

An Unnoticed Case; Hypermagnesemia at the Emergency Department

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ABSTRACT

Introduction. Hypermagnesemia generally develops in people with renal function disorders or due to exogen Mg intake for constipation. Hospitalized cases of fatal hypermagnesemia are rare in the literature. The aim of this case report was to see if fatal progression could be due to delayed diagnose.

Case Presentation. A 61 year old woman presented at the emergency department (ED) for the evaluation of her symptoms which were leg pain, weakness, nausea, constipation and general debility. In her prior history, she had used magnesia calcine for laxative until two weeks before. Electrocardiography showed atrial fibrillation with high ventricular respond (HVRAF). Initial serum magnesium (Mg) concentration was 6.80 mEq/l. 10% calcium gluconate with 20 ml used to antagonize symptoms for treatment. Intravenous (IV) metoprolol was used for HVRAF but the patient was unresponsive. On the second day Mg rose to 7.06 mEq/l. The patient's consciousness was altered, she developed lethargy, and hemodynamic instability was revealed. In addition, respiratory distress was present and patient was intubated. Therefore, she was diagnosed with a suspected Mg intoxication due to laxative use. Continuous hemodiafiltration (CHDF) was urgently used to decrease Mg. On the third day the patient was unresponsive to the treatment and died in intensive care unit (ICU).

Conclusion. Patients with nonspecific symptoms due to a prolonged laxative use can be admitted to the ED. Hypotension, altering consciousness and cardiac dysrhythmias can be revealed quickly and therefore the progress is fatal. Mg intoxication must be noticed early in the ED. IV calcium directly antagonises the effects of magnesium. It can reverse effects such as cardiac

arrhythmias. IV normal saline must be used for supportive treatment and if those not responding to intravenous calcium and other supportive measures, CHDF must be used urgently for all patients with features of life threatening hypermagnesemia.

Keywords: Emergency Department, Laxative, Hypermagnesemia

INTRODUCTION

Hypermagnesemia generally develops in people with abnormal kidney functions or in those using exogen magnesium for constipation (1,2). Severe hypermagnesemia is a rare clinical condition that often results with death in hospital. Because of previous symptoms such as altered consciousness, vomiting interferes with early symptoms of hypermagnesemia. It can't be noticed until fatal progression is present (3). Hypermagnesemia is characterized by the loss of progressive muscle strength and dysfunction of central nervous system and cardiovascular system. Clinical signs and symptoms such as sepsis, gut obstruction and first motor neuron diseases are non-specific (4). Our aim at reporting this case was to show that an unnoticed hypermagnesemia had been fatal at the emergency department (ED).

CASE PRESENTATION

A 61 year old woman known for suffering from schizophrenia presented at the ED for the evaluation of her symptoms which were leg pain, weakness, nausea, constipation and general debility. We learned that she had used magnesia calcine for laxative until two weeks before. Initially the patient's Glasgow Coma Scale was 14, blood pressure was 82/60 mmHg,

heart rate was 148 beats/minute, respiratory rate was 27 breaths/minute, temperature was 36,2 °C, oxygen saturation 88% and finger stick blood glucose was 139 mg/dl. Physical examination findings were normal auscultation lung sounds, tachycardia, normal pupils, and lower extremity strength muscle 2/5, distended abdomen and decreased bowel sounds.

