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Frequency of sarcopenia in Turkish women with systemic lupus erythematosus

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ABSTRACT

Objective This study aimed to evaluate the prevalence of sarcopenia and its clinical significance in Turkish women with SLE, exploring the association between muscle mass, muscle strength and SLE disease activity.

Methods A cross-sectional study was conducted at Gazi University Hospital's Department of Rheumatology from January to December 2020. It involved 82 patients with SLE, diagnosed according to the 2019 American College of Rheumatology/European Alliance of Associations for Rheumatology criteria, and 69 healthy controls. Sarcopenia was assessed using hand grip dynamometry (hand grip strength (HGS)) and bioelectrical impedance analysis for muscle mass, with sarcopenia defined according to the 2018 European Working Group on Sarcopenia in Older People criteria and specific cut-offs for the Turkish population. The main outcomes measured were the presence of sarcopenia and probable sarcopenia, HGS values, skeletal muscle mass index and SLE Disease Activity Index 2000 (SLEDAI-2K).

Results Among the patients with SLE, 51.2% met the criteria for probable sarcopenia and 12.9% were diagnosed with sarcopenia. The mean HGS was significantly lower in the SLE group (21.7±4.9 kg) compared with controls, indicating reduced muscle strength. The prevalence of anti-double-stranded DNA (anti-dsDNA) antibodies was 82.9%. Multivariate regression analysis identified height and levels of anti-dsDNA antibodies as independent predictors for developing probable sarcopenia. No significant association was found between clinical parameters, including SLEDAI-2K scores, and sarcopenia status.

Conclusions Sarcopenia is prevalent among Turkish women with SLE, with a significant proportion showing reduced muscle strength. The study found no direct association between sarcopenia and SLE disease activity or clinical parameters. These findings underscore the importance of including muscle strength assessments in the routine clinical evaluation of patients with SLE to potentially improve management and quality of life.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Sarcopenia impacts individuals with autoimmune diseases, including SLE, due to factors like inflammation, autoimmunity and corticosteroid use.

WHAT THIS STUDY ADDS

⇒ Reveals a high prevalence of sarcopenia (51.2%) in Turkish women with SLE, using hand grip strength and bioelectrical impedance analysis for assessment.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Highlights the need for routine sarcopenia assessments in SLE care and suggests potential benefits of exercise and nutritional strategies, advocating for a multidisciplinary approach to improve patient outcomes.

INTRODUCTION

Sarcopenia is a condition characterised by decreased muscle strength and muscle mass, which can affect physical function. It often develops as a consequence of age-related decline (primary sarcopenia) and has a major impact on physical, social and emotional well-being. In addition, patients with some other chronic conditions may suffer from sarcopenia, independent of ageing (secondary sarcopenia).[1] In particular, prior research underscores the widespread occurrence of sarcopenia across various autoimmune disorders, encompassing rheumatoid arthritis, psoriatic arthritis, spondyloarthritis, systemic sclerosis, inflammatory bowel disease and autoimmune diabetes.[1 2]

SLE is characterised by a dysregulated immune system that targets healthy cells and tissues, leading to chronic inflammation and damage.[3] In SLE, the convergence of chronic inflammation, autoimmunity, physical inactivity and the effects of long-term corticosteroid therapy could potentially lead to muscle wasting and sarcopenia. Chronic inflammation drives muscle wasting by increasing levels of inflammatory cytokines, such as tumour necrosis factor-alpha, interleukin (IL)-1 and

IL-6, which not only promote protein degradation but also impair the function of satellite cells essential for muscle repair and regeneration. Moreover, the autoimmunity in SLE might also directly target muscle tissue, further impairing muscle function and contributing to sarcopenia. In addition, SLE-related fatigue and joint pain might lead to a significant reduction in physical activity, and accelerate muscle atrophy and progression of sarcopenia. Furthermore, corticosteroids, commonly used to treat SLE, exacerbate muscle wasting by promoting protein catabolism, inhibiting protein synthesis and inducing adverse metabolic effects, such as insulin resistance. Taken together, all these mechanisms might act synergistically and result in significant sarcopenia in patients with lupus.

