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CLINICAL REPORT

Perioperative management for a patient with hypermagnesemia-induced shock with perforative peritonitis

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Abstract We present a case of hypermagnesemia accompanied by perforative peritonitis. A 79-year-old woman took magnesium citrate as part of the pretreatment on the day before a scheduled colonoscopy. She developed nausea and muscle weakness, and she was complaining of left abdominal pain. Consciousness gradually worsened and she developed shock. Intestinal obstruction was recognized on abdominal X-ray and computed tomography (CT), and peritonitis was suspected. An exploratory laparotomy was scheduled for diagnosis and treatment. In the operating room, arterial blood gas analysis showed metabolic acidosis and hypermagnesemia (Mg: 2.75 mmol/l, normal range: 0.1–1.5 mmol/l). On laparotomy, adhesion around the sigmoid colon and turbid ascites were recognized. But we could not detect the apparent region of perforation. Based on these findings and the presence of hypermagnesemia, we diagnosed that the shock was caused by peritonitis due to intestinal micro-perforation, and by hypermagnesemia due to absorption of laxative. We started to treat for metabolic acidosis, and to manage the hypermagnesemia by calcium hydrochloride administration and by continuous hemodiafiltration after the operation. On day 4 of the illness, the plasma Mg level was normalized. She was extubated on day 12, and discharged on day 84. This case with complicated clinical symptoms reaffirms the difficulty and importance of making a diagnosis quickly by collecting various data.

Keywords Hypermagnesemia · Peritonitis · Laxative

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Introduction

Hypermagnesemia is a rare disease that is caused by laxative overdose. In many cases, severe shock occurs, and early diagnosis and proper therapy are necessary. In Japan, people take Nigari (crude magnesium chloride) [1] to lose weight, and some have developed Nigari poisoning and cardiopulmonary arrest, and this has recently been viewed as a social problem. We present a case of a patient with hypermagnesemia accompanied by perforative peritonitis who took a laxative as part of the pretreatment for endoscopy.

Case report

The patient was a 79-year-old woman. She had undergone open hysterectomy due to myoma and had received 9 mg/day of prednisolone for rheumatoid polymyositis for more than 10 years. Also, she had been taking 750 mg/day of magnesium oxide for constipation (as we found out several days after admission). Because a community health screening had confirmed bloody stool, the patient was scheduled to undergo colonoscopy by a physician at another hospital. On the day before the colonoscopy, she took magnesium citrate 34 mg (MAGCOROL-P 50 mg, Horii Pharmaceutical Ind. Ltd., Osaka, Japan) as part of the pretreatment. She developed nausea and then muscle weakness, and her consciousness gradually worsened. When the patient was brought by ambulance to the hospital, her vital signs were: blood pressure 95/33 mmHg; heart rate 84 beats/min; respiratory rate 24/min; and body temperature 36.7°C. Her Glasgow Coma Scale score was 13 points ($E_3V_4M_6$), and she was complaining of left abdominal pain. Peritonitis was suspected initially; antibiotics were administered, and various tests were performed,

but her blood pressure continued to decrease gradually, and she developed shock. Despite receiving 2 μg/kg/min of norepinephrine, 15 μg/kg/min of dobutamine, and 2 u/h of vasopressin, her blood pressure was about 60/30 mmHg and her heart rate was 80–90 beats/min; because her consciousness level was decreased, artificial ventilation was started after tracheal intubation. Abdominal X-ray and computed tomography (CT) confirmed intestinal obstruction, and, based on the patient's clinical symptoms, past history, and course, she was diagnosed as having shock due to intestinal perforative peritonitis. An exploratory laparotomy was scheduled for diagnosis and treatment.

