Serum Magnesium Concentration and PTH Levels. Is Long-Term Chronic Hypermagnesemia a Risk Factor for Adynamic Bone Disease?

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The observation that some subjects with low PTH had elevated plasma magnesium (Mg) levels prompted us to analyze in 41 patients on maintenance hemodialysis for 44 ± 36 months, their serum Mg concentrations, and the relationship between plasma Mg and PTH levels. The mean serum Mg concentration was 2.4 ± 0.2 mg/dl. Twenty-four out of the 41 subjects (58.5%) had hypermagnesemia (serum Mg above 2.5 mg/dl). Patients were classified into 3 groups according to their PTH level: Group A, low PTH (below 120 pg/ml); group B, adequate PTH (120–250 pg/ml); and group C, high PTH (above 250 pg/ml). There were no differences among groups according to number of subjects, age, sex, time on dialysis, renal disease, serum calcium, phosphorus, bicarbonate, vitamin D or aluminum concentrations. Doses of calcium carbonate and aluminum hydroxide were also similar in all groups. Curiously, although the differences were not statistically significant, the total cumulative intake of calcium and aluminum were less in group A than in the other groups. Interestingly, patients with low PTH had a significantly higher serum Mg concentration than patients with adequate or high PTH (2.8 ± 0.2 mg/dl vs 2.3 ± 0.1 mg/dl and 2.2 ± 0.1 mg/dl, respectively, $p < 0.01$). Moreover, regression analysis showed a negative linear correlation between serum PTH level and plasma Mg concentration ($r = -0.6059$, $p < 0.001$). Based on these findings, chronic hypermagnesemia could have a suppressive effect on PTH secretion, and it could be a risk factor for the development of adynamic bone disease in dialysis patients.

Key words: hypermagnesemia, serum PTH, adynamic bone disease

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Renal osteodystrophy is an important complication of patients on hemodialysis (HD). The most frequent histologic patterns are osteitis fibrosa and mixed uremic osteodystrophy (19, 31, 32). During the past decade, states of low bone turnover—adynamic bone disease (ABD)—have been reported (20, 23, 30, 31), pointing to aluminum as the major pathogenic factor in these lesions (10, 19, 20, 23, 30–32). However, more recently, systematic bone biopsy studies in patients who were never exposed to aluminum demonstrated the existence of non-aluminic ABD, also called idiopathic ABD (8, 14, 23). Although several factors have been incriminated at the origin of this osteopathy (15), its exact pathophysiology remains unknown.

Magnesium (Mg) is one of the major factors controlling levels of serum calcium, and it acts mainly by regulation of parathyroid hormone (PTH). In the early 70's, some authors reported data about the bone Mg content in uremic patients (1, 9) and the effect of Mg deficiency in the progression of chronic renal disease and resistance to both vitamin D and PTH (21). Currently, a complete understanding about the influence of Mg on PTH and bone physiology in uremic patients has not yet been achieved.

We observed that some hemodialysis patients with low intact PTH (iPTH) levels had an elevated serum Mg concentration. This observation prompted us to analyze in 41 patients undergoing maintenance hemodialysis the serum Mg concentration, the relationship between Mg and PTH levels, and the possible role of hypermagnesemia as a risk factor for the development of idiopathic ABD.

MATERIAL AND METHODS

Patients

Forty-one patients (25 males and 16 females) aged

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between 20 and 80 years (mean age 55 ± 16 years) on regular hemodialysis for 44 ± 36 months (range 12–168) were assessed with a follow-up between 1 and 3 years. The causes of end stage renal disease (ESRD) were: chronic glomerulonephritis (n = 8), diabetic nephropathy (n = 8), nephroangiosclerosis (n = 7), polycystic kidney disease (n = 4), interstitial nephropathy (n = 3), other diseases (n = 5), and unknown etiology (n = 6). All patients were dialyzed 3.5 to 4.5 hours, thrice weekly using capillary dialyzers, with standard dialysis solutions containing 2.4 mg/dl of magnesium and 6 mg/dl of calcium. The blood flow rate and dialysate flow rate were 300 and 500 ml/min respectively. Thirty-eight patients received oral calcium carbonate ($CaCO_3$) supplements as primary phosphate binder, and 27 subjects also received aluminum hydroxide ($Al(OH)_3$). Only 8 cases were on treatment with vitamin D active metabolites. Doses of $CaCO_3$, $(Al(OH)_3$ and vitamin D did not experimented significant variations during the study. Cumulative $CaCO_3$ and $Al(OH)_3$ intakes were calculated from the record of each patient. None of the patients had systemic illness or organ disease other than diabetes that may affect bone metabolism.

