Effect of Peritoneal Transport Characteristics on Clinical Outcome in Nondiabetic and Diabetic Nephropathy Patients with Peritoneal Dialysis

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Introduction. This study aimed to investigate the influence of peritoneal transport characteristics on clinical outcome in nondiabetic and diabetic nephropathy peritoneal dialysis (PD) patients.
Materials and Methods. All 112 patients were from the PD Center. Peritoneal transport characteristic was assessed by peritoneal equilibration test. The patients were divided into 2 groups of high-transport group (HT) and non-high-transport group (non-HT) and followed-up till December 31st, 2010. The primary outcomes were all-cause death and technique failure.
Results. The patients were followed-up for 65.9 ± 23.9 months. Diabetic nephropathy patients with HT had a higher all-cause mortality (P = .04) and technique failure (P = .04) than those with non-HT. There were no differences in outcomes between HT and non-HT subgroups without diabetic nephropathy. Cox regression demonrtrated that high peritoneal transport (HR, 2.369; 95% CI, 1.056 to 5.311), diabetic nephropathy (HR, 2.499; 95% CI, 1.134 to 5.508), age (HR, 1.081; 95% CI, 1.032 to 1.133), and peritoneal creatinine clearance (HR, 0.962; 95% CI, 0.929 to 0.997) independently predicted all-cause mortality in continuous ambulatory PD patients. Moreover, high peritoneal transport (HR, 2.299; 95% CI, 1.079 to 4.899) and age (HR, 1.070; 95% CI, 1.026 to 1.116) predicted technique failure in continuous ambulatory PD patients.
Conclusions. Diabetic nephropathy PD patients with HT had a higher all-cause mortality and technique failure than those with non-HT, but we did not find the correlation between peritoneal transport and outcome in nondiabetic patients. The peritoneal transport was an independent predictor for outcomes in continuous ambulatory PD patients.

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INTRODUCTION

Peritoneal dialysis (PD) has been recognized as an important form of renal replacement therapy for patients with end-stage renal disease (ESRD). However, the mortality of dialysis patients remains high and the predicting factors for PD patient mortality remain to be determined.

Recently, the role of peritoneal solute transport has been extensively studied in regard to patient outcome. To our knowledge, the role of peritoneal transport characteristics on mortality and technique failure of PD patients remains controversial. The multicenter CANUSA study[1], the single-center Stoke PD study,[2,3] and some other studies[4,5] showed that a high peritoneal transport status was associated with higher mortality and technique failure in PD

patients. However, some studies failed to find a relationship between peritoneal transport and patient outcome.[6,7] The ADEMEX study[7] suggested that a high peritoneal transport status by itself was not an independent risk factor for patient survival in continuous ambulatory PD (CAPD) patients, even when the patients were stratified according to a variety of factors known to influence outcome (for example, age, diabetes mellitus, and others). Subsequent studies,[8-10] such as the EAPOS study,[9] demonstrated that high peritoneal transport status had no effect on patient survival in the patients treated with automatic PD (APD) or icodextrin. Contrarily, Guan and colleagues[11] reported that higher peritoneal transport status was a significant independent risk factor for death-censored technique failure, but not for mortality in diabetic nephropathy patients on PD.

Diabetes mellitus has become the second most common cause of ESRD in China.[12,13] Some studies indicated that the predictive value of peritoneal transport in CAPD patients was dependent on the type of comorbidity present.[14] Diabetes mellitus especially was to be reported the most important risk factor for patient mortality.[15]

Therefore, our aim was to investigate the influence of peritoneal transport characteristics on clinical outcome in nondiabetic and diabetic nephropathy patients with PD in the present study.

MATERIALS AND METHODS

Patients

All patients came from the PD Center of Peking University Third Hospital, People's Republic of China, between January 15th, 2006 and January 15th, 2007. The patient inclusion criteria were: undergoing CAPD, age greater than 18 years, having full data of peritoneal transport characteristic measurements by peritoneal equilibration test (PET) after initiating PD, and signed informed consent form. None of patients had severe malnutrition. The patients who had a history of hemodialysis or kidney transplantation prior to PD, had any clinical manifestation of congestive heart failure (New York Heart Association class III to IV), or had an underlying active malignancy or acute infection were excluded from the study. At assessment, all patients had been free of peritonitis for more than one month. Finally, a total of 112 CAPD patients completing the original study were recruited in the present study and followed up till December 31st, 2010. Patients were evaluated for clinical and biochemical data, fluid status, nutritional status, and comorbidity during a routine clinical visit. The measurements were performed at a monthly scheduled visit, during which time the abdominal cavity was empty. The ethical committee of the hospital approved the study and informed consent was obtained from each patient. The study was performed according to the recommendations of the Declaration of Helsinki.

Dialysis Therapy

Patients on PD were treated by 2-L of 2 to 4 exchanges per day, using a standard Dianeal solution (Baxter China Ltd, Shanghai, China). The dialysate glucose concentration varied depending on the individual patient's requirements for ultrafiltration. All patients underwent CAPD therapy.

