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EPIDEMIOLOGY CLINICAL PRACTICE AND HEALTH

Longitudinal Association Between the Carotid Intima-Media Thickness and Sarcopenia in an Older Japanese Population: The SONIC Study

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ABSTRACT

Aim: Sarcopenia increases the risk of long-term care, disease development, and mortality in older adults. It is also a risk factor for cardiovascular disease and vice versa. Using cross-sectional and longitudinal cohort study designs, we investigated the association between the carotid intima-media thickness (CIMT) and sarcopenia in older adults.

Methods: Data were extracted from the Septuagenarians, Octogenarians, Nonagenarians Investigation with Centenarians (SONIC) study. Using data from Wave 3 of the SONIC participants, we examined the association between CIMT and sarcopenia using cross-sectional analysis and the association between CIMT at baseline and sarcopenia status 3 years later (Wave 4) in a longitudinal study. Sarcopenia was diagnosed according to the Asian Working Group for Sarcopenia (AWGS) 2019 guidelines.

Results: The cross-sectional study included 735 participants (347 males, mean age: 76.0 ± 0.9 years); the longitudinal study included 498 participants (247 males, mean age: 75.9 ± 0.9 years). In the cross-sectional study, 130 (17.7%) participants had sarcopenia; multiple logistic regression analysis revealed an odds ratio (OR) of 1.55 (95% confidence interval [CI]: 1.15–2.07) for sarcopenia for every 1 mm increase in the maximum CIMT. In the longitudinal study, 100 participants (20.1%) had sarcopenia at Wave 4. Multiple logistic regression analysis revealed an OR of 1.63 for sarcopenia (95% CI: 1.13–2.36) for each 1 mm increase from the baseline maximum CIMT.

Conclusion: CIMT was associated with sarcopenia. These results help clarify the pathophysiology of sarcopenia and the possible use of the CIMT as a marker to identify individuals at high risk of sarcopenia.

1 | Introduction

Sarcopenia is a condition characterized by decreased skeletal muscle mass that is complicated by a decline in muscle strength and a reduction in physical performance [1]. Sarcopenia in older adults is associated with a high risk of long-term care, disease development, and even death [2]. Japan has become a super-aging society ahead of the rest of the world. Countermeasures against

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the increase in the number of older adults requiring care and the associated social problems are urgently needed. Therefore, elucidating the pathophysiology of sarcopenia and interventions will extend healthy life expectancies and help solve social issues in a super-aging society.

Sarcopenia has been associated with an increased risk of comorbid cardiovascular diseases (CVD) [3]. In a large prospective cohort study, patients with sarcopenia had a 1.33 times greater risk of developing CVD [4]. Furthermore, CVD has been reported to increase the risk of sarcopenia; in a meta-analysis by Zuo et al., the prevalence of sarcopenia was approximately twice as high in patients with CVD compared to that in the general population [5]. Sarcopenia also increases the risk of all-cause mortality and major adverse cardiac events in patients with chronic heart failure [6–8]. Thus, sarcopenia and CVD have a reciprocal cause-and-effect relationship.

The carotid intima-media thickness (CIMT) is an index measured noninvasively using ultrasonography. CIMT is a strong predictor of the development of CVD and a useful surrogate marker of systemic atherosclerosis [9]. Given the association between sarcopenia and CVD, we hypothesized that CIMT would also be associated with sarcopenia. Several cross-sectional studies have reported that CIMT is associated with reduced leg muscle mass [10], grip strength [11], and walking speed [12]. In a cross-sectional study of patients with chronic kidney disease (CKD), sarcopenia was associated with CIMT. However, these were cross-sectional studies, and thus far, no studies have examined causal relationships over time.

Since CIMT increases before the onset of CVD, we hypothesized that increased CIMT would be associated with the future development of sarcopenia. To test this hypothesis, we conducted a longitudinal analysis of a cohort study of community-dwelling older adults.

