Research Article

DOI: 10.5152/NSN.2019.10548

Sarcopenia in Parkinson's disease patients

Özge Yılmaz Küsbeci[1], Beril Dönmez Çolakoğlu[2], İpek İnci[1], Elif Duran[3], Raif Çakmur[2]

[1]Clinic of Neurology, University of Health Sciences İzmir Bozyaka Training and Research Hospital, İzmir, Turkey
[2]Department of Neurology, Dokuz Eylül University School of Medicine, İzmir, Turkey
[3]Nurse, Eczacıbaşı Company, İstanbul, Turkey

Abstract

Objective: Sarcopenia is an involuntary loss of skeletal muscle mass and strength and/or function. The identification of sarcopenia in patients with Parkinson's disease (PD) may have prognostic and therapeutic effects. In our study, we aimed to evaluate sarcopenia in patients with PD by using bioimpedance analysis (BIA).

Methods: One hundred non-demented patients with PD, and 95 healthy subjects were included in the study. Fat-free mass, weight, bone mass, fat mass, basal metabolism rate (BMR), body surface area, and body mass index (BMI) of the PD and control groups were measured using BIA.

Results: There was no statistically significant difference between the ages of the men and women in the PD and control groups p=0.19 and p=0.29, respectively. There was a statistically significant difference in the average muscle mass of the men and women in the PD and control groups: 29.83±2.13 and 31.96±1.66 kg/m^2 (p<0.001), and 25.43±2.16 and 26.82±1.69 kg/m^2 (p=0.002), respectively. There were statistically significant differences in weight, fat mass, bone mass, BMR, and BMI between the men in PD and control groups: 76.47±11.71 and 82.64±11.08 kg (p=0.005); 18.32±6.13 and 21.55±7.84 kg (p=0.01); 2.91±0.37 and 3.04±0.27 kg (p=0.03); 1679±236 and 1775±179 calories (p=0.01); 27.40±3.58 and 28.88±3.52 kg/m^2 (p=0.02), respectively. There were statistically significant differences between weight and BMR in women in the PD and control groups: 70.30±13.72 and 77.54±17.22 kg (p=0.04) and 1374±194 and 1482±244 calories (p=0.03), respectively.

Conclusion: Our study indicates that sarcopenia is not rare in PD, and early diagnosis and treatment could decrease functional decline in patients with PD.

Keywords: Bioimpedance analysis, body mass index, Parkinson's disease, sarcopenia

INTRODUCTION

Parkinson's disease (PD) is the second most common neurodegenerative disorder of aging. The average age at onset of PD is about 60 years. The prevalence of PD will increase in the coming decades with the aging of the population. The cardinal signs of PD are generally considered as rest tremor, bradykinesia, rigidity, and loss of postural reflexes (1-3). In addition, people with PD often have musculoskeletal system problems, including loss of flexibility and altered posture (4, 5).

Sarcopenia has been defined as a geriatric syndrome characterized by the involuntary loss of skeletal muscle mass and strength and/or function. Chronic diseases and related factors are potentially important contributors to sarcopenia (e.g., bed rest, suboptimal diet, and drug treatments) (6). At around the 4th decade of life, skeletal muscle mass and strength begins declining, with up to 50% of mass being lost by the 8th decade of life. Muscle mass accounts for up to 60% of total body mass. Pathologic changes to this important tissue causes disabilities, dependency, falls, loss of function, fatigue, and mortality. Environmental causes such as declines in activity and nutritional intake, disease triggers, hormonal changes, and decreased protein synthesis have been defined as factors causing sarcopenia (5, 7-10).

This manuscript has been presented as an abstract at the 19th International Congress of the Movement Disorders Society, 14-18 June 2015, San Diego, California, USA.

You may cite this article as: Yılmaz Küsbeci Ö, Dönmez Çolakoğlu B, İnci İ, Duran E, Çakmur R. Sarcopenia in Parkinson's disease patients. Neurol Sci Neurophysiol 2019; 36(1): 28-32.

