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DOES BODY FAT MASS DEFINE SURVIVAL IN PATIENTS STARTING PERITONEAL DIALYSIS?

Soo Jeong Choi, Eun Jung Kim, Moo Yong Park, Jin Kuk Kim, and Seung Duk Hwang

Department of Internal Medicine, Soonchunhyang University College of Medicine, Bucheon Hospital, Korea

♦ ***Background and Aims:*** **Peritoneal dialysis (PD) is characterized by a gain in fat mass. Unlike subcutaneous fat, visceral fat is associated with metabolic syndrome and survival. We prospectively examined whether visceral or subcutaneous fat could predict outcome in patients undergoing PD.**

♦ ***Methods:*** **We studied 117 new patients (57 men) undergoing PD between February 2006 and November 2011. Baseline body composition was measured on computed tomograms. Visceral obesity was defined as a visceral fat area exceeding 100 cm^2, and subcutaneous obesity, as a subcutaneous fat area exceeding 130 cm^2.**

♦ ***Results:*** **Among the 117 patients, 37 and 29 were diagnosed with visceral and subcutaneous obesity respectively. Visceral and subcutaneous obesity were both present in 21 patients. In the study population, the 1-year and 5-year survival rates were 94% and 59%. The rates of peritonitis and exit-infection were 0.31 and 0.14 episodes per patient–year. Mortality was greater in patients with visceral obesity than in those without visceral obesity ($p = 0.005$). Visceral obesity had no influence on peritonitis and exit-infection rates. Subcutaneous obesity was associated neither with survival nor with peritonitis or exit-site infection. In a multivariate Cox regression analysis, visceral obesity was not a risk factor for poor outcome.**

♦ ***Conclusions:*** **Increased visceral fat at PD initiation is not an independent predictor of poor survival. Any impact of visceral or subcutaneous fat mass on outcomes in patients undergoing PD would be better defined by larger, long-term studies.**

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KEY WORDS: Body composition.

Correspondence to: S.D. Hwang, Division of Nephrology, Department of Medicine, Soonchunhyang University Bucheon Hospital, 170 Jomaru-ro, Wonmi-gu, Bucheon-si, Gyeonggi-do 420–767 Republic of Korea.

sd7hwang@schmc.ac.kr

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In contrast to subcutaneous fat mass, visceral fat mass is more often associated with metabolic syndrome and atherosclerosis in both obese and normal-weight individuals (1). Additionally, compared with change in subcutaneous fat mass, change in visceral fat mass is a more reliable predictor of survival in peritoneal dialysis (PD) patients (2). Although some studies have indicated that high body mass index (BMI) is a predictor of good prognosis (3,4), obesity is a risk factor for peritonitis (5). Furthermore, BMI does not reflect hydration status in patients undergoing PD. Thus, some studies have attempted to identify the impact of visceral and subcutaneous fat on survival during PD (6,7). We examined whether either visceral or subcutaneous fat mass is associated with outcome in patients undergoing PD.

METHODS

Our study was approved by the institutional review board of Soonchunhyang University Bucheon Hospital (SCHBC-IRB-10-19). We prospectively collected data for patients who started and maintained continuous ambulatory or automated PD for at least 6 months at our hospital. Exclusion criteria were age less than 18 years, current malignancy, and a life expectancy shorter than 6 months after PD start.

The primary outcome was death from any cause. Kidney transplantation, change to hemodialysis, transfer to another hospital, and patient withdrawal were censored observations. Secondary outcomes were peritonitis and exit-site infection.

Baseline data, including age, sex, weight, underlying renal disease, and presence of comorbid diseases such as diabetes mellitus (DM), hypertension, and cardiovascular disease (CVD) were collected. Body weight was determined after the abdomen had been filled with 2 L of dialysate. Cardiovascular disease was defined as the presence of coronary artery disease, congestive heart failure, cerebrovascular accident, or peripheral vascular disease.

