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CHANGES IN ECHOCARDIOGRAPHIC PARAMETERS ACCORDING TO THE RATE OF RESIDUAL RENAL FUNCTION DECLINE IN INCIDENT PERITONEAL DIALYSIS PATIENTS

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Abstract:
Residual renal function (RRF) is associated with left ventricular (LV) hypertrophy as well as all-cause and cardiovascular (CV) mortality in patients with end-stage renal disease. However, no studies have yet examined the serial changes in echocardiographic findings according to the rate of RRF decline in incident dialysis patients. A total of 81 patients who started peritoneal dialysis (PD), and who underwent baseline and follow-up echocardiography within the first year of PD were recruited.
Patients were dichotomized into ''faster'' and ''slower'' RRF decline groups according to the median values of RRF decline slope (1.60 mL/min/y/1.73 m2). Baseline RRF and echocardiographic parameters were comparable between the 2 groups.
During the first year of PD, there were no significant changes in LV end-diastolic volume index (LVEDVI), left atrial volume index (LAVI), or LV mass index (LVMI) in the ''faster'' RRT decline group, while these indices decreased in the ''slower'' RRT decline group. The rate of RRF decline was a significant determinant of 1-year changes in LVEDVI, LAVI, and LVMI. The linear mixed model further confirmed that there were significant differences in the changes in LVEDVI, LAVI, and LVMI between the 2 groups (P ¼ 0.047, 0.048, and 0.001, respectively). During a mean follow-up duration of 31.9 months, 4 (4.9%) patients died.
Compared with the ''slower'' RRF decline group, CV composite (20.29/100 vs 7.18/100 patient-years [PY], P ¼ 0.098), technique failure (18.80/100 vs 4.19/100 PY, P ¼ 0.006), and PD peritonitis (15.73/100 vs 4.95/100 PY, P ¼ 0.064) developed more frequently in patients with ''faster'' RRF decline rate. On multivariate Cox regression analysis, patients with ''faster'' RRF decline rate showed 4.82-, 4.44-, and 7.37-fold higher risks, respectively, for each clinical outcome. Preservation of RRF is important for conserving cardiac performance, resulting in an improvement in clinical outcomes of incident PD patients.

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INTRODUCTION:

Cardiovascular (CV) disease is prevalent and is the most common cause of morbidity and mortality in patients with end-stage renal disease (ESRD). Even though coronary artery disease (CAD) and arrhythmia are not uncommon, left ventricular (LV) hypertrophy (LVH) is the most frequent CV manifestation in these patients.

LVH is known to be present in more than 70% of incident ESRD patients and to be a significant independent predictor of CV mortality not only in patients with hypertension but also in those with ESRD. Recently, several studies have demonstrated that left atrial volume (LAV) index (LAVI) is also an independent predictor of mortality in patients with ESRD.

The importance of residual renal function (RRF) has been highlighted in patients with ESRD. The loss of RRF is closely linked to fluid overload, sodium retention, hypertension, LVH, malnutrition, inflammation, endothelial dysfunction, and anaemia, all of which contribute to a higher prevalence of CV disease in dialysis patients.

In addition, lower RRF was associated with increased morbidity and mortality in ESRD patients receiving haemodialysis (HD) or peritoneal dialysis (PD). Especially in PD patients, RRF and fluid removal, but not the dose of PD, were shown to be independent predictors of mortality. Even though a number of previous studies found that RRF was a favourable factor for clinical outcomes in dialysis patients, most of these studies used only baseline RRF and clarified its impact on all-cause and/or CV mortality. RRF decreases progressively after the initiation of dialysis, and thus it has been surmised that the impact of the rate of RRF decline rather than baseline RRF is a more important determinant of clinical outcomes of ESRD patients on dialysis.