2 | Methods

2.1 | Study Design and Setting

We extracted data from participants of the Septuagenarians, Octogenarians, Nonagenarians Investigation with Centenarians (SONIC) study. SONIC is a long-term longitudinal epidemiological study of community-dwelling older adults that was designed to identify the factors associated with health and longevity. Participants were residents of four areas in western and eastern Japan: Itami, Hyogo (western urban); Asago, Hyogo (western rural); Itabashi City, Tokyo (eastern urban); and Nishitama District, Tokyo (eastern rural). The participants lived independently and were randomly selected from a resident registry. We used local survey sites to examine the participants who visited the venues. Detailed information regarding the SONIC study has been published elsewhere [13]. Participants were divided into the 70s, 80s, 90s, and 100s age groups and followed up every 3 years. Participants aged 70 years in 2010, 80 years in 2011, and 90 and 100 years in 2012 were analyzed at Wave 1. Participants aged 73 years in 2013, 83 in 2014, and 93 in 2015 were analyzed at Wave 2. Our current analysis included 76-year-old residents surveyed in 2016 as part of Wave 3. After excluding participants with missing values, we conducted a cross-sectional study with 735 participants at Wave 3. Furthermore, we conducted a longitudinal study of 498 participants who were registered at both Waves 3 and 4 in 2019 (Figure 1).

The study protocol was approved by the Institutional Review Board of Osaka University Graduate School of Medicine, Dentistry, and Human Sciences (approval numbers: 266, H22-9, and 22018). All participants provided written informed consent after obtaining detailed information. This study was conducted in accordance with the principles of the Declaration of Helsinki.

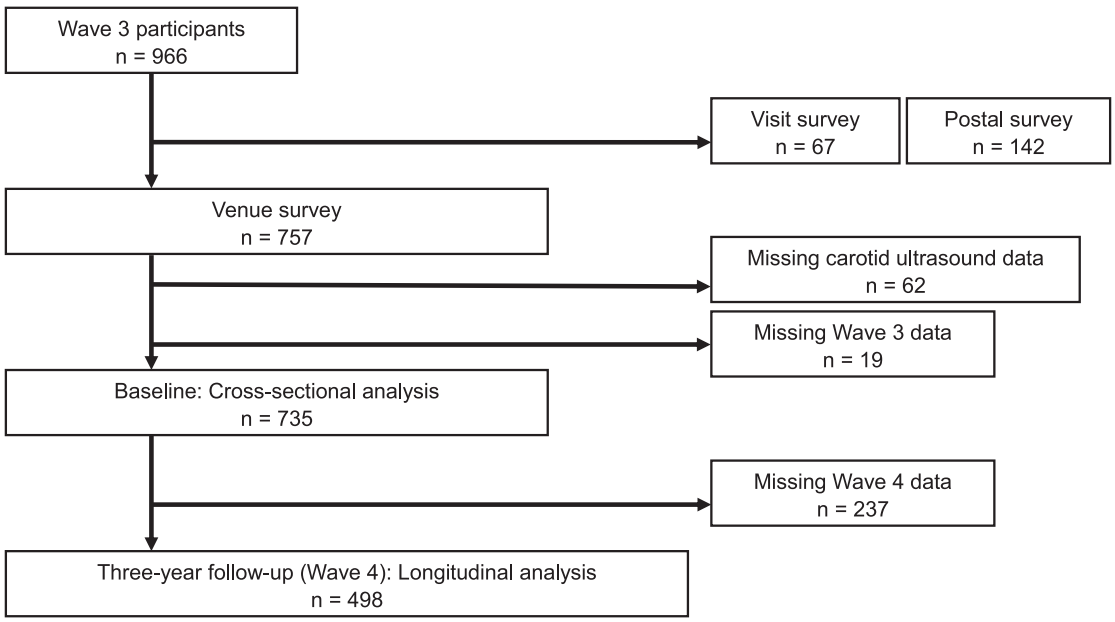

FIGURE 1 | Flow chart of study participants.

