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Sarcopenia and Physical Function in Middle-Aged and Older Stroke Survivors

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Abstract

Objective—To determine the prevalence of sarcopenia in stroke survivors using different methodologies and compared a subset of the stroke group to age, sex, and BMI-matched non-stroke control counterparts.

Design—Cohort Study

Setting—Veterans Affairs Medical Center and University Hospital

Participants—Mild to moderately disabled chronic participants with stroke aged 40–84 yrs (n=190, 61% male, 57% African American, BMI: 29±1 kg/m², X±SEM)

Interventions—Not applicable

Main Outcome Measures—DXA scans to assess appendicular lean mass (ALM). Rates of sarcopenia were determined using four established methods: 1) ALM/ht²; 2) European Working Group on Sarcopenia in Older Persons; 3) International Working Group on Sarcopenia; and 4) ALM/BMI.

Results—Sarcopenia prevalence in our stroke cohort ranged between 14% and 18%. The stroke survivor subset (n=38) matched one-for-one with control counterparts for race, sex, age±4 years and BMI±2.5 units had higher prevalence rates compared to their non-stroke counterparts (13.2% vs. 5.3%, P<0.0001). ALM/ht² was related to six-minute walking speed (r=0.28, P<0.01) and VO₂peak (l/min r=0.58, P<0.0001) for the stroke group.

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Conflict of Interest

None

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Conclusions—Stroke survivors show an elevated prevalence of sarcopenia when considering age, sex, and race to non-stroke individuals.

Keywords

sarcopenia; frailty; exercise; muscles; stroke

The prevalence of stroke, a leading cause of disability in adults, is expected to substantially increase over the next two decades¹. Approximately 50% of stroke patients suffer some degree of hemiparesis, rendering 30% unable to walk without an assistive device, and frequently resulting in long-term disability². After a stroke, the progression and consequences of muscle wasting may be compounded by their relative inactivity, reduced strength, and lower fitness levels. Our previous findings indicate that the paretic thigh of stroke survivors has 20–24% lower muscle area and muscle volume and 17–25% higher intramuscular fat than the non-paretic thigh^{3,4}. Furthermore, we have previously demonstrated that reduced muscle mass and greater severity of hemiparetic gait deficits are independent determinants of lower peak fitness⁵, suggesting that the primary components of sarcopenia after stroke translate directly into altered functional capacity and frailty.

Sarcopenia, originally described as a general loss of muscle mass with age⁶, is now more precisely characterized using clinical cutpoints that utilize lean mass measured by dual-energy x-ray absorptiometry and comparisons to a young reference population⁷. The definitions of sarcopenia continue to evolve by accounting for body size or body fat, strength and function^{8–11}. Although the prevalence of sarcopenia has been reported in the literature using accepted cut point methodologies¹², prevalence studies that are exclusive to stroke are limited. Given that sarcopenia increases the risk for subsequent injury and disability^{7,13,14} and predicts hospitalization and death¹⁵, it would be highly clinically relevant to arrive at a better understanding of sarcopenia prevalence in this at-risk population.

We tested the hypothesis that stroke survivors would have higher prevalence of sarcopenia than non-stroke controls. Thus, the aim of this study was to 1) determine the prevalence of sarcopenia in a large chronic stroke sample and 2) compare the prevalence of sarcopenia in a subset of our stroke group to age, sex and BMI-matched non-stroke controls.

