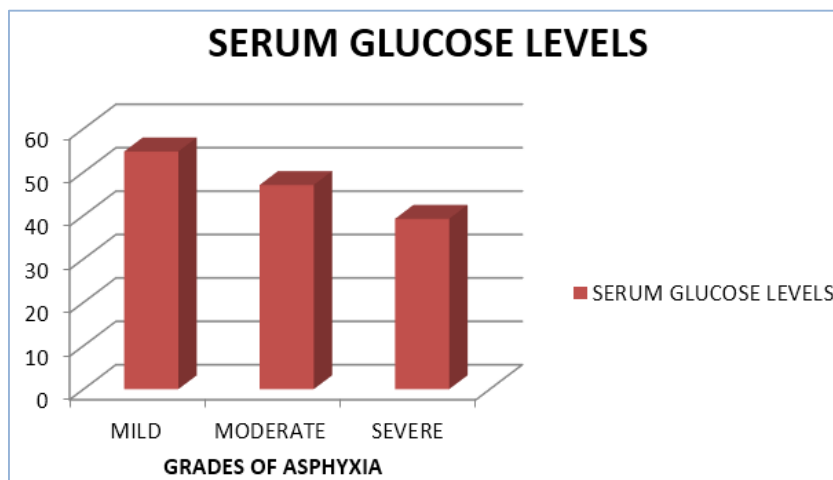
GRADING	NO.	SERUM CALCIUM LEVELS	SERUM GLUCOSE LEVELS
MILD	22	7.349±0.3399	54.785±1.771
MODERATE	11	7.157±0.09922	47.045±2.712
SEVERE	3	6.817±0.2290	39.267±1.823
TOTAL	36	7.249±0.3202	52.121±5.069
CONTROL	38	8.474±0.3640	57.409±2.302

**Table 2: Serum Calcium Levels In Study And Control According To Grades Of Asphyxia**



**Figure I: Mean Serum Calcium Levels According to Grades Of Asphyxia In Study Group**

## ORIGINAL ARTICLE



**Figure II: Mean Serum Glucose Levels According to Grades of Asphyxia in Study Group**

GRADING		
MILD X MODERATE	0.0922	> 0.05 NS
MILD X SEVERE	0.0157	< 0.05 S
MODERATE X SEVERE	0.0025	< 0.0025 VERY SIG
<b>TOTAL X CONTROL</b>	<b>&lt; 0.0001</b>	<b>HIGHLY SIGNIFICANT</b>

**Table 3: Comparison for Serum Calcium Levels in Different Grades of Asphyxia**

GRADING		
MILD X MODERATE	< 0.0001	EXTREMELY SIGNIFICANT
MILD X SEVERE	< 0.0001	EXTREMELY SIGNIFICANT
MODERATE X SEVERE	0.0006	EXTREMELY SIGNIFICANT
<b>TOTAL X CONTROL</b>	<b>&lt; 0.0001</b>	<b>EXTREMELY SIGNIFICANT</b>

**Table 4: Comparison for Serum Glucose Levels in Different Grades of Asphyxia**

**DISCUSSION:** This study was aimed to determine effect of birth asphyxia on serum calcium and serum glucose in small for gestational age newborns. This was done by comparing 36 small for gestational asphyxiated newborns with 38 non-asphyxiated small for gestational newborns.

Hypocalcemia in birth asphyxia is due to low serum calcium intake, functional hypoparathyroidism due to hypoxia and excessive bicarbonate therapy which is further exaggerated in small for gestational newborns.<sup>[11]</sup> In general, higher the calcium concentration in the umbilical cord blood, grater its decrease during the first two days of life. In asphyxiated newborns, decrease in the serum level of both total calcium and ionized calcium surpass that in non-asphyxiated newborns by approximately a third. The most striking cause for the early form of hypocalcemia are likely to be a transient hypoparathyroidism or a failure of end organ responsiveness.<sup>[12]</sup> In Present study, mean

## ORIGINAL ARTICLE

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serum calcium in SGA of study group ( $8.31 \pm 0.48$ ) was lower than control non-asphyxiated SGA group ( $9.47 \pm 0.49$ ) which was significant statistically ( $p < 0.001$ ). This is similar to that reported by Tsang et al (1975) found that total serum calcium (7.82 mg/dl) significantly lower in asphyxiated SGA babies at 24 hours of life as compared to their control group.<sup>[13]</sup> Jajoo et al (1995) also found total calcium levels (8 mg/dl) significantly low in asphyxiated SGA babies at 24 hours of as compare to their control (9.5 mg/dl).<sup>[14]</sup>

In Present study, mean serum glucose in SGA of study group ( $54.4 \pm 10.91$ ) was lower than control non-asphyxiated SGA group ( $76 \pm 15.5$ ) which was significant statistically ( $p < 0.001$ ). With increase in severity of asphyxia there was significant ( $p < 0.001$ ) fall in mean serum glucose levels. Birth asphyxia can lead to exaggerated fall in serum glucose in small for gestational newborns due to low glycogen stores in these babies.<sup>[15]</sup> Another contributory factor for hypoglycaemia in asphyxiated SGA is transient hyperinsulinemic state, deficient glycogenolysis, deficient gluconeogenesis, deficiency of oxidative enzymes for fatty acid oxidation abnormalities of counterregulatory hormoneal mechanisms like glucagon adrenalin etc. which causes further fall in the serum glucose levels.<sup>[16]</sup>

**CONCLUSION:** Among cases, hypocalcaemia and hypoglycaemia developed early and decrease in their serum levels was directly proportional to degree of asphyxia.

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