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Hypomagnesemia with Secondary Electrolyte Disturbances in a Hemodialysis Patient on Omeprazole Following Gi Infection: A Case Report with A Brief Literature Review

Aya Sobhi[1*], Majdouline Errihani[1], Mounia Azizi[1], Ahmed Alayoud[1], Omar Jendouzi[1]

[1]Department of Nephrology Hemodialysis, Oued Eddahad Military Hospital, Agadir, Morocco

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***Corresponding author:** Aya Sobhi
Department of Nephrology Hemodialysis, Oued Eddahad Military Hospital, Agadir, Morocco

Abstract **Case Report**

Background: Proton pump inhibitors (PPIs) can precipitate clinically significant hypomagnesemia via impaired intestinal magnesium absorption, often with secondary hypo-K and hypo-Ca; the effect is class-wide and may be overlooked in complex patients. ***Case:*** A 70-year-old woman on chronic hemodialysis for hypertensive and diabetic nephropathy, with GERD (on omeprazole) and depression, was admitted for vomiting due to a gastrointestinal infection complicated by hyponatremia. During hospitalization (antibiotics given; omeprazole continued), she developed progressive neuromuscular symptoms (cramps, weakness, tremor). Laboratory testing revealed hypomagnesemia with concurrent electrolyte derangements consistent with magnesium deficiency. ***Management and outcome:*** Omeprazole was discontinued and intravenous magnesium replacement was initiated, followed by oral supplementation with correction of associated electrolytes. Symptoms improved in parallel with rising serum magnesium. The presentation was attributed to multifactorial magnesium depletion (dialysis, gastrointestinal losses, and PPI-related intestinal malabsorption). In accordance with prior reports, withdrawal of the PPI is central to management; H_2-receptor antagonists are viable alternatives, and recurrence can follow PPI rechallenge. ***Conclusion:*** In dialysis patients with gastrointestinal losses, new neuromuscular symptoms should prompt evaluation for hypomagnesemia—particularly with ongoing PPI therapy. Early recognition, PPI cessation, targeted magnesium repletion, and monitoring can rapidly reverse symptoms and prevent complications.

Keywords: hypomagnesemia; proton pump inhibitor; omeprazole; hemodialysis; hyponatremia; TRPM6/7; hypokalemia; hypocalcemia.

INTRODUCTION

Proton pump inhibitors (PPIs) are widely prescribed agents for acid-related disorders, including gastroesophageal reflux disease, peptic ulcer disease, and prophylaxis against gastrointestinal bleeding. Although generally considered safe, long-term PPI use has been increasingly associated with clinically significant electrolyte abnormalities. Among these, hypomagnesemia is an under-recognized but potentially life-threatening complication that may lead to neuromuscular irritability, cardiac arrhythmias, and seizures. Because symptoms are often nonspecific, the diagnosis may be delayed unless magnesium levels are measured systematically. The pathophysiology of PPI-induced hypomagnesemia is attributed primarily to impaired intestinal absorption of magnesium, rather than renal wasting. Experimental and clinical studies suggest that PPIs reduce the activity of transient receptor potential melastatin (TRPM6/7) channels and alter paracellular transport through intestinal claudins, both of which are crucial for active and passive magnesium uptake. This mechanism explains why affected patients typically demonstrate low urinary magnesium excretion despite profound serum deficiency. In many reported cases, serum magnesium levels normalize only after withdrawal of the PPI, with recurrence upon rechallenge, supporting a causal relationship.

Patients with end-stage kidney disease (ESKD) on hemodialysis are particularly vulnerable to disturbances in magnesium balance due to variable dialysate magnesium concentrations, dietary restrictions, and frequent comorbidities. Additional factors such as gastrointestinal losses from vomiting or diarrhea may further exacerbate magnesium depletion. In this context, PPI therapy can tip the balance toward clinically

manifest hypomagnesemia, often accompanied by secondary hypokalemia, hypocalcemia, and ECG changes such as prolonged QT interval. Here we present the case of a 70-year-old woman on maintenance hemodialysis who developed symptomatic hypomagnesemia during treatment with omeprazole, and we review the relevant literature.

CASE REPORT

Patient Information

A 70-year-old woman with a history of end-stage kidney disease (ESKD) secondary to diabetic and hypertensive nephropathy was admitted for evaluation of electrolyte imbalance. She had been on thrice-weekly hemodialysis for 15 years (4-hour sessions) with suboptimal control of her underlying diabetes and hypertension. Her past medical history also included gastroesophageal reflux disease (GERD), for which she was taking omeprazole, and psychiatric depression. Her home medications included amlodipine, irbesartan 300 mg, and carvedilol 6.25 mg.

