

# **Magnesium Deficiency In Patients With Chemical Sensitivity**

William J. Rea, MD, FACS \* \*

Alfred R. Johnson, DO\*\*

Ralph E. Smiley, MD\*\*

Barbara Maynard, RD\*\*

Ollie Dawkins-Brown, MEd, MS\*\*

## Abstract

Assessment of magnesium status in 51 consecutive chemically sensitive patients (12 males, 39 females, ages ranged from 13 to 67 years) in an environmentally controlled hospital unit was undertaken. The purpose of this study was to evaluate the magnesium status in chemically sensitive patients whose disorders were closely associated with the symptoms of magnesium deficiency. The symptoms included back and neck pain, fine tremors, muscle spasm, anxiety and nervousness, spastic vascular phenomena, ventricular arrhythmia, and fatigue. Results of laboratory tests and IV magnesium challenges showed that some patients with chemical sensitivities may have a disturbance of intracellular magnesium. RBC and plasma levels proved to be poor indicators of magnesium status: 2.8% (1/35 positive) and 15% (5/33 positive) respectively. Intravenous magnesium challenge appeared to be a more accurate assessment of total body magnesium status (76% positive 39/51). Overall clinical improvement with magnesium treatment appears to be somewhat low, (45% or 23 of 51) but it is worthy of consideration in those chemically sensitive patients who are magnesium deficient.

Keywords: Magnesium deficiency, chemical sensitivity, intravenous magnesium challenge.

## Introduction

A group of chemically sensitive patients exists who exhibit many of the signs and symptoms similar to those seen in magnesium deficient patients. The following case illustrates this point.

## Case Study

A 55-year-old female treated for chemical sensitivity for 8 years developed low back pain after a fall. Medication did not relieve her pain which had persisted for two years. Her orthopedist thought that she was not absorbing sufficient amounts of magnesium. Challenge with magnesium sulfate relieved her symptoms and yielded the following results:

---

1 Serum Mg 1.6 MEq 1.4-2.5 MEq

RBC Mg 34 39.0-49.0 ppm

Mg So<sub>4</sub> IV 20 MEq

Urine retention: 100%

Symptoms relieved

12-12-1985

Mild symptoms returned

12-19-1985

Mg So<sub>4</sub> IV 20 MEq

Urine retention:

not done

Symptoms relieved

2-15-1986

Mild symptoms returned

Mg So<sub>4</sub> IV 20 MEq

Urine retention:

not done

Symptoms relieved

---

Laboratory measurements of immune parameters, vitamins and mineral levels, and presence of heavy metals yielded the following results:

IgE (25 u/ml), IgG (830 mg/dl), IgA (102 mg), IgM (109 mg/dl), and eosinophil (123 cu/mm) were found to be within normal limits. Serum levels of calcium, sodium, potassium, copper, zinc and manganese as well as chromium, phosphorus, selenium, sulfur and barium were normal. Silicon was found to be slightly low at 1.0 ppm (normal = 1.1- 1.7 ppm). All heavy metals were found to be below the element detection limit. A

Chlorinated Pesticide Screening Test, showed low levels (less than 1 ppm) of DDT, Heptaclor and Heptachlorepoide to be present in the blood serum.

Reported symptoms of magnesium deficiency include coarse twitching, hypertonicity, carpal pedal spasm, generalized convulsions, tetany, weakness, confusion, personality changes, nausea, anorexia, lack of coordination, GI disorders, alopecia, swollen gums, skin lesions, lesions of small arteries, high blood pressure, strokes, drowsiness and ventricular arrhythmia. The similarity of symptoms indicated in these seemingly disparate entities led to the thesis that some patients with chemical sensitivity may have a disturbance in their intracellular magnesium. Magnesium assessment was undertaken in a rigorous effort to find relief for this group of patients.

## Materials and Method

Fifty-one consecutive chemically sensitive patients suspected of having symptoms of magnesium deficiency were studied. There were 12 males and 39 females whose ages ranged from 13 to 67 years. The mean age was 27 years, the median was 42 years and the mode was 32 years.

