



Prevalence of dynapenic obesity and sarcopenic obesity and their associations with cardiovascular disease risk factors in peritoneal dialysis patients

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Background: Dynapenic obesity and sarcopenic obesity increase cardiovascular disease (CVD) and mortality in nonuremic patients. The present study was designed to determine the prevalence of dynapenic obesity and sarcopenic obesity and their associations with CVD risk factors in peritoneal dialysis (PD) patients.

Methods: All eligible PD patients in Tehran peritoneal dialysis centers were included in this cross-sectional study. Skeletal muscle mass and fat mass were assessed using bioelectrical impedance analysis. Muscle strength and physical performance were determined using hand grip strength and a 4-meter walk gait speed test, respectively. In addition, a 5-mL blood sample was obtained from each patient.

Results: The prevalence of dynapenic obesity and sarcopenic obesity were 11.4% and 3.8% in PD patients, respectively. Serum high-sensitive C-reactive protein (hs-CRP), soluble intercellular adhesion molecule type 1, triglyceride, total cholesterol, and low-density lipoprotein cholesterol were significantly higher in PD patients with dynapenic obesity than in dynapenic nonobese and nondynapenic nonobese patients. Similarly, serum concentrations of CVD risk factors in PD patients with sarcopenic obesity were higher than in nonsarcopenic nonobese patients, but these differences were statistically significant only for serum hs-CRP and triglyceride. In addition, muscle strength and skeletal muscle mass percentage were negatively associated with markers of inflammation and dyslipidemia, whereas body fat percentage was positively associated with these CVD risk factors.

Conclusion: This study indicates that although the prevalence of dynapenic obesity and sarcopenic obesity are relatively low in PD patients, these disorders may be associated with CVD risk factors.

Keywords: Cardiovascular risk factors, Dynapenic obesity, Peritoneal dialysis, Sarcopenic obesity

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Introduction

Dynapenia, defined as low muscle strength [1,2], and sarcopenia, characterized by low skeletal muscle mass (SMM) plus low muscle function (i.e., low muscle strength and/or low physical performance) [3,4], are common in dialysis patients and are associated with high morbidity, especially cardiovascular disease (CVD), and increased mortality [2,3,5,6]. In contrast, overweight and

obesity in dialysis patients are inversely associated with all-cause and cardiovascular mortality [7,8]. However, two studies in chronic kidney disease (CKD) patients, including dialysis patients, showed that sarcopenic obesity (i.e., the concurrence of sarcopenia and obesity) did not decrease mortality [9,10]. Also, some studies in nonuremic patients have indicated that dynapenic obesity (i.e., the concurrence of dynapenia and obesity) and sarcopenic obesity increase CVD and mortality [11–15]. To our knowledge, no study has yet been performed on associations of dynapenic obesity or sarcopenic obesity with CVD risk factors in dialysis patients, especially peritoneal dialysis (PD) patients. In addition, few studies have been conducted to assess prevalence of dynapenic obesity and sarcopenic obesity in CKD patients. One study in hemodialysis patients showed that the prevalence of sarcopenic obesity ranged from 2% to 74% in women and 12% to 62% in men based on various definitions [9]. In another study, sarcopenic obesity was present in 9.7% of predialysis CKD patients [10]. According to the available literature, no investigation has reported the prevalence of sarcopenic obesity and dynapenic obesity in PD patients. Therefore, the present study was designed to determine the prevalence of dynapenic obesity and sarcopenic obesity and their associations with CVD risk factors in PD patients.

Methods

Study design and participants

This investigation was a cross-sectional study. The study protocol was approved by the Ethics Committee of the National Nutrition and Food Technology Research Institute of Iran. The study was in adherence with the Declaration of Helsinki. Written, informed consent was obtained from all patients before initiating the study.

All eligible PD patients (n = 79) in Tehran peritoneal dialysis centers were included in this study. The causes of renal failure in the participating patients were diabetes mellitus (n = 30, 38.0%), hypertension (n = 15, 19.0%), glomerulonephritis (n = 4, 5.0%), urinary infection (n = 4, 5.1%), polycystic kidney disease (n = 3, 3.8%), nephrolithiasis (n = 2, 2.5%), nephrotic syndrome (n = 4, 5.1%) and other or unknown causes (n = 17, 21.5%).

