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Original article

Sarcopenic obesity is associated with cognitive impairment in community-dwelling older adults: The Bunkyo Health Study

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SUMMARY

Background & aims: Coexistence of obesity and decreased muscle strength, defined as sarcopenic obesity, is often observed in the older adults. The present study investigated whether sarcopenic obesity, defined as reduced handgrip strength and increased body mass index (BMI), is associated with cognitive impairment.

Methods: Study participants include 1615 older adults aged 65–84 years who lived in an urban area of Tokyo, Japan and participated in the Bunkyo Health Study. Mild cognitive impairment (MCI) and dementia were defined based on ≤22 points of Montreal Cognitive Assessment and ≤23 points of the Mini-Mental State Examination, respectively. Handgrip strength was measured using a dynamometer in a standing position. We divided participants into four groups according to their sarcopenia (probable) (handgrip strength <28 kg in men and <18 kg in women) and obesity status (BMI ≥25 kg/m^2) as control, obesity, sarcopenia and sarcopenic obesity, and investigated the association between cognitive function, sarcopenia, and obesity status.

Results: Mean age was 73.1 ± 5.4 years, and 57.6% of study participants were female. The prevalence of control, obesity, sarcopenia, and sarcopenic obesity was 59.4%, 21.2%, 14.6%, and 4.7%, respectively. The prevalence of MCI and dementia, respectively, was highest in participants with sarcopenic obesity, followed by those with sarcopenia, obesity, and control. After multivariate adjustment, sarcopenic obesity was independently associated with increased odds of MCI and dementia compared with the control (MCI: 2.11 [95% confidence interval, 1.12–3.62]; dementia: 6.17 [2.50–15.27]).

Conclusions: Sarcopenic obesity was independently associated with MCI and dementia among Japanese older adults. Future studies are necessary to clarify the causal relationship.

1. Introduction

Dementia is a major health concern associated with ageing. The prevalence of dementia, which eventually requires long-term nursing care, reached 15% among people aged over 65 years in Japan. It is projected to reach 20% in 2025 [1]. A meta-analysis reported that sarcopenia, defined as an age-related loss of muscle

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mass with reduced muscle strength and/or impaired physical performance, was independently associated with cognitive impairment, while not all studies have shown the same trend [2]. Some studies have reported that obesity determined by body mass index (BMI) has a protective effect for cognitive impairment in older adults [3,4]. In addition, subjects with obesity (fat percentile method) and sarcopenia, defined as sarcopenic obesity, had lower risk for impaired activities of daily living (ADL) compared with subjects with sarcopenia only [5]. On the other hand, sarcopenic obesity is associated with a higher risk of cognitive impairment than either alone [6–8]. However, despite the presence of criteria established by the Asian Working Group for Sarcopenia (AWGS) [9], previous studies have applied different arbitrary cut-off values for muscle mass when defining sarcopenia [10]. Thus, the impact of sarcopenic obesity as defined by AWGS criteria on cognitive impairment is difficult to estimate. Taking the data from prior studies into consideration, sarcopenic obesity as defined by AWGS criteria for confirmed sarcopenia is rare because the number of obese subjects defined by BMI that meet the definition of reduced muscle mass is very low. Recently, it has been reported that sarcopenic obesity defined by the AWGS criteria is associated with reduced ADL [11]. However, this study was performed in post-stroke patients admitted to convalescent rehabilitation wards, thus, the AWGS criteria have not been used to define sarcopenic obesity in the community-dwelling individuals.

On the other hand, decreased handgrip strength, one of the components of sarcopenia [9,12], could be a better marker of impaired cognitive function than reduced muscle mass [13–15]. Age-associated loss of hand grip strength is defined as probable sarcopenia [9,12]. Using this definition, the coexistence of obesity defined by BMI and reduced handgrip strength, defined as sarcopenic obesity, is often observed in older populations. Previous studies have shown that sarcopenic obesity (BMI$\geq$25 kg/m^2 and low handgrip strength) is associated disability [16]. However, it remains unclear whether sarcopenic obesity could be a useful indicator of probable cognitive impairment in clinical settings. In this context, the present study investigated whether sarcopenic obesity, defined as reduced handgrip strength and increased BMI, is associated with cognitive impairment in urban community-dwelling older Japanese individuals participating in the Bunkyo Health Study [17].

