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표목차

ABSTRACT

The Prevalence of Sarcopenic Obesity and Risk Factors in Korean Postmenopausal Women

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Introduction

Sarcopenic obesitycarries the cumulative risk of functional abnormality, metabolic, cardiovascular risk, and mortality compared to either sarcopenia or obesity alone. Sarcopenic obesity is a complex condition interplaying hormonal, biological, nutrition, and physical activity mechanisms. Postmenopauseis related to an increase in visceral adiposity and is associated with the risk of sarcopenic obesity.

The objective of the present study was to identify the prevalence of sarcopenic obesity and associated risk factors in Korean in postmenopausal women.

Research Design and Methods

This study was based on data from the Korean National Health and Nutrition Examination Survey (KNHANES), conducted by the Korean Ministry of Health and Welfare, from 2009 to 2010. This cross-sectional and nationally representative survey of non-institutionalized

civilians used a stratified, multistage, clustered probability sampling design. Out of 19,491 participants, the analysis included data for 2,196 postmenopausal women. Multivariate logistic regression analyses were used to determine an independent associated risk factors with sarcopenic obesity in postmenopausal women.

Results

The prevalence rates of nonsarcopenicnonobesity, nonsarcopenic obesity, sarcopenicnonobesity, and sarcopenic obesity were 48.6%, 16.1%, 15.0%, and 20.3%, respectively. Sarcopenic obesity prevalence differed significantly between the subgroups and increased with earlier age at first childbirth, with 11.7% in subjects ≥ 30 years at first childbirth and 30.7% in subjects ≤ 19 years at first childbirth.

Age at first childbirth≤ 19 years(OR 1.719 [95% confidence interval (CI) 1.091–2.711]), known hypertension (OR 1.976 [1.478–2.642]), known arthritis (OR 1.941 [1.529–2.465]), diastolic blood pressure (OR 1.027 [1.008–1.046]), vitamin D level (OR 0.962 [0.942–0.982]), and history of hormone replacement therapy(OR 0.596 [0.406-0.875])were significant associated factors with sarcopenic obesity.

Conclusions

Women's age at first childbirth and chronic disease influenced the sarcopenic obesity risk in postmenopausal women. The postmenopausal women ≤ 19 years at first childbirthwas independently associated with a higher risk of sarcopenic obesity. The vitamin D level and hormone replacement therapy were significantly associated with reduction of sarcopenic obesity risk.

Introduction

Sarcopenic obesity is a double burden for older people because it carries the cumulative risk of functional abnormality, metabolic, cardiovascular risk, and mortality compared to either sarcopenia or obesity alone(1,2). Identifying individuals at risk and precise, early intervention to prevent sarcopenic obesity can greatly improve public health in today's aging society.

Pregnancy induces dramatic alterations in a woman's physiology and metabolism(3). Although childbearing is a time-limited condition, that has been implicated in healthy consequences for women later in life (4,5),it has been suggested that the timing of childbearing is associated with women's health later in life. Self-reported health among women aged 55–74 years was negatively associated with childbirth before the age of 23(6), and women who gave birth before the age of 20 had a higher mortality risk than other parous women at ages 50–85 years (7). Women who were teenage mothers were at a higher risk of dying from diabetes at 45–74 years(8), and adolescent pregnancy was independently associated with a higher risk of diabetes in postmenopausal women(9).

However, to our knowledge, no reports have investigated the influence that age at first childbirth has on sarcopenic obesity on women later in life, particularly during postmenopause. Postmenopause is related to an increase in visceral adiposity and is associated with the risk of sarcopenia(10).

The objective of the present study was to identify the prevalence of sarcopenic obesity and associated risk factors in Korean in postmenopausal women, especially focusing on age at first childbirth.