However, the inadequate comprehension and acknowledgement of sarcopenia in autoimmune diseases, such as SLE, pose a challenge to efficient patient management. This study aims to address these issues and promote research in diagnostic and management strategies for sarcopenia associated with autoimmune diseases. The objective of this study is to evaluate muscle mass and strength in Turkish patients with lupus using different assessment methods, including bioelectrical impedance analysis (BIA) and hand grip dynamometry. Identifying and addressing sarcopenia in patients with SLE are needed for improving their quality of life and overall well-being.

MATERIALS AND METHODS

Patients and study design

In this cross-sectional investigation, we assessed 82 patients with SLE and 69 healthy individuals, as controls. The study was conducted at Gazi University Hospital Department of Rheumatology, throughout 2020, from January to December. Enrolment criteria for patients with SLE included adherence to the 2019 criteria set forth by the American College of Rheumatology and the European Alliance of Associations for Rheumatology.[4] Healthy control subjects were selected based on the absence of health conditions. Exclusion criteria were pregnancy, a history of malignancy, cognitive dysfunction, ambulatory difficulties, current arthritis, deformity of the dominant hand joint, other chronic inflammatory diseases (such as tuberculosis or inflammatory bowel disease), myositis or any rheumatological disease other than SLE, or mechanical pain, and patients who had not undergone hand or forearm surgery in the previous 6 months; exclusion criteria also included ongoing treatment with prednisone at doses greater than 10 mg/day (or equivalent dose of other corticosteroids). Comprehensive clinical and demographic information was meticulously documented for all participants. It was not appropriate or possible to involve patients or the public in the design, or conduct, or reporting, or dissemination plans of our research.

Measurements and evaluation of disease activity

The European Working Group on Sarcopenia in Older People criteria of 2018 (EWGSOP2) were used for definition. Here, three items are assessed: muscle strength, muscle mass and muscle function. Sarcopenia is determined in three stages: pre-sarcopenia, sarcopenia and severe sarcopenia, with only a reduction in muscle strength referring to probable sarcopenia. Sarcopenia is defined as a decline in muscle mass that accompanies low muscle strength. If muscle mass, strength and physical function all decrease, the condition is considered severe sarcopenia.[5] Hand grip dynamometry measurements were obtained for the entire cohort in this study; however, physical function tests and BIA were not available for all participants due to the COVID-19 pandemic. The tests were carried out by three geriatricians familiar with muscle assessmemts (OD, BC, FYB). Muscle strength was evaluated by hand grip strength (HGS) test on the dominant hand, using a TAKEI 5401 digital hand dynamometer (manufactured by Takei Scientific Instruments Co, Niigata, Japan). The protocol for the test required the participant to be seated in a relaxed posture, with the forearm flexed at a 90° angle and supported by a pillow, ensuring the hand was positioned neutrally. Subjects were instructed to exert maximum force on the dynamometer grip for three attempts. The highest value recorded from these attempts was considered as the final measurement. Sarcopenia cut-off values established by Bahat *et al* for the young population in Turkey were used to define sarcopenia.[6 7] Based on these cut-offs, an HGS of less than 22 kg for women is identified as a marker of reduced muscle strength.[6]

Height and weight were measured using a stadiometer, which is calibrated regularly. Participants wore light clothing and no shoes during the measurements. Weight was recorded to the nearest 0.1 kg and height to the nearest 0.1 cm. The body mass index (BMI) was then calculated using the following formula: BMI=weight in kilograms divided by the square of height in metres (kg/m^2). Body composition was measured using BIA, using a Tanita BC 532 body analysis monitor. According to the European Working Group on Sarcopenia in Older People (EWGSOP), BIA is suggested as a valid, reliable and practical method for measuring muscle mass in routine practice. The BIA was performed in the same way for all participants. Measurements were taken in a standing position, in the same room, early in the morning, after at least 6 hours of fasting and before any significant physical activity.

