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Reduction in Skeletal Muscle Mass in Progressive Supranuclear Palsy in Comparison with Parkinson's Disease: A Preliminary Retrospective Longitudinal Study

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Abstract: Progressive supranuclear palsy (PSP) manifests with the loss of skeletal muscle mass, but the longitudinal changes have not been investigated. We studied changes in body composition, including in skeletal muscle mass, in patients with PSP twice, approximately 1 year or more apart, and we compared these measurements with those of patients with Parkinson's disease (PD). The total number of participants was 42: 10 men had PD, 13 men had PSP, 8 women had PD, and 11 women had PSP. Using a body composition analyzer, we measured such parameters as body mass index (BMI), skeletal muscle mass, basal metabolic rate (BMR), body fat percentage (BFP), and the ratio of extracellular water to total body water. We also calculated the skeletal muscle mass index (SMI). We measured the Barthel index to assess activities of daily living. The Barthel index was lower in patients with PSP than in those with PD at the first evaluation, and it worsened by the time of the second evaluation. In men with PSP, skeletal muscle mass was far more reduced than in those with PD, but no such changes were found among women with either disease. The SMI of men with PSP was correlated significantly with BMI, BMR, BFP, and the Barthel index. Skeletal muscle mass diminished faster in patients with PSP, especially in men, than in patients with PD, probably because of inactivity.

Keywords: progressive supranuclear palsy; Parkinson's disease; parkinsonism; skeletal muscle; body composition

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1. Introduction

Progressive supranuclear palsy (PSP) is a progressive neurodegenerative disorder with parkinsonism. PSP manifests with supranuclear gaze palsy, falls, postural instability, gait disturbance, and cognitive impairment [1,2]. The symptoms and signs resemble those of Parkinson's disease (PD), which is also a progressive neurodegenerative disorder that is mainly characterized by parkinsonism, such as resting tremors, rigidity, bradykinesia, and postural instability; therefore, distinguishing between PD and PSP is often difficult in the early stages. Because PSP is rare, studies of PSP are less common than those of PD, and definitive treatment and care have not been established [3,4]. The brain lesions involved in the two diseases are different [5–7]: in PD, the substantia nigra is mainly affected, whereas in PSP, the cerebellum, pallidum, subthalamic nucleus, and frontal lobe are involved, in addition to the substantia nigra [2]. The characteristics of gait also differ between PSP and PD [8]. Therefore, an understanding of the characteristics of PSP is necessary to guide treatment and rehabilitation.

To develop further treatment, including care and rehabilitation, for PSP, it is necessary to look not only at the symptoms peculiar to the disease but also at the complications. One of these is sarcopenia. Skeletal muscle mass (SMM), muscle strength, and physical function reduce with aging, and such losses are known as sarcopenia [9]. A reduction in SMM, muscle strength, and physical function that is related to disuse, disease, or undernutrition

is known as secondary sarcopenia [9], which occurs in patients with PD [10–13]. According to the Asian Working Group on Sarcopenia, the criteria for sarcopenia in older people include low SMM, which is defined by a skeletal muscle mass index (SMI) of <7.0 kg/ m^2 in men and <5.7 kg/m^2 in women [14], in addition to low muscle strength (hand grip) and low physical function (walking speed). A previous study showed that the proportion of subjects with low SMM, defined by the above criteria, was higher among patients with PSP than among age-matched healthy elderly persons [15]. Furthermore, it reported that the prevalence of sarcopenia in PSP was higher than in PD [16]. Another study reported that low muscle mass in relation to body height (i.e., SMI) was associated with mortality in older adults [17]. Thus, SMI may also be a key tool for assessing the treatment of patients with PSP.

Generally, PSP progresses faster than PD; thus, survival is shorter with PSP than with PD [18,19], and SMM is likely to decline more quickly in PSP than in PD. However, the longitudinal changes in SMM in patients with PSP have not been investigated.

