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Case Report

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A Rare Case of Refractory Neonatal Hypocalcaemia Due to Hypomagnesemia Presenting with Feed Intolerance

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Abstract

Hypocalcaemia is a common metabolic problem for some at risk newborns admitted in NICU and SCANU.Most of the cases of hypocalcaemia usually respond with intravenous calcium administration. Hypocalcaemia not responding with adequate dose and duration of calcium, requires further evaluation for refractory hypocalcaemia. Hypomagnesemia is one of the causes of refractory hypocalcaemia. The association of hypomagnesemia with hypocalcaemia is well known for the past few years and in most of the cases disturbances of magnesium homeostasis is secondary to obvious causes. There are few case reports on primary hypomagnesemia due to disorder of magnesium metabolism [1,2]. Neonates who are both hypomagnesemic and hypocalcaemic, respond only to administration of intravenous magnesium. Nowadays hypomagnesemia is reported in neonates and during early infancy as a result of specific malabsorption of magnesium [3].

We describe a rare case of hypocalcaemia due to hypomagnesemia in a term newborn, diagnosed in SCANU of Kurmitola General Hospital, Dhaka, Bangladesh. This term, asymmetric IUGR, male newborn, was admitted with the diagnosis of perinatal asphyxia hypoxic ischaemic encephalopathy stage 1. During the hospital course the baby developed features of feed intolerance due to hypocalcaemia and hypomagnesemia. We treated the patient with intravenous calcium to which the patient was not responding, later on dramatic improvement was seen with intravenous magnesium.

Introduction

Abnormalities of magnesium and calcium metabolism are commonly seen among the admitted newborns in NICU and SCANU. Calcium disturbances is mirror image of disturbances of magnesium, as seen in hypocalcemia with hypomagnesemia or hypercalcemia with hypermagnesemia. Abnormalities in serum values for Ca2+ and Mg2+ are of great concern in any newborn and warrant further investigation.

Hypocalcemia in neonates is defined as total serum calcium concentration values of <2.0 mmol/L (<8.0 mg/dL) for a term infant or <1.75 mmol/L (<7.0 mg/dL) for a preterm infant [4].

About 80% of the calcium transfer across the placenta occurs in the last trimester that's why hypocalcaemia is invariably found in preterm babies. Parathyroid hormone-related peptide plays the main role in calcium balance in the placenta. Serum calcium level in neonates depends on certain factors like parathyroid hormone (PTH), renal and skeletal factors. Based on the timing of the presentation, hypocalcemia can be early onset (first 72 hours of life) and late onset (develops after 72 hours of life). Causes of hypocalcemia include prematurity, low birth weight, intrauterine growth restriction, perinatal asphyxia, infant of diabetic mother and preeclamptic toxemia in the mother, nutritional deprivation, hypomagnesemia, hypoparathyroidism, maternal hyperparathyroidism [5].

Normal serum levels for Mg2+ are typically 0.6 to 1.0 mmol/L (1.6–2.4 mg/dL). Hypomagnesemia is usually seen as any value <0.66 mmol/L (1.6 mg/dL). clinical signs do not manifest until levels drop below 0.5 mmol/L (1.2 mg/dL) [4].

Hypomagnesemia shares more or less same risk factors as hypocalcaemia. Risk factors are hypocalcemia, preterm and low birth weight,inadequate intake of magnesium, infant of diabetic mother, fetal growth restriction, especially if mother had preeclampsia,inherited renal wasting eg, Gitelman syndrome, hypoparathyroidism, hypocalciuria and nephrocalcinosis,magnesuria secondary to furosemide or gentamicin administration ,citrated blood exchange transfusions [4].

Clinical presentation is similar to hypocalcemia like jitteriness, apnea, seizure, feeding intolerance. If symptoms persist after adequate calcium gluconate therapy, hypomagnesemia should be considered.

We describe a case of refractory hypocalcaemia due to hypomagnesemia, presented with features of feed intolerance in Special Care Newborn Unit (SCANU) of Kurmitola General Hospital, Dhaka, Bangladesh. To identify the cause sepsis screening, blood culture and sensitivity, serum electrolytes were done.All the reports were normal except serum calcium which was repeatedly low. Later on serum magnesium was done which

was low. We studied the clinical profiles and outcomes of neonate with refractory hypocalcaemia.

Case Presentation

Our patient was term male newborn, 2nd issue of non-consanguineous parents, weighing 2000g.He was born to a 30 year old lady, who was on regular antenatal check-up. Mother had history of pregnancy induced hypertension and developed prolonged labour pain. USG of pregnancy profile showed oligohydramnios. Baby was delivered at 39 weeks by LUCS due to oligohydramnios, less fetal movement and uncontrolled pregnancy induced hypertension.Baby was admitted in SCANU after birth. due to perinatal asphyxia.Baby was kept nothing per oral; respiratory support was given in the form of oxygen by nasal cannula at the rate of 2l/min and treated by antibiotic Ceftazidim and Amikacin. Feed was started at 26hrs of age but at 50hrs of age baby developed features of feed intolerance in the form of abdominal distension and vomiting, which was not bile stained. Apart from features of feed intolerance there's no other sign & symptoms of sepsis and stool color was normal. Baby was kept nothing per oral and parental nutrition was given.Sepsis screening,blood CS,serum electrolytes, abdominal X-ray reports were normal. Serum calcium was 7.2 mg/dl. Intravenous calcium gluconate was given for 48 hrs. After that serum calcium was repeated, which showed 6.4 mg/dl. So Intravenous calcium was again given for 48hrs and repeat serum calcium was 6.8 mg/dl. The dose of intravenous calcium was checked by two doctors before administration each time. Serum magnesium, serum PTH and 1,25(OH) Vit D was sent to laboratory. Serum magnesium was 1.2 mg/dl, serum PTH and 1,25(OH) Vit D was within normal limit.

