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Acid-Base and Electrolyte Teaching Case

Treatment of Hypomagnesemia

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Serum magnesium concentration is determined by the interplay of intestinal absorption and renal excretion. Hypomagnesemia can occur as a result of insufficient magnesium intake, increased gastrointestinal or renal loss, or redistribution from extracellular to intracellular compartments. A number of drugs are known to cause hypomagnesemia, including proton pump inhibitors (PPIs). We report the case of a patient with symptomatic hypomagnesemia due to short bowel syndrome and PPI therapy. Investigations revealed low 24-hour urinary magnesium excretion and secondary hypocalcemia. PPI treatment was withdrawn and the patient was managed with intravenous and oral magnesium and calcium replacement. This teaching case provides an evidence-based discussion of the treatment of hypomagnesemia.

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INDEX WORDS: Hypomagnesemia; treatment; intravenous magnesium sulfate; oral magnesium salts; proton pump inhibitor.

Note from Feature Editor Jeffrey A. Kraut, MD: This article is part of a series of invited case discussions highlighting either the diagnosis or treatment of acid-base and electrolyte disorders. The present case discussion is the second of 2 articles describing a physiologic-based approach to the diagnosis or treatment of hypomagnesemia. In this article, Drs Ayuk and Gittoes present their approach to the treatment of hypomagnesemia; in the first teaching case, Dimke et al[1] described their approach to the evaluation of hypomagnesemia.

INTRODUCTION

Serum magnesium concentration is determined by the interplay of intestinal absorption and renal excretion. Hypomagnesemia can occur as a result of insufficient magnesium intake, increased gastrointestinal or renal loss, or redistribution from extracellular to intracellular compartments.[2] Conditions such as inflammatory bowel disease and short bowel syndrome, which are associated with diarrhea, malabsorption, or steatorrhea, can result in magnesium loss. A number of drugs are known to cause hypomagnesemia, with recent attention focusing on proton pump inhibitor (PPI) therapy. This report describes the case of a patient with inflammatory bowel disease who was on PPI therapy and presented with symptomatic hypomagnesemia. We discuss the management of hypomagnesemia, proposing treatment regimens based on available evidence.

CASE REPORT

Clinical History and Initial Laboratory Data

A 61-year-old woman presented with anorexia, vomiting, weakness, paraesthesia, and carpopedal spasm. She had longstanding Crohn disease and had undergone segmental small-bowel resection on 2 occasions, complicated by short bowel syndrome. Multiple courses of high-dose prednisolone had left her with significant dyspepsia, and in addition to mesalamine and loperamide, she had been treated with omeprazole for the previous 2 years. On examination, she was tachycardic and appeared volume depleted, with blood pressure of 90/60 mm Hg. Body temperature was 37.2°C and respiratory rate was 24 breaths/min. Chvostek sign was positive. Initial blood test results showed multiple electrolyte abnormalities (Table 1).

Additional Investigations

Subsequent investigations revealed a plasma intact parathyroid hormone (PTH) concentration of 31 (reference range, 14.6-62.7) pg/mL and 25-hydroxyvitamin D level of 21 ng/mL. The patient's 24-hour urinary magnesium excretion was 0.6 (reference range, 4-16) mEq/24 h.

Diagnosis

Hypomagnesemia due to malabsorption and PPI drug use, with secondary hypocalcemia.

Clinical Follow-up

Intravenous electrolyte replacement was undertaken with electrocardiogram monitoring. The patient received a loading dose of 2 g (16 mEq) of magnesium sulfate over 1 hour, followed by 5 g (40 mEq) of magnesium sulfate diluted in 1 L of 0.9% saline solution over 24 hours. Through separate intravenous access, one 10-mL ampule of 10% calcium gluconate diluted in 50 mL of 5% dextrose solution was infused gradually over 10 minutes, followed by an infusion of 10 ampules of 10 mL of 10% calcium gluconate in 1 L of 0.9% saline solution at a rate of 50 mL/h. Omeprazole therapy was stopped and replaced with ranitidine. Oral magnesium and calcium supplementation was initiated concurrently and continued when intravenous replacement was stopped. After 48 hours, her electrolyte levels had normalized and the patient was discharged from the hospital. A week later, serum magnesium and calcium levels were normal and treatment with oral supplements was stopped. To date, there has

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Table 1. Initial Investigation Results

Investigation	Result	Reference Range
Serum sodium (mEq/L)	131	134-146
Serum potassium (mEq/L)	3.6	3.4-5.2
Serum-adjusted calcium (mg/dL)	5.9	8.4-10.4
Serum magnesium (mEq/L)	0.36	1.4-1.9
Phosphate (mg/dL)	2.9	2.5-4.3
SUN (mg/dL)	30.8	9-21
Creatinine (mg/dL)	1.2	0.6-1.3
eGFR (mL/min/1.73 m^2)[a]	43	

Note: Conversion factors for units: serum-adjusted calcium in mg/dL to mmol/L, ×0.2495; serum magnesium in mEq/L to mmol/L, ×0.5; phosphate in mg/dL to mmol/L, ×0.3229; SUN in mg/dL to mmol/L, ×0.357; serum creatinine in mg/dL to μmol/L, ×88.4.

