Peritoneal Phosphate Clearance: The Effect of Peritoneal Dialysis Modality and Peritoneal Transport Status

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Hyper- and hypophosphatemia are recognized risk factors for all-cause mortality in peritoneal dialysis (PD) patients. Recent changes have now focused PD solute clearance targets on urea clearance, rather than on larger solutes, including phosphate. We therefore studied peritoneal phosphate clearance in a cohort of PD patients to determine which factors were clinically relevant.

We reviewed results from 451 adult PD patients who were attending for their first assessment of peritoneal membrane function [31.2% treated by continuous ambulatory PD (CAPD); 24.2.%, by automated PD (APD); and 44.6% by APD with a daytime exchange]. Demographics, PD adequacy parameters, peritoneal phosphate clearance, and transport status were reviewed.

Of the study patients, 119 (26.4%) were hyperphosphatemic, and 59 (30.1%) were hypophosphatemic; 22.2% were fast transporters. Total daily peritoneal phosphate losses were greater for the hyperphosphatemic than for the hypophosphatemic patients [15 mg/dL (range: 10.5–18.6 mg/dL) vs. 25.7 mg/dL (range: 15.5–29.8 mg/dL), $p < 0.01$*], although peritoneal phosphate clearance was less [2.7 mL/min/1.73 m^2 (range: 1.6–4.1 mL/min/1.73 m^2) vs. 4.2 mL/min/1.73 m^2 (range: 2.1–4.1 mL/min/1.73 m^2),* $p < 0.001$*]. Peritoneal phosphate clearance was greater for faster compared with slower transporters [3.5 mL/min/1.73 m^2 (range: 2.5–4.5 mL/min/1.73 m^2) vs. 1.6 mL/min/1.73 m^2 (range: 1.1–2.2 mL/min/1.73 m^2),* $p < 0.05$*] and for patients treated either with APD plus a daytime exchange or with CAPD compared with APD alone [3.44 mL/min/1.73 m^2 (range: 2.3–5.0 mL/min/1.73 m^2) vs. 2.9 mL/min/1.73 m^2 (range: 1.5–4.4 mL/min/1.73 m^2) vs. 1.6 mL/min/1.73 m^2 (range: 1.1–2.4 mL/min/1.73 m^2,* $p < 0.001$*)]. On multivariate analysis, increased peritoneal clearance was associated with faster peritoneal transport status, younger age, lower serum albumin, and lower serum phosphate.*

Peritoneal phosphate clearance depends not only PD modality, but also patient factors, including peritoneal transport status and variables associated with inflammation.

Key words
Phosphate, residual renal function, transport, APD, CAPD

Introduction

Phosphate plays a key role in human physiology, and in health, phosphate balance is therefore tightly controlled; however, chronic kidney disease leads to phosphate retention. Observational cohort studies of both hemodialysis and peritoneal dialysis (PD) patients have consistently reported an association of increased serum phosphate concentration with all-cause and cardiovascular mortality (1,2). Although studies have failed to conclusively demonstrate causality, increased serum phosphate is an important factor involved in soft-tissue and vascular calcification, suggesting a potential mechanism affecting clinical outcomes (3).

Given an observed U-shape relationship between serum phosphate concentration and survival (4), national and international clinical guideline committees have made recommendations for serum phosphate targets in dialysis patients. Some guidelines suggest maintaining serum phosphate within the normal reference range; others advocate specific cut-offs (4–6).

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All guidelines provide advice about reducing dietary intake of foodstuffs with a high phosphate content, because dietetic and other interventions designed to increase patient education and improve compliance can lead to a reduction in serum phosphate concentration, certainly in the short term (7–9).

In terms of increasing phosphate clearance with dialysis, extending hemodialysis session times and, to a lesser extent, increasing hemodialysis session frequency have been reported to lower serum phosphate concentrations (10,11). Adding convective clearance with hemodiafiltration has also been reported to improve compliance with clinical guideline targets (12), to increase phosphate clearance (13), and to reduce phosphate binder requirements (14).

