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ORIGINAL ARTICLE

Inverse correlation between serum magnesium and parathyroid hormone in peritoneal dialysis patients: a contributing factor to adynamic bone disease?

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Abstract
Background Secondary hyperparathyroidism (SHPTH) is present in many patients with end-stage renal disease (ESRD) and has been linked to uremic bone disease. Parathyroid hormone (PTH) levels are affected by calcium, vitamin D, and phosphorus. Recent data suggests that serum magnesium may also modulate PTH levels.
Objective The aim of this retrospective study was to investigate the impact of different calcium (Ca) and magnesium (Mg) concentrations of dialysis solutions on serum Mg and serum PTH levels in peritoneal dialysis (PD) patients.
Patients and methods Two groups of PD patients-group A ($n = 17$) on ''standard'' Ca and Mg dialysis solution (SCa–MgD) (Ca: 1.62 mmol/l, Mg: 0.75 mmol/l and Lactate 35 mmol/l), and group B ($n = 29$) on ''low'' Ca and Mg dialysis solution (LCa–MgD) (Ca: 1.25 mmol/l, Mg: 0.25 mmol/l and Lactate 40 mmol/l), on PD for more than 6 months, were studied. Calcium carbonate ($CaCO_3$) was used as the phosphate (P) binder in 87% (40/46) of the patients. Biochemical parameters were evaluated every 1–2 months over 6 months and the mean values were computed.
Results No significant differences were found between the two groups in all parameters except for serum Mg and PTH. Serum Mg was higher in SCa–MgD group compared to those in the LCa–MgD group (1.05 ± 0.19 vs 0.90 ± 0.23 mmol/l, respectively) and serum PTH was higher in LCa–MgD group compared to those in SCa–MgD group (72.3 ± 64.2 vs 31.1 ± 39.0 pmol/l, respectively) even though serum Ca was not different. There was a statistically significant inverse correlation between serum Mg and PTH levels ($r = -0.357$, $p < 0.05$).
Conclusion Serum Mg is lower and serum PTH higher in patients dialyzed with lower Mg concentration dialysis solution compared to those with higher Mg concentration dialysis solution. Our study confirms previous reports that serum Mg may have a suppressive role on PTH synthesis and/or secretion, and thus may play a role in pathogenesis of adynamic bone disease that often develops in patients on chronic PD with high calcium and high magnesium concentrations.

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Keywords Adynamic bone disease · Magnesium · Parathyroid hormone · Phosphate binders · Peritoneal dialysis

Introduction

Secondary hyperparathyroidism (SHPTH) is one of the most common complications in ESRD patients [1]. Serum Ca, vitamin D and P levels are considered to regulate the synthesis and secretion of PTH. Apart from these factors, it has been reported that serum Mg may be involved in regulating PTH levels [2–7]. Serum Mg concentration is closely related to the dialysis solution Mg concentration. Reduction in Mg concentration in the dialysis fluid leads to decrease in serum Mg levels, and vice versa [8–10]. We studied retrospectively two groups of patients: group A ($n = 17$) on ''standard'' Ca and Mg dialysis solution (SCa–MgD) and group B ($n = 29$) on ''low'' Ca and Mg dialysis solution (LCa–MgD) on PD for more than 6 months and evaluated the impact of dialysis solution Mg on serum Mg and the relation between serum Mg and PTH.

Patients and methods

Patients

We studied 46 patients who had started peritoneal dialysis after Jan 1, 1993 and before Dec 31, 2003. Among them, 17 were on SCa–MgD (group A: 8 males and 9 females), (Ca 1.62 mmol/l, Mg 0.75 mmol/l, and Lactate 35 mmol/l); and 29 patients were on LCa–MgD (group B: 13 males and 16 females), (Ca 1.25 mmol/l, Mg 0.25 mmol/l, and Lactate 40 mmol/l). Of these 46 patients, 40 were using $CaCO_3$ as phosphate binder, 8 were using sevelamer (Renagel), and 19 patients were receiving oral calcitriol. None of the patients received aluminum-containing agents as phosphate binder. Thirteen patients were on continuous ambulatory peritoneal dialysis (CAPD) using four or five 2-l exchanges per day, 33 patients were on automated peritoneal dialysis (APD) using 10–12l per night. Biochemical parameters, such as serum ionized calcium (Ca^{2+}), total calcium (TCa), magnesium (Mg), PTH and bicarbonate levels were measured over a 6 month period using standard laboratory methods. The average levels of these measurements were used for analysis.

Statistical analysis

Data were expressed as mean ± SD; Statistical analysis was performed with SPSS 10.0 package for Windows. Student's *t*-test was used to compare the mean of quantitative variables between groups. Chi-square test was used to compare the differences in categorical variables. Possible relationships between PTH level and other parameters were evaluated by linear regression analysis. Partial correlation analysis was used to measure each correlation while adjusting for colinearity with the other two variables. Finally, a forward stepwise multiple regression analysis was performed using PTH as the dependent variable and sex, age, duration of dialysis, Ca^{2+}, TCa, P, and Mg as independent variables. A p value less than 0.05 was considered significant.

