

# Acute Respiratory and Renal Failure due to Hypermagnesemia, Induced by Counter Laxatives in an Elderly Man

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Sir,

Increased serum magnesium levels are generally iatrogenic and rare clinical situations with significant cardiovascular, neurological and neuromuscular effects. Patients with bowel disorders, renal insufficiency, and patients with old age are especially at high risk for hypermagnesemia. The treatment of hypermagnesemia includes discontinuing the magnesium intake, gastrointestinal decontamination, intravenous calcium gluconate and hemodialysis [1]. Herein, we report a life threatening hypermagnesemia situation induced by a magnesium containing product, used for the treatment of chronic constipation.

A 70-year-old man presented with constipation, chest tightness and dyspnea. He had a history of chronic obstructive pulmonary disease, chronic kidney failure and diabetes mellitus. Contrast enema was planned on the first hospital day. The patient became lethargic in the second day of hospitalization. Abdominal pain with diminished bowel sounds was observed. The neurological examination showed symmetric decrease in muscle tone and in deep tendon reflexes. Mechanical ventilation was initiated according to arterial blood gas results. Biochemical results were as follows; magnesium, 9.07 (1.5–2.6) mEq/L; calcium, 7.78 (8.4–10.2) mEq/L; sodium, 140 (135–145) mEq/L; potassium, 5.5 (3.5–5.1) mEq/L; creatine, 3.85 (0.2–1.2) mg/dL; blood urine nitrogen, 202.5 (70–105) mg/dL. Calcium gluconate was infused for antagonizing most of the clinical effects of toxicity. A nasogastric and rectal tube were inserted for gastrointestinal decontamination. Emergency continuous veno-venous haemodialysis (CVVHD) for hypermagnesemia was performed and continued during 48 hours. Finally, after a 48 hour CVVHD treatment, the Mg level decreased to 2.0 mEq/L) and the patient was extubated. The follow up period was uneventful.

Magnesium is an important intracellular cation that functions as a co-factor in several enzyme pathways. The magnesium

plasma value is generally between 1.4 and 2.1 mEq/L and closely regulated through interaction of the gastrointestinal absorption, bone store and kidney excretion [2]. Hypermagnesemia can occur with various processes such as renal failure, Addison disease, milk alkali syndrome, hypothyroidism and with lithium therapy. Decreased elimination, magnesium overdose and increased absorption are the main causes of hypermagnesemia. Our patient had both decreased elimination as a consequence of kidney failure and magnesium overdose due to laxative usage [3]. Clinical manifestations of hypermagnesemia vary according to the serum Mg concentration. Symptomatic hypermagnesemia typically presents with neurological, neuromuscular and cardiac manifestations including hyporeflexia, sedation, muscle weakness and respiratory depression but many physicians are relatively unfamiliar with these conditions [4]. Especially elder patients are at high risk of magnesium toxicity as the kidney function declines with age [5]. Relationship between hypermagnesemia and laxative usage should induce physicians to pay more attention to hypermagnesemia especially in this subgroup of patients and urgent CVVHD is highly effective in preventing significant morbidity and mortality.

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