Peritoneal Transport Characteristics and Grouping

All patients were given a standard PET to evaluate peritoneal transport characteristic as described by Twardowski and colleagues.[16] According to Twardowski and colleagues, high was defined by 4-hour dialysate-plasma creatinine ratio as 0.81 to 1.03, high average as 0.65 to 0.80, low average as 0.50–0.64 and low as 0.34 to 0.49. On the basis of PET result, patients were divided into 2 groups of high-transport group (HT, including high) and non-high-transport group (non-HT, including high average, low and low average).[17] The time from initiation of PD to performance of the PET (PET time) was calculated.

Laboratory Measurements

Serum albumin was measured using the bromcresol green method. Intact parathyroid hormone level was determined using a commercial enzyme linked immunosorbent assay, while the remaining blood chemistry analyses were carried out using routine methods. Dialysis adequacy was assessed by Kt/V, which was the index of small molecular solute clearance.[18] The peritoneal Kt/V (pKt/V) and renal Kt/V (rKt/V) were measured separately, and total Kt/V was the sum of pKt/V and rKt/V. Urea distribution volume was calculated according to Watson formula.[19] Residual renal

function was measured as glomerular filtration rate using the mean of urea and creatinine clearance.[20]

Nutritional Assessment

Subjective global assessment was performed to assess patient's nutritional status by one experienced nurse. The patients were divided into 3 grades according to subjective global assessment: A, normal nutrition; B, mild malnutrition; and C, moderate to severe malnutrition.

Blood Pressure Measurements

The blood pressure was measured in a supine position after a fifteen-minute rest and using a mercury sphygmomanometer and a cuff of appropriate size, placed on the right arm. Phase I and V of the Korotkoff sounds were taken respectively as systolic blood pressure (Sblood pressure) and diastolic blood pressure (Dblood pressure). Three consecutive measurements were performed in each patient and the arithmetic mean of these was used. The pulse pressure (PP) was calculated as Sblood pressure minus Dblood pressure.

Bioelectrical Impedance Analysis

The patient's body fluid volumes were measured by multifrequency bioelectrical impedance analysis (Model 4200; Xitron Technologies, San Diego, CA, USA). The whole wrist-ankle method was used, as described in detail in our previous study.[21] The patient's height and weight were measured at first. The extracellular water, intracellular water, and total body water were derived by 3 consecutive measurements. The measurements were performed by the same examiner throughout the study.

Comorbidity

The comorbidity of each patient was determined according to the Charlson Comorbidity Index (CCI). The CCI was scored using the definitions of Charlson and collegues.[22]

Clinical Outcome

All patients were regularly followed up every 3 to 6 months until December 31st, 2010. The patients were followed until death, transfer to hemodialysis, kidney transplantation, transfer to other institutions, or drop-out due to renal function recovery. Patient survival, cardiovascular event, peritonitis, nonperitonitis infection, transfer to hemodialysis, kidney transplantation, transfer to other institutions, or drop-out due to recovery were recorded. All deaths that happened 3 months after transfer to hemodialysis were attributed as death event. The primary outcomes were death from any cause and technique failure. Technique failure was defined as death and permanent hemodialysis transfer.[23-27]

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation or median (interquartile range), while categorical variables were expressed as ratio or percentage. The independent-samples *t* test was used as appropriate to compare differences for continuous variables. When the variables were not normally distributed, the Mann-Whitney U test was used. The comparison of categorical variables was performed using the Chi-square test. Mortality and technique failure were plotted as Kaplan-Meier curves between the two groups, and the differences were assessed by the log-rank test. The associations between peritoneal transport and patient outcomes were analyzed in multivariable Cox regression models, after adjusting for potential confounders, including age, diabetes, nutritional status, and dialysis adequacy. Adjusted hazard ratios (HR) with 95% confidence intervals (CIs) were reported separately. A two-tailed *P* value less than .05 was considered significant.

RESULTS

Baseline characteristics of the study population

The clinical characteristics of the 112 patients were summarized in Table 1. The mean age was 61.3 ± 12.2 (26, 85) years, and the proportion of men was 41.1%. On average, the patients had been on PET for 8.4 (3.4, 25.3) months after initiating PD. The average dialysate-plasma creatinine ratio of all patients was 0.76 ± 0.13. All patients were followed up until an event happened or to the end of the study. During an average follow-up of 65.9 ± 23.9 months, 32 patients died, 4 patients transferred to hemodialysis, 2 patients transplanted, 1 patients transferred to other centers. The cumulative all-cause mortality at the 12th, 24th and 47th month was 4.5%, 15.2% and 28.6%, respectively. And the cumulative technique failure at the 12th, 24th and 47th month was 4.5%, 17.0% and 32.1%, respectively.