In PD patients, maintenance of adequate nutrition and restriction of dietary phosphate intake have to be balanced. Increased intra-abdominal pressure that places PD patients at greater risk of reflux esophagitis, coupled with the gastrointestinal side effects of phosphate binders, can lower dietary intake; additionally, peritoneal and urinary protein losses can vary.

Phosphate is located predominantly intracellularly in the body, and given that phosphate is charged, with a water shell, it diffuses more slowly than predicted based merely on its molecular weight (15). Compared with standard continuous ambulatory PD (CAPD), automated PD (APD), with its shortened intraperitoneal dwell times, is increasing in use. Traditionally, PD adequacy has included assessment of both urea and creatinine clearances, although some clinical guideline committees have dispensed with creatinine clearance, limiting targets to urea clearance alone (16,17). Because urea diffuses rapidly, peritoneal urea clearance cannot be used to estimate peritoneal phosphate clearance (18). Although APD uses shorter dwell times, not all studies have shown that phosphate clearance is less with APD than with CAPD (19). Similarly, some studies have reported that phosphate clearance in PD patients closely mirrors creatinine clearance (20), but others have found no such relationship (21). In view of the disparity in the reports, we reviewed peritoneal phosphate clearances in a large number of patients shortly after PD start.

Methods

Study population

We audited 451 adult PD outpatients attending their first assessment of peritoneal membrane function, 2–3 months after staring PD. No patient had experienced peritonitis or any acute medical illnesses before membrane testing. Patients were classified as slow, slow-average, fast-average, or fast peritoneal transporters according to their creatinine clearance as defined for the peritoneal equilibration test first described by Twardowski *et al.* (22).

Patients were being treated with CAPD or APD using lactate-based low-calcium (2.5 mEq/L) glucose and 7.5% icodextrin solutions (Baxter Health Care, Deerfield, IL, U.S.A.) as the standard of care. All patients underwent standard clinical and biologic peritoneal adequacy measurements by enzymatic creatinine assay (23) after a peritoneal equilibration test using 2.27% glucose solution (16,17). Bioimpedance to determine body composition and total body water (24) was performed using a standardized protocol (InBody 720: Biospace, Seoul, South Korea) (25,26). The protein equivalent of nitrogen appearance normalized to body weight was calculated using equations described by Bergström and colleagues (27). Clearances were normalized to a body surface area of 1.73 m^2 (16). Patient demographics were obtained from hospital computerized records, and comorbidity scores were assigned using the Stoke–Davies grading method (28).

This retrospective audit complied with National Health Service guidelines for audit and clinical service development.

Statistical analysis

Data are presented as means with standard deviation or as medians with interquartile range, according to their distribution. Differences between groups were evaluated using the chi-square test, the Student t-test, or the Mann–Whitney U-test, as appropriate. Correlations between variables were tested using Spearman rank correlation. A Kruskal–Wallis test, with appropriate *post hoc* correction, was used to compare quantitative variables in three groups. Multivariate analyses used logistic and multiple regression models (as appropriate) to analyze the determinants of peritoneal phosphate clearance (as a continuous variable), with log transformation of parametric data. A p value less than 0.05 was considered statistically significant. All analyses were performed using the Prism (version 6.0: GraphPad Software, San Diego, CA, U.S.A.), IBM SPSS Statistics (version 24.0: IBM, Armonk, NY, U.S.A.), or Analyse-it (version 3.0: Analyse-it Software, Leeds, U.K.) software applications.

Results

After their peritoneal equilibration test, patients were classified as having low, normal, or elevated serum phosphate according to U.K. Renal Association clinical guidelines (6). For most patients, serum phosphate values fell within the clinical guideline range (Table I). Fewer patients treated with CAPD had elevated serum phosphate levels. Weekly urinary urea clearances were higher in patients with normal and low serum phosphate, who were also faster peritoneal transporters. No differences in body composition, estimated dietary protein intake, or phosphate binder prescription were observed. Although residual urinary urea and creatinine clearances combined were greatest in patients with the lowest serum phosphate, their urinary phosphate clearance was lower than that in patients with the highest serum phosphate (Table I).

