

Hypocalcaemic fits not responding to intravenous calcium therapy

T M R Gunasekera¹, P A D U Chathuranga², P K Banduthilake³, D H Karunathilake⁴ and W Pathirana⁵

Introduction

Symptomatic hypocalcaemia secondary to hypomagnesaemia is a well known phenomenon. It manifests in many different ways. Unless the deficiency of magnesium is detected and treated early, long term neurodevelopmental sequelae may be unfavourable. Estimation of serum calcium and magnesium in all neonates and infants presenting with seizures of obscure aetiology should be routine. Where facilities for routine magnesium estimation are not available, even a single finding of hypocalcaemia warrants assessment of serum magnesium level in a symptomatic infant. Here we report two infants with primary hypomagnesaemia presenting with convulsions during infancy.

Case reports

Case 1

A 16-day old male neonate was admitted with

convulsions since day 12 of life. He is the first born to second degree consanguineous parents who are healthy. He was born at term with a birth weight of 2.8kg, and an uneventful perinatal period. The clinical examination was unremarkable. The biochemical investigations showed serum calcium of 1.58 (normal 2.2-2.7) mmol/l and a serum magnesium of 1.6 (normal 1.7-2.7) mg/dl.

Serum sodium, potassium, chloride and blood glucose were normal. A full septic screen was negative and ultrasound brain scan did not reveal any structural abnormalities. He was stabilised with anticonvulsants and calcitriol initially and was discharged home on oral phenobarbitone and 1 alpha calcidol. At 3 months he was readmitted with convulsions. On this occasion too, he was hypocalcaemic despite therapy and magnesium level had dropped further to 1.1 mg/dl. His convulsions settled with parenteral magnesium sulphate, and he was discharged home on oral magnesium chloride. He was readmitted at 4 months with symptomatic hypocalcaemia, and the magnesium chloride dose was readjusted. Since

^{1,3}Senior Registrar, ⁴Paediatrician, Lady Ridgeway Children's Hospital; ²BSc student, ⁵Senior Lecturer, Department of Pharmacy, Faculty of Medicine, Colombo 8.

Correspondence: TMRG, e-mail <sanjika@kodak.lk>; telephone 0777 579 656. Received 11 May, and revised version accepted 24 September 2007. Conflicts of interest: none declared.

then he has been seizure-free and developing normally at 1 year of age, and is only on a magnesium chloride supplement.

Case 2

A 9-month old previously healthy female infant of non-consanguineous parents was admitted with a prolonged convulsion on day 3 of a febrile illness. She developed severe stridor and carpopedal spasms after admission. There were no adverse perinatal events and development had been normal. The physical examination was unremarkable.

She had a serum calcium of 1.04mmol/l and magnesium of 0.45mg/dl. Other electrolytes and blood glucose were normal. The CSF examination ruled out a meningoencephalitis. The QT interval was 0.58 s (normal < 0.44). The serum immunoglobulins were in the normal range. The CT scan of the brain revealed calcification in the basal ganglia and the thalamic region. She was stabilised with parenteral magnesium sulphate and calcium gluconate. Later she was converted to oral magnesium chloride. Her subsequent development is normal. She remains seizure free and serum magnesium and calcium levels are normal.

Discussion

Magnesium is the second most abundant intracellular cation and plays a vital role in many enzymatic processes including protein synthesis, glycolysis and nucleic acid metabolism. It is distributed mainly in bone (60%) and in the intracellular compartment (38%).

The rest (2%) is in the extracellular compartment of which about 60% is unbound and metabolically active. The absorption of magnesium is mainly through the ileum and colon. The elimination is mainly renal. The kidney acts as the main conservator in deficiency states.

Magnesium deficiency could be primary or secondary. Secondary deficiency occurs in chronic diarrhoea, severe protein energy malnutrition, hypoparathyroidism, renal tubular acidosis and in any hypercalcemic state. It also occurs secondary to loop diuretics, aminoglycosides

and cephalosporins. In babies of toxæmic and diabetic women, transient hypomagnesaemia develops during the neonatal period.

Primary hypomagnesaemia is a rare genetic disorder due to a selective defect in absorption at the small intestine [1]. The inheritance is autosomal recessive [2]. The affected gene is mapped to chromosome 9. Hypomagnesaemia causes hypocalcaemia by various mechanisms, [3,4,5]. Hypomagnesaemia is associated with recurrent tetany and convulsions in early infancy, leading to permanent neurological deficits [6].

Patients with primary hypomagnesaemia usually do not require calcium supplements or vitamin D derivatives long term. The serum level of magnesium should be assessed periodically and dosage adjusted as overdosage leading to hypermagnesaemia can have serious consequences. Early treatment of primary hypomagnesaemia with magnesium supplements will result in normal neurodevelopment.

References

1. Milla PJ, Aggett PJ, Wolff OH, et al. Studies in primary hypomagnesaemia: evidence for defective carrier mediated small intestinal transport of magnesium. *Gut* 1979; **20**: 1028-33.
2. Hannerkam RCM, Donckerwolcke RA. Primary hypomagnesaemia: an autosomal recessive inherited disease. *Lancet* 1983; **1**: 927-934.
3. Anast CS, Winnacker JL, Forte LR. Impaired release of parathyroid hormone in magnesium deficiency. *Journal of Clinical Endocrinology and Metabolism* 1976; **42**: 707-17.
4. Rude RK, Oldham SB, Singer FR. Functional hypoparathyroidism and parathyroid hormone end organ resistance in human magnesium deficiency. *Clinical Endocrinology* 1976; **5**: 209-24.
5. Fatemi S, Ryzen E, Flores J. Effect of experimental human magnesium depletion on parathyroid hormone secretion and 1, 25 dihydroxyvitamin D metabolism. *Journal of Clinical Endocrinology and Metabolism* 1991; **73**: 1067-72.
6. Sang RC. Neonatal magnesium disturbances. *American Journal of Diseases of Children* 1972; **124**: 282-93.