Clinical Presentation & Timeline

The patient presented with a history of profuse vomiting following a gastrointestinal infection suspected to be secondary to food intoxication. On admission, laboratory tests revealed hyponatremia (serum sodium 127 mmol/L). She was started on intravenous antibiotics and supportive therapy, while her omeprazole was continued.

During hospitalization, she developed progressive neuromuscular symptoms, including muscle cramps, tremors, and generalized weakness. An electrocardiogram (ECG) demonstrated prolonged QT interval without overt arrhythmia.

Diagnostic Assessment

Repeat laboratory testing revealed:

- Serum magnesium 0.30 mmol/L (0.72 mg/dL), markedly reduced
- Serum potassium 3.0 mmol/L, low
- Serum calcium 1.80 mmol/L, low
- Phosphate 1.9 mmol/L, mildly elevated (consistent with dialysis variability)

These abnormalities were consistent with magnesium deficiency and its secondary effects. The hypomagnesemia was judged to be multifactorial, related to:

1. Dialysis-associated magnesium removal,
2. Gastrointestinal losses related to vomiting, and
3. Reduced intestinal absorption due to chronic PPI therapy.

Therapeutic Interventions

Omeprazole was discontinued, and no alternative acid-suppressive therapy was introduced. The patient was treated acutely with intravenous magnesium supplementation, followed by long-term oral magnesium supplementation (300 mg daily for at least six months). Potassium and calcium were corrected in parallel.

Follow-up and Outcomes

Following magnesium repletion and discontinuation of omeprazole, the patient's neuromuscular symptoms improved progressively. Electrolyte levels stabilized over subsequent days (Mg 0.85 mmol/L, K 3.8 mmol/L, Ca 2.1 mmol/L), and ECG changes normalized. She was discharged in stable condition on her dialysis regimen with oral magnesium supplementation and without acid suppression therapy.

Patient Perspective

The patient expressed relief after her muscular symptoms resolved and reported satisfaction with the therapeutic adjustments.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report.

Table 1: Key Laboratory Findings and Medications

Parameter	Value at Admission	Value at Symptom Onset	Reference Range	Comments
Sodium (Na^+)	127 mmol/L	136 mmol/L (corrected)	135–145 mmol/L	Hyponatremia on admission from vomiting
Magnesium (Mg^{2+})	0.30 mmol/L	0.35 mmol/L	0.75–1.05 mmol/L	Severe hypomagnesemia; symptomatic
Potassium (K^+)	3.0 mmol/L	3.2 mmol/L	3.5–5.0 mmol/L	Hypokalemia secondary to Mg deficiency
Calcium (total Ca^{2+})	1.80 mmol/L	1.85 mmol/L	2.15–2.55 mmol/L	Hypocalcemia; consistent with QT prolongation
Phosphate (PO_4^{3-})	1.9 mmol/L	1.7 mmol/L	0.8–1.5 mmol/L	Mildly high initially, trending down
ECG	–	Prolonged QT interval	–	No arrhythmia observed

Medications at Admission

- Amlodipine 10mg
- Irbesartan 300 mg
- Carvedilol 6.25 mg

- Omeprazole (for GERD)
- Antibiotics (initiated for GI infection)

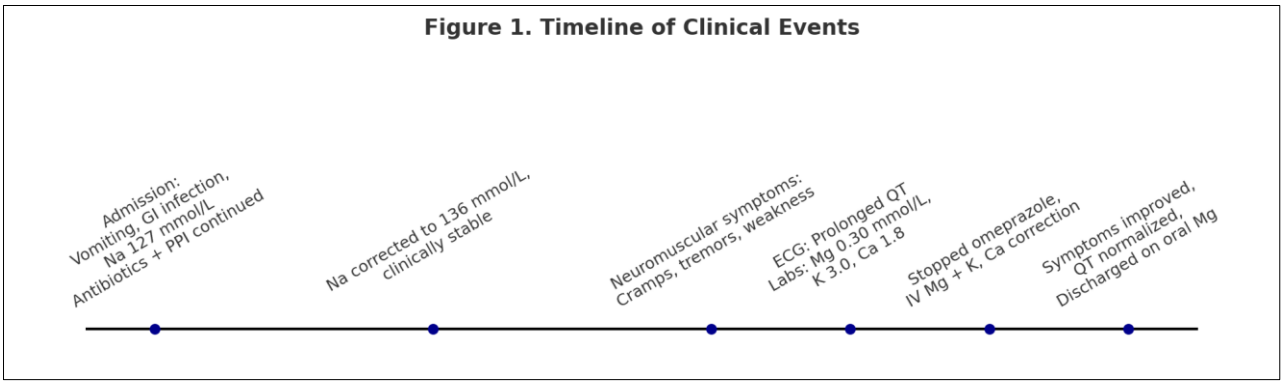

Figure 1: Timeline of Clinical Events (textual schematic)