Inclusion criteria were:

- Age \geq 18 years
- Continuous ambulatory peritoneal dialysis for at least six months

Exclusion criteria were:

- The presence of edema, based on physical examination by a physician
- Peritonitis and other infectious diseases

After the treatment of edema, peritonitis, and other infectious diseases, these PD patients were enrolled in our study.

Body composition and dietary assessments

All anthropometric and body composition measurements were performed after a 12- to 14-hour fast, with an empty urinary bladder and gastrointestinal tract, and without dialysate in the peritoneal cavity. Dry weight was measured to the nearest 0.1 kg, and height to the nearest 0.5 cm.

SMM and fat mass were assessed using bioelectrical impedance analysis by Body Composition Analyzer X-Contact 356 (Jawon Medical Co., Seoul, Korea). Skeletal muscle mass index (SMI) was calculated as the ratio of SMM in kilograms to the square of body height in meters ($SMI = SMM/height^2$) [4,16,17]. Several cutoffs have been proposed to determine low muscle mass. In the present study, we considered $SMI < 10.76 \text{ kg/m}^2$ for men and $SMI < 6.76 \text{ kg/m}^2$ for women as cutoffs to diagnose low muscle mass [16,17]. Two studies in CKD patients, including hemodialysis patients, indicated that sarcopenia based on these cutoffs was associated with mortality [16,17]. Muscle strength was assessed based on hand grip strength (HGS) by means of a hydraulic hand dynamometer (Exacta™; North Coast Medical, Gilroy, CA, USA). HGS was measured three times in the dominant hand with a 30-second rest interval between trials [11,18] and the maximum value was considered as the measure of the patient's muscle strength [11]. In our study, low muscle strength was defined as $HGS < 26 \text{ kg}$ for men and $HGS < 18 \text{ kg}$ for women [19]. Physical performance was determined by a 4-meter walk gait speed test [4,19]. Each patient was asked to walk at his/her usual speed on a 4-meter course [4]. The time was recorded by a chronometer in seconds. A gait speed lower than 0.8 m/sec was an indicator of low physical performance [4,19]. The diagnosis of sarcopenia was based on the presence of low SMM

plus low muscle function (i.e., low muscle strength and/or low physical performance) [4], whereas dynapenia was determined on the basis of low muscle strength [1,2,4]. Obesity was defined as percentage of total body fat greater than 35% in women and 25% in men [20].

The dietary intakes of patients were assessed using a three-day dietary recall, for three consecutive days [21], by a trained dietitian. Patients' diets were analyzed by Nutritionist IV software (N-Squared Computing, San Bruno, CA, USA) adjusted for some Iranian foods, especially Iranian breads and cheeses, to determine daily intake of energy and protein.

Biochemical assessments and dialysis adequacy

In this study, after a 12- to 14-hour fast, 5-mL blood was obtained from each patient. After clotting at room temperature (20–25°C), blood samples were centrifuged at 2,500 rpm for 15 minutes. The sera were separated into small aliquots and were frozen at –80°C until they were used.

The serum concentrations of high-sensitive C-reactive protein (hs-CRP), soluble intercellular adhesion molecule type 1 (sICAM-1), and lipoprotein (a) [Lp (a)] were measured using enzyme-linked immunosorbent assay (ELISA) kits (ZellBio GmbH, Ulm, Germany). The intra-assay coefficients of variations (CVs) for serum hs-CRP, sICAM-1, and Lp (a) were 4.0%, 3.3% and 5.5%, respectively. Serum malondialdehyde (MDA) concentration was assessed using colorimetry method by commercial kits (ZellBio GmbH), with an intra-assay CV of 5.8%. Serum triglyceride, total cholesterol, and serum high-density lipoprotein cholesterol (HDL-C) were assessed using various colorimetry methods by commercial kits (Pars-Azmoon, Tehran, Iran) with the aid of a Selectra 2 Autoanalyzer (Vital Scientific, Spankeren, the Netherlands). Intra-assay CVs for these biochemical parameters were less than 3%. As serum triglyceride concentrations in all participating patients were less than 400 mg/dL, serum low-density lipoprotein cholesterol (LDL-C) was estimated using the Friedwald equation [22].