Methods

Subject Selection

Stroke survivors (n=190, 116 men, 74 women) between 40–84 years and BMI between 16.5–44.6 kg/m² (range underweight to morbid obesity) with residual hemiparetic deficits >six months after onset of ischemic stroke participated in the study. Subjects were recruited through local advertisements, flyers and stroke clinics at the University of Maryland School of Medicine and Baltimore VA Medical Center. All participants with stroke had mild to moderate hemiparetic gait deficits characterized by reduced stance or reduced stance with increased swing duration, and had completed conventional rehabilitation therapy. All subjects lived independently in the community and were able to walk six minutes with their usual assistive device (s) without human assistance. Ambulation was the primary means of

mobility for participants with stroke. Use of an ankle-foot orthosis (AFO) or assistive device was documented. Evaluations included medical history, physical examination, fasting blood profile, and screening for dementia¹⁶ and depression¹⁷ to ensure adequate informed consent. Participants with stroke were excluded if they had unstable angina, congestive heart failure (NYHA II), severe peripheral arterial disease, end-stage organ disease, major post-stroke depression, dementia, severe receptive aphasia, orthopedic or chronic pain conditions. Healthy control counterparts had the same exclusion criteria as participants with stroke and were 46–80 years of age with a BMI between 21–42 kg/m². All subjects were sedentary as defined by <20 min low intensity exercise 3x/week.

The Institutional Review Board of the University of Maryland and the Baltimore VA Research & Development Committee approved all methods and procedures. Each participant provided written informed consent.

Exercise and Functional Tests

Exercise testing with open circuit spirometry was conducted to measure VO₂peak using a graded treadmill test¹⁸. A standardized six-minute walk test recorded the distance traveled, with participants with stroke walking at their comfortable self-selected walking speed using their usual assistive devices to determine gait speed. Seventeen participants with stroke did not have VO₂peak testing and 22 were missing the six-minute walk test.

Body Composition and Sarcopenia

Height (cm) and weight (kg) were measured. Fat mass, lean tissue mass and %body fat were determined by DXA (Prodigy and iDXA LUNAR GE).

Four definitions for sarcopenia were applied to classify sarcopenia^{7–11}. These include the definition as appendicular lean mass/height², ALM/ht² <7.26 kg/m² in men and <5.45 kg/m² in women⁷. Second, sarcopenia as agreed to by the European Working Group on Sarcopenia in Older People (EWGSOP)⁸, includes gait speed <0.8 m/s and ALM <7.23 kg/m² in men and ALM <5.67 kg/m² in women. Third, the International Working Group on Sarcopenia (IWG) defines sarcopenia as gait speed <1.0 m/s and ALM/ht² <7.23 kg/m² in men and <5.67 kg/m² in women⁹. Fourth, ALM divided by BMI was calculated to discriminate the presence or absence of weakness with cutpoints as follows: ALM/BMI <0.789 in men and ALM/BMI <0.512 in women¹¹. Finally, in order to eliminate potential confounders of sarcopenia, a matched pair analysis was performed wherein stroke survivors were matched with non-stroke control counterparts on the basis of race, sex, age ± 4 years and BMI ± 2.5 kg/m².

Statistical Analyses

Descriptive statistics were analyzed using SPSS (PASW Statistics, Version 18, Chicago, IL). Differences by sex and between stroke and non-stroke control counterparts for all variables were determined using unpaired Student *t*-tests and variances compared using Levine's test for equality of variances. Relationships between variables were determined by linear regression analyses with calculation of Pearson product moment correlation coefficients. Statistical assumptions are verified through tests of normality by the Kolmogorov-Smirnov

Test and random sampling of subjects. Sample size analyses conducted assuming a significance of 5% and 80% power indicate that the effect size (f^2) for the regression analyses ranged from 0.085–0.136, suggesting the need for sample sizes ranging from 27–143 stroke subjects, which is within the 190 subjects. Data are presented as means \pm SEM. P values <0.05 are statistically significant.

