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Hypomagnesemia-Induced Cardiac Arrhythmia†

Robert Chapman, MD* and Jan Rival, MD*

A case of life-threatening cardiac arrhythmia associated with magnesium deficiency is described. The patient had no history of any cardiac disease, was not taking any medication and had no other accompanying electrolyte disorder. His arrhythmia was refractory to conventional treatment but responded to parenteral magnesium therapy. The role of hypomagnesemia in the predisposition to cardiac arrhythmia is discussed. To our knowledge, there are only two well-documented cases of ventricular irritability which responded to magnesium as the sole treatment. As awareness of such a relationship increases and magnesium determinations become more available, this entity may be more commonly recognized, and the modality of therapy further substantiated.

This report describes a patient not receiving digitalis who had refractory ventricular arrhythmias secondary to magnesium deficiency. This disorder, in the absence of other contributing metabolic or physiologic factors, has rarely been reported (1). A possible cause of the problem is discussed, and the need for careful monitoring of serum magnesium is stressed.

Case Report

A 47-year-old black man, alcoholic, was brought to the Emergency Room after having passed out at home. He admitted drinking up to three pints of gin a day with very little food intake. Two days before he was admitted, he passed out while drinking heavily. He had vomited several times in the twelve hours before admission. He had a history of peptic ulcer disease but none of cardiac disease, and specifically denied symptoms of chest pain or congestive heart failure; he was taking no medications.

In the Emergency Room he had tremors and nystagmus but was alert and well oriented. Cardiac exam revealed frequent extrasystoles. Electrocardiogram showed ventricular bigeminy. He suddenly developed a generalized seizure and then went into ventricular fibrillation. After prompt and successful defibrillation he received a bolus of 75 mg of lidocaine followed by an infusion of 4 mg per minute of lidocaine. Over the next 36 hours he required three boluses of lidocaine. While still in the Emergency Room, his potassium was 3.5 mEq/l, his blood pressure was 120/80 mm Hg, and his pulse was 80 per minute.

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Fig. 1
Electrocardiogram showing sinus rhythm with ventricular bigeminy
When he was transferred to the Coronary Care Unit, monitoring revealed multiple premature ventricular contractions and frequent runs of ventricular bigeminy and trigeminy. The patient’s serum digoxin level was less than 0.3 (which in our lab cannot be distinguished from 0), his calcium was 8.6 mEq per liter, and his total protein was 7.5 mg%. His CPK and LDH isoenzymes were negative for increased cardiac fraction. He was restless and tremulous. Over 12 hours he received a total of 23 mg of Diazepam. Lidocaine failed to decrease the frequency of premature contractions.

During the second day he had delirium tremens and began receiving chlordiazepoxide in 50 mg I.V. push doses. Propranolol was started intravenously in doses of 0.5 to 5 mg for his refractory ventricular arrhythmia (which included one run of ventricular tachycardia), then increased to 20 mg every six hours; but his arrhythmia remained refractory.

On his fourth day the patient was transferred to a general medical floor. Electrocardiogram revealed that he was in ventricular bigeminy (Fig. 1). Because of the patient’s history of ethanol abuse and recent delirium tremens, a serum magnesium level was drawn; it was 0.8 mEq per liter (normal in our lab is 1.5-2.2 mEq). He was then given magnesium sulfate in 8 mEq intramuscularly every two hours times three. Within an hour after the last dose he reverted to sinus tachycardia without premature ventricular contractions; a repeat serum magnesium determination was 1.6 mEq per liter. He then received 48 mEq of magnesium sulfate over eight hours by I.V. infusion, and his rhythm reverted to normal sinus rhythm during that period. Propranolol was tapered off over the next five days, and his arrhythmia did not recur.

Fig. 2
Relation of ventricular arrhythmias to magnesium levels, magnesium administration, and other antiarrhythmics.
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Discussion
Magnesium deficiency is associated with several clinical situations. Diuretics such as furosemide, ethacrynic acid, and mercurials cause increased magnesium excretion in urine (2, 3). Alcoholics lose increased amounts of magnesium in urine, which persists briefly after ethanol withdrawal even in the face of severe hypomagnesemia. Glycosuria is associated with an increase in urinary magnesium, and if prolonged can result in marked magnesium depletion (2). Malabsorption, the diuretic phase of acute renal failure, hypoparathyroidism with hyperphosphatemia, primary aldosteronism, and acute pancreatitis have all been associated with hypomagnesemia (2).

Magnesium is a metallocoenzyme that is essential to activate adenosine triphosphatase (3, 5), which powers the transmembranous sodium-potassium pump and helps to maintain intracellular potassium concentrations. Depolarized cells, which depend on a rapid influx of potassium, might therefore be expected to be particularly sensitive to magnesium depletion. Since digitalis also inhibits the sodium-potassium pump, it is not surprising that hypomagnesemic patients are particularly prone to digitalis toxic rhythms (1).

Our patient became markedly hypokalemic immediately after magnesium replacement, even though he had no recurrent arrhythmia. This condition was probably not secondary to further magnesium depletion, but rather to intracellular mobilization of available potassium thus leading to hypokalemia. In this way, his body's total depletion of potassium was probably revealed; it may have been partially masked by his previous inability to maintain a normal transcellular potassium gradient.

Although hypomagnesemia-related cardiac arrhythmia in patients not receiving digitalis is rare (1), our patient demonstrates the effectiveness of magnesium therapy in an otherwise refractory arrhythmia. Closer monitoring of this often overlooked electrolyte may help to treat patients with refractory cardiac arrhythmias.

References