Original Research Reports

Hypomagnesemia in Patients with Eating Disorders

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One hundred seventy-five patients, admitted to the Eating Disorder Unit of Florida Hospital, Altamonte, were evaluated for electrolyte disorders, with particular reference to hypomagnesemia. Careful physical examination conducted by an internist, three separate histories, an eating-disorder questionnaire, psychometric testing, and the Beck and Zung scales for rating depression were used. The symptoms reported in the medical literature associated with hypomagnesemia were evaluated by the patients blind for the presence of this condition, using an analogue scale. Following treatment, improvement in symptoms was noted. One hundred eating-disorder patients with normal magnesium levels were used as controls. The study demonstrated an incidence of hypomagnesemia of 25%. When the hypomagnesemic patients were compared to controls and electrolyte balance, eight symptoms statistically defined the hypomagnesemic group. These included muscular weakness, cramping of the extremities, restlessness, parasthesias, diminished concentration, cardiac arrythmias, hypertension, and diminished recent memory. Magnesium replacement over several weeks was usually necessary to correct the imbalance when replacement was by the oral route. Hypomagnesemia is an important and often overlooked electrolyte abnormality that occurs in eating-disorder patients. Consequently, it should be routinely evaluated in eating-disorder patients on admission to hospital.

Considerable recent attention has been focused on the increasing prevalence and incidence of bulimia in college-aged and older women.[1-7] Although the disorder characteristically begins during adolescence, it may remain covert for many years and not be diagnosed until patients are in their late 30s or early 40s.[1] In spite of frequent visits to physicians for abdominal and cardiac complaints, as well as abnormal hematological and biochemical laboratory findings, bulimia may not be suspected as the cause of the patient's problem until its presence is called to the physician's attention by either the patient or a concerned relative or friend.[8-11]

The medical complaints most often associated with bulimia include weakness, abdominal cramps or pain, ulcer-like symptoms, finger and leg cramps, headache, nervousness, depression, dizziness, syncopal episodes, substernal pain, and dental problems. Forty percent of bulimic patients have menstrual irregularities

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or are amenorrheic.[1] Cardiac abnormalities are common at some point during the course of the illness, as are parotid enlargement and mental changes.[8,10–15]

Electrolyte abnormalities are frequently reported in bulimic patients, specifically the depletion of potassium, chloride, sodium, and calcium.[13,14,16–19] Few sources define hypomagnesemia as a significant problem requiring routine evaluation. While Mitchell and associates[20] reported hypomagnesemia as a complication of laxative abuse in eating-disorder patients, most papers did not mention magnesium as a clinically significant ion in bulimic patients.[18–23] A literature search for articles defining the incidence and complications of hypomagnesemia in bulimic patients failed to reveal any specific references in this category. Only one article, described below, reported a prospective study of magnesium levels in these patients.[11] Our report is the result of a two-year prospective investigation into the incidence, symptomatology, and management of hypomagnesemia in patients hospitalized as a result of a severe eating disorder.

METHODS

From October 1984 through August 1986, 175 patients were admitted to the eating-disorders unit at Florida Hospital, Orlando, Florida. A research consent was obtained that permitted us to analyze data obtained from laboratory and clinical evaluations, and a specific research protocol was approved by the departmental research committee and the institutional review board. All patients completed a detailed self-history and an eating-disorders questionnaire. Each was physically examined by an internist with specialized skills in the diagnosis and treatment of eating disorders. Each patient had three separate medical and psychiatric histories obtained by different investigators. Each patient also received a clinical evaluation consisting of CBC, SMAC-20, serum magnesium, urinalysis, urine toxicology, PA and lateral chest x-rays, electrocardiogram, psychological testing, and Beck and Zung depression-rating inventories. Specific inquiries were made concerning symptoms associated with electrolyte disorders. The nature, frequency, and pattern of the patients' anorectic, bulimic, or other eating disorder were carefully defined using objective criteria, as were their responses to treatment. Follow-up questionnaires were obtained six months to one year post discharge from the unit.

