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SARCOPENIA IN RHEUMATOID ARTHRITIS : IMPORTANCE OF EARLY ASSESSMENT

SARCOPENIA NA ARTRITE REUMATOIDE: IMPORTANCIA DA AVALIAÇÃO PRECOCE

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RESUMO

Introdução: A sarcopenia, caracterizada pela perda progressiva de massa e força muscular, é uma comorbidade frequente entre indivíduos com doenças reumáticas.

Objetivos: Avaliar a associação entre sarcopenia e atividade da doença em pacientes com artrite reumatoide (AR).

Métodos: Este estudo prospectivo avaliou o risco de sarcopenia em 54 mulheres com AR por meio do questionário SARC-F, da força de preensão manual (handgrip), do teste de sentar e levantar (sit-to-stand) e do teste de flexão de cotovelo. Foram coletados dados epidemiológicos e clínicos, bem como índices de atividade da doença, incluindo velocidade de hemossedimentação (VHS), proteína C-reativa (PCR), Simplified Disease Activity Index (SDAI) e Clinical Disease Activity Index (CDAI). Todas as avaliações foram repetidas após um intervalo mediano de 4 meses. Um grupo controle composto por 34 mulheres saudáveis, sem comorbidades, foi incluído para comparação.

Resultados: A idade mediana das pacientes com AR foi de 60,4 anos. A sarcopenia foi identificada em 64,8% das pacientes segundo o escore SARC-F. Além disso, 79,6% apresentaram valores de preensão manual inferiores a 16 kg/f; 62,9% realizaram menos de 12 repetições no teste de sentar e levantar; e 98,1% obtiveram menos de 22 repetições no teste de flexão de cotovelo. Quando comparadas aos controles, as pacientes com AR apresentaram desempenho significativamente inferior no SARC-F e na preensão manual ($p < 0,0001$), bem como nos testes de flexão de cotovelo e sentar e levantar ($p = 0,002$).

As análises de correlação mostraram que os escores do SARC-F correlacionaram-se com o SDAI ($r = 0,32$ e $0,29$) e o CDAI ($r = 0,47$ e $0,30$) em ambas as avaliações. Os testes de sentar e levantar e de flexão de cotovelo correlacionaram-se com o SDAI ($r = –0,46$ e $–0,41$) e com o CDAI ($r = –0,38$ para ambos) na segunda avaliação. A força de preensão manual correlacionou-se com o CDAI tanto na primeira quanto na segunda avaliação ($r = –0,34$ e $–0,29$, respectivamente). Não foi observada correlação significativa entre as variações dos parâmetros de atividade da doença e as medidas de força muscular (todas com $p > 0,05$).

Conclusão: A maioria das pacientes com AR apresentou um grau significativo de sarcopenia em comparação com os controles, e os índices de massa muscular estiveram associados às medidas de atividade inflamatória. Entretanto, não foi possível demonstrar que as variações da atividade da doença ao longo do tempo estivessem associadas a mudanças na força muscular.

Palavras-chave: Artrite reumatoide; Sarcopenia; Inflamação.

Abstract:

Introduction: Sarcopenia, characterized by the progressive loss of muscle mass and strength, is a frequent comorbidity among individuals with rheumatic diseases.

Objectives: To evaluate the association between sarcopenia and disease activity in patients with rheumatoid arthritis (RA).

Methods: This prospective study assessed the risk of sarcopenia in 54 women with RA using the SARC-F questionnaire, handgrip strength measurement, sit-to-stand test, and elbow flexion test. Epidemiological and clinical data were collected alongside disease activity indices, including erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), Simplified Disease Activity Index (SDAI), and Clinical Disease Activity Index (CDAI). All assessments were repeated after a median interval of 4 months. A control group of 34 healthy women without comorbidities was included for comparison.

Results: The median age of the RA cohort was 60.4 years. Sarcopenia was identified in 64.8% of patients according to the SARC-F score. Additionally, 79.6% exhibited handgrip strength values below 16 kg/f, 62.9% performed fewer than 12 repetitions on the sit-to-stand test, and 98.1% achieved fewer than 22 repetitions on the elbow flexion test. When compared with controls, RA patients demonstrated significantly poorer performance on the SARC-F and handgrip strength tests ($p < 0.0001$) and on the elbow flexion and sit-to-stand tests ($p = 0.002$).

Correlation analyses revealed that SARC-F scores correlated with SDAI ($r = 0.32$ and 0.29) and CDAI ($r = 0.47$ and 0.30) in both assessments. The sit-to-stand and elbow flexion tests correlated with SDAI ($r = –0.46$ and –0.41) and CDAI ($r = –0.38$ for both) in the second assessment. Handgrip strength correlated with CDAI in both the first and second assessments ($r = –0.34$ and –0.29, respectively). No significant correlation was found between changes in disease activity parameters and variations in muscle strength (all $p > 0.05$).

