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CASE REPORT

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Impaired consciousness due to hypermagnesemia associated with stercoral colitis: report of a rare case

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Abstract

Background Hypermagnesemia is a rare electrolyte abnormality that is difficult to diagnose because its symptoms are nonspecific. In addition to magnesium administration, renal dysfunction is often a major risk factor associated with the condition; severe intestinal dysfunction is also a known risk factor. However, no cases of hypermagnesemia were observed in the absence of magnesium administration.

Case presentation A 75-year-old woman with cognitive impairment presented to the emergency department with impaired consciousness. The patient was comatose and hypotensive and had a markedly distended abdomen. Her blood pressure was stabilized with infusion; however, the improvement in consciousness was insufficient and somnolence continued. Abdominal computed tomography revealed marked colonic distension due to fecal impaction in the rectum, with wall thickening and pericolonic fat stranding. Blood tests revealed elevated levels of C-reactive protein (10.2 mg/dL), lactate (6.04 mmol/L), and magnesium (5.9 mg/dL). There was no history of ingestion of magnesium-containing preparations; thus, the patient was diagnosed with hypermagnesemia associated with stercoral colitis. Magnesium levels and consciousness improved with the administration of calcium preparations, diuretics, antibiotics, and defecation control.

Conclusions Severe bowel dysfunction can cause hypermagnesemia, even in the absence of magnesium administration.

Keywords Colitis, Constipation, Hypermagnesemia, Impaired consciousness, Intestinal dysfunction, Magnesium containing cathartics, Obstructive colitis

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Background

Hypermagnesemia is a rare electrolyte abnormality that causes impaired consciousness and respiratory and circulatory disturbances. However, its diagnosis is difficult because of the absence of specific symptoms. Excretion from the kidneys and absorption from the intestinal tract are primarily involved in maintaining magnesium homeostasis [1, 2]. The kidneys play an especially important role in the process, and hypermagnesemia often develops in patients with evidence of renal dysfunction [3]. However, in the presence of gastrointestinal disorders, such as severe constipation and colitis, decreased excretion and reabsorption of residual magnesium in the intestinal tract are risk factors for hypermagnesemia [4]. Hypermagnesemia has been reported to develop in patients with renal or gastrointestinal dysfunction to whom magnesium was administered [1, 4–6]; however, the condition has not been reported in individuals who were not administered magnesium. We report a case of hypermagnesemia associated with stercoral obstructive colitis despite the absence of magnesium administration.

Case presentation

A 75-year-old woman who had been admitted to a facility for dementia was brought to the emergency department with a chief complaint of impaired consciousness. The patient's usual state of consciousness was disoriented; however, she was able to communicate. The patient had a history of surgery for uterine myoma. The only medication she prescribed was Diphenidol Hydrochloride (25 mg three times a day). Medications were administered by facility staff, and she had no history of magnesium oxide usage. Her vital signs on arrival were as follows: Glasgow Coma Scale (GCS) score of 3 (E1V1M1), blood pressure: 64/40 mmHg, heart rate: 118/min, respiratory rate: 30/min, body temperature: 36.5 ℃, and oxygenation saturation: 95% (room air). The patient's abdomen was markedly distended. Head computed tomography (CT) showed no abnormalities, and abdominal contrast-enhanced CT showed fecal impaction in the rectum with marked dilation of the proximal colon (Fig. 1). Occlusive and non-occlusive mesenteric ischemia was excluded because there was no contrast failure in the intestinal wall. Additionally, wall thickening of the rectum and increased fat tissue density were observed around the rectum (pericolonal fat stranding) (Fig. 2), which was diagnosed as stercoral colitis. Blood pressure improved quickly (101/80 mmHg) after the administration of extracellular fluid, and her consciousness improved to a GCS score of 11 (E3V3M5), which was different from the patient's usual state. Blood tests revealed elevated levels of C-reactive protein (10.2 mg/dL), lactate (6.04 mmol/L), and hypermagnesemia (5.9 mg/dL) (Table 1). Creatinine (Cr) level (0.94 mg/dL) was within the normal range. Electrocardiographic abnormalities associated with hypocalcemia or hypermagnesemia were not identified. The urine drug test results were negative.