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2.2 | Measurements

We interviewed the participants at the SONIC study venue to obtain their life and medical histories and gathered data, including anthropometric measurements, blood pressures, body composition, grip strength, gait speed, and the short physical performance battery (SPPB). A trained physician or nurse recorded the patient's life and medical history. Anthropometric measurements included height and weight, which were used to calculate the body mass index (BMI). The blood pressure was measured in the upper arm using a mercury sphygmomanometer by a trained physician or nurse after the participants were seated for at least 5 min. The body composition was measured using the bioimpedance method, and the appendicular skeletal muscle mass was calculated. We used the InBody 430 (InBody Co. Ltd., Seoul, South Korea) in the Itabashi and Nishitama areas and the MC-780A (Tanita Corporation, Tokyo, Japan) in the Itami and Asago areas. The grip strength was measured twice on each side in the standing position using a Smedley grip strength meter (YD-100; Yagami Inc., Nagoya, Japan), and the maximum value was adopted. A distance of 2.44 m was measured on a flat floor, and the starting and finishing points were marked with tape. Participants walked twice from the starting point, passed through the finishing point, and stopped beyond it. The assessor accompanied the participant and measured the walking time for a 2.44-m distance to the nearest 0.1 s using a digital stopwatch (Picco Alba W071; Seiko Holdings Corporation, Tokyo, Japan). The fastest measurement was adopted as the walking speed. The SPPB was performed according to established methods [14] and included balance, walking, and five-chair stand tests; the total score was calculated by combining the results.

Based on the physical function test results and presence of muscle weakness or performance impairment, in addition to muscle mass loss, sarcopenia was diagnosed according to the Asian Working Group for Sarcopenia (AWGS) 2019 criteria [1], as follows:

- Muscle loss was defined as an appendicular skeletal muscle mass of $< 7.0\,kg/m^2$ in males and $< 5.7\,kg/m^2$ in females.
- Muscle weakness was defined as a grip strength of $< 28\,kg$ in males and $< 18\,kg$ in females.
- Performance impairment was defined as a gait speed of $< 1.0\,m/s$, five-chair stand test of $\leq 12\,s$, or SPPB score of ≤ 9 points.

Trained physicians performed an ultrasound examination of the carotid arteries using a GE LOGIQ Book XP (GE HealthCare Japan Corporation, Tokyo, Japan). The common and internal carotid arteries were scanned in B-mode with a 7.5 MHz linear probe to measure the intima-media thickness to the nearest 0.1 mm [15]. The maximum CIMT was defined as the maximum observable intima-media thickness in the area. Bilateral measurements were taken from the near and far walls of the common carotid artery (CCA), carotid bulb, and internal carotid artery (ICA). Any focal atherosclerotic plaques were included in the measurement. All measurements were performed manually by trained physicians. The highest value among these regions was recorded as the maximum CIMT for analysis.

Blood samples were collected in a resting sitting position, and general biochemical parameters, including serum creatinine levels, were measured. The serum creatinine levels were measured using enzymatic methods. The estimated glomerular filtration rate (eGFR) was calculated from the serum creatinine level and age using the Japanese Society of Nephrology estimation formula [16]. We defined CKD as an eGFR of $< 60\,mL/min/1.73\,m^2$.

2.3 | Statistical Analysis

Data are presented as means ± standard deviation, median (interquartile range), or *n* (%). To compare the differences in clinical characteristics between male and female participants, we used the t-test as a parametric test and the *U*-test as a nonparametric test for continuous variables. We used the chi-squared test for categorical variables. In addition, multiple logistic regression analyses were performed with sarcopenia as the dependent variable. We selected independent variables associated with sarcopenia, including the maximum CIMT, sex, BMI, systolic blood pressure, CKD, history of smoking, diabetes, dyslipidemia, and history of CVD. Owing to the age-aligned nature of the SONIC study, age was not included as an independent variable. In the analysis with sarcopenia as the dependent variable in Wave 4 (longitudinal study), we added sarcopenia in Wave 3 to the aforementioned independent variables. Finally, to examine the factors associated with new-onset sarcopenia, we performed a subanalysis excluding participants with sarcopenia in Wave 3. Two-sided p values < 0.05 were considered statistically significant. All statistical analyses were performed using SPSS version 27 (IBM Corp., Armonk, NY, USA).

