DETECTING HYPOMAGNESEMIA

THE MOST OVERLOOKED ELECTROLYTE IMBALANCE

Long ignored as a cause of potentially fatal arrhythmias, hypomagnesemia is now receiving careful attention. Find out what you need to know to uncover this problem in your patients.

DAVID WALLACE, A 73-YEAR-OLD RETIRED CONstruction worker, is admitted to your coronary care unit (CCU) with a diagnosis of congestive heart failure (CHF). Although he denies having chest pain, he's pale, diaphoretic, and dyspneic. You auscultate moist crackles in his lungs and note that he's producing frothy, pink-tinged sputum.

His vital signs are: pulse, 110; blood pressure, 100/64; respirations, 28; and temperature, 97.6° F (36.4° C). Cardiac monitoring shows sinus tachycardia, with four to five ventricular ectopic beats per minute.

This is Mr. Wallace's second episode of CHF since he suffered a myocardial infarction 4 years ago. He's remained otherwise healthy on a drug regimen of digoxin (Lanoxin), furosemide (Lasix), and a potassium chloride supplement (K-Lyte/Cl). Other than a borderline low hematocrit of 39%, his lab results are within normal limits.

So what's gone wrong? The answer isn't immediately obvious because Mr. Wallace is suffering from hypomagnesemia, an often overlooked and life-threatening electrolyte deficiency, which affects many more patients than you might suspect. Recent research suggests doctors and nurses should become more aware of this potential problem.

This article will tell you what you need to know – how to identify patients at risk for hypomagnesemia, what causes to consider, what signs and symptoms to look for, and how to ad-

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minister treatment. We'll also take you through Mr. Wallace's therapy and postdischarge planning.

Associated with hypokalemia

Studies show that 42% of hypokalemic patients also have hypomagnesemia—many of them experiencing ventricular ectopy, one of Mr. Wallace's symptoms. In these studies, ventricular arrhythmias persisted after hypokalemia was corrected and continued until hypomagnesemia was eliminated. The findings prove that magnesium is as important as potassium in the etiology and treatment of these cardiac problems.

That's because a magnesium deficit can lead to intracellular potassium loss and sodium accumulation, altering membrane excitability. This creates abnormal areas of automaticity, which contribute to inefficient conduction in the heart. Of course, any changes in the normal sequence of conduction usually result in symptomatic arrhythmias.

Diagnosing Mr. Wallace

In Mr. Wallace's case, as in so many others, no one recognizes his magnesium deficit initially. Shortly after his admission, you insert an intravenous (I.V.) line and infuse furosemide (80 mg) and digoxin (0.25 mg) as ordered. Because Mr. Wallace is experiencing ventricular ectopy, the doctor also orders 250 ml of D_5W with 40 mEq potassium chloride, infused at 25 ml/hour, hoping to prevent the ventricular irritability from getting worse.

Over the next 24 hours, Mr. Wallace improves, his respirations slowing to 16 per minute, his chest sounds clearing. But the cardiac monitor reveals occasional premature ventricular contractions, so he's transferred to the telemetry unit for continued electrocardiogram monitoring. On the unit, he's started on an oral regimen of digoxin, furosemide, and a potassium supplement—the same medications he'd been taking at home. He starts to feel energetic and is allowed to resume a full activity level.

But his progress doesn't last long. On the morning of the fourth day, he complains of vague discomfort and fatigue. A nurse on the telemetry unit notes that he seems nervous and uneasy. A few hours later, he develops ventricular tachycardia, with its characteristic wide QRS complexes, missing P waves, and a heart rate of 150 to 158 beats/minute. The nurse gives a stat order of lidocaine (50 mg I.V. bolus), followed by an infusion administered at 3 mg/minute. Mr. Wallace is transferred back to the CCU, where you're at a loss to explain what went wrong again.

Hypomagnesemia discovered

The reason isn't apparent until the cardiologist asks for stat serum magnesium and potassium levels. The results show a potassium level of 3.9 mEq/liter (normal is 3.8 to 5.5) and a magnesium level of 1.3 mEq/liter (normal is 1.5 to 2.5). He orders replacement therapy—5 grams of magnesium sulfate and potassium chloride, 15 mEq in 250 ml of D_5W, to be administered over 10 hours.