Peritoneal phosphate loss was significantly associated with peritoneal creatinine loss (Table II), and on Bland–Altman analysis, mean bias for phosphate clearance was –1.5 mL/min/1.73 m^2 (95% limits of agreement: –4.8 mL/min/1.73 m^2 to 5.1 mL/min/1.73 m^2). Although the weekly peritoneal creatinine clearance was similar in all groups, the daily peritoneal phosphate loss was greater for patients with the highest serum phosphate concentration (Table I). However, when adjusted for serum phosphate, peritoneal phosphate clearance was lower in patients with the highest serum phosphate concentration (Table I). Residual renal function was greater in those with low serum phosphate, but urinary phosphate losses were greater for those with the highest serum phosphate concentration (Table I).

Peritoneal creatinine clearance was lower in patients treated using APD with no daytime exchange [1.6 mL/min/1.73m^2 (range: 1.1–2.4 mL/min/1.73m^2)] than in those treated using CAPD [2.9 mL/min/1.73m^2 (range: 1.4–4.4 mL/min/1.73m^2), $p < 0.01$] or using APD with a daytime exchange [3.4 mL/min/1.73m^2 (range: 2.3–5.0 mL/min/1.73m^2), $p < 0.001$]. Similarly, peritoneal phosphate clearance was lowest in patients treated using APD with no daytime exchange (Figure 1). However, when values were adjusted for peritoneal creatinine clearance, no difference was evident [95.8% (range: 87%–105%) with CAPD vs. 82.5% (range: 73%–93.6%) with APD alone vs. 89.4% (range: 80.6%–97.6%) with APD plus a daytime exchange]. Peritoneal phosphate clearance increased with increasing peritoneal transport status (Figure 2).

A multivariable linear regression model showed that peritoneal phosphate clearance was positively associated with faster peritoneal membrane transport and negatively associated with patient age, serum albumin, and serum phosphate concentration (Table III).

The 4-hour dialysate-to-serum (D/S) phosphate ratio obtained during the peritoneal equilibration test was most strongly positively associated with D/S creatinine ($r = 0.8$, $p < 0.0001$), D/S protein ($r = 0.34$, $p < 0.001$), but also with increasing extracellular-to-total body water ratio ($r = 0.30$, $p < 0.001$) and comorbidity score ($r = 0.13$, $p = 0.008$). It was negatively associated with serum albumin ($r = –0.4$, $p < 0.001$), serum sodium ($r = –0.28$, $p < 0.001$), hemoglobin ($r = –0.18$, $p < 0.01$), and serum phosphate ($r = –0.16$, $p = 0.006$).

Discussion and conclusions

Although observational studies report that hyperphosphatemia is a risk factor for mortality in PD patients, so is hypophosphatemia (4,5). It would therefore appear important that patients achieve normal serum phosphate targets, as advised by clinical guidelines (6).

Phosphate balance is somewhat complex, depending upon the balance between dietary phosphate intake (patients are advised to adhere to dietary phosphate intake while at the same time maintaining a daily protein intake adequate to prevent malnutrition), compliance with phosphate binders, and phosphate clearance by the peritoneal, urinary, and fecal routes. Peritoneal phosphate clearance therefore potentially has an important role in determining serum phosphate control, and a greater understanding of peritoneal phosphate clearance could aid in the adjustment of PD prescriptions to improve phosphate removal with the aim of better achieving recommended serum phosphate targets.

Approximately 30% of our cohort of patients starting PD were hypophosphatemic; 26% were hyperphosphatemic. Compared with their hypophosphatemic counterparts, hyperphosphatemic patients had lower residual renal function. Although the differences were not statistically significant, patients in the highest serum phosphate group were slightly younger, had a slightly greater normalized rate of protein nitrogen appearance, had higher serum urea and creatinine concentrations, and had higher appendicular muscle mass—all of which would be expected to be associated with greater dietary phosphate intake. In that group, 75.2% were prescribed phosphate binders; 54% in