Abbreviations: eGFR, estimated glomerular filtration rate; SUN, serum urea nitrogen.

[a]GFR estimated using the MDRD (Modification of Diet in Renal Disease) Study equation.

been no recurrence of electrolyte disturbances, and her dyspeptic symptoms are well controlled with ranitidine.

DISCUSSION

The patient presented with symptomatic hypomagnesemia. The cause of hypomagnesemia often can be obtained from the history, and a number of potential contributors were apparent immediately in our patient. Severe Crohn disease with small-bowel resection and intestinal failure can lead to malabsorption of electrolytes.[3] Hypomagnesemia may be caused by secondary hyperaldosteronism due to salt and water loss, loss of absorptive area, or nonabsorbed fatty acids binding magnesium and forming soaps within the gut lumen, preventing absorption.[4] Although there is a high prevalence of vitamin D deficiency in patients with Crohn disease, hypocalcemia in this case most likely was due to suppression of PTH secretion and action by hypomagnesemia because 25-hydroxyvitamin D concentration was satisfactory and PTH level was inappropriately normal. The normal serum phosphate concentration confirms the lack of action of PTH.

The management of hypomagnesemia is based on the severity; symptoms rarely occur at magnesium levels >1 mEq/L. General recommendations for the management of hypomagnesemia are provided in Fig 1, but local guidelines are likely to exist and should be consulted.

Due to poor absorption, large doses of oral magnesium preparations cause gastrointestinal side effects. Therefore, patients with symptomatic hypomagnesemia should receive intravenous magnesium therapy, and oral replacement should be reserved for asymptomatic patients. Mild asymptomatic hypomagnesemia

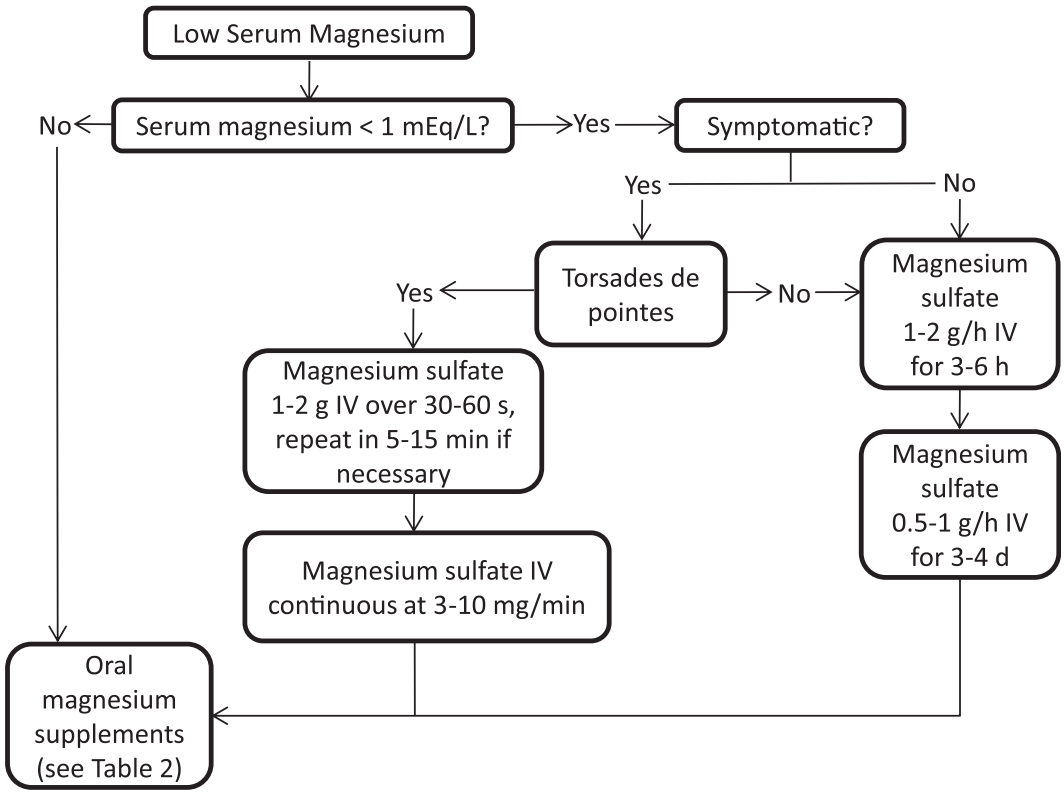

Figure 1. Treatment of hypomagnesemia. Abbreviation: IV, intravenous.