J Anesth (2011) 25:935–938
DOI 10.1007/s00540-011-1220-6

CLINICAL REPORT

A case of cardiopulmonary arrest caused by laxatives-induced hypermagnesemia in a patient with anorexia nervosa and chronic renal failure

Hiroomi Tatsumi · Yoshiki Masuda · Hitoshi Imaizumi · Hiromitsu Kuroda · Shin-ichiro Yoshida · Ryoko Kyan · Kyoko Goto · Yasufumi Asai

Received: 13 December 2010 / Accepted: 17 August 2011 / Published online: 9 September 2011

Abstract We report a case of laxatives induced severe hypermagnesemia complicated with cardiopulmonary arrest. A 55-year-old woman, with nephritic syndrome and anorexia nervosa, was later transported to our emergency room (ER) because of oliguria and consciousness disturbance. During transfer to the intensive care unit from the ER, cardiopulmonary arrest suddenly occurred. Cardiopulmonary resuscitation was immediately performed, and spontaneous circulation was restored after 3 min. Thereafter, administration of dopamine, norepinephrine, and epinephrine was required to maintain systolic blood pressure at 80 mmHg. Arterial blood gas analysis showed severe metabolic alkalosis, and blood biochemical tests revealed hypermagnesemia (serum magnesium concentration, 18.5 mg/dl) and renal dysfunction. Continuous infusion of diuretics followed by massive hydration and continuous hemodiafiltration (CHDF) was started. Five days after starting CHDF, magnesium concentration was almost normalized and administration of catecholamine was stopped. It was thought that progression of renal dysfunction that occurred in the patient taking a magnesium product for chronic constipation caused reduction in magnesium excretion ability, resulting in hypermagnesemia-induced cardiopulmonary arrest. To avoid a rebound phenomenon following magnesium flux from cells, continuous blood purification seems to be an effective treatment for symptomatic hypermagnesemia.

H. Tatsumi (✉) · Y. Masuda · H. Imaizumi · H. Kuroda · S. Yoshida · R. Kyan · K. Goto · Y. Asai
Department of Traumatology and Critical Care Medicine, School of Medicine, Sapporo Medical University, South 1, West 16, Chuo-ku, Sapporo, Hokkaido 060-8543, Japan
e-mail: htatsumi@sapmed.ac.jp

Keywords Hypermagnesemia · Continuous hemodiafiltration · Blood purification · Laxatives

Introduction

Hypermagnesemia is a rare disease caused by various conditions. We report a patient in whom severe hypermagnesemia was induced by a magnesium product taken for treatment of chronic constipation and who was saved after cardiopulmonary arrest.

Case

A 55 year-old woman was transferred to our critical care medical center because of consciousness disturbance and oliguria.

Onset and course

The patient was receiving treatment on an outpatient basis for nephrotic syndrome and anorexia nervosa. Usually, she caused herself to vomit because of anorexia nervosa. She visited a local clinic because of common cold-like symptoms and was later transported to our emergency room (ER) center due to decrease in urine volume and consciousness disorder.

Findings on admission to the ER

The patient had severe malnutrition (height 158 cm, weight 26.3 kg). At admission, Japan Coma Scale (JCS) was 0–1, Glasgow Coma Scale (GCS) was 13, blood pressure was

