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Calcium Uptake and Phosphate Removal during Hemodialysis with Variing Dialysate Calcium

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Summary. In three patients with stable uraemia the external balance of calcium and phosphate was estimated during hemodialysis with a calcium concentration of 6 mg/100 ml and 8 mg/100 ml in the dialysate, by measuring both ions in the dialysate in 100 routine dialyses.

Dialysis with a dialysate calcium of 6 mg/100 ml and 8 mg/100 ml led to a calcium uptake of 72 ± 36 mg ($\pm$ SEM, $n = 63$) and 240 ± 52 mg ($\pm$ SEM, $n = 34$), respectively, phosphate removal decreased significantly from 1019 ± 37 mg to 856 ± 50 mg respectively.

Dialysis with a dialysate calcium of 8 mg/100 ml led to post dialysis hypercalcaemia of 11.5 ± 0.9 mg/100 ml ($\pm$ SD) which lasted occasionally until the beginning of the next dialysis, the predialysis phosphate levels and predialysis calcium phosphate product decreased significantly with this regimen.

Although most of the induced biochemical changes are not against a dialysate calcium of 8 mg/100 ml, development of marked hypertension in one patient and persistent hypercalcaemia in another one after only five weeks of treatment were reason to abandon the recently suggested use of a dialysate calcium above the concentration of ultrafiltrable serum calcium, especially in the light of the comparatively small net gain of calcium during dialysis.

Key words: Haemodialysis, calcium, phosphate, osteodystrophy, hypertension.

Calciumaufnahme und Phosphatentzug während Hämodialyse mit unterschiedlichem Calciumgehalt im Dialysemedium.

Zusammenfassung. Aus den Konzentrationsunterschieden von Calcium und Phosphat im Dialysat von 100 Routinedialysen vor und nach der Dialyse wurde der Effekt von unterschiedlichen Dialysatcalciumkonzentrationen auf die Calcium- und Phosphatbilanz von chronisch-intermittierend dialysierten Patienten ermittelt.

Dialyse mit einem Dialysatcalcium von 6 mg-% führte zu einer Calciumaufnahme von 72 ± 36 mg ($\pm$ SEM, $n = 63$) und zu einem Phosphatentzug von 1019 ± 37 mg ($\pm$ SEM, $n = 63$). Während der Dialyse mit einem Dialysatcalcium von 8 mg-% stieg die Calciumaufnahme auf 240 ± 52 mg ($\pm$ SEM, $n = 34$), die Phosphatelimination fiel um denselben Betrag auf 856 ± 50 mg ($\pm$ SEM, $n = 34$).

Mit einem Dialysatcalcium von 8 mg-% kam es zwar zu einer postdialytischen Hypercalcämie von $11{,}5 \pm 0{,}9$ mg-% ($\pm$ SD), jedoch wurde ein signifikantes Absinken der prädialytischen Serumphosphatwerte und des für die Entstehung von metastatischen Calcifikationen entscheidenden Calciumphosphatproduktes beobachtet.

Obwohl die Mehrzahl der biochemischen Veränderungen nicht gegen die Verwendung einer höheren Dialysatcalciumkonzentration sprechen würde, wird eine dem ultrafiltrierbaren Serumcalcium entsprechende Dialysatcalciumkonzentration von 6 mg-% empfohlen, nachdem sich bei einem Patienten nach wenigen Wochen der Verwendung einer höheren Dialysatcalciumkonzentration eine ausgeprägte und später reversible Hypertonie, bei einem anderen eine persistierende Hypercalcämie entwickelten.

Schlüsselwörter: Calcium, Phosphat, Hämodialyse, Osteodystrophie, Hypertonie.