Body mass index was calculated using the formula

BMI = weight (kg) / height (m^2).

Obesity was defined as a BMI exceeding 25 kg/m^2.

NUTRITION PARAMETERS AND SEROLOGY MARKERS

Blood samples were obtained on day 7 after the start of PD. The subjective global assessment for nutrition status was performed at the same time. Normalized protein equivalent of nitrogen appearance (8) and lean body mass (9), based on creatinine kinetics, were estimated from the results of an adequacy assessment using 24-hour effluent and urine.

For the subjective global assessment, we used a 7-point Likert-type scale to assess four items (10): weight loss, anorexia, subcutaneous fat, and muscle mass. The item scores were totalled to produce a global assessment. Scores of 1 – 2 represent severe malnutrition; 3 – 5, moderate-to-mild malnutrition; and 6 – 7, normal nutrition.

Skinfold thickness was measured at the biceps using conventional skinfold calipers (Skyndex: Caldwell, Justice and Co., Fayetteville, AR, USA). Mid-arm circumference was measured three times, and the mean value was recorded.

BODY COMPOSITION ASSESSMENT

We measured body composition using bioelectric impedance analysis (BIA: Inbody, Biospace, Seoul, Korea) on day 7 after PD start. Patients underwent BIA without socks and shoes after a dialysate fill. Multi-frequency BIA (5, 50, 250, and 500 kHz) reveals extracellular fluid, intracellular fluid, protein, mineral, and fat mass.

We measured visceral and subcutaneous fat mass using abdominal computed tomography (CT/1 Pro: GE Healthcare, Milwaukee, WI, USA) on day 7 after PD start. All subjects were examined in the supine position with both arms stretched above the head. One acquisition was made at the umbilicus level, with a slice thickness of 10 mm; exposure time varied from 3 s to 11 s. The radiation dose for the fat imaging was 1.15 mSv. Total adipose tissue area was calculated by delineating the abdomen with an electronic graph pen and computing the adipose tissue surface using an attenuation range of –150 HU to –50 HU. Visceral fat was distinguished from subcutaneous fat by tracing along the facial plane to define the internal abdominal wall. The area of each compartment was measured in square centimeters.

We defined visceral obesity as a visceral fat area (VFA) exceeding 100 cm^2 (11,12) and subcutaneous obesity as a subcutaneous fat area (SFA) exceeding 130 cm^2 (13).

STATISTICAL ANALYSIS

Data are expressed as mean ± standard deviation. Statistical differences were analyzed using the Student t-test, the chi-square test, and the Pearson correlation, as appropriate. Logistic regression analysis was used to predict variables of visceral and subcutaneous obesity. Survival rates were analyzed using Kaplan–Meier survival curves. The log-rank test was used to test differences between survival curves. A Cox proportional hazards model was used to identify independent predictors of primary and secondary outcomes. Values of $p < 0.05$ were taken to indicate statistical significance. All statistical calculations were performed using the SPSS software package (version 14.0: SPSS, Chicago, IL, USA).

RESULTS

PATIENT CHARACTERISTICS

Of 126 patients who started PD between February 2006 and November 2011, 4 were excluded because of a life expectancy of less than 6 months, and 5, because of refusal of informed consent. Thus, 117 patients were included in the study.

Table 1 shows the baseline clinical characteristics for the study patients. Mean age was 54.0 ± 13.3 years (range: 23 – 91 years). In this group, 59 patients (50.4%) had DM, and 43 (36.8%) had CVD.

NUTRITION STATUS AND FAT MASS AT PD START

Table 2 shows body composition, anthropometrics, and laboratory results for the patients at the start of PD. Compared with female patients, male patients weighed more and had higher serum creatinine and lower hematocrit and cholesterol. Body composition—including intracellular fluid, extracellular fluid, protein, and mineral mass—was greater in men than in women. Percentage body fat was greater in women than in men.