Electrolytes, including magnesium, were drawn immediately upon admission to the hospital. Results were generally available within 24 hours. Patients found to be hypomagnesemic were specifically questioned against a checklist of symptoms previously associated with hypomagnesemia in medical patients. The severity of each symptom was defined on an analogue scale of one to ten. Magnesium was then replaced either orally or, if symptoms were severe, by intramuscular injection. The analogue-symptom checklist was readministered, serum magnesium levels were monitored, and the duration of symptoms was noted as serum magnesium levels changed. Following replacement, patients were encouraged to report the redevelopment of symptoms previously experienced. If symptoms returned, serum magnesium levels were again obtained. The analogue-symptom checklist was also administered to 100 control patients having eating disorders whose magnesium and other electrolyte levels were within normal limits.

RESULTS

During the 22 months of this study, 42 of the 175 individuals admitted were found to have laboratory evidence of hypomagnesemia, defined as a serum magnesium level of 1.8 or below. Of these, 41 patients gave consent to be reported in the group data. Four patients had serum magnesium levels of 1.5 meq/l; seven of 1.6 meq/l; 18 of 1.7 meq/l; and 13 of 1.8 meq/l. Four of the patients with hypomagnesemia had restrictive anorexia nervosa, 19 had bulimia with purging, and two were diagnosed as having anorexia nervosa alternating with periods of bulimia with purging. Thirteen patients were admitted with exogenous obesity and depression, while two had unusual eating patterns that were subsequently found to be symptomatic of a

TABLE 1. Statistical comparison of eating-disorder patients with hypomagnesemia versus those with no electrolyte abnormality

Symptoms	Hypomagnesemia patients (total n=41)		Control patients (total n=100)		χ^2
Muscular weakness	30	(73%)	17	(17%)	38.797***
Restlessness	28	(68%)	21	(21%)	26.634***
Leg cramps	29	(70%)	27	(27%)	21.436***
Paresthesias	15	(36%)	6	(6%)	19.115***
Diminished concentration	35	(85%)	48	(48%)	15.259***
Cardiac arrythmias	8	(20%)	4	(4%)	7.104**
Hypertension	10	(24%)	7	(7%)	6.735**
Diminished memory	21	(44%)	28	(28%)	5.928*
Lability of mood	5	(12%)	16	(16%)	0.700
Irritability	24	(58%)	39	(39%)	3.735
Confusion	21	(44%)	35	(35%)	2.554
Anxiety	28	(68%)	62	(62%)	0.263
Depression	17	(41%)	36	(36%)	0.174
Fasciculations	6	(15%)	0	0	—
Seizures (grand mal)	2	(5%)	0	0	—

*$p<0.05$
**$p<0.01$
***$p<0.001$

thought disorder. One patient was admitted with psychogenic vomiting.

Two hypomagnesemic patients were chronic, severe, laxative abusers; one patient had gout; one had sarcoidosis; one had a gluten enteropathy. Three patients had severe irritable-bowel syndrome with profuse diarrhea. Six patients had diabetes mellitus; of these, five presented with obesity and depression, while one presented with bulimia with purging, the diabetes being unrecognized at the time of admission. Five patients chronically abused diuretics; three of these patients were hypokalemic with potassium levels of 1.8, 3.1, and 3.4 meq/l respectively (normal range 3.6 to 5.1 meq/l). The frequencies of symptoms associated with hypomagnesemia for both the study and control groups are shown in Table 1. The control population also had eating disorders but were known to have normal electrolyte (including magnesium) levels.

