RATE OF DECLINE OF RESIDUAL KIDNEY FUNCTION BEFORE AND AFTER THE START OF PERITONEAL DIALYSIS

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◆ **Background:** There is a paucity of information on whether peritoneal dialysis (PD) slows the decline of residual kidney function (RKF) compared to the natural slope of RKF decline prior to dialysis start. Our aim was to analyze the RKF decline before and after initiating PD, and to determine the principal factors affecting this decline during the PD period.

◆ **Methods:** We determined individual glomerular filtration rates (GFR) for approximately 12 months before and after PD in 77 new PD patients in a large academic medical center (2008 – 2012). The GFR was estimated by the Modification of Diet in Renal Disease (MDRD) equation in the predialysis period and by averaging 24-hour urine creatinine and urea clearances in the PD period. The rate of RKF decline was calculated using unadjusted linear regression analysis. Wilcoxon signed rank test was used to compare RKF decline before and after PD initiation. Multivariate linear regression was used to identify independent risk factors for RKF decline in the PD phase.

◆ **Results:** A significantly slower mean rate of RKF decline was observed in the PD period compared with the predialysis period (-0.21 ± 0.30 vs -0.59 ± 0.55 mL/min/1.73 m²/month, $p < 0.01$). Higher baseline RKF, higher serum phosphate, and older age were independently associated with faster decline of RKF (all $p < 0.01$).

◆ **Conclusions:** In patients with advanced chronic kidney disease, initiating PD was associated with a slower rate of RKF decline compared to the rate in the predialysis period.

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The presence of residual kidney function (RKF) has important clinical consequences in dialysis patients because it is associated with prolonged survival and better quality of life (1–3). Several studies have shown that the rate of RKF decline is slower in patients on peritoneal dialysis (PD) than in patients on hemodialysis (HD) (4–6). However, there is a paucity of information on whether PD itself affects the rate of RKF decline

compared to the natural slope of kidney function decline prior to dialysis start. Clinically, we have observed that many PD patients maintain urine volume and GFR for a long time on this therapy. In addition, Berlanga *et al.* reported slower rates of RKF decline in patients commenced on PD compared to the rate in the predialysis period (7).

The primary objective of this retrospective study was to explore the course of the RKF decline in the 12 months before and after initiating PD, using prospectively collected data from a large academic medical center between 2008 and 2012. The secondary objective of this study was to examine the principal risk factors influencing RKF decline during the PD period.

STUDY POPULATION AND METHODS

STUDY POPULATION

We conducted a single-center, retrospective study of prospectively collected data at the Home Peritoneal Dialysis Unit of the University Health Network – Toronto General Hospital, Toronto, Canada. This study was approved by the University Health Network's Research Ethics Board. All patients starting PD between January 1, 2008, and December 31, 2012, were eligible for the study. Exclusion criteria were: (i) age < 18 years old; (ii) less than 6 months of follow-up before or after initiating PD; (iii) fewer than 3 measurements of RKF in the 12 months before or after initiating PD; (iv) anuria at the start of PD; (v) prior kidney replacement therapy (i.e. a patient who transitioned to PD from a failing kidney allograft or from chronic HD); (vi) incomplete data for study.

Data collected included patient demographics such as age at the start of PD, sex, height, weight, underlying cause of end-stage kidney disease (ESKD), comorbid conditions (e.g. diabetes mellitus, hypertension), PD modality (continuous cyclic PD [CCPD], continuous ambulatory PD [CAPD], nocturnal intermittent PD [NIPD]), type of PD fluid (with or without icodextrin). Laboratory data collected at baseline (initiation of PD) included hemoglobin, albumin, corrected calcium, phosphorus, intact parathyroid hormone (iPTH) level, C-reactive protein (CRP), lipid profile and peritoneal equilibration test values (PET, corrected dialysate/plasma creatinine ratio) using

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standard methodology. Additional clinical data included blood pressure measurements and medications (e.g. angiotensin-converting enzyme inhibitors [ACEI]/angiotensin receptor blockers [ARB], diuretics) and episodes of peritonitis.

GLOMERULAR FILTRATION RATE (GFR)

In the predialysis period, the GFR was estimated by the Modification of Diet in Renal Disease (MDRD) equation (8). In the PD period, GFR was calculated as the average of creatinine clearance (Ccr) and urea clearance (C_{BUN}) from a 24-hour urine collection and corrected for the body surface area ($\text{mL}/\text{min}/1.73 \text{ m}^2$) (9,10). All patients had 3 to 6 measurements of GFR at intervals of 1 – 3 months in the 6 – 12 months before dialysis or in the PD period respectively.

STATISTICAL ANALYSIS

Results are expressed as proportions (percentages) for categorical variables, mean $\pm$ standard deviation (SD) for continuous normally distributed variables, and median (range) for continuous non-normally distributed variables.