BASELINE FACTORS ASSOCIATED WITH FAT COMPOSITION

In 38 patients (32.5%), BMI exceeded 25 kg/m^2 [Figure 1(A)]. Visceral obesity was diagnosed in 37 patients (31.6%), and subcutaneous obesity, in 29 [24.8%, Figure 1(B,C)]. Visceral and subcutaneous obesity were both present in 21 patients (17.9%). After adjustments for age, sex, DM, and CVD, baseline VFA and SFA were associated with weight, triglycerides, intracellular fluid, protein mass, fat mass, mid-arm circumference, skinfold thickness, and BMI (data not shown). Baseline

TABLE 1
Characteristics of the Study Population

Variable	Value
Patients (*n*)	117
Sex [*n* (%)]	
Men	57 (48.7)
Women	60 (51.3)
Diabetes [*n* (%)]	59 (50.9)
CVD [*n* (%)]	43 (36.8)
Modality (*n* CAPD/APD/combined)	50/53/14
Age (years)	54.0±13.3
Height (cm)	161.2±11.5
Weight (kg)	61.2±11.5
Body mass index (kg/m^2)	23.5±3.2
Hematocrit (%)	31.0±4.6
Creatinine (mg/dL)	8.2±2.8
Albumin (g/dL)	3.9±0.8
Cholesterol (mg/dL)	152.5±42.9
Triglycerides (mg/dL)	121.6±86.7
hs-CRP (mg/dL)	0.8±1.5
HbA1c (%)	5.8±1.2

CVD = cardiovascular disease; CAPD = continuous ambulatory peritoneal dialysis; APD = automated peritoneal dialysis; hs-CRP = high sensitivity C-reactive protein.

VFA was correlated with SFA. Table 3 shows the predictors for visceral and subcutaneous obesity at the start of PD. Although many variables were predictors of visceral and subcutaneous obesity by univariate logistic regression analysis, multivariate logistic regression analysis showed that female sex, weight, and visceral-to-subcutaneous fat ratio were predictors of visceral and subcutaneous obesity. Age was a risk factor for visceral obesity.

FOLLOW-UP NUTRITION STATUS AND FAT MASS

Patient Survival: During the observation period [36.1 ± 20.2 months (range: 1 – 76 months)], 23 PD patients (19.7%) died. Another 5 patients underwent kidney transplantation, 22 changed to hemodialysis (HD), and 5 transferred to other hospitals. In the study cohort, the 1-year and 5-year survival rates were 94% and 59% respectively.

Univariate Cox regression analysis showed that several variables at the start of PD—including age, DM, CVD, triglycerides, creatinine, fat mass, fat mass percentage, visceral-to-subcutaneous fat ratio, and visceral obesity—were closely related to survival. Compared with patients not having visceral obesity, those with visceral obesity experienced poorer survival (Figure 2). However, a multivariate Cox regression analysis model that included age, sex, DM, CVD, visceral obesity, creatinine, triglycerides, high-sensitivity C-reactive protein, and normalized protein equivalent of nitrogen appearance revealed that only age and serum creatinine were independent risk factors for a poor prognosis (Table 4).

TABLE 2
Body Composition and Anthropometrics of the Study Population at Peritoneal Dialysis Start