You use a controller to administer the infusion and monitor the infusion site carefully for signs of infiltration. You also check and document Mr. Wallace's heart rate and blood pressure hourly, aware that a drop in either vital sign could mean that excess magnesium is slowing conduction in the myocardium, compromising cardiac output. This is possible because hypermagnesemia, like hypomagnesemia, slows conduction in the myocardium. (See *Managing Complications of Magnesium Therapy.*)

Two hours after starting the infusion, you find Mr. Wallace's vital signs are stable. A rhythm strip reveals a normal sinus rhythm with an occasional but rare premature ventricular contraction.

Mr. Wallace returns to the telemetry unit after spending 24 hours on the CCU. His serum magnesium and potassium levels are checked daily. Discharge is planned within a few days because his electrolyte levels return to normal and his CHF resolves.

His diuretic is changed from furosemide to triamterene/hydrochlorothiazide (Maxzide), which spares potassium and magnesium. He'll no longer need to take potassium supplements but will

MANAGING COMPLICATIONS OF MAGNESIUM THERAPY

With the clinical significance of hypomagnesemia finally being recognized, you can expect to administer magnesium supplements more often to your patients. Here are some guidelines to help you detect complications during magnesium replacement therapy:

- Monitor the patient's vital signs, checking for an irregular or decreased heart rate, lowered blood pressure, and depressed respirations. Also watch ECG tracings closely, looking for peaked T waves and widened QRS complexes, both of which may indicate too *much* magnesium.
- Magnesium infusions can be toxic for patients with renal problems, so keep in mind that decreased cardiac output could signal a problem. Also monitor intake and output carefully before, during, and after the infusion.
- Assess the patient for impaired neuromuscular reactions, which may indicate an increased magnesium level. Look for muscle weakness, respiratory depression, and, ultimately, flaccid paralysis. Ongoing evaluation of deep tendon reflexes before and during magnesium infusions will help you identify any problem before it gets too severe.
- Keep calcium gluconate and calcium gluceptate on hand for rapid reversal of toxic effects. Also keep resuscitation equipment available. As you're watching for potentially toxic magnesium levels, remember that your patient's level may also remain normal despite continuous infusion. That's because serum levels are a poor indicator of intracellular magnesium levels, which fluctuate independently from serum levels.
- You may also be asked to administer I.M. magnesium, usually ordered as magnesium sulfate (20 grams in 2 ml). But some experts question the effectiveness of this therapy because many patients show no change in serum levels after injection. One reason may be that the injectable concentration of magnesium exceeds the renal tubular threshold. You can address this problem by getting an order to administer injections more frequently.
- Oral magnesium therapy poses problems too. Because of its laxative effect, magnesium hydroxide can exacerbate the deficiency through gastrointestinal loss. Magnesium chloride avoids this problem but is unpalatable. Timed-release tablets of magnesium chloride are available, but using them for continuous therapy is expensive.

need monthly serum electrolyte screenings to detect any future abnormalities.

Who's at risk?

Now that you're aware of the dangers of hypomagnesemia, what can you do to avoid it? Start by identifying patients at risk. First off, you should be suspicious whenever you see a case of hypokalemia or multiple electrolyte deficiencies (such as hyponatremia and hypocalcemia). Also be on guard when you administer diuretic therapy, a common cause of magnesium deficiencies. (Keep in mind, though, that a change of diuretics is not always advisable.)

Loop and thiazide diuretics are usually responsible. With the growth of the elderly population and a rise in associated cases of hypertension and coronary disease, more people take these diuretics. In addition to furosemide, other magnesium-depleting diuretics include ethacrynic acid (Edecrin), hydrochlorothiazide (Hydro-Diuril and Esidrix), and bumetanide (Bumex).

What to look for

You may be surprised to learn that neuromuscular irritability, the hallmark of calcium and potassium deficiencies, is also a sign of hypomagnesemia. Check for leg cramps, muscle spasms, twitching, and tetany. Also watch for central nervous system symptoms such as confusion and disorientation.

