CASE REPORT
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마그네슘 포함 비료 음독 후 발생한 중증의 고마그네슘혈증의 1례

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Severe Case of Hypermagnesemia Caused by Ingesting Magnesium Containing Fertilizer

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Hypermagnesemia is a rare condition that is usually iatrogenic in patients with elderly or renal failure. Severe hypermagnesemia is uncommon in patients with a normal renal function. Symptoms due to hypermagnesemia can range from mild symptoms, such as nausea, to severe symptoms, such as cardiac and respiratory arrest. This paper describes a case of a 49-year-old woman who ingested a magnesium-containing fertilizer with normal renal function. Cardiac arrest occurred eight hours after poisoning. Electrocardiography changed from a narrow QRS to a wide QRS and then to a complete atrioventricular block. Her hemodynamic state was unstable. Continuous renal replacement therapy was performed to remove magnesium from the blood, with the subsequent resolution of arrhythmia and hemodynamic stabilization. This paper reviews the pathophysiologic effects of magnesium on the cardiovascular system, clinical manifestation, and treatment of hypermagnesemia.

Keywords: Hypermagnesemia, Poisoning, Fertilizers

INTRODUCTION

Magnesium is an essential intracellular ion that has many different functions such as, acting as a cofactor for many enzymes, a binding partner of nucleotides, stabilizing nucleic acids and membranes and antagonizing the actions of calcium[1].

Hypermagnesemia is relatively uncommon but can be seen in patients with chronic and acute renal failure who are taking magnesium-containing antacids, laxatives, enemas, or infusions[2]. Renal function is important factor because renal excretion is the major route of magnesium elimination from the body and a positive magnesium balance would be expected in patients with renal impairment. Fatal case due to hypermagnesemia is uncommon in patients with normal renal function[3].

In hypermagnesemia, because magnesium acts as a calcium channel blocker, the blood vessels tend do dilate, which leads to lower blood pressure[4]. Cardioinhibitory actions include prolongation of PR interval, increase in QT interval, complete atrioventricular block and cardiac arrest[5-7]. Furthermore, patients with hypermagnesemia present a disturbance of consciousness[8], respiratory failure due to respiratory muscle paralysis[4]. Therefore mild hypermagnesemia presented general malaise, such as nausea and vomiting, whereas serious hypermagnesemia can result in death[9].

Fertilizer, natural or artificial substance containing the chemical elements that

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improve growth and productiveness of plants. Magnesium fertilizers vary greatly in their solubility in water, affecting the availability to plants once applied to the soil. Fertilizer poisoning is rare and clinical course might depend on composition of fertilizer.

We describe a successful treatment of hypermagnesemia using continuous renal replacement therapy (CRRT) in patient with normal renal function, who showed severe hypermagnesemia, presented with mental change, paralysis, cardiopulmonary dysfunction with variable Electrocardiography (ECG) changes after poisoning with magnesium containing fertilizer.

CASE REPORT

A 49-year-old woman with no known co-morbid was referred to the emergency department for drowsy mentality after taking magnesium containing fertilizer. She ingested 300 ml fertilizer (17.1 mg magnesium) in an attempted suicide following a familial dispute.

On arrival at our emergency department, the patient was hypothermic (35.4° C) with a respiratory rate of 20 breaths/min, heart rate of 105 beats/min, and blood pressure of 140/90 mm Hg. The physical examination was unremarkable. Arterial blood gas analysis in O_2 4L showed a pH of 7.08, pO_2 203 mm Hg, pCO_2 91 mmHg, HCO_3^- 27 mEq/L and O_2 saturation 99%. The results were interpreted as indicative of acute severe respiratory acidosis associated with respiratory depression. Intubation was performed and placed on mechanical ventilation. Laboratory values were WBC 27170/mm^3 (neutrophils, 78.1%), hemoglobin 16.7 g/dl, BUN 28 mg/dl, serum creatinine 0.92 mg/dl (estimated glomerular filtration rate: 73.35 ml/min/1.73 m^2), sodium 137 mEq/dl, potassium 4.0 mEq/dl, chloride 148 mEq/dl, calcium 10.2 mg/dl, phosphorus 6.8 mg/dl, and lactate dehydrogenase 483 IU/L. We did not checked for magnesium level. Initial ECG showed first degree atrioventricular block (PR interval 218 msec). In neurologic examination, there is loss of deep tendon reflexes and flaccid paralysis. Computed tomography of the brain showed no acute lesion. Her ECG and vital sign were close monitored. Cardiac arrest occurred 8 hours after poisoning. ECG changed from narrow QRS tachycardia to wide QRS, and then to complete atrioventricular block (Fig. 1). As the heart rate slows, she was resuscitated with chest compression, epinephrine bolus and atropine. vasopressors were infused for hypotension. Her pulse rate fluctuated from 40 to 70 beats/min and ECG showed junctional bradycardia. Afterward cardiac arrest occurred, we checked magnesium level. Her magnesium level was too high to be unmeasurable (〉14 mg/dl). We started CRRT (continuous veno-venous hemodiafiltration) in an attempt to decrease magnesium urgently. Her serum mag-