3 | Results

Figure 1 illustrates the flow of the study participants. Of the participants aged approximately 76 years in Wave 3 of the SONIC study, 757 were tested at the study venue. After excluding participants with missing data, 735 participants were included in the cross-sectional analysis; of them, 498 with no missing data were included in Wave 4, 3 years later, in the longitudinal study.

Baseline clinical characteristics of participants included in the longitudinal analysis ($n = 498$) and those lost to follow-up ($n = 237$) are summarized in Table S1. Walking speed and grip strength showed significant variations between groups; no other variables showed significant differences.

Of the 735 participants included in the cross-sectional analysis, 130 (17.7%) met the inclusion criteria for sarcopenia. Table 1A shows the clinical characteristics of participants in the cross-sectional analysis. Age, BMI, blood pressure, eGFR, prevalence of hypertension, and criteria for sarcopenia differed between the sarcopenia and non-sarcopenia groups. Multiple logistic regression analysis was performed with the presence or absence of sarcopenia as the dependent variable (Table 2), and the maximum CIMT was independently associated with sarcopenia. The odds ratio was 1.55 (95% confidence

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TABLE 1A | Baseline clinical characteristics of the study participants in the cross-sectional analysis.

	No sarcopenia ($n=605$)	Sarcopenia ($n=130$)	p
Age, years	75.9 ± 0.9	76.1 ± 0.9	0.009
Male, *n* (%)	289 (47.8)	58 (44.6)	0.514
Body mass index, kg/m^2	23.5 ± 2.9	20.4 ± 2.4	< 0.001
Systolic BP, mmHg	138 ± 16	133 ± 18	0.010
Diastolic BP, mmHg	77 ± 10	72 ± 10	< 0.001
Estimated GFR, mL/min/1.73 m^2	64.1 [56.7–73.5]	69.1 [59.2–80.2]	< 0.001
CKD, *n* (%)	208 (34.7)	36 (27.7)	0.124
Maximum CIMT, mm	1.40 [1.10–1.90]	1.50 [1.00–2.10]	0.368
Skeletal muscle mass index, kg/m^2	7.0 ± 1.0	5.9 ± 0.7	< 0.001
Walking speed, m/s	1.1 ± 0.2	1.0 ± 0.2	< 0.001
Grip strength, kg	26.3 ± 7.9	21.7 ± 7.9	< 0.001
History of smoking, *n* (%)	227 (37.9)	52 (40.0)	0.655
Hypertension, *n* (%)	216 (36.1)	33 (25.4)	0.019
Diabetes, *n* (%)	61 (10.3)	13 (10.0)	0.931
Dyslipidemia, *n* (%)	212 (35.5)	37 (28.5)	0.128
History of stroke, *n* (%)	32 (5.4)	6 (4.7)	0.754
History of heart failure, *n* (%)	5 (0.8)	0 (0.0)	0.381
History of myocardial infarction, *n* (%)	14 (2.3)	5 (3.9)	0.235
History of cardiovascular disease, *n* (%)	46 (7.9)	10 (8.1)	0.940

Note: Data are presented as mean ± SD, median [interquartile range], or *n* (%).
Abbreviations: BP, blood pressure; CIMT, carotid intima-media thickness; CKD, chronic kidney disease (defined as an eGFR < 60 mL/min/1.73 m^2); GFR, glomerular filtration ratio.

interval [CI]: 1.15–2.07) for each 1 mm increase in the maximum CIMT.

