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Original Article

ORIGINAL ARTICLE

The centre-calculated cutoff value is better for identifying fast peritoneal solute transfer of patients on peritoneal dialysis than the traditional value: a retrospective cohort study

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ABSTRACT

Background. The mean 4-h dialysate to plasma ratio of creatinine (4-h D/Pcr) is a vital cutoff value for recognizing the fast peritoneal solute transfer rate (PSTR) in patients on peritoneal dialysis (PD); however, it shows a noticeable centre effect. We aimed to investigate our centre-calculated cutoff value (CCV) of 4-h D/Pcr and compare it with the traditional cutoff value (TCV) (0.65).

Methods. In this study, we enrolled incident PD patients at our centre from 2008 to 2019, and divided them into fast or non-fast PSTR groups according to baseline 4-h D/Pcr–based CCV or TCV. We compared the efficiency of the fast PSTR recognized by two cutoff values in predicting mortality, ultrafiltration (UF) insufficiency and technical survival.

Results. In total, 1905 patients were enrolled, with a mean 4-h D/Pcr of 0.71 ± 0.11. Compared with TCV (0.65), CCV (0.71) showed superiority in predicting mortality of PD patients [hazard ratio (HR) 1.27, 95% confidence interval (CI) 1.02–1.59 vs HR 1.24, 95% CI 0.97–1.59]. The odds ratio (OR) of the fast PSTR in centre classification was slightly higher than traditional classification in predicting UF insufficiency (OR 1.67, 95% CI 1.25–2.24 vs OR 1.60, 95% CI 1.15–2.22). Additionally, the restricted cubic splines 4-h D/Pcr has an S-shaped association with mortality and UF insufficiency, and the inflection points of 4-h D/Pcr were 0.71 (equal to CCV).

Conclusions. The CCV of 4-h D/Pcr for identifying fast PSTR was 0.71. It was superior to TCV in predicting mortality and UF insufficiency.

Keywords: fast peritoneal solute transfer rate, mortality, peritoneal dialysis, peritoneal equilibration test, ultrafiltration insufficiency

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INTRODUCTION

As a filter membrane, the peritoneum determines the solute transfer and ultrafiltration (UF) volume in patients undergoing peritoneal dialysis (PD) and dominates the effect of PD [1]. Therefore, identifying the status of peritoneal transfer function is of paramount importance for optimizing and individualizing PD prescriptions in patients with PD. The peritoneal equilibration test (PET) is used to characterize peritoneal UF and solute transfer [2]. The test yields the dialysate to plasma ratio of creatinine (D/Pcr), dialysate glucose ratio and UF volume [2], among which 4-h D/Pcr is the most frequently used to evaluate the peritoneal solute transfer rate (PSTR) of patients [3]. In addition, D/Pcr is a robust predictor of clinical outcomes among these three parameters of PET [3–11].

Studies have clarified the detrimental effects of fast PSTR (PSTR is higher than the mean 4-h D/Pcr in the PD population) on clinical outcomes in patients undergoing PD [4, 5, 12]. Accordingly, the mean 4-h D/Pcr ratio has become a clinically important cutoff value. Initially, the PSTR of PD patients was categorized into high, high average, low average and low peritoneal transport rates according to the values of 4-h D/Pcr among 86 PD patients (0.65 ± 0.15), which was widely used in clinical practice [2]. However, because of the diverse methods for measuring creatinine levels and different dextrose/glucose concentrations used for PET, there is a noticeable region/centre effect on the 4-h D/Pcr value [12, 13]. Therefore, the actualized cutoff value of 4-h D/Pcr (0.65) may not apply to all centres. In this context, the latest guideline of the International Society of Peritoneal Dialysis (ISPD) recommends that centres establish their own normal range of 4-h D/Pcr value [12].

We assume that our centre-calculated cutoff value (CCV) is different from the traditional cutoff value (TCV) (0.65), and that it is better in predicting the clinical outcomes of PD patients than the TCV. We undertook this study to calculate CCV of 4-h D/Pcr to accurately identify PD patients with a fast PSTR in our centre. We intend to further analyse and compare the efficacy of CCV and TCV in predicting mortality, UF insufficiency and technical survival.

MATERIALS AND METHODS

Patients and study design

All patients initiating PD therapy at our centre between 2008 and 2019 were included in this retrospective cohort study. Patients who missed the baseline PET data, were aged <18 years, withdrew from PD within 6 months of initiation, were transferred from permanent haemodialysis, underwent automatized PD, used icodextrin PD solution or had failed renal transplantation were excluded. All participants were followed-up until 31 December 2021. Patients were assigned to the fast PSTR group or non-fast PSTR group according to baseline 4-h D/Pcr values, which had two different cutoff values (CCV and TCV). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and approved by the Human Ethics Committee of the First Affiliated Hospital, Sun Yat-sen University.