Results

Physical Characteristics (Table 1)

Subjects with stroke are 61% male and 39% female. The group is also racially mixed with 42% Caucasian (n=78), 57% African-American (n=108), and the remaining 2% (n=4) other nationalities. Latency since stroke averaged 51 ± 7 months (range 6 – 375 months). Twenty-three percent of participants with stroke had diagnosed type 2 diabetes upon study entry. Approximately 57% of participants with stroke used a single point cane, 14% used a quad cane, 14% used a walker, and 15% did not use any assistive device for ambulation and 50% used an AFO. Men have higher body weight than women ($P<0.005$) but BMI is not different. Women have higher percent body fat and fat mass ($P<0.0001$) and lower total lean mass, paretic and non-paretic lean mass ($P<0.0001$) than men. ALM and ALM/ ht^2 are higher in men than women ($P<0.0001$). VO_2 peak (l/min) is 1.3 fold-higher in men than women ($P<0.0001$) but is not significantly different when expressed per body weight (ml/kg/min). Gait speed is also ~ 1.3 fold higher in men than women ($P<0.005$). Paretic leg lean mass by DXA is lower than non-paretic leg lean mass in the total group ($P<0.0001$), and in both men ($P<0.0001$) and women ($P<0.05$).

The total prevalence of sarcopenia, independent from sex, ranged from 14.3% – 17.9%, depending on the method used for the diagnosis (Figure 1). The lowest prevalence in the total group is found when using the EWGSOP criteria (14.3%) and the highest prevalence is found when using the ALM/BMI criteria (17.9%). When stratified by sex, the prevalence of sarcopenia is greater in women than men using the criteria of EWGSOP (19 vs.11.5%) and the IWG (22.2 vs.13.5%) but higher in men than women by ALM/BMI (23.3 vs. 9.5%) criteria. By the ALM/ ht^2 criteria, there is the smallest difference by sex (men vs. women: 17.2 vs. 16.2%).

The prevalence of sarcopenia in the total group increases with age using all the criteria except the IWG (Table 2). The highest prevalence in individuals over the age of 60 years is 23% using the ALM/BMI criteria and is twice that of individuals younger than 60 years (10%). In a breakdown by age and sex, the prevalence of sarcopenia is slightly greater in women and men older than 60 years (both 18%) than those younger than 60 years by ALM/ ht^2 (15 and 16%, respectively) By the EWGSOP, prevalence is similar between age groups in men (12vs. 11%) but about 6% higher in older than younger women. In contrast, the prevalence of sarcopenia with the IWG criteria is slightly higher in both men and women younger than 60 than those older than 60 years of age. With the ALM/BMI criteria, the prevalence increases with age in men but declines in women.

The non-stroke control counterparts are well matched to the stroke group (n=38 per group) in terms of age (controls: 63 ± 1 years, range 46–80 years and stroke: 63 ± 1 years, range

49–81 years) and BMI (controls: 32 ± 1 kg/m², range 21–42 kg/m² and stroke: 32 ± 1 kg/m², range 21–41 kg/m²). None of the control subjects had type 2 diabetes and 31% of the matched stroke group had diagnosed type 2 diabetes. After matched pair analysis, 5.3% of the control counterparts have sarcopenia while 13.2% of the stroke group have sarcopenia ($P < 0.0001$). Lean mass of both the paretic and non-paretic leg are lower than lean mass of the right side of the control counterparts (both $P < 0.0001$), suggesting that the frequency of the sarcopenia did not result only from the loss of muscle mass of the affected side.

ALM/ht² is related to gait speed ($r = 0.28$, $P < 0.0001$) and VO₂peak (l/min $r = 0.58$, $P < 0.0001$ and ml/kg/min $r = 0.26$, $P < 0.0005$) independent of sex in the stroke population. Likewise, ALM/BMI is related to gait speed ($r = 0.34$, $P < 0.0001$) and VO₂peak (l/min $r = 0.47$, $P < 0.0001$ and ml/kg/min $r = 0.48$, $P < 0.0001$) independent of sex.

Discussion

The present study is the first to show that the prevalence of sarcopenia in stroke varies between 14% and 18%. Further, we show differences in sarcopenia by sex. When matched to non-stroke control counterparts, stroke survivors may be at an elevated risk for sarcopenia when considering age, sex, and race.