On entry into the operating room, the patient's blood pressure was 40/20 mmHg, heart rate was 85 beats/min, and peripheral oxygen saturation (SpO_2)was 90% (fractional inspired oxygen [F_1O_2] = 1.0). Arterial blood gas analysis was performed, confirming metabolic acidosis: pH, 7.396; PO_2, 57.1 mmHg; PCO_2, 28.5 mmHg; Na, 130 mmol/l; K, 3.49 mmol/l; Ca, 1.22 mmol/l; HCO_3^-, 17.7 mmol/l; base excess (BE), −7.5 mmol/l, and the ionized Mg level was 2.75 mmol/l (normal range: 0.1–1.5 mmol/l). The operation was performed under general anesthesia (fentanyl and low dose sevoflurane: 0.5–1.0%). On laparotomy, no clear signs of intestinal perforation were seen, but the ascites was turbid. The previous surgery for uterine myoma had caused adhesions around the sigmoid colon, and this had led to intestinal obstruction. Based on the laparotomy findings and the presence of hypermagnesemia, we deduced that the patient was at risk of intestinal obstruction, and laxative ingestion had increased the internal intestinal pressure to cause micro-perforations. At the same time, the patient had developed shock and hypermagnesemia due to the absorption of Mg from the laxative by the intestinal canal or peritoneum. To manage the hypermagnesemia, calcium chloride and furosemide were administered, and at the same time, the acidosis was treated by high-volume fluid and sodium bicarbonate administration. At the end of the surgery, blood pressure and heart rate had improved to 100/40 mmHg and 80 beats/min, respectively, and blood gas analysis had also improved (pH, 7.382; PaO_2, 119 mmHg; $PaCO_2$, 37.6 mmHg; Na, 134 mmol/l; K, 3.8 mmol/l; Ca, 1.52 mmol/l; HCO_3^-, 22.5 mmol/l; BE, −2.8 mmol/l). However, the ionized Mg level was still high (2.54 mmol/l). After the end of the surgery, the patient was transferred to the intensive care unit (ICU), and continuous hemodiafiltration (CHDF) was immediately started (conditions of the CHDF: filter, polyacrylonitrile membrane; dialysis rate, 500 ml/h; hemofiltration rate, 1000 ml/h). At that point, 1.5 μg/kg/min of norepinephrine and 2 u/h of vasopressin were required. The plasma Mg level was 11.4 mg/dl, but then it gradually decreased. At the same time, the circulation dynamics began to stabilize. On the next day, the plasma Mg level was 5.9 mg/dl, and the blood pressure fluctuated at 100–110/50–60 mmHg. On day 4 of the illness, the plasma Mg level had normalized at 1.8 mg/dl, and CHDF was discontinued. At this point, blood pressure was stable at about 120/60 mmHg with 0.06 μg/kg/min of norepinephrine. On day 6 of the illness, norepinephrine administration was discontinued (Fig. 1), and on day 12 of the illness, the patient was extubated. On day 15 of the illness, the patient was transferred to a general ward. The patient had mild motor impairment in the left upper and lower extremities due to brain infarction that was believed to have been caused by intracranial circulation failure during shock. The patient was discharged on day 84 of the illness without any other major problems.

Discussion

Reports on hypermagnesemia have been sporadic: there have been reports of people who had taken laxatives for a long time but did not develop hypermagnesemia and reports of patients who developed hypermagnesemia due to an underlying disease, such as delayed Mg excretion associated with renal failure [2]; and, as was the case with the present patient, hypermagnesemia was induced when a laxative was administered to patients with intestinal obstruction [3]. Some patients have died of hypermagnesemia, and pharmaceutical companies have warned about renal dysfunction and long-term administration of magnesium preparations. With regard to symptoms, hypermagnesemia patients first experience muscle weakness and consciousness disturbance and then respiratory depression

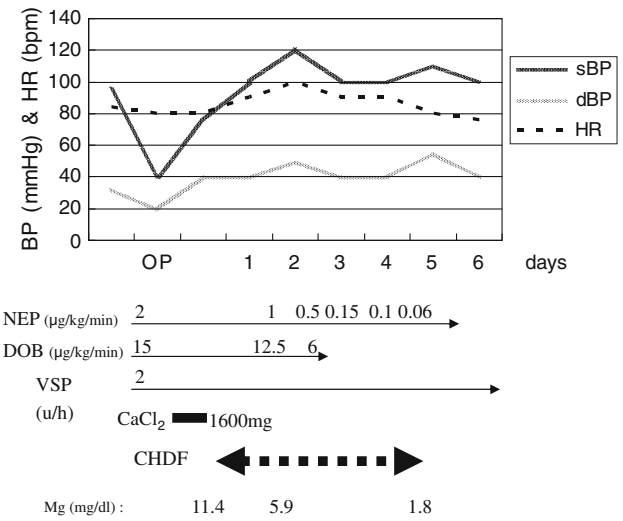

Fig. 1 Clinical course of the patient. *BP* blood pressure, *HR* heart rate, *sBP* systolic blood pressure, *dBP* diastolic blood pressure, *NEP* norepinephrine, *DOB* dobutamine, *VSP* vasopressin, *CHDF* continuous hemodiafiltration, *OP* operation