Data collection and measurements

All participants underwent at least one standard PET during the first 6 months of PD initiation, which was recorded as the baseline PET in this study. The PET was routinely done for them every 6 months during the follow-up period. At our centre, standard PET was performed using 2.5% dextrose at baseline. During the PET, dialysate samples at 0, 120 and 240 min and venous blood samples at 120 min were collected. All samples were sent to the laboratory of the First Affiliated Hospital, Sun Yat-sen University, to analyse the concentrations of creatinine (enzyme colorimetric method), glucose (glucose oxidase method) and urea nitrogen (enzyme colorimetric method). The UF volume within 240 min was also calculated and recorded. For the interference of glucose in the effluent, the concentration of creatinine in the dialysate was corrected using a correction factor of 0.000531415. The 4-h D/Pcr ration was calculated as the ratio of the corrected creatinine concentration in the dialysate (240 min) to the creatinine concentration in the serum (120 min). Additionally, the 240-min UF volume during the follow-up period was collected to determine the occurrence of insufficient UF.

We recorded the demographics and comorbidities of participants at the initiation of PD therapy, including age, gender, primary cause of renal failure, history of cardiovascular disease (CVD), complicated with diabetes, body mass index (BMI) and Charlson Comorbidity Index (CCI). Other data, including 24-h urine volume, 24-h UF volume, haemoglobin (Hb), serum albumin, serum calcium, serum phosphorus, blood urea nitrogen (BUN) and intact parathyroid hormone (iPTH) performed during the first 1–3 months of PD initiation, were collected as baseline data. Urea clearance normalized to total body water (Kt/V) were performed and analysed together with PET.

Definition and study endpoints

Fast PSTR was defined as the 4-h D/Pcr above the PD population mean value calculated by PET using either a 2.5% or 4.25% glucose-based dialysate [12]. Therefore, CCV for recognizing the fast PSTR patients was the mean 4-h D/Pcr value based on our centre PET data. Because the purpose of this study was to compare the efficacy of different cutoff values (CCV and TCV), these two cutoff values were used for further statistical analysis. The CCI was calculated by weighting 19 comorbid conditions and age, and utilized to evaluate comorbidity status [14]. Patients with CVD were defined as having angina, myocardial infarction, congestive heart failure, coronary heart disease, cerebrovascular events or peripheral vascular disease [15]. All-cause mortality refers to the death of an individual owing to various causes. Death-censored technical failure was defined as the transfer of PD to haemodialysis for more than 3 months. Death, kidney transplantation, transfer to other centres, loss to follow-up and follow-up until the study completion date were treated as censoring events. UF insufficiency refers to a 240-min UF volume of <100 mL on 2.5% glucose solution or <400 mL on 4.25% glucose solution [12].

The primary endpoint of this study was all-cause mortality. The secondary endpoints were UF insufficiency and technical survival.

Statistical analysis

The participants were divided into two groups (fast PSTR and non-fast PSTR) according to their baseline 4-h D/Pcr values. Individuals with 4-h D/Pcr values above the cutoff values were assigned to the fast PSTR group, whereas the others were assigned to the non-fast PSTR group. Data are presented as mean ± standard deviation (SD), median with interquartile range (IQR) or frequency. To determine the differences in variables between the two groups, the Wilcoxon test, chi-square test or *t*-test was used, as appropriate.