Research design and methods

Study population

This study was based on data from the KNHANES, which was conducted by the Korean Ministry of Health and Welfare from 2009 to 2010. This cross-sectional and nationally representative survey of non-institutionalized civilians used a stratified, multistage, clustered probability sampling design. The sampling units are defined based on data from household registries, including geographical area, sex, and age groups. The KNHANES is composed of a health interview survey, a nutrition survey, and a health examination survey conducted by trained investigators. All participants provided signed informed consent. From the 19,491 participants in the 2009–2010 survey, we used data collected from 3,136 women who were naturally postmenopausal. Menopause was defined as the absence of menses for 12 consecutive months. We excluded women who experienced the onset of menopause when they were younger than 40 years old (n = 135). We also excluded women with no history of childbirth (n = 45), no data for reproductive factors (n = 185), no data for dual-energy X-ray absorptiometry (n = 157), any malignancy (n = 126),and missing or incomplete data for analysis (n = 292). Finally, data for 2,196 postmenopausal women were retained for analysis.

Measurement and classification of variables

Height was measured to the nearest 0.1 cm using a portable stadiometer while the participants were in an upright position. Body weight was measured to the nearest 0.1 kg on a balanced. Body mass index (BMI) was calculated as weight in kilograms divided by the

square of the height in meters. Waist circumference (WC) was measured midway between the costal margin and the iliac crest at the end of a normal expiration. Blood pressure (BP) was measured from the right arm using a standard mercury sphygmomanometer after 5 minutes of rest in a sitting position. The mean value of two separate BPs was used for analysis. Venous blood samples were obtained after a minimum fasting time of 8 hours. The plasma level of fasting glucose was measured using a Hitachi Automatic Analyzer 7600 (Hitachi, Tokyo, Japan). Serum 25-hydroxyvitamin D (25[OH]D) concentration was measured by immunoradiometric assay using 1470 WIZARD gamma-counter (PerkinElmer, Finland).

Appendicular skeletal muscle mass (ASM) was measured using whole-body, dual-energy X-ray absorptiometry (DXA; Discovery-W, Hologic, Inc., Waltham, MA, USA). ASM was calculated as the sum of skeletal muscles in the arms and legs, assuming that all non-fat and none-bone tissue was skeletal muscle. We defined sarcopenia using ASM as a percentage of body weight (ASM/Wt), modified from the report of Janssen et al.(11). Sarcopenia was defined as ASM/Wt of 1 standard deviation (SD) below the sex-specific mean value for the young reference group aged 20–39 years from the same 2009–2010 KNHANES database(12). The cut-off value for sarcopenia was 24.65% (ASM/Wt).

Obesity was defined according to the criteria recommended by the Korean Society for the Study of Obesity, defining a BMI≥25.0 kg/m^2 as obese(13).

Sarcopenic obesity was defined when a subject satisfied the criteria for both sarcopenia and obesity.

Self-reported questionnaires were administered to determine smoking status, alcohol use, family income, education level, residential area, and regular exercise, as well as intake of total energy, carbohydrates, proteins, and fats. Residential area was categorized according to the Korean administrative district as urban and rural areas. Regular exercise is indicated as

"yes" if the subject does moderate exercise on a regular basis (for more than 30 minutes at a time, more than 5 times per week). Diabetes was defined as having a fasting plasma glucose (FPG) ≥ 126 mg/dL, using insulin or antidiabetic medication, or being diagnosed as diabetic by a physician. Known hypertension was defined as undergoing treatment for hypertension or being diagnosed with hypertension by a physician. Subjects were also asked to recall reproductive factors, including age at menarche, age at menopause, number of pregnancies, age at first childbirth, oral contraceptive use, and hormone replacement therapy.

Statistical analysis

The complex sample analysis was used for the KNHANES data for weighting all values following the guidance of statistics from the Korea Centers for Disease Control and Prevention. Continuous variables were reported as mean ± SD, and categorical variables were reported as weighted percentages. The comparisons among groups were performed using *t*-tests for continuous variables, and a chi-squared test was used for categorical variables.