Dialysis adequacy (as total Kt/V per week) was determined for each patient by a Kt/V calculator using information recorded in patient files, including blood urea concentration, 24-hour urine volume, urine urea concentration, 24-hour dialysate drain volume, dialysate

urea concentration, weight, height, and age [23]. From among the 79 PD patients, information regarding Kt/V index was available only for 65 patients. The peritoneal equilibration test for glucose was performed for each patient based on a 2-L 4.25% dextrose dwell with dialysate samples at 0 and 4 hours during the dwell period. The ratio of dialysate glucose concentration at time 4 to dialysate glucose level at time zero (D4/D0) was determined and percentage of glucose absorbed from the dialysate was then calculated based on the $1 - D4/D0$ formula [24].

Statistical analysis

Quantitative data are displayed as the mean \pm standard error. Statistical analysis of the data was performed using Statistical Package for the Social Sciences for Windows (version 21.0; IBM Co., Armonk, NY, USA). A chi-square test was used to determine associations between qualitative variables. All quantitative parameters had normal distribution according to the Kolmogorov–Smirnov test. We used one-way analysis of variance (ANOVA) to compare quantitative parameters among the groups. If the result of the ANOVA test was significant, the Bonferroni test was used for multiple comparisons. In addition, we adjusted the effects of two confounding factors; gender and diabetes, on serum concentrations of CVD risk factors by multiple linear regression analysis, and then compared CVD risk factors among the groups. Pearson's correlation coefficient was used to determine associations between quantitative variables. A *P* value less than 0.05 was considered statistically significant.

Results

In this study, the prevalence of dynapenic obesity in PD patients was 11.4%. Obesity without dynapenia and dynapenia without obesity were present in 12.7% and 31.6% of PD patients, respectively. In addition, 44.3% of PD patients were nondynapenic and nonobese (Table 1). There were no significant differences among these four categories of PD patients with regard to age, gender, dialysis vintage, dialysis adequacy, percentage of absorbed glucose from dialysis solution, total intake of energy and protein, or intake of statins, gemfibrozil, and levothyroxine, whereas a significant difference was observed among these four categories of PD patients with regard to the

Table 1. Characteristics of the peritoneal dialysis patients classified according to dynapenia and obesity

Characteristic	Dynapenic obesity	Nondynapenic obesity	Dynapenia & nonobesity	Nondynapenic & nonobesity
Prevalence	9 (11.4)	10 (12.7)	25 (31.6)	35 (44.3)
Age (yr)	56.0 ± 3.0	51.0 ± 6.0	59.5 ± 3.0	50.0 ± 2.0
Gender, male	3 (33.3)	3 (30.0)	10 (40.0)	19 (54.3)
Diabetes*	6 (66.7)	2 (20.0)	13 (52.0)	9 (25.7)
Dialysis vintage (yr)	2.6 ± 0.5	2.4 ± 0.4	3.7 ± 0.6	2.9 ± 0.4
Dialysis adequacy (Kt/V)	1.8 ± 0.2	2.2 ± 0.3	2.3 ± 0.1	2.0 ± 0.1
Percent of absorbed glucose from dialysis solution (%)	72.0 ± 0.0	68.0 ± 0.0	67.0 ± 0.0	69.0 ± 0.0
Total energy intake (kcal/kg/d)	28.0 ± 2.5	28.0 ± 2.0	28.0 ± 1.5	29.0 ± 1.0
Total protein intake (g/kg/d)	0.8 ± 0.1	0.8 ± 0.1	0.8 ± 0.1	0.9 ± 0.1
Intake of statins	5 (55.6)	7 (70.0)	14 (56.0)	19 (54.3)
Intake of gemfibrozil	0	0	0	0
Intake of levothyroxine	1 (11.1)	1 (10.0)	3 (12.0)	2 (5.7)
Body mass index (kg/m ²)	30.0 ± 2.0 [†]	29.0 ± 1.0 [†]	24.0 ± 1.0	24.0 ± 0.5
Body fat percentage (%)	34.0 ± 2.0 [†]	34.5 ± 1.0 [†]	20.0 ± 2.0	19.0 ± 2.0
Skeletal muscle percentage (%)	36.0 ± 1.0 [†]	36.0 ± 1.0 [†]	44.0 ± 1.0	45.0 ± 1.0
Body fat mass (kg)	27.0 ± 2.0 [†]	26.0 ± 1.0 [†]	12.0 ± 1.0	12.0 ± 1.0
Skeletal muscle mass (kg)	26.0 ± 2.0	27.0 ± 2.0	26.0 ± 1.0	29.0 ± 1.0
Skeletal muscle mass index (kg/m ²)	10.5 ± 0.6	10.5 ± 0.5	11.0 ± 0.4	11.0 ± 0.3
Muscle strength (kg)	12.0 ± 2.0 [†]	25.0 ± 3.0	15.0 ± 1.0 [‡]	28.0 ± 1.5
4-meter walk gait speed (m/sec)	7.0 ± 2.0	6.0 ± 1.0	7.0 ± 1.0	4.5 ± 0.2