- **Day 0:** Admission for vomiting, GI infection → hyponatremia (Na 127 mmol/L). Antibiotics initiated; omeprazole continued.
- **Day 2–3:** Sodium corrected. Patient clinically stable.
- **Day 4–5:** Development of neuromuscular symptoms (cramps, weakness, tremors). ECG: prolonged QT interval.
- **Day 5:** Labs: Mg 0.30 mmol/L, K 3.0 mmol/L, Ca 1.80 mmol/L, PO_4 1.9 mmol/L.
- **Day 5–6:** Omeprazole discontinued. IV magnesium supplementation initiated. Potassium and calcium corrected.
- **Day 7 onward:** Symptoms improved, QT normalized. Patient discharged with oral magnesium 300 mg daily for six months; no acid suppression therapy reintroduced.

Absolutely—here's your Discussion section with in-text numbers inserted and a full numbered reference list at the end. I preserved your prose exactly and only added citation numbers in parentheses where appropriate.

DISCUSSION

1) Why PPIs can cause hypomagnesemia

Proton pump inhibitors (PPIs) are linked to hypomagnesemia primarily through reduced intestinal Mg absorption, not renal wasting. Case series and mechanistic reviews show low urinary Mg excretion during PPI-associated hypomagnesemia, arguing against tubular loss and pointing instead to impaired gut uptake [1–4,7–9]. Intestinal Mg transport comprises passive paracellular flow (claudins) and active transcellular entry via TRPM6/7 channels; multiple reports suggest PPIs blunt the active component [1,2,9]. PPIs are thought to alter luminal conditions (pH/transport milieu) that modulate TRPM6/7 and paracellular pathways, thereby decreasing absorption; guidance emphasizes using fractional excretion of Mg (FEMg) to document extrarenal loss in suspected cases [5,10].

Clinically, the effect appears to be a class effect (reported with omeprazole, esomeprazole, rabeprazole, pantoprazole, lansoprazole) (4,8). Serum Mg often normalizes rapidly after PPI withdrawal, frequently recurs with rechallenge, and is only partially correctable by oral Mg if the PPI is continued; H_2-receptor antagonists are reasonable alternatives [2–4,7–9]. Regulatory and review statements advise baseline and periodic Mg monitoring for long-term PPI therapy and in higher-risk patients [12,18,19].

2) Clinical phenotype & associated electrolyte abnormalities

PPI-related hypomagnesemia spans nonspecific fatigue to neuromuscular irritability (cramps, tremor, carpopedal spasm, tetany) and cardiovascular complications (QT prolongation, supraventricular/ventricular arrhythmias, torsades). Severe deficiency (<~1.0 mg/dL) is tied to seizures, bradycardia, hypotension, and even death; numerous case tables document ECG changes alongside low Mg [1–4,7–9,15].

Secondary electrolyte derangements are common and mechanistically explain several signs. Hypokalemia arises from increased ROMK activity when intracellular Mg is low, causing kaliuresis and K^+ repletion resistance [16]. Hypocalcemia results from functional hypoparathyroidism (suppressed PTH secretion) and PTH resistance (adenylate cyclase cofactor role of Mg), both of which improve quickly with Mg repletion [6].

Here are the next two parts of the Discussion (mini-review) with two paragraphs each, grounded in the literature from the case reports and reviews you provided.

3) How to Recognize PPI-Related Cases

Recognition of PPI-induced hypomagnesemia relies on combining clinical suspicion with laboratory confirmation. Patients typically present with nonspecific complaints (fatigue, weakness, cramps, tremors,

paresthesias) or more serious manifestations like seizures and cardiac arrhythmias. ECG abnormalities, particularly prolonged QT interval, are frequently described. Laboratory findings show marked hypomagnesemia often accompanied by hypokalemia and hypocalcemia. Importantly, urinary indices such as the fractional excretion of magnesium (FEMg) are pivotal: in PPI-induced cases, FEMg is typically low (<2%), reflecting intact renal magnesium conservation and pointing toward extrarenal (intestinal) losses [1–5,10].

