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Case Report



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Hypermagnesemia–induced pseudonecrotizing enterocolitis and respiratory distress in a preterm infant: A case report

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ABSTRACT

Rationale: This case highlights the rare yet critical diagnosis of hypermagnesemia in a preterm infant, who presented with symptoms mimicking necrotizing enterocolitis. It underscores the importance of considering hypermagnesemia as a differential diagnosis in neonatal respiratory distress and gastrointestinal symptoms, even when the cause is not immediately apparent.

Patient's Concern: An extremely low birth infant with respiratory distress further had episodes of apnoea and cyanosis. The infant eventually exhibited abdominal distension and bilious vomiting, symptoms mimicking necrotizing enterocolitis.

Diagnosis: The infant's clinical features were attributed to hypermagnesemia, despite no magnesium being administered and no apparent cause identified. Screening the apparently asymptomatic mother confirmed a serum magnesium level of 4 mg/dL, suggesting transplacental transfer as the likely cause of the neonate's condition.

Interventions: The newborn was treated supportively with adequate hydration and maintenance calcium gluconate.

Outcomes: Magnesium levels decreased, and the baby showed signs of improvement.

Lessons: Hypermagnesemia, though uncommon, should be investigated and considered even when the symptoms are not apparent.

KEYWORDS: Necrotizing enterocolitis; Sepsis; Preterm; Respiratory distress; Calcium

1. Introduction

Hypermagnesemia in infants is usually caused due to an increased magnesium load or reduced renal magnesium excretion due to

prematurity and asphyxia[1]. Hypermagnesemia, potentially fatal if overlooked, is a rare condition whose symptoms such as respiratory depression, hypotonia, hypotension, bradycardia mimicking septic shock syndrome or gastrointestinal hypomotility mimicking intestinal obstruction, are fairly ambiguous and are often seen in premature neonates[1–3]. This urges a vigilant eye considering the possibility of hypermagnesemia in infants. Early diagnosis is a key factor in treating hypermagnesemia, as this illness is known to have a poor prognostic factor if left untreated[4].

Here, we present a case of a newborn with hypermagnesemia with significant respiratory depression and gastrointestinal hypomotility mimicking necrotizing enterocolitis (NEC).

2. Case report

Written informed consent was obtained from the patient's parents for participation and case report publication.

An extremely low birth weight preterm neonate weighing 1430 grams was delivered at 31 weeks of gestation through an emergency caesarean section due to preterm premature rupture of membranes with fetal distress. The baby had a spontaneous cry with good respiratory efforts but had respiratory distress in the form of nasal

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flaring and chest indrawing with a Silvermann-Anderson score of 5. Continuous positive airway pressure (CPAP) at a positive end-expiratory pressure of 5 cm water was given in the delivery room for 30 minutes. As the distress did not subside, the baby was shifted to NICU and continued on CPAP at a positive end-expiratory pressure of 5 cm water. The baby was started on intravenous 10% dextrose with maintenance calcium gluconate. Prophylactic 1st line antibiotics ampicillin and gentamicin, as per unit policy, were administered considering spontaneous premature delivery. By the 2nd day of life, the baby was started on minimal enteral nutrition with expressed breast milk. The infant initially showed improvement and was gradually weaned off bubble CPAP on the 3rd day of life. The patient was relatively stable until the 3rd day of life.

Despite a negative sepsis screen, sterile blood culture, and normal chest radiograph, the infant worsened by the 4th day of life with marked episodes of apnoea and cyanosis. The infant eventually exhibited abdominal distension and bilious vomiting, symptoms mimicking NEC. The radiograph suggested gaseous distension of the small bowel with inter-bowel free fluid (Figure 1). Sonography revealed indistinct echogenic foci in the periphery of the left lobe of the liver, suggestive of portal venous gas, gaseous distension of small bowel loops, and faecal loading in large bowel loops, with mild inter-bowel free fluid. Considering NEC stage 2B as per modified Bell's staging, the infant was kept nil per oral, and metronidazole was added. Serial blood cultures were sterile, and inflammatory markers were normal. These complications were initially attributed to prematurity, prompting the decision to initiate total parenteral nutrition. The baseline investigations are summarised in Table 1.