The adjusted skeletal muscle mass index, referred to as SMMI (BMI), was calculated by dividing skeletal muscle mass (in kilograms) by BMI, measured in kg/m^2. This formula gives a BMI-based SMMI. A study by Bahat *et al* established specific cut-off values for SMMI (BMI) in the Turkish population. According to their findings, SMMI (BMI) values below 0.823 (kg/(kg/m^2)) in women indicate low muscle mass.[7]

The SLE Disease Activity Index 2000 (SLEDAI-2K) is a comprehensive tool used to evaluate the activity of SLE. This index encompasses 24 items, including general symptoms like fever and fatigue, lymph node swelling, joint pain, skin rashes, pulmonary and renal complications, muscle weakness, peripheral and central nervous system complications, blood disorders and specific laboratory markers for SLE. Each item is graded to quantitatively assess the severity and extent of the disease, playing a crucial role in the management of treatment and monitoring disease progression. At each outpatient visit, disease activity over the previous 30 days, a Physician Global Assessment (PGA) (0–3 points, on a 10 cm scale) and SLEDAI-2K (0–105) were obtained by the same senior rheumatologist (RB) with extensive SLE management experience.

Statistical analysis

Statistical analyses were conducted using SPSS software V.22. The normality assumption was assessed using the Shapiro-Wilk test. Continuous variables with a normal distribution are reported as the mean±SD. For those not following a normal distribution, the median (25th–75th percentile) is used. Categorical variables are presented as counts (percentages). To determine the significance of differences between the two groups, either the Student's t-test or the Mann-Whitney U test is employed. Statistical analyses include the use of either the Pearson $\chi 2$ test or the Fisher's exact test, based on the data characteristics. The univariate analyses to identify variables associated with probable sarcopenia in patients with SLE were investigated using $\chi 2$, Fisher's exact, Student's t and Mann-Whitney U tests, where appropriate. For the multivariate analysis, the possible factors identified with univariate analyses were further entered into the logistic regression analysis to determine independent predictors of probable sarcopenia. Hosmer-Lemeshow goodness-of-fit statistics was used to assess model fit. A 5% type I error level was used to infer statistical significance.

RESULTS

The study included 82 women with SLE and 54 age-matched healthy women; mean±SD ages were 39.2±10.8 and 40.2±14.3 years, respectively. HGS was statistically significantly lower in the SLE cohort compared with the control group (p<0.001). Although the SMMI and the SMMI (BMI) were lower in the SLE group, these differences did not reach statistical significance. By definition, the SLE group had a significantly higher rate of probable sarcopenia (51.2%) compared with the control group (25.9%) (p=0.003). The frequency of sarcopenia was also higher in the SLE group (12.9%) than the control group (5.8%); however, this difference was not statistically significant (table 1).

In the studied cohort, 68 patients (82.9%) tested positive for anti-double-stranded DNA (anti-dsDNA) antibodies. Ribonucleoprotein (RNP) antibodies were confirmed in 23 patients, who represent 28% of the cohort.

The clinical manifestations observed in the patients with SLE, as well as autoantibody profiles, are given in table 2. Treatment regimens included hydroxychloroquine administration in 69 patients (84.1%) and the use of low-dose corticosteroids in 26 patients (31.7%). Furthermore, 37 patients (45.1%) with evidence of organ involvement were undergoing treatment with immunosuppressive agents, such as azathioprine, mycophenolate mofetil, ciclosporin or rituximab.

The mean age did not significantly differ in those with and without probable sarcopenia, in patients with SLE (p=0.18) (table 3). Patients with probable sarcopenia had a significantly lower mean HGS score compared with those patients with lupus with normal muscle assessments (17.7±2.7 vs 25.8±2.8 kg, p<0.001). However, there were no statistically significant differences between the two groups in terms of BMI, SMMI (BMI), PGA and SLEDAI-2K scores (p>0.05). Regarding immunological parameters, mean anti-dsDNA titres were lower in the pre-sarcopenic group (p=0.01). However, when the analyses were done based on the presence or absence of autoantibodies, none of the autoantibodies revealed a statistically significant relationship, except for anti-RNP (p=0.01). Additionally, no significant link between the administration of low-dose corticosteroids and the incidence of probable sarcopenia was observed (p>0.05).