Introduction

After initially great variations in reported dialysate calcium concentrations [2, 3] the optimum calcium concentration seemed clearly defined at 6 mg/100 ml [36], this concentration preventing calcium losses during dialysis, arresting bone changes and preventing a great increase in post dialysis serum calcium. The question of optimal calcium concentration has been reopened recently since various authors [7, 9, 20] dialysing patients against a calcium concentration higher than 6 mg/100 ml found a marked decrease in serum parathyroid hormone levels with consequent improvement in bone histology [11]. Calcium gains of between 700 and 1100 mg per dialysis were calculated by means of arteriovenous concentration difference and blood flow measurements [9, 20], a method which will always remain open to criticism [18]. Apart from three isolated measurements of the external calcium balance during hemodialysis against the calcium concentration at question [36] we are unaware of any other calcium and phosphate balance data in the literature. This gap of information has also been expressed in a recent review of this subject [31].

We have investigated the external balance and internal distribution of calcium and phosphate during 100 routine dialyses at a dialysate calcium of 6 mg/100 ml and 8 mg/100 ml and this paper presents our findings.

Material and Methods

Patients

1. 30 year old male with Good Pasture Syndrome, anuric and on hemodialysis for 18 months.
2. 40 year old woman with chronic pyelonephritis, on hemodialysis for 6 months, creatinine clearance between 2 and 3 ml/min.
3. 47 year old male with nephrolithiasis and chronic pyelonephritis, on hemodialysis for 14 months, creatinine clearance less than 2 ml/min.

The patients were dialysed twice a week for eight hours with a blood flow rate between 120 and 180 ml/min using a

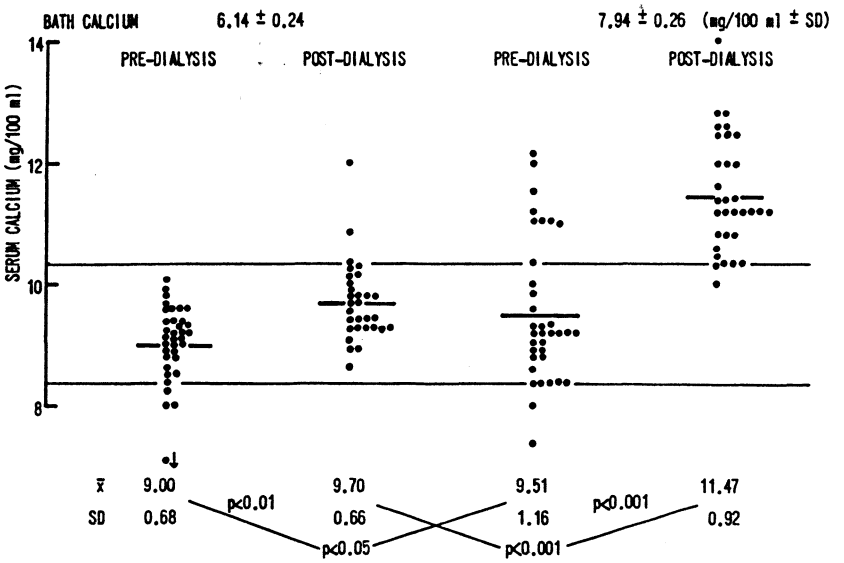

Fig. 1. Serum calcium before and after dialysis with a dialysate calcium of 6 mg/100 ml and 8 mg/100 ml

twin coil kidney, the dialysate composition being as follows: sodium 125 mEq/l, potassium 3 mEq/l, calcium 6 mg/100 ml, magnesium 2.5 mg/100 ml, chloride 100 mEq/l, acetate 30 mEq/l, glucose 200 mg/100 ml. Throughout the patients received 1.5 g aluminum hydroxide daily.

Investigations

For the purpose of the study the patients were dialysed for a total of 64 dialyses using the Travenol standard tank and changing the dialysate every two hours. An aliquot of each dialysate pre and post dialysis was analysed for calcium by a routine complexometric method (Titriplex) and for phosphate by the method of Fiske [38]. Total transfer of calcium and phosphate was calculated from the initial and final concentration in the bath fluid. The adequacy of the mixing of the concentrated salt solution and water was checked by determination of its electrical conductivity [19].