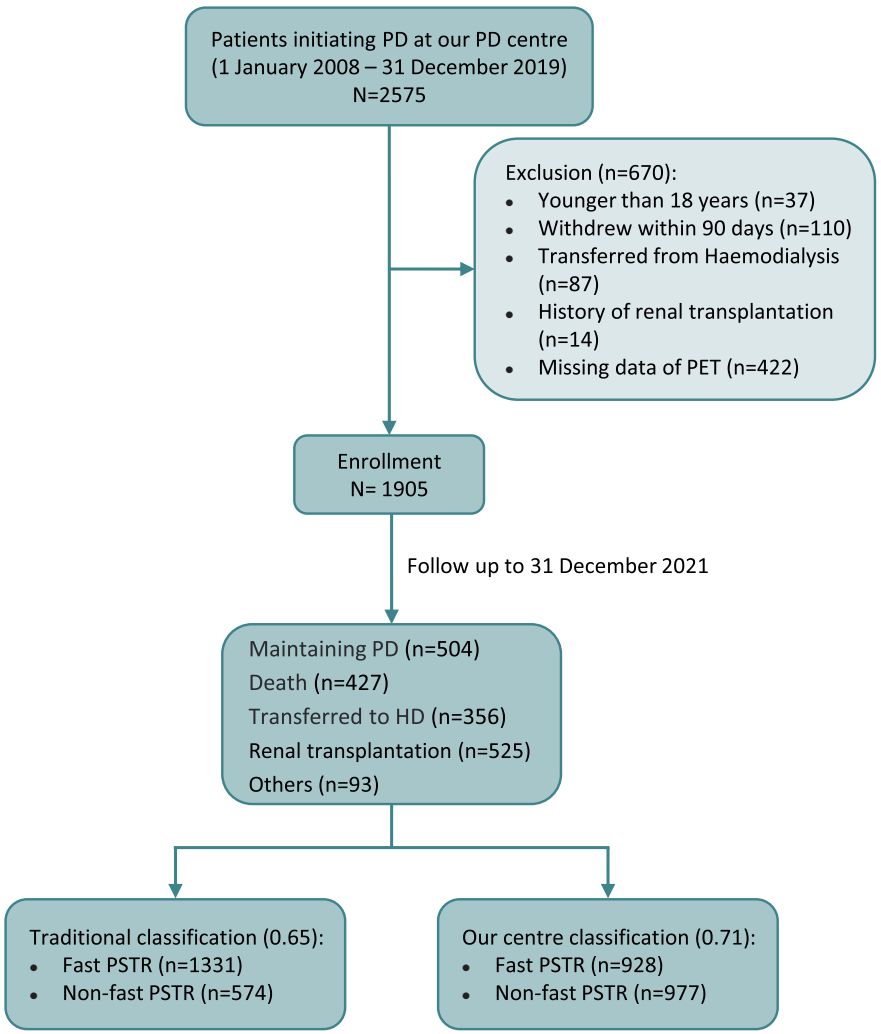

Figure 1: Flow chart of the study population. HD, haemodialysis.

Kaplan–Meier curves were used to depict the cumulative survival rate of the groups and to further estimate the differences in survival distributions between the two groups using log-rank tests. Adjusted Cox models were used to calculate hazard ratios (HRs) for mortality. Variables with $P < .1$ in the univariate regression model or those considered as potential confounders for mortality were adjusted, including age, gender, BMI, CCI, 24-h urine volume, BUN, peritoneal Kt/V, Hb, serum calcium, serum phosphorus and iPTH. In addition, the association between 4-h D/Pcr on a continuous scale and HRs for mortality was plotted using a Cox model with a restricted cubic spline (RCS). To compare the efficacy of the calculated cutoff value based on CCV (0.71) and TCV (0.65) in predicting mortality of patients with PD, we used a time-dependent receiver operating characteristic (ROC) curve. We calculated the area under the curve (AUC), plotted its changes over time and compared the AUC of the two classification methods.

Pearson's and Spearman's correlation analyses were used to determine the relationship between the 4-h D/Pcr and UF status. Furthermore, we used multiple logistic regression to test the association between fast PSTR and UF insufficiency and calculated the odds ratios (ORs). In the adjusted logistic regression models, the covariates ($P < .1$) in the univariate regression model or those considered as risk factors for UF insufficiency were finally adjusted, including age, gender, BMI, GFR, glucose exposure and PD vintage. RCS with four knots was also performed to explore the association between 4-h D/Pcr and UF insufficiency.

SPSS software (version 22.0) and R (version 4.1.2) were used to perform statistical analysis. Statistical significance was set at $P < .05$.

RESULTS

Demographic and clinical characteristics

A total of 2575 patients initiated PD therapy at our centre between 2008 and 2019. After screening, 1905 patients were enrolled in the cohort, and 670 patients were excluded (Fig. 1). The baseline data of patients are shown in Table 1. Of 1905 participants, the mean age was 45.5 ± 14.4 years, 40.7% were female, 22.8% had diabetes and 49.8% had a history of CVD. In both methods of classification, compared with the patients in the non-fast PSTR group, those in the fast PSTR group were older; more likely to be male and develop diabetes; had higher CCI scores, BMI, GFR, 24-h urine volume and glucose exposure; and had lower Hb, serum albumin, serum calcium, serum BUN, peritoneal Kt/V and 24-h UF volume.

Table 1: Baseline characteristics of individuals according to categories of traditional classification (0.65) and our centre classification (0.71).