Supporting this point of view, a study by Liao et al revealed that patients with faster RRF decline after PD initiation had worse clinical outcomes, and that the rate of RRF decline rather than baseline RRF was an independent risk factor for technique failure in incident PD patients. The authors also found that the rate of RRF decline was superior to baseline RRF in predicting patient and technique survival in these patients. However, they did not elucidate the mechanism of poor clinical outcomes in patients with a rapid decline of RRF. Since there is a close relationship between RRF and cardiac dysfunction in ESRD patients, the rate of RRF decline may have an effect on the changes in cardiac function, which is closely associated with patient morbidity and mortality. The specifics of this hypothesis have never been explored.

METHODS:

Patients:

Initial recruitment for this retrospective cohort study included 148 patients who started PD and who underwent echocardiography, urea kinetic study, and peritoneal equilibration test within 1 month of PD initiation. Among these patients, we excluded those who were younger than 18 years, were anuric (<100 mL/d) at the time of PD initiation, had a history of HD or kidney transplantation prior to PD, had an underlying active malignancy or acute infection, or died within 3 months of PD initiation. Patients who did not receive follow-up echocardiography within the first year of PD treatment were also excluded. Thus, a total of 81 incident PD patients were included in the final analysis.

Collection of Data:

Demographic and clinical data, including age, gender, body mass index (calculated as weight/height), primary renal disease, comorbidities, and medications, were recorded at the time of PD initiation. CAD was defined as a history of angioplasty, coronary artery bypass grafts, myocardial infarction, or angina, while peripheral arterial disease (PAD) was defined as a history of claudication, ischemic limb loss and/or ulceration, or peripheral revascularization procedure. Laboratory data were measured from fasting blood samples, which were drawn at 2 hours after the first PD exchange with 1.5% dextrose dialysate on the day when the first urea kinetic study was performed. The following variables were included: haemoglobin, blood urea nitrogen, creatinine, calcium, phosphorus, alkaline phosphatase, intact parathyroid hormone (iPTH), glucose, haemoglobin A1c (HbA1c), albumin, lipid profile, sodium, potassium, bicarbonate, iron profile, N-terminal pro–B-type natriuretic peptide, cardiac troponin T, and high-sensitivity C-reactive protein (hs-CRP). Estimated glomerular filtration rate was calculated using the 4-variable Modification of Diet in Renal Disease study equation. A peritoneal equilibrium test was conducted to determine peritoneal transport characteristics, and dialysis adequacy and RRF were measured within 1 month of beginning PD and every 6 months thereafter. Peritoneal transport characteristics were assessed using equilibration ratios between dialysate and plasma creatinine, which were determined by a standardized peritoneal equilibration test using 2 L 4.25% dextrose dwell

with dialysate samples taken at 0, 2, and 4 hours and a plasma sample at 2 hours.

Echocardiographic Measurements:

Echocardiography was performed in the morning with an empty abdomen, based on the imaging protocol recommended by the health care officials. LV systolic function was estimated by LV ejection fraction using a modified biplane Simpson method from the apical 2-chamber and 4-chamber views. LV mass was determined using the method described by Devereux et al,24 and the LV mass index (LVMI) was calculated by dividing LV mass by BSA. LVH was defined as LVMI > 131 g/m2 for men and >100 g/m2 for women.

Outcome Measures:

The aim of this study was to clarify time-dependent 1-year changes in echocardiographic parameters according to the rate of RRF decline. Independent factors associated with the RRF decline rate and the impact of RRF decline rate on clinical outcomes, such as all-cause mortality, composite of death or hospitalization, CV composite, infection composite, new-onset CV diseases, technique failure, and PD peritonitis, were also elucidated. A CV event was defined as death or hospitalization from CAD, congestive heart failure (CHF), arrhythmia, pulmonary edema, cerebrovascular disease, or PAD.