and circulatory collapse; as the disorder progresses, the ECG shows prolonged QT [4] and cardiac arrest [5]. Although hypermagnesemia requires early diagnosis and a rapid response, it is often diagnosed only when the condition is severe [6]. The present patient had typical hypermagnesemia; her consciousness level decreased, she had muscle weakness as the chief complaint, and her blood pressure gradually decreased, but at the same time, she strongly complained of left abdominal pain and had bloody stool. As a result, we incorrectly assumed the presence of peritonitis due to an occlusive lesion in the colon, thus delaying the correct diagnosis. Furthermore, because the patient had been on oral steroid therapy for a long time and suddenly developed hypermagnesemia, we erroneously diagnosed the patient with intestinal perforation. In fact, the present patient had turbid ascites, and fine colon perforation had occurred, and the two overlapping conditions complicated the diagnosis.

The present patient had been using a laxative for a long period (magnesium oxide 750 mg/day), but it is not clear whether or not renal dysfunction existedoriginally. On admission, the plasma blood urea nitrogen (BUN) and creatinine (Cr) levels were 19 and 1.29 mg/dl; however, these findings might been caused by shock or dehydration. Additionally, Mg was not included in this patient's transfusion. We cannot specify the conclusive cause of hypermagnesemia in this case, but as mentioned above, we suggest the following; in addition to a daily intake of Mg, rapid absorption of Mg by the intestinal canal or peritoneum had occurred because of intestinal obstruction and micro-perforations.

Moreover, in diagnosing, it was important that the patient did not have a high fever or an increased heart rate due to decreased blood pressure, which is associated with septic shock. If the delay of inflammatory reactions was caused by steroid, WBC and C-reactive protein (CRP) might have shown low values and the temperature would not have changed at the beginning of this event. However, during a state of shock, the heart rate would usually have increased in a patient without heart disease or neurological disturbance.

The common treatments for hypermagnesemia include high-dose Ca administration, Mg washout by high-volume fluid transfusion and diuretic administration, and hemopurification by dialysis. When the present patient was diagnosed, the initial therapy consisted of high-volume fluid transfusion and 1600 mg $CaCl_2$ administration, but only 5 mg furosemide administration. Administering a high-dose diuretic to a patient with severe shock can exacerbate circulatory dynamics, and making decisions under such circumstances is very difficult. However, by actively administering a diuretic and a large quantity of fluid, Mg can be washed out, possibly stabilizing circulatory dynamics earlier. This is a lesson that we learned from treating this patient. And CHDF was very effective in treating hypermagnesemia in this patient without causing circulation instability; indeed, Morimatsu et al. [7] reported that CHDF reduced serum magnesium in critically ill patients with acute renal failure.

In the present case, we provided perioperative management for a patient with hypermagnesemia and peritonitis. The findings in the present patient with complicated clinical symptoms reaffirm the difficulty and importance of making a diagnosis quickly by collecting various data.

References

1. Ogimoto M, Uematsu Y, Kabashima J, Suzuki K, Ito K. Survey on the quality of commercial Nigari (crude magnesium chloride [sea water]). Shokuhin Eiseigaku Zasshi (Japan). 2006;47:296–301. (in Japanese with English abstract).
2. Schlling JR. Fatal hypermagnesemia. Clin Nephrol. 2000;53:61–5.
3. Kontani M, Hara A, Ohta S, Ikeda T. Hypermagnesemia induced by massive cathartic ingestion in an elderly woman without preexisting renal dysfunction. Intern Med. 2005;44:448–52.
4. Kutsal E, Aydemir C, Eldes N, Demirel F, Polat R, Taspnar O, Kulah E. Severe hypermagnesemia as a result of excessive cathartic ingestion in a child without renal failure. Pediatr Emerg Care. 2007;23:570–2.
5. Qureshi T, Melonakos TK. Acute hypermagnesemia after laxative use. Ann Emerg Med. 1996;28:552–5.
6. So M, Ito H, Sobue K, Tsuda T, Katsuya H. Circulatory collapse caused by unnoticed hypermagnesemia in a hospitalized patient. J Anesth. 2007;21:273–6.
7. Morimatsu H, Uchino S, Bellomo R, Ronco C. Continuous venovenous hemodiafiltration or hemofiltration: impact on calcium, phosphate and magnesium concentrations. Int J Artif Organs. 2002;25:512–9.