Table 1. Baseline Characteristics of all Patients Grouped According to the Peritoneal Transport Characteristics

Parameters	Total (n = 112)	Non-High-Transport Group (n = 70)	High-Transport Group (n = 42)	*P*
Age, y	61.3 ± 12.2	63.0 ± 11.3	58.7 ± 13.3	.08
Sex				
Male	46	29	17	
Female	66	41	25	.92
Body mass index, kg/m^2	24.0 ± 3.8	24.2 ± 3.8	23.8 ± 3.9	.59
Time on dialysis, mo	7.5 (3.5, 25.6)	4.6 (3.4, 21.9)	10.6 (4.0, 27.8)	.12
Peritoneal equilibration test time, mo	8.4 (3.4, 25.3)	4.8 (3.2, 23.9)	10.6 (4.2, 27.1)	.09
Follow-up time, mo	65.9 ± 23.9	65.5 ± 22.8	66.6 ± 25.8	.81
Etiologies of uremia				
Chronic glomerulonephritis, %	31 (27.7)	17 (24.3)	14 (33.3)	
Diabetes mellitus, %	29 (25.9)	20 (28.6)	9 (21.4)	
Hypertension, %	19 (17.0)	14 (20.0)	5 (11.9)	
Tubulointerstitial nephritis, %	19 (17.0)	11 (15.7)	8 (19.0)	
Polycystic kidney disease, %	4 (3.6)	4 (5.7)	0 (0)	
Obstructive nephropathy, %	1 (0.9)	0 (0)	1 (2.4)	
Unknown, %	9 (8.0)	4 (5.7)	5 (11.9)	.62
Systolic blood pressure, mm Hg	146.3 ± 22.7	145.1 ± 23.0	148.4 ± 22.5	.47
Diastolic blood pressure, mm Hg	82.4 ± 13.4	80.8 ± 12.7	85.3 ± 14.4	.09
Pulse pressure, mm Hg	63.8 ± 19.8	64.3 ± 19.9	63.1 ± 19.9	.75
Extracellular water, L	14.85 ± 3.17	14.76 ± 3.07	14.99 ± 3.37	.72
Intracellular water, L	13.85 ± 4.06	13.73 ± 4.22	14.04 ± 3.83	.70
Total body water, L	28.69 ± 6.75	28.49 ± 6.83	29.03 ± 6.68	.69
Instilled dialysate volume, mL/d	5022.9 ± 1864.0	5045.9 ± 1891.6	4984.4 ± 1839.1	.87
Urine volume, ml/d	656.3 ± 499.0	643.6 ± 521.1	677.6 ± 465.2	.73
Ultrafiltration volume, ml/d	551.5 ± 478.0	556.5 ± 485.3	543.1 ± 471.2	.89
Hemoglobin, g/L	116.9 ± 21.0	118.2 ± 22.8	114.7 ± 17.4	.40
Serum albumin, g/L	37.1 ± 3.9	37.6 ± 3.5	36.5 ± 4.3	.18
Serum sodium, mmol/L	138.1 ± 2.8	138.7 ± 2.3	137.3 ± 3.3	.02
Serum calcium, mmol/L	2.18 ± 0.25	2.21 ± 0.23	2.13 ± 0.26	.11
Serum glucose, mmol/L	6.00 ± 2.13	5.97 ± 1.77	6.04 ± 2.56	.87
Triglycerides, mmol/L	2.06 ± 1.13	2.41 ± 1.27	1.60 ± 0.72	.003
Total cholesterol, mmol/L	5.14 ± 1.05	5.04 ± 0.99	5.28 ± 1.14	.39
Low-density lipoprotein cholesterol, mmol/L	3.26 ± 0.97	3.14 ± 0.89	3.41 ± 1.06	.27
High-density lipoprotein cholesterol, mmol/L	1.36 ± 0.46	1.25 ± 0.41	1.49 ± 0.48	.04
Urea, mmol/L	21.7 ± 5.6	21.7 ± 5.3	21.6 ± 6.1	.89
Creatinine, μmol/L	769.5 ± 263.0	768.6 ± 277.2	771.0 ± 241.5	.96
Sensitive C-reactive protein, mg/L	2.0 (0.9, 8.5)	2.5 (1.1, 11.0)	1.5 (0.7, 6.1)	.18
Parathyroid hormone, pg/mL	237.0 ± 205.0	226.1 ± 177.6	252.5 ± 240.9	.60
Peritoneal Kt/V urea/wk	1.20 ± 0.46	1.17 ± 0.48	1.26 ± 0.44	.29
Renal Kt/V urea/wk	0.69 ± 0.61	0.75 ± 0.69	0.58 ± 0.44	.11
Total Kt/V urea/wk	1.89 ± 0.52	1.92 ± 0.58	1.85 ± 0.38	.47
Peritoneal creatinine clearance, L/wk/1.73 m^2	31.6 ± 13.1	28.4 ± 12.3	36.9 ± 12.7	.001
Renal creatinine clearance, L/wk/1.73 m^2	29.0 ± 28.3	32.3 ± 32.4	23.6 ± 18.9	.08
Total creatinine clearance, L/wk/1.73 m^2	60.6 ± 23.9	60.7 ± 27.7	60.5 ± 16.2	.97
Dialysate-plasma creatinine ratio	0.76 ± 0.13	0.68 ± 0.09	0.90 ± 0.06	< .001
Residual renal function, mL/min/1.73m^2	2.72 ± 2.67	3.01 ± 3.05	2.22 ± 1.81	.09
Dialysate protein loss	54.6 ± 12.5	55.1 ± 12.2	53.7 ± 13.1	.59
Subjective global assessment (B) %	31.5	27.5	38.1	.25
Charlson comorbidity index	4.0 (3.0, 6.0)	5.0 (3.0, 6.8)	4.0 (3.0, 6.0)	.39
Death, %	32 (28.6)	18 (25.7)	14 (33.3)	.39
Technique failure, %	36 (32.1)	20 (28.6)	16 (38.1)	.30