TABLE 1. Baseline Demographic and Clinical Characteristics of Study Patients

Variables	Total (N = 81)	Faster RRF Decline Group (N = 41)	Slower RRF Decline Group (N = 40)	*P*
Age, y	51.5 ± 11.7	51.0 ± 12.4	52.1 ± 11.2	0.702
Sex (male)	40 (49.4%)	23 (56.1%)	17 (42.5%)	0.221
BMI, kg/m^2	23.2 ± 3.5	23.0 ± 2.8	23.4 ± 4.1	0.670
Systolic BP, mm Hg	133.5 ± 21.6	139.7 ± 19.7	127.1 ± 21.9	0.021
Diastolic BP, mm Hg	77.3 ± 12.4	77.5 ± 12.4	77.1 ± 12.6	0.905
Pulse pressure, mm Hg	56.2 ± 16.9	62.2 ± 17.3	50.0 ± 14.1	0.004
Primary cause of end-stage renal disease				0.095
Diabetes	31 (38.3%)	20 (48.8%)	11 (27.5%)	
Hypertension	12 (14.8%)	5 (12.2%)	7 (17.5%)	
Glomerulonephritis	20 (24.7%)	9 (22.0%)	11 (27.5%)	
Interstitial nephritis	3 (3.7%)	2 (4.9%)	1 (2.5%)	
Congenital/hereditary disease	3 (3.7%)	2 (4.9%)	1 (2.5%)	
Others	6 (7.4%)	3 (7.3%)	3 (7.5%)	
Unknown	6 (7.4%)	0 (0.0%)	6 (15.0%)	
Comorbid disease				
Hypertension	73 (90.1%)	38 (92.7%)	35 (87.5%)	0.482
Diabetes	37 (45.7%)	23 (56.1%)	14 (35.0%)	0.057
Chronic lung disease	12 (14.8%)	7 (17.1%)	5 (12.5%)	0.562
CAD	19 (23.5%)	11 (26.8%)	8 (20.0%)	0.468
PAD	3 (3.7%)	2 (4.9%)	1 (2.5%)	0.999
Congestive heart failure	17 (21.0%)	9 (22.0%)	8 (20.0%)	0.829
Arrhythmia	7 (8.6%)	3 (7.3%)	4 (10.0%)	0.712
Cerebrovascular disease	17 (21.0%)	11 (26.8%)	6 (15.0%)	0.191
Connective tissue disease	11 (13.6%)	4 (9.8%)	7 (17.5%)	0.309
Liver disease	1 (1.2%)	0 (0.0%)	1 (2.5%)	0.494
Malignancy	1 (1.2%)	0 (0.0%)	1 (2.5%)	0.494
All CVD*	32 (39.5%)	17 (41.5%)	15 (37.5%)	0.715
Heart disease†	27 (33.3%)	17 (41.5%)	10 (25.0%)	0.116
Modified CCI	5.49 ± 2.47	5.77 ± 2.84	5.20 ± 2.04	0.367
Medications				
RAS blockers	72 (88.9%)	36 (87.8%)	36 (90.0%)	0.999
Diuretics	49 (60.5%)	30 (73.2%)	19 (47.5%)	0.018
β blockers	49 (60.5%)	23 (56.1%)	26 (65.0%)	0.413
CCB	62 (76.5%)	32 (78.0%)	30 (75.0%)	0.746
Nitrate	9 (11.1%)	5 (12.2%)	4 (10.0%)	0.999
Other BP medications	9 (11.1%)	6 (14.6%)	3 (7.5%)	0.482
Statin	25 (30.9%)	11 (26.8%)	14 (35.0%)	0.426
Aspirin	19 (23.5%)	10 (24.4%)	9 (22.5%)	0.841
Plavix	9 (11.1%)	6 (14.6%)	3 (7.5%)	0.482
Vitamin D	25 (30.9%)	12 (29.3%)	13 (32.5%)	0.753
Ca-based P binder	58 (71.6%)	27 (65.9%)	31 (77.5%)	0.245
Non-Ca-based P binder	7 (8.6%)	2 (4.9%)	5 (12.5%)	0.264
ESA	64 (79.0%)	35 (85.4%)	29 (72.5%)	0.155
Iron agents	66 (81.5%)	33 (80.5%)	33 (82.5%)	0.816

BMI = body mass index, BP = blood pressure, Ca = calcium, CAD = coronary artery disease, CCB = calcium channel blocker, CCI = Charlson comorbidity index, CVD = cardiovascular disease, ESA = erythropoiesis-stimulating agent, P = phosphorus, PAD = peripheral artery disease, RAS = renin–angiotensin system, RRF = residual renal function.