Variable	Men (*n*=57)	Women (*n*=60)	*p* Value
Age (years)	51.6±13.0	56.2±13.2	0.064
Weight (kg)	68.1±10.0	54.8±8.8	0.000
Body mass index (kg/m^2)	24.1±3.1	23.0±3.3	0.090
Hematocrit (%)	30.1±4.1	32.0±4.9	0.026
Creatinine (mg/dL)	9.7±2.8	6.9±1.9	0.000
Albumin (g/dL)	3.8±0.5	3.9±0.9	0.411
Cholesterol (mg/dL)	133.6±35.0	170.4±42.3	0.000
Triglycerides (mg/dL)	105.5±74.9	136.6±94.6	0.055
hs-CRP (mg/dL)	1.0±1.3	0.6±1.7	0.227
HbA1c (%)	5.9±1.1	5.7±1.2	0.364
Intracellular fluid (L)	24.5±3.6	17.7±2.9	0.000
Extracellular fluid (L)	14.0±2.6	11.5±6.2	0.008
Protein mass (kg)	13.3±2.0	9.7±1.7	0.000
Mineral mass (kg)	3.4±1.4	2.7±1.2	0.011
Fat mass (kg)	13.1±5.7	13.8±6.2	0.502
Percentage body fat (%)	18.8±6.2	25.1±8.1	0.000
Visceral fat area (cm^2)	85.7±58.1	84.2±56.1	0.885
Subcutaneous fat area (cm^2)	94.7±52.5	110.9±67.9	0.153
Mid-arm circumference (cm)	28.5±3.0	27.2±7.2	0.110
Skinfold thickness (cm)	22.4±7.2	22.4±7.2	0.962
SGA score	26.2±7.5	25.5±2.9	0.098
nPNA (g/kg/day)	1.04±0.24	1.08±0.30	0.453
Total Kt/V	2.18±1.80	2.53±1.31	0.236
Diabetes	29	30	0.855
Cardiovascular disease	23	20	0.450

hs-CRP = high sensitivity C-reactive protein; SGA = subjective global assessment; nPNA = normalized equivalent of protein nitrogen appearance.

Peritonitis and Exit-Site Infection: During the study period, 49 patients experienced a mean of 2.3 ± 1.5 episodes of peritonitis (range: 1 – 6 episodes), and 25 patients experienced a mean of 2.0 ± 1.4 exit-site infections (range: 1 – 6 infections). The peritonitis and exit-site infection rates were, respectively, 0.31 and 0.14 episodes per patient–year. Univariate Cox regression

