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Original Article

ORIGINAL ARTICLE

Change in adiposity indices after 1 year of peritoneal dialysis: a single-center cohort study

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ABSTRACT

Background. Weight gain is common after starting peritoneal dialysis (PD). Several adiposity indices have been developed recently as potential indicators of visceral adiposity and lipid accumulation. We aim to investigate the prevalence and prognostic implications of the change in adiposity indices after 1 year of PD.

Methods. We recruited 110 patients treated with PD for 12 months. Adiposity indices, including triglyceride glucose index, lipid accumulation product, visceral adiposity index and conicity index, were measured at baseline and then 1 year after PD started. The relation between their changes (Δ) and other clinical and biochemical parameters, as well as survival and hospitalization rates were analyzed.

Results. After 1 year of PD, more than half of the patients had increased adiposity indices. The change in adipose tissue mass significantly correlated with the concomitant changes in triglyceride glucose index (ΔTyGI) ($r = 0.25$, $P = .01$), lipid accumulation product (ΔLAP) ($r = 0.27$, $P = .007$) and visceral adiposity index (ΔVAI) ($r = 0.26$, $P = .01$). ΔTyGI significantly correlated with the change in insulin resistance as represented by homeostasis model assessment of insulin resistance (HOMA-IR) ($r = 0.22$, $P = .02$), while ΔLAP and change in conicity index (ΔCI) correlated with the changes in various anthropometric parameters. However, no indices variation was associated with patient survival, technique survival or hospitalization rate.

Conclusions. Increased adiposity indices were common after 1 year of PD. The changes in adiposity indices had variable correlation with the change in adipose tissue mass, insulin resistance and anthropometric parameters. Further studies are required to identify simple metabolic parameters with a prognostic impact that could be suitable for serial monitoring.

Keywords: lipid-related indices, peritoneal dialysis, renal failure, variation

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KEY LEARNING POINTS

What was known:

- Weight gain is common after peritoneal dialysis (PD).
- Several adiposity indices are recently developed as potential indicators of visceral adiposity and lipid accumulation.

This study adds:

- After 1 year of PD, more than a half of the patients had increased adiposity indices.
- ΔTyGI significantly correlated with the change in insulin resistance, while ΔLAP and ΔCI correlated with the changes in anthropometric parameters.

Potential impact:

- Simple metabolic parameters may be prognostically relevant and are suitable for serial monitoring.

INTRODUCTION

The global prevalence of obesity and end-stage kidney disease has increased in the past decades [1, 2]. Our previous study in Hong Kong over the past 25 years showed that the prevalence of obesity substantially increased in both diabetic and non-diabetic new PD patients, and that the incidence of new-onset diabetes was significantly higher in new PD patients with pre-existing obesity or overweight than those without [3].

Along with obesity being frequent among patients starting dialysis, weight gain is common during the first 2 years of PD [4, 5]. However, the prognostic significance of weight gain after PD is still controversial. Although Castro *et al.* [6] noted that increase in waist circumference during the first 6 months of PD was associated with a higher mortality, waist circumference and weight gain cannot be compared directly. Our recent study found that weight gain during the first 2 years of PD did not appear to have any significant impact on the subsequent outcome [5].

The reason for the inconsistent relationship between weight gain and PD patients' outcome remains unclear, but the variation in the pattern of body fat distribution could be a contributing factor. In essence, visceral and subcutaneous adipose tissues have different metabolic profiles and probably prognostic implications [7, 8]. Although the degree of visceral adiposity is traditionally assessed by imaging techniques [9], several adiposity indices, such as triglyceride glucose index [10], lipid accumulation product [11], visceral adiposity index [12] and conicity index [13], have recently been developed as potential indicators of visceral adiposity and lipid accumulation. Previous studies in non-dialysis patients showed that these indices were significantly associated with visceral obesity, insulin resistance, mortality and cardiovascular events [14–18]. Notably, Cui *et al.* reported that the cumulative triglyceride glucose index in a non-dialysis cohort was associated with an increased risk of cardiovascular disease [19], suggesting that serial monitoring of adiposity indices may be a valuable clinical tool. However, the prognostic significance of adiposity indices in PD patients has not been fully explored. It has been reported that baseline triglyceride glucose index predicted cardiovascular mortality in incident PD patients [20] and increased fat mass was associated with the conicity index and a pro-inflammatory state [21]. In the present study, we investigated the prevalence and prognostic implications of the change in adiposity indices after 1 year of PD.

MATERIALS AND METHODS

The study was approved by the Joint Chinese University of Hong Kong—New Territories East Cluster Clinical Research Ethics Committee (approval numbers CREC-2008.276 and CREC-2021.367). All study procedures followed the Declaration of Helsinki. Written informed consent was obtained in all patients.