TABLE I Patient demographics

Variable	*Serum phosphate group*[a]		
	<3.4 mg/dL	*3.4–5.26 mg/dL*	*>5.26 mg/dL*
Patients [*n* (%)]	59 (30.1)	273 (60.5)	119 (26.4)
Men	37 (45.8)	156 (57.1)	74 (66.4)
White	22 (37.3)	129 (47.3)	65 (54.6)
With diabetes	27 (45.8)	112 (41.0)	36 (30.3)
Mean age (years)	60±19	58±17	54±17
Dialysis vintage (months)			
Median	2	2	2
Range	2–3	2–3	2–3
Dialysis modality [*n* (%)]			
CAPD	15 (25.4)	63 (23.1)[b]	18 (15.1)
APD	11 (18.6)	56 (20.5)	42 (35.3)
CCPD	33 (55.9)	154 (56.4)	59 (49.6)
Daily icodextrin use (L)			
Median	1.1	1.2[c]	1.0
Range	0.7–2.0	0.7–2.0	0–1.5
Daily 2.27% dextrose use (L)			
Median	0	0	0
Range	0–4.4	0–3.2	0–4.6
Weekly Kt/V_{urea}			
Urinary			
Median	1.59[c]	1.46[d]	0.96
Range	1.01–2.34	0.9–2.1	0.57–1.64
Peritoneal			
Median	1.21	1.1	1.09
Range	0.98–1.49	0.82–1.38	0.92–1.37
Weekly creatine clearance (L)			
Urinary			
Median	64.6	58.5	41.1
Range	33.8–89	33.1–87	23.8–61.3
Peritoneal			
Median	35.6	33.9	29.3
Range	19.5–47.6	22.1–44	21.3–42.2
Daily peritoneal phosphate loss (mg)			
Median	15.5[d]	17.4[c]	21.7
Range	10.5–18.6	12.4–23.9	15.5–29.8
Mean 4-hour D/S			
Creatinine	0.74±0.14[d]	0.73±0.13[d]	0.71±0.15
Sodium	0.95±0.14[b]	0.94±0.13	0.93±0.04
Phosphate	0.73±0.14	0.64±0.14	0.61±0.17
Mean 4-hour D/D_0 glucose	0.33±0.09	0.33±0.08	0.34±0.10
Body composition			
Weight (kg)	70.1±13.5	74.3±15.7	73.3±18.6
Appendicular muscle mass (kg)	20.9±5.4	21.6±5.9	22.4±7.3
Body cell mass (kg)	30.9±7.3	33.2±9.5	33.5±7.8
Body surface area (m^2)	1.81±0.21	1.86±0.23	1.85±0.26
ECW/TBW	39.9±1.4	39.6±1.4	39.6±1.6

TABLE I Continued

Variable	*Serum phosphate group*[a]		
	<3.4 mg/dL	*3.4–5.26 mg/dL*	*>5.26 mg/dL*
Mean daily nPNA (g/kg)	0.88±0.23	0.90±0.25	0.95±0.26
Laboratory parameters			
Mean hemoglobin (g/dL)	11.1±1.7	11.2±1.6[b]	10.8±1.4
Mean serum albumin (g/L)	35±6	37±5	37±5
Mean serum urea (mg/dL)	95.2±32.5[d]	108.4±31.3[d]	121.7±33.1
Serum creatinine (mg/dL)			
Median	4.93[d]	5.80[d]	7.86
Range	4.15–6.74	4.76–7.61	6.06–9.89
C-Reactive protein (mg/L)			
Median	4	4	4
Range	1–13	1–8	1–12
Mean serum calcium (mg/dL)	9.36±0.6	9.32±0.6	9.28±0.9
Parathyroid hormone (pmol/L)			
Median	18.2[b]	25.3[b]	31.1
Range	10–9-35.5	14.7–39.6	13.8–46.9
Mean serum phosphate (mg/dL)	2.98±0.43[d]	4.31±0.53[d]	5.92±0.9[d]
Medications			
Phosphate binders [*n* (%)]	32 (54.2)	182 (66.7)	82 (75.2)
Weekly alfacalcidol (μg)			
Median	1.0	0.75	0.75
Range	0–1.75	0–3.0	0–3.5
Clearances (mL/min/1.73 m^2)			
Combined urea and creatinine			
Median	6.3[c]	5.5[d]	3.6
Range	3.7–8.7	3.2–7.8	2.1–5.7
Urinary phosphate			
Median	4.1[c]	3.8	2.7
Range	2.1–4.1	2.4–5.4	1.6–4.1