Figure 3 shows the patient's clinical course after hospital admission. After admission, manual disimpaction was performed, which led to large bowel movements. Calcium gluconate hydrate and diuretics were administered for hypermagnesemia. Although intestinal ischemia was unlikely based on CT images, the patient was hypotensive at arrival with elevated levels of lactate, so antibiotic therapy (meropenem) was initiated for stercoral obstructive colitis. During hospitalization, regular

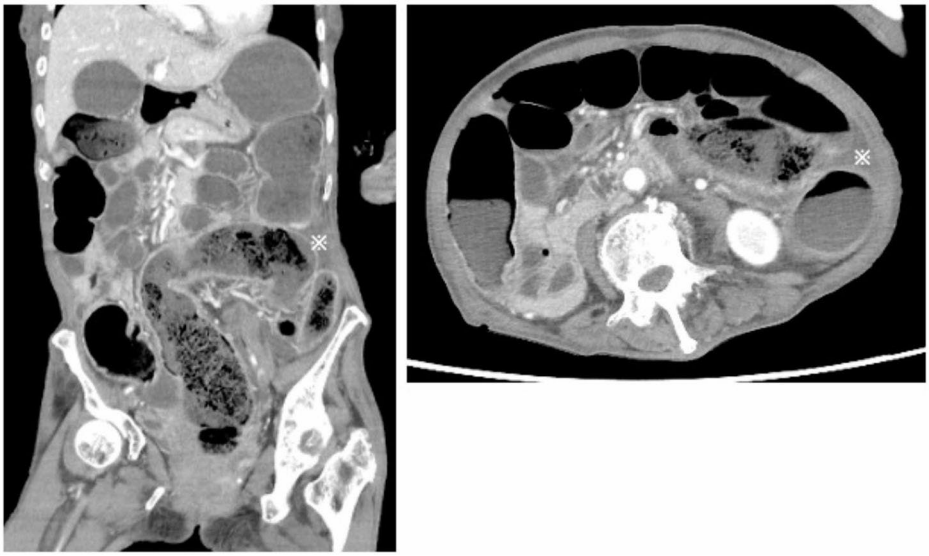

Fig. 1 Abdominal contrast-enhanced computed tomography performed in emergency department
Fecal impaction from the rectum to the sigmoid colon with marked dilation of the proximal colon. A small amount of ascites was detected (※)

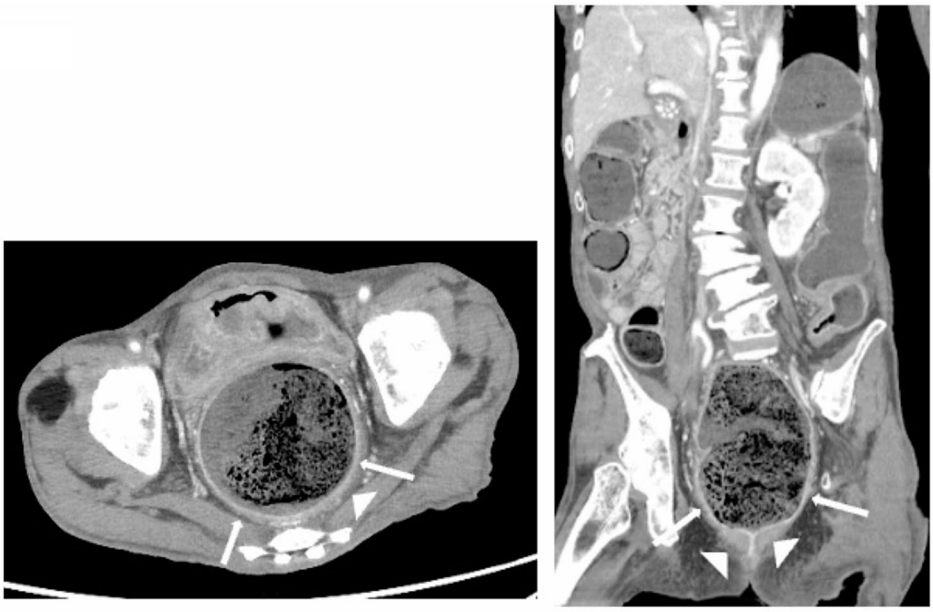

Fig. 2 Wall thickening of the rectum (arrows) and increased fat tissue density around the rectum (arrowheads)