Among the 32 patients who died, the causes of death were cardiovascular disease in 11 (34.4%) patients, peritonitis in 3 (9.4%) patients, malignancy in 3 (9.4%) patients, severe nonperitonitis infection in 5 (15.6%) patients, gastrointestinal bleeding in 4 (12.5%) patients, multiple organ failure in 4 (12.5%) patients, and unknown causes in 2 (6.3%) patients. The reasons for transfer to hemodialysis included 3 (75.0%) peritonitis and 1 (25.0%) urological carcinoma.

The patients were divided into two groups according to PET results: non-HT group (70 patients, including high average 52 patients, L 15 patients and low average 3 patients) and HT group (including high, 42 patients). Compared with those with non-HT group, patients with HT group had higher high-density lipoprotein cholesterol and peritoneal creatinine clearance, lower serum sodium and triglycerides ($P < .05$). There were no significant differences in age, blood pressure, volume, serum albumin, mortality or technique failure between the two groups at baseline. Kaplan-Meier curves were shown in Figure 1. There were no differences in events-free survival (log-rank chi-square = 0.637, $P = .43$) or technique failure (log-rank chi-square = 0.856, $P = .36$) between the non-HT and HT groups in all patients.

Comparison Between Nondiabetic Nephropathy Subgroups

The PD patients were stratified according to diabetic nephropathy or not. Table 2 showed the subgroup analysis on the patients without diabetic nephropathy (n = 83). The HT patients without diabetic nephropathy were younger, had higher diastolic blood pressure and peritoneal creatinine clearance, and lower serum sodium and triglycerides as compared with those in non-HT group ($P < .05$). No differences were seen in volume, serum albumin, subjective global assessment, CCI, mortality or technique failure between the two groups. Kaplan-Meier analysis (Figure 2) found that there were no differences in events-free survival (log-rank chi-square = 0.071, $P = .79$) or technique failure (log-rank chi-square = 0.010, $P = .92$) between the subgroups without diabetic nephropathy.

Comparison Between Diabetic Nephropathy Subgroups

The 29 PD patients with diabetic nephropathy were included in another subgroup analysis (Table 3). The diabetic patients with HT had a higher prevalence of malnutrition, mortality and technique failure, higher peritoneal Kt/V, and peritoneal creatinine clearance than those in non-HT subgroup ($P < .05$). Significantly lower serum albumin, triglycerides, renal Kt/V, renal creatinine clearance, and Residual renal function were observed in HT diabetic patients as compared to the non-HT group ($P < .05$). Kaplan-Meier curves were reported in Figure 3. The diabetic nephropathy patients with HT showed a higher all-cause mortality (log-rank

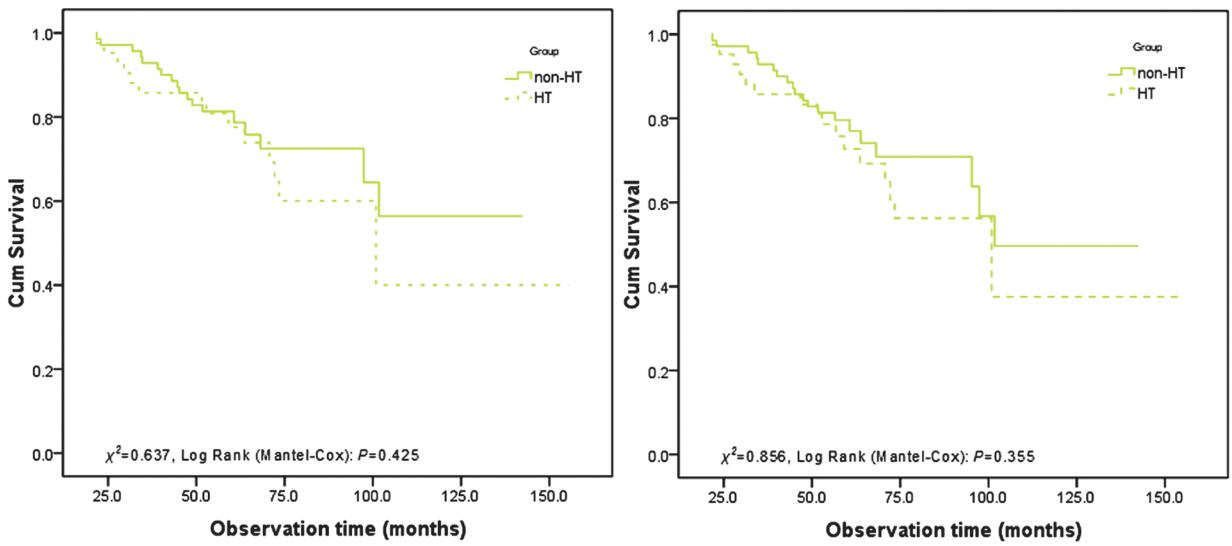

Figure 1. Kaplan-Meier survival curves for all patients for all-cause mortality (Left) and technique failure (Right) according to peritoneal membrane transport characteristic.