Study population

This is a retrospective analysis of a prospective, observational, cohort on incident adult PD patients in a single center. Consecutive patients who started PD between January 2011 and February 2014 were recruited. Patients who were expected to receive a living donor kidney transplant or who were transferred to other renal centers within 6 months were excluded. Assessment of adiposity indices and other clinical assessment (see below) were performed at baseline and 12 months after PD started.

Clinical data collection

Baseline demographic and clinical data were collected by chart review. After the patients were on PD for around 4 weeks, we performed baseline assessment of adiposity indices, insulin resistance indices, lipid profile, other standard biochemistry, dialysis adequacy, nutrition status, anthropometric measurements, multi-frequency bioimpedance analysis and assessment of peritoneal transport status. After 1 year of PD, all the assessments (except for peritoneal transport status) were repeated and variation was determined.

Anthropometric measurements

Anthropometric measurements, including body weight (BW), height, waist circumference (WC) and hip circumference (HC), mid-arm circumference, and triceps and subscapular skin-fold thickness were performed by a single examiner with standard methods. Body mass index (BMI) and the waist-to-hip ratio (WHR) were computed.

Adiposity indices and insulin resistance

Four adiposity indices, namely triglyceride glucose index, lipid accumulation product, visceral adiposity index and conicity index, were determined at baseline and 1 year after PD by the following formulae:

$$\text{triglyceride glucose index} = \ln\left[\frac{\text{TG (mg/dL)} \times \text{FG (mg/dL)}}{2}\right].$$

$$\text{lipid accumulation product (male)} = [\text{WC (cm)} - 65] \times \text{TG (mmol/L)}$$

$$\text{lipid accumulation product (female)} = [\text{WC (cm)} - 58] \times \text{TG (mmol/L)}.$$

$$\text{visceral adiposity index (male)} = \frac{\text{WC (cm)}}{39.68 + 1.88 \times \text{BMI}} \times \frac{\text{TG (mmol/L)}}{1.03} \times \frac{1.31}{\text{HDL (mmol/L)}}$$

$$\text{visceral adiposity index (female)} = \frac{\text{WC (cm)}}{36.58 + 1.89 \times \text{BMI}} \times \frac{\text{TG (mmol/L)}}{0.81} \times \frac{1.52}{\text{HDL (mmol/L)}}.$$

$$\text{conicity index} = \frac{\text{WC (m)}}{0.109 \times \sqrt{\frac{\text{BW (kg)}}{\text{height (m)}}}}$$

Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated by the following formula [22]:

$$\text{HOMA-IR} = \text{fasting insulin (mU/L)} \times \text{FG (mmol/L)} / 22.5.$$

Removal of toxins and nutritional assessment

In essence, total Kt/V was obtained from 24-h urine and dialysate samples collection as described previously [23]. Residual glomerular filtration rate (GFR) was calculated by the average of 24-h urinary urea and creatinine clearance [24]. Nutrition status was represented by serum albumin level, subjective global assessment (SGA) score [25], comprehensive malnutrition inflammation score (MIS) [26], normalized protein nitrogen appearance (NPNA) [27] and fat-free edema-free body mass (FEBM) [28] as determined by creatinine kinetics.

Peritoneal glucose exposure

Daily peritoneal glucose absorption ($G_{absorbed}$) was calculated by the difference between the daily glucose instilled dialysate ($G_{instilled}$), determined by the PD regimen, and the daily glucose in the PD effluent ($G_{drained}$) was determined directly by 24-h dialysate collection during the assessment of toxins remova. (see above).

Bioimpedance analysis

Multi-frequency bioimpedance spectroscopy device (Body Composition Monitor, Fresenius Medical Care, Germany) was used for the assessment of body composition as described previously [29]. In this study, we analyzed the data on lean tissue mass (LTM), adipose tissue mass (ATM), volume of over-hydration and extracellular-to-intracellular volume (E:I) ratio.

Peritoneal transport status

The standard peritoneal equilibration test [30] was performed at baseline, usually 4–6 weeks after the patients were stable on PD. The dialysate-to-plasma creatinine ratio of creatinine was computed after adjusting for the influence of glucose. The mass transfer area coefficients (MTAC) of creatinine normalized for body surface area [31] was obtained by a standard formula [32].