Fourteen patients were hypertensive at the time of admission. Of these, six showed a dramatic reduction in blood pressure immediately following the administration of intramuscular magnesium sulfate. One patient, a 14-year-old, morbidly obese, depressed, white female with type II diabetes mellitus, showed a decline of blood pressure from 150/90 to 100/80. The second, a 13-year-old, morbidly obese, depressed teenager, experienced a blood pressure change from 152/92 to 120/60 following intramuscular magnesium replacement. A 19-year-old, obese, depressed teenager who had been maintaining blood pressures in the range of 140/80 to 150/90 maintained a consistent blood pressure of 100/60 following magnesium replacement. A 44-year-old, chronically hypertensive, type II diabetic patient, whose blood pressure was maintained at 150/90 with antihypertensive medications prior to magnesium treatment, maintained a pressure of 136/70 without medication after magnesium replacement. Two other patients showed a con-

siderable lowering of blood pressure with magnesium replacement, with pressures changing from 150/90 to 130/70 and 168/94 to 138/80. The latter case was particularly interesting, as blood pressure rose to pretreatment levels (160/90) when serum magnesium levels again fell below normal range. When magnesium was readministered, blood pressure again reverted to the 136/80 range.

Contrary to our expectations, associated electrolyte abnormalities were relatively uncommon. Three patients were found to be hypokalemic while two were hyperkalemic. Four patients had hypocalcemia while one was hypercalcemic (the patient with sarcoidosis). Eight patients were hyperphosphatemic while one was hypophosphatemic. Nineteen patients evidenced low serum protein, while one had elevated serum protein levels. Six patients had diminished albumin levels.

Five patients were taking diuretics to help control hypertension. All also used a potassium supplement. None of these patients were hypokalemic. As previously noted, five additional patients abused diuretics; two of these patients were hypokalemic.

DISCUSSION

Hirschfelder[24] first described tremor and seizures as signs of hypomagnesemia in 1934. Since that time, considerable clinical and experimental work related to hypomagnesemia has appeared in the literature.[25–51] Magnesium follows calcium, sodium, and potassium as the body's fourth most abundant cation, and it is the second most plentiful intracellular cation. Only 1% of the body's magnesium stores are found in the extracellular compartment, and of these only 50% are available as a reservoir for depleted cellular stores.

Magnesium is distributed primarily as an intracellular ion, with highest concentrations being found in bone, liver, kidney, and brain. Serum magnesium concentrations consequently, like those of potassium, do not accurately reflect the intracellular concentration of the ion and symptomatic, total-body deficiency can occur in the presence of normal serum magnesium levels. Conversely, when serum magnesium levels are low, profound, total-body hypomagnesemia usually exists.

The magnesium content of the average American diet is approximately 250 mg/day, the richest dietary sources being cereals, nuts, and legumes. Drinking water is another important source. The present recommended daily allowance (RDA) for magnesium is 350 mg/day for men and 250 mg/day for women. Significant differences on a mg/kg basis exist, and balance studies suggest that ranges of optimal dietary intake vary from as little as 200 mg/day to as much as 700 mg/day. Thus there is potential for deficit in even a normal diet, much less that of the anorectic or bulimic. Hypomagnesemia may develop from any condition producing excessive gastrointestinal or renal losses or from inadequate dietary intake. It is commonly seen in patients with malabsorption syndrome, cancer, sprue, Crohn's disease, following gastric or enteric surgery, and in patients receiving prolonged parenteral nutrition. It also occurs in patients who chronically use or abuse laxatives and diuretics, in diabetics and alcoholics, and following the rehydration of dehydrated or starved patients.

Magnesium is principally absorbed in the small intestine, with approximately 30% of the ingested magnesium passing into the serum. Magnesium absorption is reduced in patients with chronic renal disease, perhaps because of chronic vitamin D deficiency. These effects are independent of calcium absorption, as calcium and magnesium have separate transport systems in the gut. Small concentrations of magnesium in the gut, however, do reduce calcium absorption by approximately 40%.

Magnesium is excreted primarily by the kidney, although the ion is actively reabsorbed by the nephron. As serum levels fall, renal reabsorption increases dramatically, with urinary losses reduced to less than 1 meq per day. Tubular reabsorption is diminished by extracellular fluid-volume expansion, alcohol intake, and high solute loads passing through the nephron, such as occurs in starving patients and those with diabetes. Renal magnesium loss is enhanced by

renal vasodilatation from any cause, diuretic use, hypercalcemia, elevated sodium intake (such as occurs in anorexic and bulimic patients who ingest large quantities of diet soda), thyroid hormone excess, digitalis administration, increased growth-hormone secretion, and the effects of high levels of mineralocorticoids. Conversely, increased levels of parathormone increase the tubular reabsorption of magnesium.