Table 2. Comparison between the nondiabetic nephropathy subgroup.

Parameters	Non-High-Transport Group (n = 50)	High-Transport Group (n = 33)	*P*
Age, y	62.9 ± 12.0	55.8 ± 13.0	.01
Sex			
Male	19	12	
Female	31	21	.88
Body mass index, kg/m^2	24.0 ± 4.0	24.1 ± 4.2	.96
Time on dialysis, mo	7.2 (3.7, 22.5)	15.0 (4.1, 30.9)	.20
Peritoneal equilibration test time, mo	8.7 (3.4, 24.7)	15.6 (4.2, 30.2)	.23
Follow-up time, mo	66.7 ± 20.8	71.0 ± 26.2	.41
Systolic blood pressure, mm Hg	141.9 ± 24.2	146.6 ± 22.2	.38
Diastolic blood pressure, mm Hg	80.6 ± 13.1	87.1 ± 15.0	.04
Pulse pressure, mm Hg	61.3 ± 20.7	59.5 ± 17.9	.69
Extracellular water, L	14.10 ± 2.63	14.56 ± 3.17	.47
Intracellular water, L	13.66 ± 4.34	14.48 ± 3.89	.39
Total body water, L	27.75 ± 6.59	29.04 ± 6.74	.39
Instilled dialysate volume(ml/d	5129.3 ± 1827.4	4795.3 ± 1920.3	.43
Urine volume, ml/d	566.4 ± 523.8	710.6 ± 495.3	.21
Ultrafiltration volume, ml/d	596.6 ± 503.5	520.6 ± 515.6	.51
Hemoglobin, g/L	114.5 ± 20.4	114.0 ± 18.9	.92
Serum albumin, g/L	38.3 ± 3.0	37.8 ± 3.5	.59
Serum sodium, mmol/L	139.0 ± 2.3	137.6 ± 3.3	.04
Serum calcium, mmol/L	2.24 ± 0.25	2.13 ± 0.27	.05
Serum glucose, mmol/L	5.39 ± 1.37	5.30 ± 1.45	.81
Triglycerides, mmol/L	2.32 ± 1.33	1.64 ± 0.75	.03
Total cholesterol, mmol/L	4.83 ± 0.98	5.27 ± 1.15	.16
Low-density lipoprotein cholesterol, mmol/L	2.95 ± 0.90	3.40 ± 1.14	.13
High-density lipoprotein cholesterol, mmol/L	1.26 ± 0.44	1.51 ± 0.48	.06
Urea, mmol/L	22.4 ± 5.3	22.5 ± 6.3	.98
Creatinine, μmol/L	832.2 ± 271.8	833.4 ± 231.7	.99
Sensitive C-reactive protein, mg/L	2.3 (1.0, 11.4)	1.1 (0.6, 4.8)	.10
Parathyroid hormone, pg/mL	209.8 ± 163.0	273.2 ± 253.1	.26
Peritoneal Kt/V urea/wk	1.22 ± 0.48	1.22 ± 0.48	> .99
Renal Kt/V urea/wk	0.59 ± 0.59	0.59 ± 0.47	.96
Total Kt/V urea/wk	1.80 ± 0.52	1.81 ± 0.40	.96
Peritoneal creatinine clearance, L/wk/1.73 m^2	29.64 ± 12.21	35.44 ± 13.65	.046
Renal creatinine clearance, L/wk/1.73 m^2	23.71 ± 25.54	23.60 ± 19.98	.98
Total creatinine clearance, L/wk/1.73 m^2	53.35 ± 20.33	59.05 ± 16.31	.18
Dialysate-plasma creatinine ratio	0.69 ± 0.08	0.90 ± 0.07	<.001
Residual renal function, mL/min/1.73 m^2	2.19 ± 2.41	2.21 ± 1.92	.97
Dialysate protein loss	53.84 ± 11.85	54.34 ± 13.55	.86
Subjective global assessment, B	26.5	24.2	.82
Charlson comorbidity index	4.0 (3.0, 5.8)	3.0 (2.0, 5.0)	.54
Death, %	12 (24.0)	7 (21.2)	.77
Technique failure, %	14 (28.0)	9 (27.3)	.94

chi-square = 4.305, *P* = .04) and technique failure (log-rank chi-square = 4.305, *P* = .04) than those with non-HT.