For data analysis, subjects were subdivided into four groups according to the women's age at first childbirth as follows: ≤ 19 years, 20–24 years, 25–29 years, and ≥ 30 years.

Multivariate logistic regression analyses were used to identify the association between age at first childbirth subgroups and sarcopenic obesity. This was conducted by evaluating the OR after adjusting for confounding factors associated with an increased risk of sarcopenic obesity. Analyses were adjusted for potential confounders in a series of models. Covariates were added to the model in the following order: age, chronic disease (stroke, cardiovascular disease, diabetes, hypertension, and arthritis), systolic and diastolic blood pressure, lifestyle behaviors (smoking status, alcohol use, and regular exercise, as well as intake of total energy,

carbohydrates, proteins, and fats), sociodermographic factors (residential area, family income, and education), serum 25(OH)D levels, and reproductive factors (age at menarche, age at menopause, number of pregnancies, oral contraceptive use, and hormone replacement therapy).

Statistical analyses were performed using SPSS software (version 18.0), and a *P*-value of < 0.05 was considered statistically significant.

Results

Table 1 presents the subjects' clinical and biochemical characteristics according to the presence of sarcopenia and obesity. The prevalence rates of nonsarcopenicnonobesity, nonsarcopenic obesity, sarcopenicnonobesity, and sarcopenic obesity were 48.6%, 16.1%, 15.0%, and 20.3%, respectively.

Among the subgroups, significant differences were found regarding variables, such as age, systolic and diastolic blood pressure, body composition parameters, education, and diet pattern. Chronic diseases, such as diabetes, known hypertension, known arthritis, previous stroke, and reproductive factors (such as number of pregnancies and hormone replacement therapy) also differed between groups. Lower levels of 25(OH)D were more common among subjects with sarcopenia.

The mean age of women at first childbirth differed significantly between the subgroups and was more likely to be lower in subjects with sarcopenic obesity. In addition, distribution of the age group at first childbirth differed significantly between the subgroups, and the proportion of earlier age at first childbirth increased in the sarcopenic obesity group.

Comparison of clinical characteristics and prevalence of sarcopenic obesity among age at first childbirth subgroup

Table 2 shows characteristics of the subjects stratified into four groups by age at first childbirth. Subjects with an earlier age at first childbirth were older and had higher BMIs, WCs, systolic blood pressures, and had higher number of pregnancies than those with a later age at first childbirth. In addition, subjects with an earlier age at first childbirth were more

likely to have a lower family income and education level and to reside in rural areas. Smoking, alcohol use, known hypertension, known arthritis, and reproductive factors (such as age at menarche, age at menopause, and oral contraceptive use) differed between subgroups.

The prevalence of sarcopenia was more likely to increase with earlier age at first childbirth (31.4% in subjects ≥ 30 years and 46.8% in subjects ≤ 19 years, respectively). Sarcopenic obesity prevalence differed significantly between the subgroups and increased with an earlier age at first childbirth, with 11.7% in subjects ≥ 30 years at first childbirth and 30.7% in subjects ≤ 19 years at first childbirth.

Associated risk factors of sarcopenic obesity in postmenopausal womens

Table 3 presents the results of the logistic regression analyses designed to examine the association between variables and sarcopenic obesity. Age at first childbirth≤ 19 years(OR 1.719 [95% confidence interval (CI) 1.091–2.711]), known hypertension (OR 1.976 [1.478–2.642]), known arthritis (OR 1.941 [1.529–2.465]), diastolic blood pressure (OR 1.027 [1.008–1.046]), vitamin D level (OR 0.962 [0.942–0.982]), and history of hormone replacement therapy(OR 0.596 [0.406-0.875])were significant associated factors with sarcopenic obesity.

