SYMPTOMATIC TRANSIENT IDIOPATHIC HYPOMAGNESAEMIA IN A NEONATE

Dear Sir,

Neonatal seizures are common emergencies encountered by a paediatrician or neonatologist. Among the various causes of neonatal seizures, hypocalcaemia and hypomagnesaemia carry a good prognosis. Magnesium plays an important role in the stabilisation of membranes and nerve conduction. Small intestine is the major site of its absorption, and magnesium homeostasis (normal serum magnesium concentration 0.62–0.94 mmol/L) is mainly regulated by renal excretion.[1-4]

In most cases of neonatal hypomagnesaemia, the aetiology is either gastrointestinal or renal loss, and the underlying disease may be genetic, acquired or iatrogenic (prolonged use of magnesium-free parenteral fluids, drugs, insulin administration, exchange transfusion or hypercalcaemia).[1-4] It may be apparently but not truly idiopathic, as in association with prematurity, low birth weight, intrauterine growth restriction and diabetic mothers, where the exact pathogenesis for the hypomagnesaemia may not be immediately apparent.[4-6] These idiopathic events are usually transient, and the fall in serum magnesium is typically less severe than magnesium transport defects.[1-6] The symptoms of hypomagnesaemia are lethargy, muscle cramps, tetany and seizures. One important guide for the aetiology of hypomagnesaemia is the fractional excretion of magnesium (FeMg). In children, FeMg range is 1%–8%. A low FeMg suggests extrarenal losses, whereas a high FeMg suggests renal loss of magnesium.[1-4]

The index case of hypomagnesaemia was born to a booked, normally nourished, non-anaemic 23-year-old primigravida at 38 weeks of gestation after an uneventful antenatal period. There was no medical illness in the mother. Hypertension and gestational diabetes mellitus had been ruled out during antenatal visits. There was no consanguinity or family history of hypomagnesaemic illnesses, epilepsy, developmental delay, renal or gastrointestinal diseases. The baby was a healthy 2.75-kg boy with normal Apgar scores, facies and head circumference. He had been on exclusive breast feeds since birth and presented with poor sucking and a shrill cry on Day 8 of life. He developed multifocal spasms (seizures) and was given 10% calcium gluconate at 2 mL/kg, as he was euglycaemic. The non-invasive blood pressure recording immediately after the seizure activity was normal at 56/23 mm Hg. Pre-bolus serum calcium (both total and ionised) and magnesium samples were collected, which revealed total calcium, ionised calcium and magnesium levels as 9.5 mg/dL, 4.5 mg/dL and 0.48 mmol/L, respectively. Sepsis and meningitis were ruled out by negative sepsis screen, sterile blood culture and normal cerebrospinal fluid examination findings. Ultrasonography of the skull was also normal. Seizures recurred twice by the time serum magnesium report was received. The baby received 0.2 mL/kg of 50% magnesium sulfate, which controlled the seizures, and the baby became well with no fresh seizures and good activity/suck within 12 hours of magnesium administration. Serum magnesium was found to be normal after 12 hours of the first injection (1.11 mmol/L) and again after seven days (0.82 mmol/L). Thus, magnesium was discontinued.

A diagnosis of transient symptomatic hypomagnesaemia was made. Normal serum calcium, inorganic phosphorus, alkaline phosphatase, sodium, potassium, creatinine, blood gases, urine routine microscopy and renal ultrasonography, along with a normal FeMg (3.3%), ruled out most causes of hypomagnesaemia. The mother's serum magnesium, calcium, phosphorus, alkaline phosphatase and parathormone were also within the normal range. Thus, we were left only with the idiopathic variety, but peculiarly, there was no risk factor. This baby was still on follow-up at 6.5 months of age, when this report was being written. At 6.5 months, he weighed 8 kg, the head circumference was 42.3 cm, and he was achieving his developmental milestones normally in all spheres (he could sit well with support and transiently without support, could feed himself a biscuit, babbled [monosyllables] and patted his own image in the mirror). The serum for magnesium level was re-tested at 6.5 months of age in view of parental concerns, and it was found to be 0.93 mmol/L. These facts confirm the transient nature of the disturbance during the neonatal period.

This letter describes an unusual case of neonatal seizure in an otherwise healthy newborn, where the aetiology was hypomagnesaemia and the event was transient and truly idiopathic, i.e. without any risk factor.

Yours sincerely,

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