Clinical outcomes

After the second assessment 1 year after PD start, all patients were followed until 31 July 2023. During the follow-up period, the clinical management was at the discretion of the treating physician and was not affected by the study. Primary outcome measures of this study included patient survival, technique survival and peritonitis-free survival, all counted from the second assessment (i.e. after 1 year of PD). Secondary outcome measures included the number of hospital admission and total duration of hospitalization, both adjusted for the duration of follow-up. For patient survival analysis, recovery of kidney function, loss to follow-up, transfer to other dialysis centers, conversion to long-term hemodialysis and kidney transplant were censored. For the technique survival analysis, recovery of kidney function, loss to follow-up and transfer to other dialysis centers were censored.

Statistical analysis

Statistical analysis was performed by SPSS for Windows software version 26.0 (IBM, Armonk, NY, USA). The normality of data distribution was checked by the Kolmogorov–Smirnov test. Summary statistics were described as frequency (%) for categorical variables and mean ± standard deviation or median [interquartile range (IQR)] for continuous variables as appropriate. Paired *t*-test or Wilcoxon rank sum test was used to compare the result of baseline and first-year assessment as appropriate. Correlation between clinical parameters and the change (denoted as Δ) in individual adiposity index was explored by Pearson correlation coefficient or Spearman's rank correlation coefficient as appropriate. Survival rates were represented by the Kaplan–Meier survival curves and grouped by the quartile of the change in individual adiposity index. Univariate Cox regression analysis was further performed for the change in each adiposity index. The relation between hospitalization and the change of individual adiposity index, grouped into quartiles, was explored by linear regression model with log transformation of the hospitalization data. A *P*-value of $<.05$ was considered statistically significant. All probabilities were two-tailed.

RESULTS

We studied 110 eligible incident PD patients. Their baseline characteristics are summarized in Table 1. Their anthropometric, removal of toxins, nutrition, and body composition parameters at baseline and changes after 1 year of PD are summarized in Table 2.

Adiposity indices and their changes with PD

The baseline triglyceride glucose index, lipid accumulation product, visceral adiposity index and conicity index were 8.66 ± 0.61, 37.09 ± 26.47, 2.13 ± 1.74 and 1.29 ± 0.09, respectively. After 1 year of PD, more than a half of the patients had their adiposity indices increased. The mean changes in triglyceride glucose index (ΔTyGI), lipid accumulation product (ΔLAP), visceral adiposity index (ΔVAI) and conicity index (ΔCI) were 0.18 ± 0.69, 10.06 ± 43.44, 0.50 ± 3.35 and 0.03 ± 0.07, respectively ($P = .007$, .001, .07 and .001, respectively). There was a modest but significant inverse correlation between baseline value and the change in triglyceride glucose index ($r = -0.50$, $P < .001$)

Table 1: Baseline demographic and clinical characteristics.

No. of patients	110
Age (years)	59.53 ± 11.02
Sex, no. (%)	
Female	28 (25.5)
Male	82 (74.5)
Blood pressure (mmHg)	
Systolic	142.15 ± 20.40
Diastolic	75.53 ± 13.19
Cause of end-stage kidney disease, no. (%)	
Diabetes mellitus	60 (54.5)
Glomerulonephritis	19 (17.3)
Hypertension	11 (10.0)
Polycystic kidney disease	3 (2.7)
Urological	6 (5.5)
Unknown	11 (10.0)
Comorbidities, no. (%)	
Diabetes mellitus	69 (62.7)
Ischemic heart disease	32 (29.1)
Cerebrovascular accident	23 (20.9)
Peripheral vascular disease	9 (8.2)
Cancer	7 (6.4)
Charlson's comorbidity score	6.25 ± 2.51
Baseline peritoneal transport	
D/P4	0.69 ± 0.13
MTAC (mL/min/1.73 m^2)	10.94 ± 5.21
Nutritional indices	
MIS	6.79 ± 3.44
SGA score	5.31 ± 0.85

Data are presented as mean ± standard deviation or no. (%).
D/P4, dialysate-to-plasma ratios of creatinine at 4 h.

and in visceral adiposity index (r = –0.27, $P = .004$), while baseline lipid accumulation product and conicity index did not have significant correlation with their corresponding changes after 1 year of PD (details not shown). The internal correlation between the changes of the four adiposity indices are summarized in Supplementary data, Table S1. In essence, there were good internal correlations between ΔTyGI, ΔLAP and ΔVAI, while the ΔCI only correlated with ΔLAP.