Loop diuretics are particularly potent in producing hypomagnesemia, as they inhibit reabsorption of the ion from the ascending portion of Henley's loop. Thiazide diuretics cause an immediate hypermagnesuria of a few days duration, which is followed by a period of renal compensation. Long-term thiazide diuretic use is therefore more likely to produce hypokalemia than hypomagnesemia.

During starvation, magnesium continues to be excreted in the urine. Muscle magnesium levels drop dramatically, while only small decreases are seen in serum magnesium levels until the total-body stores are seriously depleted. When starved patients are rehydrated by the administration of large quantities of fluids (particularly saline), a marked increase in urinary magnesium excretion, producing the acute onset of symptoms, can occur.[35] Several of our patients were asymptomatic until rehydrated.

Thirty percent of magnesium is bound to serum albumin. Serum stores are thus lowered in hypoalbuminemic patients. Moderate to severe hypomagnesemia tends to result in significant hypocalcemia. Magnesium deficiency impairs the release of parathyroid hormone and alters end-organ response to circulating parahormone.

Hypokalemia is frequently associated with magnesium deficiency, as the kidneys are unable to adequately reabsorb potassium in the face of low magnesium levels. Consequently, clinically refractory hypokalemia should suggest to the clinician the coexistence of a magnesium deficiency.[36]

Whang[36] has shown that the depletion of both potassium and magnesium plays a significant role in producing the ventricular tachyarrhythmias that occur in alcoholic patients, patients treated with diuretics, and patients with digitalis toxicity. Whang reported a 38% to 42% incidence of concurrent hypomagnesemia in hypokalemic patients. He concluded that magnesium as well as potassium should be replaced in all hypokalemic patients who suffer from alcoholism or who are receiving diuretics and/or digitalis because of the ventricular tachyarrhythmias that accompany these clinical states. He also believed that serum magnesium determinations would contribute significantly to the identification of patients at risk for cardiac arrhythmias due to magnesium depletion.

Whang and colleagues[37] conducted four studies to determine the frequency of hypomagnesemia in patients already found to have at least one abnormal electrolyte determination. They noted that hypomagnesemia occurred in 42% of patients with hypokalemia, 29% of patients with hypophosphatemia, 27% of patients with hyponatremia, and 22% of patients with hypocalcemia. They suggested that the detection of any of these electrolyte imbalances should alert the clinician to order a serum magnesium level because of their frequent association with hypomagnesemia. They argued that, optimally, serum magnesium levels should be obtained routinely because of the frequency of deficiencies found in hospitalized patients.

Many of the symptoms hypomagnesemic patients experience are produced by interference with magnesium's function as an essential cofactor in a variety of metabolic processes. Magnesium is necessary for the activation of alkaline phosphatase and the pyrophosphatases that are critical for energy storage and transfer. Low magnesium concentrations consequently affect a variety of cellular processes moderated by adenosinetriphosphate. Magnesium is an essential cofactor for oxidative phosphorylation and is necessary for the maintenance and integrity of ribosomes. It is crucial for protein synthesis and for the synthesis and degradation of DNA.[38]

Twenty-four percent (42/175) of the patients admitted to our eating-disorders unit had clinically significant hypomagnesemia, while the prevalence of clinical hypomagnesemia reported following the routine testing of hospitalized general medical patients ranges from 6% to

11%.[38] All of our patients were symptomatic, and symptoms improved following replacement of magnesium with magnesium gluconate or sulfate, as documented by return of serum magnesium levels to normal range. Symptoms recurred in several cases and were again associated with a diminished serum magnesium level. When levels were returned to normal, symptoms again abated. It should be noted, however, that other nonspecific treatments were begun that might also have influenced the patient's response to treatment. They included such events as the patients' decision to seek hospitalization, the initiation of specific individual, group, and family psychotherapy, the institution of a normal diet, and fluid and vitamin replacement.