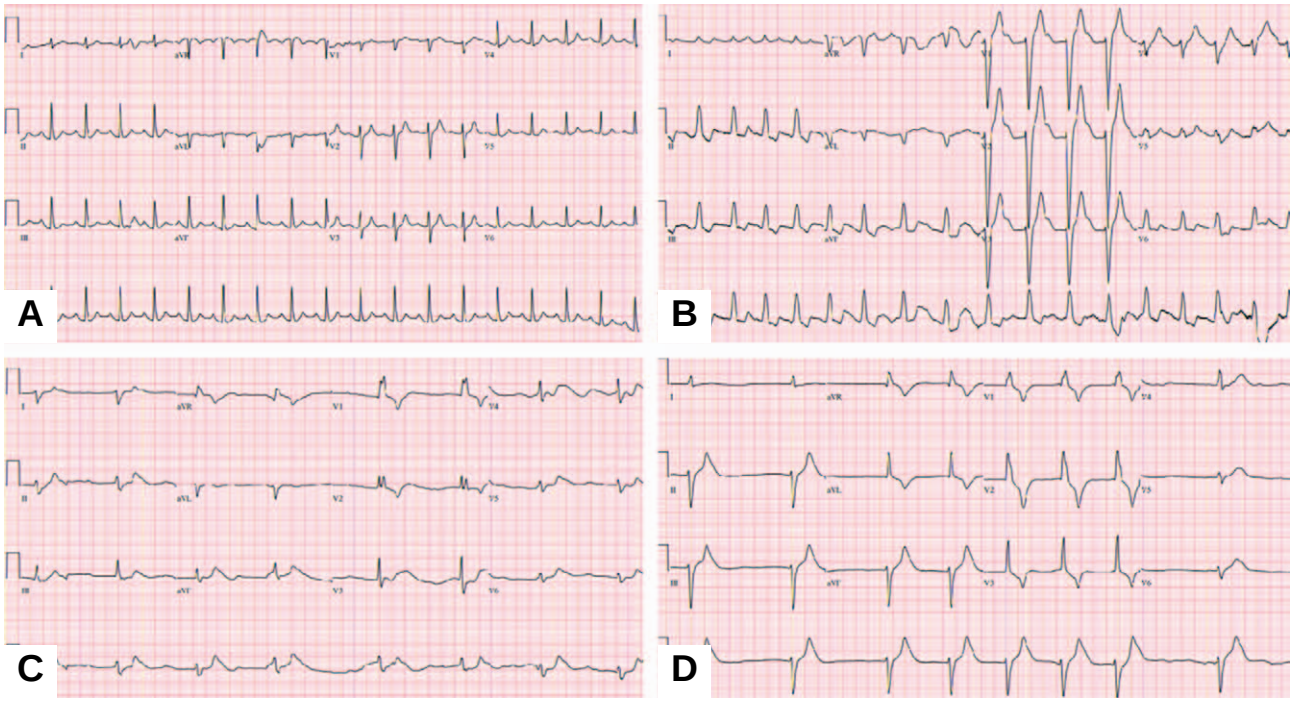

Fig. 1. Electrocardiography changes over times (**A**) On admission, 1st degree atrioventricular block and prolonged PR interval were observed (**B**) 8 hours after poisoning, QRS widening, T-wave inversion and prolonged QTc interval were observed (**C**) 9 hours after poisoning, complete atrioventricular block and (**D**) 12 hours after poisoning, junctional rhythm was observed.

nesium had decreased to 9.43 mg/dl at 8 hours after CRRT (Fig. 2).