A simple (unadjusted) linear regression analysis was performed in each patient between the calculated GFR and time in the period of 12 months before and 12 months after the start of PD. The regression coefficient of time against GFR was used to give an estimated rate of GFR decline for each individual patient in each period. The linearity of the individual GFR slopes was accepted as an adequate description of the rate of GFR decline. Wilcoxon signed rank test was used to compare the rate of GFR decline before and after PD for each individual.

Simple correlation analysis was used to evaluate the relationship between the rate of RKF decline and exploratory variables, including age, sex, diabetes mellitus, baseline blood pressure, hemoglobin, serum albumin, calcium, phosphate, C-reactive protein, peritoneal equilibration test, PD modality, and use of ACEI/ARB during the PD period. Multivariate linear regression was used to determine the independent risk factors for RKF decline during the PD period using the significant factors ($p < 0.05$) found on univariate analyses and factors that were deemed to be clinically plausible. SPSS version 11.5 software was used.

RESULTS

PATIENT CHARACTERISTICS

Seventy-seven patients who started PD between January 2008 and December 2012 fit the inclusion and exclusion criteria and were studied. Characteristics of those patients at the start of PD are shown in Table 1.

RATES OF RKF DECLINE

The rate of RKF decline was estimated for all patients both in the predialysis period and during PD. A significantly

TABLE 1
Demographic and Clinical Characteristics of
Study Population at Start of PD

Characteristics	Values
Number of patients (n)	77
Age (years; mean $\pm$ SD)	63.1 $\pm$ 15.1
Male/female (male %)	42/35(54.5)
Etiology of end stage kidney failure (%)	
Diabetic nephropathy	24.7
Hypertension	13.0
Glomerulonephritis	40.3
Tubulointerstitial nephritis	3.9
Other	14.2
Unknown	3.9
Comorbidity	
Diabetes mellitus (%)	37.7
Hypertension (%)	92.2
BMI (kg/m^2)	28.1 $\pm$ 7.0
Systolic blood pressure (mmHg; mean $\pm$ SD)	134.7 $\pm$ 16.6
Diastolic blood pressure (mmHg; mean $\pm$ SD)	75.5 $\pm$ 11.2
Hemoglobin (g/L)	113.8 $\pm$ 11.7
Serum albumin (g/L)	36.2 $\pm$ 3.7
Serum corrected calcium (mmol/L)	2.36 $\pm$ 0.19
Serum phosphate (mmol/L)	1.69 $\pm$ 0.51
Serum CRP (mg/L)	3 (1–82)
Serum PTH (pmol/L)	30 (1–179)
Serum LDL-C (mmol/L)	2.45 $\pm$ 0.90
Serum HDL-C (mmol/L)	1.12 $\pm$ 0.36
Serum triglycerides (mmol/L)	1.84 $\pm$ 1.14
Baseline RKF initiating PD ($\text{mL}/\text{min}/1.73 \text{ m}^2$)	7.4 $\pm$ 3.2
PET (dialysate/plasma creatinine ratio at 4 hours)	0.69 $\pm$ 0.12
PD modality (automated PD %)	52
Type of dialysate (icodextrin %)	27.3
Medications: diuretics (%)	90.9
ACEI/ARB (%)	76.6
Peritonitis in the 1 st year of PD (%)	17.1

PD = peritoneal dialysis; SD = standard deviation; BMI = body mass index; CRP = C-reactive protein; PTH = parathyroid hormone; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; RKF = residual kidney function; PET = peritoneal equilibration test; ACEI/ARB = angiotensin converting enzyme inhibitor/angiotensin receptor blocker.

slower mean rate of RKF decline was observed in the PD period compared with the predialysis period (-0.21 ± 0.30 vs $-0.59 \pm 0.55 \text{ mL}/\text{min}/1.73 \text{ m}^2/\text{month}$, $Z = -5.532$, $p < 0.01$).

INDEPENDENT RISK FACTORS INFLUENCING THE RKF DECLINE DURING THE PD PERIOD

Simple correlation analysis showed that the higher the baseline hemoglobin, the slower the rate of RKF decline ($p < 0.05$). On the other hand, the higher the baseline serum phosphate level ($p < 0.01$) and the higher the baseline RKF at initiation of PD, the faster the rate of RKF decline ($p < 0.05$).

Although not statistically significant, the higher the D/P creatinine, the slower the rate of RKF decline ($p = 0.05$). There were no other clinical associations with the rate of RKF decline. These results are listed in Table 2.

Multiple linear regression analysis found that the independent risk factors for the rate of RKF decline in the first year of PD were age, sex, diabetes mellitus, hemoglobin, serum phosphate, PET value and baseline RKF. Of these factors, higher baseline RKF, higher serum phosphate and older age were independently associated with faster decline of RKF (all $p < 0.01$). See Table 3.