Correlation with clinical parameters

The correlation between the changes in adiposity indices and baseline clinical and biochemical parameters are summarized in Supplementary data, Table S2, and their correlations with the corresponding changes of clinical and biochemical parameters after 1 year of PD are summarized in Table 3. In essence, ΔTyGI, ΔLAP and ΔVAI had modest but significant correlations with baseline triglyceride levels, as well as the changes in hemoglobin, lipid profiles, HbA1C, and adipose tissue mass after PD for 1 year. No variation of the adiposity indices had correlation with any baseline anthropometric measurements, bioimpedance spectroscopy findings or other nutritional parameters. ΔTyGI also had significant correlations with baseline fasting glucose, the changes in fasting glucose and HOMA-IR, while ΔLAP and ΔCI had significant correlations with the changes in anthropometric measurements but not with their baseline values. No variation of the adiposity indices correlated with the peritoneal glucose load (Table 3).

Clinical outcomes

After 1 year of PD, all patients were further followed for 35.5 (IQR 18.1 to 61.9) months (3170.8 patient-months in total). During the subsequent follow-up, 84 patients died, 9 were switched to long-term hemodialysis, 9 had kidney transplantation and 3 were transferred to other centers. The causes of death were ischemic heart diseases (22 cases), cerebrovascular accidents (7 cases), sudden cardiac arrest (4 cases), peritonitis (8 cases), non-peritonitis infection (34 cases), malignancy (3 cases), termination of dialysis (2 cases) and other specific causes (4 cases). During the follow-up period, 128 episodes of peritonitis developed in 65 patients; the peritonitis rate was 0.48 episodes per patient-year. There were 1065 hospital admissions for a total of 9178 days. The median rate of hospital admission was 2.52 episodes per year (IQR 1.24 to 4.74), and the median duration of hospitalization was 19.93 days per year (IQR 8.16 to 38.50).

The relation between the change in adiposity indices after 1 year of PD, divided into quartiles, and the clinical outcome is summarized in Table 4. The corresponding Kaplan–Meier plots of patient and technique survival are shown in Figs 1 and 2, respectively. Although the lowest quartile of ΔLAP and ΔVAI seemed to be associated with worse patient and technique survival rates (see Figs 1 and 2), a statistically significant difference was not confirmed by univariable Cox analysis (Table 4). Multivariable Cox regression analysis to adjust for other clinical factors was therefore not performed. No variation in the adiposity indices was associated with peritonitis-free survival or hospitalization.

DISCUSSION

In the present study, we found that after 1 year of PD, more than half of the patients experienced an increase in adiposity indices. The changes in triglyceride glucose index, lipid accumulation product and visceral adiposity index were internally correlated, while conicity index only correlated with lipid accumulation product. The changes in lipid accumulation product and conicity index significantly correlated with the concomitant changes in various anthropometric measurements, but not with the glucose exposure. No variation in the adiposity indices was associated with survival or hospitalization.

This is the first study to investigate the relationship between the change in adiposity indices after 1 year of PD and the subsequent clinical outcome. Similar to previous studies [4, 5], we noted that weight gain was common after PD. Specifically, we noted that LTM as determined by multi-frequency bioimpedance was not significantly changed over a year of PD, while ATM was markedly increased, which is consistent with the previous report of Verger *et al.* [33]. We also found substantial increases in waist, hip and mid-arm girths, and triceps and subscapular skin-fold thickness, as well as cholesterol levels, fasting glucose, HbA1C and HOMA-IR. However, the magnitude of weight gain only correlated with the concomitant increase in lipid accumulation product but not with other adiposity indices, and the change in an adiposity index generally correlated with the change in the parameters that constitute the calculation of the index, which is an expected phenomenon. Contrary to our expectations, we did not find any correlation between peritoneal glucose load, which we believe that could be the major

Table 2: Baseline nutrition and biochemical parameters, and their changes after 1 year of PD.