Discussion

In the current study, we found positivesignificant association between chronic disease and sarcopenic obesity risk in postmenopausal women. The age of women at first childbirth was significantly associated with sarcopenic obesity in postmenopausal women.Postmenopausal women with adolescent pregnancy (age at first childbirth ≤ 19 years) had a higher risk of sarcopenic obesity after adjusting for multiple conditions potentially affecting the results. To our knowledge, this is the first large, population-based study that investigated the association between age at first childbirth and sarcopenic obesity in postmenopausal women. Our results indicate long-term effects of age at first childbirth on incidental sarcopenic obesity. The vitamin D level and hormone replacement therapy were negatively associated with sarcopenic obesity risk.

Sarcopenic obesity is a complex condition interplaying hormonal, biological, nutrition, and physical activity mechanisms(14). This study has a cross-sectional design; therefore, it was not possible to establish pathophysiological mechanisms that connect adolescent pregnancy and an increased risk of sarcopenic obesity. Several possibilities may be suggested.

First, pregnancy can affect the physiology of women depending on a given age[3]. Pregnancy during adolescence is a significant challenge to the organisms that is not physiologically prepared(15,16). Insulin resistance is associated with the augmentation of intramyocellular fat mass and loss of muscle function(17), and pregnancy is related to increased insulin resistance(3). These early exposures to biologically immature organs may induce subtle and deleterious outcomes in physiology and metabolism, including muscle, which is still growing,that might be sustained and susceptible to an increased risk of sarcopenic obesity later in life when facing vulnerable and not compensated period such as postmenopause.

Postmenopauseis related to an increase in visceral adiposity, as well as a decrease in muscle mass and muscle strength(10).

Second, obesity, especially postpartum weight retention and the development of obesity, may act as an intermediating or provoking factor between adolescent pregnancy and the risk of sarcopenic obesity later in life. Several studies suggested that excess gestational weight gain contributes to an increased risk of being overweight during subsequent life, and adolescent pregnancies result in higher weight gain compared with adult pregnancies(18-20). Moreover, a teenage mother may encourage unhealthy lifestyle habits, such as less physical activities, adding to an increased risk of obesity later in life (21). In obesity, high circulating levels of lipids accumulate in skeletal muscle, and these intramyocellular lipids may alter muscle morphology, size, and function, leading to a reduced muscular renewal capacity(22). An increase in obesity also expression of myostatin, which is a muscle growth factor regulating muscle growth, suppressing differentiation and proliferation of myocytes, and inversely correlating with skeletal muscle mass(23). In addition, visceral fat produces more pro-inflammatory adipokines, leading to a low-grade inflammatory status, which is related to a loss of skeletal mass by triggering muscle proteolysis and myocyte apoptosis(24). Longstanding and cumulative exposure to obesity seems to create a vicious cycle of fat gain and muscle loss, which could contribute to the development of sarcopenia. Chronic exposure to high amounts of fat induces de-differentiation of myotube progenitor cells to mesenchymal adipocyte-like default cells. This process could decrease the regeneration capacity of the skeletal muscle with aging(25). However, information is not available on gestational weight gain, postpartum weight retention, and lifetime weight change for the current study. Additional studies are needed to explore the exact causal relationships in more detail.

Third, women who are pregnant during adolescence may have higher parity, as early age at

first childbirth was correlated with increasing parity(26). In our study, women with who were younger at first childbirth had more pregnancies than those who were older at first childbirth. Each pregnancy permanently resets ovarian function, and increasing pregnancies can lead to a reduced lifetime exposure to estrogen(27). A decline in estrogen may lead to increased pro-inflammatory cytokines, such as tumor necrosis factor alpha (TNF-α) or interleukin-6 (IL-6), which contribute to muscle catabolism(28). In addition, IL-6 and TNF-α could lower the anabolic hormone insulin-like growth factor-1 (IGF-1), leading to a catabolic state(29). These activities may be associated with developing sarcopenia later in life. Moreover, skeletal muscle has estrogen beta-receptors(30). Although further studies are needed, there is a potential direct effect of low estrogen levels on a decrease in protein synthesis. In the present study, the association of adolescent pregnancy with sarcopenic obesity remained after adjusting for the number of pregnancies. These results suggest that adolescent pregnancy may itself influence sarcopenic obesity via pathophysiological mechanisms other than the number of pregnancies.