Multivariate regression analysis was used to investigate the factors that potentially might influence HGS test scores. The analysis revealed a strong correlation between HGS scores and the SMMI, with a correlation coefficient (r) of 0.49 and a p value of less than 0.001. However, SMMI was excluded from the regression analysis due to concerns about the adequacy of the model. Status and levels of anti-dsDNA antibodies were identified as independent predictors for the likelihood of developing probable sarcopenia, according to further

Table 1 Comparison of lupus and control group according to the presence of sarcopenia and probable sarcopenia

	Lupus (n=82)	Control group (n=54)	P value
Probable sarcopenia presence	42 (51.2)	14 (25.9)	0.003
Probable sarcopenia absence	40 (48.8)	40 (74.1)	
Sarcopenia presence	4 (12.9)	3 (5.8)	0.25
Sarcopenia absence	27 (87.1)	49 (94.2)	

Table 2 Demographic and clinical characteristics of patients with SLE and control groups

	Lupus (n=82)	Control group (n=54)	P value
Age, years	39.2±10.8	40,2±14.3	0.65
BMI, kg/m^2	25.2±4.6	24.8±5.2	0.59
Hand grip, kg	21.7±4.9	25.1±6.6	<0.001
SMMI*, kg	24 (21–26.3)	24.4 (21.6–25.9)	0.89
SMMI (BMI)*, kg/(kg/m^2)	0.98±0.18	1.03±0.33	0.47
ANA, n	77 (93.9)		
Anti-dsDNA, IU/µL	275 (18–544)		
C3, mg/dL	72.3 (56–92)		
C4, mg/dL	11.4 (8.4–15.6)		
PGA	1 (0–1)		
SLEDAI-2K score	8 (6–15.2)		
Neurological disorder, n (%)	10 (12.2)		
Articular involvement, n (%)	51 (62.2)		
Mucocutaneous involvement, n (%)	62 (75.6)		
Serosal involvement, n (%)	9 (11)		
Renal involvement, n (%)	21 (25.6)		
Haematological involvement, n (%)	17 (20.7)		
Anti-Ro (SS-A) positivity, n (%)	30 (36.6)		
Anti-La (SS-B) positivity, n (%)	10 (12.2)		
Anti-Ro52 positivity, n (%)	19 (23.2)		
Anti-Sm positivity, n (%)	13 (15.9)		
Anti-RNP positivity, n (%)	23 (28)		
Anti-dsDNA positivity, n (%)	68 (82.9)		

*Bioimpedance analysis was conducted on 31 patients with SLE (37.8%) and 52 healthy controls (96.3%).
anti-dsDNA, anti-double-stranded DNA; BMI, body mass index; PGA, Physician Global Assessment; RNP, ribonucleoprotein; SLEDAI-2K, SLE Disease Activity Index 2000; SMMI, skeletal muscle mass index.

examination using multivariable logistic regression models (table 4).

DISCUSSION

In this study, we assessed the presence of sarcopenia and probable sarcopenia in patients with SLE, using HGS and SMMI (BMI). The percentage of probable sarcopenia according to HGS was 51.2%, while the percentage of sarcopenia according to HGS and SMMI (BMI) together was 12.9%. A meta-analysis conducted in 2023 included 11 studies that evaluated muscle strength in patients with lupus. HGS was assessed in patients with SLE and it was found that muscle strength was lower than in healthy controls. In this meta-analysis, HGS in patients with lupus was calculated to be 21.74 kg.[8] The muscle strength in patients with SLE in our study was similar (21.7±4.9) and was found to be significantly lower than in the healthy control group.

