J Anesth (2007) 21:273–276
DOI 10.1007/s00540-006-0492-8

Journal of
Anesthesia

Circulatory collapse caused by unnoticed hypermagnesemia in a hospitalized patient

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Abstract
We report a case of hypermagnesemia in a hospitalized patient after prolonged laxative use; due to preexisting impaired consciousness and digestive problems, the hypermagnesemia was difficult to detect until it almost became fatal. A 64-year-old man who was a patient at another hospital for treatment of head injury and gastric ulcer had developed circulatory collapse and was transferred to our hospital. Hypermagnesemia (serum magnesium concentration 11.0 mg·dl^{-1}) was thought to be the cause of the circulatory collapse and treatments were successful. A magnesium laxative had been administered for more than a month at the previous hospital, but the patient's serum magnesium level was never measured. Care should be taken when a magnesium laxative is administered to patients who already have impaired consciousness and digestive problems that are early symptoms of hypermagnesemia.

Key words Hypermagnesemia · Bowel hypomotility · Head injury · Normal renal function · Laxative

Case report

A 64-year-old man (height, 158 cm; weight, 47.4 kg) was transferred to our hospital from another hospital because of circulatory collapse. He had no relevant medical history, with no renal dysfunction. At the previous hospital, he had received treatment for a head injury for 6 weeks, though slight consciousness disorder, of Glasgow Coma Scale (GCS) 14 (E4V4M6), had remained. During the course of treatment, he had developed a gastric ulcer and often vomited. He had complained of constipation all the time during this 6-week period and had been given 1.5 g·day^{-1} magnesium oxide (0.9 g·day^{-1} magnesium) for a month. Despite this, the constipation had continued. On the day before he was transferred to our hospital, he had complained of abdominal pain. At first, he had been treated with an enema, for constipation, and a small amount of stool was excreted. But his abdomen was hard and distended, which indicated acute abdomen. Though an electrocardiogram (ECG) during his first admission to that hospital showed a normal sinus rhythm (Fig. 1A), ECG on the day before transfer showed heart rate (HR) of 72 bpm, with complete left bundle branch block; the HR was slower than expected, and hypovolemia due to acute abdomen was suspected (Fig. 1B). Meanwhile, impaired consciousness and hypotension had developed progressively, resulting in circulatory collapse, and he was transferred to our hospital. When he was brought to the emergency room, he was in a state of pulseless electrical activity, and cardiopulmonary resuscitation was performed. One mg of epinephrine IV was successful in restoring circulation, but his ECG showed junctional rhythm of 50–60 bpm with multifocal ventricular premature contractions (VPCs; Fig. 1C). He remained hypotensive despite having received 30 mg·h^{-1} dopamine and an infusion of 2500 ml saline. Echocardiogram revealed that the left ventricle was small in size without asynergy, which indicated hypovolemia. His abdomen was hard and distended, and computed tomography showed megacolon, and free air was suspected in the intraperitoneal cavity around the prostate. Laboratory examination showed hypermagnesemia (serum magnesium concentration, 11.0 mg·dl^{-1}), renal dysfunction, and systemic inflammation (Table 1). Because hypermagnesemia was thought to be the cause of his bradycardia, 850 mg intravenous calcium gluconate was administered, and he was admitted to our intensive care unit (ICU) to prepare for emergency surgery for suspected gastrointestinal perforation. In the ICU, 8500 mg intravenous calcium gluconate was administered to antagonize magnesium, and glucose-insulin therapy was performed to shift magnesium into the intracellular space. His serum magnesium concentration decreased

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Received: July 31, 2006 / Accepted: December 6, 2006

Table 1. Laboratory examination of the patient

	Previous hospital[a]	ER	ICU (postoperative)
Mg (1.7–2.6 mg·dl^{-1})	—	11.0	7.4
BUN (8–22 mg·dl^{-1})	11	44	42
Cre (0.6–1.1 mg·dl^{-1})	0.6	2.8	2.1
K (3.6–4.9 mEq·l^{-1})	4.2	5.1	3.7
Ca (9.0–10.6 mEq·l^{-1})	—	8.7	10.6
Ca^{2+} (1.13–1.32 mmol·l^{-1})	—	1.17	1.40
WBC (3.6–9.6 × 10^3·μl^{-1})	32.2	20.7	4.3
CRP (≦0.3 mg·dl^{-1})	0.4	16.1	16.3

Figures in parentheses are normal ranges
[a] Day before transfer to our hospital
ER, emergency room; ICU, intensive care unit