Fourth, a pregnant teenager experiences several role changes relating to childcare. These changes can lead to the mother becoming more and more prone to an impaired nutrition status and lower physical performance(21), leading to the deterioration of muscle quantity and quality.

Fifth, socioeconomic status can affect both adolescent pregnancy and the risk of sarcopenic obesity as selection effects. In the present study, education, family income, and residential area are considerable confounders. After these variables are added to the multivariable models, the magnitude of the association between subjects≤ 19 years at first childbirth and sarcopenic obesity attenuated but continued to be statistically significant. These results should be interpreted carefully because our data on socioeconomic factors reflect the status at

enrollment but not during the adolescent or lifetime period.

The strength of this study is that it was a large, population-based national representative study that considered a comprehensive range of possible confounding and mediating factors, including chronic disease, sociodemographic influences, lifestyle differences, serum 25(OH)D levels, and reproductive factors. Recently, a possible role of vitamin D in sarcopenia has been postulated, suggesting vitamin D deficiency as a risk factor for sarcopenia(31). In the currentstudy, we added a serum 25(OH)D level to the multivariable models; a magnitude of association between subjects ≤ 19 years age at first childbirth and sarcopenic obesity attenuated but continued to be statistically significant.

This study had several limitations. The measurements were performed at a certain time in a cross-sectional design; thus, a causal relationship could not be clearly determined. We also applied measurements of muscle mass only to define sarcopenia because the KNHANES did not examine muscle strength or performance, although the European Working Group on Sarcopenia in Older People developed diagnostic criteria for sarcopenia, including low muscle function(32). However, we used muscle mass as an index in which ASM was calculated as a percentage of body weight, which has been known to be associated with metabolic parameters(12).Another limitation was that age at first childbirth, age at menopause, and age at menarche were based on self-report, which is prone to recall bias. However, the recall of reproductive factors is expected to be valid and reliable(33). Finally, these results can be potentially less generalized to today's women because the adolescent environmental conditions of this study population were different from those of today's adolescents.

Conclusion

In conclusion, age at first childbirth and chronic diseases influenced sarcopenic obesity in postmenopausal women, and adolescent pregnancy was independently associated with a higher risk of sarcopenic obesity in postmenopausal women. The vitamin D level and hormone replacement therapy influence on the reduction of sarcopenic obesity risk.

Our data propose that adolescent pregnancy may contribute to the development of sarcopenic obesity later in life and should be considered a risk factor. Therefore, to effectively prevent sarcopenic obesity in postmenopausal women, more precise consideration should be focused on women who are pregnant during adolescence. A prospective study is needed to explore the possible causal relationship between adolescent pregnancy and the risk of subsequent sarcopenic obesity later in life.

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Table 1. Characteristics of the study population