Table 1 Laboratory data at the emergency department

Biochemistry	Value	Normal range	CBC/Coagulopathy	Value	Normal range	Endocrinology Arterial blood gas	Value	Normal range
TP, g/dL	6.4	6.6–8.1	WBC, $\times 10^3/\mu L$	6.88	3.3–8.6	TSH, μIU	4.84	0.61–4.23
Alb, g/dL	3.6	4.1–5.1	Neu, %	75	30–70	FT3, pg/mL	0.898	2.3-4
TB, mg/dL	0.7	0.4–1.5	Lymph, %	18	19–61	FT4, ng/dL	0.992	0.9–1.7
AST, IU/L	35	13–30	Mono, %	7	2–12			
ALT, IU/L	38	7–23	Eos, %	0	0–8	pH	7.276	
ALP, IU/L	54	38–113	Baso, %	0	0–2	BE, mmol/L	-11.6	-3-3
LDH, IU/L	344	124–222	RBC, $\times 10^4/\mu L$	559	350–510	Lactate, mmol/L	6.04	
Amy, IU/L	77	44–132	Hb, g/dL	17.6	11.6–14.8	PCO2, mmHg	30.4	35–45
BUN, mg/dL	42	8–20	Hct, %	51.6	35.1–44.4	HCO3, mmol/L	13.8	20–26
Cr, mg/dL	0.94	0.46–0.79	Plt, $\times 10^4/\mu L$	28.9	15.8–34.8			
CK, IU/L	35	41–153	PT, %	126	70–130			
Na, mEq/L	136	138–145	PT-INR	0.92				
K, mEq/L	5.3	3.6–4.8	APTT	29.7	23–38			
Ca, mg/dL	6.9	8–10						
Mg, mg/dL	5.9	1.8–2.4						
Glu, mg/dL	192	70–110						
CRP, mg/dL	10.2	0-0.5						

Abbreviations - CBC: complete blood count; TP: total protein; Alb: albumin; TB: total bilirubin; AST: aspartic aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase; LDH: lactate dehydrogenase; Amy: amylase; BUN; blood urea nitrogen; Cr: creatinine; CK: creatine kinase; Na: sodium; K: potassium; Ca: calcium; Mg: magnesium; Glu: glucose; CRP: C-reactive protein; WBC: white blood cell; Neu: neutrophil; Lymph: lymphocyte; Mono: monocyte; Eo: eosinophil; Baso: basophil; RBC: red blood cell; Hb: hemoglobin; Hct: hematocrit; Plt: platelet; PT: prothorombin time; PT-INR: prothorombin time-international normalized ratio; APTT: activated partial thromboplastin time; TSH: thyroid stimulating hormone; FT3: free triiodothyronine; FT4: free thyroxine; BE: base excess; PCO2: partial pressure of carbon dioxygen, HCO3: bicarbonate

bowel movements were observed and serum magnesium levels gradually improved. As the patient's consciousness gradually improved, impaired consciousness caused by hypermagnesemia associated with stercoral colitis was noticed. On hospital day 11, the magnesium level and the GCS score had improved to 1.7 mg/dL and 14 (E4V4M6), respectively, which was comparable to the patient's baseline condition. The patient was discharged on the hospital day 18.