Biochemical parameters

Blood samples for serum calcium, phosphate and alkaline phosphatase measurements were obtained monthly. Serum Mg, iPTH, and 1,25 dihydroxivitamin D ($1,25(OH)_2D$) were measured every 2 months, and aluminum every 6 months. All samples were collected immediately before the first dialysis session of the week. Serum calcium, phosphorus and alkaline phosphatase concentrations were quantified using standard laboratory techniques. Serum iPTH level was determinated by an immunoradiometric assay (IRMA), $1,25(OH)_2D$ by radioimmunoassay, and Mg and aluminum were measured by atomic absorption spectroscopy. The normal values for serum calcitriol are between 18 and 78 pg/ml. The normal level of serum Mg in our laboratory is 1.5 to 2.5 mg/dl. The normal range of alkaline phosphatase and iPTH varies between 98 and 279 IU/L and 15 and 60 pg/ml, respectively. Since patients with ESRD need higher PTH values to maintain a normal bone remodelling (33, 34), we considered as "adequate" a iPTH concentration between 120 and 250 pg/ml (2–4 times the upper limit of the normal range).

Statistical analysis

Results are expressed as mean ± SEM. Student's t-test was used to analyze differences between means of two quantitative variables. One-way analysis of variance (ANOVA) was performed to compare more than two quantitatives variables, with multiple range analysis (Newman Keuls test) when appropriate. Qualitative variables were analyzed using chi-square test. Linear regression analysis was used to correlate two quantitative variables. P values below 0.05 were considered as significant.

Table I. *Demographic and clinical characteristics of 41 hemodialyzed patients according to their serum iPTH levels*

Patients	Group A* (Low PTH)	Group B* (Normal PTH)	Group C* (High PTH)
n	13	16	12
Age (yrs)	57 ± 15	59 ± 15	49 ± 18
Male/Female	9/4	10/6	6/6
T° HD (months)	33 ± 20	59 ± 8	37 ± 31
D/ND	2/11	4/12	2/10

T° HD: Time on hemodialysis; D: Diabetics, ND: Non diabetics
* Not significant

RESULTS

The mean concentrations of serum calcium, phosphate, magnesium, alkaline phosphatase (AP), ferritin, $1,25(OH)_2D$ and iPTH in the group of 41 patients during the study were 9.9 ± 0.6 mg/dl, 5.6 ± 1.9 mg/dl, 2.4 ± 0.2 mg/dl, 230 ± 98 U/L, 218 ± 137 µg/L, 49.1 ± 10 pg/ml and 210 ± 173 pg/ml, respectively. There were no significant variations in these parameters during the study.

The patients were classified into 3 groups according to the serum iPTH level: Group A, low iPTH (below 120 pg/ml); Group B, adequate iPTH (between 120 and 250 pg/ml), and Group C, high iPTH (above 250 pg/ml). To better ascertain potential risk factors of the reduced iPTH levels, we compared subjects on group A to the patients with normal or high iPTH concentrations. There were no differences between

Table II. *Biochemical parameters in 41 hemodialyzed patients classified according to their serum iPTH levels*

	Group A (Low PTH)	Group B (Normal PTH)	Group C (High PTH)
Ca (mmol/L)	2.4 ± 0.2	2.42 ± 0.1	2.55 ± 0.1
P (mmol/L)	1.8 ± 0.6	1.6 ± 0.4	2 ± 0.7
HCO_3 (mmol/L)	23 ± 2.8	23 ± 4	22 ± 3.8
Al (µg/ml)	23 ± 14	27 ± 19	26 ± 38
$CaCO_3$ (g/d)	5.9 ± 1.5	5.3 ± 1.1	6 ± 1.8
$Al(OH)_3$ (g/d)	2.1 ± 0.8	1.9 ± 0.6	2.3 ± 0.9
Vit D (ng/L)	52.2 ± 14	48.4 ± 9	50.6 ± 11
Ferr (µg/L)	235 ± 120	205 ± 134	216 ± 165
AP (U/L)	170 ± 60[a,b]	239 ± 54[a]	283 ± 141[b]
iPTH (pg/ml)	67.5 ± 31[b]	175 ± 31[b]	411 ± 192[b]