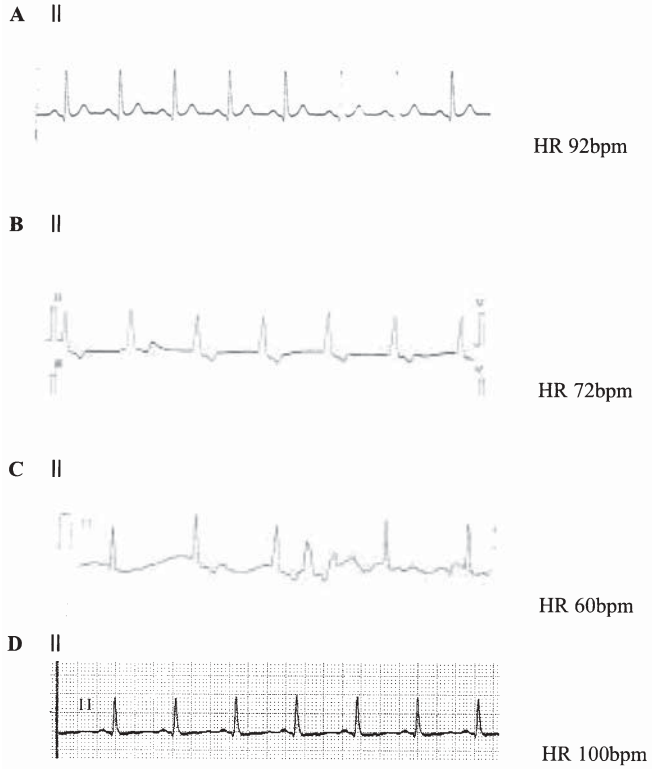

Fig. 1A–D. Electrocardiogram of the patient (lead II). **A** First admission to the previous hospital; **B** in the previous hospital just before transfer; **C** after resuscitation in emergency room (ER); **D** recovery to sinus rhythm in the operating room. *HR*, heart rate

to 8.6 mg·dl^{-1} 2 h after his admission to our hospital. Then he was transferred to the operating room, although his ECG showed junctional rhythm with multifocal VPCs. In the operating room, volume loading and furosemide administration were effective for magnesium washout, and his ECG recovered to a normal sinus rhythm (Fig. 1D). Serum magnesium concentration at that time was 7.6 mg·dl^{-1}. Surgeons found necrosis in a large area of the colon, without perforation, and a colostomy was performed. He was readmitted to the ICU postoperatively. On postoperative day 1, he recovered consciousness, to GCS 14, which was equal to the level shown in the previous hospital, and extubation was done (Fig. 2). On postoperative day 2, he was discharged from the ICU with his serum magnesium concentration further decreased, at 3.2 mg·dl^{-1}.

Discussion

There have been many reports of hypermagnesemia caused by magnesium laxatives [1–3]. Most reports describe outpatients who showed some kinds of symptoms of hypermagnesemia and were admitted to hospitals, while there have been few reports of fatal hypermagnesemia in hospitalized patients. In the hospitalized patient described here, hypermagnesemia was not detected until it had almost become fatal, because his preexisting symptoms—impaired consciousness, nausea, and vomiting—were similar to some of the early symptoms of hypermagnesemia. Hypermagnesemia is characterized by the progressive loss of neuromuscular, cardiovascular, and central nervous system function. Nausea, vomiting, cutaneous flushing, lethargy, and hyporeflexia are usually the earliest symptoms, and the clinical manifestations are correlated with serum magnesium levels. The clinical picture of hypermagnesemia is nonspecific and may be similar to the presentation of many other syndromes—sepsis, bowel obstruction, ethanol intoxication, and primary central nervous system events [4]. Consequently, the symptoms of hypermagnesemia tend to be easily overlooked. In fact, because our patient had impaired consciousness secondary to head injury, as well as bowel hypomotility, the early symptoms of hypermagnesemia were masked until circulatory collapse had occurred. In addition a symptom resembling septic shock, due to the abdominal lesion, was a confusing factor.