Sarcopenia is shown to increase with age¹⁹ with loss of muscle mass associated with an increased risk of incident disability and all-cause mortality¹³. The increase in mortality risk with sarcopenia is independent of age, sex, education, activities of daily living, and disease¹⁹. Sarcopenia also leads to higher health care costs in the US²⁰. Risk factors for sarcopenia include advancing age, low levels of physical activity, inadequate nutrition, and co-morbid disease (i.e. type 2 diabetes mellitus and end-stage organ diseases)¹³.

We find a 14–18% prevalence rate in the group as a whole. Our findings in stroke survivors generally fall within the sarcopenia prevalence range (1–29%) reported in other populations¹²; however, prevalence rates can depend on the age of the subjects and the diagnostic criteria. In the Leiden Longevity Study in the Netherlands²¹, there was a 0% to ~45% prevalence rate depending on the criteria. Community-dwelling men in the Hertfordshire Cohort Study in the United Kingdom²² have a prevalence of <7% using the EWGSOP definition. In a report of individuals aged 65 and older in Belgium, prevalence rates of sarcopenia varied between 9% and 18%²³. In comparison to our study, the prevalence of sarcopenia for our comparable age group of individuals with stroke (those 60 years and older), is slightly higher, ranging between 15 and 23%. Furthermore, results from our study are in line with reports whose subjects have a mean age that is higher than ours of 63 years suggesting that individuals with stroke are at a higher risk for sarcopenia at a younger age. For example, in persons older than 80 years in the BELFRAIL study in Belgium, the 12.5% prevalence of sarcopenia by EWGSOP²⁴ is slightly lower than the prevalence in our stroke cohort despite the fact that our mean age is almost 20 years younger. Furthermore, in the iISIRENTE study, Italians aged 80 and older have a ~22% sarcopenia prevalence using the EWGSOP criteria¹⁹. In another study, women and men older than 80 years have prevalence rates of sarcopenia of ~32% and 17%, respectively²⁵.

Comparing these results to ours suggests that the consequences of stroke likely outweigh the aging associated increase in sarcopenia.

The sarcopenia definition from Cawthon et al.¹¹ allowed us to examine the prevalence of sarcopenia that discriminates weakness. With this method, we find the greatest variance in prevalence between men and women stroke survivors, e.g. about a two-fold greater prevalence in men than women. Despite not having measured grip strength to determine if this influences the results, we have previously shown that paretic leg strength is significantly lower than the non-paretic leg^{4,26}. Presumably grip strength is likewise reduced on the paretic side. Further, we have previously show that hemiparesis is associated with lower gait speed in subjects with stroke.²⁷ Thus, it is reasonable to suggest that any calculation of sarcopenia prevalence involving grip strength and gait speed would be influenced in stroke.

In addition to the physical inactivity and disuse observed after stroke, we can speculate some possible skeletal muscle mechanisms for the elevated prevalence of sarcopenia observed in this stroke population. In this larger sample, we confirm our previous findings³ of significantly lower lean tissue mass in the paretic compared to the non-paretic leg by DXA which also supports our findings of lower muscle area and muscle volume by computed tomography on the paretic leg.^{3,4} Increased infiltration of adipose tissue in skeletal muscle can occur during the atrophy process in stroke and is higher in paretic than non-paretic skeletal muscle^{3,4}. Not only could the increased intramuscular fat impact the amount of lean mass, higher intramuscular fat is associated with slow gait speed²⁸; thus, two contributors to sarcopenia definitions could be affected. Sarcopenia in stroke could also potentially be the result of increased skeletal muscle protein breakdown or a decrease in skeletal muscle protein synthesis. Our findings of 40% higher myostatin mRNA levels in the paretic vs. nonparetic vastus lateralis skeletal muscle suggest a protein synthesis/breakdown imbalance²⁶. Yet, myostatin levels are modifiable and our data indicates that resistive training results in a 49% reduction in myostatin in stroke paretic skeletal muscle²⁶. Inflammatory processes could also play a role in sarcopenia in stroke as tumor necrosis factor- α mRNA levels is increased on the paretic leg compared to controls²⁹.