* Composite of CAD, PAD, congestive heart failure, arrhythmia, and cerebrovascular disease.

† Composite of CAD, congestive heart failure, and arrhythmia.

RESULTS:

The baseline demographic and clinical characteristics are shown in Table 1. The mean age was 51.5 11.8 years, and 49.4% of patients were males. The most common cause of ESRD was diabetes mellitus (DM, 38.3%), followed by glomerulonephritis (24.7%) and hypertension (14.8%). When patients were dichotomized into 2 groups according to the median value of RRF slope (1.60 mL/min/y/1.73 m2), systolic blood pressure, pulse pressure (PP), and the proportion of patients on diuretics were significantly higher in the ''faster'' RRF decline group compared with those in the ''slower'' RRF decline group. Among laboratory variables, HbA1c and iPTH levels were significantly higher in the ''faster'' RRF decline group compared with those in the ''slower'' RRF decline group, while serum ferritin levels were significantly lower. There was a trend of higher hs-CRP levels in patients with rapid RRF decline compared with those in the ''slower'' RRF decline group, but it did not reach statistical significance (Table 2).

TABLE 2. Baseline Laboratory and Peritoneal Dialysis–Related Parameters

Variables	Total (N = 81)	Faster RRF Decline Group (N = 41)	Slower RRF Decline Group (N = 40)	*P*
Hemoglobin, g/dL	10.80 ± 1.68	11.02 ± 1.69	10.58 ± 1.66	0.310
Ca, mg/dL	9.06 ± 0.57	8.98 ± 0.47	9.14 ± 0.66	0.284
P, mg/dL	4.35 ± 0.97	4.40 ± 0.96	4.31 ± 0.99	0.730
Uric acid, mg/dL	6.89 ± 1.59	6.84 ± 1.78	6.94 ± 1.40	0.812
Glucose, mg/dL	109.3 ± 45.4	117.2 ± 56.9	101.1 ± 28.1	0.167
HbA1c, %	6.22 ± 1.20	6.60 ± 1.36	5.90 ± 0.97	0.044
ALP, IU/L	67.9 ± 22.8	66.5 ± 21.5	69.4 ± 24.4	0.626
Protein, g/dL	6.16 ± 0.71	6.22 ± 0.77	6.10 ± 0.66	0.518
Albumin, g/dL	3.40 ± 0.51	3.45 ± 0.59	3.36 ± 0.43	0.507
BUN, mg/dL	49.0 ± 14.5	48.1 ± 14.7	50.0 ± 14.6	0.611
Creatinine, mg/dL	6.84 ± 2.30	6.58 ± 2.02	7.10 ± 2.57	0.378
GFR, mL/min/1.73 m^2	9.47 ± 6.54	9.44 ± 3.94	9.50 ± 8.51	0.974
Sodium, mmol/L	139.1 ± 3.3	138.9 ± 3.7	139.2 ± 3.0	0.730
Potassium, mmol/L	4.00 ± 0.63	3.92 ± 0.65	4.08 ± 0.61	0.344
Chloride, mmol/L	99.5 ± 3.9	99.3 ± 4.0	99.8 ± 3.8	0.569
Bicarbonate, mmol/L	27.2 ± 3.2	27.4 ± 3.5	27.0 ± 2.8	0.640
Intact PTH, pg/mL	164.9 (74.3–290.3)	209.4 (121.9–323.2)	117.8 (43.5–245.3)	0.049
B2MG, mg/L	21.8 ± 8.1	24.6 ± 9.0	19.8 ± 7.0	0.157
Cholesterol, mg/dL	177.9 ± 45.8	179.5 ± 48.2	176.3 ± 43.9	0.788
Triglyceride, mg/dL	131.6 ± 68.3	117.6 ± 52.8	146.6 ± 80.0	0.101
