Reminder of important clinical lesson

Dyselectrolytemia in acute kidney injury causing tetany and quadriparesis

Atul Vijay Palkar,¹ Mayur Mewada,² Sonal Thakur,² Makardhwaj Sarvadaman Shrivastava²¹Department of Medicine, Lokmanya Tilak Municipal Medical College and General Hospital, Mumbai, Maharashtra, India;²Department of Internal Medicine, Lokmanya Tilak Municipal Medical College and General Hospital, Mumbai, Maharashtra, India**Correspondence to** Dr Makardhwaj Sarvadaman Shrivastava, makar_in@rediffmail.com**Summary**

A 40-year-old female, presented with prerenal acute kidney injury secondary to diarrhoea. With appropriate hydration, she went into diuretic phase and subsequently developed hypokalemic quadriparesis with hypocalcaemic tetany due to hypomagnesemia and subclinical vitamin D deficiency. The patient improved with oral potassium, magnesium, calcium and vitamin D supplementation.

BACKGROUND

Hypomagnesemia is a common entity occurring up to 12% of hospitalised patients and up to 60% of patients in intensive care settings though often missed.¹ Symptomatic magnesium depletion may be due to gastrointestinal or renal losses. It may present with hypokalemia, hypocalcemia and tetany. Hypocalcemia and hypokalemia in hypomagnesemia are often refractory to treatment with vitamin D or parathyroid hormone (PTH) or potassium supplementation unless the underlying magnesium deficit is corrected.

CASE PRESENTATION

A 40-year-old perimenopausal female, widow, was brought to the casualty by her daughter with history of multiple episodes of watery diarrhoea for 5 days with decreased urine output. A provisional diagnosis of acute gastroenteritis with oliguric acute kidney injury was made and the patient was started on intravenous fluid therapy. There was no history of pain in abdomen, vomiting or fever. There was no history of chronic diarrhoea, bloating or significant illness in the past. The patient was the only earning member of the family and used to work indoors for most part of the day as a housemaid. On examination, the patient was average built and nourished. She was afebrile with respiratory rate of 14 per min, pulse rate of 96 per min and blood pressure 90/60 mm Hg in the right arm in supine position. She was grossly dehydrated with dry tongue, sunken eyes and decreased skin turgor. There was no pallor, icterus or palpable lymphadenopathy. Her abdomen was scaphoid, soft with no organomegaly. There were no uraemic signs or symptoms. Cardiovascular, respiratory and central nervous system examination were unremarkable.

On day 3 of admission, the patient went in diuretic phase of acute kidney injury (AKI) with more than 3 litres of urine output daily.

On day 6 of admission, the patient developed flaccid quadriparesis and tetany. On clinical examination, Chvostek sign and Trousseau's sign were present. The patient was conscious, oriented. Higher functions were

normal and there were no cranial nerve deficits. Tone was decreased in all limbs with power symmetrically reduced in both upper and lower limbs to 3/5 proximally and 4/5 distally. There was weakness of neck and trunk muscles to grade 3/5. Muscle stretch reflexes were normal. Single breath count was 24.

Final diagnosis of prerenal AKI with hypokalemic quadriparesis with symptomatic hypocalcemia due to hypomagnesemia with underlying vitamin D deficiency and secondary hyperparathyroidism was made. The cause of hypomagnesemia was acute gastroenteritis and diuretic phase of AKI.

INVESTIGATIONS

The investigations on admission revealed blood urea nitrogen 85 mg/dl (30.35 mmol/l), creatinine 3.6 mg/dl (318.24 µmol/l), serum potassium 3.1 mmol/l and serum sodium 139 mmol/l. Haemogram showed haemoglobin 12 g/dl (7.45 pmol/l), total leucocyte count $9.5 \times 10^9/l$ with differential of 60%/35%/2% (polymorphs/lymphocytes/eosinophils) and platelets $2.2 \times 10^9/l$. Arterial blood gas showed normal anion gap metabolic acidosis. Renal ultrasound revealed normal sized kidneys with grade I increased echogenicity.

Investigations done when patient developed flaccid paralysis and tetany, revealed, ST segment prolongation with flattening of T waves on ECG (figure 1), serum calcium 4.8 mg/dl (1.2 mmol/l), phosphate 2.0 mg/dl (0.6458 mmol/l), albumin 41g/l, potassium 2.1 mmol/l and uric acid 2.1 mg/dl (124.95 µmol/l). Patient was euthyroid. Serum 25 hydroxy vitamin D level was 5.26 ng/ml (less than 10 ng/ml indicates deficiency) (figure 2) and serum PTH was 279.10 pg/ml (normal range 11.1 to 79.5 pg/ml). The serum magnesium level was 1.1 mg/dl (0.55 mmol/l) (normal range 1.7 to 2.4 mg/dl).