2. Materials and methods

2.1. Study design and participants

The Bunkyo Health Study is an ongoing prospective cohort study designed to clarify how muscle mass, muscle strength, and insulin sensitivity are associated with multiple diseases that necessitate long-term care [17]. In this study, we recruited older subjects aged 65–84 years living in Bunkyo-ku, an urban area in Tokyo, Japan. All subjects participated in examinations over two visits at the Sportology Center from October 15, 2015, to October 1, 2018. Briefly, we evaluated cognitive function using questionnaires, physical fitness using dynamometry and physical performance tests, brain lesions using magnetic resonance imaging (MRI), body composition and bone mineral density using dual-energy X-ray absorptiometry (DXA), arteriosclerosis using the cardio-ankle vascular index, and abdominal fat distribution by MRI. Next, we administered a 75-g oral glucose tolerance test (OGTT). The present study was post-hoc analysis using baseline data of the Bunkyo Health Study.

The study protocol was approved by the ethics committee of Juntendo University in November 2015 (Nos. 2 015 078, 2 016 138, 2 016 131, 2 017 121, and 2 019 085). This study was carried out in accordance with the principles outlined in the Declaration of Helsinki. All participants gave written informed consent and were informed that they had the right to withdraw from the trial at any time.

Among 1629 subjects enrolled in the Bunkyo Health Study, we excluded 2 patients with unavailable DXA data on body composition. Among the remaining 1627 subjects, 12 subjects previously diagnosed with dementia (n = 1), or depression (n = 11) were excluded. Therefore, 1615 subjects were included in this analysis (Fig. 1).

2.2. Muscle strength measurement and maximum gait speed

We evaluated handgrip strength using a dynamometer (T. K. K. 5401, Takei Scientific Instruments, Niigata, Japan) in a standing position [18]. Subjects held the dynamometer at thigh level and two measurements were taken for each hand. The maximum grip strength value averaged across both hand was used in the analysis. In addition, we evaluated maximum gait speed using the 10-m walking test. Maximum gait speed was calculated by dividing walking distance (m) by walking time (sec) during the middle 5 m.

2.3. Anthropometric measurements

Height was measured within 0.1 cm using a stadiometer in the upright position in the morning. Body weight, skeletal muscle mass, and body fat mass were measured within 0.1 kg using DXA [19]. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2). Skeletal muscle mass index (SMI) was calculated by dividing appendicular muscle mass (kg) by height squared (m^2) [9]. Percent body fat was calculated by dividing body fat mass (kg) by body weight (kg). Waist circumference at the level of umbilical was measured within 0.5 cm with a plastic tape measure.

2.4. Cognitive function

Cognitive function was primarily evaluated using the Montreal Cognitive Assessment (MoCA) [20] and the Mini-Mental State Examination (MMSE) [21]. The MoCA and MMSE contain 9 and 11 items, respectively. Possible scores range from 0 to 30 points for each scale. In this study, we used MoCA score $\leq$22 as the cut-off for mild cognitive impairment (MCI) [22] and MMSE score $\leq$23 as the cut-off for dementia [21]. We also used the short version of the Geriatric Depression Scale (GDS) [23] to assess depressive symptoms; depression was defined as a score $\geq$6 points.

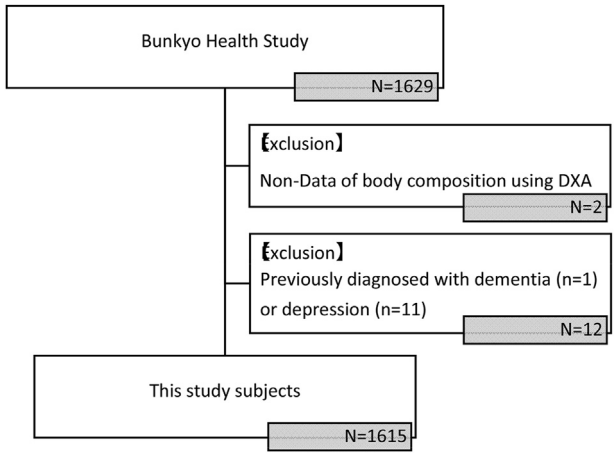

Fig. 1. Flowchart of study subjects.

