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Severe Hypermagnesemia Following Rectal Magnesium Laxative Use Complicated by Non-Occlusive Mesenteric Ischemia: A Case Report

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Abstract

Magnesium-containing laxatives are commonly used in elderly patients for the treatment of constipation; however, excessive magnesium absorption, particularly in individuals with impaired renal function, may result in severe hypermagnesemia that can lead to life-threatening complications. Non-occlusive mesenteric ischemia (NOMI) is a rare but often fatal condition characterized by intestinal hypoperfusion without major arterial occlusion and is typically associated with systemic hypotension and low-flow states. We report the case of an 83-year-old female with a history of hypertension and chronic neurological sequelae who presented to the emergency department with altered mental status, abdominal pain, hypotension, and bradycardia following rectal administration of a magnesium-containing laxative. Laboratory analysis revealed severe hypermagnesemia (8.56 mg/dL), acute kidney injury, metabolic acidosis, elevated lactate (10 mmol/L), leukocytosis, and markedly increased inflammatory markers. Contrast-enhanced abdominal computed tomography demonstrated diffuse small bowel wall thickening with decreased mural enhancement and mesenteric edema, while CT angiography showed patent mesenteric arteries without occlusion, findings consistent with non-occlusive mesenteric ischemia. Despite aggressive fluid resuscitation, vasopressor therapy, broad-spectrum antibiotics, and emergency surgical resection of

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necrotic bowel segments, the patient developed multiorgan failure and died on postoperative day nine. Severe hypermagnesemia in elderly patients with renal impairment may therefore lead to profound hemodynamic instability and systemic hypoperfusion, potentially contributing to the development of NOMI; early recognition of hypermagnesemia, prompt measurement of serum magnesium levels, and aggressive management are essential to reduce morbidity and mortality. In addition, the markedly elevated inflammatory markers raise the possibility of a mixed shock state, in which both hypermagnesemia-induced cardiovascular depression and a concomitant septic process may have contributed to systemic hypoperfusion and the development of non-occlusive mesenteric ischemia.

Keywords: Hypermagnesemia; Mesenteric Ischemia; Laxatives; Aged; Acute Kidney Injury

Introduction

Constipation is a common condition among elderly individuals and is frequently managed with osmotic laxatives, including magnesium-containing preparations due to their efficacy and accessibility. Magnesium oxide and other magnesium-based agents are widely prescribed for the treatment of chronic constipation (1). However, excessive magnesium intake or impaired renal excretion may result in hypermagnesemia, a potentially life-threatening electrolyte disorder characterized by neuromuscular, cardiovascular, and respiratory complications (2). Elderly patients are particularly vulnerable because of age-related decline in renal function, multiple comorbidities, and the frequent use of magnesium-containing medications or laxatives (3).

Magnesium plays an essential role in numerous physiological processes, including neuromuscular transmission, vascular tone regulation, and cardiac electrophysiology (4). Elevated serum magnesium levels exert calcium antagonist effects that can cause hypotension, bradycardia, and decreased myocardial contractility, potentially leading to cardiovascular collapse in severe cases (2,4).

Non-occlusive mesenteric ischemia (NOMI) is a subtype of acute mesenteric ischemia characterized by intestinal hypoperfusion in the absence of major arterial obstruction (5). It is typically associated with systemic hypotension, heart failure, sepsis, or other low-flow states that compromise mesenteric blood supply (6). NOMI accounts for approximately 20–30% of acute mesenteric ischemia cases and carries a high mortality rate, particularly when diagnosis and treatment are delayed (6,7).

Although hypermagnesemia has been reported following the use of magnesium-containing cathartics and enemas, reports describing severe hypermagnesemia associated with the development of NOMI are extremely limited. Here we present a fatal case of severe hypermagnesemia following rectal magnesium laxative use complicated by non-occlusive mesenteric ischemia in an elderly patient with acute kidney injury.

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Case Presentation

An 83-year-old woman with a history of hypertension and chronic neurological sequelae presented to the emergency department. Her neurological deficits included permanent right-sided visual impairment and right upper extremity motor deficit secondary to a previous cerebrovascular event.