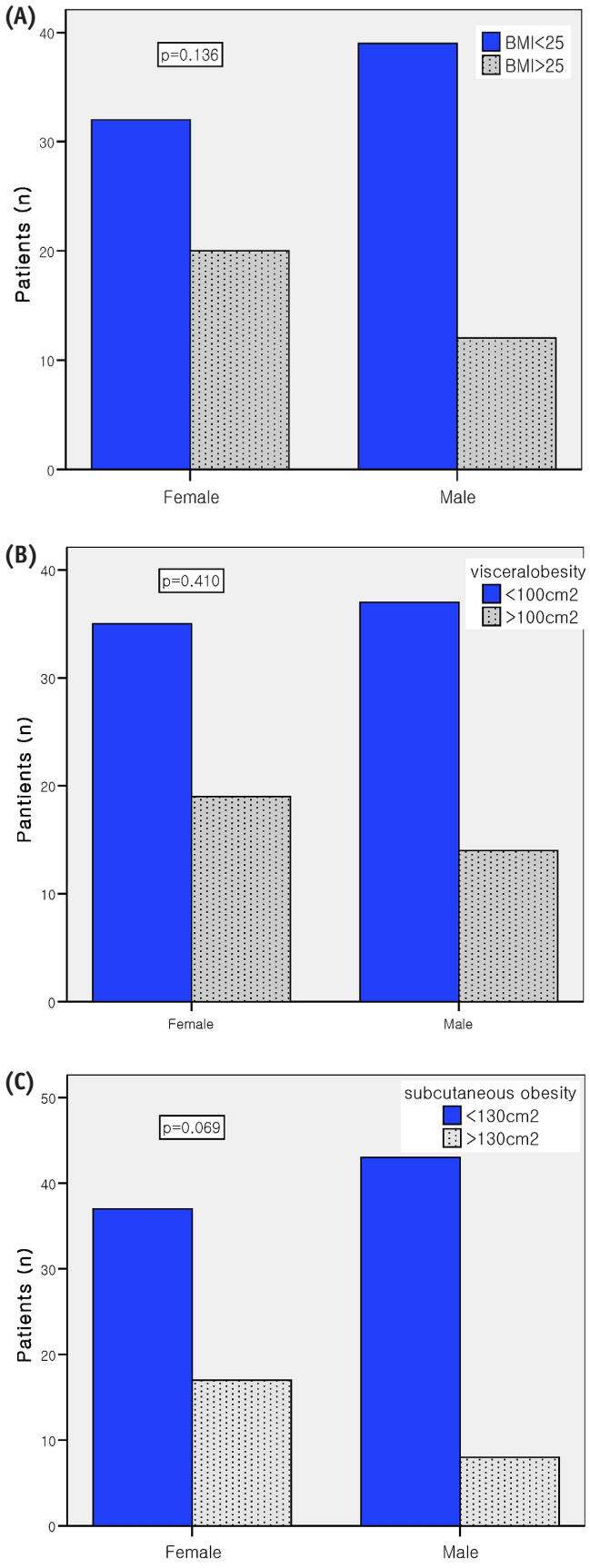

Figure 1 — Comparison of (A) obesity, (B) visceral obesity, and (C) subcutaneous obesity, by sex.

analysis showed that none of the studied variables were associated with peritonitis and exit-site infection.

DISCUSSION

An increase in body fat is a common finding in patients undergoing PD (14,15). Excess adipose tissue, particularly visceral fat, is associated with insulin resistance, hyperglycemia, dyslipidemia, hypertension, and prothrombotic and proinflammatory states in patients undergoing dialysis (1,16–18). Those conditions may predict the development of protein–energy wasting (19).

Some reports have indicated that a high BMI is a predictor of good prognosis (3,4); however, BMI does not reflect hydration status, and being overweight is a known risk factor for survival (5,20,21). Given that patients undergoing PD have continuously fluctuating weight according to use of dialysate, we analyzed visceral and subcutaneous fat areas by computed tomography. In our previous study (7), body weight increased continuously during a 12-month study period, but visceral and subcutaneous fat mass did not change during that time. In the present study, we showed the clinical significance of separating visceral and subcutaneous fat. Neither visceral nor subcutaneous fat mass differed according to sex (Table 2), but female sex was associated with visceral and subcutaneous obesity (Table 3). Those findings contrast with the results published by Sanches *et al.* (17), who conducted a cross-sectional study in patients with non-dialysis chronic kidney disease, showing that visceral fat was greater in men than in women and that subcutaneous fat was greater in women.

We determined visceral and subcutaneous obesity based on the definitions proposed by other investigators—that is, VFA greater than 100 cm^2 and SFA greater than 130 cm^2 (11–13). In the present study, visceral obesity was associated with poorer survival (Figure 2), but it was not a risk factor for poor survival according to the multivariate Cox regression analysis. Visceral obesity was not associated with peritonitis and exit-infection (data not shown). Obesity (BMI > 25) was not associated with mortality in our study (data not shown). Those results are consistent with the results of Cordeiro *et al.* (22), who used a conicity index for abdominal fat deposition. Because BMI does not distinguish between muscle and fat, those authors proposed a useful tool to identify individuals who have abdominal obesity, but who are not necessarily obese or overweight. In contrast, we analyzed the impact of volume overload, as in a previous report (23). However, the extracellular-to-intracellular water ratio is not a predictor of survival in patients undergoing PD. Caution should be used when interpreting volume

TABLE 3
Predictors of Visceral and Subcutaneous Obesity at Peritoneal Dialysis Start