	Nonsarcopenic nonobesity	Nonsarcopenic obesity	Sarcopenicnon obesity	Sarcopenic obesity	*P*-value
N (%)	1,078(48.6)	367 (16.1)	295 (15.0)	456 (20.3)	
Age (years)	61.66 ± 0.38	61.52 ± 0.54	62.67 ± 0.78	64.15 ± 0.53	<0.001
BMI (kg/m^2)	22.17 ± 0.68	27.01 ± 0.10	23.22 ± 0.97	27.98 ± 0.15	<0.001
WC (cm)	76.97 ± 0.23	89.00 ± 0.37	80.68 ± 0.48	91.49 ± 0.45	<0.001
ASW (kg)	13.84 ± 0.06	16.08 ± 0.09	12.04 ± 0.10	14.19 ± 0.10	<0.001
ASW/Wt (%)	26.30 ± 0.08	25.13 ± 0.08	22.26 ± 0.11	21.90 ± 0.08	<0.001
Total body fat (%)	31.28 ± 0.18	35.12 ± 0.19	38.34 ± 0.34	40.32 ± 0.21	<0.001
SBP (mmHg)	125.27 ± 0.67	130.73 ± 0.87	127.90 ± 1.26	133.04 ± 0.99	<0.001
DBP(mmHg)	76.79 ± 0.37	80.66 ± 0.57	78.41 ± 0.67	80.82 ± 0.56	<0.001
25(OH)D (ng/mL)	18.75 ± 0.33	19.35 ± 0.48	17.07 ± 0.50	17.15 ± 0.36	<0.001
Smoking (%)					0.517
None	90.9	93.3	89.7	91.1	
Ex	2.1	1.0	2.0	3.4	
Current	6.9	5.7	8.3	5.5	
Alcohol use (%)					0.079
None	54.1	42.1	50.3	49.9	
≤ 1/week	39.1	52.2	41.2	43.2	
2-3/week	4.3	3.7	6.9	4.1	
≥ 4/week	2.6	2.0	1.6	2.7	
Family income[a] (%)					0.606
< 100	27.9	31.3	27.5	32.0	
100-199	20.1	21.5	18.8	20.0	
200-299	15.2	17.8	18.1	14.9	
≥ 300	36.8	29.3	35.6	33.1	
Less than high school education (%)	73.7	86.6	71.9	85.6	<0.001
Residence in urban area (%)	45.5	45.1	44.3	42.7	0.885
Regular exercise[b] (yes, %)	13.2	18.2	11.7	13.1	0.153
Total energy intake (kcal)	1,641.92 ± 30.78	1,654.40 ± 46.56	1,484.78 ± 42.65	1,547.75 ± 44.01	0.010
Carbohydrate intake	306.20 ± 6.17	306.60 ± 8.94	271.88 ± 8.02	280.61 ± 6.87	0.001
Protein intake (g/day)	54.47 ± 1.16	54.85 ± 1.83	51.04 ± 1.98	53.84 ± 2.01	0.452
Fat intake (g/day)	24.38 ± 0.81	24.12 ± 1.26	23.30 ± 1.21	24.68 ± 1.72	0.884
Diabetes (%)	11.1	19.2	13.3	19.2	0.001

Known hypertension (%)	29.7	46.5	35.1	56.8	<0.001
Known arthritis (%)	27.8	41.7	35.8	49.5	<0.001
Previous stroke (%)	2.3	4.8	1.0	3.1	0.033
Previous CVD (%)	3.1	3.2	3.2	3.7	0.962
Age at menarche (years)	15.87 ± 0.07	15.70 ± 0.12	15.82 ± 0.14	15.90 ± 0.10	0.620
Age at menopause (years)	49.62 ± 0.13	50.36 ± 0.26	49.80 ± 0.28	49.68 ± 0.21	0.115
Pregnancies (n)	4.80 ± 0.08	5.27 ± 0.15	4.75 ± 0.14	5.35 ± 0.12	<0.001
OC (ever, %)	21.5	25.7	22.9	23.2	0.561
HRT (ever, %)	16.0	16.1	17.6	10.0	0.044
Age at first childbirth (years)	23.96 ± 0.13	23.53 ± 0.21	24.04 ± 0.29	22.90 ± 0.17	<0.001
Age group at first childbirth (%)					0.011
≤ 19	8.0	7.5	10.3	14.5	
20–24	51.7	56.7	49.0	56.6	
25–29	33.9	31.6	33.4	25.7	
≥ 30	6.4	4.1	7.3	3.2	

Data are expressed as mean ± SD for continuous variables and as weighted percentages for categorical variables. Body mass index (BMI), cardiovascular disease (CVD), diastolic blood pressure (DBP), hormone replacement therapy (HRT), oral contraceptive (OC), systolic blood pressure (SBP), waist circumference (WC), body weight. (Wt).

[a]Unit is thousands Korean won/month.