To our knowledge, the only other prospective study dealing with the incidence of hypomagnesemia in eating-disorder patients is that of Jacobs and Schneider,[11] who reported on 24 of 39 consecutive bulimic patients who had magnesium levels drawn. Of these, five (21%) were hypomagnesemic. The patients in that study were similar in both age and frequency of purging to those in our sample, with the average frequency of purging being almost three times daily in the Jacobs and Schneider study and slightly more than three times daily in our population. They did not believe that the presence of hypomagnesemia warranted routine laboratory determination. Our study results suggest a different conclusion concerning hypomagnesemia. Clinical symptoms potentially attributable to hypomagnesemia were present in all of our patients with low serum magnesium levels and improved following magnesium replacement, as serum magnesium levels returned to normal.

The symptoms that responded to treatment included diminished concentration, muscular weakness, anxiety, restlessness, muscular cramps, and irritability and confusion associated with memory impairment and loss. As many of these symptoms are associated with primary psychiatric disease, their presence may not in and of themselves raise the possibility of hypomagnesemia. Routine laboratory surveillance of serum magnesium levels, however, will define the presence of hypomagnesemia in a high-risk population. Consequently, we suggest ordering magnesium levels routinely for all patients admitted to eating-disorder programs. Clinically, we were struck by the fact that 15 patients experienced significant parasthesias that cleared with the replacement of magnesium, while 10 patients were found to be hypertensive. Six of the 10 showed a significant decline of blood pressure following magnesium replacement.

Eight hypomagnesemic patients experienced cardiac arrhythmias that remitted following magnesium replacement. These included superventricular tachycardia, PVCs, coupled PVCs, PACs, bigeminy, atrial fibrillation, and multiple atrial and ventricular premature beats. Other ECG changes included widening of the QRS interval with peaked T-waves, prolongation of the P-R interval, and S-T segment depression. All of these changes have been reported in association with clinical hypomagnesemia.[29] Whang and associates[36] point out that these arrhythmias may precede the development of ventricular tachyarrhythmias.

Hollifield[39] notes that exercise-induced, premature ventricular contractions correlate significantly with hypokalemia and hypomagnesemia. He states further that the treatment of the hypokalemic-hypomagnesemic hypertensive patient with exercise-induced ventricular ectopy can be effected by the combined administration of potassium and magnesium, but not by either given alone. Hollifield cautions that potassium and magnesium repletion should be considered whenever potassium repletion is clinically indicated in patients with ventricular ectopy. As the presence of cardiac arrhythmias in anoretic and bulimic patients can be life threatening, this association requires careful note.

Isner and coworkers[41] have recommended routine electrocardiographic monitoring for anoretic patients with prolonged Q-T intervals, because ventricular tachyarrhythmias related to Q-T interval prolongation were seen in three of their patients who died suddenly. Both Thurston and Marks[42] and Pálossy and Oö[43] have pointed out the similarities between electrocardiographic findings noted in patients with anorexia nervosa

and those with intracranial injury. They have suggested that the hypothalamic abnormalities associated with anorexia nervosa may result in electrocardiographic changes mediated through the sympathetic nervous system as well as through direct effects on cardiac catecholamine levels. Magnesium may be involved in this sequence, as it is important in the physiological processes of several critical hypothalamic nuclei.

Other recent work has shown the specific effect of hypomagnesemia in the production of coronary-artery spasm and cardiac arrhythmia. Chadda[44] documented electrocardiographic evidence of acute myocardial ischemia secondary to coronary-artery spasm in a hypomagnesemic patient. Oral magnesium sulfate replacement successfully reversed the findings. Chadda suggested that magnesium therapy is indicated in patients who present with prolonged Q-T intervals, arrhythmias, and hypomagnesemia—a triad seen in eight of our patients.