LDL-cholesterol, mg/dL	101.0 ± 39.8	106.2 ± 41.9	95.5 ± 37.4	0.306
HDL-cholesterol, mg/dL	48.1 ± 15.1	47.6 ± 14.4	48.7 ± 16.2	0.769
hs-CRP, mg/dL	4.49 (0.70–19.60)	9.57 (0.93–24.58)	1.37 (0.60–12.07)	0.057
Troponin T, ng/mL	0.034 (0.014–0.115)	0.054 (0.014–0.107)	0.031 (0.013–0.134)	0.741
NT-proBNP, pg/mL	5,278.0 (2,713.0–35,000.0)	11,211.0 (3,004.8–35,000.0)	5,278.0 (1,138.0–35,000.0)	0.740
Serum iron, µg/dL	73.8 ± 40.9	71.6 ± 45.4	76.0 ± 36.4	0.677
TIBC, µg/dL	230.0 ± 47.8	239.3 ± 47.8	220.4 ± 46.6	0.123
TSAT, %	32.3 ± 17.2	30.1 ± 18.7	34.6 ± 15.4	0.316
Ferritin, ng/mL	147.2 (68.9–264.5)	90.7 (54.4–191.6)	211.8 (91.6–374.8)	0.017
Peritoneal dialysis–related parameters				
APD	6 (7.4%)	4 (9.8%)	2 (5.0%)	0.675
Biocompatible solution	6 (7.4%)	4 (9.8%)	2 (5.0%)	0.675
Dialysate volume, mL/d	7,452.8 ± 1,444.2	7,561.9 ± 1,393.8	7,340.0 ± 1,509.8	0.553
Urine volume, mL/d	1,121.2 ± 968.4	1,176.8 ± 1,149.3	1,063.7 ± 753.1	0.652
Total weekly Kt/V urea	2.41 ± 0.62	2.40 ± 0.53	2.43 ± 0.71	0.852
Peritoneal Kt/V urea	1.50 ± 0.43	1.52 ± 0.44	1.48 ± 0.42	0.725
Renal Kt/V urea	0.91 ± 0.66	0.88 ± 0.44	0.95 ± 0.83	0.687
Total weekly CCr, L/wk/1.73 m^2	108.2 ± 36.9	112.6 ± 30.6	103.4 ± 42.6	0.335
Peritoneal CCr, L/wk/1.73 m^2	41.7 ± 10.2	42.9 ± 12.6	40.5 ± 7.0	0.364
Renal CCr, L/wk/1.73 m^2	66.4 ± 37.3	69.7 ± 29.5	62.8 ± 44.5	0.479
RRF, mL/min/1.73 m^2	4.68 ± 2.88	4.83 ± 2.15	4.52 ± 3.53	0.680
RRF slope, mL/min/y/1.73 m^2	−1.91 ± 3.40	−4.29 ± 2.86	0.54 ± 1.79	<0.001
LBM-Cr, kg	40.1 ± 10.1	40.3 ± 10.9	39.9 ± 9.3	0.872
Lean body mass, %	64.8 ± 12.5	65.8 ± 13.8	63.9 ± 11.1	0.561
nPNA, g/kg/d	0.99 ± 0.21	0.97 ± 0.21	1.01 ± 0.22	0.432
D/P creatinine, 4 h	0.6959 ± 0.1027	0.6957 ± 0.1002	0.6962 ± 0.1069	0.984
Groups of peritoneal equilibration test				0.765
High	12 (14.8%)	5 (12.2%)	7 (17.5%)	
High average	44 (54.3%)	23 (56.1%)	21 (52.5%)	
Low average	24 (29.6%)	13 (31.7%)	11 (27.5%)	
Low	1 (1.2%)	0 (0.0%)	1 (2.5%)	

On multivariate linear regression analysis, natural log values of hs-CRP (Ln hs-CRP) and baseline E/A values were significant independent factors associated with the rate of RRF decline during the first year of PD. After including the 1-year changes in echocardiographic parameters (LVEDVI, LAVI, or LVMI) into the successive models, PP, Ln hs-CRP levels, baseline E/A, and the slope of each echocardiographic parameter were found to be significant determinants of the RRF decline rate. Among 3 variances in cardiac performance, only the changes in LAVI remained statistically significant in the final model.