[b]Regular exercise is indicated as “yes” when the subject does moderate exercise on a regular basis (for more than 30 minutes at a time, more than 5 times per week).

Table 2. Characteristics of the study population according to age at first childbirth

	Age at first childbirth (years)				
	≤ 19	20–24	25–29	≥ 30	*P*-value
N (%)	201 (9.6)	1212 (53.1)	673 (31.8)	110 (5.5)	
Age (years)	70.24 ± 0.87	62.98 ± 0.39	59.38 ± 0.34	58.67 ± 0.87	<0.001
BMI (kg/m^2)	24.80 ± 0.27	24.47 ± 0.11	23.92 ± 0.14	23.71 ± 0.32	0.002
WC (cm)	85.14 ± 0.74	83.13 ± 0.33	80.92 ± 0.43	79.33 ± 0.88	<0.001
ASW (kg)	13.89 ± 0.18	14.01 ± 0.08	14.07 ± 0.09	13.77 ± 0.22	0.480
ASW/Wt (%)	24.34 ± 0.23	24.61 ± 0.11	24.71 ± 0.13	24.63 ± 0.25	0.597
Total body fat (%)	35.21 ± 0.41	34.77 ± 0.24	34.78 ± 0.26	34.31 ± 0.52	0.603
SBP (mmHg)	133.86 ± 1.29	128.26 ± 0.64	126.43 ± 0.88	126.53 ± 2.85	<0.001
DBP(mmHg)	78.12 ± 0.74	78.34 ± 0.36	78.59 ± 0.47	79.66 ± 1.32	0.724
25(OH)D (ng/mL)	17.69 ± 0.60	18.64 ± 0.29	17.82 ± 0.37	18.37 ± 0.73	0.091
Smoking (%)					
None	78.2	91.6	94.7	89.8	<0.001
Ex	4.8	2.1	1.7	1.3	
Current	17.0	6.4	3.6	8.9	
Alcohol use (%)					0.002
None	66.5	45.8	53.5	54.1	
≤ 1/week	29.9	45.5	41.3	39.2	
2–3/week	1.9	5.8	3.3	5.0	
≥ 4/week	1.7	2.9	1.9	1.6	
Family income[a] (%)					<0.001
< 100	42.8	34.6	18.7	14.8	
100–199	21.7	20.1	19.1	23.7	
200–299	10.6	15.4	18.4	17.7	
≥ 300	24.9	29.9	43.9	43.8	
Less than high school education (%)	98.2	87.2	60.4	54.7	<0.001
Residence in urban area (%)	29.5	41.2	54.1	50.8	<0.001
Regular exercise[b] (yes, %)	17.0	13.6	13.3	13.0	0.656
Total energy intake (kcal)	1,446.05 ± 48.11	1,610.98 ± 26.19	1,627.13 ± 33.05	1,628.50 ± 84.30	0.010
Carbohydrate intake	276.83 ± 9.50	298.68 ± 4.99	297.42 ± 7.09	294.15 ± 16.52	0.220
Protein intake (g/day)	44.85 ± 1.73	53.85 ± 1.15	56.16 ± 1.29	56.87 ± 3.28	<0.001
Fat intake (g/day)	18.78 ± 1.42	23.88 ± 0.88	26.02 ± 0.81	26.82 ± 2.40	<0.001
Diabetes (%)	17.1	15.9	11.5	12.1	0.144
Known hypertension (%)	58.8	40.4	31.9	26.4	<0.001

Known arthritis (%)	41.6	39.0	29.5	28.7	0.003
Previous stroke (%)	4.2	2.8	2.2	1.8	0.609
Previous CVD (%)	7.0	2.9	3.1	2.0	0.053
Age at menarche (years)	15.65 ± 0.12	16.14 ± 0.07	15.51 ± 0.10	15.24 ± 0.24	<0.001
Age at menopause (years)	49.02 ± 0.36	49.70 ± 0.13	50.09 ± 0.15	49.99 ± 0.38	0.020
Pregnancies (*n*)	6.33 ± 0.22	5.30 ± 0.08	4.33 ± 0.09	3.35 ± 0.17	<0.001
OC (ever, %)	14.8	26.8	19.9	13.4	0.001
HRT (ever,%)	9.8	14.0	16.8	23.8	0.054
Sarcopenia (%)	46.8	35.5	32.1	31.4	0.013
Sarcopenic obesity (%)	30.7	21.6	16.4	11.7	<0.001