Among the 498 participants who underwent longitudinal analysis, 81 (16.2%) met the criteria for sarcopenia at Wave 3, and 100 (20.1%) met the criteria for sarcopenia 3 years later at Wave 4. The baseline clinical characteristics of the participants, classified according to the presence or absence of sarcopenia at Wave 4, are shown in Table 1B. Age, BMI, diastolic blood pressure, and dyslipidemia differed between the sarcopenia group at Wave 4 and the non-sarcopenia group. Multiple logistic regression analysis with sarcopenia at Wave 4 as the dependent variable identified an independent association with the maximum CIMT at baseline. The odds ratio was 1.63 (95% CI: 1.13–2.36) for each 1 mm increase in maximum CIMT (Table 3A).

Furthermore, baseline clinical characteristics of participants without sarcopenia at baseline, stratified by sarcopenia status at Wave 4, are shown in Table 1C. Only BMI and skeletal muscle mass index showed variation, whereas other variables showed no significant differences. When we performed the same multiple logistic regression analysis excluding participants with sarcopenia at Wave 3 and using new-onset sarcopenia as the dependent variable, the odds ratio for each 1-mm increase in baseline maximum CIMT was 1.60 (95% CI: 1.04–2.47), as shown in Table 3B.

4 | Discussion

Using cross-sectional and longitudinal study designs, we analyzed the association between sarcopenia and the maximum CIMT measured using ultrasound in community-dwelling older adults in the SONIC study. In both cross-sectional and longitudinal studies, the maximum baseline CIMT was independently associated with sarcopenia, suggesting that subclinical atherosclerosis, expressed as CIMT thickening, may be involved in the pathogenesis of sarcopenia. The maximum CIMT may also be a marker for identifying individuals at high risk of sarcopenia. We selected the maximum CIMT, rather than the mean CIMT, as it reflects focal arterial wall thickening and includes plaque burden. Prior studies have shown that the maximum CIMT is more strongly associated with adverse outcomes such as CVD, cognitive decline, and sarcopenia, compared to the mean CIMT [9].

To date, studies with different research designs have reported the relationship between CIMT and sarcopenia, muscle strength, and physical performance. In studies of the general population, CIMT has been related to grip strength [11], and

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TABLE 1B | Baseline clinical characteristics of the study participants in the longitudinal analysis.

	No sarcopenia after 3 years ($n = 398$)	Sarcopenia after 3 years ($n = 100$)	p
Age, years	75.9 ± 0.9	76.0 ± 1.0	0.049
Male, *n* (%)	199 (50.0)	48 (48.0)	0.721
Body mass index, kg/m^2	23.4 ± 2.9	21.0 ± 2.5	< 0.001
Systolic BP, mmHg	138 ± 15	135 ± 18	0.11
Diastolic BP, mmHg	77 ± 10	74 ± 10	0.037
Estimated GFR, mL/min/1.73 m^2	65.2 [58.4–73.7]	68.2 [57.6–79.6]	0.103
CKD, *n* (%)	124 (31.5)	34 (34.0)	0.628
Maximum CIMT, mm	1.40 [1.10–1.90]	1.50 [1.10–2.08]	0.107
Skeletal muscle mass index, kg/m^2	7.0 ± 1.0	6.1 ± 0.8	< 0.001
Walking speed, m/s	1.1 ± 0.2	1.0 ± 0.2	0.037
Grip strength, kg	26.8 ± 7.8	23.9 ± 7.5	0.001
Sarcopenia at baseline, *n* (%)	28 (7.0)	53 (53.0)	< 0.001
History of smoking, *n* (%)	154 (39.0)	41 (41.0)	0.713
Hypertension, *n* (%)	148 (37.3)	28 (28.0)	0.083
Diabetes, *n* (%)	41 (10.4)	5 (5.1)	0.104
Dyslipidemia, *n* (%)	150 (37.9)	27 (27.0)	0.042
History of stroke, *n* (%)	20 (5.1)	6 (6.1)	0.704
History of heart failure, *n* (%)	2 (0.5)	2 (2.0)	0.182
History of myocardial infarction, *n* (%)	8 (2.0)	3 (3.0)	0.385
History of cardiovascular disease, *n* (%)	28 (7.3)	8 (8.2)	0.764

Note: Data are presented as mean ± SD, median [interquartile range], or *n* (%).
Abbreviations: BP, blood pressure; CIMT, carotid intima-media thickness; CKD, chronic kidney disease (defined as an eGFR < 60 mL/min/1.73 m^2); GFR, glomerular filtration ratio.