Data are presented as number (%), mean ± standard error, or number only.

*A significant difference among four categories of the peritoneal dialysis patients ($P < 0.05$).

[†] $P < 0.05$ versus dynapenia & nonobesity, and versus nondynapenic & nonobesity; [‡] $P < 0.01$ versus nondynapenic obesity, and versus nondynapenic & nonobesity.

presence of diabetes ($P < 0.05$, Table 1).

Mean body mass index (BMI) was significantly higher in PD patients with dynapenic obesity and nondynapenic obesity as compared with dynapenic nonobese patients and nondynapenic nonobese patients ($P < 0.05$, Table 1). Body fat percentage was significantly higher in PD patients with dynapenic obesity and nondynapenic obesity as compared with dynapenic nonobese and nondynapenic nonobese patients ($P < 0.05$, Table 1). In contrast, skeletal muscle percentage was significantly lower in PD patients with dynapenic obesity and nondynapenic obesity in comparison with dynapenic nonobese and nondynapenic nonobese patients ($P < 0.05$, Table 1). Muscle strength was significantly lower in dynapenic obese and dynapenic nonobese patients as compared with nondynapenic obese and nondynapenic nonobese patients ($P < 0.01$, Table 1).

Serum concentrations of hs-CRP, sICAM-1, triglyceride, total cholesterol, and LDL-C were significantly higher in PD patients with dynapenic obesity than in dynapenic

nonobese and nondynapenic nonobese patients ($P < 0.05$, Table 2). There were no significant differences among PD patients with dynapenic obesity and the other three categories of PD patients with regard to serum concentrations of MDA, Lp (a), and HDL-C (Table 2).

In PD patients with nondynapenic obesity, serum hs-CRP concentration was significantly higher as compared with nondynapenic nonobese patients ($P < 0.05$, Table 2). Serum sICAM-1 concentration was significantly higher in PD patients with nondynapenic obesity than in dynapenic nonobese and nondynapenic nonobese patients ($P < 0.05$). Also, serum HDL-C was lower in PD patients with nondynapenic obesity than in dynapenic nonobese and nondynapenic nonobese patients ($P < 0.05$, Table 2).

Muscle strength was significantly negatively correlated with serum hs-CRP ($r = -0.25$, $P = 0.03$), sICAM-1 ($r = -0.24$, $P = 0.03$), triglyceride ($r = -0.28$, $P = 0.01$), and total cholesterol ($r = -0.25$, $P = 0.03$), whereas there were no significant correlations between muscle strength and serum concentrations of MDA, Lp (a), LDL-C, and HDL-

Table 2. Serum concentrations of cardiovascular disease risk factors in the peritoneal dialysis patients classified according to dynapenia and obesity

Parameter	Dynapenic obesity	Nodynapenic obesity	Dynapenia & nonobesity	Nodynapenic & nonobesity
hs-CRP (mg/L)	8.0 ± 0.4 ^{†‡}	6.8 ± 0.7 [†]	4.5 ± 0.6	4.4 ± 0.6
sICAM-1 (ng/mL)	288 ± 41 ^{†‡}	385 ± 29 ^{†‡}	177 ± 25	142 ± 22
MDA (μmol/L)	7.3 ± 1.0	5.2 ± 1.2	8.2 ± 1.1	6.3 ± 0.6
Lp (a) (mg/dL)	48.0 ± 11.0	62.0 ± 12.0	56.0 ± 10.0	60.0 ± 6.0
Triglyceride (mg/dL)	244 ± 29 ^{†‡}	185 ± 30	164 ± 16	148 ± 10
Total cholesterol (mg/dL)	225 ± 37 ^{†‡}	182 ± 26	171 ± 7	153 ± 6
LDL-C (mg/dL)	139 ± 39 ^{†‡}	110 ± 26	90 ± 7	80 ± 5
HDL-C (mg/dL)	41.0 ± 4.0	35.0 ± 2.0 ^{†‡}	48.0 ± 3.0	44.0 ± 1.5

Data are presented as mean ± standard error.

HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitive C-reactive protein; LDL-C, low-density lipoprotein cholesterol; Lp (a), lipoprotein (a); MDA, malondialdehyde; sICAM-1, soluble intercellular adhesion molecule type 1.

[†]*P* < 0.05 versus dynapenia & nonobesity; [‡]*P* < 0.05 versus nondynapenic & nonobesity.

Table 3. Correlations of body composition indicators and muscle strength with serum concentrations of cardiovascular disease risk factors in the peritoneal dialysis patients

Parameter	Body fat percentage		Muscle strength		Skeletal muscle percentage	
	r	<i>P</i> value	r	<i>P</i> value	r	<i>P</i> value
hs-CRP	0.29	0.01	-0.25	0.03	-0.29	0.01
sICAM-1	0.60	0.001	-0.24	0.03	-0.60	0.001
MDA		NS		NS		NS
Lp (a)		NS		NS		NS
Triglyceride	0.39	0.001	-0.28	0.01	-0.38	0.001
Total cholesterol	0.24	0.03	-0.25	0.03	-0.24	0.03
LDL-C		NS		NS		NS
HDL-C	-0.22	0.05		NS	0.22	0.06

HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitive C-reactive protein; LDL-C, low-density lipoprotein cholesterol; Lp (a), lipoprotein (a); MDA, malondialdehyde; NS, not significant; sICAM-1, soluble intercellular adhesion molecule type 1.

C (Table 3). In addition, body fat percentage was significantly positively correlated with serum hs-CRP ($r = 0.29$, $P = 0.01$), sICAM-1 ($r = 0.60$, $P < 0.01$), triglyceride ($r = 0.39$, $P < 0.01$), and total cholesterol ($r = 0.24$, $P = 0.03$), whereas it was significantly negatively correlated with serum HDL-C ($r = -0.22$, $P = 0.05$). There were no significant correlations between body fat percentage and serum concentrations of MDA, Lp (a), and LDL-C (Table 3).

Of all the PD patients, 3.8% had sarcopenic obesity, 20.2% had nonsarcopenic obesity, 7.6% were sarcopenic nonobese patients, and 68.4% were nonsarcopenic nonobese patients (Table 4). There were no significant differences among these four categories of PD patients with regard to age, presence of diabetes, dialysis vintage, dialysis adequacy, percentage of absorbed glucose from dialysis solution, total intake of energy and protein, or intake of statins, gemfibrozil, and levothyroxine, whereas

a significant difference was observed among these four categories with regard to gender ($P < 0.05$, Table 4).

Mean BMI in PD patients with sarcopenic obesity was not significantly different from nonsarcopenic obese, sarcopenic nonobese, and nonsarcopenic nonobese patients, whereas BMI was significantly higher in PD patients with nonsarcopenic obesity as compared with sarcopenic nonobese and nonsarcopenic nonobese patients ($P < 0.01$, Table 4). Body fat percentage was significantly higher in PD patients with sarcopenic obesity and nonsarcopenic obesity as compared with sarcopenic nonobese and nonsarcopenic nonobese patients ($P < 0.01$, Table 4). In contrast, skeletal muscle percentage was significantly lower in PD patients with sarcopenic obesity and nonsarcopenic obesity in comparison with sarcopenic nonobese and nonsarcopenic nonobese patients ($P < 0.01$, Table 4).