2.5. Other measurements

Physical activity and sedentary time were evaluated using the International Physical Activity Questionnaire, which assesses different types of physical activity, such as walking and moderate- and high−intensity activities [24]. Nutritional status was evaluated using a brief-type self-administered diet history questionnaire (BDHQ) [25]. The BDHQ is a four-page structured self-administered questionnaire and asks about the consumption frequency of the selected 58 food and beverage items to estimate the dietary intake of those. The use of BDHQ in very older adults was already validated [26]. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg [27], or current use of antihypertensive medications. Diabetes was defined as fasting plasma glucose ≥126 mg/dL, glucose level ≥200 mg/dL at 2 h after the OGTT, hemoglobin A1c ≥ 6.5% [28], or current use of medications for diabetes mellitus. Dyslipidemia was defined as low-density lipoprotein cholesterol ≥140 mg/dL, high density lipoprotein cholesterol <40 mg/dL, triglycerides ≥150 mg/dL [29], or currently use of lipid-lowering agents.

2.6. Statistical analysis

In this study, sarcopenia (probable) was defined as handgrip strength <28 kg in men and <18 kg in women based on AWGS criteria [9], and it was expressed as “sarcopenia”. Obesity was defined as BMI ≥25 kg/m^2 [30]. The participants were divided into four groups according to their sarcopenia and obesity status: non-sarcopenia/non-obese group (control), non-sarcopenia/obese group (obesity), sarcopenia/non-obese group (sarcopenia), sarcopenia/obese group (sarcopenic obesity).

Normal distribution was assessed by histogram. Then, data are presented as means ± standard division, SD or median (Interquartile Range, IQR) on continuous variable, and number (%) on categorical variable, appropriately. Differences between the four groups were compared using one-way analysis of variance or Kruskal−Wallis test for continuous variables and the chi-square test for categorical variables. All statistical tests were two-sided with a 5% significant level. Logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for cognitive impairment in each group, with adjustment for age (continuous variable), sex (male or female), and other potential confounders. In this study, year of education (continuous variable) [31], depression (normal or suspected depression; GDS ≥6 points) [32], physical activity (continuous variable) [33], hypertension (yes or no) [34], diabetes (yes or no) [35], and dyslipidemia (yes or no) [36] were included as potential confounders, because these factors have been reported to be associated with cognitive impairment. In addition, the association between the combination of sarcopenia and obesity status and cognitive impairment was also analyzed separately in men and women because the prevalence and etiology of cognitive impairment differ by sex [37]. All statistical analyses were conducted using the IBM SPSS Statistics (version 26.0).

3. Results

3.1. Characteristics by sarcopenia and obesity status

Characteristics of this cohort based on the presence of sarcopenia and obesity status are shown in Table 1. The prevalence of no obesity or sarcopenia, obesity, sarcopenia, and sarcopenic obesity was 59.4% (51.0% in men and 65.6% in women), 21.2% (26.6% in men and 17.3% in women), 14.6% (17.3% in men and 12.7% in women), and 4.7% (5.1% in men and 4.4% in women), respectively. Of note, only 0.1% (0.3% in men and 0% in women) of study participants were categorized as having both confirmed sarcopenia and obesity, defined as the copresence of low SMI (SMI <7.0 kg/m^2 for men or SMI <5.4 kg/m^2 for women) and weak handgrip strength based on AWGS criteria [9] and BMI ≥25.0 kg/m^2 [38].

The study participants in the sarcopenia and sarcopenic obesity groups were older than those in the control and obesity groups. Body composition parameters, including BMI, percent body fat, and SMI were comparable between the control and sarcopenia groups. The obesity and sarcopenic obesity groups had higher BMI, percent body fat, and SMI than the control and sarcopenia groups. The sarcopenic obesity group had higher percent body fat and lower SMI than the obesity group. In addition, handgrip strength in the sarcopenia and sarcopenic obesity groups was lower than the handgrip strength of the control and obesity groups. Handgrip strength was higher in the obesity group than the control group. Rank order for maximum gait speed were similar to rank order for handgrip strength. The sarcopenic obesity group had the highest prevalence of hypertension, diabetes, dyslipidemia, and cardiovascular disease among the groups.