One week prior to admission, the patient had sustained a lumbar vertebral fracture following a fall and was advised bed rest with corset immobilization. She had a history of chronic constipation and intermittently used oral and rectal magnesium-containing laxatives.

Shortly after rectal administration of a magnesium-containing laxative for constipation, she developed diffuse erythematous skin rash, abdominal pain, progressive weakness, and altered mental status.

Physical Examination

At presentation, the patient appeared confused and lethargic with slow responses to verbal stimuli.

Skin examination revealed diffuse erythematous rash. This rash may represent a systemic or hypersensitivity reaction following laxative administration. Abdominal examination demonstrated diffuse tenderness with guarding and rebound tenderness.

Vital signs were as follows:

Blood pressure: 88/59 mmHg
Pulse: 54 beats/min
Temperature: 35.5°C
Respiratory rate: 14/min
Oxygen saturation: 86% on room air
Blood glucose: 158 mg/dL

Electrocardiography demonstrated sinus rhythm with T-wave inversion in lead DIII.

Laboratory Findings

Laboratory tests revealed severe metabolic and inflammatory abnormalities (Table 1).

Table 1. Laboratory findings with reference values

PARAMETER	PATİENT VALUE	REFERENCE RANGE
MAGNESİUM	8.56 mg/dL	1.7–2.4 mg/dL
CREATİNİNE	2.11 mg/dL	0.6–1.2 mg/dL
LACTATE	10 mmol/L	0.5–2.0 mmol/L
BİCARBONATE (HCO_3^-)	14 mmol/L	22–28 mmol/L
WHİTE BLOOD CELL COUNT	27.5 $\times 10^3/\mu L$	4.0–10.0 $\times 10^3/\mu L$
PROCALCİTONİN	>100 ng/mL	<0.05 ng/mL
C-REACTİVE PROTEİN (CRP)	56 mg/L	<5 mg/L
D-DİMER	14,230 ng/mL	<500 ng/mL

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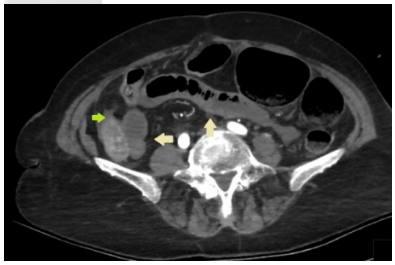
These findings were consistent with severe hypermagnesemia, acute kidney injury, metabolic acidosis, and systemic inflammatory response.

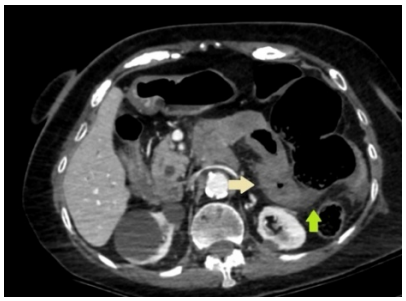
Imaging Studies

Contrast-enhanced abdominal computed tomography demonstrated diffuse circumferential wall thickening of proximal jejunal and ileal loops with reduced bowel wall enhancement and submucosal edema. Mesenteric fat stranding and interloop free fluid were also present.

CT angiography revealed diffuse atherosclerotic plaques in the abdominal aorta; however, the celiac trunk, superior mesenteric artery, and inferior mesenteric artery were patent without evidence of arterial occlusion (Figure 1).

Figure 1. Figure 1. Contrast-enhanced abdominal CT and CT angiography findings.





These findings were considered compatible with non-occlusive mesenteric ischemia.

Management and Clinical Course

The patient received aggressive intravenous fluid resuscitation, norepinephrine infusion

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for hemodynamic support, and broad-spectrum antimicrobial therapy including meropenem, teicoplanin, and fluconazole.

Supportive treatment for hypermagnesemia included intravenous hydration and diuretics to enhance renal magnesium excretion. Intravenous calcium gluconate 10% 20 mL was administered to counteract the cardiodepressant effects of hypermagnesemia.