Discussion and conclusions

We report a case that highlights an important clinical learning point: severe intestinal dysfunction and inflammation, such as stercoral colitis, are important risk

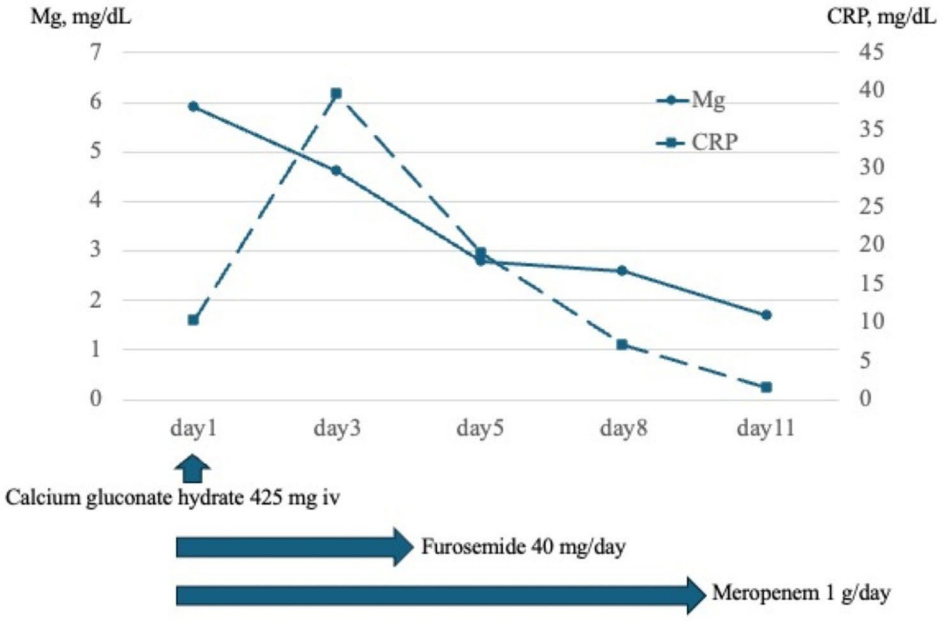

Fig. 3 Course of treatment and changes in magnesium and C-reactive protein levels
Calcium gluconate hydrate and diuretics were administered to treat hypermagnesemia. For stercoral obstructive colitis, antibiotic therapy was initiated after manual disimpaction
Mg; magnesium: CRP; C-reactive protein

factors for hypermagnesemia, even without administering magnesium preparation. The mechanism of hypermagnesemia involves a combination of factors, including excessive administration of magnesium preparations, increased absorption, and decreased excretion [7]. Mori et al. reported that the average serum concentration in patients taking prescribed doses of magnesium preparations was 2.2 mg/dL and stated that hypermagnesemia is very rare with regular oral doses [8]. Renal function is a very significant factor affecting magnesium concentrations; 60–65% of plasma magnesium is filtered by glomeruli, and up to 6 g/day of magnesium can be excreted under normal conditions [9, 10]. Therefore, renal dysfunction is a major risk factor for hypermagnesemia. Serum Cr levels are commonly used as indicators of renal function; however, they are also influenced by muscle mass, making them less likely to increase in elderly patients. In this case, the patient's Cr levels were normal. In contrast, an estimated glomerular filtration rate (eGFR) of less than 30 ml/min/1.73 m^2 is considered a risk factor for hypermagnesemia [2]. The eGFR in this case was 44.4 ml/min/1.73 m^2; however, as evidenced by elevated blood urea nitrogen in this patient, it is important to consider the possibility of latent renal dysfunction in elderly patients should be considered rather than assessed only based on test results.