Results

The demographic characteristics and the mean concentrations of serum Ca^{2+}, TCa, P, Mg and PTH in SCa–MgD group and LCa–MgD group are shown in Table 1.

No significant differences were found between SCa–MgD group and LCa–MgD group except for serum Mg and PTH levels. Serum Mg was significantly lower and serum PTH was significant higher in patients with LCa–MgD group. When patients were divided into two groups (above or below the upper limit of normal serum Mg concentration). (Group 1 ($n = 30$) serum Mg ≤ 1.02 mmol/l, group 2 ($n = 16$) serum Mg > 1.02 mmol/l), there was a significant difference in serum PTH levels between these two groups ($p < 0.01$), even though there were no significant differences in serum Ca levels (Table 2). Serum PTH was higher in patients with lower serum Mg, and PTH was lower in patients with higher serum Mg (PTH 85.7 ± 67.8 vs 30.2 ± 20.5 pM/l, respectively). Linear regression analysis showed an inverse relationship between serum Mg and PTH ($r = -0.357$, $p = 0.032$). Also we found an inverse relationship between age and serum PTH

Table 1 Demographic characteristics and laboratory parameters of 46 patients included in the study

Parameters	SCa–MgD group (n = 17)	LCa–MgD group (n = 29)
Male/female	8/9	13/16
Diabetes/nondiabetes	3/14	4/25
Age (years)	56.3 ± 18.3	57.0 ± 17.3
Time on dialysis (months)	76.7 ± 38.2	57.0 ± 28.1
Ionized calcium (mmol/l)	1.24 ± 0.07	1.22 ± 0.09
Total calcium (mmol/l)	2.40 ± 0.16	2.36 ± 0.21
Serum albumin (g/l)	**36.7 ± 3.3**	**35.9 ± 3.1**
Phosphorous (mmol/l)	1.73 ± 0.65	1.76 ± 0.61
Magnesium (mmol/l)	1.05 ± 0.19	0.90 ± 0.23[a]
PTH (pmol/l)	31.1 ± 39.0	72.3 ± 64.2[a]
Bicarbonate (mmol/l)	25.76 ± 2.51	25.52 ± 2.32
Doses of $CaCO_3$ (mg/day) (n_1/n_2 = 14/26)	1660.7 ± 824.0	2076.9 ± 2309.5
Dose of Calcitriol (mcg/week) (n_1/n_2 = 8/11)	2.16 ± 2.38	1.95 ± 0.80
Dose of Sevelamer(mg/day) (n_1/n_2 = 1/7)	7200	3347.1 ± 3073.3

n_1/n_2: sample size of SCa–MgD group/ LCa–MgD group in using $CaCO_3$, Calcitriol and Sevelamer

a: $p < 0.05$, (SCa–MgD group versus LCa–MgD group)

($r = -0.323$, $p = 0.044$). After controlling for age the inverse correlation between serum Mg and PTH was still present ($r = -0.36$, $p = 0.015$). Fig. 1 shows the correlation between serum Mg and PTH in these 46 PD patients (Pearson's $r = -0.357$, $p = 0.032$).

Discussion

Renal osteodystrophy is one of the most common complications in patients with end stage renal diseases. Bone disorders are categorized into two principal types: a high-turnover osteodystrophy, known as ostelitis fibrosa cystica and a low-turnover state characterized by adynamic bone disease [11], that occurs in 50–66% dialysis patients, especially in CAPD patients [12–15]. Recent studies suggested that adynamic bone disease is linked with low serum PTH [12, 16–22], and is characterized by reduced osteblasts and osteoclasts, no accumulation of osteoid and markedly low bone turnover on bone histology. [23, 24], and prevalence of bone pains, fractures and hypercalcemias clinically [25]. Torres et al [18] reported that in dialysis patients not using vitamin D, serum PTH level lower than 120 pg/ml (13.2 pmol/l) was highly predictive of low bone turnover with a positive predictive value of 90%. It is well documented that serum PTH is regulated by serum Ca, P and vitamin D [26–35]. However, apart from these factors, Mg may play an important role in regulating serum PTH level. It was reported that intravenous Mg sulfate infusion depressed iPTH levels in a healthy population [36, 37]. Gough et al [38] reported that intravenous Mg sulfate infusion significantly

Table 2 Characteristics of 46 patients on PD classified according to their serum Mg level

	Group1 (n = 30) (Mg ≤ 1.02 mmol/l)	Group 2 (n = 16) (Mg > 1.02 mmol/l)
Male/female	11/19	10/6
Age (years)	57.7 ± 17.2	54.9 ± 18.4
Time on dialysis (months)	66.2 ± 29.7	60.6 ± 39.8
Time on low Mg dialysis (months)	30.8 ± 24.0	29.4 ± 32.9
Ionized calcium (mmol/l)	1.23 ± 0.10	1.23 ± 0.06
Total calcium (mmol/l)	2.40 ± 0.19	2.42 ± 0.20
Serum albumin (g/l)	36.4 ± 2.9	35.7 ± 3.8
Phosphorous (mmol/l)	1.68 ± 0.58	1.87 ± 0.67
Bicarbonate (mmol/l)	25.53 ± 2.46	25.75 ± 2.27
PTH (pmol/l)	73.2 ± 66.0	26.9 ± 24.9[a]
Doses of $CaCO_3$ (mg/day) (N_1/N_2 = 24/15)	2239.6 ± 2406.4	1500.0 ± 574.8
Dose of Calcitriol (mcg/week) (N_1/N_2 = 13/6)	1.69 ± 0.94	2.79 ± 2.48
Dose of Sevelamer(mg/day) (N_1/N_2 = 4/4)	4600.0 ± 3600.0	3057.5 ± 2945.0