These patients were deemed too ill to be diagnosed and treated on an outpatient basis by the usual criteria. Patients were admitted to an environmentally controlled hospital unit which was designed to minimize patient exposure to the environmental factors suspected of triggering adverse reactions. A baseline for the purpose of measurement was obtained in the following way. Pre- and post-challenge signs and symptoms scores were noted. Forty-seven patients were maintained on their usual diets while four were fasted. Twenty-four hour food intake was recorded for each patient. Efforts were made to obtain as stable a magnesium intake baseline as was possible. Serum and red blood cell magnesium levels were measured by the atomic absorption mass spectrometry method<sup>1</sup> before challenge. Twenty-four hour urine analysis was undertaken for calcium, magnesium and creatinine. Analysis of urine for magnesium content was also by the atomic absorption mass spectrometry method.<sup>1</sup> Each patient was challenged intravenously with either magnesium chloride or magnesium sulfate (.2 meq/kg of body wt.) over a 4 hour period. A second urine collection began at challenge and continued over a 24 hour period. A percentage of magnesium excretion was calculated for each patient in the following manner. The challenged amount of magnesium was added to the amount of baseline excretion and divided into the challenge excretion.

The figure was divided into the challenge excretion. This figure was then multiplied by 100.

### Challenge Excretion

% of excretion =  $\frac{\text{Challenge Excretion}}{\text{Baseline excretion plus challenge dose}} \times 100$

Baseline excretion plus challenge dose

Magnesium deficiency was defined as less than 80% excretion of the amount of challenged magnesium.<sup>2</sup> Pre- and post-magnesium challenge signs and symptoms were recorded. Pre- and post-magnesium challenge urine excretion values were

compared and pre- and post-magnesium challenge serum and RBC levels were compared. Urine excretion values were also compared to serum RBC values.

## Results

Symptoms and Signs Relieved. Forty-eight percent or 23 of the 47 non-fasted patients showed immediate improvement.

Table 1

### Changes in Signs and Symptoms

---

Changes in Signs Number of  
& Symptoms Changes\*

---

Reduced dizziness 1

Improvement in depression 2

Reduced itching 1

Decreased tension 6

Reduced pain 7

Stronger, more energy 6

Decreased anxiety 3

Improvement in sleep 3

No change 17 (14 non-fasted,  
3 fasted)

Reaction to magnesium 8+ (7 non-fasted,  
1 fasted)

1) extreme weakness

2) felt drugged,

unable to move

### 3) metallic taste, heavy heart

\* more than one symptom was relieved in some patients.

+ immediate severe reactions in 4 patients required cessation of magnesium challenge.

Symptoms relieved included back and neck pain, fine tremors, muscle spasm, anxiety and nervousness, vascular spasms, ventricular arrhythmia and fatigue. Adverse reactions to chemical exposures were decreased. No fasted patients showed immediate relief.

Excretion of Challenged Magnesium. Less than 40% excretion of challenged magnesium was found in 11% or 5 of 47 non-fasted patients. Less than 60% excretion was found in 25% or 12 of 47 non-fasted patients. Excretion of 60-80% was found in 42% or 20 of the 47 non-fasted patients. Four were unable to tolerate the magnesium. Seventy-nine percent or 37 of the 47 non-fasted patients showed below 80% excretion of magnesium which is suggestive of magnesium deficiency. Less than 10% excretion of magnesium was found in 50% or 2 of 4 fasted patients. Less than 20% excretion was found in 25% or 1 of 4 fasted patients. Less than 60% excretion was found in 25% or 1 of 4 fasted patients. Four patients were unable to tolerate the magnesium challenge due to immediate severe reactions and the administration was stopped.

Table 2

#### Results of Magnesium Evaluation in 51 Chemically Sensitive Patients

##### Number of Magnesium Control

##### Patients Levels

1 Patient Serum 2 S.D. below control  $2 \pm .6$  Meq

5/33 (15%) R.B.C. 2 S.D. below control  $44 \pm 5$  ppm

37/47 (79%) Mg excretion below 20%

4/51 (7.8%) No Mg excretion level measured\*

20/47 (42%) Mg excretion between 60-80%

12/47 (26%) Mg excretion between 40-60%

5/47 (11%) Mg excretion below 40%

\*4 patients were unable to tolerate the magnesium challenge due to immediate severe reactions. The challenge was terminated and no excretion measurement was obtained.

Serum and RBC Magnesium. Serum magnesium was found to be below the normal range of 1.4-2.5 meq in 2.8% or in 1 of 35 patients. Red blood cell magnesium levels were found to be below the normal range of 39-49 ppm in 15% or in 5 of 33 patients.