Hypermagnesemia without renal dysfunction is often associated with intestinal obstruction or severe constipation [1, 4, 6]. Magnesium reabsorption from the intestines occurs primarily in the proximal small intestine, with an absorption rate that varies markedly between 25% and 65% [11]. Intestinal dysfunction and inflammation can lead to prolonged magnesium retention, resulting in increased magnesium reabsorption [12, 13]. This involves the following factors. First, passive magnesium absorption via the paracellular pathway, which exists as a magnesium transport pathway in the intestinal tract, increases linearly with increasing amounts of magnesium in the intestinal tract, with no apparent threshold [14]. Second, hypermagnesemia itself exacerbates the neuromuscular blocking effects of magnesium, leading to impaired intestinal function and further promoting magnesium reabsorption [2]. The magnesium absorption mechanism from the intestines shares some aspects with calcium absorption mechanism in terms of absorption sites and transporters, so increased intestinal magnesium absorption may be one factor in hypocalcemia, as in this case [15]. However, there have been no reports of hypermagnesemia due to severe intestinal dysfunction in the absence of oral magnesium preparations, as observed in this case. Based on marked colonic dilatation due to fecal impaction in the rectum, wall thickening of the rectum, and pericolonal fat stranding, the patient was diagnosed with stercoral colitis [16]. Stercoral colitis is strongly associated with chronic constipation, which is influenced by various factors, including intrinsic factors such as functional, neurological, and metabolic, and extrinsic factors such as drugs. The patient was at high

risk for functional constipation, as she was institutionalized and predictably tended to be bedridden. Conversely, medication for constipation was not given, suggesting a delay in intervention for intestinal tract dysfunction. The combination of intestinal dysfunction due to obstruction and severe inflammation resulted in excessive magnesium reabsorption, leading to hypermagnesemia despite the absence of magnesium preparations.

In this case, because there were no signs of peritonitis, the stercoral colitis was managed with antibiotics and appropriate defecation control. In cases of fever, leukocytosis, and elevated lactate, antibiotics are strongly recommended. In cases of suspected perforation, broad-spectrum antibiotics are selected to cover gram-negative rods and anaerobic bacteria [16]. Meropenem was initiated because the organisms that was considerd to be covered were the same even if the case was not perforated.

Calcium preparations and diuretics were administered to treat hypermagnesemia. In the absence of renal dysfunction, treatment of the underlying diseases causing intestinal dysfunction and symptomatic therapy can lead to rapid improvement in hypermagnesemia.

Although the symptoms of hypermagnesemia are nonspecific, it is not particularly difficult to consider hypermagnesemia in the differential diagnosis if magnesium preparations have been administered. However, in elderly patients with severe intestinal inflammation or dysfunction, as in this case, hypermagnesemia should be considered in the differential diagnosis, even if magnesium preparations have not been administered.

This case has several limitations. First, blood test results do not exclude the possibility of hypothyroidism. However, since the level of free thyroxin (fT4) was low but within the reference range and the level of thyroid-stimulating hormone (TSH) was only mildly elevated, we considered the low level of free triiodothyronine (fT3) to be a low T3 syndrome due to the patient's general condition. Subsequent levels of thyroid hormone were not measured; however, the patient's condition, including consciousness level, improved without thyroid hormone supplementation. Therefore, hypothyroidism was not significantly associated with impaired consciousness in this case. Second, it was difficult to clearly differentiate whether the patient's impaired consciousness was associated with hypermagnesemia and with a systemic condition due to stercoral colitis. Third, we checked the patient's oral medications, but not the magnesium levels in her usual food and drink. Finally, tumors have not been evaluated by colonoscopy or other means, and paraneoplastic syndromes, which can cause various metabolic abnormalities, have not been evaluated.

In conclusion, hypermagnesemia is a rare electrolyte abnormality that often develops in patients who have used magnesium preparations and in those with risk factors such as renal dysfunction and decreased intestinal function. However, in cases of severe intestinal dysfunction, such as stercoral colitis, hypermagnesemia can develop on its own. Regarding nonspecific symptoms, it is important to thoroughly assess risk factors and consider hypermagnesemia in the differential diagnosis, even if magnesium preparations have not been used.

Abbreviations

GCS	Glasgow coma scale
CT	Computed tomography
Cr	Creatinine
eGFR	Estimated glomerular filtration rate
fT3	free triiodothyronine
fT4	free thyroxin
TSH	thyroid-stimulating hormone

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Author contributions

KY, DM and HH provided emergency care to the patient; analyzed the patient data; and contributed to the diagnosis. KY and DM drafted the manuscript. HH, MM, and MN supervised the preparation of this manuscript. All the authors have read and approved the final version of the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

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