Corresponding Author: Özge Yılmaz Küsbeci **E-mail:** yilmazozge@hotmail.com **Submitted:** 13 August 2017 **Accepted:** 11 December 2018

Parkinson's disease and sarcopenia have a predilection for the elderly. The presence of sarcopenia may cause poor therapeutic control of PD and identification of sarcopenia in patients with PD may have therapeutic effects (1, 5). Various methods are available to evaluate muscle mass. For example, computed tomography (CT) or magnetic resonance imaging (MRI) and dual-energy X-ray absorptiometry (DEXA) have a high reliability and precision in determining lipid mass and fat mass (FM) and thus estimating lean body mass (LBM) (11-13). Nevertheless, these methods are not applicable on a bedside basis. Bioimpedance analysis (BIA) is inexpensive, easy to use, readily reproducible, and appropriate for both ambulatory and bedridden patients for estimating the volume of fat and lean body mass, and have been widely used in studies for the assessment of muscle mass for more than 10 years (14). Under standard conditions BIA results have been found to correlate well with MRI predictions (15, 16)

In this study, we aimed to estimate the prevalence of sarcopenia in patients with PD using BIA and to determine probable relations between the two disorders.

METHODS

One hundred right-handed non-demented patients with PD who were diagnosed in accordance with the United Kingdom Parkinson's Disease Society Brain Bank Clinical Diagnostic Criteria were included in the study (17). Ninety-five right-handed healthy subjects were evaluated as the control group. All patients were recruited from the outpatient clinic for movement disorders of University of Health Sciences İzmir Bozyaka Training and Research Hospital and Dokuz Eylül University School of Medicine Department of Neurology. We evaluated patients with PD who had been diagnosed at least 1 year prior to the study. All patients were examined at least 3 times per year by movement disorder specialists. Only optimally controlled patients were included in the study. This study was approved by the local Ethics Committee of University of Health Sciences İzmir Bozyaka Training and Research Hospital and informed consent was obtained from all participants. Subjects both in the PD and control groups were excluded if they aged 80 years or over, had experienced an acute infection within the previous month, had malignancy, chronic diseases known to affect muscle mass, had undergone any surgery in the last 6 months, or had stroke during the previous 6 months. Fat-free mass (FFM), weight, bone mass, fat mass, basal metabolism rate (BMR), body mass index (BMI) of PD and control groups were measured using a bioimpedance analysis device (body composition analyzer SC-330 Tanita Corp., Tokyo, Japan). Body surface area (BSA) was calculated using the DuBois formula ($BSA = kg^{0.425} \times cm^{0.725} \times 0.007184$). Fat-free mass was divided by BSA to determine muscle mass (18). Body mass index was obtained by dividing weight by height squared. We did not measure the patient and control group's walking speed and muscle strength because of the rigidity and bradykinesia in the PD group, the results would not be objective.

Statistical Analysis

All analyses were performed using IBM Statistical Package for the Social Sciences Statistics version 20.0 (SPSS IBM Corp.; Armonk, NY, USA). Categorical variables are expressed as frequency and percentages. Numeric variables are expressed as mean ± standard deviation. Two-group comparison tests (Student's t-test and Mann-Whitney) were used to analyze the types of variables. Chi-square analysis and two-group comparison tests were used as appropriate for the variable types. P values ≤0.05 were considered significantly significant.

RESULTS

The mean ages of the men and women in the PD and control groups were 67.37±8.47 and 65.27±8.72 years and 63.35±9.01 and 61.63±5.22 years (p=0.19 and p=0.29, respectively). There was a statistically significant difference between the average muscle mass (FFM/BSA= kg/m^2) of the men and women in the PD and control groups (29.83±2.13 kg/m^2 and 31.96±1.66 kg/m^2, and (25.43±2.16 kg/m^2 and 26.82±1.69 kg/m^2 (p<0.001 and p=0.002, respectively). There were statistically significant differences in weight,

Table 1. Demographic data of the PD and control groups

	Men			Women		
	Parkinson's disease (n=61)	Control (n=54)	p	Parkinson's disease (n=39)	Control (n=41)	p
Age	67.37±8.47	65.27±8.72	0.19	63.35±9.01	61.63±5.22	0.29
Weight	76.47±11.71	82.64±11.08	0.005	70.30±13.72	77.54±17.22	0.04
Avarage Muscle mass						
FFM/BSA (kg/m^2)	29.83±2.13	31.96±1.66	<0.001	25.43±2.16	26.82±1.69	0.002
Fat mass (kg)	18.32±6.13	21.55±7.84	0.01	26.50±13.22	28.25±11.88	0.536
Bone mass (kg)	2.91±0.37	3.04±0.27	0.03	2.33±0.33	2.47±0.38	0.07
BMR	1679±236	1775±179	0.01	1374±194	1482±244	0.03
BMI	27.40±3.58	28.88±3.52	0.02	28.89±5.29	29.85±6.54	0.47