Risk factors for death and technique failure

Upon Cox proportional hazards models analysis, high peritoneal transport (HR, 2.369; 95% CI, 1.056 to 5.311; *P* = .036), diabetic nephropathy (HR, 2.499; 95% CI, 1.134 to 5.508; *P* = .023), age (HR, 1.081; 95% CI, 1.032 to 1.133; *P* = .001) and peritoneal creatinine clearance (HR, 0.962; 95% CI, 0.929 to 0.997; *P* = .033) independently predicted all-cause mortality in CAPD patients after adjusting for potential confounders including serum albumin at baseline (Table 4). Moreover, high peritoneal transport (HR, 2.299; 95% CI, 1.079 to 4.899;

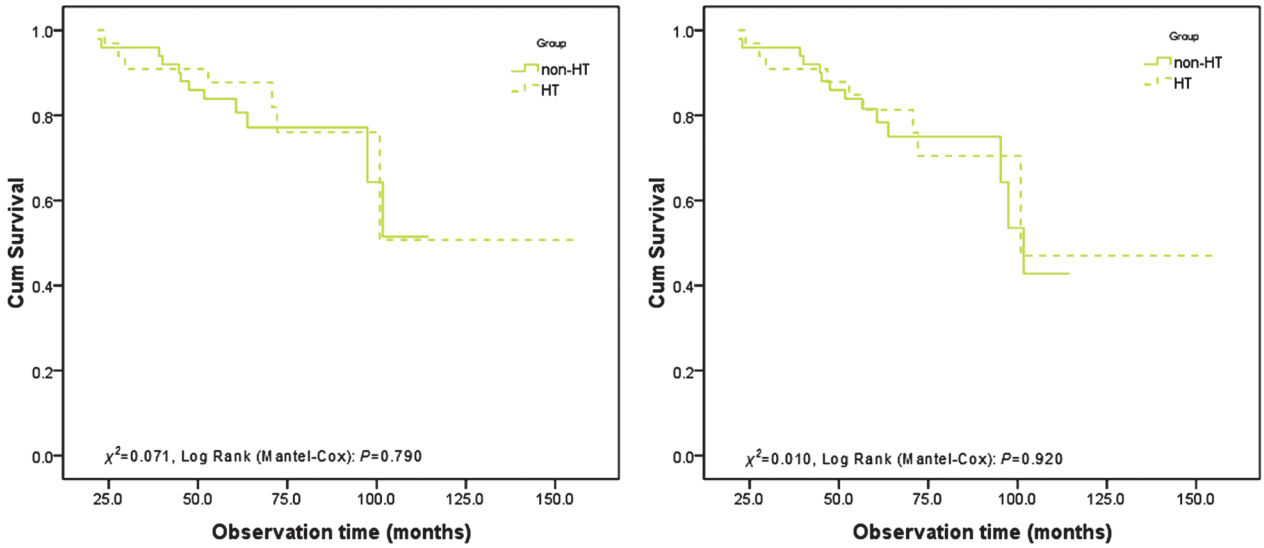

Figure 2. Kaplan-Meier survival curves for the nondiabetic nephropathy patients for all-cause mortality (Left) and technique failure (Right) according to peritoneal membrane transport characteristic.

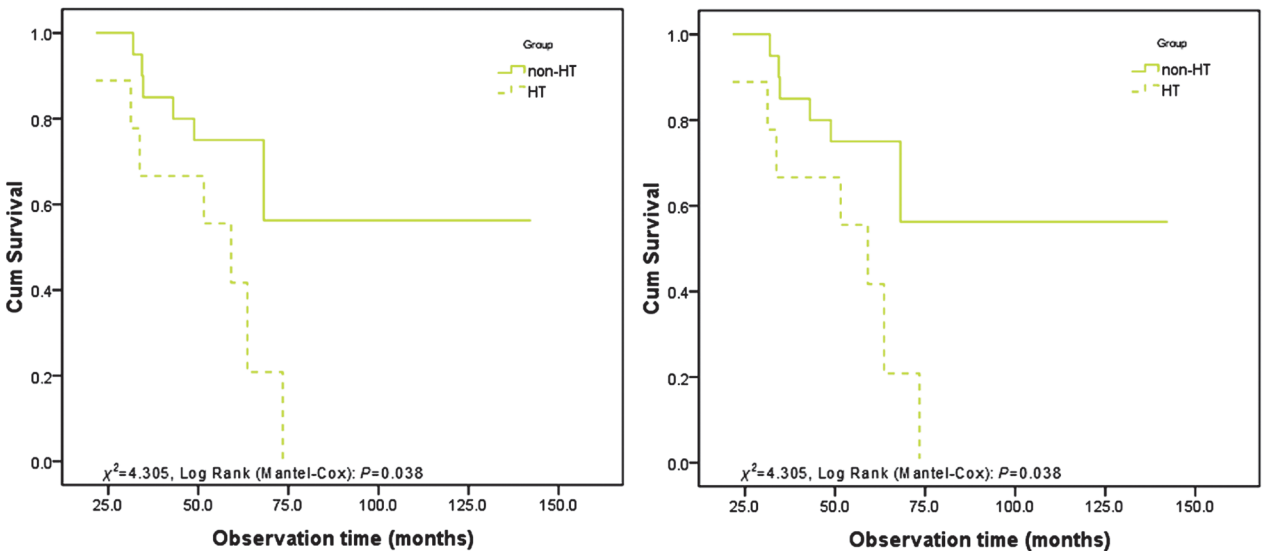

Figure 3. Kaplan-Meier survival curves for the diabetic nephropathy patients for all-cause mortality (Left) and technique failure (Right) according to peritoneal membrane transport characteristic.