Total plasma calcium and phosphate was measured using the above methods immediately before and at the end of dialysis, the sample being taken from the arterial cannula. In addition urea nitrogen, creatinine and bicarbonate was measured before and at the end of a representative number of dialyses by routine laboratory methods [23].

After a total of 64 dialyses the concentration of calcium was changed to 8 mg/100 ml leaving the other components unchanged and in a total of 34 dialyses the above measurements were carried out.

Results

All results are expressed as mean values $\pm$ SEM. Dialyses using a calcium concentration of 6 mg/100 ml and 8 mg/100 ml are referred to in this paper as "normal calcium" and "high calcium" dialyses respectively.

Effects of Dialysate Calcium of 6 mg/100 ml

Predialysis serum calcium levels during "normal calcium" dialysis (dialysate calcium 6.14 ± 0.09 mg/100 ml, $n = 64$) were 9.5 ± 0.11 mg/100 ml and increased significantly ($p < 0.01$) to 9.7 ± 0.12 mg/100 ml after 8 hours dialysis (Fig. 1). Pre- and postdialysis serum phosphate levels were 8.26 ± 0.26 mg/100 ml and 4.74 ± 0.14 mg/100 ml respectively (Fig. 2). The mean external balance of calcium as calculated from changes in dialysate calcium concentration was positive with 72 ± 36 mg ($n = 63$). The total elimination of phosphate in this group was 1019 ± 37 mg ($n = 64$). The time course of elimination is shown in Fig. 3.

Effects of a Dialysate Calcium of 8 mg/100 ml

During high calcium dialysis (dialysate calcium 7.94 ± 0.10 mg/100 ml, $n = 34$) predialysis serum calcium levels rose significantly ($p < 0.05$) from previously 9.0 mg/100 ml to 9.5 ± 0.19 mg ($n = 36$) in some instances frank hypercalcemia being observed (Fig. 1). At the end of high calcium dialysis serum calcium levels were 11.5 ± 0.16 mg/100 ml nearly all values being outside the normal range. Predialysis serum phosphate levels fell significantly ($p < 0.01$) from the previous mean value of 8.26 to 6.39 ± 0.38 mg/100 ml. At the end of dialysis the serum phosphate was 4.19 ± 0.17 mg/100 ml which is not significantly different from the value after "normal calcium" dialysis. The calcium phosphate product before dialysis decreased ($p < 0.01$) from 74.6 ± 2.1 on normal calcium dialysis to 60.9 ± 4.2 on high calcium dialysis (Fig. 4). Postdialysis calcium phosphate products on "normal calcium" and "high calcium" dialysis were 46.4 ± 1.4 and 48.0 ± 1.6 respectively which is statistically not different. The calcium uptake during high calcium dialysis increased ($p < 0.05$) by $\sim$170 mg to 240 ± 52 mg ($n = 34$), the total elimination of phosphate decreased by the same amount to 856 ± 52 mg ($n = 32$), the greatest difference being observed after the first two hours of dialysis (Fig. 3).