Table 4. Characteristics of the peritoneal dialysis patients classified according to sarcopenia and obesity

Parameter	Sarcopenic obesity	Nonsarcopenic obesity	Sarcopenia & nonobesity	Nonsarcopenic & nonobesity
Prevalence (%)	3 (3.8)	16 (20.2)	6 (7.6)	54 (68.4)
Age (yr)	52.0 ± 7.0	53.0 ± 4.0	56.0 ± 6.0	53.0 ± 2.0
Gender, male*	2 (66.7)	4 (25.0)	6 (100.0)	23 (42.6)
Dialysis vintage (yr)	2.5 ± 1.3	2.5 ± 0.3	5.0 ± 1.5	3.0 ± 0.3
Diabetes	2 (66.7)	6 (37.5)	0	22 (40.7)
Dialysis adequacy (Kt/V)	1.3 ± 0.1	2.1 ± 0.2	2.0 ± 0.2	2.1 ± 0.1
Percent of absorbed glucose from dialysis solution (%)	78 ± 0.1	68 ± 0.0	66 ± 0.0	68 ± 0.0
Total energy intake (kcal/kg/d)	25 ± 3.0	29 ± 2.0	30.0 ± 1.5	29 ± 1.0
Total protein intake (g/kg/d)	0.6 ± 0.1	0.8 ± 0.1	0.9 ± 0.06	0.8 ± 0.04
Intake of statins	2 (66.7)	10 (62.5)	2 (33.3)	31 (57.4)
Intake of gemfibrosil	0	0	0	0
Intake of levothyroxine	0	2 (12.5)	2 (33.3)	3 (5.6)
Body mass index (kg/m ²)	26.0 ± 2.0	30.0 ± 1.0 [†]	22.0 ± 1.0	24.0 ± 0.5
Body fat percentage (%)	36.0 ± 5.0 [†]	35.0 ± 1.0 [†]	20.0 ± 2.0	20.0 ± 1.0
Skeletal muscle percentage (%)	34.5 ± 3.0 [†]	35.5 ± 1.0 [†]	44.5 ± 1.0	45.0 ± 1.0
Body fat mass (kg)	27.0 ± 7.0 [†]	26.0 ± 1.0 [†]	13.0 ± 2.0	12.0 ± 1.0
Skeletal muscle mass (kg)	26.0 ± 5.0	27.0 ± 1.0	28.0 ± 2.0	28.0 ± 1.0
Skeletal muscle mass index (kg/m ²)	9.0 ± 0.8	11 ± 0.4	10 ± 0.2	11.0 ± 0.3
Muscle strength (kg)	11.0 ± 6.0	20.5 ± 2.0	24.0 ± 3.0	23.0 ± 1.0
4-meter walk gait speed (m/sec)	4.5 ± 2.5	6.5 ± 1.0	4.0 ± 0.5	5.5 ± 0.5

Data are presented as number (%), mean ± standard error, or number only .

*A significant difference among the four categories of peritoneal dialysis patients ($P < 0.05$).

[†] $P < 0.001$ versus sarcopenia & nonobesity, and versus nonsarcopenic & nonobesity.

Table 5. Serum concentrations of cardiovascular disease risk factors in the peritoneal dialysis patients classified according to sarcopenia and obesity

Parameter	Sarcopenic obesity	Nonsarcopenic obesity	Sarcopenia & nonobesity	Nonsarcopenic & nonobesity
hs-CRP (mg/L)	8.0 ± 0.3 [†]	6.8 ± 0.7 [†]	6.0 ± 1.4	4.3 ± 0.5
sICAM-1 (ng/mL)	251.0 ± 95.0	385.0 ± 29.0 ^{††}	180.0 ± 52.0	153.0 ± 17.0
MDA (μmol/L)	8.9 ± 0.2	5.2 ± 1.2	7.2 ± 2.0	7.0 ± 0.6
Lp (a) (mg/dL)	56.0 ± 2.0	62.0 ± 12.0	61.0 ± 17.0	58 ± 6.0
Triglyceride (mg/dL)	234.0 ± 61.0 [†]	185.0 ± 30.0 [†]	186.0 ± 28.0	150.0 ± 9.0
Total cholesterol (mg/dL)	220.0 ± 26.0	182.0 ± 26.0	154.0 ± 11.0	161.0 ± 5.0
LDL-C (mg/dL)	129.0 ± 13.0	110.0 ± 26.0	67.0 ± 8.5	86.0 ± 4.5
HDL-C (mg/dL)	45.0 ± 10.0	35.0 ± 2.0 ^{††}	50.0 ± 8.0	45.0 ± 2.0

Data are presented as mean ± standard error.

HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitive C-reactive protein; LDL-C, low-density lipoprotein cholesterol; Lp (a), lipoprotein (a); MDA, malondialdehyde; sICAM-1, soluble intercellular adhesion molecule type 1.

[†] $P < 0.05$ versus nonsarcopenic & nonobesity; ^{††} $P < 0.05$ versus sarcopenia & nonobesity.

Serum concentrations of hs-CRP and triglyceride were significantly higher in sarcopenic obese and nonsarcopenic obese patients than in nonsarcopenic nonobese patients ($P < 0.05$, Table 5). Serum sICAM-1 was signifi-

cantly higher in nonsarcopenic obese patients as compared with sarcopenic nonobese and nonsarcopenic nonobese patients ($P < 0.05$, Table 5). In addition, serum HDL-C was lower in PD patients with nonsarcopenic

obesity than in sarcopenic nonobese and nonsarcopenic nonobese patients ($P < 0.05$, Table 5).

Skeletal muscle percentage was significantly negatively correlated with serum hs-CRP ($r = -0.29$, $P = 0.01$), sICAM-1 ($r = -0.60$, $P < 0.01$), triglyceride ($r = -0.38$, $P = 0.001$), and total cholesterol ($r = -0.24$, $P = 0.03$). In addition, skeletal muscle percentage was marginally positively correlated with serum HDL-C ($r = 0.22$, $P = 0.06$). There were no significant correlations between skeletal muscle percentage and serum concentrations of MDA, Lp (a), and LDL-C (Table 3).

Discussion

Dynapenic obesity and sarcopenic obesity are mainly observed in older people [4,25]; however, they can develop in younger adults with catabolic diseases such as CKD [4,10]. In CKD patients, factors including inflammation, oxidative stress, metabolic acidosis, decreased secretion of testosterone, insulin resistance, growth hormone resistance, physical inactivity, inadequate energy and protein intake, and vitamin D deficiency [1–3] increase protein catabolism and lead to dynapenia and sarcopenia [1–4]. The simultaneous presence of dynapenia or sarcopenia with obesity results in dynapenic obesity or sarcopenic obesity in CKD patients [4,10]. Because muscle strength decreases more rapidly than muscle mass [2], dynapenic obesity occurs earlier than sarcopenic obesity. Our study showed that the prevalence of dynapenic obesity and sarcopenic obesity were 11.4% and 3.8%, respectively, in adult PD patients in peritoneal dialysis centers in Tehran, Iran. The reason for this low prevalence of obesity, including dynapenic obesity and sarcopenic obesity, in Iranian PD patients is a high prevalence of protein-energy wasting in dialysis patients in Iran [26]. We found no study on the prevalence of dynapenic obesity in CKD patients, including PD patients, to compare with the results of our study. However, a few studies have assessed the prevalence of sarcopenic obesity in predialysis and hemodialysis patients. In agreement with our study, Malhotra et al [9] showed that the prevalence of sarcopenic obesity in hemodialysis patients ranged from 12% to 62% in men and 2% to 74% in women based on different definitions. Androga et al [10] reported that the prevalence of sarcopenic obesity was 9.7% in predialysis CKD patients.

Dynapenia and sarcopenia are associated with in-

creased morbidity, especially CVD, and high mortality [2,3,5,6]. In dialysis patients, obesity is negatively correlated with all-cause and cardiovascular mortality [7,8]. In contrast, two studies in CKD patients indicated that sarcopenic obesity did not reduce mortality [9,10]. In addition, some investigations in non-CKD patients showed that dynapenic obesity and sarcopenic obesity increased CVD and mortality [11–15].