	Baseline	Change after 1 year	P-value
Anthropometry			
Body weight (kg)	64.78 ± 13.61	1.95 ± 5.24	$P < .001^a$
BMI (kg/m^2)	24.1 (21.2–26.1)	+0.76 (–0.48 to +1.90)	$P < .001^b$
WC (cm)	88.0 (80.3–95.0)	+2.0 (–1.0 to +6.0)	$P < .001^b$
HC (cm)	95.0 (89.0–99.0)	+2.0 (–1.0 to +6.0)	$P < .001^b$
WHR	0.94 (0.90–0.98)	0.00 (–0.01 to +0.02)	$P = .28^b$
Mid-arm circumference (cm)	25.0 (23.1–27.0)	+0.4 (–0.5 to +1.0)	$P < .001^b$
Triceps skin fold (mm)	9.8 (8.0–12.2)	+0.4 (–0.2 to +1.6)	$P < .001^b$
Subscapular skin fold (mm)	12.4 (6.9–15.2)	+0.3 (–0.2 to +1.4)	$P < .001^b$
Hemoglobin (g/dL)	8.92 ± 1.16	0.52 ± 1.58	$P = .001^a$
Serum albumin (g/L)	38.0 (33.3–40.0)	–2.0 (–5.0 to +0.8)	$P < .001^b$
Lipid profile (mmol/L)			
Total cholesterol	4.30 (3.63–5.10)	+0.55 (–0.20 to +1.10)	$P < .001^b$
Triglyceride	1.30 (0.90–1.90)	+0.10 (–0.30 to +0.50)	$P = .11^b$
HDL cholesterol	1.20 (1.00–1.50)	–0.10 (–0.20 to +0.20)	$P = .41^b$
LDL cholesterol	2.30 (1.70–3.10)	+0.30 (–0.10 to +0.90)	$P < .001^b$
Fasting glucose (mmol/L)	5.2 (4.8–6.4)	+0.5 (–0.1 to +1.8)	$P < .001^b$
HbA1C (%)	6.10 (5.50–6.98)	–0.10 (–0.50 to +0.10)	$P = .02^b$
HOMA-IR	3.56 (2.07–6.14)	+1.08 (–0.08 to +3.92)	$P < .001^b$
hsCRP (mg/L)	2.70 (1.10–8.90)	–0.35 (–5.01 to +1.19)	$P = .03^b$
Total Kt/V	2.09 (1.72–2.40)	–0.26 (–0.64 to +0.12)	$P = .004^b$
Residual GFR (mL/min/1.73m^2)	3.96 (2.20–6.11)	–1.21 (–0.26 to –2.50)	$P < .001^b$
NPNA (g/kg/day)	1.14 ± 0.22	–0.03 ± 0.25	$P = .32^a$
FEBM (kg)	37.0 (30.7–43.2)	+5.04 (1.70 to 11.87)	$P < .001^b$
Bioimpedance spectroscopy			
Overhydration (L)	3.9 (2.3–5.7)	–0.60 (–2.08 to +0.90)	$P = .03^b$
E:I ratio	1.02 ± 0.17	–0.01 ± 0.16	$P = .42^a$
Lean tissue mass (kg)	40.95 ± 11.02	0.05 ± 7.63	$P = .95^a$
Adipose tissue mass (kg)	19.47 ± 10.83	2.86 ± 7.55	$P < .001^a$
Peritoneal glucose exposure (mmol/day)			
$G_{instilled}$	755.6 (750.0–777.8)	–255.6 (–111.0 to –250.0)	$P < .001^b$
$G_{drained}$	158.6 (116.4–219.8)	+5.9 (–9.3 to +38.9)	$P = .01^b$
$G_{absorbed}$	579.9 (445.5–645.1)	–215.3 (–11.7 to –294.2)	$P < .001^b$

Data are presented as mean ± standard deviation or median (inter-quartile range) after normality of distribution was checked by the Kolmogorov-Smirnov Test, and compared by [a]paired Student's t test or [b]Wilcoxon rank sum test.
HDL, high-density lipoprotein; LDL, low-density lipoprotein; HbA1C, glycosylated hemoglobin; hsCRP, high sensitivity C-reactive protein; Kt/V, dialysis adequacy assessment by total urea clearance; FEBM, fat-free edema-free body mass.

contributing factor for the weight gain, and the change in the adiposity indices. The peritoneal glucose absorption was around 350–550 mmol/day, corresponding to an additional intake of 270–400 kcal per day in our cohort.

The lack of association between the change in adiposity indices and clinical outcomes of PD patients may be considered unexpected. In this study, the 2-year patient survival rate was 73.3%, which is similar to the overall PD population in Hong Kong [34]. Previous studies in non-dialysis patients have shown a significant association between these indices and visceral obesity, insulin resistance, mortality and the risk of cardiovascular events [14–18]. Theoretically, the calculation of lipid accumulation product, visceral adiposity index and conicity index include parameters related to metabolic disturbance and central obesity, while triglyceride glucose index was originally designed as a simple marker of insulin resistance [10, 35]. It would be expected that the combination of several relevant parameters could allow a more comprehensive assessment of the nutritional and metabolic profile that could be relevant to patient outcomes. Previous studies showed that baseline adiposity indices were associated with clinical outcomes. For example, triglyceride glucose index was noted to predict cardiovascular outcomes in patients with coronary artery disease [36]; lipid accumulation product, but not BMI, predicted mortality in nondiabetic patients at high risk for cardiovascular diseases [37, 38]; visceral adiposity index levels were significantly associated with all-cause and cardiovascular disease mortality in patients without kidney disease [39, 40]; and conicity index was an independent risk factor for all-cause mortality in Chinese older people [41]. Specifically in incident PD patients, baseline triglyceride glucose index predicted cardiovascular mortality [20]. The result of our present study, however, indicated that serial measurement of adiposity indices did not provide additional prognostic information to incident PD patients.