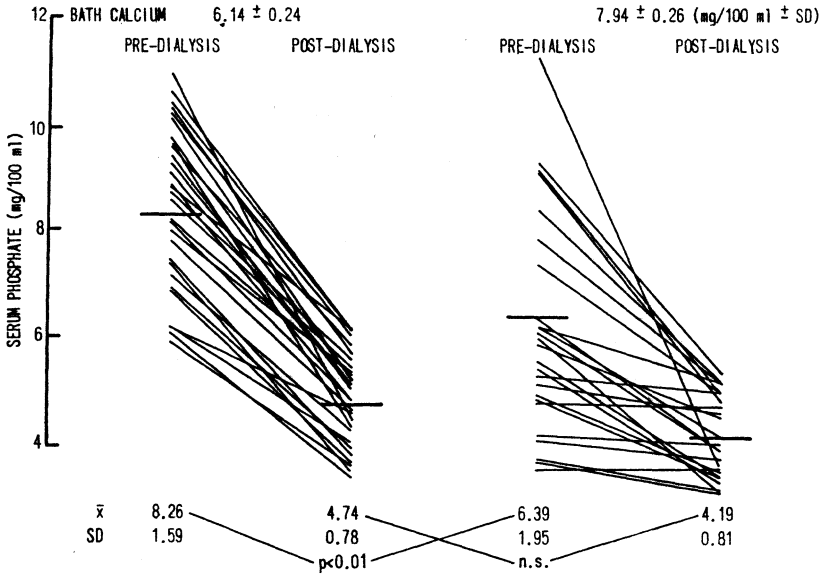

Fig. 2. Serum phosphate values before and after dialysis with a dialysate calcium of 6 mg/100 ml and 8 mg/100 ml

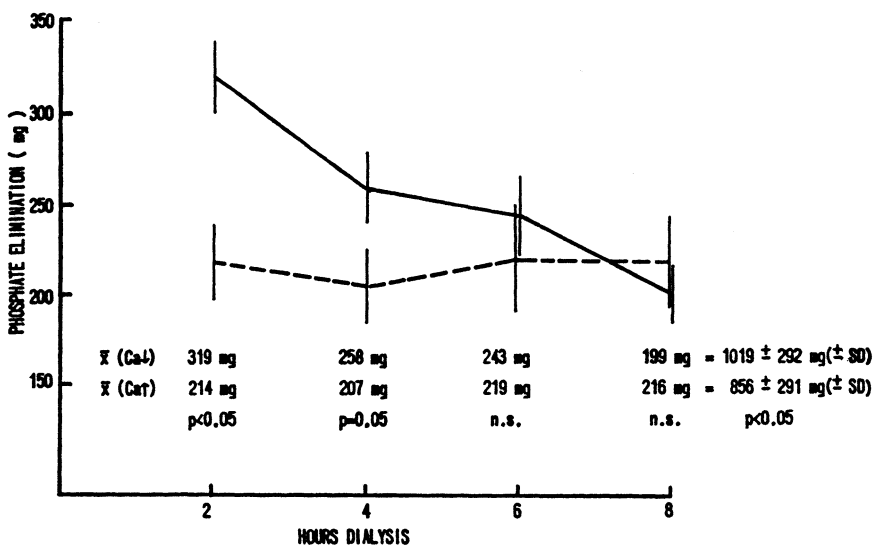

Fig. 3. Time course and total elimination of phosphate during dialysis with a dialysate calcium of 6 mg/100 ml and 8 mg/100 ml. — Bath calcium 6.14 mg/100 ml. --- Bath calcium 7.94 mg/100 ml

There was no significant difference in pre and post dialysis urea nitrogen, serum creatinine and bicarbonate between the two regimes.

Discussion

As the balance of calcium and phosphate is certainly one of the important criteria for the development or arrest of uremic osteodystrophy in hemodialysed patients [31], it is surprising that besides a few acute experiments [36] no comprehensive data have been published. Although it seems difficult to define calcium and phosphate balance during a single dialysis due to the error induced by small differences in dialysate calcium and phosphate concentrations and large dialysate volumes, our results and confidence limits indicate that the large number of specimens used in our study has supervened this difficulty.

Calcium Uptake during Dialysis

Our results confirm that during dialysis using a dialysate calcium of 6 mg/100 ml significant uptake of calcium does not take place, since the dialysate calcium equilibrates with the ultrafiltrable serum calcium [36].

