Magnesium and Hypertension

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Zusammenfassung

Epidemiologische Studien und der Magnesium-Metabolismus beim Bluthochdruck im Menschen zeigen eine direkte positive Korrelation zwischen Magnesiummangel und Bluthochdruck. Trotzdem ist die Hochdruckbehandlung mit Magnesiumsalzen unsicher.

Summary

Epidemiological studies and magnesium metabolism in human hypertension demonstrate a direct positive correlation between magnesium deficiency and hypertension. Yet, treatment of hypertension with magnesium salts is equivocal.

Résumé

Des études épidémiologiques et le métabolisme du magnésium dans l'hypertension humaine démontrent une corrélation positive entre le déficit magnésique et l'hypertension. Cependant le traitement de l'hypertension par les sels de Mg suscite des doutes.

Epidemiological studies

In Newfoundland, Finland and South Carolina there appears to be a strong association between low dietary Mg intake, low soil Mg, low water Mg and high incidence of blood pressure elevation in human subjects living in these diverse geographical regions [2].

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In Texas the drinking waters are the most heavily mineralized in the U.S.A. but differ in composition. These waters contain from 285 to 750 mg/l sodium and several of them contain from 30 to 100 mg/l Mg. In the studies conducted by *Dawson* et al. [6] in Texas, they included consideration of various ratios in their epidemiological assessment of drinking water or urine composition. They found that water-borne Mg was inversely correlated with hypertension. The water-borne Na/Mg ratio was directly correlated with hypertension. A similar pattern was seen in relation to urinary Mg and urinary Na/Mg ratio.

Kesteloot [9] found that the 24-hour urinary Mg excretion significantly and negatively correlated with diastolic blood pressure both in Belgian men and women. *Levin* et al. [10] studied the influence of drinking water hardness and blood pressure in USSR populations. The strongest correlations were with water-borne Mg. There was a 3-fold increased prevalence of high arterial blood pressure at the lowest water-borne Mg level (2.4 mg/l) in comparison with the control locality (26.2 mg/l). Thus moderate increments of water-borne Mg can have a noticeable effect on blood pressure among long-term residents.

McCarron [11] in a survey of Mg intake and hypertension found a difference in Mg consumption (normotensive, 261 ± 116 mg; hypertensive 206 ± 60 mg; mean $\pm$ SD).

Mg metabolism in hypertension

Albert et al. [1] found the mean serum Mg concentration in 26 uncomplicated hypertensive patients, to be significantly lower than in normal individuals. *Smith* at al. [18] observed hypertension in 9 out of 18 hypomagnesemic patients, known to be normotensive previously. *Petersen* et al. [12] found a significant inverse correlation between serum Mg level and mean blood pressure.

Silver et al. [17] studied the Mg turnover in 9 hypertensive patients. According to the values of exchangeable Mg found, the group could be subdivided into two groups, suggesting that a biological invariant was being measured. *Bauer* et al. [3] found a significant decrease in the 24-hour exchangeable Mg in 17 male patients with essential hypertension. The hypertensive men had also a lower serum Mg level compared to control subjects.

Resnick et al. [13] found higher serum Mg levels in patients with low-renin hypertension than in those with normal-renin hypertension or in normotensive controls. Untreated hypertensive individuals consistently demonstrated lower level of erythrocyte free Mg than either normotensive

or hypertensive subjects whose blood pressure had been normalized on therapy [4].

A strong relationship was found to exist between intracellular free Mg and diastolic and systolic blood pressure. A significant relationship was also observed between intracellular free Mg levels and serum concentration of total Mg. Insufficient numbers of subjects within differing renin subgroups have been evaluated to establish a relationship with intracellular free Mg [14].

Treatment of Hypertension with Mg salts

Dyckner and *Wester* [7] gave Mg supplementation as aspartate hydrochloride (15 mmol/day) for 6 months for arterial hypertension to 18 patients receiving long-term thiazide treatment. Both systolic and diastolic pressures decreased significantly by a mean of 12/8 mmHg. *Reyes* et al. [16] found that early supplementation with Mg chloride (15.78 mmol/day) during hydrochlorothiazide monotherapy of essential hypertension resulted in a potentiation of the antihypertensive effect of the diuretic. *Resnick* et al. [15] demonstrated that short-term Mg therapy in essential hypertensive subjects preferentially lowered blood pressure in patients with higher levels of plasma renin activity. *Whang* et al. [19] found that hypertensive patients with hypomagnesemia required more drug treatment to control blood pressure. Mg deficiency may therefore induce resistance to the effects of antihypertensive agents.

The effect of a new table salt with a composition of 65 % NaCl 25 % KCl and 10 % $MgSO_4 \times 7\ H_2O$ was introduced in Northern Karelia in Finland [8] and in Japan [20]. This new salt was well accepted by the consumers and was found to be safe. The studies revealed that use of the new salt resulted in a significant reduction of dietary sodium, a lowering of the Na/K ratio, and an increase in Mg. In addition, a significant reduction in the level of the blood pressure was noted in the hypertensive individuals. Average systolic blood pressure was lowered. The fall in diastolic pressure, although statistically significant, was very small.

Cohen et al. [5] found no effect of Mg on high-renin essential hypertension (750 mg Mg-oxide/day). *Cappuccio* et al. [4] also found no effect of Mg on high blood pressure (15 mmol Mg aspartate hydrochloride/day).

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