P = .031) and age (HR, 1.070; 95% CI, 1.026 to 1.116; *P* = .002) were also independent predictors of technique failure in CAPD patients (Table 5).

DISCUSSION

The present study demonstrated that there were no significant differences in all-cause mortality or technique failure between all patients and nondiabetic nephropathy subgroup with high peritoneal transport and those with non-high peritoneal transport in Kaplan-Meier analysis. In contrast, diabetic nephropathy subgroup with high peritoneal transport showed a significantly higher all-cause mortality and technique failure than those with non-high peritoneal transport. Furthermore, Cox regression analysis found that high peritoneal transport independently predicted all-cause mortality and technique failure in CAPD patients after adjustment for confounders at baseline.

Our result was consistent with some previous studies.[1-5] In the CANUSA study,[1] high peritoneal transport was associated with increased risk of death and technique failure in CAPD patients. Our

Table 3. Comparison Between Diabetic Nephropathy Subgroups

Parameters	Non-High-Transport Group (n = 20)	High-Transport Group (n = 9)	*P*
Age, years	63.1 ± 9.5	69.4 ± 8.4	.10
Sex			
Male	10	5	
Female	10	4	> .99
Body mass index, kg/m^2	24.6 ± 3.4	22.7 ± 2.2	.14
Time on dialysis, mo	3.4 (3.2, 19.0)	10.2 (3.1, 12.8)	.59
PET duration, mo	3.4 (1.6, 19.0)	10.2 (4.3, 12.9)	.10
Follow-up time, mo	62.4 ± 27.6	50.3 ± 17.5	.24
Systolic blood pressure, mm Hg	152.7 ± 18.1	156.6 ± 24.1	.65
Diastolic blood pressure, mm Hg	81.2 ± 11.9	76.9 ± 6.9	.38
Pulse pressure, mm Hg	71.5 ± 16.1	79.7 ± 21.8	.30
Extracellular water, L	16.40 ± 3.49	16.56 ± 3.77	.91
Intracellular water, L	13.90 ± 4.04	12.43 ± 3.31	.35
Total body water, L	30.31 ± 7.22	28.99 ± 6.86	.65
Instilled dialysate volume, mL/d	4837.5 ± 2078.1	5677.8 ± 1377.3	.28
Urine volume, mL/d	836.5 ± 472.9	556.7 ± 327.3	.12
Ultrafiltration volume, mL/d	456.3 ± 432.2	625.6 ± 254.7	.29
Hemoglobin, g/L	127.5 ± 26.3	117.1 ± 10.2	.15
Serum albumin, g/L	35.9 ± 4.2	32.2 ± 3.8	.05
Serum sodium, mmol/L	137.9 ± 2.1	136.0 ± 3.2	.09
Serum calcium, mmol/L	2.13 ± 0.16	2.15 ± 0.25	.82
Serum glucose, mmol/L	7.08 ± 1.96	9.11 ± 3.83	.22
Triglycerides, mmol/L	2.69 ± 1.09	1.48 ± 0.64	.03
Total cholesterol, mmol/L	5.74 ± 0.68	5.30 ± 1.24	.42
Low-density lipoprotein cholesterol, mmol/L	3.74 ± 0.54	3.45 ± 0.80	.43
High-density lipoprotein cholesterol, mmol/L	1.23 ± 0.33	1.42 ± 0.54	.43
Urea, mmol/L	20.0 ± 5.0	18.3 ± 4.0	.36
Creatinine, μmol/L	616.1 ± 231.5	549.7 ± 118.6	.43
Sensitive C-reactive protein, mg/L	2.5 (1.3, 11.8)	3.0 (2.1, 14.4)	.72
Parathyroid hormone, pg/mL	284.0 ± 223.6	123.0 ± 56.1	.19
Peritoneal Kt/V urea/wk	1.03 ± 0.46	1.43 ± 0.20	.004
Renal Kt/V urea/wk	1.17 ± 0.75	0.55 ± 0.32	.004
Total Kt/V urea/wk	2.21 ± 0.64	1.98 ± 0.29	.32
Peritoneal creatinine clearance, L/wk/1.73 m^2	25.38 ± 12.45	42.24 ± 6.60	<.001
Renal creatinine clearance, L/wk/1.73 m^2	53.72 ± 38.07	23.74 ± 15.45	.03
Total creatinine clearance, L/wk/1.73 m^2	79.09 ± 34.88	65.99 ± 15.18	.29
Dialysate-plasma creatinine ratio	0.66 ± 0.10	0.91 ± 0.03	<.001
Residual renal function, mL/min/1.73m^2	5.07 ± 3.56	2.28 ± 1.43	.006
Dialysate protein loss	58.09 ± 12.76	51.43 ± 11.49	.19
Subjective global assessment (B), %	30.0	88.9	.005
Charlson comorbidity index	7.0 (6.0, 7.8)	6.5 (5.5, 10.8)	.96
Death, %	6 (30.0)	7 (77.8)	.04
Technique failure, %	6 (30.0)	7 (77.8)	.04