Among the 1615 participants, 292 (18.1%) were classified as having MCI and 53 (3.3%) were classified as having dementia. In increasing order, the prevalence of MCI was 14.5% in the control group, 16.9% in the obesity group, 27.1% in the sarcopenia group, and 40.8% in the sarcopenic obesity group ($P < 0.001$) (Fig. 2). Similarly, the prevalence of dementia was 1.6% in the control group, 2.6% in the obesity group, 7.6% in the sarcopenia group, and 14.5% in the sarcopenic obesity group ($P < 0.001$) (Fig. 2).

3.2. Sarcopenia and obesity status and odds ratio for mild cognitive impairment and dementia

Table 2 shows the ORs for MCI and dementia by group. The model with adjustment for age and sex revealed an association between sarcopenic obesity and MCI (Control: OR, 1.00 [referent]; Obesity: OR, 1.06 [95% CI, 0.75−1.50]; sarcopenia: OR, 1.33 [95% CI, 0.92−1.91]; and sarcopenic obesity: OR, 2.41 [95% CI, 1.43−4.07]). Even after adjusting for all confounders, sarcopenic obesity was independently associated with MCI (Control: OR, 1.00 [Referent]; Obesity: OR, 0.96 [95% CI, 0.67−1.37]; Dynapenia: OR, 1.30 [95% CI, 0.90−1.89]; and sarcopenic obesity: OR, 2.08 [95% CI, 1.22−3.57]). When analyses were stratified by sex, sarcopenic obesity was independently associated with MCI in females, but not in males.

In terms of dementia, the fully adjusted model revealed an association between sarcopenia or sarcopenic obesity and dementia (Control, 1.00 [Referent]; obesity, 1.69 [95% CI: 0.71−4.02]; sarcopenia: 3.22 [95% CI: 1.57−7.02]; and sarcopenic obesity: 6.08 [95% CI: 2.47−15.96]). When analyses were stratified by sex, sarcopenic obesity was independently associated with dementia in males, and sarcopenia and sarcopenic obesity were each independently associated with dementia in females.

4. Discussion

In the present study, we investigated the association between the combination of sarcopenia and obesity status and cognitive impairment in 1615 older adults living in an urban area of Tokyo. In this cohort, 292 (18.1%) of participants were classified as having MCI and 53 (3.3%) were classified as having dementia. After full adjustment for potential risk factors, we found that sarcopenic obesity was independently associated with MCI and dementia. On the other hand, sarcopenia was independently associated only with dementia, and obesity alone was not associated with either MCI or dementia.

We defined obesity as BMI of ≥25 kg/m^2 in the present study. It has been proposed that thresholds of obesity in Asians should be

Table 1
Characteristics of study participants by sarcopenia and obesity status.