Pre-TPN assessments showed hypermagnesemia, with a 5.6 mg/dL serum magnesium level. A repeat test also indicated high levels of magnesium. Notably, there was no history of magnesium-containing compounds administered to the mother. The baby had not received any exogenous fluids/therapy containing magnesium. The other causes of neonatal hypermagnesemia were looked for; baseline investigations revealed normal renal function with good urine output. The baby had normal vitamin D, parathyroid hormone, and calcium levels. Since no cause for hypermagnesemia could be delineated, the possibility of maternal hypermagnesemia was considered, and investigations sent in an otherwise asymptomatic mother revealed hypermagnesemia of 4 mg/dL.

Subsequently, the newborn was treated supportively with adequate hydration and maintenance calcium gluconate. Magnesium levels decreased, and the baby showed signs of improvement, strongly indicating the cause of symptoms was hypermagnesemia. As the baby clinically improved, he was initiated on enteral feeds; breastfeeding was initiated by the 9th day of life. Along with feed fortification, the baby had consistent weight gain and was



Figure 1. Radiograph showing gaseous small bowel distension with mild inter-bowel free fluid.

Table 1. Initial laboratory parameters.

Laboratory parameters	Results	Reference range
Hemoglobin, g/d	17.3	15.0-24.0
Hematocrit, %	53.5	44.0-70.0
Total leucocyte Count, μ L	9200.0	9100.0-34000.0
Platelets, μ L	151000.0	84000.0-478000.0
C-reactive protein, mg/L	0.5	0.3-6.1
Sodium, mmol/L	138.0	133.0-146.0
Potassium, mmol/L	4.3	3.0-6.0
Chloride, mmol/L	101.0	95.0-105.0
Total serum calcium, mmol/L	2.3	2.1-2.7
Magnesium, mg/dL	5.6	1.2-2.6
Vitamin D, nmol/L	61.3	>50.0
Parathyroid hormone, pg/mL	15.2	15.1-87.7
Creatinine, μ mol/L	61.2	28.3-81.3
Urea, mmol/L	5.9	1.0-8.2
ALT, IU/L	8.5	5.0-33.0
AST, IU/L	40.0	32.0-162.0
ALP, IU/L	236.0	90.0-273.0
Total protein, g/L	66.0	46.0-74.0
Albumin, g/L	38.0	33.0-45.0
TSH, mIU/L	1.8	<37.0

ALT: Alanine transaminase; AST: Aspartate transaminase; ALP: Alkaline phosphatase; TSH: Thyroid stimulating hormone

discharged on the 43rd day of life. The baby is thriving well on monthly follow up.

3. Discussion

Neonatal hypermagnesemia is defined as serum Magnesium above 3 mg/dL[5]. The most common cause in a neonate is antenatal magnesium sulphate administration[2]. Other causes include newborn magnesium therapy, decreased renal magnesium

excretion due to prematurity, and asphyxia. Notably, in our case, the mother did not undergo any form of magnesium sulphate therapy nor had any comorbidities. One potential cause of maternal hypermagnesemia is maternal renal insufficiency or renal failure, which can lead to decreased excretion and subsequent accumulation of magnesium[6]. However, a workup in the mother revealed normal kidney functions with normal urine output. Mother received fluid flush therapy, resulting in normalised magnesium levels, and was opined to have idiopathic hypermagnesemia by the nephrologist. Further analysis was planned but not conducted due to financial constraints. Hypermagnesemia noted in this infant could be attributed to either the abnormal massive shift of Mg from the intracellular fluid to the extracellular fluid and perturbations in the Mg influx/efflux regulation systems due to non-oliguric hypermagnesemia or could be secondary transplacental transfer from mother[2,7]. This case has shown the presence of hypermagnesemia with few apparent reasons, emphasising the need for a comprehensive evaluation in neonates and mothers when hypermagnesemia is suspected.

While further genetic analysis and investigations may provide additional insights into the underlying mechanisms of hypermagnesemia in such cases, supportive treatment remains paramount; however, in severe cases, dialysis or exchange transfusion is needed[2].

Overall, this case report emphasises the need for heightened awareness and consideration of this condition in clinical practice, particularly in neonatal intensive care settings where prompt diagnosis and management are crucial for optimising outcomes.

Conflict of interest statement

The authors report no conflict of interest.

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Data availability statement

The data supporting the findings of this study are available from the corresponding authors upon request.

Authors' contributions

NSK analysed patient data and contributed to writing the manuscript. DRR and NK analysed and interpreted the patient data, managed the case and contributed to writing the manuscript.

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