Neonatal Hypocalcemia - A Study of 43 Cases in Dinajpur Medical College

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Neonatal seizure is a common manifestation of neurological dysfunction. It constitutes a medical emergency requiring urgent etiology specific therapy to prevent further brain injury. The aim of this study was to observe clinical presentation, etiology and outcome of neonatal hypocalcemia. This was an observational study done in the department of pediatrics, Dinajpur medical College Hospital from January 2012 to December 2012. Out of 1280 total admitted newborns, convulsion developed in 130 cases. Among the 130 cases neonatal hypocalcemia (total serum calcium <7 mEq/L) found in 43 (33%) cases. Term normal birth weight newborns(58%) were the main the victim. Hypoxic Ischaemic Encephalopathy (HIE) was the main cause of hypocalcemia in majority (67%) cases followed by infection. Convulsions were well controlled by injection Calcium Gluconate 1 mg/kg 6 hourly following a bolus dose of 2 mg/kg intravenous calcium gluconate. Majority required more than 2 days intravenous calcium gluconate

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Introduction

Biochemical disturbances occur frequently in neonatal seizure. It is difficult to control seizure without correcting biochemical disturbances.[1] Early detection and prompt treatment of biochemical disturbances are essential for optimal management and satisfactory longterm outcome.[2,3,4] Neonatal hypocalcemia is a very common metabolic cause of convulsion. It is clinically divided into early and late presentation. Early neonatal hypocalcemia is defined as occurring within first 48 hrs of life and causes are prematurity, intrauterine growth retardation and birth asphyxia.[5] Maternal factors including gestational diabetes, vitamin D deficiency and hyperparathyroidism can also cause early neonatal hypocalcemia. Late neonatal hypocalcemia is defined as hypocalcemia presenting with seizure after 3rd day of life.[6] Hypocalcemia is defined as total serum calcium <7 mg/dl or ionized calcium <4 mg/dl.[7] Ionized calcium is the preferred mode in defining hypocalcemia. Though several studies have been carried out in our country on neonatal hypocalcemia, very few references exist on it. It is very important to make up an study on neonatal hypocalcemia as because early recognition and treatment of hypocalcemia can significantly influence the better outcome.

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Methods

This study was done in Pediatric department of Dinajpur Medical College Hospital during January, 2012 to December, 2012. Neonates developed overt convulsion prior or after admission showing serum calcium ‹7mg/dl were enrolled within the study. A structured form containing gestational age, birth weight, time of onset of seizure, type of seizure, obstetric history including maternal blood sugar status, drug response and clinical presentation was made and data were taken from hospital records. EEG criteria of hypocalcemia as well as electrical seizure (EEG) were not utilized due to poor facility. Intravenous 10% calcium gluconate 2 ml/kg diluted with equal volume of normal saline or 5% DA as a bolus dose followed by 1 ml/kg 6hourly doses were given. Monitoring of heart rate always done during intravenous calcium therapy. Continuous therapy was not given randomly to prevent tissue necrosis following accidental leakage of infusion fluid. Continuous therapy was given to only severely hypocalcemic babies. All data records and investigation records were examined and statistical analysis was done.

Results

Out of 43 cases of neonatal hypocalcemia, seizure developed during first 24 hrs of life among 53% cases. Term babies (58%) having normal birth weight (60%) were the main victims. The main causes were HIE (67.5%) and IUGR (23%). Subtle seizures were noted among 59.5% patients and carpopedal spasms were observed in 35% cases. Reduced spontaneous movements were noted in 83.5% cases. Serum calcium level was <6 mg/dl in 36% and <7 mg/dl in 64% cases. Seizures were controlled by equally diluted intravenous 10% calcium gluconate 2 ml/kg bolus dose. In some cases intravenous Phenobarbitone 20 mg/kg bolus dose was given where HIE was the cause. Serum calcium was maintained by intravenous calcium 1 ml/kg/6 hourly for next 2-3 days .

Table I: Distribution of gestational age and birth wt in neonatal hypocalcemia

Birth weight in gm	Seizures Number (%)
≤2500	26 (60)
1500 - 2499	15 (35.5)
> 1499	2 (4.5)
Gestational age in week	
<37	25 (58)
34 - 37	14 (32.5)
>34	4 (9.5)

Table II: Time of onset of seizure (n=4)

Time	Seizures Number (%)
> 24 hr	23 (53)
24 – 28 hr	18 (42.5)
< 48 hr	2 (4.5)

Table III: Types of seizure (n=43)

Types of seizure	Seizures Number (%)
Subtle	26 (59.5)
Tonic	15 (35)
Clonic	2 (4.5)
Myoclonic	0 (0)

Table IV: Duration of Convulsion (n=43)

Duration in minute	Seizures Number (%)
>5	29 (67.5)
5 - 30	14 (32.5)

Table V: Clinical Manifestation (n=43)

Clinical Features	Seizures Number (%)
Reduced spontaneous movement	36(83.5)
Feeding problem	38(8)
Carpopedal Spasm	15(3)
Stridor	2(4.5)
Irritability/Excessive cry	9(21)
Abdominal Distension	5(12)

Table VI: Serum Calcium Level (n=43)

Serum level of calcium	Seizures Number (%)
>7 mg/dl	27(64)
> 6 mg/dl	16(36)

Table VII: Etiology of Neonatal Hypocalcemia (n=43)

Etiology	Number (%)
HIE	29(67.5)
IUGR	10(23)
Infection	2(4.5)
Infant of Diabetic mother	1(2.5)
Others(undetected)	1(2.5)

Discussion

In this study only biochemically proved neonatal hypocalcemia were studied. Kumar et al showed that primary metabolic disorder accounted for 25% causes of neonatal convulsion.[7] Cockburn et al found primary disturbance of mineral metabolism were responsible for 55% cases of neonatal convulsion over a long 2 yrs study.[6] Metabolic causes of convulsion is very common in Bangladesh. Faulty feeding practices in addition to high incidence of HIE which predisposes to hypocalcemia is likely the explanation.[8] This is in contrast to report from western countries is very uncommon due to less incidence of HIE and proper feeding practices.