N_1/N_2: sample size of group1/group 2 in using $CaCO_3$, Calcitriol and Sevelamer

a: $p < 0.01$ (group 1 versus group 2)

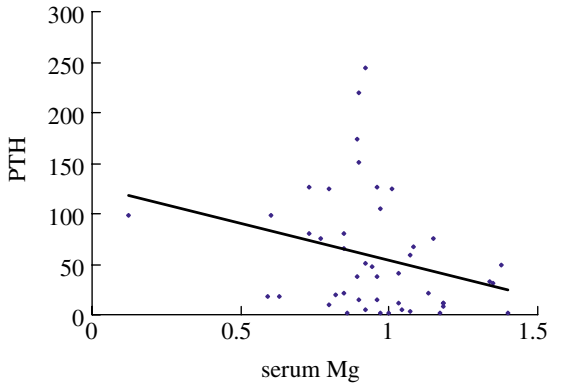

Fig. 1 Correlation between serum Mg and PTH in 46 PD patients (Pearson's $r = -0.357$, $p < 0.05$)

suppressed iPTH levels in patients with primary hyperparathyroidism. Also there are some reports [39, 40] indicating that magnesium carbonate ($MgCO_3$) as a phosphate binder depressed serum PTH level. Normalization of serum Mg by using low Mg dialysis solution in hypermagnesemic hemodialysis patients was followed by a rise in serum PTH level [41, 42]. Navarro et al. [4, 5] reported that there was a significant inverse correlation between serum Mg and PTH in both HD and CAPD patients. Similar results were reported by other authors [3, 43–46]. In our study, we found that serum Mg was higher in patients on SCa–MgD solution group compared to those on LCa–MgD solution group (mean serum Mg was 1.05 ± 0.19 mmol/l vs 0.90 ± 0.23 mmol/l, respectively, $p < 0.05$). Our findings confirm previous observations that dialysis solution Mg plays a critical role in determining serum Mg level in PD patients [8, 47, 48].

There was an inverse relationship between serum Mg and PTH in our study, similar to that reported by other authors [3, 4, 48, 49]. All these findings suggest that high serum Mg may be suppressing the synthesis and/or secretion of serum PTH, and low serum Mg stimulates PTH secretion independently of the serum Ca and P concentration. It is possible that the hyperparathyroidism that developed in PD patients after conversion from high Ca (and Mg) to low Ca (and Mg) solution [47] is due to the decrease in serum Mg. Furthermore, since low PTH has been considered an important factor contributing to adynamic bone disease, it is possible that elevated serum Mg inhibits parathyroid hormone (PTH) synthesis and/or secretion and may play a role in the pathogenesis of adynamic bone disease. In our study, 7 patients, (41.2%) in SCa–MgD group had low serum PTH (PTH < 7.6 pmol/l, whereas, in the LCa–MgD group, only one patient (3.5%) had a serum PTH < 7.6 pmol/l (PTH 5.6 pmol/l). There was no significant difference in the percentage of vitamin D use between patients with low PTH and normal or above normal PTH levels. None of these 46 patients had received Al-containing agents previously. There were no significant differences in age, diabetes/non-diabetes patients, and in serum Ca and P levels between SCa–MgD group and LCa–MgD group. Therefore, it is unlikely that these factors explain the low PTH level in the SCa–MgD group. These data, together with the results of the regression analysis showing an independent inverse relationship between serum Mg and PTH, strongly suggest that an elevated serum Mg level might be a factor responsible for the low PTH level. The mechanism of action of Mg on PTH is unknown; an in vitro study has demonstrated that modifications of extracellular Mg may induce changes in intracellular Ca by two independent mechanisms that regulate PTH secretion [50]. In vitro [6] and in vivo [36, 38, 51] (animal and human) studies have demonstrated that Mg modulates PTH secretion in a way similar to that of Ca. This hypothesis was supported by recent studies on successful use of Mg salts to control hyperparathyroidism, and the use of Mg-containing agents as phosphate binders [39, 40, 52–56]. These studies indicate that, as a phosphate binder, $MgCO_3$ may be an alternative phosphate binder.

In conclusion, our study demonstrates that concentration of Mg in dialysis solution may affect serum Mg and PTH levels in PD patients without changing their serum Ca. There is an inverse relationship between serum Mg and PTH. Elevated serum Mg may have a suppressive role on PTH synthesis and/or secretion, and an elevated serum Mg may play a role in pathogenesis of adynamic bone disease. If this can be confirmed in prospective studies, $MgCO_3$ may be an alternative phosphate binder in patients on low Ca–M dialysis solution.

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