Our data show a higher prevalence of probable sarcopenia and sarcopenia in patients with SLE than expected for this age group. The higher prevalence of patients with probable sarcopenia in our study may be attributed to the use of sarcopenia indices defined by Bahat *et al* from the EWGSOP Study group in the Turkish population, which differ from those recommended by the EWGSOP.

The prevalence of sarcopenia in systemic sclerosis is 22.5%, which is consistent with other rheumatic diseases such as rheumatoid arthritis (20.8%), psoriatic arthritis (20%) and ankylosing spondylitis (22.7%).[2 9 10] However, there are no studies on the prevalence of sarcopenia in patients with lupus. Sumantri *et al* conducted the only study on the Asian study group based on the HGS cut-off. The group of patients defined as having low HGS was found to be 44%, and the sarcopenia quality of life indices were reported to be lower in the low HGS group.[11] The relationship between sarcopenia and disease markers has been extensively studied in other rheumatic diseases such as osteoarthritis, rheumatoid arthritis, systemic sclerosis, Sjögren's syndrome and ankylosing spondylitis. However, little is known about the relationship between SLE-specific markers and sarcopenia. This study focused on demographic, clinical and immunological variables to investigate the relationship between sarcopenia and SLE.

Table 3 Factors associated with probable sarcopenia in patients with SLE (n=82)

Lupus (n=82)	No sarcopenia (n=40)	Probable sarcopenia (n=42)	P value
Age, years	38.5±9.2	40.7±11.9	0.18
Height, cm	163.6±4.9	160.3±6.6	0.01
Weight, kg	67.9±13.2	64.8±12.5	0.28
BMI, kg/m^2	25.3±4.9	25.2±4.3	0.87
Hand grip, kg	25.8±2.8	17.7±2.7	<0.001
SMMI*, kg	25.3±3.0	22.7±3.3	0.03
SMMI (BMI)*, kg/(kg/m^2)	0.98±0.16	0.98±0.20	0.92
PGA	1 (0–1)	1 (0–1)	0.09
SLEDAI-2K score	9 (4.2–18)	8 (6–14)	0.58
ANA positivity, n (%)			
Yes	38 (95)	39 (92.9)	0.68
No	2 (5)	3 (7.1)	
Anti-dsDNA, IU/µL	415 (140–731)	212 (100–358)	0.01
C3, mg/dL	70.5 (56.2–85.1)	77.5 (59.1–93.5)	0.32
C4, mg/dL	11.1 (8.5–15.4)	12.2 (8.2–15.6)	0.63
Symptom duration, months	120 (60–156)	114 (60–159)	0.80
Disease duration, months	102 (39–144)	90 (36–144)	0.58
Being married, n (%)			
Yes	26 (65)	31 (73.8)	0.38
No	14 (35)	11 (26.2)	
In employment, n (%)			
Yes	9 (22.5)	9 (21.4)	0.90
No	31 (77.5)	33 (78.6)	
Smoking, n (%)			
Yes	8 (20)	8 (19)	0.91
No	32 (80)	34 (81)	
Delay in diagnosis, n (%)			
Yes	16 (40)	17 (40.5)	0.96
No	24 (60)	25 (59.5)	
Neurological disorder, n (%)			
Yes	5 (12.5)	5 (11.9)	0.93
No	35 (87.5)	37 (88.1)	
Articular involvement, n (%)			
Yes	23 (57.5)	28 (66.7)	0.73
No	17 (42.5)	14 (33.3)	
Mucocutaneous involvement, n (%)			
Yes	30 (75)	32 (76.2)	0.90
No	10 (25)	10 (23.8)	
Serosal involvement, n (%)			
Yes	4 (10)	9 (11.9)	0.78
No	36 (90)	73 (88.1)	
Renal involvement, n (%)			
Yes	12 (30)	9 (21.4)	0.37
No	28 (70)	33 (78.6)	
Haematological involvement, n (%)			