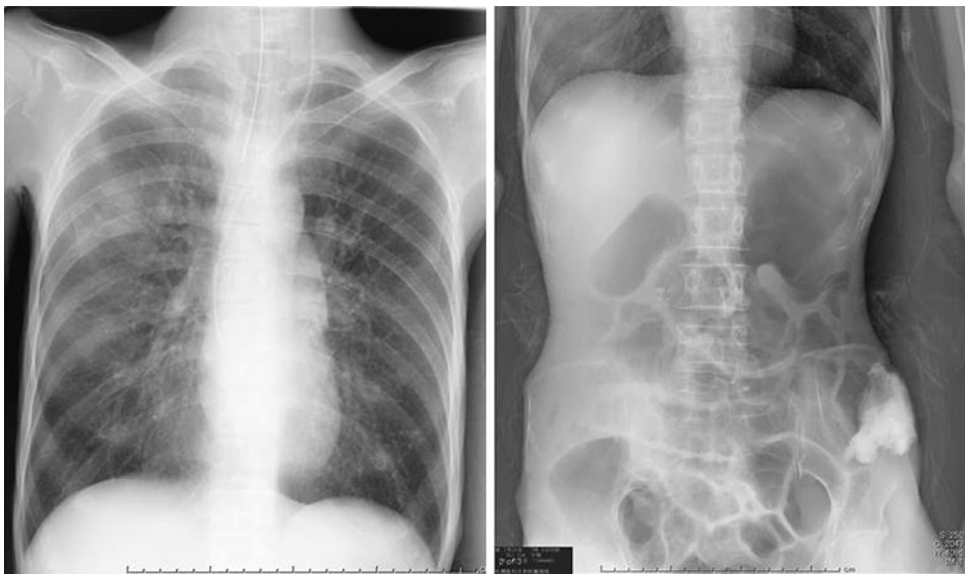

Fig. 1 Chest and abnormal X-ray on admission to the emergency room (ER). Chest X-ray showed no notable abnormalities, but intestinal paralysis was suspected by the findings of abdominal X-ray

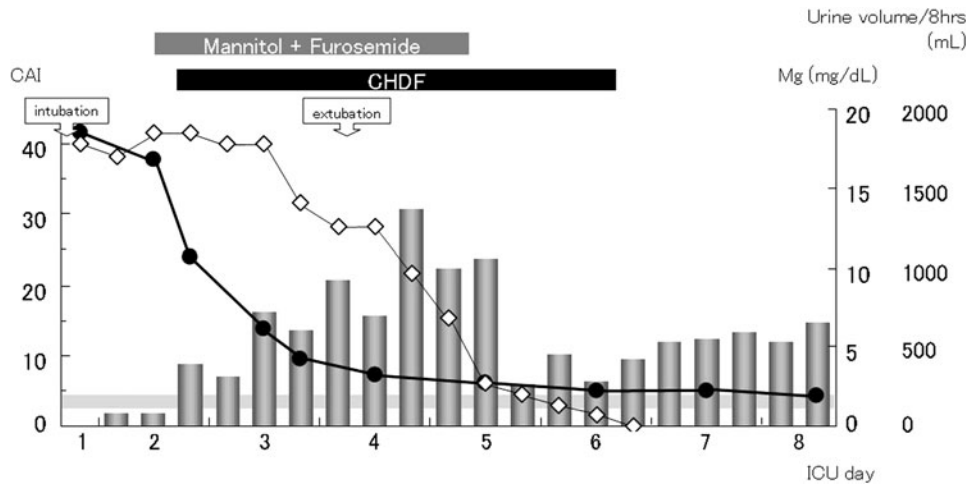

Fig. 2 Clinical course. *Filled circles* magnesium (Mg) (mg/dl), *open diamonds* cathecholamine index (CAI), *light and dark grey-shaded bars* urine volume/8 h, *light grey-shaded bar* normal range of magnesium concentration. Cathecholamine index = dopamine [μg/kg/min] + dobutamine [μg/kg/min] + (epinephrine [μg/kg/min] + norepinephrine [μg/kg/min]) × 100. *CHDF* continuous hemodiafiltration, *ICU* intensive care unit