Hashizume and Mori [5] have claimed that hypermagnesemia is easily overlooked. They measured serum magnesium levels in 6252 patients and found 51 patients (0.8%) who had hypermagnesemia (serum magnesium,

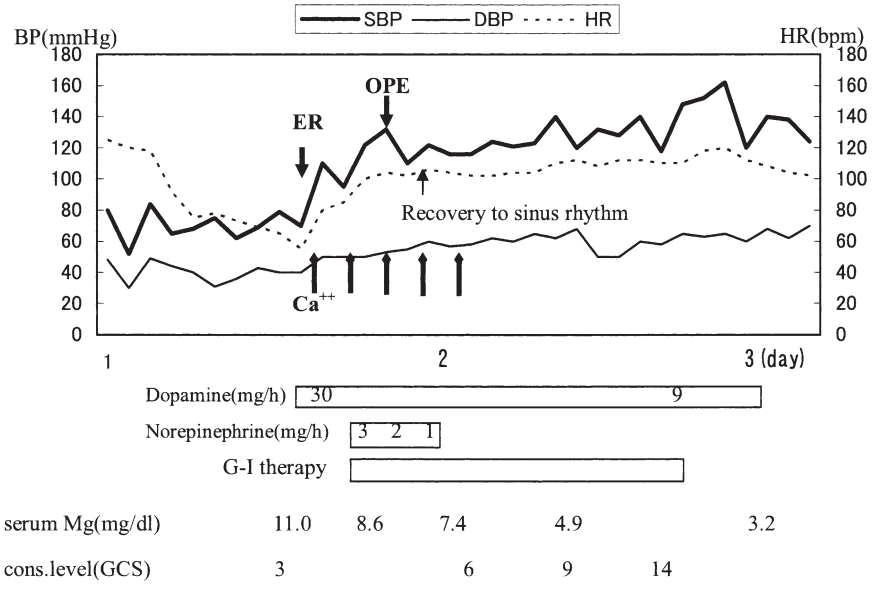

Fig. 2. Clinical course of the patient. The vital signs became stable after the initiation of therapy for hypermagnesemia. With the reduction in the serum magnesium level, the consciousness (*cons.*) level improved. *SBP/DBP*, systolic/diastolic blood pressure; *HR*, heart rate; *G-I*, glucose- insulin therapy; *GCS*, Glasgow Coma Scale; *ER*, emergency room; *OPE*, operation

≥3.9 mg·dl^{-1}). Though more than half of these hypermagnesemic patients had renal dysfunction (60%) or had received magnesium preparations (data not shown), most physicians in charge of the patients did not pay attention to the serum magnesium levels. Even when they realized that some symptoms of hypermagnesemia were present, they tended to consider that the symptoms were caused by an underlying disease.

The causes of hypermagnesemia are, theoretically, as follows: magnesium overdose, increased absorption, and decreased elimination. In our patient, the hypermagnesemia was attributed to a combination of these factors; that is, long-term administration of a magnesium laxative, preexisting bowel hypomotility, and acute renal failure.

The total amount of magnesium laxative used seemed relatively excessive for him. He had been receiving 0.9 g·day^{-1} of magnesium for a month. The maximum dose of magnesium set by the Japanese Pharmacopoeia is 1.2 g·day^{-1} (2 g·day^{-1} magnesium oxide). Therefore, the dose given at the previous hospital (1.5 g·day^{-1} magnesium oxide) may not be have been excessive. However, the magnesium administration was continued for a month without any effect on his constipation. In consequence, his serum magnesium level increased insidiously. Nevertheless, his serum magnesium level was never measured at the previous hospital. The magnesium administration should have been discontinued soon after it proved to be ineffective.

There are several reports of hypermagnesemia in patients with normal renal function [6–8]. In these patients, there was often underlying bowel hypomotility. It is reported that prolonged contact of ingested magnesium with the mucosa due to depressed bowel motility or mechanical bowel obstruction facilitates the absorption of magnesium [4]. Hypermagnesemia itself may have contributed to intestinal smooth-muscle dysfunction in our patient [9].

Another factor which contributed to the hypermagnesemia in our patient may have been acute renal dysfunction, which would have decreased the elimination of magnesium. Most patients with hypermagnesemia have renal dysfunction [10]. Our patient did not have renal dysfunction until the day before transfer to our hospital (Table 1). The hypovolemia due to ischemic colitis and the circulatory collapse due to his hypermagnesemia may have caused acute renal failure. His serum magnesium level the day before transfer to our hospital may have been already high due to the chronic administration of the magnesium laxative for his refractory constipation.

Hypermagnesemia can be fatal if early symptoms are overlooked. Therefore, we should know the symptoms of hypermagnesemia, and close observation should be done when a magnesium laxative is administered to patients who have factors predisposing to hypermagnesemia. Regular checking of serum magnesium levels is advisable, especially when patients have persistent constipation.

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