Ca: calcium; P: phosphorus; HCO_3: bicarbonate; Al: aluminum; $CaCO_3$: dose of calcium carbonate; $Al(OH)_3$: dose of aluminum hydroxide; Vit D: 1,25 vitamin D; Ferr: ferritin; AP: alkaline phosphatase; iPTH: intact PTH
[a] $p < 0.05$, ANOVA, Newman-Keuls test
[b] $p < 0.01$, ANOVA, Newman-Keuls test

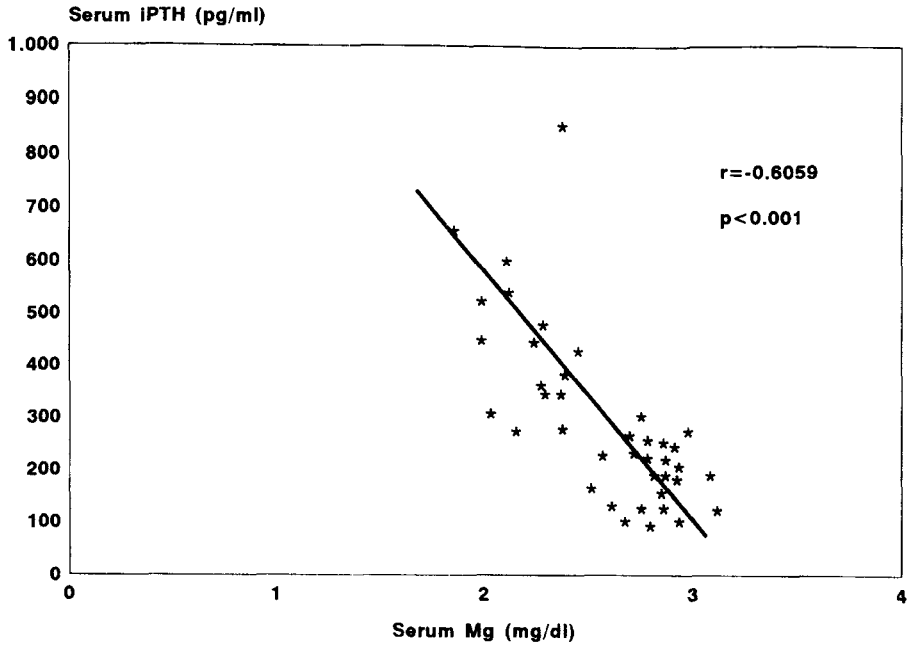

Fig. 1. Relationship between plasma intact PTH and serum magnesium (Mg) concentration.

groups related to number of patients, age, sex, time on dialysis or renal disease (Table I). Biochemical parameters are presented in Table II. Serum $1,25(OH)_2D$, ferritin, calcium, phosphate, bicarbonate and aluminum concentration, and daily doses of $CaCo_3$ and $Al(OH)_3$ were similar in all the groups. Cumulative doses of $Al(OH)_3$ and $CaCO_3$ were lower in patients with low iPTH respect to patients on groups B and C (62 ± 53 vs 128.5 ± 86 and 109.4 ± 80 g; and 144.3 ± 102 vs 284.6 ± 170 and 195.8 ± 163 g, respectively), but the differences were not statistically significant. Markers of bone turnover such as PTH and AP were significantly lower in group A than in patients on groups B and C (Table II). Interestingly, when compared to patients with normal or high iPTH, those with low iPTH had a higher serum Mg concentration (2.8 ± 0.2 vs 2.3 ± 0.1 and 2.2 ± 0.1 mg/dl, respectively; $p < 0.01$). Moreover, regression analysis showed a negative lineal correlation between the concentration of serum iPTH and the level of serum Mg ($r = -0.6059$, $p < 0.001$) (Fig. 1).