There are several limitations of our present study. First, there was likely a selection bias of our cohort because only patients who survived the first year of PD were eligible for analysis. As compared with previous studies [19, 33, 42], our present one had a smaller sample size and may not have adequate statistical power. The choice of repeating the measurement of adiposity indices after 1 year of PD was also empirical. In retrospect, it is possible that the change in adiposity indices within an early timeframe (e.g. 3 or 6 months after PD) could serve as a relevant indicator of patients' response to PD treatment. This could offer

Table 3: Correlation between change in adiposity indices over 1 year of PD and the concomitant change in clinical parameters.

Changes after 1 year	ΔTyGI	ΔLAP	ΔVAI	ΔCI
Blood pressure				
Systolic	r = 0.02, P = .86	r = 0.05, P = .61	r = –0.00, P = .97	r = –0.01, P = .89
Diastolic	r = 0.21, P = .03	r = 0.13, P = .17	r = 0.09, P = .34	r = 0.15, P = .12
Anthropometry				
Body weight	r = 0.01, P = .92	r = 0.34, P < .001	r = 0.12, P = .21	r = 0.04, P = .71
BMI	r = -0.02, P = .86	r = 0.30, P = .002	r = 0.08, P = .43	r = 0.02, P = .87
WC	r = 0.09, P = .34	r = 0.44, P < .001	r = 0.19, P = .053	r = 0.71, P < .001
HC	r = 0.08, P = .41	r = 0.39, P < .001	r = 0.15, P = .13	r = 0.49, P < .001
WHR	r = 0.10, P = .32	r = 0.21, P = .03	r = 0.13, P = .18	r = 0.49, P < .001
Mid-arm circumference	r = 0.02, P = .84	r = 0.29, P = .002	r = 0.15, P = .12	r = 0.37, P < .001
Triceps skin-fold	r = 0.01, P = .89	r = 0.26, P = .005	r = 0.08, P = .43	r = 0.32, P = .001
Subscapular skin fold	r = 0.13, P = .17	r = 0.30, P = .001	r = 0.16, P = .10	r = 0.37, P < .001
Blood result				
Hemoglobin	r = 0.27, P = .005	r = 0.28, P = .003	r = 0.23, P = .02	r = –0.08, P = .39
Serum albumin	r = 0.02, P = .87	r = 0.08, P = .38	r = 0.10, P = .32	r = –0.15, P = .12
Total cholesterol	r = 0.41, P < .001	r = 0.43, P < .001	r = 0.27, P = .004	r = –0.07, P = .50
Triglycerides	r = 0.85, P < .001	r = 0.89, P < .001	r = 0.88, P < .001	r = 0.07, P = .45
HDL cholesterol	r = –0.50, P < .001	r = –0.42, P < .001	r = –0.72, P < .001	r = –0.13, P = .17
LDL cholesterol	r = 0.32, P = .001	r = 0.30, P = .002	r = 0.23, P = .02	r = –0.05, P = .59
Fasting glucose	r = 0.58, P < .001	r = 0.13, P = .16	r = 0.16, P = .09	r = 0.10, P = .31
HbA1C	r = 0.31, P = .001	r = 0.25, P = .009	r = 0.25, P = .009	r = 0.03, P = .74
HOMA-IR	r = 0.22, P = .02	r = –0.01, P = .94	r = –0.01, P = .92	r = 0.05, P = .60
hsCRP	r = 0.00, P = .99	r = –0.09, P = .35	r = 0.04, P = .67	r = 0.05, P = .62
Total Kt/V	r = 0.03, P = .79	r = –0.17, P = .17	r = 0.04, P = .75	r = –0.09, P = .46
Residual GFR	r = –0.04, P = .77	r = –0.15, P = .23	r = –0.06, P = .63	r = –0.00, P = .99
NPNA	r = –0.17, P = .16	r = –0.23, P = .06	r = –0.18, P = .14	r = 0.05, P = .70
FEBM	r = –0.09, P = .45	r = –0.12, P = .31	r = –0.17, P = .16	r = 0.04, P = .73
Bioimpedance spectroscopy				
Over-hydration	r = –0.06, P = .54	r = –0.03, P = .79	r = –0.07, P = .50	r = 0.18, P = .07
E:I ratio	r = –0.02, P = .86	r = 0.02, P = .83	r = –0.02, P = .86	r = 0.18, P = .07
Lean tissue mass	r = –0.09, P = .39	r = –0.05, P = .65	r = –0.10, P = .33	r = –0.08, P = .44
Adipose tissue mass	r = 0.25, P = .01	r = 0.27, P = .007	r = 0.26, P = .01	r = 0.10, P = .34
Cumulative peritoneal glucose exposure in 1 year				
$G_{instilled}$	r = –0.09, P = .38	r = –0.04, P = .68	r = –0.02, P = .87	r = –0.08, P = .44
$G_{drained}$	r = 0.06, P = .53	r = 0.14, P = .16	r = 0.12, P = .21	r = –0.04, P = .71
$G_{absorbed}$	r = –0.09, P = .37	r = –0.07, P = .49	r = –0.03, P = .79	r = –0.09, P = .37