To gain insights into the loss of SMM in PSP and to refine current treatments, we investigated changes in body composition, including the SMM of the upper extremities (U/E), lower extremities (L/E), and upper and lower extremities (U&L/E), twice (T0 was the first measurement, and T1 was the second, performed approximately 1 year or more later) in patients with PSP and PD.

2. Results

Table 1 (men) and Table 2 (women) list the participants' demographic and clinical characteristics by disease and the time of evaluation. The total number of participants was 42: 10 men had PD, 13 men had PSP, 8 women had PD, and 11 women had PSP. The interval from the first evaluation (T0) to the second (T1) did not differ significantly among these four groups.

Table 1. Male patients' demographic and clinical characteristics by disease and time of evaluation.

	Men			
	PD (*n* = 10)		**PSP (*n* = 13)**	
	T0	**T1**	**T0**	**T1**
Interval (years) [a]	1.9 ± 0.7		1.4 ± 0.5	
Age (years) [a]	71.7 ± 7.4	73.7 ± 6.9	73.8 ± 6.3	75.5 ± 6.1
Disease duration (years) [a]	5.1 ± 2.7	7.1 ± 2.8	3.9 ± 2.3	5.7 ± 2.6
Height (cm) [a]	165.8 ± 8.8	165.6 ± 7.0	165.7 ± 6.9	164.8 ± 6.7
Weight (kg) [a]	59.6 ± 8.9	58.6 ± 8.6	57.8 ± 8.3	49.2 ± 8.9 *,#
BMI (kg/m^2) [a]	21.6 ± 2.1	21.3 ± 2.1	21.1 ± 3.0	18.1 ± 3.4 *,#
BFP (%) [a]	20.0 ± 5.9	19.3 ± 4.5	19.7 ± 7.8	13.7 ± 7.4
BMR (kcal) [a]	1398.0 ± 149.3	1379.5 ± 143.8	1363.8 ± 114.0	1277.0 ± 118.5 *
ECW/TBW [a]	0.392 ± 0.009	0.394 ± 0.010	0.390 ± 0.010	0.404 ± 0.014 *
SMM U/E (kg) [a]	4.7 ± 1.0	4.6 ± 1.1	4.7 ± 0.6	4.1 ± 0.7
SMM L/E (kg) [a]	15.5 ± 3.0	15.2 ± 2.5	15.0 ± 2.3	13.4 ± 1.9 *,#
SMM U&L/E (kg) [a]	20.2 ± 3.9	19.7 ± 3.5	19.7 ± 2.9	17.5 ± 2.4 *
SMI U/E (kg/m^2) [a]	1.7 ± 0.2	1.6 ± 0.3	1.7 ± 0.2	1.5 ± 0.2
SMI L/E (kg/m^2) [a]	5.6 ± 0.7	5.5 ± 0.6	5.4 ± 0.7	4.9 ± 0.6
SMI U&L/E (kg/m^2) [a]	7.3 ± 0.9	7.1 ± 0.9	7.2 ± 0.8	6.4 ± 0.7 *
Modified Rankin scale score [b]	2.0 (2–3)	2.5 (2–3.3)	4.0 (3–4)	4.0 (4–5)
Barthel index [a]	82.0 ± 22.8	80.0 ± 21.9	53.8 ± 24.1 #	32.7 ± 32.1 *,#

PD, Parkinson's disease; PSP, progressive supranuclear palsy group; T0, first evaluation; T1, second evaluation; BMI, body mass index; BFP, body fat percentage; BMR, basal metabolic rate; ECW/TBW, ratio of extracellular water to total body water; SMM, skeletal muscle mass; SMI, skeletal muscle mass index; U/E, upper extremities; L/E, lower extremities; U&L/E, upper and lower extremities. [a] Results are reported as means ± standard deviations. [b] Results are reported as medians (interquartile ranges). * $p < 0.05$ in comparisons with T0 (two-way analysis of variance (ANOVA), post hoc Bonferroni test). # $p < 0.05$ in comparisons with PD (two-way ANOVA, post hoc Bonferroni test).