		Traditional classification			Our centre classification		
Variables	All (N = 1905)	Non-fast PSTR (*n* = 574)	Fast PSTR (*n* = 1331)	*P*-value	Non-fast PSTR (*n* = 977)	Fast PSTR (*n* = 928)	*P*-value
Age (years)	45.5 ± 14.4	44.4 ± 14.7	46.0 ± 14.2	**.019**	44.2 ± 14.2	46.9 ± 14.4	**<.001**
Female (%)	40.7	52.4	47.6	**<.001**	48.9	31.3	**<.001**
BMI (kg/m^2)	21.7 ± 3.14	21.4 ± 3.29	21.8 ± 3.07	**.007**	21.5 ± 3.27	21.9 ± 2.99	**.016**
Primary kidney disease [*n* (%)]				**<.001**			**<.001**
Glomerulonephritis	1171 (61.5)	372 (64.8)	799 (60.0)		642 (65.7)	529 (57.0)	
Diabetic kidney disease	372 (19.5)	79 (13.8)	293 (22.0)		131 (13.4)	241 (26.0)	
Renal vascular disease	145 (7.6)	43 (7.5)	102 (7.7)		69 (7.1)	76 (8.2)	
Other	217 (11.4)	80 (13.9)	137 (10.3)		135 (13.8)	82 (8.8)	
Diabetes [*n* (%)]	434 (22.8)	101 (17.6)	333 (25)	**<.001**	162 (16.6)	272 (29.3)	**<.001**
CVD [*n* (%)]	949 (49.8)	259 (45.1)	690 (51.8)	**.007**	466 (47.7)	483 (52.0)	.058
CCI score	3.35 ± 1.68	3.21 ± 1.64	3.41 ± 1.70	**.016**	3.16 ± 1.56	3.55 ± 1.78	**<.001**
Hb (g/L)	107 ± 19	110 ± 17	106 ± 19	**<.001**	109 ± 18	105 ± 19	**<.001**
Albumin (g/L)	36.9 ± 4.7	38.8 ± 4.2	36.1 ± 4.7	**<.001**	38.4 ± 4.2	35.4 ± 4.7	**<.001**
Potassium (mmol/L)	3.80 ± 0.66	3.79 ± 0.63	3.81 ± 0.67	.487	3.80 ± 0.64	3.81 ± 0.68	.844
Calcium (mmol/L)	2.25 ± 0.20	2.30 ± 0.20	2.23 ± 0.19	**<.001**	2.28 ± 0.20	2.22 ± 0.19	**<.001**
Phosphorus (mmol/L)	1.34 ± 0.35	1.45 ± 0.38	1.29 ± 0.32	**<.001**	1.40 ± 0.37	1.28 ± 0.32	**<.001**
iPTH (pg/mL)	243 (121–404)	242 (105–401)	245 (127–403)	.374	244 (123–402)	242 (119–405)	.477
BUN (mmol/L)	16.0 ± 5.4	16.9 ± 5.7	15.7 ± 5.3	**<.001**	16.3 ± 5.4	15.7 ± 5.4	**.023**
Total Kt/V	2.49 ± 0.64	2.44 ± 0.62	2.51 ± 0.64	**.018**	2.48 ± 0.63	2.50 ± 0.65	.416
Peritoneal Kt/V	1.66 ± 0.39	1.70 ± 0.40	1.65 ± 0.39	**.008**	1.69 ± 0.40	1.64 ± 0.38	**.002**
eGFR (mL/min/1.73 m^2)	4.01 ± 2.75	3.63 ± 2.61	4.19 ± 2.79	**<.001**	3.82 ± 2.67	4.23 ± 2.82	**.002**
24-h urine volume (mL/day)	1100 (700–1600)	1000 (600–1600)	1200 (750–1700)	**.001**	1050 (600–1600)	1200 (800–1675)	**.019**
Glucose exposure (g/day)	128 ± 20	123 ± 15	130 ± 22	**<.001**	124 ± 17	132 ± 23	**<.001**
24-h UF volume (mL)	140 (–120 to 450)	370 (95–650)	40 (–250 to 350)	**.004**	260 (0–535)	0 (–320 to 300)	**<.001**

Data are presented as frequency (%), mean ± SD or median (interquartile range).
Traditional classification: cutoff value, 0.65; fast PSTR group, >0.65; non-fast PSTR group, ≤0.65. Our centre classification: cutoff value, 0.71; fast PSTR group, >0.71; non-fast PSTR group, ≤0.71.
eGFR, estimated glomerular filtration rate.
Bold means P-value < 0.05

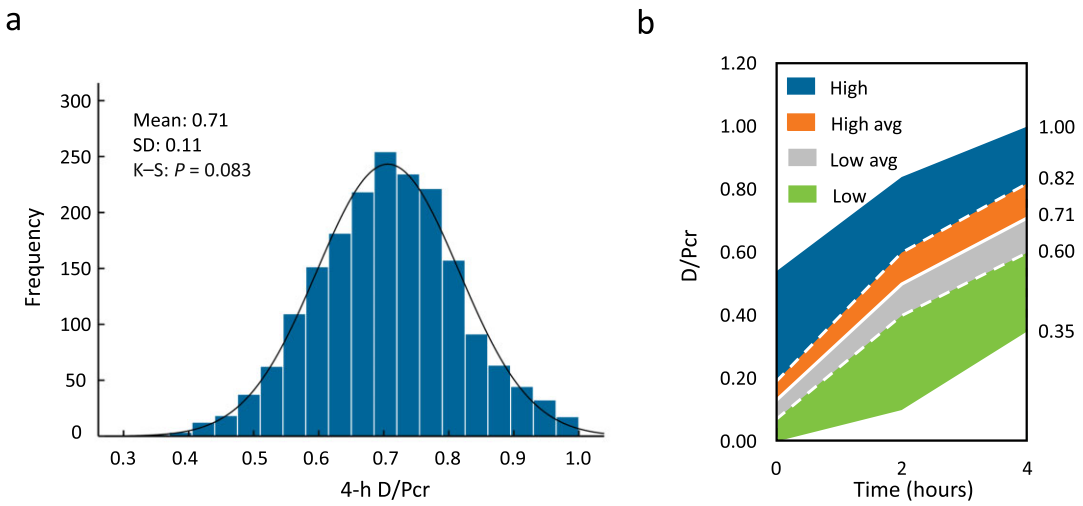

Figure 2: The distribution (**a**) and categories (**b**) of 4-h D/Pcr in PD patients.