CIMT and the number of plaques in the carotid artery have been related to walking speed and balance function [12]. In a study of males who underwent anti-aging medical checkups, Ochi et al. reported that the CIMT, brachial-ankle pulse wave velocity (baPWV), and mid-thigh muscle cross-sectional areas are related [10]. A cross-sectional clinical study of patients with CKD undergoing conservative and replacement therapy reported that sarcopenia is associated with increased CIMT and pericardial fat and decreased endothelial function [17]. In addition, CIMT has been associated with grip strength and walking speed but not with sarcopenia in older patients with Type 2 diabetes [18]. In contrast, one study reported that CIMT is not associated with muscle mass [19]. This may be explained by the relatively young age of the participants (most were 20–40 years old), and most had CIMT of < 1.0 mm. Such a narrow distribution may have limited the detection of any association due to the so-called "floor effect." Nevertheless, these were all cross-sectional studies, and we are the first to report a causal relationship using a longitudinal study design. Furthermore, we used the gait speed, five-chair stand test, and SPPB scores to diagnose sarcopenia based on the AWGS 2019 criteria. As a result, we were able to diagnose sarcopenia in more detail than in previous studies.

One possible mechanism linking CIMT and sarcopenia involves vascular and skeletal muscle interactions. Muscle satellite cells, which are tissue stem cells in skeletal muscle, play an essential role in the growth and hypertrophy of skeletal muscle and in the regeneration of damaged skeletal muscle, and satellite cell dysfunction contributes to the development of sarcopenia [20]. Satellite cells promote angiogenesis and myogenesis through crosstalk with vascular endothelial cells via vascular endothelial growth factor (VEGF), hepatocyte growth factor (HGF), and insulin-like growth factor-1 (IGF-1) [21, 22]. In cellular experiments, the proliferation and differentiation of muscle satellite cells are negatively impacted by high glucose concentration-induced endothelial cell damage [23]. In animal experiments using transgenic mice with upregulated VEGF, Grunewald et al. reported that atrophic changes in skeletal muscle and muscle weakness were suppressed with a concurrent decrease in age-related capillary suppression [24]. Therefore, we hypothesized that damage to the vascular endothelium leads to a decline in muscle satellite cell function, which may lead to sarcopenia. In atherosclerosis, CIMT may increase as damage to the vascular endothelium progresses [25]. The decline in muscle satellite cell function may mediate the association between CIMT and sarcopenia.

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TABLE 1C | Baseline clinical characteristics of the study participants in the longitudinal analysis, excluding participants with sarcopenia at baseline.

	No sarcopenia after 3 years ($n = 370$)	Sarcopenia after 3 years ($n = 47$)	p
Age, years	75.9 ± 0.9	76.1 ± 1.0	0.161
Male, *n* (%)	185 (50.0)	23 (48.9)	0.891
Body mass index, kg/m^2	23.7 ± 2.8	21.8 ± 2.5	< 0.001
Systolic BP, mmHg	138 ± 15	139 ± 18	0.797
Diastolic BP, mmHg	77 ± 10	75 ± 10	0.171
Estimated GFR, mL/min/1.73 m^2	64.1 [50.9–73.7]	64.1 [55.1–74.5]	0.822
CKD, *n* (%)	117 (32.0)	19 (40.4)	0.245
Maximum CIMT, mm	1.40 [1.10–1.90]	1.50 [1.10–2.20]	0.293
Skeletal muscle mass index, kg/m^2	7.1 ± 1.0	6.4 ± 0.8	< 0.001
Walking speed, m/s	1.1 ± 0.2	1.1 ± 0.2	0.472
Grip strength, kg	26.9 ± 7.8	25.2 ± 6.8	0.16
History of smoking, *n* (%)	143 (39.0)	18 (38.3)	0.93
Hypertension, *n* (%)	140 (37.9)	15 (31.9)	0.421
Diabetes, *n* (%)	38 (10.3)	3 (6.5)	0.601
Dyslipidemia, *n* (%)	138 (37.5)	13 (27.7)	0.187
History of stroke, *n* (%)	19 (5.2)	4 (8.7)	0.308
History of heart failure, *n* (%)	2 (0.6)	2 (4.3)	0.064
History of myocardial infarction, *n* (%)	8 (2.2)	1 (2.2)	1.00
History of cardiovascular disease, *n* (%)	27 (7.5)	5 (10.9)	0.388