## Discussion

Magnesium deficiency is probably more wide spread in the general population than originally suspected. This appears to be primarily due to poor dietary intake.<sup>2, 4</sup> Increased phosphate also contributes to magnesium deficiency states.<sup>5</sup> Segments of the population are at risk due to the large consumption of high phosphate containing soft drinks. Others who have poor protein or high fat<sup>6</sup> metabolism tend to be more likely afflicted with magnesium deficiency. Some disease states such as advancing age<sup>7</sup> diabetes mellitus,<sup>8, 9</sup> alcoholism,<sup>10</sup> heart failure,<sup>11, 12</sup> or use of diuretics,<sup>13, 14</sup> may cause magnesium depletion. Hypoxia and drugs such as gentamycin, cyclosporin and angiotensin<sup>15-17</sup> can also deplete the body of magnesium. Excess vitamin C may effect a drop in magnesium levels.<sup>18, 19</sup> Malabsorption of magnesium has been associated with gastrointestinal problems such as functional bowel disorder,<sup>20</sup> ulcerative colitis,<sup>20</sup> and Crohn's disease.<sup>21, 22</sup> Acute magnesium deficiency can occur after epinephrine, cold stress, and stress of serious injury or extensive surgery.<sup>23</sup> The average daily amount of magnesium intake is around 120 mg/100 calories in the adult U.S. population.<sup>24</sup> The need may be increased with any of the aforementioned conditions.

Many patients with chemical sensitivity have poor dietary intake, poor protein metabolism, or fall within the aforementioned conditions. Recent research has suggested that certain chemicals tiny alter the body's magnesium or its catalyzed products. For example, ethanol and carbon tetrachloride<sup>24</sup> have been shown to disturb liver collagen in rats. Exposures to some pesticides can disturb muscle and nerve physiology with muscular spasm and tetany occurring.<sup>26, 27</sup> Most chlorinated hydrocarbons are also lipophilic. Exposures can lead to disturbances in membrane stability. Magnesium plays an integral role in membrane stability. It is also a co-factor in many metabolic reactions.<sup>28, 29</sup> Additionally, it has been shown to counteract the diuretic inducer of catecholamine.<sup>30</sup> Consideration of the above facts suggests that some chemically sensitive patients may be magnesium deficient.

Some interesting facts came to the forefront as we further considered this group of patients. It appeared that serum and red blood cell measurements of magnesium may not reflect a true state of magnesium depletion unless it is extremely low. Further, if serum levels fell within the "normal" range, a depleted state may still be a possibility. This was in agreement with the World Conference on Magnesium (1985) consensus that the best way to define deficiency was by magnesium challenge.<sup>31</sup>

We found a great disparity between alleviation of signs and symptoms control (45%) and calculated magnesium deficiency (76%). There may be several reasons for this disparity: 1) The patient's symptoms may have been caused by other factors; 2) In some cases, the magnesium deficiency may not have been severe enough in light of biochemical individuality to produce symptoms originally; 3) The patient may not have been treated with sufficient amount of magnesium to relieve the symptoms; 4) Lower levels of magnesium retention may not reflect a true magnesium deficiency based on current thinking of what normal values of retention are; and 5) The lack of a precise calculated value for dietary intake may be a further complication although assessment of oral intake would have tended to bias the study toward higher levels of magnesium excretion.

The results of this study suggest that there possibly be some degree of magnesium deficiency in people who are chemically sensitive and that for enhancement of their treatment, attention should be paid to their magnesium intake.

## References

1. Tietz NW. *Fundamentals of Clinical Chemistry*. Philadelphia: WB Saunders, 1976.
2. Jones JE, Manalo R, Flink EB. Magnesium requirements in adults. *Med J Clin Nutr*, 20:632-35, 1967.
3. Laseter JL. Chlorinated hydrocarbon pesticides in environmentally sensitive patients. *Clin Ecol*, 2:3-1 2, 1983.
4. Marier JR and Neri LC. Quantifying the role of magnesium in the interrelationship between human mortality/morbidity and water hardness. *Magnesium*, 4:53-9, 1985.
5. Franz KB. Magnesium intake during pregnancy. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 27 in *J Am Coll Nutr*, 4:319, 1985.
6. Review. Hypomagnesemia in protein-caloric malnutrition. *Nutr Rev*, 29:89-90, 1971.
7. Mountokalakis T. Effects of Aging, chronic disease and multiple supplements on magnesium requirements. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 40 in *J Am Coll Nutr*, 4:326, 1985.
8. Rapado A, Herrera JL, Piedra C, et al. Corporal magnesium deficiency in diabetes mellitus after parenteral magnesium loading. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 143 in *J Am Coll Nutr*, 4:373, 1985.
9. Ewald U, Gebre-Meahin M, Tuvemo T. Hypomagnesemia in diabetic children. *Acta Paediatr Scand*, 72:367-71, 1983.
10. Cohen L, Laior A, Kitzes R. Lymphocyte and bone magnesium in alcohol-associated osteoporosis. *Magnesium*, 4:148-52, 1985.
11. Bloom B. Cardiomyopathy of magnesium deficiency and ischemia. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 19 in *J Am Coll Nutr*, 4:314, 1985.
12. Whang R. The need for routine serum magnesium: Clinical observations. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 49 in *J Am Coll Nutr*, 4:330, 1985.