On hospital day 3, magnesium concentration decreased to 5.0 mg/dl and an ECG showed a sinus rhythm with QT prolongation. Neither significant arrhythmias nor blood pressure fluctuations were observed. She was able to move and recover consciousness to the extent of responding to stimuli as 24 hrs passed after starting CRRT. Her magnesium concentration and ECG completely normalized within 48 hours, thus, CRRT and vasopressors were stopped. She extubated on day 8 and left the intensive care unit on day 10. At the 15 day of admission she was discharged without any sequelae or complaint. This report is approved by IRB of Soonchunhyang university Cheonan hospital. (2020-05-010)

DISCUSSION

Magnesium homeostasis is mainly dependent on gastrointestinal absorption and renal excretion, with the kidneys playing a major role in magnesium regulation[10]. Unlike most other filtered solutes, only 10% of filtered magnesium is reabsorbed in the proximal tubule of the kidney and approximately 50-70% of magnesium is reabsorbed in the thick ascending limb of Henle[11]. Therefore, the cause of hypermagnesemia is usually iatrogenic and there is a higher risk associated with elderly patients, chronic renal failure with reduced excretion and gastrointestinal disorders with increased absorption[12,13]. Although our patient was a normal renal function, hypermagnesemia occurred due to a large amount of magnesium poisoning.

Patients with symptomatic hypermagnesemia can present different clinical manifestation depending on the level. When the plasma magnesium concentration reaches levels of 4.8-7.2 mg/dl symptoms including nausea, flushing, headache, lethargy, and diminished deep tendon reflexes are noticed[14]. Magnesium levels of 7.2-12 mg/dl result in hypocalcemia, absent deep tendon reflexes, hypotension and bradycardia[15]. Magnesium levels above 12 mg/dl cause muscle paralysis, complete atrioventricular block, respiratory and cardiac arrest[16].

Treatment approach for hypermagnesemia is dependent on the renal function, magnesium concentration, and clinical symptoms[17]. Patients with normal renal function and mild asymptomatic hypermagnesemia require no treatment except the removal of all sources of exogenous magnesium. In severe cases, close monitoring of the ECG, blood pressure, and neuromuscular function and early treatment are necessary. To improve the renal clearance of magnesium, loop diuretics and saline diuresis are intuitive options[18]. Calcium acts as an antagonist in hypermagnesemia[4,19]. Administration of Intravenous calcium reverse the neuromuscular and cardiac effects of hypermagnesemia[4] and bridges life-threatening hypermagnesemia to dialysis. This should be given 1g of intravenous calcium gluconate, followed by the infusion of 150-100 mg of calcium over 5-10 minutes[20]. When kidney function is impaired or the patient is symptomatic from severe hypermagnesemia, hemodialysis is needed. Hemodialysis, with its higher flow rates, would be preferred in acute magnesium intoxication because it can lower serum magnesium levels more efficiently to safer levels[17]. When hemodynamic state is unstable in severe hypermagnesemia, hemodialysis is impossible. So CRRT might be good alternative treatment. The most common sources of nutrients in mineral fertilizers

Hospital day	1	2	3	4	5-8	9-15
Mental state						
Drowsy						
Alert						
Blood pressure						
SBP (mmHg)	140	80	120	130	130	110
DBP (mmHg)	90	50	80	80	80	60
Heart rate	105	29	75	110	98	78
Magnesium level (mg/dl)	>14	9.43	5	2.7	2	1.7
QTc interval (ms)	418	595	500	385	400	400
Treatment						
CVVHDF						
Mechanical ventilation						

Fig. 2. Summary of the clinical course of the patient and treatment. SBP: systolic blood pressure, DBP: diastolic blood pressure, CVVHDF: continuous veno-veno hemodiafiltration

are nitrogen, potassium and phosphate. Therefore, at the time of admission, we checked for phosphate level and did not recognize hypermagnesemia. Afterward, hypermagnesemia was confirmed, cardiac arrest occurred, and CRRT was quickly applied to quickly remove magnesium. Our patient responded well to CRRT and her magnesium levels normalized after 2 days.

CONCLUSION

CRRT must be considered to drop magnesium levels to a non-toxic level because hemodynamic state is unstable in severe hypermagnesemia.

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