

Case Report

Clinically significant hypermagnesemia in a healthy individual: a rare entity

Smita Manjavkar^{1*}, Jayashree Kharaje¹, Aniruddha Malgaonkar²

¹Department of Medicine, ESI-PGIMSR, Andheri, Mumbai, Maharashtra, India

²Department of Community Medicine, Rajiv Gandhi Medical College, Kalwa, Thane, Maharashtra 400606, India

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*Correspondence:

Dr. Smita Manjavkar,

E-mail: smita.davari@gmail.com

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ABSTRACT

Clinically significant hypermagnesemia is rare in individuals with normal renal functions. Its cause is often iatrogenic. In this case a young married female had significant hypermagnesemia with normal renal function. The clinical spectrum of hypermagnesemia can range from subtle neurological signs to major life threatening shock, dysarrhythmias and coma. The patient was treated with intravenous hydration, Ca-gluconate and furosemide. Patient responded to the treatment and her serum magnesium levels decreased to 4.00mEq/l.

Keywords: Hypermagnesemia, Cathartics, Refractory hypotension

INTRODUCTION

Magnesium is a major intracellular divalent cation. It is the fourth most common cation in the body and the second most intracellular cation after potassium.¹⁻⁴ Only 1% of it is present in extracellular fluid and of this, one-third is protein bound with a normal serum level of 1.5 to 2.2 mg/dl.^{5,6} Magnesium is absorbed in proximal small intestine.⁷⁻⁹ It is a physiological calcium channel blocker and renal homeostasis maintains body serum magnesium levels. Hypermagnesemia is rare in individuals with normal renal function with its cause often iatrogenic. Patients with hypermagnesemia present with wide spectrum of neurological and cardiovascular manifestations with the hallmark being refractory hypotension and fatal cases of cardiac arrest being reported.¹⁰

CASE REPORT

16 years old young unmarried female presented with chief complaints of 5 to 6 episodes of vomiting associated with pain in abdomen, generalised weakness,

fatigue, headache followed by altered sensorium. On detail history, vomiting was non-bilious, non-projectile with no blood in vomitus. Pain in abdomen was predominantly in epigastric region, non-colicky, non-radiating with generalized tenderness over the entire abdomen. There was no history of fever, loose motions, jaundice, convulsions or any bleeding diathesis. The patient was not a known case of any major medical illness with no history of drug ingestion, suicide or homicide attempt.

On examination the patient presented with vital signs of 36.8°C temperature, a feeble pulse with a rate of 50/minute, systolic blood pressure of 60mm of Hg with dry tongue, low Jugular Venous Pulse, all peripheral pulses felt but feeble and cold and clammy extremities. Patient was pale but had no icterus, cyanosis or lymphadenopathy. No neuro-cutaneous markers or stigmata of tuberculosis were seen. Bone and spine was normal. Cardio-respiratory system was normal. Per abdominal examination revealed tenderness over entire abdomen with good bowel peristalsis. On Central Nervous System examination patient was in altered sensorium & irritable with grade II power in both limbs

with absent deep tendon reflexes and plantars were flexor bilaterally. Both pupils were equal, sluggishly reacting to light. There was no cranial nerve involvement and no neck stiffness.

Laboratory Investigations revealed a hemoglobin of 10gm%, a total leucocyte count of 14,100/cmm, platelet count of 207000/cmm. Malaria antigen and peripheral smear for malarial parasite was negative. Serum Sodium level was 140mEq/L, Serum Potassium- 4.1 mEq/L, Serum chloride- 101.5 mEq/L and Serum Calcium level was 9.8 mg%. Creatine phosphokinase level was 34 IU/L, ESR-15mm/hr, blood urea nitrogen was 34 mg% and serum creatinine level was 0.8mg%. SGOT recorded was 47 IU/L, SGPT-19 IU/L, serum bilirubin was 0.8mg%, serum lipase- 40U/L and serum amylase level was 52 IU/L.

Cerebrospinal fluid study was normal with adenosine deaminase at 48U/L. Random blood sugar level was 198mg%. Arterial blood gas analysis showed a pH of 7.4 with a $p\text{CO}_2$ - 27 mm of Hg, $p\text{O}_2$ - 127 mm of Hg, HCO_3 - 20.6 mmol/L, SpO_2 - 100%.

Electrocardiogram showed sino-atrial node block. A two dimensional echocardiograph showed an ejection fraction of 60%, good biventricular function, no regional wall motion abnormality and no evidence of pericardial effusion. Fundoscopy showed no evidence of papilloedema, X-ray chest PA view was normal, and MRI scan of the brain was normal.

Urine Toxicology screen (with A and B group toxins) did not reveal anything. Dengue and leptospira IgG and IgM antibodies were negative and so were viral markers HBsAg, anti- HCV and HIV were negative. T_3 level was 160mg/100ml, T_4 - 5.4 mcg/100ml and TSH level was 1.5 IU/ml. Serum uric acid level was 10mg/dl, Serum phosphate - 3.2mg/dl and Serum parathormone level was 28ng/l. Blood group of the patient was O Rh +ve.

Ultrasound of chest, abdomen and pelvis revealed bilateral pleural effusion and organised sludge in gall-bladder. Gestational sac was present and minimal free fluid in abdomen was seen. Serum cortisol level was 25mg/dl at 8.00 am.

Urine pregnancy test was positive. Sickling test was negative.