[a] Divided according to U.K. clinical practice guidelines for serum phosphate (6), with dialysis variables calculated based on 4-hour peritoneal equilibration test dialysate-to-serum (D/S) ratios.
[b] $p<0.05$ compared with the highest tertile (>5.26 mg/dL).
[c] $p<0.01$ compared with the highest tertile (>5.26 mg/dL).
[d] $p<0.001$ compared with the highest tertile (>5.26 mg/dL).
CAPD = continuous ambulatory peritoneal dialysis; APD = automated peritoneal dialysis; CCPD = continuous cycling peritoneal dialysis; D/S = dialysate-to-serum concentration ratio; D/D_0 = final-to-initial dialysate concentration ratio; ECW = extracellular water; TBW = total body water; nPNA = normalized protein nitrogen appearance.

the low-phosphate group were prescribed binders. As was therefore expected, total peritoneal phosphate losses were greatest in patients with the highest serum phosphate; however, when adjustments for serum phosphate and body surface area were applied, peritoneal phosphate clearance was then actually greater for the group with the lowest serum phosphate.

Results in earlier studies of whether peritoneal transport status has an effect on phosphate clearance are discordant, with some studies reporting no effect (29) and others showing an effect (30). We observed that increasing peritoneal transport status was associated with increasing peritoneal phosphate clearance. We observed increased clearance with lower serum phosphate—an apparent paradox that could potentially reflect an association between lower serum phosphate and an inflammatory state. Faster peritoneal transport status has been found with inflammatory states

TABLE II Univariate associations[a] with daily peritoneal phosphate losses

Variable	r *Value*	p *Value*
Weekly creatinine clearance (L/1.73 m^2)	0.74	<0.0001
4-Hour D/S phosphate	0.55	<0.0001
Weekly peritoneal Kt/V_{urea}	0.48	<0.0001
Daily icodextrin use (L)	0.40	<0.0001
4-Hour D/S sodium	0.40	<0.0001
Daily 2.27% dextrose use (L)	0.37	<0.0001
4-Hour D/S creatinine	0.37	<0.0001
4-Hour D/D_0 glucose	–0.36	<0.0001
Serum albumin (g/L)	–0.30	<0.0001
Appendicular muscle mass index (kg/m^2)	0.30	<0.0001
Serum phosphate (mg/dL)	0.30	<0.0001
Serum creatinine (mg/dL)	0.28	<0.0001
24-Hour peritoneal ultrafiltration (L)	0.28	<0.0001
24-Hour urinary phosphate (mg)	–0.23	<0.0001
Extracellular water (L)	0.26	<0.0001
Intracellular water (L)	0.23	<0.0001
Body surface area (m^2)	0.19	<0.0001
Hemoglobin (g/L)	–0.19	<0.0001
Extracellular-to-total body water ratio	0.15	0.002
C-Reactive protein (mg/L)	0.12	0.010
Body mass index (kg/m^2)	0.10	0.035

[a] By Spearman rank correlation, based on the 4-hour dialysate-to-serum (D/S) concentration ratio in a peritoneal dialysis equilibration test.

D/S = dialysate-to-serum concentration ratio; D/D_0 = final-to-initial dialysate concentration ratio.

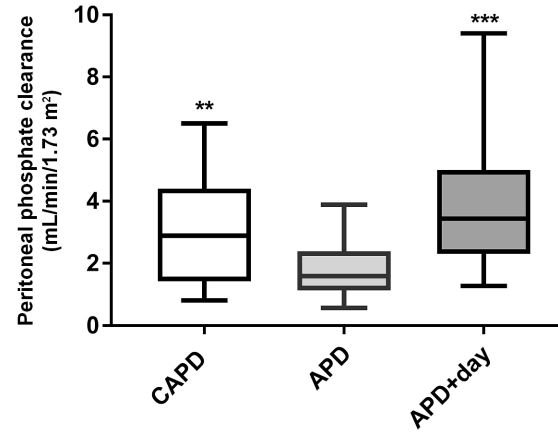

FIGURE 1 Peritoneal phosphate clearance adjusted for body surface area (median, interquartile range, and 95% confidence limits). CAPD = patients treated by continuous ambulatory peritoneal dialysis; APD = patients treated by automated cycler peritoneal dialysis with no daytime exchange; APD+day = patients treated by APD with a daytime exchange. $^{**}p < 0.015$ and $^{***}p < 0.01$ compared with APD.

