Understanding Hypomagnesemia in Patients with Short Bowel Syndrome and Renal Disease: a Case Report

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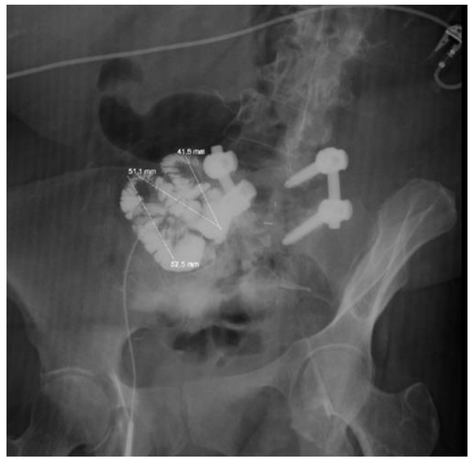
Introduction

- Loss of magnesium in acute patients may be multi-factorial
- Understanding the mechanism of deficiency is helpful in the treatment of patients with hypomagnesemia
- Mechanisms of hypomagnesemia: renal vs non-renal
  - Non-renal: reduced GI absorption, malnutrition, GI losses (diarrhea, vomiting), alcohol use, redistributive (refeeding, insulin therapy, blood transfusions
  - Renal: diuretics, Bartter vs Gitelman syndrome, medications (anti-microbials, calcineurin inhibitors, growth factor receptor inhibitors, cytotoxic drugs), hypercalcemia, hyperthyroidism, hyperparathyroidism, alcohol use
- Patients with post-surgical GI anatomy and/or stomas, in conjunction with kidney disease are more prone to deficiency
  - Short gut syndrome defined as <180–200cm of remaining small bowel (normal length = 275–850cm)
  - High-output stomas = >1.5-2L/day output primary consisting of H2O, Na, Mg
- Patients typically present with symptoms of weight loss, diarrhea, steatorrhea, dehydration, malnutrition and notably, electrolyte imbalance

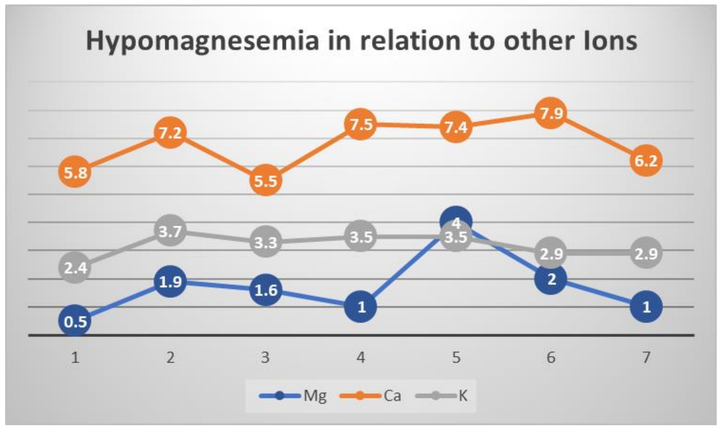
Case Presentation

- 62 year old female presented with (1) generalized weakness, (2) lethargy, (3) cramping for a few days with c/o increased NB ileostomy output, decreased appetite, severe nausea and NBNB vomiting.
- PMH: (1) Crohn's disease s/p panproctocolectomy with end ileostomy – RLQ in 1990, (2) short bowel syndrome s/p small bowel resection in 2020 (125cm remaining), (3) peritoneal and intestinal adhesions s/p numerous correctional abdominal surgeries, (4) long-term TPN use, (5) CKD stage 3 (GFR = 30-59 ml/min), (6) hyponatremia, (7) dementia, (8) small bowel perforation, (9) multiple sepsis (w/wo shock) admissions, (10) hypothyroidism, (11) hyperlipidemia
- Patient was treated for septic shock; was given IV fluids, started on PPI, empiric micafungin, cefepime and vancomycin. Patient had seizure-like activity with hypotension which resolved without pressors.
- Initial labs: Na=135, K=2.4, corrected Ca=5.8, Mg=0.5, BUN/Cr=45/2.9 (baseline Cr~1)