Table 2. Female patients' demographic and clinical characteristics by disease and time of evaluation.

	Women			
	PD (*n* = 8)		**PSP (*n* = 11)**	
	T0	**T1**	**T0**	**T1**
Interval (years) [a]	1.7 ± 1.0		1.6 ± 0.8	
Age (years) [a]	75.1 ± 12.3	77.1 ± 12.3	68.9 ± 5.2	70.4 ± 5.4
Disease duration (years) [a]	6.0 ± 3.6	8.0 ± 3.7	5.0 ± 2.9	6.5 ± 3.3
Height (cm) [a]	150.9 ± 7.8	150.1 ± 7.8	153.2 ± 3.6	153.1 ± 3.5
Weight (kg) [a]	45.6 ± 9.1	41.7 ± 5.1	53.3 ± 12.7	50.9 ± 13.5
BMI (kg/m^2) [a]	20.0 ± 3.6	18.7 ± 3.5	22.7 ± 5.2	21.8 ± 5.8
BFP (%) [a]	28.0 ± 10.5	24.5 ± 12.2	33.4 ± 12.4	30.4 ± 12.0
BMR (kcal) [a]	1068.4 ± 109.0	1042.5 ± 85.1	1111.8 ± 95.7	1088.7 ± 91.4
ECW/TBW [a]	0.391 ± 0.007	0.395 ± 0.008	0.393 ± 0.010	0.400 ± 0.013
SMM U/E (kg) [a]	2.6 ± 0.7	2.5 ± 0.5	3.2 ± 0.7	3.0 ± 0.7
SMM L/E (kg) [a]	10.1 ± 2.2	9.1 ± 2.1	10.3 ± 1.7	9.6 ± 1.7
SMM U&L/E (kg) [a]	12.6 ± 2.9	11.6 ± 2.5	13.4 ± 2.3	12.6 ± 2.3
SMI U/E (kg/m^2) [a]	1.1 ± 0.2	1.1 ± 0.2	1.3 ± 0.3	1.3 ± 0.3
SMI L/E (kg/m^2) [a]	4.4 ± 0.6	4.0 ± 0.5	4.4 ± 0.7	4.1 ± 0.7
SMI U&L/E (kg/m^2) [a]	5.4 ± 0.8	5.0 ± 0.7	5.7 ± 0.9	5.4 ± 1.0
Modified Rankin scale score [b]	2.0 (2–3)	2.0 (2–3)	3.0 (3–4) [#]	4.0 (3–5) [*,#]
Barthel index [a]	82.5 ± 6.3	72.5 ± 29.9	57.3 ± 27.3	40.6 ± 31.9

PD, Parkinson's disease; PSP, progressive supranuclear palsy group; T0, first evaluation; T1, second evaluation; BMI, body mass index; BFP, body fat percentage; BMR, basal metabolic rate; ECW/TBW, ratio of extracellular water to total body water; SMM, skeletal muscle mass; SMI, skeletal muscle mass index; U/E, upper extremities; L/E, lower extremities; U&L/E, upper and lower extremities. [a] Results are reported as means ± standard deviations. [b] Results are reported as medians (interquartile ranges). * $p < 0.05$ in comparisons with T0 (two-way analysis of variance (ANOVA), post hoc Bonferroni test). [#] $p < 0.05$ in comparisons with PD (two-way ANOVA, post hoc Bonferroni test).