Study Limitations

Limitations of this study include the lack of grip strength measures, stratification by comorbid conditions (type 2 diabetes), and investigator blinding to stroke and control groups, and a generally lower age range than some population studies. Another limitation is that the participants with stroke and the control cohorts were not matched on diabetes status which could confound the comparison between these two groups. A larger sample size may provide greater power for examining the relationships between sarcopenia and fitness. However, this is a chronically disabled and unique study population in terms of examining sarcopenia. The subjects studied represent a racially and sex mixed group, which is statistically similar to stroke survivors in the U.S.³⁰ Moreover, a broad range of body composition and gait speed represent strengths. It is also important to recognize that the control counterparts were sedentary as were the participants with stroke. Sedentary behavior is associated with sarcopenia in community-dwelling men and women 60–86 years of age³¹. The measurement of gait speed and its relationship with ALM/ht² indicates that functional

impairment is one factor related to sarcopenia in stroke, although deficits could be related to other central motor and sensory factors such as spasticity.

Conclusions

Future studies could be directed at interventions such as exercise to reduce sarcopenia in stroke survivors as well as include an in-depth examination of the mechanisms for the muscle atrophy and weakness observed in this disabled population. In conclusion, stroke survivors show an elevated prevalence of sarcopenia when considering age, sex, and race to non-stroke individuals, which are related to reduced fitness in this vulnerable population.

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Abbreviations

BMI	body mass index
DXA	dual-energy x-ray absorptiometry
ALM	appendicular lean mass
EWGSOP	European Working Group on Sarcopenia in Older Persons
IWG	International Working Group on Sarcopenia
VO₂peak	peak oxygen consumption

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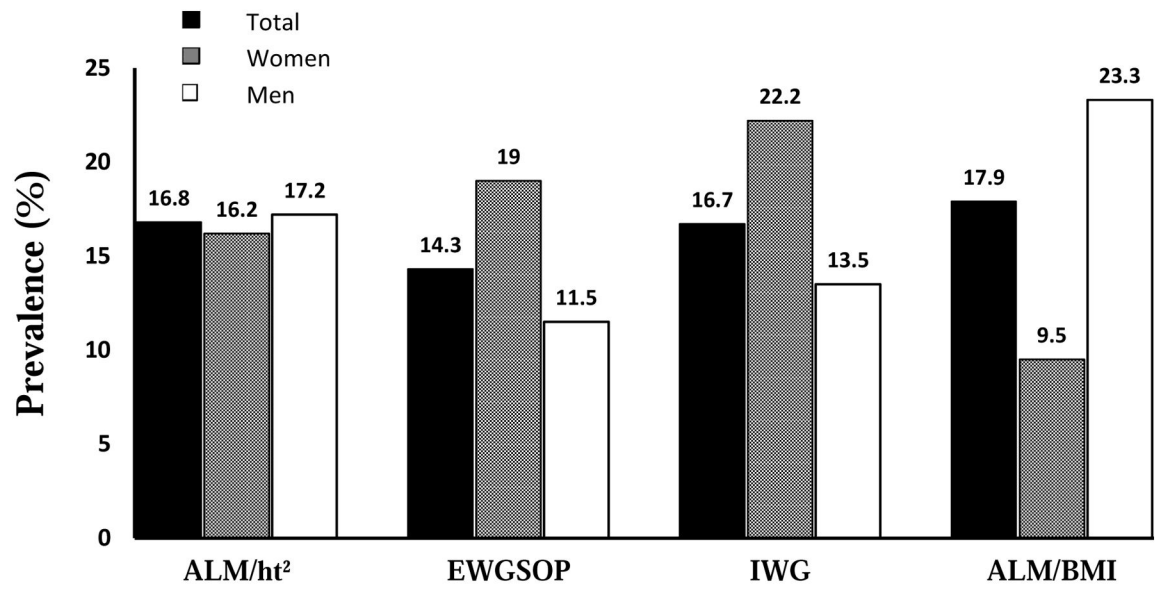


Figure 1. The prevalence of sarcopenia in the total group, in women, and in men with criteria of appendicular lean mass (ALM)/ht², the European Working Group on Sarcopenia in Older People (EWGSOP), the International Working Group (IWG), and ALM/body mass index.