Data were compared by the Spearman's rank correlation coefficient.
HDL, high-density lipoprotein; LDL, low-density lipoprotein; HbA1C, glycated hemoglobin; hsCRP, high sensitivity C-reactive protein; Kt/V, dialysis adequacy assessment by total urea clearance; FEBM, fat-free edema-free body mass.

Table 4: Relation between the change in adiposity indices after 1 year of PD and the subsequent clinical outcome.

	ΔTyGI quartile	ΔLAP quartile	ΔVAI quartile	ΔCI quartile
Survival[a]				
Patient survival	1.02 (0.84–1.24), P = .83	1.03 (0.83–1.27), P = .81	1.07 (0.86–1.33), P = .57	1.02 (0.85–1.24), P = .81
Technique survival	0.98 (0.83–1.17), P = .86	0.93 (0.77–1.13), P = .46	1.02 (0.83–1.24), P = .87	0.97 (0.82–1.16), P = .74
Peritonitis-free survival	1.01 (0.88–1.36), P = .43	1.12 (0.89–1.41), P = .32	1.10 (0.861.41), P = .44	1.04 (0.84–1.29), P = .70
Hospitalization[b]				
No. of hospital admission	+0.00 (–0.12 to +0.12), P = .97	–0.01 (–0.13 to +0.11), P = .89	+0.03 (–0.08 to +0.15), P = .59	+0.03 (–0.09 to +0.13), P = .59
Duration of hospitalization	+0.05 (–0.15 to +0.26), P = .60	+0.11 (–0.10 to +0.31), P = .28	+0.09 (–0.13 to +0.31), P = .45	+0.09 (–0.10 to +0.27), P = .34

[a]Data were analyzed by univariate Cox regression, and expressed as hazard ratio (95% confidence interval) and its P-value.
[b]Data were analyzed by linear regression following log-transformation of the hospitalization data, and expressed as β value (95% confidence interval) and its P-value.

valuable insights for continuing PD in frail patients with multiple comorbidities, especially when a time-limited trial of PD is originally planned. However, this hypothesis needs to be tested by further studies.

In conclusion, we found that after 1 year of PD, more than half of the patients had an increase in adiposity indices, and changes in lipid accumulation product and conicity index significantly correlated with the concomitant changes in anthropometric measurements but not glucose exposure. However, in none of the adiposity indices was a change was associated with the subsequent survival or need of hospitalization. Further studies are required to identify simple metabolic parameters that are prognostically relevant and are suitable for serial monitoring.