A two-way repeated-measures ANOVA showed no significant interactions between time (T0 and T1) and disease (PD and PSP) with regard to age and disease duration. Among the men, the two-way repeated-measures ANOVA showed significant interactions between time and disease with regard to weight ($F_{1,21} = 11.320$, $p = 0.003$, $\eta p^2 = 0.350$), body mass index (BMI) ($F_{1,21} = 9.043$, $p = 0.007$, $\eta p^2 = 0.301$), basal metabolic rate (BMR) ($F_{1,21} = 5.983$, $p = 0.023$, $\eta p^2 = 0.222$), the ratio of extracellular water to total body water (ECW/TBW) ($F_{1,21} = 9.820$, $p = 0.005$, $\eta p^2 = 0.319$), SMM L/E ($F_{1,21} = 5.969$, $p = 0.023$, $\eta p^2 = 0.221$), SMM U&L/E ($F_{1,21} = 9.114$, $p = 0.007$, $\eta p^2 = 0.303$), SMI U&L/E ($F_{1,21} = 8.157$, $p = 0.009$, $\eta p^2 = 0.280$), and the Barthel index ($F_{1,21} = 5.235$, $p = 0.033$, $\eta p^2 = 0.200$). At T0, the afore-mentioned elements of body composition did not differ significantly between patients with PSP and those with PD. By contrast, according to a post hoc test, significant changes occurred between T0 and T1 in patients with PSP ($p < 0.05$) but not in those with PD. Furthermore, at T1, the two patient groups differed significantly in weight, BMI, and SMM L/E ($p < 0.05$). The Barthel index was significantly lower in patients with PSP than in those with PD at both T0 and T1, and in patients with PSP, the Barthel index was lower at T1 than at T0 ($p < 0.05$).

For the women, the two-way repeated-measures ANOVA only showed significant interactions between time and disease in the modified Rankin scale scores. Post hoc tests revealed significant changes between T0 and T1 in patients with PSP ($p < 0.05$).

Table 3 lists the rate of change in each outcome per year by sex and disease. The men with PSP showed significantly greater changes in weight, BMI, BMR, ECW/TBW, SMM L/E, SMI U&L/E, and the Barthel index ($p < 0.05$) than did the men with PD. In contrast, among the women, only the Barthel index differed significantly between patients with PD and those with PSP ($p < 0.05$).

Table 3. Rate of change in each outcome per year by sex and disease.

	Men		Women	
	PD (*n* = 10)	**PSP (*n* = 13)**	**PD (*n* = 8)**	**PSP (*n* = 11)**
Height	0.2 ± 1.2	−0.4 ± 1.1	−0.5 ± 1.2	−0.1 ± 0.3
Weight	−1.1 ± 4.4	−10.8 ± 7.1 *	−2.7 ± 5.7	−3.0 ± 12.9
BMI	−1.4 ± 5.2	−9.9 ± 8.3 *	−1.5 ± 6.7	−2.9 ± 12.7
BFP	1.2 ± 30.3	−20.8 ± 26.5	2.0 ± 22.3	−8.2 ± 17.3
BMR	−0.9 ± 5.2	−4.8 ± 3.5 *	−1.5 ± 2.1	−1.3 ± 2.9
ECW/TBW	0.1 ± 1.9	2.8 ± 1.9 *	0.5 ± 0.8	1.3 ± 1.3
SMM U/E	−1.5 ± 8.8	−8.8 ± 7.4	−0.6 ± 5.5	−4.2 ± 10.5
SMM L/E	−0.7 ± 9.2	−8.2 ± 7.5	−6.2 ± 4.3	−5.0 ± 5.8
SMM U&L/E	−0.8 ± 8.1	−8.5 ± 6.0 *	−5.1 ± 3.5	−4.8 ± 5.8
SMI U/E	−1.7 ± 9.4	−8.0 ± 7.4	0.5 ± 4.9	−4.0 ± 10.3
SMI L/E	−1.1 ± 7.8	−7.4 ± 7.9	−5.2 ± 3.6	−4.8 ± 5.7
SMI U&L/E	−1.2 ± 7.2	−7.7 ± 6.4 *	−4.7 ± 2.0	−4.6 ± 5.6
Modified Rankin scale	8.1 ± 17.2	14.9 ± 17.8	0.0 ± 0.0	13.3 ± 13.5
Barthel index	0.7 ± 24.8	−36.9 ± 41.2 *	−7.7 ± 13.8	−39.5 ± 31.6 *

PD, Parkinson's disease; PSP, progressive supranuclear palsy group; T0, first evaluation; T1, second evaluation; BMI, body mass index; BFP, body fat per-centage; BMR, basal metabolic rate; ECW/TBW, ratio of extracellular water to total body water; SMM, skeletal muscle mass; SMI, skeletal muscle mass index; U/E, upper extremities; L/E, lower extremities; U&L/E, upper and lower extremities. Results are reported as mean ± standard deviation of rate of change. * $p < 0.05$ in comparisons with PD (Mann–Whitney U test).