FFM: fat-free mass; BSA: body surface area; BMI: Body mass index; BMR: basal metabolism rate; PD: Parkinson's disease

fat mass, bone mass, BMR, and BMI between men in PD and control groups: 76.47±11.71 and 82.64±11.08 kg (p=0.005); 18.32±6.13 and 21.55±7.84 (p=0.01); 2.91±0.37 and 3.04±0.27 (p=0.03); 1679±236 and 1775±179 (p=0.01); and 27.40±3.58 and 28.88±3.52 kg/m^2 (p=0.02), respectively. There were statistically significant differences between the weight and BMR of women in the PD and control groups: 70.30±13.72 and 77.54±17.22 kg, and 1374±194 and 1482±244 (p=0.04 and p=0.03, respectively). There were no statistically significant differences between the prevalences of sarcopenia in patients with early and advanced-stage PD (n=13.3, 8.2% and n=29, 43.9%) (p=0.584), respectively. Additionally, we found no significant difference between the mean UPDRS-III scores and sarcopenia in patients with PD: 24.54±8.48 and 23.01±8.38 (p=0.372). The demographic data and clinical characteristics of the study participants are provided in Table 1.

DISCUSSION

Muscle mass declines at a rate of about 1% per year after 30 years of age (19). The rate of muscle mass decline is approximately 8% per decade from the age of 50 years until the age of 70 years (20). After 70 years, muscle mass loss reaches rates of 15% per decade (21). Sarcopenia is an important predictor of disability for the future. Epidemiologic studies have demonstrated associations between sarcopenia and future functional decline, physical disability, increased morbidity and mortality (9, 21-23). Sarcopenia has multifactorial causes and the major cause appears to be related to a decrease of physical activity with advancing age (24). Poor protein and calorie intake can aggravate sarcopenia (25).

The prevalence of sarcopenia changes depending on the population sample and definition used (26, 27). The prevalences of sarcopenia in nursing home residents were found as 85%, 68%, and 38.9% in 3 Turkish studies (8, 28, 29). There are few studies evaluating sarcopenia and prevalence of sarcopenia in patients with PD (30-32). In one study, researchers found a significant association of UPDRS-III scores with early-stage sarcopenia and the authors concluded that this significance might indicate a common and early pathway in both diseases and supports the existence of sarcopenia (30). In another study, researchers found sarcopenia as a prevalent condition among older adults with PD and severe sarcopenia was associated with disease severity. Additionally, they concluded that sarcopenia might present the common downstream pathway that leads from non-motor and motor PD symptoms to the progressive frailty and disability, and offered that further studies should explore the potential close link between PD and sarcopenia. Slow movements and weakness are common features of PD and they are responsible for the functional decline and loss of independence experienced by patients with PD. However, as stated previously, the criteria that were tested in the general population may not be appropriate for patients with PD (31).

The mean age of our subjects in both groups was over 60 years. Therefore, in our study, both groups had the risk of sarcopenia because of their ages, and we found a statistically significant difference between the FFM of men and women in both the PD and control groups (p<0.001 and p=0.002, respectively). This result showed that PD could increase the risk of sarcopenia. In our study, we accepted two standard deviations below the mean fat- free muscle mass of the healthy control group as a cut-off value for sarcopenia. According to these cut-off values, the prevalences of sarcopenia among the men and women in the PD and control groups were 34.4% (n=21) and 16% (n=9), and 25.6% (n=10) and 7.3% (n=3) (p=0.001 and p=0.009, respectively). When we compared patients with early and advanced-stage PD, the prevalences of sarcopenia were 38.2% (n=13) and 43.9% (n=29) (p=0.584), respectively. Additionally, we found no significant association between the UPDRS-III scores and sarcopenia inpatients with PD (p=0.372). Our results show that sarcopenia can be seen at early stages of PD and there may be a common and early pathway in both diseases.

Cellular and tissue changes, and environmental and behavioral factors contribute to the loss of muscle mass. The factors could be given in 3 groups as potentially treatable (e.g. social factors and isolation), medical (e.g. gastrointestinal diseases such as vomiting, diarrhea, swallowing problems, dysphagia, poor pain management or constipation) and more difficult to treat (e.g. loss of taste and smell, diet restrictions). Therefore, the high prevalence of sarcopenia in PD could be a result of a potentially shared pathophysiology (30-33).