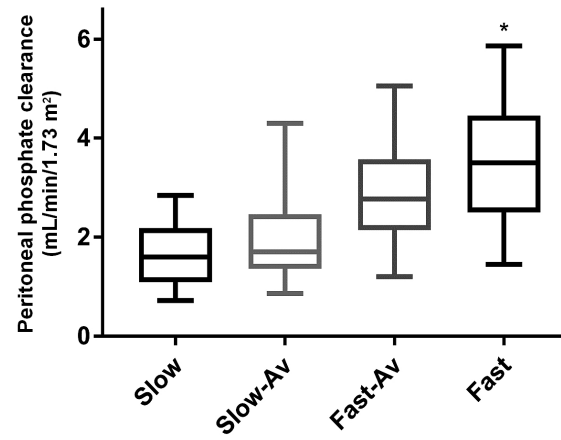

FIGURE 2 Peritoneal phosphate clearance adjusted for body surface area (median, interquartile range, and 95% confidence limits), by peritoneal membrane transport status (22). Av = average. $^{*}p < 0.05$ compared with slow transport.

(31,32). The potential presence of an inflammatory state is supported by the trend toward lower serum albumin, higher extracellular-to-total body water ratio (33,34), higher D/S creatinine and D/S protein (35), and greater comorbidity in the group with the lowest serum phosphate.

We observed that peritoneal phosphate clearance was lower for patients treated with the shorter dwell times associated with APD cycles than for their counterparts treated with CAPD or APD plus a daytime exchange, which accords with some earlier reports (20). Similarly, we observed a correlation between peritoneal phosphate and creatinine transport (20), although after adjustment for peritoneal creatinine clearance, we found no difference in phosphate clearance between the various PD modalities. The latter observation suggests that the differences between study reports about whether peritoneal phosphate clearance is lower with APD treatment depend on the characteristics of the particular patient population studied (20,21).

In Europe and North America compared with Asia, a greater proportion of PD patients are now treated with APD cyclers. Patient preference and lifestyle

TABLE III Multivariable linear regression model for peritoneal phosphate clearance[a]

Variable	*β*	*SE*	*Standardized β*	*t*	*95% CI*	p *Value*
Phosphate	–1.22	0.30	–0.20	–4.0	–1.82 to –0.62	0.000
Age	–0.19	0.01	–0.13	–2.1	–0.03 to –0.01	0.009
Albumin	–0.09	0.03	–0.17	–3.1	–0.42 to –0.329	0.002
Creatinine	2.17	0.95	0.12	2.3	0.30 to 4.0	0.023

[a] Based on the 4-hour dialysate-to-serum concentration ratios of creatinine, phosphate, and albumin in a peritoneal equilibration test. Tolerance > 0.9; variance inflation factor < 1.2.
SE = standard error; CI = confidence interval.

are the most common reasons that patients decide to choose APD rather than CAPD. As a result, many slow-average and slow transporters are now treated with APD cyclers. Once residual renal function is lost, peritoneal clearance of phosphate becomes more important in determining phosphate balance. Peritoneal phosphate loss is greater with the longer dwell times of CAPD and APD plus a daytime exchange, and also with faster peritoneal transport status.

Although it has been suggested that creatinine can be used as a surrogate marker of phosphate transport, we found that peritoneal phosphate and creatinine clearances differed, suggesting that measurement of creatinine alone cannot replace direct measurements of peritoneal phosphate transport status and clearance.

As is the case with any observational data, future studies are required to determine whether increasing phosphate removal will result in improved patient outcomes.

Disclosures

I understand that *Advances in Peritoneal Dialysis* requires disclosure of any conflicts of interest, and I declare that I have no conflicts to disclose.

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