Thus, on comparing patients with low iPTH level with subjects with normal or high iPTH, the only cut difference was that the low iPTH group had a higher serum Mg concentration. Based on this findings we classified the 41 patients into 2 groups according to their serum Mg level: Group I, normal Mg (1.5–2.5 mg/dl, N = 17), and group II, high Mg (>2.5 mg/dl, N = 24). Plama Mg was significantly higher in group II than in group I (2.65 ± 0.2 vs 2.23 ± 0.1 mg/dl, $p < 0.001$). There were no differences concerning the following parameters: age, sex, time on dialysis, renal disease, serum phosphate, bicarbonate, aluminum, ferritin, $1,25(OH)_2D$, dose of $CaCO_3$ or $Al(OH)_3$, and cumulative aluminum and calcium intake (Table III). However, patients in group II had a significant lower iPTH concentration (116 ± 61 vs 342 ± 194, $p < 0.001$), and curiously, they also had a significant lower serum calcium level (9.6 ± 0.6 vs 10.2 ± 0.5, $p < 0.05$).

DISCUSSION

Patients with ESRD on dialysis need a serum level of

Table III. *Demographic, clinical, and biochemical parameters in 41 hemodialyzed patients according to their serum magnesium (Mg)*

	Group I* (Normal Mg)	Group II* (Elevated Mg)
Age (yrs)	53 ± 17	57 ± 16
Males/Females	8/9	17/7
T° HD (months)	42 ± 40	43 ± 33
D/ND	2/17	6/24
P (mmol/L)	1.9 ± 0.6	1.6 ± 0.5
HCO_3 (mmol/L)	22.7 ± 3.3	23.3 ± 3.7
Al (μg/ml)	26 ± 23	25 ± 17)
Ferr (μg/L)	187 ± 150	239 ± 125
Vit D (ng/L)	53.7 ± 12	51.4 ± 11
$CaCO_3$ (g/d)	5.6 ± 1.8	5.6 ± 1.2
$Al(OH)_3$ (g/d)	2.2 ± 0.8	2 ± 0.8
Cumulative Al intake (g)	103 ± 78	80.5 ± 73
Cumulative Ca intake (g)	218.6 ± 166	221.5 ± 169

T° HD: Time on hemodialysis; D/ND: Diabetics/Nondiabetics; P: phosphorus; HCO_3: bicarbonate; Al: aluminum; Ferr: ferritin; Vit D: vitamin D; $CaCO_3$: dose of calcium carbonate; $Al(OH)_3$: dose of aluminum hydroxide
* Not significant

iPTH between 2 and 4 times the upper limit of the normal range to avoid both low turnover and hyperparathyroid bone disease (14–17). ABD was described for the first time in 1982 by Sherrard et al. (31). Many authors have studied the correlation between iPTH level and bone biopsy findings and, they have found that ABD is always associated with relatively low plasma PTH levels, ie, iPTH in the normal range or slightly increased (7, 8, 16–18, 22, 23, 29, 30, 34). In our study, 13 out of the 41 subjects (31.7%) had a decreased serum iPTH concentration. Although bone biopsy was not performed, we considered that these patients (group A), based on their iPTH level, are or may be at risk to develop an adynamic bone disease. Moreover, these patients had a significant lower serum AP concentration that patients on groups B and C (Table II). The serum level of this enzyme activity has been also found to be significantly decreased in low turnover renal osteodystrophy compared to hyperparathyroid bone disease (11, 13).

ABD has been classically associated with aluminum accumulation (10, 19, 20, 23, 30–32). However, in 1989, Morinière et al. (23) reported the first cases of non-aluminic ABD in patients undergoing dialysis. Twenty-seven out of the 41 patients included in the study (65.8%) were treated with $Al(OH)_3$, with a similar distribution: 9 on group A, 10 on group B, and 8 on group C. Dose of $Al(OH)_3$ was similar in all groups (Table II), and furthermore, although the differences were not statistically significant, patients with low iPTH had a mean cumulative intake of $Al(OH)_3$ lower than patients with normal or high iPTH (62 ± 53 vs 128.5 ± 86 and 109.4 ± 80 g, respectively). As a result of these findings, it is extremely unlikely that aluminum played a significant role in the genesis of ABD in our patients.

