Study Of Blood Glucose Levels In Birth Asphyxiated Newborn: A Hospital Based Study

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I. Introduction:
World Health Organization Has Defined Birth Asphyxia As “Failure To Initiate And Sustain Breathing At Birth.” Perinatal Asphyxia Is One Of The Most Common Primary Cause Of Mortality (28.8%) And Morbidity Among Neonates In India And Is The Commonest Cause Of Stillbirths (45.1%). An Apgar Score Of <7 At 1 Min And At 5 Min Respectively Is Seen In 8.4% And 2.45% Cases In India. The Primary Cause Of This Condition Is Systemic Hypoxemia And/Or Reduced Cerebral Blood Flow. Birth Asphyxia Causes 23% Of All Neonatal Death Worldwide. Birth Asphyxia Is Associated Frequently With Metabolic Changes Like Hypoglycemia, Hypocalcemia, Hyponatremia, Hyperphosphatemia And Metabolic Acidosis. 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. Then Levels Spontaneously Increase In Normal Infants. In Healthy Term Infants, Serum Glucose Values Are Rarely <35 Mg/Dl Between 1 And 3 H Of Life, <40 Mg/Dl From 3 To 24 H And <45 Mg/Dl After 24 H Of Life. 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 Min Following An Acute Event Due To Catecholamine Surge Which Inhibits Insulin Release And Stimulates Glucagon Release) May Be Followed Within 2-3 Hours By Profound Hypoglycaemia. This Study Was Undertaken To Detect The Incidence Of Hypoglycemia In Asphyxiated Babies As To Prevent The Adverse Effects Of These Biochemical Abnormalities In The Newborns.

II. Materials And Methods
The Study Was Conducted On 135 Newborns Delivered In The Obstetrics Department And Admitted To Neonatology Section Of Department Of Pediatrics, Rajendra Institute Of Medical Sciences, Ranchi During July 2016 To December 2017. In A Series Of 135 Newborns Selected, 100 Newborns Were Asphyxiated, And 35 Served As Control Group. Informed Consent Was Obtained From The Parents Of Each Subject. In This Study, 100 Asphyxiated Neonates (Apgar Score At 1 Min 7 Or Less) Were Taken As Cases Of Study. 35 Normal Neonates (Apgar Score At 1 Min More Than 7) Were Taken As Control. Total Serum Glucose Levels Were Determined At 24 H Of Life In All The Newborns. Blood Glucose Estimation Was Done By One Touch Strip Glucometer. Babies With Congenital Malformations, Serum Creatinine Levels More Than 1.5 Mg/Dl, Suspected Metabolic Disease, Treated With Diuretics And Those Born To Mothers Having Hypertension, Diabetes Mellitus, Toxaemia Of Pregnancy Were Excluded From The Study.

III. Results

Figure 1: Glucose Levels In Study And Control Group

DOI: 10.9790/0853-1705070507 www.iosrjournals.org
Fig. 2: Glucose Levels According To Grades Of Asphyxia

Of the 100 cases, 55 were male newborns and 45 were females. Mean birth weight in the study group was 2405.70 ± 638.32 g. 36 newborns were delivered by normal vaginal delivery, 64 were lower segment caesarean section. The study group was comprised of 53 cases of mild birth asphyxia, 26 cases of moderate birth asphyxia and 21 cases of severe birth asphyxia.

In the control group, 18 were males and 17 newborns were female newborns. Mean birth weight was 2624.28 ± 555.76 g respectively. 29 newborns were delivered by normal vaginal delivery, 6 were by lower segment caesarean section.

IV. METHOD:

Statistical comparison of measured values between two groups were performed by the unpaired T-test of the means and ANOVA test of the groups.

In the present study, mean glucose levels of the study and control groups were 54.4 ± 10.91 and 76 ± 15.5 mg/dl respectively. The mean serum glucose level in the study was lower as compared to the control group and the statistical difference was highly significant (P < 0.001) as shown in figure 1. It was found that mean glucose level of severely asphyxiated babies was 46.8 ± 4.58 mg/dl and was significantly lower than mean glucose level of mild and moderately asphyxiated babies 59.71 ± 11.47 mg/dl and 53.69 ± 6.97 mg/dl respectively (P < 0.05). Mean glucose level of moderately asphyxiated newborns were lower than mildly asphyxiated newborns (P < 0.05). This shows that glucose levels at 24 h of life were inversely related to severity of asphyxia (P < 0.05) as shown in figure 2. Serum glucose level had significant positive correlation with gestational age and birth weight (P < 0.01) and significant negative correlation with asphyxia.

V. DISCUSSION

Perinatal asphyxia is a common neonatal problem and contributes significantly to neonatal mortality and morbidity. Hypoxic ischemic brain injury is the most important consequence of perinatal asphyxia.

Out of 100 cases, 44 babies had fetal asphyxia. Babies with Apgar score ≤7 were defined as asphyxiated and babies with Apgar score more than 7 constituted the control group. All the neonates (both study group and control group) were subjected to determination of glucose at 24 h of life.

In the present study, decrease in the serum glucose in asphyxiated babies was directly proportional to the degree of asphyxia. This is similar to that observed by Xu et al. and Davis et al. observed in their study, at day 1 of life. In asphyxiated neonates, temporary hyperinsulinism contributed to hypoglycaemia in these babies. Davis et al. had also reported in their study that there was severe asphyxia. Singhal et al. in their study concluded that out of 2/3 hypoglycaemic babies, birth asphyxia contributed to 24.2% of cases. The present study showed that there was significant negative correlation between serum glucose level and severity of asphyxia (P < 0.01), whereas highly significant positive correlation of serum glucose with gestational age and birth weight (P < 0.01).

VI. CONCLUSION

It can be concluded from the present study that with perinatal asphyxia, hypoglycaemia (when serum glucose < 40 mg/dl) after birth is directly proportional to severity of asphyxia. Severely asphyxiated babies develop hypoglycaemia which may require medical intervention.
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References