Table 4 lists the numbers of patients with low SMI U&L/E (defined as <7.0 kg/m^2 in men and as <5.7 kg/m^2 in women) and the changes in SMI U&L/E. Low SMI U&E/L was present in significantly more men with PSP than those with PD ($p < 0.05$). In fact, SMI U&L/E changed from normal to low at T1 in more men with PSP (no statistical processing). Conversely, SMI U&L/E did not differ significantly between the women with PD and the women with PSP, but approximately half the women in both groups had SMI U&L/E values less than the cut-off.

Table 4. Numbers of patients with low SMI U&E/L and change in SMI U&E/L.

			Men				Women			
			PD (*n* = 10)		**PSP (*n* = 13)**		**PD (*n* = 8)**		**PSP (*n* = 11)**	
Numbers and Rates of Low SMI U&L/E			**T0**	**T1**	**T0**	**T1**	**T0**	**T1**	**T0**	**T1**
			2 (20.0)	2 (20.0)	7 (53.8)	10 (76.9) *	4 (50.0)	7 (87.5)	5 (45.5)	6 (54.5)
The Change in Classification										
Normal	→	**Normal**	8 (80.0)		3 (23.1)		1 (12.5)		5 (45.5)	
Normal	→	**Low**	0 (0.0)		3 (23.1)		3 (37.5)		1 (9.1)	
Low	→	**Low**	2 (20.0)		7 (54.0)		4 (50.0)		5 (45.5)	
Low	→	**Normal**	0 (0.0)		0 (0.0)		0 (0.0)		0 (0.0)	

PD, Parkinson's disease; PSP, progressive supranuclear palsy group; SMI, skeletal muscle mass index; U&L/E, upper and lower extremities; T0, first evaluation; T1, second evaluation. Low SMI U&L/E was defined as <7.0 kg/m^2 in men and <5.7 kg/m^2 in women; normal SMI U&L/E was defined as >7.0 kg/m^2 in men and >5.7 kg/m^2 in women. * $p < 0.05$ in comparisons with PD (Fisher's exact test).

Table 5 lists the correlations between the rate of change in SMI U&L/E and the other parameters by sex and disease. SMI U&L/E was correlated significantly with (1) BMI in the men with PSP; (2) BMR in the men with PD, in the men with PSP, and in the women with PSP; (3) BFP in the men with PSP; (4) ECW/TBW in the men with PD; and (5) the Barthel index in the men with PD and in the men with PSP (all $p < 0.05$). Conversely, the changes in SMI U&L/E were not correlated significantly with age.

Table 5. Correlations between the rate of change in SMI U&L/E and other parameters by sex and disease.

	Sex	Disease		Age	BMI	BMR	BFP	ECW/TBW	Barthel Index
SMI U&L/E	Men	PD	*r*	−0.261	0.467	0.903 *	−0.370	0.697 *	0.650 *
			p	0.467	0.174	<0.001	0.293	0.025	0.042
		PSP	*r*	0.159	0.709 *	0.714 *	0.588 *	−0.247	0.720 *
			p	0.603	0.007	0.006	0.035	0.425	0.006
	Women	PD	*r*	0.429	0.143	−0.214	0.000	−0.095	0.439
			p	0.289	0.736	0.610	1.000	0.823	0.276
		PSP	*r*	0.218	0.482	0.709 *	−0.373	−0.209	0.436
			p	0.519	0.133	0.015	0.259	0.537	0.180

SMI, skeletal muscle mass index; U&L/E, upper and lower extremity; PD, Parkinson's disease; PSP, progressive supranuclear palsy group; BMI, body mass index; BMR, basal metabolic rate; BFP, body fat percentage; ECW/TBW, the ratio of extracellular water to total body water. * $p < 0.05$ (Spearman's rank correlation coefficient).

