The town of Epsom lies only 20 miles from London, England. In the year 1618, a farmer in Epsom tried to give his cows water from a local well. The cows were thirsty, but they refused to drink the water because of the water’s bitter taste. Although his cows found the water distasteful, the farmer discovered that when he applied this water to his skin, rashes and scratches were quickly healed. Thus was the discovery of Epsom salts, which continue to be used medicinally to this day.1

We now know that the active ingredient in Epsom salts is magnesium sulphate, and we have learned that magnesium is an essential element for proper human cellular function. Hypomagnesemia has been recognized as a serious disorder that can affect nearly every organ system and lead to potentially life-threatening complications.2

A patient seen in our practice was found to have symptomatic hypomagnesemia.

**Case Report**

A 56-year-old white female patient was evaluated in our cardiology specialty office for recurrent symptoms of palpitations associated with supra-ventricular arrhythmias and hypomagnesemia. She had recently been evaluated on three separate occasions in the hospital emergency department (ED) for symptoms of palpitations and lightheadedness. At each hospital presentation, the patient was found to have an abnormal cardiac rhythm, with the electrocardiogram demonstrating supra-ventricular tachycardia leading to a resultant heart rate range of 160 to 180. While in the ED, the cardiac rhythm converted promptly to sinus rhythm on each occasion following the intravenous administration of Adenosine or Diltiazem.

Laboratory evaluation revealed normal serum electrolytes, renal function, complete blood count, ultrasensitive thyroid stimulating hormone level, ionized calcium and serial cardiac enzymes, including creatine kinase (CK-MB) and troponin-I levels. However, the patient was found to have a decreased serum magnesium level, ranging from 1.3 to 1.5 mg/dL (normal 1.7-2.7 mg/dL), and she was treated with oral or intravenous magnesium supplementation.

The patient’s history was significant for a cardiac evaluation 3 years earlier for symptoms of recurrent chest discomfort and electrocardiographic abnormalities during an exercise stress test. She subsequently underwent diagnostic cardiac catheterization, which demonstrated normal left ventricular function and no evidence of significant coronary disease. She had noted occasional symptoms of shortness of breath with activity, but no symptoms of dyspnea at rest, orthopnea or paroxysmal nocturnal dyspnea. The palpitations usually resolved spontaneously within 5 minutes, but had persisted on the three occasions that resulted in presentation to the ED.

The patient’s cardiac risk factors included a history of hypertension, type 2 diabetes, hypercholesterolemia and a family history of cardiac disease.

Her current cardiac medications included a calcium-blocking agent, Diltiazem, and...
an angiotensin converting enzyme inhibitor, Enalapril. She was also receiving an oral diabetes medication, Metformin, and Feno-fibrate for treatment of lipid abnormalities.

A magnesium supplement, Magnesium Oxide, had been recently added to her medication regimen following the ED evaluation and treatment.

On physical examination, the blood pressure was 128/72, the heart rate was 70 and the respiratory rate was 16. The ophthalmological and fundoscopic examination were normal. The lungs were clear to auscultation. On cardiac examination, there was a regular rhythm with a soft systolic murmur at the left sternal border. The murmur was unchanged from previous examinations. There was no abdominal bruit present. On extremity examination, peripheral pulses were present and there was no edema.

The electrocardiogram demonstrated sinus rhythm, left axis deviation and minor non-specific ST-T changes. A chest X-ray showed no acute abnormalities. An echocardiogram showed normal left ventricular function with an ejection fraction of 58 percent and mild valvular insufficiency. A stress test performed with digital echocardiography demonstrated no stress induced abnormal symptoms, arrhythmias or echocardiographic evidence of myocardial ischemia.

It was assessed that the episodes of supraventricular tachycardia were associated with the patient’s recurrent propensity for hypomagnesemia. She subsequently was referred for an extensive metabolic, gastrointestinal and renal evaluation to evaluate the cause of the recurrent hypomagnesemia. The metabolic evaluation was normal except for a mildly decreased vitamin D level, with a 25-hydroxy D total of 23 ng/mL (normal 25-80 ng/mL). The gastrointestinal evaluation to evaluate the cause of the recurrent hypomagnesemia was entirely satisfactory, including upper and lower endoscopic examinations. The renal evaluation, including urinary electrolyte and magnesium excretion, indicated that the hypomagnesemia was most likely a result of diabetic nephropathy, possibly exacerbated by the Vitamin D deficiency.

The patient was treated with oral magnesium and vitamin D supplementation as well as dietary recommendations for foods rich in magnesium and vitamin D. She has subsequently maintained serum magnesium and vitamin D levels that are within normal limits. She has recently had only rare episodes of palpitations, and no further episodes of sustained palpitations or documentation of supraventricular tachycardia.

**Discussion**

Magnesium plays a critical role in cellular biochemical function. It is an integral component of many metabolic enzyme reactions and is also essential for the maintenance of proper intracellular and vascular ionic balance. Hypomagnesemia is considered present when the serum magnesium concentration is measured as less than 1.7 mg/dL, with a normal range of 1.7 to 2.7 mg/dL.

In our patient, hypomagnesemia was associated with the onset of symptomatic supraventricular arrhythmias. Decreased serum magnesium levels have also been associated with other cardiac effects, including conduction abnormalities and ventricular arrhythmias. Torsade de pointes, or polymorphous ventricular tachycardia, has been associated with hypomagnesemia, and the American Heart Association now recommends magnesium sulfate be included in the acute treatment of this life-threatening arrhythmia.

In addition to cardiac abnormalities, hypomagnesemia is also known to have neuromuscular effects, including convulsions, muscle cramping and neuropsychiatric disturbances. Other organ systems known to be affected by hypomagnesemia include orthopedic abnormalities, with increased incidence of osteoporosis, pulmonary abnormalities with exacerbation of asthma, and renal effects, including development of nephrolithiasis.

In our patient, the etiology of the decreased magnesium level was thought to be diabetic nephropathy as well as possible deficiency of vitamin D. Other causes of hypomagnesemia include decreased oral intake of magnesium from poor nutrition or prolonged treatment with magnesium deficient intravenous fluids. Decreased intestinal absorption of magnesium may result from malabsorption syndromes, diarrhea or an intestinal fistula. Hypomagnesemia may also result from increased renal excretion of magnesium in patients receiving prolonged treatment with diuretics and in association with the diuretic phase of acute renal insufficiency. Other conditions associated with decreased serum magnesium include hypocalcemia, parathyroid abnormalities and pancreatitis. Several identified genetic abnormalities may also lead to a predisposition for hypomagnesemia.

Hypomagnesemia has been found to have a surprisingly high prevalence in patients in acute-care settings. Decreased serum magnesium levels have been reported in as many as 12 percent of patients admitted to a general hospital, and in as many as 65 percent of patients within an intensive care unit.

Magnesium levels are often not routinely obtained in hospitalized patients, and this has led to magnesium being termed the “forgotten cation.” Our case illustrates the importance of obtaining serum magnesium levels in hospitalized or ambulatory care patients whose symptoms are suggestive of possible hypomagnesemia.

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