76/48 mmHg, pulse was 70/min, and body temperature was 37.4°C. Results of arterial blood gas analysis in room air [pH, 7.54; PaO_2, 78 mmHg; $PaCO_2$, 89 mmHg; bicarbonate ion (HCO_3^-), 80 mmol/l; base excess (BE), +51 mmol/l; lactate, 28 mg/dl] indicated severe metabolic alkalosis followed by compensation of respiratory acidosis. Breathing was shallow (18 breaths per minute). Biochemical tests were as follows: albumin, 3.9 mg/dl; blood urea nitrogen (BUN), 49 mg/dl; creatinine (Cr), 3.3 mg/dl; sodium ion (Na^+), 143 mEq/l; potassium ion (K^+), 2.5 mEq/l; chloride ion (Cl^-), 63 mEq/l; calcium ion (Ca^{2+}), 11.2 mg/dl. Ultrasonic examination showed that the inferior vena cava diameter was 11 mm, indicating slight dehydration. A chest X-ray showed no notable abnormalities, but an abdominal X-ray showed accumulation of intestinal gas, and intestinal paralysis was suspected (Fig. 1).

Clinical course (Fig. 2)

Infusion of acetated Ringer's solution was started for fluid resuscitation. Because SpO_2 gradually decreased, oxygen was given via a face mask at 5 l/min. During transfer to the intensive care unit (ICU) for examination and treatment, respiratory arrest followed by cardiac arrest suddenly occurred. Intubation was immediately performed, and cardiac massage was started. Breathing and heartbeat were restored following intravenous injection of 1 mg epinephrine. Thereafter, maintenance of systolic blood pressure at 80 mmHg required administration of dopamine, norepinephrine, and epinephrine at maximum doses of 15, 0.2, and 0.06 μg/kg/min, respectively (catecholamine index: CAI = 41).

Results of blood gas analysis and biochemical tests on admission to the ICU were as follows: pH, 7.70; HCO_3^-,

65.7 mmol/l; BE, 35.5 mmol/l (indicating severe metabolic alkalosis); albumin, 3.4 mg/dl; BUN, 55 mg/dl; Cr, 3.6 mg/dl; Na^+, 141 mEq/l; K^+, 3.1 mEq/l; Cl^-, 78 mEq/l; Ca^{2+}, 9.1 mg/dl; magnesium ion (Mg^{2+}), 18.5 mg/dl (normal range, 1.5–2.0); phosphorus (P), 6.9 mg/dl (normal range, 2.5–4.5), suggesting acute renal failure and hypermagnesemia complicating chronic renal failure and with accompanying metabolic alkalosis. Strong ion difference (SID) was 66.2 mEq/l, which also indicated metabolic alkalosis. As urine volume was less than 10 ml/h, continuous infusion of mannitol/furosemide was started after rapid and massive hydration (3 l acetate Ringer's solution for 2 h), and continuous hemodiafiltration (CHDF) was started for fluid management and electrolyte adjustment. After starting CHDF, magnesium concentration gradually decreased and was 10.7 mg/dl at 24 h later. Magnesium concentration had decreased to 4.2 mg/dl at 48 h after starting CHDF, and the dose of norepinephrine was reduced to 0.17 μg/kg/min and administration of epinephrine was stopped. Five days after starting CHDF (ICU day 6), magnesium had decreased to an almost normal level (2.2 mg/dl) and administration of dopamine and norepinephrine was stopped. CHDF was continued until ICU day 6, and the patient was transferred to a general ward on ICU day 14 without any consciousness disorder or other sequelae.

Discussion

Hypermagnesemia is a rare disease. The normal range of serum magnesium is 1.5–2.0 mg/dl (normal range in our hospital), and initial symptoms appear when magnesium concentration exceeds 5 mg/dl [1]. Initial symptoms include low blood pressure, nausea/vomiting, flushed face, urinary retention, and ileus. Progression of hypermagnesemia results in flaccid paralysis of skeletal muscle, decrease in deep tendon reflexes, respiratory depression, coma, and bradycardia arrhythmia and even cardiac arrest in some cases [2]. Initial symptoms of hypermagnesemia including decrease in blood pressure, decrease in urine volume, consciousness disorder, and ileus were seen in our patient when she was brought to our hospital, and respiratory suppression and cardiac arrest later occurred.