To our knowledge, no studies have determined associations of dynapenic obesity and sarcopenic obesity with CVD risk factors in dialysis patients. Our study showed that serum hs-CRP, a systemic inflammation marker, and serum sICAM-1, a vascular inflammation marker, were significantly higher in PD patients with dynapenic obesity as compared with dynapenic nonobese and nondynapenic nonobese patients. Chronic inflammation is a common complication in PD patients which results from decreased clearance of inflammatory cytokines because of kidney failure, increased synthesis of inflammatory cytokines due to accumulation of various compounds, and bioincompatibility of PD solutions [27]. Inflammation increases the release of inflammatory cytokines by leukocytes, leading to synthesis of CRP and sICAM-1 [28]. Chronic inflammation is an important cause for dynapenia in PD patients [1–3]. In addition, obesity itself results in an inflammatory state [29]. Therefore, in dynapenic obese patients, the simultaneous presence of dynapenia and obesity is the main reason for higher serum concentrations of hs-CRP and sICAM-1 in comparison with dynapenic nonobese and nondynapenic nonobese patients. In agreement with this fact, our study showed that muscle strength was significantly negatively correlated with serum hs-CRP and sICAM-1, whereas body fat percentage was significantly positively correlated with serum hs-CRP and sICAM-1. In PD patients with nondynapenic obesity, serum concentrations of hs-CRP and sICAM-1 were significantly higher as compared to nondynapenic nonobese patients. This may be due to obesity itself [29].

Oxidative stress and high serum Lp (a) levels are two common complications in PD patients [30,31]; however, our study showed that dynapenic obesity had no effect on serum concentrations of MDA, an oxidative stress marker, or Lp (a) in PD patients. In the present study, serum triglyceride, total cholesterol, and LDL-C were significantly higher in PD patients with dynapenic obesity

in comparison with dynapenic nonobese and nondynapenic nonobese patients. High serum concentrations of triglyceride, total cholesterol, and LDL-C are common lipid abnormalities in PD patients [32,33]. In PD patients, the absorption of glucose from PD solutions and high serum glucose concentration lead to increased hepatic synthesis of triglycerides, cholesterol, and LDL-C [31,32]. In addition, high serum levels of total cholesterol and LDL-C in PD patients may be due to increased hepatic synthesis of apoprotein B100, and consequently LDL-C, following loss of amino acids and proteins through PD [31].

Other causes for high serum triglyceride, total cholesterol, and LDL-C in PD patients with dynapenic obesity are obesity and inflammation. Obesity itself leads to hypertriglyceridemia and hypercholesterolemia [34]. Inflammatory cytokines cause high serum triglyceride, total cholesterol, and LDL-C by several mechanisms [35]. First, they increase lipolysis in adipose tissue and result in increased synthesis of triglycerides and very low density lipoprotein (VLDL) in liver [35]. Second, inflammatory cytokines decrease the activity of lipoprotein lipase and cause an increase in serum concentration of triglyceride-rich lipoproteins such as VLDL [35]. Third, they increase the activity of hydroxy-methyl-glutaryl-coenzyme A reductase in the cholesterol biosynthesis pathway [36,37] and decrease cholesterol 7 α -hydroxylase activity in the conversion pathway of cholesterol into bile acids [37], resulting in a rise in serum total cholesterol and LDL-C. In our study, serum HDL-C levels were lower than the normal range in each of the four categories of PD patients. There were no significant differences between dynapenic obese PD patients and the three other categories of PD patients with regard to serum HDL-C concentration. However, serum HDL-C was lower in PD patients with nondynapenic obesity than in dynapenic nonobese and nondynapenic nonobese patients. Low serum HDL-C concentration in PD patients may be due to decreased activities of lipoprotein lipase, hepatic lipase, and lecithin-cholesterol acyl transferase, and reduced synthesis of apoprotein AI [38,39].

Similar to PD patients with dynapenic obesity, serum concentrations of CVD risk factors in PD patients with sarcopenic obesity were higher in comparison with non-sarcopenic nonobese patients, but these differences were statistically significant only for serum hs-CRP and triglyc-

eride. This may be due to the small number of sarcopenic obese patients, because only three PD patients had sarcopenic obesity in our study. Like muscle strength, SMM percentage as an indicator for sarcopenia was significantly negatively correlated with serum hs-CRP, sICAM-1, triglyceride, and total cholesterol. The mechanisms by which SMM and sarcopenic obesity affect CVD risk factors are similar to the mentioned mechanisms for muscle strength and dynapenic obesity. A limitation of our study was small sample size.

In conclusion, this study indicates that although the prevalence of dynapenic obesity and sarcopenic obesity are relatively low in PD patients, these conditions may be associated with CVD risk factors.

Conflicts of interest

All authors have no conflicts of interest to declare.

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