DISCUSSION

Residual kidney function has been associated with longer survival and a better quality of life in dialysis patients (1–3). Many studies have shown that the decline of RKF in HD patients is faster than the decline in PD patients (4–6). However, few studies have focused on the effect of the PD procedure itself on RKF decline. Our study of 77 incident PD patients showed that the mean rate of RKF decline was slower in the first year of PD compared with that in the year prior to starting dialysis. This finding is consistent with the only other study performed solely in PD patients more than a decade ago in a smaller cohort by Berlanga *et al.*, who also found that the rate of RKF decline was faster in every patient during the predialysis period than during PD (7). The NECOSAD group also examined rates of decline in both HD and PD patients (11). The rate of decline in the predialysis period was nearly identical to the findings in this study, and the PD subcohort experienced an even slower rate of decline in the ensuing months (-0.11 mL/min/ 1.73 m²/month) (11).

Several mechanisms could account for the beneficial effect of PD on the progression of CKD. Firstly, PD may off-load the hyperfiltration of the remnant kidneys by contributing to solute and fluid removal in a gentle, continuous manner. This is akin to the nephroprotective effect of ACEI/ARBs by reducing glomerular capillary hypertension and hyperfiltration (12–14). Secondly, PD may slow the progression of CKD through removal of excess fluid and uremic toxins. Peritoneal dialysis may help to clear nephrotoxic solutes that may be contributing to the loss of RKF. This is supported by the experimental data in rats (15), where an oral charcoal adsorbent (AST-120), theoretically chelating uremic toxins, ameliorated kidney injury. The benefit may also be related to correction of metabolic acidosis leading to slower CKD progression (16–18) by mitigating the tubulointerstitial injury induced by acidosis through ammonia-induced complement activation and endothelin and aldosterone activation (19,20). Lastly, PD may have a beneficial hemodynamic effect in removing excess total body water without large hemodynamic fluctuations in contrast to the predialysis phase, where wide fluctuations might occur with aggressive diuretic bolus prescriptions. As such, PD may be better at decreasing the risk of ischemic injury due to hypotension, thus facilitating the adaptation of surviving nephrons to maintain glomerular filtration (21).

TABLE 2
Correlation of Rate of RKF Decline and Other Variables in PD Period

	R (Pearson or Spearman correlation)	P
Age (years)	-0.038	0.7
Gender (male/female)	0.088 ^a	0.4
Diabetes mellitus/non-diabetic mellitus	0.099 ^a	0.4
With hypertension/without hypertension	0.036 ^a	0.8
BMI (kg/m ²)	0.013	0.9
Systolic blood pressure (mmHg)	-0.082	0.5
Diastolic blood pressure (mmHg)	0.104	0.4
Hemoglobin (g/L)	0.275	0.02 ^b
Serum albumin (g/L)	-0.072	0.5
Serum corrected calcium (mmol/L)	0.042	0.7
Serum phosphate (mmol/L)	-0.35	0.002 ^c
Serum CRP (mg/L)	-0.136 ^a	0.3
Serum PTH (pmol/L)	-0.127 ^a	0.3
Serum LDL-C (mmol/L)	-0.001	0.9
Serum HDL-C (mmol/L)	0.055	0.7
Serum triglycerides (mmol/L)	-0.042	0.7
PET (dialysate/plasma creatinine ratio at 4 hours)	0.254	0.05
Baseline RKF initiating PD	-0.254	0.04 ^b
PD modality (APD/CAPD)	0.114 ^a	0.3
with icodextrin/without icodextrin	0.045 ^a	0.7
with diuretics /without diuretics	0.047 ^a	0.7
with ACEI/ARB /without ACEI/ARB	0.028 ^a	0.8
with peritonitis/without peritonitis in the 1 st year of PD	-0.057 ^a	0.6

RKF = residual kidney function; PD = peritoneal dialysis; BMI = body mass index; CRP = C-reactive protein; PTH = parathyroid hormone; LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; PET = peritoneal equilibration test; ACEI/ARB = angiotensin converting enzyme inhibitor/angiotensin receptor blocker; APD = automatic PD; CAPD = continuous ambulatory PD.

^a Spearman correlation.

^b $p < 0.05$.

^c $p < 0.01$.

In terms of risk factors for accelerated decline in RKF during PD, the present study showed that older age was related to a faster rate of RKF decline. This finding has been reported previously (4, 22). It is possible that age-related renovascular changes make older people more vulnerable to shifts in water balance and uremic toxins.