Hypocalcemia is the most common metabolic error in term babies in this study as 60% cases were term babies. Almost similar finding were found by Kumar et al.[7] Arvind Sood et al found hypoglycemia as the most common cause.[5] Hypocalcemia is common between 12-72 hrs of life specially in asphyxiated baby, preterm babies and babies of diabetic mothers which is well consistent with this study.[6,7,8] Definitions of neonatal hypocalcemia is very controversial in literatures.[9,10] Some authors have defined it as <8 mg/dl, <7.5 mg/dl, <7 mg/dl. In this study neonatal hypocalcemia was defined as plasma calcium level less than 1.75 mmol/l (7 mg/dl). .

Duration of convulsion has prognostic value. Prolonged seizure lasting >30 min bears a bad prognosis.[11] In this study, 67.5 % babies had < 5 min duration convulsion and 32.5% had 5-30 min convulsion. Clinical observation without EEG monitoring may underrate the total incidence and duration of convulsion .EEG monitoring was not available in this setting.

Stoliar et al studied 115 neonatal seizure showing higher incidence of hypocalcemia in full term babies.[11] Jajoo et al showed 13 out of 35 babies with convulsion had serum calcium level <7.5 mg/dl.[12] Tsang et al showed reduced spontaneous movement and feeding problem were the main clinical presentation.[13] Carpopedal spasms were found in 33% cases as described by Jajoo et al which is more or less consistent with our finding.[12] Intravenous calcium gluconate was given very cautiously under close monitoring of heart rate. 64% patients were given calcium as 6hrly dosage following bolus dose and 36% patients who showed S. calcium <6 mg/dl were given continuous infusion. Clinical improvement and normalization of serum calcium did not vary considerably. Findings of our study were consistent with majority study groups with some negligible variation which may be attributed to racial and geographical issues. A study with more number of cases over prolong period might overcome these

Conclusion

This observational study revealed HIE as the main cause of neonatal hypocalcemia which is largely preventable by improving obstetric care and mass people awareness regarding safe delivery. Intravenous calcium gluconate can be safely given 6 hourly as bolus dosage to prevent accidental tissue damage. All

categories of hypocalcemia should be treated for 72 hrs to prevent recurrence of seizure and further neuronal damage which might add to more favorable outcome.

References

1. Halsam RHA. Neonatal seizure. In: Behrman RE, Kliegman RM, Jenson HB. Nelson text book of pediatrics, 17th edn. Philadelphia, W B Saunders Company .2004. p.1813-28.
2. Rennic JM. Neonatal neurology. In: Mclntosh N,Helms PJ, Smyth RL. Forfar and Arneil's Textbook of Pediatrics, 6th edn. Newyork, Churchill Livingston.2003.p. 244-49.
3. Gupte S. Neonatal convulsions. In: Gupte S. The short textbook of pediatrics, 4 th edn.New Delhi, Jaypee. 2003.p. 342.
4. Seay AR, Bary PF. Significance of seizures in infants weighing <2500 gms. Arch-neurol 1977; 34:381-82.
5. Sood A, Grover N, Sharma R. Biochemical abnormalities in neonatal seizures. Indian J Pediatr 2003; 3:221-24.
6. Brown JK, Cockburn F, Forfur JO. Clinical and chemical correlaters in convulsions of the newborns. Lancet 1972; 1:135-39.
7. Kumar A, Gupta V, Kachhawaha JS, Singla PN. Biochemical abnormalities in neonatal seizure. Indian J of pediatr 1995; 323: 424-28.
8. Kliegmen RM. Fetal and Neonatal Medicine. In : Behrman RE, Kilegmen RM. Nelson Essentials of Paediatrics,10thedn.Philadelphia,WB Sunders company. 2002. p.202-206.
9. Cockburn F, Brown JK, Belton NR, Forfar JO. Neonatal Convulsions associated with primary disturbance of calcium, phosphorous and magnesium metabolism. Arch of disease of childhood 1973; 48:99-108.
10. Raisz LG. Physiology and pharmacologic regulation of bone absorption. N Engl J Med 1997; 282:909-15
11. Stoliar O, Largnia M, Languia AE, Ruiz B. Studies in 115 neonates with one minute Apgar score of three or less: early neonatal hypocalcaemia. J Pediatr 1971; 78:906.
12. Jajoo D, Kumar A, Shankar R, Bhargana V. Effect of birth asphyxia in serum calcium levels in neonates. Indian J pediatr 1995; 62: 455-9.
13. Tsang RC, Chen IVY, Hayes W, Atkinson W, Atherton H, Edward N. Neonatal hypocalcaemia in infants with birth asphyxia. J Pediatr 1974; 48:428-33.