Table 1

Body Composition and Physical Function (n=190)

	Women X±SEM	Men X±SEM	Total Group X±SEM	Total Group Range
Age (yr)	62 ± 1	63 ± 1	63 ± 1	(40 – 84)
Weight (kg)	77.4 ± 1.9	85.2 ± 1.4 [*]	82.2 ± 1.2	(40 – 134)
BMI (kg/m ²)	29.1 ± 0.7	28.3 ± 0.5	28.6 ± 0.4	(16 – 45)
VO ₂ peak (L/min) (n=173)	0.91 ± 0.05	1.40 ± 0.05	1.21 ± 0.04	(0.29 – 3.29)
VO ₂ peak (ml/kg/min) (n=173)	11.8 ± 0.6	16.2 ± 0.5	14.5 ± 0.4	(4.6 – 34.2)
Gait speed (m/s) (n=168)	0.56 ± 0.04	0.72 ± 0.04 [*]	0.66 ± 0.03	(0.07 – 1.51)
Body fat (%)	43.4 ± 0.7	30.5 ± 0.6 [†]	35.6 ± 0.6	(11 – 58)
Fat mass (kg)	34.1 ± 1.3	26.6 ± 0.9 [†]	29.5 ± 0.8	(7.67 – 64.2)
Lean mass (kg)	40.6 ± 0.8	55.3 ± 0.7 [†]	49.5 ± 0.8	(27.0 – 75.6)
Paretic leg lean mass (kg)	6.58 ± 0.18 ^a	8.96 ± 0.16 ^{†b}	8.05 ± 0.15 ^b	(3.9 – 13.1)
Non-Paretic leg lean mass (kg)	6.73 ± 0.17	9.32 ± 0.15 [†]	8.32 ± 0.15	(4.4 – 14.4)
Appendicular lean mass (kg)	17.8 ± 0.4	25.0 ± 0.4 [†]	22.2 ± 0.4	(11.2 – 36.9)
ALM/ht ² (kg/m ²)	6.69 ± 0.15	8.29 ± 0.12 [†]	7.66 ± 0.11	(4.60 – 11.89)

BMI= body mass index; ALM=appendicular lean mass

Men vs. Women,

^{*} P <0.005;[†] P <0.0001

Paretic vs. Non-Paretic,

^a P <0.05;^b P <0.0001

Table 2

Sarcopenia Status

	Women	Men	Total Group
Stroke			
ALM/ht ² (n=190)			
< 60 yrs (n=77)	15% (n=5)	16% (n=7)	16% (n=12)
60 yrs (n=113)	18% (n=7)	18% (n=13)	18% (n=20)
EWGSOP (n=168)			
< 60 yrs (n=72)	16% (n=5)	12% (n=5)	14% (n=10)
60 yrs (n=96)	21% (n=7)	11% (n=7)	15% (n=14)
IWG (n=168)			
< 60 yrs (n=72)	23% (n=7)	15% (n=6)	18% (n=13)
60 yrs (n=96)	21% (n=7)	13% (n=8)	16% (n=15)
ALM/BMI (n=190)			
< 60 yrs (n=77)	13% (n=4)	10% (n=4)	10% (n=8)
60 yrs (n=113)	9% (n=3)	33% (n=21)	23% (n=26)

ALM=appendicular lean mass; EWGSOP: European Working Group on Sarcopenia in Older People; IWG=International Working Group; BMI=body mass index

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