During a mean follow-up duration of 31.9 months, 4 (4.9%) patients died. The event rates for all-cause mortality, composite of death or hospitalization, infection composite, and new-onset CV disease were not different between the 2 groups. In contrast, compared with patients with ''slower'' RRF decline, the ''faster'' RRF decline group showed a significantly higher rate of technique failure (18.80 vs 4.19 events/100 patient-years [PY], P ¼ 0.006). CV composite (20.29 vs 7.18 events/100 PY, P ¼ 0.098) and PD peritonitis (15.73 vs 4.95 events/100 PY, P ¼ 0.064) also developed more frequently in the ''faster'' RRF decline group, but the differences did not reach statistical significance.

DISCUSSION:

A number of previous studies have shown that lower RRF is associated with increased morbidity and mortality in ESRD patients on HD or PD. Furthermore, RRF and cardiac function are known to influence each other. Therefore, some changes in echocardiographic findings are expected over time as RRF declines in incident dialysis patients, but this trend has not previously been explored. In the current study, we demonstrate for the first time that slow RRF decline is significantly associated with time-dependent decreases in LVEDVI, LAVI, and LVMI during the first year of PD, indicating an improvement in cardiac morphology and function. Furthermore, a rapid decline in RRF independently predicted adverse clinical outcomes such as CV composite, technique failure, and PD peritonitis. RRF plays a pivotal role in maintaining sodium and water balance of dialysis patients, and thus loss of RRF leads to chronic volume expansion and hypertension.

The severity of anaemia is also known to correlate with RRF. All these factors contribute to the development of LVH and left atrial enlargement (LAE). Therefore, it is a matter of course that LVH and LAE progress as RRF declines, but this trend has not previously been explored in dialysis patients. As expected, the present study found that there were increases in LVEDVI, LVESVI, and LAVI in patients with rapid RRF decline. However, these parameters were relatively decreased in the ''slower'' RRF decline group, which seemed to be partly attributed to an increase in mean RRF in this group. In contrast, LVMI was decreased in both groups even though the reduction of LVMI was significantly greater in patients with ''slower'' RRF decline. We inferred that correction of uremia and hyperparathyroidism rather than improvement in fluid balance and/or anemia contributed to the regression of LVMI in the ''faster'' RRF decline group in spite of a significant decrease in RRF.

Since the first study by Maiorca et al, which demonstrated that RRF was significantly higher in patients who survived compared with that in those who died (2.73 2.49 vs 0.33 0.86 mL/min, P ¼ 0.0005), and that RRF was an independent predictor of survival in 102 prevalent dialysis patients, many subsequent studies have shown a significant impact of RRF on the clinical outcomes in ESRD patients on HD or PD.

According to another demonstration which was based on 56% decrease in relative risk of death ($P < 0.0001$) and with an increase of 1/week in renal Kt/V urea in 740 incident HD patients may also considered.

In that study, the influence of dialysis dose on mortality was found to be significant only in anuric patients. Termorshuizen et al14 also showed that a 12% reduction in mortality rate (P ¼ 0.039) was observed for each mL/min/1.73 m2 increase in RRF in 413 incident PD patients, and that there was no significant effect of peritoneal creatinine clearance on patient survival, which was consistent with the results of previous studies by Szeto et al and Rocco et al.

CONCLUSION:

Despite these limitations, we believe that the present study is a meaningful investigation demonstrating for the first time that cardiac performance is significantly worsened or less improved in incident PD patients with rapid RRF decline. In addition, rapid RRF decline rate is found to be a significant independent predictor of adverse clinical outcomes including CV composite, technique failure, and PD peritonitis. Based on these findings, preservation of RRF is

important for conserving cardiac performance, resulting in an improvement in clinical outcome in incident PD patients.

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