Note: Data are presented as mean ± SD, median [interquartile range], or *n* (%). BP, blood pressure; GFR, glomerular filtration ratio; CKD, chronic kidney disease (defined as an eGFR < 60 mL/min/1.73 m^2); CIMT, carotid intima-media thickness.
Abbreviations: BP, blood pressure; CIMT, carotid intima-media thickness; CKD, chronic kidney disease (defined as an eGFR < 60 mL/min/1.73 m^2); GFR, glomerular filtration ratio.

TABLE 2 | Multiple logistic regression analysis of sarcopenia at baseline (cross-sectional analysis).

	OR	95% CI	p
Sex, male	1.23	0.68–2.24	0.50
Body mass index, kg/m^2	0.61	0.54–0.67	< 0.0001
Systolic BP, mmHg	0.99	0.98–1.01	0.44
CKD	0.74	0.45–1.21	0.22
History of smoking	1.97	1.08–3.59	0.027
Diabetes	0.78	0.36–1.70	0.53
Dyslipidemia	0.77	0.48–1.26	0.30
CVD	0.88	0.37–2.09	0.78
Maximum CIMT, mm	1.55	1.15–2.07	0.004

Abbreviations: BP, blood pressure; CI, confidence interval; CIMT, carotid intima-media thickness; CKD, chronic kidney disease (defined as an eGFR < 60 mL/min/1.73 m^2); CVD, cardiovascular disease; OR, odds ratio.

Furthermore, the SONIC study group has previously reported additional mechanisms that may link CIMT and sarcopenia. These include systemic inflammation, periodontal disease, nutritional status, and hyperuricemia. Chronic inflammation and periodontal disease can accelerate the progression of atherosclerosis, which may subsequently contribute to sarcopenia through impaired muscle function, potentially increasing the CIMT. The SONIC study group has demonstrated an association between periodontal inflamed surface area and systemic inflammation markers, such as high-sensitive C-reacting protein (hs-CRP), which suggests a potential link between oral health and atherosclerosis [26]. Moreover, elevated uric acid levels have been shown to be an independent risk factor for increasing CIMT in females, which may further mediate the association between atherosclerosis and sarcopenia [27]. Additionally, malnutrition, which is often associated with diminished occlusal support, is a contributory factor in the progression of both atherosclerosis and sarcopenia [28]. These findings demonstrate the existence of multiple interconnected pathways through which atherosclerosis and related systemic conditions may influence the development and progression of sarcopenia.

This study had several limitations. First, the participants were limited to older adults living in the designated areas who voluntarily visited the health survey venue, which might have led to selection bias. In addition, approximately 40% of the participants at baseline were lost to follow-up; therefore, the

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TABLE 3A | Multiple logistic regression analysis of sarcopenia after 3 years (longitudinal analysis).