In the medical intensive care unit, patient was started on intravenous voluven (6% hydroxyethyl starch in 0.9% sodium chloride) injection, along with broad spectrum antibiotic injection Ceftriaxone sodium. Fluid challenge with IV fluids of Normal Saline and Dextrose Normal Saline was given and CVP (central venous pressure) was corrected. In spite of this the patient had hypotension.

Hence, patient was started on intravenous dopamine at 3mcg/kg/min. In addition to this intravenous

noradrenaline was given at the rate of 4mcg/kg/min with both the ionotropes up titrated to the maximum dose as per the patient's weight. Even then no urine output was seen after 24hrs and hypotension was persistent. Hence urgent serum magnesium level was done which was 16mg/dl.

Intravenous calcium gluconate was deferred initially with normal potassium and calcium but was subsequently started in view of hypermagnesemia. 10% Ca-gluconate in the dose of 100mg was given over 2hrs and followed by 2mg/kg/hr over 24hrs. Patient showed improvement in the form of increase in systolic blood pressure to 100mmHg. Intravenous lasix infusion was given at 2-3 mg/hr. Patient's sensorium improved gradually. Subsequently patient was conscious and oriented. Vitals signs were stable and the patient was hemodynamically stable.

DISCUSSION

Magnesium is the second most prevalent intracellular cation and it plays vital role in many cellular metabolic pathways. It is required for DNA, protein and parathyroid hormone synthesis and it is an essential co-factor for most enzymatic phosphorylation reactions. Normal serum magnesium concentration is 1.5 to 2.0 mEq/l. Of this 25-30% is protein bound, 10-15% is complexed, and rest 50-60% is ionized. Bone and skeletal muscle are major reservoirs of magnesium.

Approximately 30-40% of normal dietary magnesium is absorbed in the ileum and jejunum.^{9,11,12} 70-80% magnesium is filtered at the glomerulus and approximately 5-15% is reabsorbed in the proximal tubule, 60-70% in thick ascending Loop of Henle & 10-15% in distal convoluted tubule.

Kidney is the main regulator of magnesium concentration. Hypermagnesemia is a rare entity as kidney is very effective in excreting excess magnesium and hence hypermagnesemia is most commonly observed in renal insufficiency. Massive exogenous magnesium exposures through gastrointestinal tract can cause hypermagnesemia. Due to this even normal amount of magnesium containing cathartics retained in gastrointestinal tract for prolonged period in case of intestinal obstruction, ileus or perforation can cause hypermagnesemia.

Magnesium excess affects the central nervous system, neuromuscular and cardiovascular system. Apart from this various disease states like Addison's disease, familial hypocalcemic hypercalcemia, hyperparathyroidism, and tumour lysis syndrome may present with or result in hypermagnesemia. Lithium and theophylline intoxication causes hypermagnesemia.

Metabolic acidosis, hypocalciuria, hypokalemia, phosphate depletion, alcohol use and volume expansion

all produce an immediate increase in magnesium excretion in the urine. Degree of elevation of magnesium levels determines the symptoms. Sudden rise of magnesium levels are more symptomatic than slow rise in the levels.

Vasodilatation and neuromuscular blockade are most prominent at serum magnesium level of more than 4mEq/l. Nausea, lethargy and weakness may progress to respiratory failure, paralysis and hypoactive tendon reflexes and coma at serum magnesium level of more than 8mEq/l. In addition there is gastrointestinal hypomotility, ileus, facial flushing, papillary dilatation, paradoxical bradycardia and prolongation of PR, QRS & QT interval. Heart block and asystole occurs at serum magnesium level of more than 20mEq/l.

Majority of reported cases of hypermagnesemia have occurred in patients with impaired renal function or are iatrogenic in nature. Magnesium has been used as an anticonvulsant in pre-eclampsia and eclampsia, as antidysrhythmic, as an antacid, a laxative, an antihistaminic and as a cathartic to treat some poisonings.¹³ It has been recommended as routine replacement therapy in hypomagnesaemia states such as chronic alcoholism.

In this case an incidental finding of a gestational sac with raised β -HCG levels was present. So a possibility of use of some kind of abortifacient or alternative medicine or over the counter medication with higher magnesium content by the patient cannot be totally denied and this may be the reason for hypermagnesemia in this patient. Possibility of poisoning followed by use of cathartic cannot be ruled out. However toxicology report was inconclusive and this could be attributed to the significant time gap between poisoning and presentation with hypermagnesemia.

Magnesium is a natural physiological calcium channel blocker hence calcium can reverse this antagonistic action. Calcium is especially effective for hypotension, dysarrhythmias and respiratory distress and it also enhances the excretion of magnesium.^{12,14,15} Commonly used dose for the treatment of hypermagnesemia is 5-10mEq/l of calcium (CaCl_2) rapidly intravenous over 30 seconds and repeated as necessary every 5-10 minutes or 100-200mg of calcium over 1-2 hour as 10% calcium gluconate followed by 2-4mEq/kg/hr is given. Vigorous intravenous hydration should be attempted and intravenous furesomide can be given to accelerate magnesium elimination.^{16,17} Haemodialysis is effective and may be required in significant renal insufficiency.

CONCLUSION

This is a case of severe hypermagnesemia with normal renal function and refractory hypotension in young unmarried primigravida. All workup for cause was inconclusive but may be due to the use of cathartic

poisoning or magnesium containing abortifacient. Patient responded to intravenous calcium gluconate, furesomide and intravenous hydration and her serum magnesium levels decreased to 4.00mEq/l.

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