n	All	Control	Obesity	Sarcopenia	Sarcopenic Obesity	*P* value
	1615	960	343	236	76	
Female sex	931 (57.6%)	611 (63.6%)	161 (46.9%)	118 (50.0%)	41 (53.9%)	<0.001
Age, y	73 (68–77)	72 (68–76)	72 (68–77)	77 (72–81)[a,b]	79 (74–80)[a,b]	<0.001
Body mass index, kg/m^2	23.2 ± 3.1	21.9 ± 2.0	27.1 ± 2.0[a]	21.6 ± 2.1[b]	26.9 ± 1.9[a,c]	<0.001
Percent body fat, %	22.8 ± 6.2	22.0 ± 5.7	24.7 ± 6.7[a]	21.5 ± 5.8[b]	27.3 ± 6.1[a,b,c]	<0.001
Skeletal muscle index, kg/m^2	7.1 ± 1.1	6.8 ± 0.9	8.2 ± 1.0[a]	6.7 ± 0.9[b]	7.7 ± 0.9[a,b,c]	<0.001
Year of education year, n	13.9 ± 2.5	14.0 ± 2.4	14.0 ± 2.6	13.9 ± 2.6	13.2 ± 3.1	0.202
Montreal Cognitive Assessment, score	26 (23–27)	26 (24–28)	26 (23–27)	23 (20–27)[a,b]	23 (20–27)[a,b]	<0.001
Mini-Mental State Examination, score	28 (27–29)	28 827–29)	28 (27–29)	27 (25–28)[a,b]	27 (25–28)[a,b]	<0.001
Hand grip strength, kg	25.9 ± 7.1	26.4 ± 6.4	29.5 ± 7.5[a]	20.3 ± 5.2[a,b]	20.0 ± 5.3[a,b]	<0.001
Maximum gait speed, m/s	1.88 ± 0.35	1.95 ± 0.33	1.88 ± 0.36[a]	1.73 ± 0.35[a,b]	1.61 ± 0.36[a,b]	<0.001
Physical activity, METs·hour/week	29.9 (16.5–54.2)	33.0 (19.8–56.7)	29.7 (15.4–55.8)	23.1 (11.6–42.8)[a]	27.1 (12.6–48.3)	<0.001
Sedentary time, hour/day	6.0 ± 3.6	5.9 ± 3.6	6.2 ± 3.7	6.1 ± 3.6	6.2 ± 3.3	0.469
Dietary intake, kcal/day	1962.1 ± 597.7	1949.8 ± 583.0	1976.6 ± 620.4	1952.3 ± 596.1	2082.5 ± 675.5	0.289
Protein intake, g/day	83.0 ± 30.6	83.1 ± 29.8	82.6 ± 32.8	80.9 ± 27.8	91.0 ± 37.4	0.189
Fat intake, g/day	61.6 ± 22.0	61.5 ± 21.2	61.7 ± 23.2	60.5 ± 21.4	66.8 ± 27.9	0.182
Carbohydrate intake, g/day	242.1 ± 83.3	241.0 ± 81.4	241.4 ± 85.1	245.0 ± 87.3	250.4 ± 87.4	0.745
Hypertension	1067 (66.1%)	565 (58.9%)	277 (80.8%)	160 (67.8%)	65 (85.5%)	<0.001
Diabetes	208 (12.9%)	88 (9.2%)	55 (16.0%)	46 (19.5%)	19 (25.0%)	<0.001
Hyperlipidemia	1013 (62.7%)	567 (59.1%)	251 (73.2%)	137 (58.1%)	58 (76.3%)	<0.001
Cerebrovascular disease	67 (4.1%)	34 (3.5%)	18 (5.2%)	10 (4.2%)	5 (6.6%)	0.382
Cardiovascular disease	75 (4.6%)	33 (3.4%)	23 (6.7%)	11 (4.7%)	8 (10.5%)	0.006
Current smoking	122 (7.6%)	70 (7.3%)	33 (9.6%)	16 (6.8%)	3 (3.9%)	0.277
Past smoking	663 (41.1%)	385 (40.1%)	160 (46.6%)	87 (36.9%)	31 (40.8%)	0.089
Depression (GDS ≥6 points)	221 (13.7%)	118 (12.3%)	44 (12.8%)	42 (17.8%)	17 (22.4%)	0.018

GDS: Geriatric depression scale.
Data are means ± SD, median (IQR), or n (%).
P values from one-way analysis of variance or Kruskal–Wallis test for continuous variables, and the chi-square test for categorical variables.
$P < 0.05$.
[a] Vs control.
[b] Vs. Obese.
[c] Vs Dynapenia from the Tukey–Kramer,Games–Howell test, or Bonferroni correction.

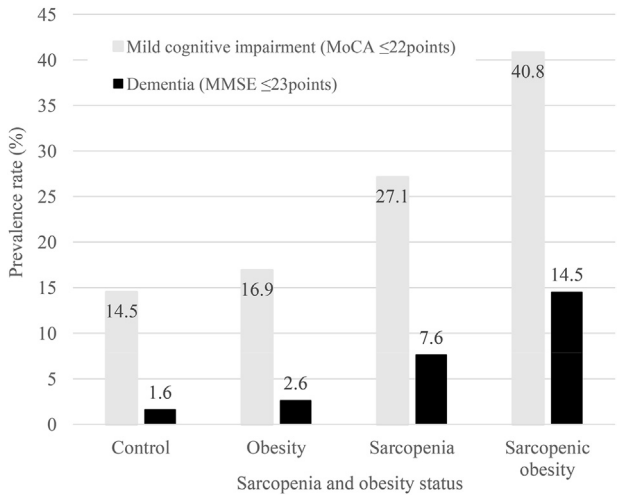

Fig. 2. Prevalence of cognitive impairment by sarcopenia and obesity status.