	OR	95% CI	*p*
Sex, male	1.42	0.68–2.94	0.35
Body mass index, kg/m^2	0.79	0.70–0.89	<0.0001
Systolic BP, mmHg	1.00	0.98–1.02	0.96
CKD	1.32	0.73–2.37	0.36
History of Smoking	1.46	0.71–3.03	0.31
Diabetes	0.44	0.14–1.38	0.16
Dyslipidemia	0.61	0.33–1.10	0.10
CVD	1.33	0.49–3.60	0.58
Sarcopenia at baseline	9.07	4.77–17.3	<0.0001
Maximum CIMT, mm	1.63	1.13–2.36	0.010

Abbreviations: BP, blood pressure; CI, confidence interval; CIMT, carotid intima-media thickness; CKD, chronic kidney disease (defined as an eGFR <60 mL/min/1.73 m^2); CVD, cardiovascular disease; OR, odds ratio.

TABLE 3B | Multiple logistic regression analysis of sarcopenia after 3 years, excluding participants with sarcopenia at baseline (longitudinal analysis).

	OR	95% CI	*p*
Sex, male	1.43	0.59–3.49	0.43
Body mass index, kg/m^2	0.72	0.62–0.83	<0.0001
Systolic BP, mmHg	1.01	0.99–1.03	0.53
CKD	1.58	0.80–3.14	0.19
History of smoking	1.62	0.67–3.91	0.29
Diabetes	0.48	0.13–1.77	0.27
Dyslipidemia	0.73	0.36–1.49	0.39
CVD	1.31	0.43–4.00	0.64
Maximum CIMT, mm	1.60	1.04–2.47	0.034

Abbreviations: BP, blood pressure; CI, confidence interval; CIMT, carotid intima-media thickness; CKD, chronic kidney disease (defined as an eGFR <60 mL/min/1.73 m^2); CVD, cardiovascular disease; OR, odds ratio.

longitudinal analysis may have included only healthier participants. Consistent with this concern, participants lost to follow-up had significantly lower baseline walking speed and grip strength compared to those who were retained (Table S1). Furthermore, in our cohort, baseline systolic blood pressure, CKD, history of smoking, diabetes, dyslipidemia, and CVD were not significantly associated with sarcopenia (Table 3A) or with new-onset sarcopenia (Table 3B). Second, although carotid ultrasound examinations were performed by trained physicians using a standardized method, multiple examiners performed the examinations, and inter-examiner errors were possible. Third, although bioelectrical impedance analysis was performed using InBody devices, the measurement conditions (e.g., fasting state, time of day, hydration status) were not standardized across the participants. This lack of uniformity may have introduced variability in the assessment of skeletal muscle mass and potentially influenced the results. Fourth, the medical histories of the study participants were based on interviews and were not corroborated by medical records from hospitals or clinics. Therefore, a history of CVD, which served as a covariate in the multivariate analysis, might have been overlooked.

In conclusion, we have identified that the maximum CIMT is associated with the existence and onset of sarcopenia. These results help clarify the pathophysiology of sarcopenia. CIMT may also be a marker for identifying groups at high risk of sarcopenia. Future interventional studies targeting subclinical arteriosclerosis are expected to help prevent and treat sarcopenia.

Author Contributions

All authors critically revised the manuscript for important intellectual content and gave their final approval for submission. Additionally, the authors provided the following specific contributions: K.T. and H.A. collected the data, analyzed the data, and drafted the article. Y.O., T.F., Sh.Y., R.O., T.M., Y.Y., M.I., K.G., Y.A., Mi.K., Ma.K., Sa.Y., M.O., T.N., and Y.M. collected the data. T.H. and Y.A. conceived of the research. Y.G., K.I., K.K., and K.Y. designed the study and interpreted the data.

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Ethics Statement

The study protocol was approved by the Institutional Review Board of Osaka University Graduate School of Medicine, Dentistry, and Human Sciences (approval numbers 266, H22-9, and 22018, respectively). The study was conducted in accordance with the Declaration of Helsinki and its later amendments.

Consent

Written informed consent was obtained from all participants.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Table S1:** Baseline clinical characteristics of the study participants in the longitudinal analysis and participants lost to follow-up.

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