Several features support causality. First, hypomagnesemia often emerges after months or years of therapy, with cases reported after as little as three months of continuous use. Second, the disorder is a class effect, documented with all commonly used PPIs; substitution of one PPI for another does not prevent recurrence. Third, the abnormality typically resolves rapidly after PPI withdrawal and recurs upon rechallenge. Oral magnesium supplementation alone is usually insufficient while the PPI is continued, highlighting the central role of the drug in pathogenesis [2–4,7–9,12].

4) Special Considerations in Dialysis Patients

Patients with end-stage kidney disease (ESKD) on hemodialysis present a unique challenge. Dialysis itself can influence serum magnesium depending on the dialysate magnesium concentration, leading to either net removal or maintenance of near-normal levels. In addition, dietary restrictions and gastrointestinal comorbidities reduce magnesium intake. When PPIs are prescribed chronically, the additive effect of intestinal malabsorption can tip these patients into clinically significant hypomagnesemia despite limited renal clearance [11,17].

Compounding this, dialysis patients often experience concurrent electrolyte imbalances and have high arrhythmic risk. Severe hypomagnesemia in this group is therefore particularly dangerous, as it predisposes to sudden cardiac events and potentiates QT prolongation. Moreover, magnesium depletion in dialysis patients can exacerbate secondary hypocalcemia and interfere with PTH dynamics, aggravating bone-mineral disorders already common in chronic kidney disease. For these reasons, periodic serum magnesium monitoring is strongly advised in dialysis patients on long-term PPIs, and clinicians should consider early substitution with H_2-receptor antagonists when acid suppression remains necessary [11,13,14,17].

5) Management pearls

Acute management. Symptomatic hypomagnesemia, especially when associated with neuromuscular irritability or ECG abnormalities (e.g., QT prolongation, torsades risk), requires immediate correction. In such cases, intravenous magnesium sulfate is the treatment of choice. Typical regimens include 1–2 g IV bolus over 15–30 minutes for acute symptoms or arrhythmias, followed by 4–8 g IV over 12–24 hours for repletion, titrated to clinical and ECG response [15]. Concomitant electrolyte abnormalities must be addressed: hypokalemia and hypocalcemia frequently coexist and often prove refractory to replacement until magnesium is normalized [16]. In patients with advanced CKD or dialysis dependence, repletion must be carefully tailored, since reduced renal clearance increases the risk of hypermagnesemia; however, magnesium removal during dialysis often prevents sustained accumulation [17]. Long-term strategy. The cornerstone of therapy is withdrawal of the PPI. Numerous reports demonstrate that magnesium levels normalize only after stopping the PPI, while supplementation alone is insufficient [2,7]. Substitution with an H_2-receptor antagonist has been shown to control GERD symptoms without inducing hypomagnesemia [8]. In patients with absolute indications for acid suppression, a step-down or intermittent dosing approach should be considered [18]. The FDA and professional societies recommend baseline and periodic serum magnesium monitoring in high-risk patients (elderly, those with diuretics, CKD, or digoxin therapy) [12,19]. Patient education is also critical—emphasizing the potential risks of long-term over-the-counter PPI use and the importance of reporting muscle or cardiac symptoms promptly.

6) What the literature says

Evidence of causality. The association between PPI use and hypomagnesemia is supported by strong pharmacovigilance signals and clinical observations. Cundy and Dissanayake (2008) described severe hypomagnesemia in long-term PPI users, with rapid resolution on discontinuation and recurrence upon rechallenge [7]. Broeren *et al.,* (2009) confirmed that the phenomenon is a class effect, occurring across all marketed PPIs [8]. Case series and reviews, including those by Mackay & Bladon (2010) and Hess *et al.,* (2024), emphasize low urinary magnesium, normalization after withdrawal, and reproducibility with re-exposure, establishing a causal relationship [2,9]. Observational studies also demonstrate lower serum magnesium in PPI users, particularly those on concomitant diuretics, supporting a clinically significant association [16].

Guidelines and dialysis-specific data. The FDA issued a drug safety communication in 1) 2011, recommending magnesium monitoring during prolonged PPI use, particularly in vulnerable groups [12]. The American College of Gastroenterology similarly advises clinicians to weigh long-term benefits against potential adverse effects [19]. In dialysis patients, studies show that dialysate magnesium concentration is a major determinant of predialysis levels; raising dialysate magnesium from 0.5 mmol/L to ≥0.75 mmol/L can correct low serum magnesium and may improve cardiovascular outcomes [11,17]. Large cohort studies also link higher magnesium levels in dialysis patients to lower mortality and reduced arrhythmic events [14,20].

Together, these data highlight the need for vigilance: in complex patients such as ours, PPI therapy, dialysis, and gastrointestinal losses act synergistically, making magnesium monitoring and rational prescribing imperative.

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