3. Discussion

This study is the first investigation of longitudinal changes in body composition in PSP in comparison with PD. Our results show that the changes in body composition over time were greater in patients with PSP than in those with PD, especially in men. Moreover, SMM in the men with PSP decreased to the state of sarcopenia.

The patients with PSP already exhibited severe dysfunction in mobility (according to the modified Rankin scale) or in activities of daily living (according to the Barthel index) at the time of the first evaluation, and these manifestations worsened by the time of the second evaluation. PSP progressed more quickly than PD in both the men and women. In the patients with PSP, daily activity decreased with disease progression, which accelerated muscle atrophy. Disorders of postural control and gait and frequent falls are more severe in PSP than in PD [1,2,8], and patients might become inactive to prevent falls. Additionally, PSP responds poorly to levodopa, unlike PD, which might also explain why the patients with PSP would have reduced their activities.

SMM was far more reduced in the men with PSP than in the men with PD, which was a novel finding in this study. Such a reduction was probably associated with disuse but not with aging, since the decrease in SMI was not correlated significantly with age in the men with PSP. A previous study showed that sarcopenia was associated with reduced daily activity and severe gait/midline symptoms in PSP [16]; therefore, the impact of inactivity would have also been significant in this study. The reduction in SMM with disuse paralleled the reductions in BMR and weight in this study. Furthermore, PSP was characterized by a vicious cycle of weakening muscles, an increase in inactivity, and a further decline in the ability to perform activities of daily living. Generally, muscle atrophy caused by disuse predominantly involves slow muscle fibers, whereas that caused by aging predominantly involves fast muscle fibers [20,21]. In this study, the decrease in slow muscle fibers may have been associated with inactivity; however, the muscle fiber types (fast and slow) involved in atrophy must be analyzed through a muscle biopsy to determine which type is involved in PSP.

By contrast, the women with PD and PSP in this study did not exhibit significant changes in body composition, including in SMM. The proportions of the women with low SMM were already similar among those with PD and PSP at T0; therefore, changes in body composition may not have differed between the groups. Among the men with PSP, however, the proportion of those with low SMM (53.8%) was fairly similar to the proportion of the women with PSP who had low SMM (45.5%) at the time of the first evaluation. An animal study of disuse by hindlimb suspension showed that female rats had less muscle deconditioning than did male rats [22]. The same mechanism might apply to humans: Even if disuse was augmented by a decline in activities of daily living, muscle atrophy in the women with PSP was not as drastic as in the men with PSP. Furthermore, a study in Japan showed that quadriceps mass decreased with age at a faster rate in men than in

women [23]. In our study, the rate of decline in SMM might have been greater in the men than in the women over a short time. The potential mechanisms of differences in muscle deconditioning by sex may be attributed to a variety of factors, including differences in hormones [24,25] and muscle composition [26]. However, our findings could not explain this sex difference; thus, further studies are needed.

This study had several limitations. First, the sample size was small and might have influenced the results. This study is a preliminary investigation; therefore, care must be taken in interpreting the results, and further research is needed. Multi-center research with larger patient populations is necessary. Second, we did not collect data about the activity of each participant between the two evaluations. Medical treatment, inpatient therapy, meals, and exercise have been reported to be related to SMM [9,27–31]. In future studies, researchers should assess these factors to clarify the mechanism of rapid SMM reduction. Third, the difference in the rate of disease progression and the decline in ADL function was not investigated. The present study suggests considering a wide range of ADL for comparing patient groups within 10 years of onset. It is important to compare them with those adjusted for the time of disease onset. However, it is quite difficult to discriminate between PSP and PD because both disorders do not manifest typical neurological signs in the early stages of the disease. It is necessary to carry out comparative verification by employing patients with similar activity levels within a few years after the onset in a multi-center study. Finally, body composition was measured using a multi-frequency bioelectrical impedance analysis (BIA), which could not be used on patients with pacemakers and other electronic devices. Therefore, we did not measure body composition in patients with pacemakers or after deep brain stimulation. Furthermore, it has been reported that BIA measurements are affected by body water status [32], and this effect was also included in the results of this study.