Higher baseline RKF at the start of PD was another identified predictor of more rapid RKF decline during the PD period in this study, similar to other studies (22,23). A nonlinear decline in RKF in PD patients has been described (24). This phenomenon is somewhat akin to the progressive slowing of the rate of RKF decline with increasing time on PD noted by Lysaght *et al.* (25). This nonlinear decline is a potential source of confounding in our study. Our study found that there was a faster decline of RKF before PD and then the decline slowed

TABLE 3
Multiple Linear Regression Analysis of Rate of RKF Decrease in Year 1 of PD Period

	For each 1 mL/min/1.73 m ² /month decline of RKF	B (Unstandardized coefficients)	Beta (Standardized coefficients)	t	P
Constant		0.936		3.878	<0.001
Baseline RKF (mL/min/1.73 m ² /month)	1.362	-0.047	-0.481	-4.215	<0.001
Phosphate (mmol/L)	0.244	-0.262	-0.475	-3.836	<0.001
Age (years)	10.6	-0.006	-0.304	-2.478	0.02

R²=0.622, adjust 0.348; F=10.093, p<0.01

RKF = residual kidney function; PD = peritoneal dialysis.

during the PD period, which we attribute to the PD. An alternative hypothesis is that patients who started PD (at a lower RKF than in the predialysis period) were just showing the natural lessening of the slope of decline during the PD phase that has more to do with an intrinsic dual phase (faster, then slower) of decline, rather than any particular nephroprotective effect of the PD itself.

We found an association of higher serum phosphate level at baseline with a more rapid rate of RKF loss in the PD period. Contrary to our results, Noordzij *et al.* did not find disordered mineral metabolism to be associated with the rate of RKF decline in dialysis (PD and HD) patients (26). Given that higher baseline RKF is associated with a higher rate of RKF decline (23) and better phosphate clearance and lower serum phosphate, the positive correlation of phosphate and RKF decline cannot be explained by more severe kidney failure at the start of PD. In pre-dialysis patients, Voormolen also found that higher serum phosphate was an independent risk factor for a more rapid decline in renal function (27). In an animal study, rats fed with a diet rich in phosphate showed a faster decline in kidney function compared with rats fed with a low phosphate diet (28). The mechanism may relate to enhanced extracellular and intracellular phosphate concentrations that may engender endothelial dysfunction and oxidative stress (19), 2 potential risk factors for progression of kidney injury.

A more clinical explanation could be that patients who were more non compliant with dietary phosphate control and phosphate binding were also non-compliant with other nephroprotective maneuvers that led to a more rapid decline in kidney function.

In addition, we did not find that PD sub-modality was a risk factor for loss of RKF in the first year of PD. This agrees with most (23,29), but not all (30–32), studies. The use of icodextrin also did not influence RKF. Once again, this is in agreement with most (33,34), but not all (35), studies using this dialysis fluid.

Previous studies have associated the use of ACEI/ARB with a slower rate of RKF decline (36–39). We did not find this. About 3/4 of our PD patients were taking ACEI/ARB and only those who had a contraindication or had suffered side effects of ACEI/ARB were not prescribed these classes of drugs.

Perhaps this high use accounted for our inability to detect an effect.

The role of diuretics in the preservation of RKF in PD patients is unclear. Liao *et al.* found that the use of diuretics was independently associated with a faster decline of RKF in PD patients in Taiwan (40). However, our results and those of Medcalf reported that furosemide had no effect on RKF despite maintaining diuresis (41).

Contrary to the previous studies (42,43), we did not observe that peritonitis in the 1st year of PD was a risk factor for RKF decline. Perhaps the observation period was too short, and the number of patients experiencing peritonitis too small to observe an effect.

The main limitations of our study include its retrospective cohort design and single-center experience. We excluded patients on PD less than 6 months because of insufficient data to accurately determine the rate of RKF decline. Our comparison of RKF decline was not ideal due to differing GFR baselines 6–12 months before and at the time of PD initiation. A more ideal study would compare the RKF decline between 2 groups with the same baseline RKF, one not starting dialysis, the other starting PD in a randomized controlled trial design. As pointed out in a recent review (44), these kinds of studies are confounded by lead-time bias determined by when they start PD and whether the start was precipitated by an episode of acute or chronic kidney injury. Two different calculations were used for RKF in the predialysis and the PD periods, but the slope was measured only within the same calculation methods. Finally, there is a risk that the change of slope represented regression to the mean, but given the reproducible finding among the whole cohort of patients, we think that this is unlikely, especially when there is a physiologic explanation for our findings.

CONCLUSIONS

In patients with advanced chronic kidney disease, initiating PD was associated with a slower mean rate of RKF decline compared to the rate of RKF decline in the predialysis period. Higher RKF, higher serum phosphate and older age at PD initiation are independent factors for a faster decline of RKF in the PD phase.

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DISCLOSURES

The authors have no financial conflicts of interest to declare.

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