Radiographic signs were normal. Electrocardiography (ECG) showed atrial fibrillation with high ventricular respond (HVRAF). Initially, laboratory results were as follows; normal complete blood count, BUN: 32.91 mg/dl, creatinine (cre): 1.31 mg/dl, magnesium (Mg): 6.80 mEq/l, potassium: 5.66 mEq/l, calcium: 6.74 mEq/l. Hypocalcaemia was treated with 20 ml of 10% calcium gluconate. Intravenous (IV) metoprolol was used for HVRAF. Normal saline, dopamine and noradrenalin IV infusion were started to improve perfusion. The following morning, the patient's consciousness was altered, she developed lethargy, and hemodynamic instability was revealed. In addition, respiratory distress was present and patient was intubated. The patient's condition worsened quickly and she developed shock and acute kidney failure. HVRAF was unresponsive to electrical and medical cardioversion. Serum Mg level rose to 7.06 mEq/l. Therefore, she was diagnosed with a suspected Mg intoxication due to laxative use. A nephrologist was then consulted for the treatment of symptomatic hypermagnesemia. Continuous hemodiafiltration (CHDF) was used to decrease Mg urgently. Following CHDF, laboratory results were BUN: 25.39 mg/dl, cre:0.96 mg/dl and Mg:4.47 mEq/l respectively. Atrial fibrillation didn't improve and overdrive pacing was performed. On the third day the patient was transferred to ICU and hemodynamic parameters became worse. Serum Mg level didn't decrease and HVRAF didn't improve. The patient died in ICU.

DISCUSSION

Normal serum Mg concentration is between 0.7-1.1 mmol/L. Kidneys provide autoregulation of serum Mg levels (5). Hypermagnesemia is a problem if an excessive Mg intake or renal failure is present. Hypermagnesemia is defined as serum Mg levels over 1.5 mmol/L. Mg levels rise in plasma as kidney function decreases (6). When exogenous Mg intake is given as antacids or laxatives at usual therapeutic doses especially in patients with renal failure, severe and symptomatic hypermagnesemia can also be induced (7).

Mg intakes may be administered intravenously, orally and via rectal enema; hypermagnesemia can occur at usual therapeutic doses especially in patients with dysfunction in regulation of Mg levels (2,8,9).

Clinical symptoms are present when Mg levels are over 2 mmol/L in plasma (6). A correlation is seen between plasma Mg levels and clinical presentation of hypermagnesemia (9–11).

Clinical correlations of plasma Mg levels are showed in Table 1.

Hypermagnesemia inhibits parathyroid hormone secretions and creates a decrease in calcium levels in plasma. Hypocalcemia can cause of ECG changes (12).

Treatment of hypermagnesemia changes according to renal function and plasma Mg levels. Loop diuretics increase Mg excretion in patients who prevent renal functions or with mild or moderate re-

Table 1. The Correlation between Plasma Mg Level and Clinical Symptoms in Hypermagnesemia

Plasma Mg Level	Clinic presentation
2-3mmol/L	nausea, headache, paraesthesia, erythema on body and face
3-5 mmol/L	Somnolence, loss of deep tendon reflex, hypotension and hypocalcaemia, ECG changes
>4-5 mmol/L	Hypotension, conduction disorders and bradycardia, a prolonged PR, β wide QRS, μ prolonged QTc, complete heart block in ECG, and cardiac arrest.

ECG: Electrocardiogram, μ Prolonged corrected QT interval: QTc >480 ms
 β Wide QRS complex duration \geq 120 ms, α Prolonged PR interval >200 ms

nal failure. Loop diuretics can be used for treatment of mild or moderate hypermagnesemia but CDHF is needed in presence of neurological and cardiovascular symptoms for treatment of hypermagnesemia. In symptomatic patients, before CDHF, calcium is used to treat the symptoms (13).

CONCLUSION(S)

Patients with nonspecific symptoms due to a prolonged laxative use can be admitted to the ED. Hypotension, altering consciousness and cardiac dysthymias develop quickly and, therefore, often results death in hospital. Mg intoxication must be noticed early in the ED. Iv calcium reverse cardiac arrhythmia, iv normal saline and CHDF must be used urgently.

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Contribution of authors

Mustafa Korkut examined the patient, diagnosed the case, wrote the first version and approved the final version of the paper and edited it, Ömer Faruk Karakoyun read the literature and participated in writing.

Conflict-of-interest

All authors declare that they have no conflict of interest.

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