

Symptomatic Hypermagnesemia in the Absence of Renal Failure

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Abstract

Hypermagnesemia is an uncommon clinical finding, and symptomatic hypermagnesemia is even rarer. Symptomatic hypermagnesemia has a low incidence of occurrence, because the kidney is able to eliminate excess magnesium by rapidly reducing its tubular reabsorption to almost negligible amounts. Renal failure is the most common cause of hypermagnesemia.^[1] Other causes of hypermagnesemia such as lithium therapy, Milk alkali syndrome, exogenous intake, Addison disease, tumor lysis syndrome and hypothyroidism are much rarer.^[1] We report a case of symptomatic hypermagnesemia, in a sixty-one year old woman that has been taking milk of magnesia for chronic constipation.

INTRODUCTION

Magnesium is one of the body's major electrolytes. It is the second most common intracellular cation in the body and plays a crucial role in deoxyribonucleic acid (DNA), and protein synthesis. In addition, magnesium is a necessary cofactor for most enzymes in phosphorylation reactions and parathyroid hormone synthesis. [2]

Magnesium is absorbed in the ileum and excreted in stool and urine. The minimum daily requirement of magnesium is 300-350 mg, or 15 mmol; this amount is easily obtainable with a normal daily intake of fruits, seeds, and vegetables because magnesium is a component of chlorophyll and is present in high concentrations in all green plants. [2]

Kidneys are the main organ involved in magnesium homeostasis. Magnesium absorption occurs primarily in the proximal tubule and thick ascending limb of the loop of Henle. Kidney is very effective in excreting excess magnesium. Therefore, hypermagnesemia is very rare and symptomatic hypermagnesemia is even rarer in absences of renal insufficiency. [2] This case illustrates the potential for severe hypermagnesemia in patients with normal renal function and the importance of a complete history. Recognition of Hypermagnesemia and its effects is critical to institution of appropriate therapy and prevention of its lethal effects.

CASE

A 61-year old woman with a past medical history of chronic

constipation and hypertension presented to the emergency department with generalized weakness, body aches, and abdominal discomfort for duration of one week. Her home medications included the following: Verapamil, Lovastatin, and Hydrochlorothiazide. She was also taking over the counter milk of magnesia for chronic constipation. Her physical examination was unremarkable except for signs of mild dehydration.

Her Laboratory investigation revealed a normal EKG and serum levels of the following: Magnesium 8.8 mg/dl (1.8-2.6 mg/dl), Phosphate 2.2 mg/dl (2.5-4.5 mg/dl), and Calcium 7.3 mg/dl (8.6-10.0). Her serum Creatinine was 0.9 mg/dl (0.5-1.1 mg/dl) with blood urea nitrogen level of 17 mg/dl (2-22 mg/dl).

During her hospital stay she was placed on telemetry, her diuretics were held, and milk of magnesia was discontinued. She was re-hydrated with isotonic fluids. Serum Magnesium levels were followed every twenty-four hours and normalized to 1.5 mg/dl, on day three. By forty-eight hours her symptoms had diminished greatly and by seventy-two hours had resolved completely. Patient was discharged with instructions to avoid Milk of Magnesia. A follow up visit was scheduled in 2 weeks and she was found to be completely symptoms free.

DISCUSSION

Magnesium homeostasis, like that of other ions, is maintained by the kidneys. Severe hypermagnesemia with

life-threatening symptoms is usually seen in one of two settings: Renal impairment, or when a large magnesium load is given, such as in cases of eclampsia, over the counter laxatives, and enemas. [3]

In healthy individuals, plasma magnesium concentration ranges from 1.8-2.6 mg/dl. Approximately 70% (15% complexed and 55% free Mg²⁺ ions) of total body magnesium is filterable through artificial membranes and remaining 30% is protein bound. Normally, 97% is reabsorbed by the renal tubules: thus only 3% of filtered magnesium appears in the urine. The majority of filtered magnesium is reabsorbed in the thick ascending loop of Henle and the remaining is reabsorbed in the proximal tubules and distal tubules. The cellular magnesium transport mechanism is not well understood. [2]

Hypermagnesemia prevents presynaptic acetylcholine release by competitively inhibiting calcium influx into presynaptic nerve channels via the voltage dependent calcium channel resulting in blockage of neuromuscular transmission. [1] Therefore, neuromuscular conduction symptoms such as delayed deep tendon reflexes, facial paresthesia, muscle weakness, flaccid paralysis, and respiratory depression and apnea at higher magnesium levels can ensue. [1]

Magnesium is cardiotoxic and in moderate to high concentration, can cause hypotension, bradycardia, complete heart block, and cardiac arrest. Hypermagnesemia depresses the conducting system of the heart and sympathetic ganglia. [1]

Hypocalcaemia can develop due to hypermagnesemia because of reduction in parathyroid hormone (PTH) secretion or end-organ resistance to PTH. This will lead to paralytic ileus from smooth muscle paralysis. [1]

Hypermagnesemia interferes with blood clotting mechanisms and causes impairment in platelet adhesiveness, thrombin generation time and clotting time. [1]

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 [4]. Magnesium levels of 7.2 to 12mg/dl result in hypocalcaemia, absent deep tendon reflexes, hypotension and bradycardia. [5] Magnesium levels above 12mg/dl cause muscle paralysis, complete heart block, respiratory and cardiac arrest. Magnesium preparations and parenteral magnesium in renal insufficiency patients should be avoided. If renal function is normal, cessation of magnesium therapy promptly restores normal magnesium levels. However, patients with renal failure may require urgent dialysis. For rapid reversal of potentially life threatening side effects of hypermagnesemia, intravenous calcium gluconate can be given. [6] Calcium antagonizes the toxic effect of magnesium, and these ions electrically oppose each other at their sites of action. This treatment usually leads to prompt symptomatic improvement.

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