The methods used to measure muscle mass are DEXA, BIA, CT, and MRI. Bioimpedance analysis has gained some popularity because of its portability. In our study, we preferred to use BIA because it was non-radiating, relatively cheap, portable, and an inexpensive method to measure body composition in our outpatient clinic (28).

In our study, we did not evaluate walking speed and muscle strength of the PD and control groups. All of the cardinal clinical manifestations of PD could affect the walking speed and muscle strength of patients (1). Walking speed in particular could be affected because of bradykinesia.

The relatively small sample size in our study may limit the generalizability of our findings. Also, due to the dehydration in older patients, BIA may cause fat tissue to be underestimated resulting in artificially high FFM values. A third limitation could be the muscle mass estimation method. The gold standard for that approach is DEXA. Nevertheless, BIA appears to be an adequate alternative.

In conclusion, our study indicated that sarcopenia is not rare among patients with PD. Therefore, prevention, early diagnosis, and intervention for sarcopenia could decrease functional decline in patients with PD.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of University of Health Sciences İzmir Bozyaka Training and Research Hospital.

Informed Consent: Written informed consent was obtained from patients participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – Ö.Y.K., İ.İ., B.D.Ç.; Design – Ö.Y.K., İ.İ., B.D.Ç., R.Ç.; Supervision – Ö.Y.K., R.Ç., B.D.Ç..; Resources – Ö.Y.K., E.D., İ.İ., B.D.Ç.; Materials – Ö.Y.K., E.D., İ.İ., R.Ç.; Data Collection and/or Processing – Ö.Y.K., İ.İ., E.D., B.D.Ç.; Analysis and/or Interpretation – Ö.Y.K., B.D.Ç.; Literature Search – Ö.Y.K., B.D.Ç.; Writing Manuscript – Ö.Y.K.; Critical Review – Ö.Y.K., B.D.Ç.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Nutt JG, Wooten GF. Clinical practice. Diagnosis and initial management of Parkinson's disease. N Engl J Med 2005; 353: 1021-1027. [CrossRef]
2. De Lau LM, Breteler MM. Epidemiology of Parkinson's disease. Lancet Neurol 2006; 5: 525-535. [CrossRef]
3. Lilienfeld DE, Perl DP. Projected neurodegenerative disease mortality in the United States, 1990-2040. Neuroepidemiology 1993; 12: 219-228. [CrossRef]
4. Schenkman M. Physical therapy intervention for the ambulatory patient. In: Turnbull G, ed. Physical Therapy Management of Parkinson's Disease. New York: Churchill Livingstone; 1992, pp.137-192.
5. Schenkman M, Cutson TM, Kuchibhatla M, Chandler J, Pieper C. Reliability of impairment and physical performance measures for persons with Parkinson's disease. Phys Ther 1997; 77: 19-27.
6. Cruz-Jentoft AJ, Landi F, Schneider SM, et al. Prevalence of and interventions for sarcopenia in ageing adults: a systematic review. Report of the International Sarcopenia Initiative (EWGSOP and IWGS). Age Ageing 2014; 43: 748-759. [CrossRef]
7. Goodpaster BH, Park SW, Harris TB, et al. The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. J Gerontol A Biol Sci Med Sci 2006; 61: 1059-1064. [CrossRef]
8. Tasar PT, Sahin S, Karaman E, et al. Prevalence and risk factors of sarcopenia in elderly nursing home residents. Eur Geriatr Med 2015; 6: 214-219. [CrossRef]
9. Landi F, Liperoti R, Russo A, et al. Sarcopenia as a risk factor for falls in elderly individuals: results from the ilSIRENTE study. Clin Nutr 2012; 31: 652-658. [CrossRef]
10. Walston JD. Sarcopenia in older adults. Curr Opin Rheumatol 2012; 24: 623-627. [CrossRef]
11. Mazess RB, Barden HS, Bisek JP, Hanson J. Dual-energy x-ray absorptiometry for total-body and regional bone-mineral and soft-tissue composition. Am J Clin Nutr 1990; 51: 1106-1112. [CrossRef]