Hemodialysis was also considered; however, it could not be performed due to the patient's rapidly deteriorating hemodynamic status and the need for urgent surgical intervention. Hemodialysis could not be initiated due to profound hemodynamic instability and the urgent need for surgical intervention.

Emergency exploratory laparotomy revealed extensive small bowel ischemia and necrosis. The affected intestinal segments were resected and an ileostomy was performed.

Despite intensive postoperative care, the patient developed progressive multiorgan failure and died on postoperative day nine due to cardiac arrest.

Discussion

Hypermagnesemia is an uncommon but potentially life-threatening electrolyte disorder that usually develops in patients with impaired renal excretion or excessive magnesium intake (2, 4). The kidneys play a central role in maintaining magnesium homeostasis, and decreased renal function significantly increases the risk of magnesium accumulation, particularly in elderly patients receiving magnesium-containing medications (3).

Magnesium acts as a physiological calcium antagonist and suppresses neuromuscular transmission as well as myocardial contractility (4). As serum magnesium levels rise, patients may develop hypotension, bradycardia, and cardiac conduction abnormalities. Severe hypermagnesemia, generally defined as serum levels exceeding 6 mg/dL, may lead to profound cardiovascular depression, respiratory failure, and cardiac arrest (2,4). In this case, the clinical picture likely represents a multifactorial mechanism, with both hypermagnesemia-induced cardiovascular depression and a concomitant septic process contributing to systemic hypoperfusion and the development of non-occlusive mesenteric ischemia.

Magnesium-containing laxatives and enemas are frequently used to treat constipation, especially among elderly or immobilized patients. However, several studies have documented severe hypermagnesemia associated with magnesium-based cathartics, particularly in individuals with impaired renal function. Rectal administration may result in significant systemic absorption, especially when large doses are used or when intestinal mucosal integrity is compromised (1-3).

In the present case, the patient developed severe hypermagnesemia accompanied by hypotension and bradycardia, suggesting significant cardiovascular depression. These hemodynamic disturbances may have

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contributed to systemic hypoperfusion and subsequent development of non-occlusive mesenteric ischemia.

NOMI occurs as a consequence of intestinal hypoperfusion without mechanical arterial obstruction. The underlying mechanism involves mesenteric vasoconstriction and reduced intestinal blood flow triggered by systemic hypotension, heart failure, or shock states (8,9). Early diagnosis is often challenging, and delayed recognition significantly increases mortality (9).

Another important consideration in this case is the presence of markedly elevated inflammatory markers including leukocytosis, elevated procalcitonin levels, and high lactate concentration. These findings raise the possibility of septic shock. It is therefore plausible that the patient developed a mixed shock state in which both hypermagnesemia-induced cardiovascular depression and systemic inflammatory response contributed to mesenteric hypoperfusion and intestinal ischemia (8,9).

Management of severe hypermagnesemia focuses on stabilizing cardiovascular function and enhancing magnesium elimination. Initial treatment typically includes intravenous calcium administration to antagonize the cardiac effects of magnesium, aggressive fluid resuscitation, and loop diuretics to promote renal magnesium excretion (4). In patients with severe hypermagnesemia or renal failure, hemodialysis is considered the most effective method for rapidly reducing serum magnesium levels.

NOMI remains a highly lethal condition with reported mortality rates exceeding 50%, particularly when diagnosis and treatment are delayed (6,7). Early recognition of hemodynamic instability and prompt restoration of mesenteric perfusion are therefore essential to improve patient outcomes.

This case highlights the importance of considering hypermagnesemia in elderly patients presenting with unexplained hypotension and bradycardia, especially in the setting of recent magnesium-containing laxative use.

Conclusion

Magnesium-containing laxatives should be used with caution in elderly patients, particularly in those with impaired renal function. Severe hypermagnesemia may cause significant cardiovascular depression and systemic hypoperfusion, potentially contributing to the development of non-occlusive mesenteric ischemia.

Early recognition of hypermagnesemia, prompt measurement of serum magnesium levels, and aggressive hemodynamic management are essential for reducing morbidity and mortality in such patients.

Informed Consent

Written informed consent was obtained from the patient's legal representatives for publication of this case report and any accompanying images.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

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