As shown in Fig. 2a, the distribution of 4-h D/Pcr was normal (P = .083), and the mean 4-h D/Pcr of participants was 0.71 (± 0.11). Therefore, CCV for recognizing patients with fast PSTR was 0.71 in our centre. We first classified the patients into four categories according to the baseline 4-h D/Pcr values determined using the method of Twardowski *et al.* [2].: high (0.82–1.00), high average (0.71–0.82), low average (0.60–0.71) and low (0.35–0.60) peritoneal transport rates (Fig. 2b). Then, patients were assigned to the fast PSTR or non-fast PSTR groups according to their baseline 4-h D/Pcr values based on the different cutoff values (CCV: fast PSTR group, >0.71; non-fast PSTR group, ≤0.71; TCV: fast PSTR group, >0.65; non-fast PSTR group, ≤0.65), which is described in detail in Fig. 1.

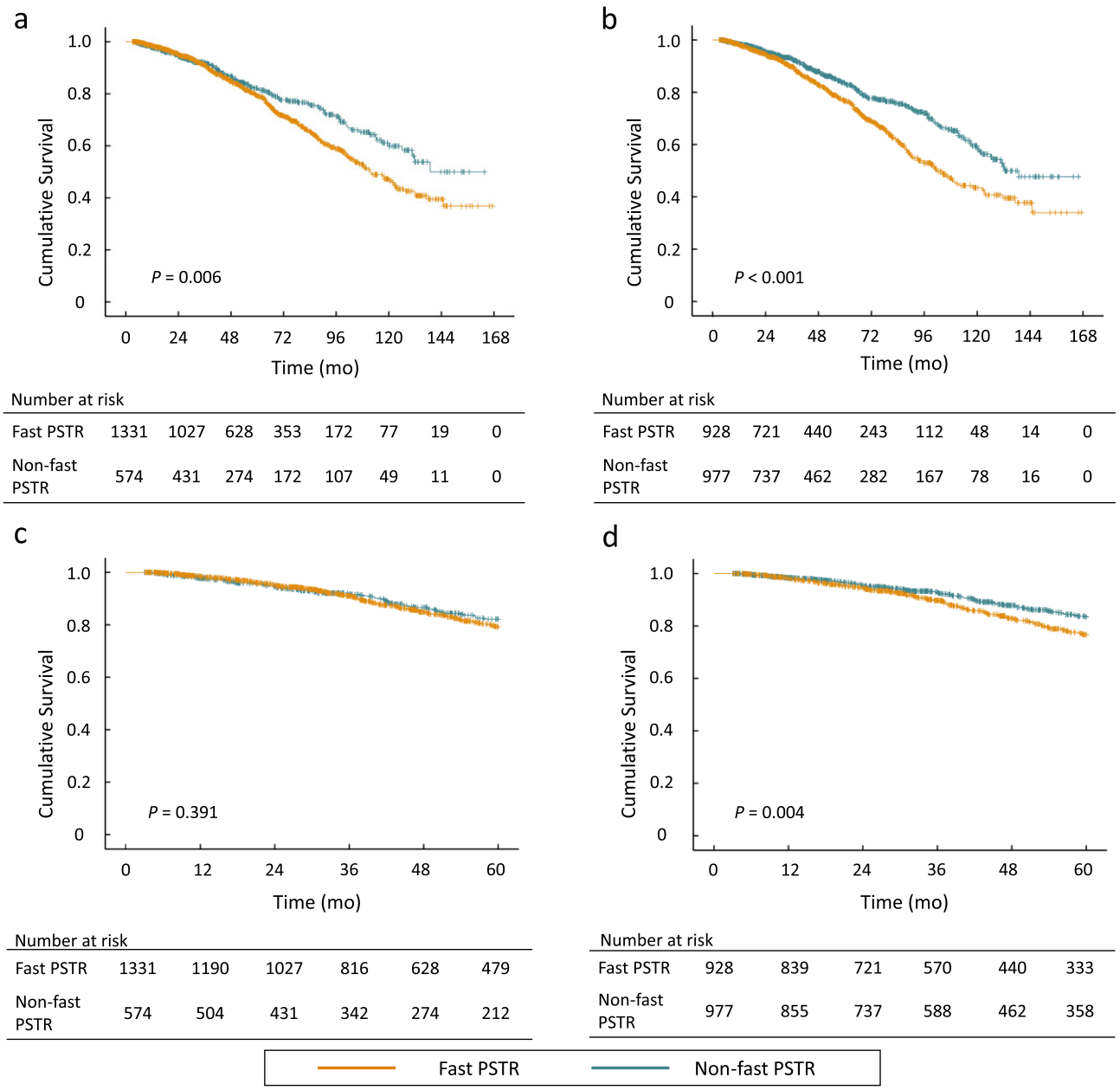

Figure 3: Kaplan–Meier curve of patient survival under different classification methods. The Kaplan–Meier curve illustrated the patient survival between fast PSTR and non-fast PSTR groups under traditional classification (**a**) and our centre classification (**b**) for the whole follow-up period, and those under traditional classification (**c**) and our centre classification (**d**) for 5-year follow-up period.

Primary endpoint—all-cause mortality

During a median of 45.7 (24.9–76.3) months of follow-up, 427 (22.3%) deaths were recorded. As shown in the Kaplan–Meier curves of the two groups under the two classification methods, the accumulated incidence of death for patients in the fast PSTR group was higher than that in the non-fast PSTR