Predictor	Visceral obesity			Subcutaneous obesity		
	p Value	Exp(B)	95% CI	*p* Value	Exp(B)	95% CI
Univariate						
Age (years)	0.006	1.047	1.014 to 1.082	0.722	1.006	0.974 to 1.038
Sex (female)	0.421	1.380	0.630 to 3.023	0.080	2.178	0.910 to 5.213
Weight (kg)	0.001	1.068	1.026 to 1.111	0.001	1.079	1.032 to 1.128
History of CVD	0.028	2.463	1.104 to 5.496	0.300	0.637	0.271 to 1.496
Hematocrit (%)	0.031	0.903	0.823 to 0.991	0.092	0.919	0.834 to 1.014
Triglycerides (mg/dL)	0.004	1.008	1.003 to 1.014	0.013	1.006	1.001 to 1.012
BMI (kg/m^2)	0.000	1.496	1.257 to 1.781	0.000	1.710	1.369 to 2.135
Fat mass (kg)	0.000	1.328	1.158 to 1.522	0.000	1.369	1.187 to 1.576
V/S ratio	0.001	7.475	2.563 to 27.796	0.057	0.319	0.098 to 1.035
MAC (cm)	0.001	1.340	1.121 to 1.601	0.001	1.926	1.315 to 2.819
Skinfold thickness (cm)	0.039	1.067	1.003 to 1.136	0.002	1.114	1.039 to 1.195
Multivariate[a]						
Sex (female)	0.001	35.435	4.675 to 268.572	0.009	20.375	2.106 to 197.09
Age	0.019	1.077	1.012 to 1.146	0.162	1.050	0.981 to 1.124
Weight	0.000	1.253	1.119 to 1.403	0.028	1.165	1.016 to 1.336
V/S ratio	0.000	15.458	3.446 to 69.329	0.008	0.017	0.001 to 0.350

CI = confidence interval; CVD = cardiovascular disease; BMI = body mass index; V/S ratio = visceral-to-subcutaneous fat ratio; MAC = mid-arm circumference.
[a] Model included sex, age, weight, history of CVD, diabetes, hematocrit, triglycerides, and visceral-to-subcutaneous fat ratio.

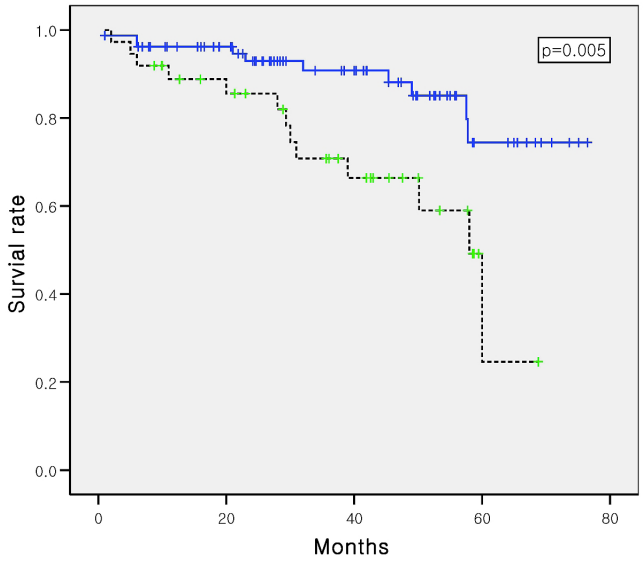

Figure 2 — Kaplan–Meier survival curves for patients with (dotted line) and without (solid line) visceral obesity (log-rank $p = 0.005$).

status in PD patients, because body fluid distribution is influenced by nutrition status, aging, and sex (24,25).

As in our previous study (7), we found that visceral and subcutaneous fat did not change, leading us to hypothesize that initial visceral or subcutaneous fat mass influences patient outcome. The visceral and subcutaneous fat masses are biologically distinct (26,27). Axelsson *et al.* (28) showed that the relationship between fat mass and inflammatory biomarkers differed for truncal and non-truncal fat mass. McDonald *et al.* (5) showed that a higher BMI at the commencement of PD is a significant risk factor for peritonitis, although the mechanism is unknown. Those authors suggested some possible reasons, including the greater difficulty that obese patients might experience with exit-site care and assessment, and a greater tendency toward underdialysis in obese patients. Our study did not find visceral or subcutaneous obesity to be a risk factor for exit-site infection, peritonitis, or survival. Other reports using body weight have been unable to determine a significant effect of body size on the risk for peritonitis (29,30). Twardowski and Prowant reported that a high BMI was associated with an increased risk for early exit-site infection (31).