Continued

Table 3 Continued

Lupus (n=82)	No sarcopenia (n=40)	Probable sarcopenia (n=42)	P value
Yes	10 (25)	7 (16.7)	0.35
No	30 (75)	35 (83.3)	
Immunological involvement, n (%)			
Yes	39 (97.5)	39 (92.9)	0.61
No	1 (2.5)	3 (7.1)	
Fatigue, n (%)			
Yes	18 (45)	24 (57.1)	0.27
No	22 (55)	18 (42.9)	
Weight loss, n (%)			
Yes	4 (10)	7 (16.7)	0.37
No	36 (90)	35 (83.3)	
Anti-Ro (SS-A) positivity, n (%)			
Yes	14 (35)	16 (38.1)	0.77
No	26 (65)	26 (61.9)	
Anti-La (SS-B) positivity, n (%)			
Yes	6 (15)	4 (9.5)	0.51
No	34 (85)	38 (90.5)	
Anti-Ro52 positivity, n (%)			
Yes	9 (22.5)	10 (23.8	0.88
No	31 (77.5)	32 (76.2)	
Anti-Sm positivity, n (%)			
Yes	8 (20)	5 (11.9)	0.31
No	32 (80)	37 (88.1)	
Anti-RNP positivity, n (%)			0.01
Yes	16 (40)	7 (16.7)	
No	24 (60)	35 (83.3)	
Anti-dsDNA positivity, n (%)			
Yes	36 (90)	32 (76.2)	0.09
No	4 (10)	10 (23.8)	
Low-dose corticosteroid use, n (%)			
Yes	11 (27.5)	15 (35.7)	0.42
No	29 (72.5)	27 (64.3)	

Values are given as n (%), unless otherwise stated.
Median (25th–75th percentile).
*Bioimpedance analysis was conducted on 31 patients with SLE, representing 37.8% of the study population.
anti-dsDNA, anti-double-stranded DNA; BMI, body mass index; PGA, Physician Global Assessment; RNP, ribonucleoprotein; SLEDAI-2K, SLE Disease Activity Index 2000; SMMI, skeletal muscle mass index.

There were no significant differences in SMMI (BMI), PGA and SLEDAI-2K scores between patients with and without probable sarcopenia. The frequency of organ involvement was also not significantly different between the groups. Mahran *et al* found a correlation between HGS and disease activity, while Keramiotou *et al* did not.[12 13] In particular, patients defined as having probable sarcopenia had lower anti-dsDNA levels and higher complement levels. This suggests that the immunological burden of the disease is not associated with sarcopenia. When regression analysis was performed with the significant variables in our study, height and anti-dsDNA antibody levels were found to be independent predictors of sarcopenia in these patients.

Balsamo *et al* showed increased fatigue, decreased functional performance and poorer quality of life with lower dynamic muscle strength in 25 premenopausal patients with SLE with low disease activity compared with 25 controls matched for age, physical characteristics and physical activity level.[14] Another study from our centre

Table 4 Multivariable logistic regression analysis for predictive factors of probable sarcopenia in patients with SLE

	Adjusted OR*	95% CI	P value
Age, years†	1.004	0.95 to 1.05	0.89
Height, cm†	0.91	0.84 to 0.99	0.03
PGA†	1.58	0.77 to 3.22	0.20
Anti-RNP positivity	0.37	0.12 to 1.12	0.08
Anti-dsDNA, IU/µL†	0.99	0.996 to 0.999	0.01

*Age-adjusted OR.
†Continuous variables; otherwise, categorical variables.
anti-dsDNA, anti-double-stranded DNA; PGA, Physician Global Assessment.

demonstrated worse hand functions in 46 patients with SLE (all women), but reported that it was better compared with 51 patients with rheumatoid arthritis.[15] Stockton *et al* found that muscle strength was decreased in 24 patients with SLE compared with the control group.[16] In the study conducted by Keramiotou *et al*, it was found that arthritis was reported by 18% of the participants, while joint pain was indicated by 48% of them.[13] We believe that such a high rate is related to the presence of arthritis and arthralgia in about half of the group. The grip strength of the lupus low disease activity state (LLDAS) group was higher than that of the non-LLDAS group. In the model constructed according to compressive strength, increasing age, female sex, painful joints and use of immunosuppressants were statistically significant, but no relationship was shown with disease activity scores. In our study, no significant association was found between low-dose steroid use and the probable sarcopenia.