For dialysis against a calcium of 8 mg/100 ml a positive calcium balance between 700 mg [20] and

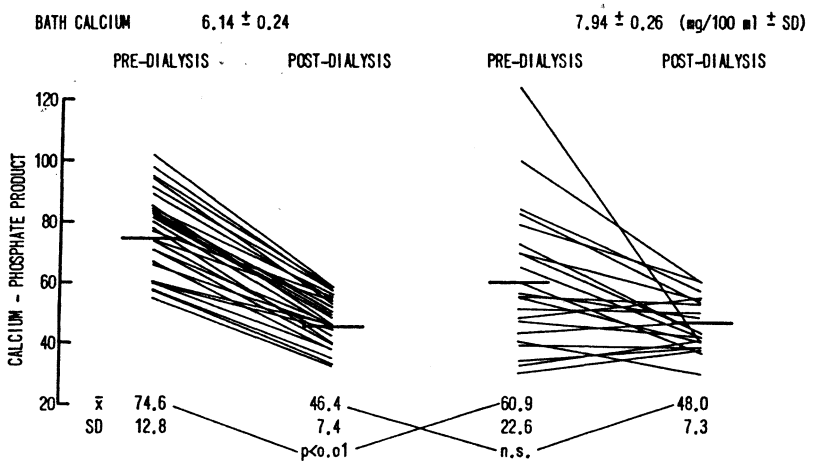

Fig. 4. Serum calcium — phosphate product before and after dialysis with a dialysate calcium of 6 mg/100 ml and 8 mg/100 ml

1100 mg [9] has been calculated by means of arteriovenous calcium concentration difference and simultaneous blood flow measurements. The mean figure of 250 mg derived by us by direct measurement is very much lower but agrees well with the isolated figure reported by Wing [36]. From the mean rise of serum calcium of 20 mg/l one can calculate that about a third of the infused calcium is in the intravascular pool at the end of dialysis. The remainder of 160 mg is apparently placed outside the extracellular pool since systemic ionised calcium does not increase during dialysis [32] although there is some controversy [21]. It is interesting to note that comparable infusions of calcium over shorter periods in the interdialytic period do not lead to comparable increases in serum calcium regardless as to whether bone disease is present or not [8, 12]. Although dialysis itself produces hypercalcemia independent of calcium balance due to increased calcium binding by plasma proteins [32] this effect does not explain the observed difference in increase of serum calcium between normal and high calcium dialysis. The hemodialysis itself, either the simultaneous removal of phosphate and/or the change in acid base balance might be responsible for the possibly impaired removal of infused calcium from the intravascular pool.

The figures derived by us are unlikely to have been caused by systematic errors. The errors most likely to occur (such as calcium uptake by the dialysis membrane, which has been found to be negligible [36], precipitation of calcium in the bath fluid or uptake of calcium by bacteria) would lead to overestimation of calcium uptake during dialysis.

Our results contrast with the experience of others [9] who found decreased serum calcium levels at the beginning of the next dialysis and explained this by suppression of parathyroid hormone secretion. There was no indication of excessive (and non suppressible) parathyroid hormone activity as the cause of hypercalcemia as judged by micromorphometric measurements in iliac crest biopsies obtained from the patients. Especially in the patient who developed persistent hypercalcemia on high calcium dialysis the percentage of Howship lacunae occupying the total trabecular surface was within the normal range [26] and well below the values reported by Ritz [24] for hemodialysed patients. The histological data go beyond the scope of this paper and will be presented elsewhere.

Taking the few reported gastrointestinal balance figures [3, 13, 30, 34] as representative, which indicate a faecal loss in excess of intake of at least 100 mg calcium per day, neither normal calcium dialysis nor high calcium dialysis would be able to compensate for faecal calcium losses in hemodialysed patients. The consistently suppressed parathyroidhormone levels [30] in patients on high calcium dialysis could either worsen faecal losses of calcium by diminshing intestinal absorption [22, 28] or improve balance by diminshing intestinal secretion [10]. Needless to say, gastrointestinal balance studies in patients on high calcium dialysis will be very interesting.