group ($P = .006$ and $P < .001$, respectively) (Fig. 3a and b). However, after shortening the follow-up time to 5 years, patients in the non-fast PSTR group no longer showed a survival advantage under the traditional classification ($P = .391$), whereas a survival advantage still existed under the classification based on our centre data ($P = .004$) (Fig. 3c and d). We then used 4-h D/Pcr as a continuous variable to analyse the relationship between 4-h D/Pcr and mortality using the RCS. The RCS curve showed an S-shape, with the inflection point of 4-h D/Pcr being 0.71 (Fig. 4a). Patients with a 4-h D/Pcr higher than 0.71 had an increased mortality risk, whereas those with a 4-h D/Pcr lower than 0.71 had a decreased mortality risk. After adjusting for age, gender, BMI, CCI, 24-h urine volume, BUN, peritoneal Kt/V, Hb, serum calcium, serum phosphorus and iPTH in Cox proportional hazards regression models, the fast PSTR group showed an increased mortality risk [HR 1.27, 95% confidence interval (CI) 1.02–1.59] compared with non-fast PSTR group under the classification of CCV (0.71), whereas the risk of mortality failed to show any statistical difference (HR 1.24, 95% CI 0.97–1.59) between the two

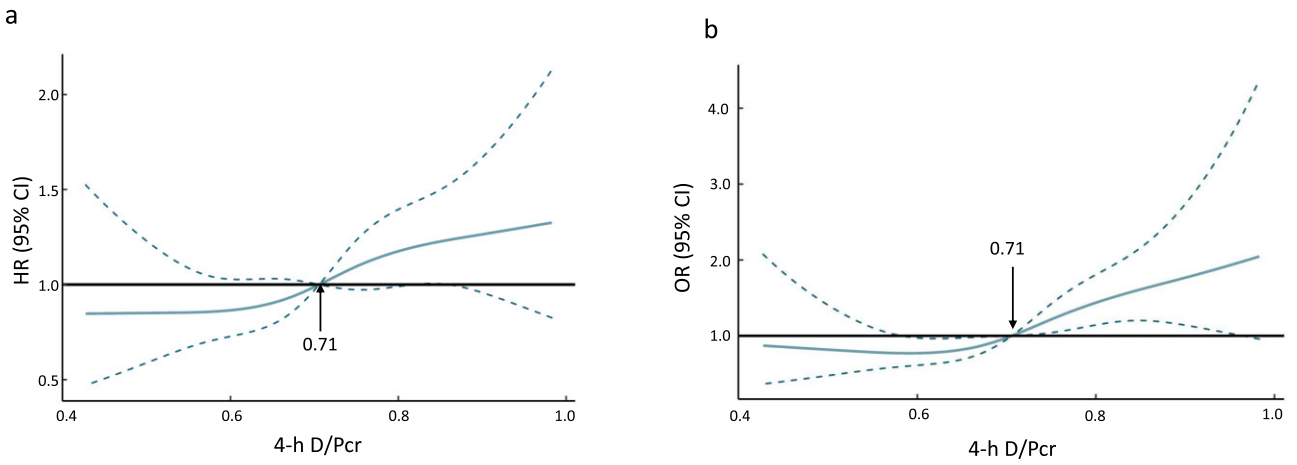

Figure 4: Association of 4-h D/Pcr with mortality (**a**) and UF insufficiency (**b**). The risk of mortality was adjusted for age, gender, BMI, CCI, 14-h urine volume, BUN, peritoneal Kt/V, Hb, serum calcium, serum phosphorus and iPTH. The risk of UF insufficiency was adjusted for age, gender, BMI, GFR, glucose exposure and PD vintage.