Data are expressed as mean ± SD for continuous variables and as weighted percentages for categorical variables. Body mass index (BMI), cardiovascular disease (CVD), diastolic blood pressure (DBP), hormone replacement therapy (HRT), oral contraceptive (OC), systolic blood pressure (SBP), waist circumference (WC), body weight. (Wt).

[a]Unit is thousands Korean won/month.

[b]Regular exercise is indicated as “yes” when the subject does moderate exercise on a regular basis (for more than 30 minutes at a time, more than 5 times per week).

Table 3. Association of variables with sarcopenic obesity in postmenopausal women

	Odds ratio	95% CI	*P*-value
Age (years)	1.007	0.987-1.027	0.501
SBP (mmHg)	1.000	0.990-1.010	0.942
DBP (mmHg)	1.027	1.008-1.046	0.005
25(OH)D (ng/mL)	0.962	0.942-0.982	<0.001
Smoking			
None	1.00(ref.)	1.00(ref.)	
Ex	1.519	0.617-3.740	0.362
Current	0.671	0.369-1.219	0.189
Alcohol use			
None	1.00(ref.)	1.00(ref.)	
≤ 1/week	1.240	0.934-1.647	0.136
2-3/week	1.123	0.496-2.540	0.780
≥ 4/week	1.662	0.602-4.592	0.326
Family income[a]			
< 100	0.804	0.570-1.133	0.211
100-199	0.911	0.659-1.260	0.573
200-299	0.806	0.521-1.247	0.332
≥ 300	1.00(ref.)	1.00(ref.)	
Education			
More than high school education	1.00(ref.)	1.00(ref.)	
Less than high school education	1.497	0.966-2.321	0.071
Residence			
Rural area	1.00(ref.)	1.00(ref.)	
Urban area	1.034	0.769-1.390	0.826
Regular exercise[b]			
No	1.00(ref.)	1.00(ref.)	
Yes	1.039	0.714-2.512	0.842
Total energy intake (kcal)	0.999	0.997-1.001	0.532
Carbohydrate intake	1.000	0.992-1.009	0.952
Protein intake (g/day)	1.012	1.001-1.023	0.040
Fat intake (g/day)	1.010	0.988-1.032	0.377
Diabetes	1.290	0.924-1.800	0.134
Known hypertension	1.976	1.478-2.642	<0.001
Known arthritis	1.941	1.529-2.465	<0.001
Previous stroke	0.861	0.431-1.719	0.670

Previous CVD	0.936	0.498-1.761	0.838
Age at menarche (years)	0.981	0.919-1.046	0.554
Age at menopause (years)	0.987	0.854-1.022	0.454
Pregnancies (n)	1.012	0.957-1.070	0.669
OC (ever)	0.995	0.761-1.301	0.970
HRT (ever)	0.596	0.406-0.875	0.008
Age group at first childbirth			
≤ 19	1.719	1.091-2.711	0.020
20–24	1.209	0.893-1.638	0.219
25–29	1.00(ref.)	1.00(ref.)	
≥ 30	0.748	0.359-1.559	0.437

Cardiovascular disease (CVD), diastolic blood pressure (DBP), hormone replacement therapy (HRT), oral contraceptive (OC), systolic blood pressure (SBP).

[a]Unit is thousands Korean won/month.

[b]Regular exercise is indicated as "yes" when the subject does moderate exercise on a regular basis (for more than 30 minutes at a time, more than 5 times per week).