Sarcopenia, characterised by the loss of skeletal muscle mass and strength, is greatly influenced by non-disease-related factors, particularly physical exercise. Structured and consistent exercise programmes are crucial for preventing and managing sarcopenia, enhancing mobility and improving overall quality of life. Systematic reviews highlight that aerobic exercises boost cardiovascular health, while resistance training enhances physical function in patients, including those with SLE. Exercise also effectively reduces fatigue and depressive symptoms, which contributes to improved aerobic capacity and overall physical functioning.[17] Considering the diverse effects of SLE on various organ systems and physical functions, personalised physical activity recommendations are vital for effective disease management. According to Blaess *et al*, integrating safe and specific exercises tailored to the patient's disease severity, comorbidities and overall health can significantly improve health outcomes, helping patients with SLE to lead an active and healthier life.[18]

The strengths of this study include the availability of a large and well-characterised population with SLE and the detailed use of sarcopenia and clinical features, immunological parameters and demographic data. However, our study has several limitations. First, we were not able to assess all our patients by BIA due to the restrictions imposed by the pandemic. Second, all our patients were Turkish, as SLE is a predominantly female disease due to the gender trend, which may limit the applicability of the findings to male patients with SLE of other ethnicities. The study was cross-sectional; SLEDAI-2K was analysed, but given that sarcopenia is a progressive disease, it would have been better to look at Systemic Lupus International Collaborating Clinics Damage Index. Furthermore, although we believe that nutritional parameters and physical activity may have an effect on sarcopenia, it is not easy to collect such information in a standardised way, so this could not be done in this study. Evaluation of the same parameters in a larger group of patients from multiple centres is necessary to confirm our findings.

This observational study emphasises that probable sarcopenia is a common and often underestimated complication in patients with SLE. Clinicians should be aware that, according to our findings, probable sarcopenia and sarcopenia may start at a young age in patients with lupus.

Importantly, this study points to significant gaps in current research, particularly in longitudinal studies assessing sarcopenia's progression in patients with SLE and the effectiveness of integrated treatment protocols. Future research should aim at elucidating the precise molecular mechanisms linking SLE and sarcopenia, exploring novel therapeutic targets and developing standardised assessment tools for sarcopenia in the context of autoimmune diseases. By fostering a multidisciplinary approach that integrates rheumatology, geriatrics and physical therapy, we can enhance patient care, improve quality of life and potentially extend disease-free survival in individuals with SLE. Thus, this study not only broadens our understanding of sarcopenia's impact on autoimmune diseases but also sets the stage for innovative research and clinical practice paradigms aimed at addressing this complex challenge.

Conclusions

Our results suggest that sarcopenia is a prevalent condition among patients with SLE. The frequency of probable sarcopenia was 51.2%, and sarcopenia was 12.9% in our patients with lupus. Even if BIA cannot be performed, HGS may be an indicator for the possibility of sarcopenia in patients with lupus. Therefore, we think it is important to evaluate it. Since anti-dsDNA is a predictive factor of probable sarcopenia, physicians should pay attention when evaluating the findings of patients. In addition, the HGS showed low muscle strength in almost half of the patients. Despite this, probable sarcopenia was not found to be associated with any clinical parameters.

Contributors RB and HDV designed the study and wrote the initial draft of the manuscript. MAO, BG and AT contributed to the design of the study, the collection and interpretation of the data, and revising the manuscript for improving intellectual content. RB, AT and BG also wrote the statistical analysis plan and analysed

the data. BC, HS, GTA, FYB, OD, AAG and HK contributed to the data collection and interpretation and revised the manuscript. RB is responsible for the overall content as guarantor. The literature was searched and analysed by all authors. All authors approved the final version to be submitted for publication and agree to be accountable for all aspects of the work.

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