Table 4. Cox Regression Analysis of All-cause Mortality in All Patients*

Covariate	Hazard Ratio	95% Confidence Interval	*P*
High dialysate-plasma creatinine ratio	2.369	1.056 to 5.311	.04
Diabetic nephropathy	2.499	1.134 to 5.508	.02
Age, y	1.081	1.032 to 1.133	.001
Peritoneal creatinine clearance, L/wk/1.73 m^2	0.962	0.929 to 0.997	.03

*Adjusted for serum albumin (*P* = .29)

Table 5. Cox Regression Analysis of Technique Failure in All Patients*

Covariate	Hazard Ratio	95% Confidence Interval	*P*
High dialysate-plasma creatinine ratio	2.299	1.079 to 4.899	.03
Diabetic nephropathy	2.066	0.971 to 4.398	.06
Age, y	1.070	1.026 to 1.116	.002
Peritoneal creatinine clearance, L/wk/1.73 m^2	0.970	0.939 to 1.002	.07

*Adjusted for serum albumin (*P* = .85)

previous study[4] also indicated that high peritoneal transport was associated with decreased fluid and small-solute removal and higher mortality in CAPD patients. The previous explanation was that high peritoneal transporters were prone to fluid overload, low serum albumin, inadequate dialysis, malnutrition, and cardiovascular disease, especially with long dwell CAPD cycles.

On the contrary, the ADEMEX study[7] showed that peritoneal small solute clearance had a neutral effect on patient survival. Chung and coworkers[10] investigated 82 PD patients in a prospective, single center study and found that peritoneal transport was not significantly associated with nutritional status at more than 6 months' treatment with PD and was not associated with subsequent patient survival. A similar finding was reported by Szeto and colleagues,[28] who also found no significant correlation between peritoneal transport and nutritional status and patient survival in new and prevalent patients. In addition, a large registry report from Australian and New Zealand confirmed that high peritoneal transport predicted death in CAPD patients, but not in APD patients.[29] Subsequently, Yang and coworkers[30] demonstrated that higher peritoneal transport was not a significant independent risk factor for mortality in APD patients. However, Guan and coworkers[11] reported that higher peritoneal transport status was a significantly independent risk factor for death-censored technique failure, but not for mortality in diabetic nephropathy patients on PD.

There are some differences between our study and the previous reports. Firstly, many previous studies have demonstrated the profound influence of comorbid disease such as diabetes mellitus on mortality in CAPD patients.[6,15] Contrarily, some studies did not find the association between diabetes mellitus and death or technique failure.[1,7] In the CANUSA study, the relationship between diabetes mellitus and patient outcome was not seen, although high peritoneal transport patients had a greater proportion of type I (14.0%) and type II (19.4%) diabetes mellitus. In the current study, the effect of diabetes mellitus on patient outcome was noticed and patients were stratified to compare according to diabetic nephropathy or not. Secondly, our data showed a higher percentage of high peritoneal transporter type (37.5%), which was obviously greater than that in other studies[1,6,31] and might lead to our present results. Thirdly, CAPD patients, but not APD patients, were observed in this study. Patients were grouped in different methods as compared with other studies.[1,4] In addition, our result contradicted Guan's reports[11] in part although diabetic PD patients were observed in the two studies. On the whole, this finding seemed to be affected by the high prevalence of diabetes mellitus and high peritoneal transporter in this study. The PD patients with diabetes mellitus had peritoneal histological changes such as mesothelial basement membrane thickening, vascular wall thickening, and inflammatory infiltrate with long-term exposure to glucose-containing dialysis solution.[6] These changes can lead to increased peritoneal transport, fluid overload, malnutrition, eventually poor outcome in the diabetic PD patients.

In our study, the age was an independent predictor for all-cause death and technique failure in PD patients. Our result was in agreement with the previous reports.[2,6] Peritoneal creatinine clearance was also a predictor for all-cause death in PD patients, which was similar to the CANUSA study.[1] Furthermore, serum albumin, often used as a nutritional marker, was significantly lower in the diabetic nephropathy patients with high transport in the current study. However, Cox regression found that serum albumin could not predict clinical outcome in PD patients. Davies and colleagues[2] indicated that plasma albumin was not an independent predictor although albumin declined with time. Our result was consistent with Davies' reports.[2] Additionally, HT diabetic subgroup had a higher prevalence of malnutrition

and lower Residual renal function than non-HT subgroup.

It should be noted that there are some limitations in our study. Firstly, this study is a single-center and retrospective study. Patient selection biases cannot be avoided. Secondly, there was no data on volume status at the end of follow-up, although the HT group had no significant difference in volume status at baseline as compared with the non-HT group. In addition, the present study comprised a relatively small number of patients. Finally, inflammatory factors have also been described in incident PD patients with a high peritoneal transport status, which maybe is related to PD patient outcome.[10,32] Unfortunately, inflammatory factors were not included in this study.

CONCLUSIONS

In summary, our study showed that diabetic nephropathy PD patients with high peritoneal transport had a higher all-cause mortality and technique failure than those with non-high peritoneal transport, but we did not find the correlation between peritoneal transport and patient outcome in nondiabetic nephropathy PD patients. The peritoneal transport was a strong independent predictor for all-cause mortality and technique failure in CAPD patients.

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CONFLICT OF INTEREST

None declared.

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