13. Ryan P. Magnesium and potassium-sparing diuretics. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 33 in *J Am Coll Nutr*, 4:322, 1985.
14. Kuller L, Farrier N, Caggiula, et al. Relationship of diuretic therapy and serum magnesium levels among participants in the Multiple Risk Factor Intervention Trial. *Am J Epidemiol*, 122:1045-59, 1985.
15. Finton CK, Bjorkland S, Zaloga GP, et al. Gentamicin-induced hypomagnesemia. *Am Surg*, 49:576-8, 1983.
16. Zumkley H, Loose H, Spieker C, Zidek. Effects of drugs on magnesium requirements. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 39 in *J Am Coll Nutr*, 4:325-26, 1985.
17. Quamme GA. Renal Handling of Magnesium: Drug Interactions. Paper presented at the Fourth International Symposium on Magnesium. *J Am Coll Nutr*, 4:322, 1985.
18. Hsu JM, Smith JC, Yunice AA, et al. Impairment of ascorbic acid synthesis in liver extracts of magnesium deficient rats. *J Nutr*, 113:2041-7, 1983.
19. Kassouny ME, Coen CH and Bebok ST. The influence of vitamin C and magnesium on calcium, magnesium, and copper contents of guinea pig. *Intern J Vitamin Nutr Research*, 55:295-300, 1985.
20. Dumitrascu D, Lencu M, Stanciu L, et al. Hypomagnesemia and functional digestive disorders. Paper presented at the Fourth International Symposium on Magnesium. Abstract number 199 in *J Am Coll Nutr*, 4:394, 1985.
21. Dyckner T, Nyhlin H, Ek B, et al. Aggravation of thiamine deficiency by magnesium depletion: A case report. *Acta Med Scand*, 218:129-31, 1985.
22. Main AN, Morgan RJ, Russell RI, et al. Magnesium deficiency in chronic inflammatory bowel disease and requirements during intravenous nutrition. *J Parenteral Nutr*, 5:15-9, 1985.
23. Flink EB. Magnesium deficiency. Etiology and clinical spectrum. *Acta Med Scand* (Suppl), 647:125-37, 1981.
24. Ammerman CB. Magnesium requirements of animals and man. Paper presented at the Fourth International Symposium on Man. Abstract number 20 in *J Am Coll Nutr*, 4:325, 1985.
25. Rayssiguier Y, Chevalier F, Bonnet M, et al. Influence of magnesium deficiency on liver collagen after carbon tetrachloride or ethanol administration to rats. *J Nutr*, 115:1656-62, 1985.
26. James MF and Wright GA. Tetany and myocardial arrhythmia due to hypomagnesaemia: A case report. *South African Med J*, 69:48-9, 1985.

27. Durlach J, Duirlach V, Poenaru S, et al. Physiologic tracings and ionic evaluation of latent tetany due to magnesium deficit. Paper presented at the Fourth International Symposium on Magnesium. Abstract 54 in *J Am Coll Nutr*, 4:33, 1985.

28. Wacker WEC and Parisi AF. Magnesium metabolism. *N Engl J Med*, 278:658-63, 712-17, 727-76, 1968.

29. Rayssiguier Y, Gueux E, Weiser D. Effect of magnesium deficiency on lipid metabolism in rats fed a high carbohydrate diet. *J Nutr*, 111: 1876-83, 1981.

30. Dhalla NS. Role of Sarcolemmal  $Ca^{2+}/Mg^{2+}$  + ATPase in Health and Disease. Paper presented at the Fourth International Symposium on Magnesium. Abstract 21 in *J Am Coll Nutr*, 4:33, 1985.

31. American College of Nutrition Twenty-sixth Annual Meeting and the Fourth International Symposium on Magnesium. *J Am Coll Nutr*, 4:303-405, 1985.

Source: <http://www.aehf.com/articles/A27.htm>

---

This PDF file is provided here as a public service under a banner of "Fair Use" by:

[www.mcs-international.org](http://www.mcs-international.org)

Bringing the hidden dangers  
of modern synthetic chemicals  
out into the Light worldwide.

Contact: [webmaster@mcs-international.org](mailto:webmaster@mcs-international.org)

---