Besides aluminum, several factors have been implicated at the origin of ABD, namely, diabetes, iron overload, acidosis and increasing age (20, 28). In dialysis population, both insulin and non-insulin dependent diabetes mellitus has been associated with low PTH secretion and low turnover bone disease (28, 35). In our study, it is unlikely that diabetes could explain the low iPTH concentration in subjects on group A, since only 8 patients were diabetic and there was a uniform distribution among all the groups (Table I). Furthermore, serum iPTH level was similar in diabetic and non-diabetic patients (196 ± 140 pg/ml vs 216 ± 184 pg/ml). The possible contribution of iron overload as a cause of the low iPTH in patients on group A can be ruled out since the serum ferritin level, the best parameter to monitor iron stores in dialysis population (2), was lower than 300 μg/L (the upper limit of the normal range) and there were no significant differences among groups (Table II). Serum bicarbonate was not decreased, ruling out acidosis as cause of depressed serum iPTH. Some authors have also noted the emergence of the aplastic lesion in subjects treated with calcium salts as a phosphate binder (20, 23). Although the most of our patients were received $CaCO_3$, the distribution among the groups was similar (10 in group A, 16 in group B, and 11 in group C), with no differences concerning the dose of calcium supplements (Table II). Moreover, the total cumulative intake of calcium was lower in patients with decreased iPTH than in patients on groups B and C (144.3 ± 102 vs 284.6 ± 170 and 195.8 ± 163 g), although the differences did not reach statistical signification. On the other hand, other authors have hypothesized that vitamin D might have a pathogenic role (23). However, only 8 (19%) were on treatment with vitamin D active metabolites, and only one of them was on group A. Recently, Malluche and Faugere (20) have reported that the prevalence of ABD increased from 9% in patients less than 25 years to 25% in those 75 years and older, suggesting the role of increasing age in the occurrence of ABD. However, the mean age in patients on group A was 57 ± 15 years, without significant differences with group B (59 ± 15) and C (49 ± 18).

Magnesium is the fourth most abundant cation in the human body. In the normal individual, Mg has effects on PTH secretion similar to those of calcium, but at similar concentrations, Mg is two to three times less effective than calcium in inhibiting PTH release (3–5). However, little is known about the effects of Mg on PTH and bone metabolism in patients with renal disease. Increased Mg levels have been related with suppression of PTH release, but conflicting results have been reported (6, 16). In our study, 24 subjects (58.5%) had a chronically increased level of serum Mg (above 2.5 mg/dl). Interestingly, patients with low iPTH had a higher serum Mg concentration than patients with normal or high iPTH levels, and moreover, a negative linear correlation was observed between serum iPTH and Mg concentration (Figure 1). Several works have showed the successful control of hyperparathyroidism in dialysis patients using Mg salts as phosphate binder (24–26). In these studies, meanwhile plasma Mg concentration increased, elevated PTH level decreased, and in some cases, the control of hyperparathyroidism was better in patients receiving Mg salts than in those subjects only treated with $CaCO_3$ or $Al(OH)_3$. Morinière et al. (24) administered Mg salts to control hyperphosphatemia in 20 dialysis patients. Serum Mg concentration raised from 1.89 to 2.89 mg/dl and remained stable during 18 months. They concluded that mild hypermagnesemia had no noxius effects on bone mineralisation, but in this study, the mean PTH level decreased from 260 pg/

ml to 185 pg/ml. The mean hemodialysis time in our patients was very higher (44 months), and it is possible that chronic hypermagnesemia during a long-term period can have deletereous consequences on PTH secretion and bone metabolism.

Recently, Felsenfeld (12) has performed a study to determinate whether a reduction in dialysate calcium increases PTH levels and improves bone turnover in diabetic dialysis patients with relative deficiency of PTH. He found that after 6 months of dialysis with a low dialysate calcium, PTH increased from 139 to 242 pg/ml. Based on our findings, could be important to analyze the Mg concentration in patients with low PTH levels, and to evaluate the effect of a reduction in Mg dialysate on parathyroid function in these subjects.

Chronic Mg excess can have an eventual noxius effect on PTH secretion. Long-term mild chronic hypermagnesemia in uremic patients could be a risk factor for the development of ABD. Based on our data, several questions focusing on the relevance of Mg in renal bone disease need to be answer: (1) What is the exact relationship between serum Mg concentration and iPTH levels in patients with renal disease?, (2) Should Mg levels be periodically measured in these patients?, (3) Is chronic hypermagnesemia a risk factor for ABD in dialysis patients?, and (4) Need dialysis patients with unappropriately low iPTH level a reduction in the concentration of dialysate Mg in order to increase serum PTH and to improve bone turnover?

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