Table 2: Association between PSTR of PD patients and all-cause mortality according to categories of traditional classification (0.65) and our centre classification (0.71).

Model	HR (95% CI)	P-value
Traditional classification		
Unadjusted	1.36 (1.09–1.69)	.006
Model 1[a]	1.19 (0.95–1.49)	.122
Model 2[b]	1.25 (0.99–1.58)	.065
Model 3[c]	1.24 (0.97–1.59)	.086
Our centre classification		
Unadjusted	1.55 (1.28–1.88)	<.001
Model 1[a]	1.26 (1.03–1.54)	.026
Model 2[b]	1.30 (1.05–1.60)	.015
Model 3[c]	1.27 (1.02–1.59)	.033

Traditional classification: cutoff value, 0.65; fast PSTR group, >0.65; non-fast PSTR group, ≤0.65. Our centre classification: cutoff value, 0.71; fast PSTR group, >0.71; non-fast PSTR group, ≤0.71.
[a]Adjusted for age, gender, BMI and CCI score.
[b]Adjusted for Model 1 variables plus 24-h urine volume, BUN and peritoneal Kt/V.
[c]Adjusted for Model 2 variables plus Hb, potassium, calcium, phosphorus and iPTH.

Table 3: The association between PSTR of PD patients and UF insufficiency according to categories of traditional classification (0.65) and our centre classification (0.71).

Model	OR (95% CI)	P-value
Traditional classification		
Unadjusted	1.74 (1.34–2.26)	<.001
Adjusted[a]	1.60 (1.15–2.22)	.005
Our centre classification		
Unadjusted	1.84 (1.47–2.31)	<.001
Adjusted[a]	1.67 (1.25–2.24)	.001

Traditional classification: cutoff value, 0.65; fast PSTR group, > 0.65; non-fast PSTR group, ≤0.65. Our centre classification: cutoff value, 0.71; fast PSTR group, > 0.71; non-fast PSTR group, ≤0.71.
[a]Adjusted for age, gender, BMI, GFR, glucose exposure and PD vintage.

groups under the classification of TCV (0.65) (Table 2). The time-dependent ROC illustrated that the AUC of our centre classification was higher than that of the traditional classification, but failed to show any statistical significance after adjustment (Supplementary data, Fig. S1).

Secondary endpoint—UF insufficiency and technical survival

The baseline median 24-h UF volume was 140 (–120 to 450) mL, and the baseline median 4-h UF volume of PET was 250 (140–350) mL. UF insufficiency occurred in 394 (20.7%) patients in this cohort. We first treated 4-h D/Pcr as a continuous variable and found a modest inverse correlation between 4-h D/Pcr and the 24-h UF volume ($r = –0.335$, $P < .001$) (Supplementary data, Fig. S2). We then used different classification methods and found that the fast PSTR group had a lower 24-h UF volume (CCV: $r = –0.324$, $P < .001$; TCV: $r = –0.317$, $P < .001$) (Supplementary data, Table S1). In adjusted logistic regressions, both methods of classification showed that the fast PSTR groups had a higher UF insufficiency risk after adjusting for age, gender, BMI, GFR, glucose exposure and PD vintage (CCV: OR 1.67, 95% CI 1.25–2.24, $P = .001$; TCV: OR 1.60, 95% CI 1.15–2.22, $P = .005$), but the OR of fast PSTR groups under CCV was slightly higher than that under TCV (Table 3). We then used 4-h D/Pcr as a continuous variable to analyse the relationship between 4-h D/Pcr and UF insufficiency using an RCS curve. Interestingly, the RCS curve also showed an S-shape with an inflection point at 4-h D/Pcr 0.71 (Fig. 4b). When the 4-h D/Pcr was ≤0.71, the change in UF insufficiency risk with 4-h D/Pcr was not obvious. However, the risk of UF insufficiency increased with the 4-h D/Pcr after the 4-h D/Pcr above 0.71.

During the follow-up period, 356 individuals (18.7%) transferred to haemodialysis. Of 356 patients who transferred to haemodialysis, 153 (43.0%) were ascribed to infection, 68 (19.1%) to inadequacy dialysis, 37 (10.4%) to dialysate complications (including pleuro-abdominal fistula, hernia, peritoneal-scrotal dialysate leakage), 35 (9.8%) to UF insufficient, 6 (1.7%) to catheter dysfunction, 3 (0.8%) to encapsulating peritoneal sclerosis and 54 (15.2%) to other causes (Supplementary data, Fig. S3). As shown in Supplementary data, Fig. S4, neither of the two classification methods showed a difference in technique survival rates between the groups ($P = .842$ and $P = .744$, respectively).