BMI of ≥25 kg/m^2 [30], because metabolic abnormalities are often developed in Asians with BMI of ≥25 kg/m^2. Using this definition, the present study demonstrated that obesity alone is not associated with MCI or dementia, which is consistent with a meta-analysis showing obesity evaluated based on late-life BMI is not associated with dementia [39,40]. However, obesity, as defined by BMI, may be associated with adverse outcomes differently in different types of outcomes and different populations. For example, obese subjects living in nursing home had better functional status [39]; however, in community-dwelling older people, BMI was negatively correlated with ADL score. In terms of cognitive function, it remains unclear whether the association between obesity and MCI or dementia depends on the study population. Further study is clearly required to clarify these issues.

On the other hand, sarcopenic obesity was associated with the presence of MCI and dementia, and the association of coexisting sarcopenia and obesity on the presence of MCI or dementia seemed to synergistic rather than additive. Similarly, previous study showed that increased handgrip strength is associated with reduced risk of cognitive impairment among obese women (BMI≥25 kg/m^2), but not in non-obese women [41]. The mechanism underlying the synergistic effect is unclear. However, several studies have shown that chronic inflammation is present as the basic common pathophysiology in dementia, obesity, and sarcopenia. Persons with sarcopenic obesity might be in a high chronic inflammation state, which leads to a strong association between sarcopenic obesity and dementia. In addition, since cognitive and motor performance rely on the nervous system, an impaired nervous system may result in decreased cognitive and motor function simultaneously. Higher BMI is associated with brain atrophy [42,43] and also with brain volume deficits in the frontal, temporal, parietal, and occipital lobes in both Alzheimer's disease and MCI [44]. It may also be possible that cognitive impairment reduces physical activity levels in the older adults, resulting in muscle weakness. Furthermore, a previous study demonstrated that obesity determined by percent body fat is a more sensitive marker for cognitive impairment than obesity defined by BMI [6]. This data indicates that high levels of body fat level are strongly associated with cognitive impairment. In this case, the sarcopenic obesity group had higher percent body fat and lower SMI than the obesity group, since muscle strength is positively associated with muscle mass and negatively associated with fat mass in general. Thus, compared with obesity alone, higher fat mass could be detected

Table 2
Adjusted odds ratio and 95% confidence intervals for cognitive impairment by sarcopenia and obesity status.

	Control	Obesity	Sarcopenia	Sarcopenic obesity
Mild cognitive impairment (MoCA≤22 points)				
Age and sex adjustment	1.00 (Referent)	1.06 (0.75–1.50)	1.33 (0.92–1.91)	**2.41 (1.43–4.07)**
Multiple adjustment[a]	1.00 (Referent)	0.95 (0.66–1.35)	1.33 (0.92–1.93)	**2.11 (1.23–3.62)**
Male	1.00 (Referent)	0.98 (0.60–1.616)	1.48 (0.87–2.50)	1.17 (0.51–2.66)
Female	1.00 (Referent)	0.93 (0.54–1.60)	1.17 (0.68–2.01)	**3.25 (1.61–6.60)**
Dementia (MMSE ≤23 points)				
Age and sex adjustment	1.00 (Referent)	1.61 (0.69–3.74)	**3.21 (1.54–6.69)**	**6.33 (2.70–14.80)**
Multiple adjustment[a]	1.00 (Referent)	1.67 (0.69–3.96)	**3.40 (1.61–7.20)**	**6.17 (2.50–15.27)**
Male	1.00 (Referent)	1.85 (0.46–7.36)	2.84 (0.83–9.67)	**7.97 (1.82–34.87)**
Female	1.00 (Referent)	1.69 (0.54–5.28)	**3.56 (1.36–9.33)**	**5.49 (1.65–18.29)**

MoCA: Montreal cognitive assessment, MMSE: Mini-mental state examination.
[a] Multiple adjustment included adjustment for age, sex, year of education, physical activity, hypertension, diabetes, dyslipidemia, and depression status. Bold values indicate statistical significance ($P < 0.05$).

using the definition of sarcopenic obesity, which might be more strongly associated with cognitive impairment in sarcopenic obesity than in obesity alone.