Creatinine clearance in our patient was low (38.1 ml/min) as a result of nephrotic syndrome, and she also had chronic renal failure (Cr level, 3.6 mg/dl). Generally, serum magnesium does not increase unless creatinine clearance decreases to below 20 ml/min, and magnesium level in chronic renal failure patients on maintenance hemodialysis can be high (2.4–3.6 mg/dl), but symptoms do not appear. Thus, even in cases of renal dysfunction, severe hypermagnesemia with symptoms will not occur unless magnesium medication is taken [3]. In our patient, it is thought that progression of renal dysfunction in response to infection that occurred while she was taking a magnesium product (magnesium oxide, 1.2 g/day) for treatment of chronic constipation resulted in reduction in magnesium excretion ability and eventually led to respiratory and cardiac arrest as a result of hypermagnesemia. Moreover, it might be another possible mechanism of hypermagnesemia that severe constipation needed further laxatives ingestion and brought about the long stay of magnesium in the intestine, which caused increase in magnesium absorption and deterioration of hypermagnesemia. It was reported that hypermagnesemia itself could cause bowel hypomotility [4]; consequently, this mechanism was thought to produce a "vicious circle" in hypermagnesemia.

Acute treatments for hypermagnesemia include inhibition of the action of magnesium and elimination of magnesium from the blood [5, 6]. The former treatment is carried out using a calcium preparation, and the latter is performed by blood purification. In the case of blood purification, because most of the magnesium in the body exists in cells, it is possible that the serum magnesium level will increase again after hemodialysis for a short duration [7]. In our case, continuous blood purification was performed and magnesium decreased from the pre-CHDF level of 18.5–10.7 mg/dl at 24 h after the start of CHDF. Magnesium further decreased to 4.2 mg/dl after CHDF for 48 h, and tapering of the doses of catecholamines was started. Magnesium had decreased to almost 2.2 mg/dl after 5 days, and CHDF was stopped. Because the patient could be weaned from catecholamines around the same time, it is thought that the reduction in magnesium concentration was related to the improvement in clinical symptoms.

Our patient had nephritic syndrome with renal dysfunction and was regularly taking magnesium oxide for chronic constipation, and it is thought that hypermagnesemia occurred as a result of progression of renal dysfunction caused by infection and eventually led to respiratory and cardiac arrest. The patient was successfully treated using CHDF for 5 days without any recurrent rise in magnesium level because of rebound phenomenon. Our case indicates the need to consider the risk of hypermagnesemia occurring in patients with renal disorder who have been taking a magnesium product for a long time and the importance of monitoring renal function and electrolytes in such patients. The results in our case also suggest that continuous blood purification is effective for preventing the rebound phenomenon if hypermagnesemia with clinical symptoms occurs.

Conclusion

Acute deterioration of chronic renal dysfunction occurred in a patient in whom severe hypermagnesemia developed because of magnesium medication for constipation and

eventually led to cardiac arrest. There is a risk of hypermagnesemia occurring in patients with renal disorder who are regularly taking magnesium, and continuous blood purification is thought to be effective for the treatment of hypermagnesemia with clinical symptoms.

References

1. Kontani M, Hara A, Ohta S, Ikeda T. Hypermagnesemia induced by massive cathartic ingestion in an elderly woman without pre-existing renal dysfunction. Intern Med. 2005;44:448–52.
2. 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.
3. Navarro-González JF. Magnesium in dialysis patients: serum levels and clinical implications. Clin Nephrol. 1998;49:373–8.
4. Mordes JP, Wacker WEC. Excess magnesium. Pharmacol Rev. 1977;29:273–300.
5. Vissers RJ, Purssell R. Iatrogenic magnesium overdose: two case reports. J Emerg Med. 1996;14:187–91.
6. Razavi B, Somers D. Hypermagnesemia-induced multiorgan failure. Am J Med. 2000;108:686–7.
7. Schelling JR. Fatal hypermagnesemia. Clin Nephrol. 2000;53:61–5.