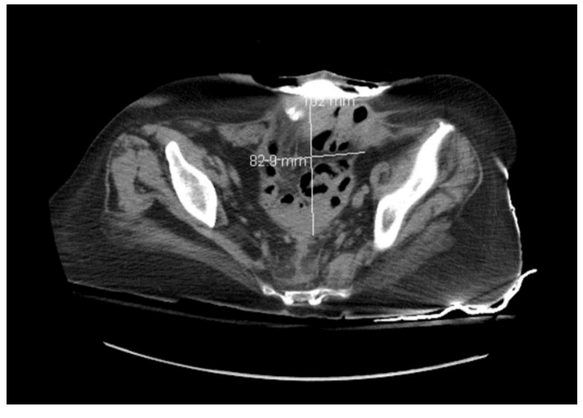
Figures



Fluoroscopic KUB X-ray showing shortened small bowel anatomy (<125cm)



Patient's electrolyte trend through hospitalization



CT A/P showing residual small bowel with an area of 10x10x10cm

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Discussion

Differentiating mechanisms of renal vs non-renal (GI) losses of magnesium is important in the clinical setting:

Renal:

- Nearly all renal Mg is filtered at glomerulus and reabsorbed from the loop of Henle and downstream
- Kidney damage, hypercalcemia, metabolic acidosis, hypokalemia or phosphate depletion can reduce physiological reabsorption

Non-renal:

- Nearly 90% of Mg of the GI tract is reabsorbed in the terminal ileum and colon
- Reduced, damaged or infiltrated lumenal surfaces of the GI tract will cause significant losses through malabsorption
- Increased transit times through the GI tract (seen in SBS) limits absorption of vital minerals (Mg, Ca)
- Prolonged diarrhea increases the loss of Mg
- Certain medications such as PPIs can cause loss of magnesium due to reduced gastrointestinal absorption

Urinary magnesium analysis is helpful in the assessment of renal vs non-renal etiology of hypomagnesemia:

- Quantifying the Fractional Excretion of Mg gives insight into whether Mg deficiency is due to renal wasting
- Diuretics or Mg supplements must be stopped for accurate analysis
- Urinary Mg must be adjusted for urinary creatinine (Cr)
- Fractional Excretion of Mg (FEMg) = serum Cr x urine Mg/0.7 x serum Mg x urine Cr
- Renal Mg wasting = FEMg > 0.4% or daily Mg excretion of 1mmol/day (24.31 mg/d)

Evaluation of other ions that may be deficient due to hypomagnesemia must be assessed:

- Potassium: decreased Mg causes K wasting into the lumen of the collecting duct via ROMK channels
- Calcium: severe deficiencies of Mg causes impaired PTH secretion which decreases bone resorption and Ca reabsorption

Correlation with bariatric surgery, osteomies and other causes

- Shortened bowel, such as in patients with pan-proctocolectomy, can lead to fat malabsorption; excess fatty acids bind cations (e.g., Mg, Ca) creating soaps which hinder magnesium reabsorption.

Conclusion

- The etiology of hypomagnesemia in this patient was believed to be multifactorial.
- Measuring urinary magnesium is beneficial in such cases, as it helps distinguish renal versus non-renal causes of hypomagnesemia. Increased excretion suggests renal wasting, while low or normal values indicate non-renal causes, such as gastrointestinal losses or inadequate intake.
- Integrating urinary and serum Mg analysis in conjunction with clinical presentation is helpful in developing customized management strategies for hypomagnesemia.
- Treatment for hypomagnesemia in patients with short bowel syndrome varies based on the underlying etiology: renal magnesium wasting necessitates managing kidney dysfunction and magnesium supplementation, while non-renal causes involve dietary adjustments, magnesium supplements, or addressing conditions leading to gastrointestinal magnesium loss.
- Modes of magnesium repletion may include oral magnesium supplements, intravenous magnesium administration, incorporation of enteral nutrition formulations, topical magnesium application, and combination therapy tailored to individual patient needs.

References