DIFFERENTIAL DIAGNOSIS

- Osteomalatic myopathy
- Periodic paralysis
- Giltelman syndrome

Figure 1 (A) ECG showing ST segment elongation and T wave inversion and flattening. (B) Chest leads, showing same ECG changes.

- Bartter syndrome
- Isolated dominant hypomagnesemia (IDH) with hypocalciuria.

TREATMENT

AKI in the patient was treated conservatively with intravenous and oral hydration. When she developed hypokalemic quadriparesis with hypocalcemic tetany, she was started on oral and intravenous potassium

supplementation along with oral magnesium, calcium and vitamin D.

OUTCOME AND FOLLOW-UP

The patient improved with resolution of limb weakness and tetany and was discharged after normalisation of creatinine and electrolytes without any residual abnormality. She was given vitamin D supplements and calcium at discharge.

Figure 2 X-ray knee joint. Please note marked osteopenia.

DISCUSSION

Vitamin D deficiency is an important cause of hypocalcemia. Patients with hypocalcemia may be asymptomatic if the decrease in serum calcium is relatively mild and chronic, or they may present with life-threatening complications like seizures, laryngeal spasm or cardiac dysrhythmias.² Symptomatic hypocalcemia in vitamin D deficiency may be precipitated by hypoparathyroidism, hypomagnesemia or bisphosphonate therapy.

Low plasma levels of calcitriol (1,25-dihydroxyvitamin D) have been noted in hypocalcemic, hypomagnesemic subjects and can contribute to the fall in the plasma calcium concentration. In one study, several days of magnesium replacement normalised plasma calcium and PTH levels but not the concentration of calcitriol.³

Immunoreactive PTH levels in most hypomagnesemic-hypocalcemic patients are either normal or low (and in some cases undetectable), indicating inappropriately low PTH secretion.^{4 5} However, PTH resistance may play more important role in causing hypocalcemia. PTH-induced release of calcium from bone is substantially impaired when the plasma magnesium concentration falls below 0.8 mEq/l (1 mg/dl or 0.4 mmol/l); in comparison, diminished PTH secretion appears to require more severe hypomagnesemia.

Hypokalemia is a common event in hypomagnesemic patients, occurring in 40% to 60% of cases.⁶ This may be partly due to underlying disorders that cause both magnesium and potassium loss, such as diuretic therapy and diarrhoea. There is also evidence of renal potassium wasting in hypomagnesemic patients,⁷ that is likely due

to increased potassium secretion in the loop of Henle and perhaps the cortical collecting tubule. The hypokalemia in this setting is relatively refractory to potassium supplementation and requires correction of the magnesium deficit.⁸

Though molecular genetic studies were not done, Gitelman syndrome as the cause of hypomagnesemia was ruled out due to lack of metabolic alkalosis and normalisation of serum magnesium on repeated follow-up without magnesium supplementation. Bartter syndrome was ruled out due to older age of presentation, absence of typical clinical features and hypocalcemia. IDH with hypocalcemia was ruled out due to lack of family history and absence of chondrocalcinosis.

Our patient had presented with oliguric AKI secondary to acute gastroenteritis. Diuretic phase of AKI along with diarrhoea caused significant hypomagnesemia and hypokalemia causing hypocalcemic tetany and quadriparesis respectively. Due to poor sun exposure despite tropical climate, patient had underlying vitamin D deficiency which was unmasked due to hypomagnesemia. Hypokalemia could have been easily attributed to diarrhoea and diuretic phase of AKI while hypocalcemia to vitamin D deficiency. Serum magnesium was detected to be low and underlying cause of dyselectrolytemia. Appropriate oral magnesium correction along with potassium, calcium and vitamin D supplementation resolved tetany and flaccid quadriparesis.

In literature, quadriparesis in the setting of hypokalemia, hypocalcemia and hypomagnesemia has been reported in diabetes mellitus⁹ and Gitelman's syndrome.¹⁰

Learning points

- ▶ Hypomagnesaemia is a common cause of hypokalaemia and hypocalcaemia though often missed.
- ▶ Magnesium deficiency may cause subclinical vitamin D deficiency manifested due to PTH deficiency or resistance precipitating hypocalcaemia which may be refractory to supplements unless magnesium is replenished.
- ▶ Vitamin D deficiency may be present even in patients belonging to tropical countries despite adequate sunlight throughout the year which may be unmasked by hypomagnesaemia, hypoparathyroidism or bisphosphonate therapy.

Competing interests None.

Patient consent Obtained.

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