Inj magnesium sulphate was given in proper dose and duration. It usually given as 25-50mg/kg/dose (0.2–0.4 mEq/kg/dose) IV every 8 to 12 hours for 2 to 3 doses until magnesium level is normal or symptoms resolve. We gave Inj. magnesium sulphate 25 mg/kg/dose 8hrly for 3 doses.During administration of magnesium sulphate baby was closely monitored to avoid potential complications of magnesium sulphate infusion. Repeat serum magnesium and calcium was 1.5 mg/dl and 7.1 mg/dl respectively.So another 3 doses of magnesium sulphate was given.Repeat serum magnesium was 1.9 mg/dl and serum calcium was 8.1 mg/dl. There was gradual improvement of abdominal distension, abdominal girth was decreased and gastric aspirate was nil after completing the magnesium sulphate dose. Feed was resumed and baby's reflex activity was good. We were able to discharge the patient after 10 days of age with advice.

Discussion

Calcium and magnesium are important for many physiological processes including haemostasis, hormone secretion and action, enzyme activation and inhibition, nerve conduction, transmission across cellular membranes and muscle contraction. Calcium is also key to the formation of mineralised connective tissue that provides the skeleton with its structural integrity. Fetus accumulates 30 g of calcium by term, with 80% of this process occurring during the third trimester. Active transport of calcium and phosphate to the fetus across the placenta is driven by a magnesium adenosine triphosphate dependent pump.

Hypocalcaemia and hypomagnesemia share more or less same risk factors and symptoms. The main risk factors for both hypomagnesemia and hypocalcaemia in newborn are maternal diabetes & pregnancy induced hypertension, prematurity, fetal growth restriction, perinatal asphyxia, diuretics, rapid extracellular volume expansion and immature tubular function [2] Moreover, recent genetic studies have identified several Mg transporting proteins, whose alteration may affect magnesium homeostasis. In our patient, some risk factors for hypomagnesemia and hypocalcaemia were present like maternal pregnancy induced hypertension, fetal growth restriction and perinatal asphyxia.

Initially we tried to treat hypocalcaemia with intravenous calcium, but both the clinical symptoms and biochemical parameters were not responding. Later on we treat the baby with intravenous magnesium to which the baby responded very well. Both clinical and biochemical parameters improved dramatically.

The clinical features include irritability, tremors, twitching and seizures, lethargy, poor feeding, abdominal distension, vomiting. Our patient presented with features of feed intolerance in the form of abdominal distension,vomiting and increased gastric aspirate.

Agarwal el al. described a case report of a 6-week-old baby born to a consanguineous parent presented with repeated tonic clonic seizure since 4 weeks of age, not responding with any anticonvulsant. They diagnosed the case as primary hypomagnesemia with secondary hypocalcaemia and treated with intravenous magnesium to which the baby responded [6].

Another case report highlighted that infantile hypomagnesemia may be associated with symptoms and signs mimicking sepsis and seizures. They reported a case of 26days old term neonate presented with apnea, cyanosis, nasal bleeding, and diminished alertness. Laboratory examination revealed severe hypomagnesemia and hypocalcemia, which were promptly ameliorated following intravenous administration of magnesium and calcium [7].

The above-mentioned cases were Familial hypomagnesemia with secondary hypocalcemia, a rare autosomal recessive disorder caused by mutations in the gene encoding transient receptor potential melastatin 6 on chromosome 9q22, a channel involved in epithelial magnesium resorption [7]. On the other hand, in our case the baby born to non-consanguineous parents and there was no family history of such type of illness.

Capillari et al described a case of neonatal seizure due to transient neonatal hypomagnesemia due to a transient immature tubular function, considering that no risk factor for magnesium deficiency was present [8].

Seizure related to hypomagnesemia not responding to conventional anticonvulsants is found in some case reports and case series but features of feed intolerance associated with hypomagnesemia is not frequently seen.

So, feed intolerance not responding with gut rest, with normal septic screening and serum electrolytes reports after excluding common causes hypomagnesemia should be considered.

Conclusion

Calcium and magnesium are important for many physiological processes. Hypomagnesemia presents clinical findings indistinguishable from those of neonatal hypocalcaemia. Hypomagnesemia may cause hypocalcemia. So, whenever we get a case of hypocalcaemia not responding to intravenous administration of calcium gluconate, the possibility of hypomagnesemia should be considered.

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