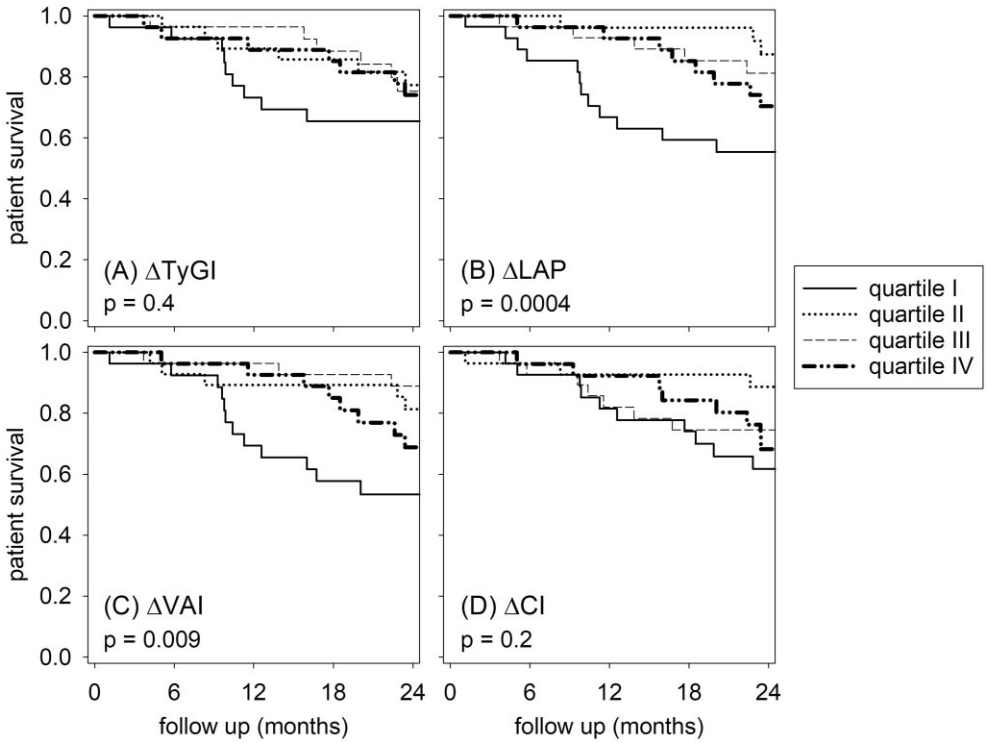

Figure 1: Kaplan–Meier plot for patient survival according to the quartile of change in (**A**) triglyceride glucose index (ΔTyGI); (**B**) lipid accumulation product (ΔLAP); (**C**) visceral adiposity index (ΔVAI); and (**D**) conicity index (ΔCI) over 1 year. Quartile I indicates the group with the lowest level. Data were compared by log-rank test.

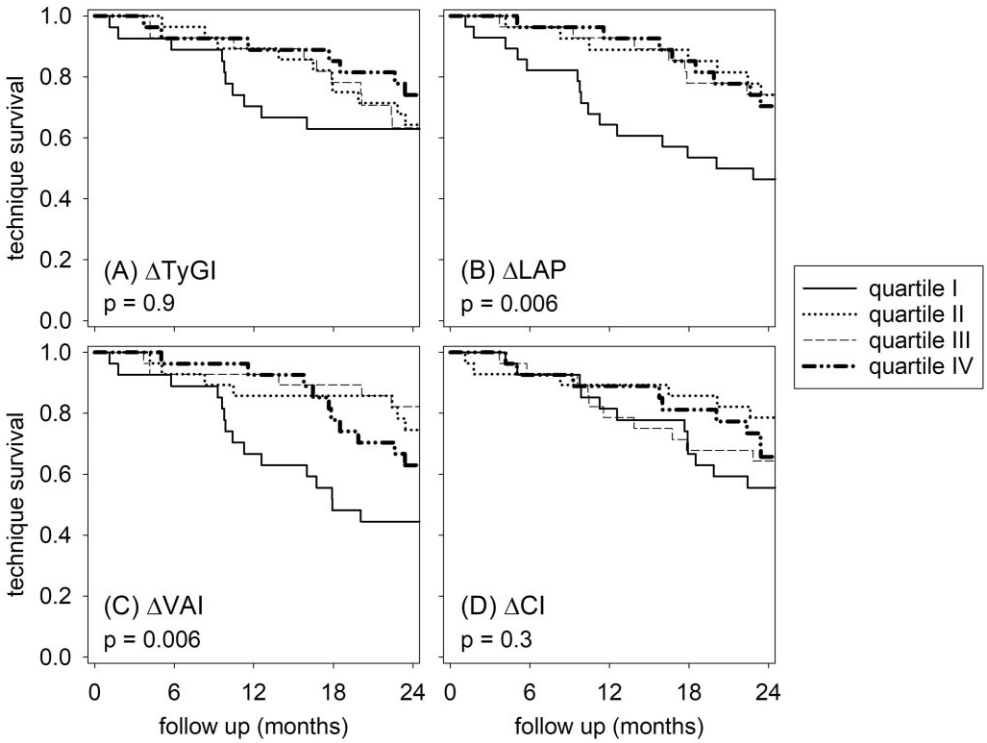

Figure 2: Kaplan–Meier plot for technique survival according to the quartile of change in (**A**) triglyceride glucose index (ΔTyGI); (**B**) lipid accumulation product (ΔLAP); (**C**) visceral adiposity index (ΔVAI); and (**D**) conicity index (ΔCI) over 1 year. Quartile I indicates the group with the lowest level. Data were compared by log-rank test.

SUPPLEMENTARY DATA

Supplementary data are available at *Clinical Kidney Journal* online.

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AUTHORS' CONTRIBUTIONS

Z.K.Y. analyzed the data and wrote the first draft of the manuscript. J.K.-C.N., G.C.-K.C. and W.W.-S.F. collected and validated the data. K.-M.C. was responsible for database maintenance and project administration. C.-C.S. was responsible for the original idea, overall supervision and writing the final version of the manuscript.

DATA AVAILABILITY STATEMENT

All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

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