Other authors have demonstrated a relationship between hypomagnesemia, coronary vasospasm, acute myocardial infarction, and sudden death syndromes.[45–48] Sheehan and Seelig[49] point out the importance of assaying serum magnesium levels, as deficiencies result in the intracellular loss of magnesium from the heart and arteries. This depletion contributes significantly to the propagation of cardiovascular damage resulting from functional rhythm abnormalities. These investigators have demonstrated that the heart, with its high metabolic activity, is particularly vulnerable to magnesium deficiency because magnesium is essential for the maintenance of cardiac mitochondrial structure and the normal enzymatic function of myocardial tissue. Hypomagnesemia also interferes with the sodium/potassium pump and the production of ATPase, necessary for energy transfer in the myocardium.

Recently magnesium has been shown to be necessary for the modulation of potassium proton (H+) exchange. The selectivity of this sodium/potassium H+ exchange is highly dependent upon serum magnesium concentration. Magnesium deficiency through this mechanism causes the loss of myocardial potassium and thus contributes to future electrophysiologic disturbance and cellular damage. In addition, a high calcium/magnesium ratio predisposes arterial spasm and increased cardiac catecholamine release. Sheehan and Seelig[49] caution that the replacement of potassium and calcium in patients with an undiagnosed magnesium deficiency is not only often unsuccessful, but carries with it an inherent risk for further intensification of the patient's magnesium depletion. Such replacement may precipitate further arterial contractility, ischemia, and arrhythmias. Should the patient go into heart failure, the administration of digitalis to a hypomagnesemic patient can produce both acute digitalis intoxication and further symptoms of hypomagnesemia. Thus, magnesium deficiency in patients with myocardial infarction increases the risk of both malignant ventricular arrhythmias and sudden cardiac death.

The Altruras[50] have shown that patients who experience an acute loss of potassium, magnesium, and glucose are likely to develop cerebral ischemia and stroke-like events. Stroke patients have been reported with low serum and cerebrospinal fluid magnesium concentrations. These investigators have also shown that experimentally induced magnesium and potassium deficiencies can produce cerebral vasospasm.

CONCLUSION

It is widely acknowledged that patients with eating disorders suffer from electrolyte abnormalities. We believe that the role of hypomagnesemia in these patients has been underestimated; in our study, as well as in the one other prospective study available, we found that overall incidence of hypomagnesemia approached 25%. Additionally, our hypomagnesemic patients were symptomatic, and hypomagnesemic symptoms cleared rapidly following adequate magnesium replacement.

A single-dose, intramuscular, magnesium-replacement regimen was inadequate to maintain normal serum levels over time. Replacement either intramuscularly or orally over several days to weeks was essential. Several days' therapy

with total replacement doses of 32 to 46 meq/l of magnesium per day worked well and is recommended. This can be accomplished through the administration of 500 mg tablets of magnesium gluconate or magnesium sulfate, or by the intramuscular injection of 2 cc to 3 cc of 50% magnesium sulfate deep im. Replacement can also be accomplished by giving the patient 5 ml of milk of magnesia or one milk of magnesia tablet daily, with an increase to four times daily as tolerated. We feel treatment with magnesium sulfate or gluconate tablets is preferable, as many of these patients are prone to laxative abuse.

The majority of our hypomagnesemic patients had normal serum potassium levels. Thus we believe that magnesium levels should be obtained routinely, independent of potassium levels, as a regular part of the admission workup for patients with eating disorders. Hypomagnesemia was common in eating-disorder patients taking diuretics and potassium supplements, particularly in patients using loop diuretics.

Replacement of magnesium may control hypertension in patients who have previously been inadequately controlled on standard antihypertensive medications, diets, or salt restriction. Further prospective studies are needed to define both the incidence and types of symptoms seen in eating-disorder patients with hypomagnesemia. The incidence of the condition in less severely ill outpatients and in medical and psychiatric controls also requires further definition. If our findings are confirmed, one might prophylactically treat anorectic patients having prolonged Q-T intervals and low or low-normal serum magnesium levels with magnesium sulfate and thus reduce or abort the development of the sudden and severe arrhythmias thought to produce sudden cardiac death in these patients.

When hypomagnesemic patients were compared to controls for electrolyte balance, eight symptoms statistically defined the hypomagnesemic group: muscular weakness, leg cramps, restlessness, paresthesias, diminished concentration, cardiac arrhythmias, hypertension, and diminished recent memory.

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