DISCUSSION

In this observational cohort study, 4-h D/Pcr was normally distributed, with a mean of 0.71 (± 0.11), which was quite different from that traditionally used (0.65). Thus, the cutoff value of 4-h D/Pcr for recognizing patients with fast PSTR in our centre was 0.71. CCV showed better predicted all-cause mortality than TCV. Furthermore, under both classification methods, the fast PSTR group showed a higher risk of UF insufficiency than the non-fast PSTR group, but the OR of our centre classification for UF insufficiency was slightly higher than that of the traditional classification.

Previous studies have reported a large discrepancy in baseline mean 4-h D/Pcr (0.56–0.81) [1, 3, 4, 13, 16–22]. A study including 764 dialysis facilities in the USA reported a mean 4-h D/Pcr of 0.65 (±0.12) [3]. However, a multicentre study involving Italy, Korea, and the UK reported their mean 4-h D/Pcr was 0.71 (±0.12) [13]. In our PD centre, 4-h D/Pcr was normally distributed with a mean of 0.71 (±0.11). It is speculated that the varying race, methods for creatinine measurement, dextrose/glucose concentrations used for PET, etc., accounted for the discrepancy. Thus, the traditionally used cutoff value may be not applicable to all centres and regions. Our study provide data supporting the suggestion by ISPD guideline that PD centres should find a cutoff value suitable for their own centres.

The increased mortality risk associated with fast PSTR has been well documented [3, 4, 11]. However, some studies also claimed that fast PSTR was not an independent risk factor for mortality [23, 24]. As our results showed, the fast PSTR group in our centre classification had a higher mortality risk than the non-fast PSTR group, but this was not presented under the traditional classification. Numerous previous studies have elucidated that faster PSTR is associated with lower fluid removal [10], higher glucose exposure [25], incidence of peritonitis [5], development of encapsulating peritoneal sclerosis [6], increased peritoneal protein clearance [26], malnutrition [27] and development of encapsulating peritoneal sclerosis [6], all of which are closely related to mortality in PD patients. We speculate that unreasonable cutoff value selection may explain the inconsistency in mortality risk among studies. In addition, the inflection point of 4-h D/Pcr was 0.71 in RCS of mortality, which is equal to CCV. These results further support the ISPD guideline and the reasonability of CCV.

In our cohort, fast PSTR was an independent risk factor for UF insufficiency, which is consistent with previous studies [28, 29]. Smit *et al*. reported that patients with UF failure had higher D/Pcr values than those without UF failure (0.86 vs 0.71), and that a high mass transfer area coefficient for creatinine was one of the causes of UF failure [28]. This can be explained using the three-pore model of the peritoneal membrane [30]. More specifically, a fast PSTR can cause an early loss of the osmotic gradient, which further results in less water transport through ultra-small and small pores [12]. Additionally, the different UF insufficiency risks of fast PSTR identified by the two classification methods suggest that CCV may have a better ability to predict UF insufficiency, and is more reasonable. However, unlike previous studies [4, 25], neither our centre classification nor the traditional classification showed a difference in technical survival rates between the groups. We think that the main reason for this was that only a small minority of patients (9.8%) suffer technical failure due to UF insufficiency in our PD centre.

Our study is the first to investigate the CCV of 4-h D/Pcr in identifying PD patients with fast PSTR, and to compare its efficacy with the TCV in predicting clinical outcomes. However, this study had some limitations. First, we only used PET data obtained at baseline to predict mortality, UF insufficiency and technical survival. As we known, the peritoneal function of patients varied over follow-up, which may influence the outcomes. Second, the AUC for mortality was low for both classification methods, which may mean that the selected variables were not perfect. However, our target was to compare the efficacy of different cutoff values in predicting mortality, while did not fit a sophisticated model. Moreover, as an observational study, we were unable to conclude a causal relationship between PSTR status and clinical outcomes.

CONCLUSION

In summary, the cutoff value of 4-h D/Pcr for identifying patients with fast PSTR was 0.71 in our centre. Compared with the TCV, it was superior in predicting mortality in patients with PD, especially in terms of short-term mortality. The CCV also had an advantage in predicting UF insufficiency. It is concluded that centres should establish their own cutoff values to accurately assess the peritoneal transfer status of patients on PD, which will assist in achieving optimal dialysis effects and improving outcomes.

SUPPLEMENTARY DATA

Supplementary data are available at *ckj* online.

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AUTHORS' CONTRIBUTIONS

X.Y. proposed the concept of the study. J.G. analysed and interpreted the data, and prepared the draft of the article and revised it under the supervision of X.Y. and Q.G. X.Y. was responsible for the management of the PD centre and setting up the PD database. X.Y., Q.G., H.M., F.H. and H.Y. were in charge of the treatment of PD patients. R.L., J.L., C.Y., X.D., Y.P. and H.W. collected the data. All authors approved the final version of the manuscript.

CONFLICT OF INTEREST STATEMENT

The authors have no financial conflicts of interest to declare. The results presented in this article have not been published previously in whole or part, except in abstract format.

DATA AVAILABILITY STATEMENT

The data underlying this article will be shared on reasonable request to the corresponding author.

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