But the most life-threatening effects of a magnesium deficit are slowed cardiac rhythms and conduction time. Ventricular and atrial premature contractions may develop and progress to fatal arrhythmias. The electrocardiogram changes are similar to those seen in hypokalemia. A deficit can also lead to prolonged complexes and prolonged PR intervals. You might also see ST depressions and flattened T waves.

Dietary evaluation

You should evaluate the diets of patients who are at risk. What a patient eats can play a large role in preventing a deficiency. Recommend inexpensive and easily prepared meals that contain a lot of magnesium and potassium, including beans, corn, bananas, wholegrain breads, cereals, and green, leafy vegetables. Also suggest the use of herbs and other low-salt seasonings.

Recommended daily allowance standards call for consumption of 350 mg of magnesium for adult males and 280 mg for adult females. Keep in mind that only one-third of dietary magnesium is absorbed by the gastrointestinal tract.

You can't depend on diet alone to replenish magnesium when a patient has a malabsorption problem, the leading cause of hypomagnesemia. (There are other causes to consider too. For a complete overview, refer to *Causes of Hypomagnesemia.*)

In Mr. Wallace's case, you also need to teach him about his new diuretic. He was placed on triamterene/hydrochlorothiazide because it spares potassium and magnesium. You may see patients in similar circumstances receive such alternatives as Dyazide, which combines different concentrations of triamterene and hydrochlorothiazide, and Moduretic, a mixture of amiloride and hydrochlorothiazide.

You explain the reason for the change in diuretics to Mr. Wallace, emphasizing that he should take the new pill once a day. You also make sure he understands that this medication will replace – not supplement – his old regimen of furosemide and potassium.

Ongoing assessment needed

Once a patient has experienced this electrolyte imbalance, ongoing assessment is important. Frequent monitoring of serum magnesium and potassium levels should be done to ensure that they remain stable. With this in mind, you arrange a follow-up doctor's appointment for Mr. Wallace, scheduling it for 1 month after discharge so his magnesium and potassium levels can be checked. You also set up transportation for him and make sure he realizes the importance of the appointment.

Mr. Wallace is doing well, and his magnesium levels remain steady at 1.5 to 2.5 mg/dl. But if his deficiency hadn't been discovered and treated, it could have been fatal. You'll no doubt hear about other patients like him as hypomagnesemia attracts more attention. Routine testing for the condition will become widespread as more doctors and nurses recognize the need for it. Now you'll be prepared. N

CAUSES OF HYPOMAGNESEMIA

GASTROINTESTINAL
- Alcoholism
- Protein-calorie malnutrition
- Prolonged I.V. therapy (without magnesium replacement)
- Gastric suction
- Intestinal bypass for obesity
- Diarrhea
- Colonic neoplasms
- Laxative abuse
- Bulimarexia
- Short-bowel syndrome
- Malabsorption

RENAL
- Diuretics
- Antibiotics (ticarcillin, gentamicin, carbenicillin)
- Cisplatin
- Cyclosporine
- Hypercalcemic states (including malignancies)
- Postobstructive diuresis
- Acute tubular necrosis (diuretic phase)
- Hereditary renal magnesium wasting
- Aldosteronism

MISCELLANEOUS
- Excessive lactation
- Exchange transfusion
- Acute intermittent porphyria

SELECTED REFERENCES

Borris, M., and Papa, L.: "Magnesium: A Discussion of Its Role in the Treatment of Ventricular Dysrhythmia," *Critical Care Medicine.* 16(3):292-293, March 1988.

Martin, B., and Milligan, K.: "Diuretic-Associated Hypomagnesemia in the Elderly," *Archives of Internal Medicine.* 147(10):1768-1771, October 1987.

Ryzen, E., et al.: "Magnesium Deficiency in a Medical ICU Population," *Critical Care Medicine.* 13(1):19-21, January 1985.

Whang, R.: "Magnesium Deficiency: Pathogenesis, Prevalence, and Clinical Implications," *American Journal of Medicine.* 82(3A):24-29, March 20, 1987.