Sarcopenia alone was associated with dementia, but not MCI, in the present study. We found a stronger association between sarcopenia as determined by handgrip strength and dementia than between sarcopenia and MCI. This data is consistent with a recent meta-analysis of longitudinal cohort studies that revealed a positive association between linear rates of change in handgrip strength and changes in cognitive function [45].

In the present study, we defined subjects with low handgrip strength as sarcopenia (probable) [9], and it was expressed as "sarcopenia", because the number of obese subjects with reduced muscle mass is very small. Accordingly, some reports defined sarcopenia by skeletal muscle mass adjusted by BMI or body surface area or height squared [46–48]. This index could be an alternative method to define sarcopenia in obese subjects; however, the association between sarcopenic obesity defined by this index and cognitive impairment remains unclear. Further study is clearly required to clarify the association.

Our additional analyses showed that sarcopenic obesity is significantly associated with MCI and dementia, and sarcopenia is significantly associated with dementia in females, but not in males. These findings align with a previous report that showed a stronger association between weak handgrip strength and cognitive impairment in females than in males [49]. It remains unclear why sarcopenia or sarcopenic obesity is more strongly associated with cognitive impairment in females than males, although multiple cognitive functions are affected both more severely and more widely in women with Alzheimer's disease than men [50].

The current study has several limitations. We used MoCA and MMSE for definitions of MCI and dementia, respectively. Although these tests are screening methods, these definitions for MCI and dementia were generally used in epidemiological studies [20,21]. This cohort included only participants living in an urban part of Japan and the study participants might be relatively healthy and high education level. The prevalence of dementia (3.3%) in the present study was numerically lower than that in 23 community-setting studies (4.9%) [51], which may be due in part to differences in such study populations and/or definition of dementia (e.g. MMSE or ICD or DSM). In addition, the prevalence of probable sarcopenia (19.3%) in the present study was also numerically different from previous studies in Japan (15.2%) [52], Greek (25.4%) [53] and Turkey (11.8%) [5]. These differences of the prevalence of probable sarcopenia may be also due to the differences in study populations and/or definition of probable sarcopenia (e.g. AWGS criteria [9] or EWGSOP criteria [12]). Thus, our results may not be applicable to other populations. Next, this study used BMI to define obesity. Instead of BMI, body fat percentage could be used for the definition of obesity [5,54] and we might identify different results from the present study with this alternate definition [54], because some people who are defined as obese by BMI have a low body fat percentage. On the other hand, measurement of body fat percentage requires a specific device, and in this respect, BMI is advantageous in defining obesity because it can be easily assessed. In this study, handgrip strength was assessed using the average value of both hands. Thus, it may be underestimated compared to the maximum value. We evaluated maximum gait speed in the present study. Therefore, this study might underestimate the prevalence of sarcopenia, because we could not use gait speed for the definition of confirmed sarcopenia. Furthermore, this study was a cross-sectional study, so causal relationships cannot be established. Further prospective and interventional studies are required to clarify the effect of sarcopenic obesity on the incidence of cognitive impairment.

5. Conclusion

Sarcopenic obesity as evaluated based on BMI and handgrip strength was independently associated with MCI and dementia among older Japanese individuals. Future studies are necessary to clarify the causal relationship.

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Author contributions

Y.S., H.K., and Y.T. contributed to study design, participated in data collection, interpreted the results, and wrote and edited the manuscript. Y.S., H.K., D.S., S.K., and R.S. participated in data collection and analysis, and contributed to the discussion. N.H., S.A., Y.M., K.S., H.D., M.I., and K.K. reviewed and revised the manuscript. S.N. provided advice regarding the statistical analysis. R.K. contributed to the discussion. H.W. contributed to the study design, and reviewed and edited the manuscript.

Conflict of Interest

The authors have nothing to disclose.

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