Phosphate Removal during Dialysis

Phosphate dialysance is reported to vary widely between 40 and 80 ml/min [16, 27, 37], comprehensive removal figures have not been reported. Fig. 1 shows the time course and total elimination on normal and high calcium dialyses comparatively. Whereas phosphate removal during normal calcium dialysis decreases significantly from the beginning to the end of dialysis, this effect is not demonstrable on high calcium dialysis. The total removal of 1100 mg phosphate on normal calcium dialysis exceeds the decrease of extracellular phosphate by a calculated 500 mg, an amount which has been derived from the intracellular pool. The diminished phosphate removal during high calcium dialysis is evident only during the first four hours of dialysis and is apparantly caused by the

lower initial serum phosphate values which were observed at the beginning of high calcium dialysis. Verberckmoes and coworkers [34] investigated the intestinal balance of phosphate in hemodialysed patients which were on a very similar regimen to ours (twice weekly 8 hours hemodialysis with a dialysate calcium of 5.2 mg/100 ml, twin coil kidney and none or very little oral aluminum hydroxide) and found a mean positive phosphate balance of 500 mg/day (range 320–770 mg). If these figures can be applied to our patients the amount of phosphate removed by twice weekly dialysis of 2.2 g could not compensate for the intestinal retention of 3.5 g phosphate. Thrice weekly hemodialysis and/or high oral doses of aluminumhydroxide could well induce negative phosphate balance [1].

Side Effects

The patients did not experience any subjective symptoms while on high calcium dialysis which is contrary to the finding of Wing [36] and in agreement with the experience of others [20]. One patient (No. 1) developed marked hypertension while on high calcium dialysis, the systolic and diastolic pressure increasing ($p < 0.001$) to 190 ± 13 mm Hg ($\pm$ SD, $n = 28$) from a previous value of 162 ± 17 mm Hg ($n = 39$) and to 98 ± 7 mm Hg from 80 ± 9 mm Hg respectively. The blood pressure returned to normal after being again on normal calcium dialysis for 3 months. The blood pressure remained unchanged throughout the study in the other two patients. Hypercalcemia may have profound effects on blood pressure [4] and seems to have been responsible for the development of hypertension in patient No. 1, since it reversed to normal after stopping high calcium dialysis. Since renal failure predisposes to a hypertensive response to acute hypercalcemia [35] it would seem the most likely explanation for the development of hypertension in the above patient. However, it would be difficult to exclude other causes of hypertension, for example the progress of natural history of renal hypertension [5].

It is interesting to note that hypertension developed in the patient with complete loss of excretory kidney function and not in the other two patients with some remaining kidney function and that the development of hypertension was not related to the development of persistent hypercalcemia.

Besides the hazard of arhythmias in patients receiving glucosides, the observed increase in serum calcium on high calcium dialysis does not necessarily appear to be a contraindication for the use of a high calcium concentration in the dialysate even in the presence of an increased content of skin calcium [15], since only the calcium phosphate product is apparently the critical point in the development of metastatic calcifications [29].

Conclusions

Although raising dialysate calcium above the ultrafiltrable serum calcium is effective to suppress parathyroid hormone levels [6], it does not induce the strongly positive calcium balance during dialysis which has been claimed in the past [9, 20]. Although this study shows that dialysis with a high calcium dialysate somewhat increases the retention of calcium and phosphorus during dialysis, it will have to be shown by gastrointestinal balance studies whether it improves overall calcium balance in hemodialysed patients or even to the contrary induces increased overall losses of calcium by diminishing intestinal absorption.

Disabling complications caused by renal osteodystrophy are rare [24] and cardiac complications due to hypertension and/or metastatic calcifications of the cardiovascular system still present the most life-threatening complication in hemodialysed patients. Before possible adverse effects of high calcium dialysis such as myocardial and vascular calcification [14, 33] and development of hypertension have been excluded we would disencourage the use of a dialysate Calcium concentration above that of ultrafiltrable serum calcium. Treatment other than high calcium dialysis, such as administration of active vitamin D metabolites [14], carefully adjusted elimination of phosphate and possibly rigorous elimination of diphosphonates [25] seems to be the more rational approach to the problem of renal osteodystrophy. Supported by the Österreichische Forschungsfonds, Grant No. 1971.

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