Contrary to our expectation, multivariate Cox regression did not demonstrate a clinical impact of visceral or subcutaneous fat mass on outcome. It showed that age and serum creatinine at the start of PD were associated with survival. We therefore suggest that increased visceral fat at the initiation of PD is not an independent predictor of poor outcome.

TABLE 4
Predictors of Mortality by Univariate and Multivariate Cox Analysis

Predictor	Univariate			Multivariate[a]		
	p Value	Exp(B)	95% CI	*p* Value	Exp(B)	95% CI
Age (years)	0.000	1.104	1.062 to 1.148	0.034	1.058	1.004 to 1.115
Sex (female)	0.285	1.601	0.676 to 3.791	0.508	1.465	0.473 to 4.533
Diabetes (present)	0.007	3.635	1.423 to 9.286	0.605	1.413	0.381 to 5.239
History of CVD	0.003	3.615	1.485 to 8.803	0.360	1.721	0.539 to 5.499
BMI (kg/m^2)	0.783	1.016	0.907 to 1.138			
Weight (kg)	0.697	0.993	0.956 to 1.031			
Creatinine (mg/dL)	0.000	0.649	0.510 to 0.827	0.049	0.725	0.527 to 0.999
hs-CRP (mg/dL)	0.118	1.198	0.955 to 1.501	0.469	1.108	0.840 to 1.462
Triglycerides (mg/dL)	0.045	1.004	1.000 to 1.007	0.291	1.002	0.998 to 1.007
Obesity						
Visceral	0.007	3.098	1.353 to 7.094	0.655	1.320	0.391 to 4.458
Subcutaneous	0.358	1.519	0.623 to 3.702			
Intracellular fluid (L)	0.058	0.893	0.794 to 1.004			
Fat mass (kg)	0.009	1.074	1.018 to 1.133			
Fat percentage (%)	0.000	1.098	1.042 to 1.156			
V/S ratio	0.001	1.389	1.146 to 1.684			
nPNA (g/kg/day)	0.021	0.112	0.017 to 0.717	0.071	0.208	0.038 to 1.142

CI = confidence interval; CVD = cardiovascular disease; BMI = body mass index; hs-CRP = high sensitivity C-reactive protein; V/S ratio = visceral-to-subcutaneous fat ratio; nPNA = normalized equivalent of protein nitrogen appearance.
[a] Model included age, sex, diabetes status, history of CVD, visceral obesity, creatinine, triglycerides, hs-CRP, and nPNA.

To the best of our knowledge, this report is the first to use determination of fat mass by computed tomography to investigate the clinical significance of visceral and subcutaneous fat in patients with PD. Despite subanalyses of the results using factors such as age less than 65 years, non-DM status, and stable PD, we could not show any clinical significance for visceral and subcutaneous obesity in PD patients.

Our study has some limitations. We did not determine levels of inflammatory cytokines and adipokines or of insulin resistance, which might have identified an effect of visceral and subcutaneous fat. The small size of the patient sample resulted in low statistical power. Furthermore, we selected patients with a life expectancy of 6 months or more for the study, and those patients had a nutrition status that was better than the nutrition status in patients having a life expectancy of less than 6 months.

CONCLUSIONS

Increased visceral fat at the initiation of PD is not an independent predictor of poor survival. The impact of visceral or subcutaneous mass on outcomes in patients undergoing PD would be better defined by larger, long-term studies.

DISCLOSURES

The authors have no conflicts of interest to declare.

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