12. Dufour AB, Hannan MT, Murabito JM, Kiel DP, McLean RR. Sarcopenia definitions considering body size and fat mass are associated with mobility limitations: the Framingham Study. J Gerontol A Biol Sci Med Sci 2013; 68: 168-174. [CrossRef]
13. Ellis KJ. Human body composition: in vivo methods. Physiol Rev 2000; 80: 649-680. [CrossRef]
14. Kyle UG, Bosaeus I, De Lorenzo AD, et al. Bioelectrical impedance analysis--part I: review of principles and methods. Clin Nutr 2004; 23: 1226-1243. [CrossRef]
15. Bioelectrical impedance analysis in body composition measurement: National Institutes of Health Technology Assessment Conference Statement. Am J Clin Nutr 1996; 64: 524S-532S. [CrossRef]
16. Janssen I, Heymsfield SB, Baumgartner RN, Ross R. Estimation of skeletal muscle mass by bioelectrical impedance analysis. J Appl Physiol (1985) 2000; 89: 465-471. [CrossRef]
17. Caslake R, Moore JN, Gordon JC, Harris CE, Counsell C. Changes in diagnosis with follow-up in an incident cohort of patients with parkinsonism. J Neurol Neurosurg Psychiatry 2008; 79: 1202-1207. [CrossRef]
18. Yu R, Wong M, Leung J, Lee J, Auyeung TW, Woo J. Incidence, reversibility, risk factors and the protective effect of high body mass index against sarcopenia in community-dwelling older Chinese adults. Geriatr Gerontol Int 2014; 14(Suppl 1): 15-28. [CrossRef]
19. Michel JP. Sarcopenia: there is a need for some steps forward. J Am Med Dir Assoc 2014; 15: 379-380. [CrossRef]
20. Grimby G, Saltin B. The ageing muscle. Clin Physiol 1983; 3: 209-218. [CrossRef]
21. Yu S, Umapathysivam K, Visvanathan R. Sarcopenia in older people. Int J Evid Based Healthc 2014; 12: 227-243. [CrossRef]
22. Landi F, Liperoti R, Fusco D, et al. Sarcopenia and mortality among older nursing home residents. J Am Med Dir Assoc 2012; 13: 121-126. [CrossRef]
23. Janssen I, Heymsfield SB, Ross R. Low relative skeletal muscle mass (sarcopenia) in older persons is associated with functional impairment and physical disability. J Am Geriatr Soc 2002; 50: 889-896. [CrossRef]
24. Morley JE. Sarcopenia in the elderly. Fam Pract 2012; 29(Suppl 1): 44-48. [CrossRef]
25. Rolland Y, Onder G, Morley JE, Gillette-Guyonet S, Abellan van Kan G, Vellas B. Current and future pharmacologic treatment of sarcopenia. Clin Geriatr Med 2011; 27: 423-447. [CrossRef]
26. Iannuzzi-Sucich M, Prestwood KM, Kenny AM. Prevalence of sarcopenia and predictors of skeletal muscle mass in healthy, older men and women. J Gerontol A Biol Sci Med Sci 2002; 57: M772-M777. [CrossRef]
27. Di Monaco M, Vallero F, Di Monaco R, Tappero R. Prevalence of sarcopenia and its association with osteoporosis in 313 older women following a hip fracture. Arch Gerontol Geriatr 2011; 52: 71-74. [CrossRef]
28. Halil M, Ulger Z, Varlı M, et al. Sarcopenia assessment project in the nursing homes in Turkey. Eur J Clin Nutr 2014; 68: 690-694. [CrossRef]
29. Bahat G, Saka B, Tufan F, et al. Prevalence of sarcopenia and its association with functional and nutritional status among male

residents in a nursing home in Turkey. Aging Male 2010; 13: 211-214. [CrossRef]
30. Drey M, Hasmann SE, Krenovsky JP, et al. Associations between early markers of Parkinson's disease and sarcopenia. Front Aging Neurosci 2017; 9: 53. [CrossRef]
31. Vetrano DL, Pisciotta MS, Laudisio A, et al. Sarcopenia in Parkinson disease: comparison of different criteria and association with disease severity. J Am Med Dir Assoc 2018; 19: 523-527. [CrossRef]
32. Lee JJ, Oh JS, Ham JH, et al. Association of body mass index and the depletion of nigrostriatal dopamine in Parkinson's disease. Neurobiol Aging 2016; 38: 197-204. [CrossRef]
33. Boirie Y. Physiopathological mechanism of sarcopenia. J Nutr Health Aging 2009; 13: 717-723. [CrossRef]