4. Materials and Methods

We measured body composition twice; the second evaluation (T1) was conducted approximately 1 year or more after the first (T0) to investigate the changes in SMM in patients with PSP and, for comparison, in patients with PD. This preliminary retrospective longitudinal study was conducted at the National Hospital Organization Higashinagoya National Hospital in Nagoya, Japan, between June 2017 and September 2021. The participants included patients with probable or possible PSP, diagnosed according to the 2017 Movement Disorder Society Criteria for the Clinical Diagnosis of PSP [1,33], and patients with PD, diagnosed according to the United Kingdom Brain Bank criteria [34]. The inclusion criteria were (1) a disease duration of $\leq$10 years at T0; (2) a modified Rankin scale score [35] of <5 at T0; and (3) available measurements of body composition at T0 and T1. The patients' informed consent to participate was obtained in the form of an opt-out option on a website (https://higashinagoya.hosp.go.jp/). The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Ethics Committee of the National Hospital Organization Higashinagoya National Hospital (approval number: 28-13, 30-11).

The age, sex, disease duration, height, weight, BMI, modified Rankin scale score, and Barthel index of all participants were documented from medical records. Body composition was measured with the InBody S10 body composition analyzer (InBody Japan, Inc., Tokyo, Japan), which enables multi-frequency BIA [15,36–38]. We recorded the BMR; BFP; ECW/TBW; and SMMs of U/E, L/E, and U&L/E. We calculated the SMI of U/E, L/E, and U&L/E according to the formula [appendicular SMM (in kilograms) divided by square of height (in meters)]. According to the Asian Working Group on Sarcopenia criteria for sarcopenia in older people [14], we defined low muscle mass as an SMI U&L/E of <7.0 kg/m^2 in men and <5.7 kg/m^2 in women, and we documented the numbers and proportions of men and women with each disease who had low SMI U&L/E. We did not survey the activities (e.g., medical treatment, inpatient therapy, meals, and exercise) of the participants between the two evaluations.

To conduct statistical analyses, we used SPSS software, version 26 (IBM, Inc., Armonk, NY, USA); missing values were excluded. We conducted a two-way repeated-measures analysis of variance (ANOVA), with time (T0 and T1) as the within-subject factor and disease (PD and PSP) as the between-subjects factor by sex. If interactions were significant, we conducted a post hoc analysis by using the Bonferroni correction for multiple comparisons. To analyze the proportions of male and female participants with low muscle mass, we used Fisher's exact method. To analyze the rate of change in SMM per year by sex from T0 to T1, we used the Mann–Whitney U test, and the calculation formula was as follows: $[(T1 - T0)/T1]/\text{evaluation interval (1 year)} \times 100$. We also used Spearman's rank correlation coefficient to calculate the correlations between the rate of change in SMI U&L/E and the other parameters. A p value of <0.05 was considered significant.

5. Conclusions

In conclusion, we compared changes in body composition in patients with PSP to those in patients with PD, focusing on SMM. We found that the reduction in SMM was faster in patients with PSP, especially in men, than in patients with PD.

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Abbreviations

PSP	progressive supranuclear palsy group
PD	Parkinson's disease
BIA	bioelectrical impedance analysis
BMI	body mass index
BFP	body fat percentage
BMR	basal metabolic rate
ECW/TBW	ratio of extracellular water to total body water
SMM	skeletal muscle mass
SMI	skeletal muscle mass index
U/E	upper extremities
L/E	lower extremities
U&L/E	upper and lower extremities

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