Conclusion: The majority of RA patients exhibited a substantial degree of sarcopenia compared with controls, and muscle mass indices were associated with inflammatory activity measures. However, changes in disease activity over time were not associated with changes in muscle strength.

Keywords: Rheumatoid arthritis; Sarcopenia; Inflammation.

INTRODUCTION

Sarcopenia refers to a generalized loss of skeletal muscle mass and strength that may occur in the elderly as well as in individuals with chronic inflammatory diseases. It is associated with progressive declines in muscle quantity and function[1], predisposing affected individuals to disability, falls, and loss of independence. In rheumatoid arthritis (RA), sarcopenia is a multifactorial process. Pro-inflammatory cytokines—particularly tumor necrosis factor (TNF) and interleukin-6 (IL-6)—are considered key mediators of skeletal muscle degradation through mechanisms involving enhanced proteolysis, disruption of muscle regeneration, and impairment of myofiber contractility. The use of glucocorticoids and physical inactivity resulting from pain, joint stiffness, and deformities further exacerbate this risk [1-3] .

Epidemiological studies have demonstrated that the prevalence of sarcopenia among RA patients is approximately 2.5 times higher than that observed in the general population [3]. Early recognition and management of sarcopenia are essential, given its

association with adverse health outcomes such as functional disability, insulin resistance, falls, frailty, and increased mortality [3,4].

The present study aimed to evaluate the prevalence of sarcopenia in a cohort of Brazilian patients with RA and to investigate its association with disease activity.

METHODS

This prospective study included a total of 88 participants: 54 women with rheumatoid arthritis and 34 healthy controls. The study protocol was approved by the local Research Ethics Committee (protocol number 5.936.828), and all participants provided written informed consent prior to enrollment.

A convenience sample was used, comprising all RA patients from a single rheumatology center who attended routine follow-up visits over a two-year period and consented to participate. Patients' companions were invited to participate as control subjects.

RA patients underwent two evaluations, with the second assessment performed at a median interval of 4.0 (4.0–7.0) months from the first. Epidemiological, clinical, and treatment data were collected, and disease activity was assessed using the Simplified Disease Activity Index (SDAI), Clinical Disease Activity Index (CDAI), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) levels.

The CDAI was calculated based on the 28-joint tender and swollen joint counts, the patient's global disease activity assessment (0–10), and the evaluator's global assessment (0–10) [5]. The SDAI was determined by summing the 28-joint tender and swollen joint counts, patient and evaluator global assessments (each 0–10), and CRP in mg/dL [5].

Muscle Strength Assessment

Muscle strength was evaluated at both time points using the following instruments:

a) SARC-F (Simple Questionnaire to Rapidly Diagnose Sarcopenia): A five-item questionnaire assessing strength, assistance with walking, ability to climb stairs, rise from a chair, and frequency of falls. Each item is scored from 0 to 2, with a total score ranging from 0 to 10. A score ≥ 4 indicates risk of sarcopenia [6, 7].

b) Sit-to-Stand Test: Assesses lower limb strength by recording the number of times the individual can rise from a seated position within 30 seconds. The expected normal range is 12 to 17 repetitions [8].

c) Elbow Flexion Test: Evaluates upper limb strength by counting the number of flexion movements performed in 30 seconds. The standard load is approximately 2 kg for women and 4 kg for men, with 22 repetitions considered normal [8].

d) Handgrip Strength: Measures static grip force using a Jamar® dynamometer. Participants were seated with feet flat on the floor, knees and elbows flexed at approximately 90°, and the forearm close to the trunk. Maximal grip strength was maintained for about three seconds, with at least 15 seconds of rest between trials. Three repetitions were performed using the dominant hand, and the highest value was recorded for analysis (1, 7). The cut-off values adopted were those proposed by the European Working Group on Sarcopenia in Older People (EWGSOP2): <27 kg for men and <16 kg for women [9].

Inclusion and Exclusion Criteria: Eligible participants were female, aged 45 years or older, and fulfilled the 2010 ACR/EULAR classification criteria for RA with a minimum score of 10 points [10]. Individuals with renal insufficiency (glomerular filtration rate ≤ 20 mL/min), heart failure (New York Heart Association Class ≥ III), eating disorders, malabsorption syndromes, neoplasms, or those receiving treatment for chronic infectious diseases were excluded.

Statistical Analysis: Data were organized into frequency and contingency tables. Nominal variables were expressed as percentages, while numerical data were summarized as mean ± standard deviation (for parametric samples) or median and interquartile range (for non-parametric samples). Data distribution was assessed using the Shapiro–Wilk test. Comparisons between the first and second evaluations were made using Fisher's exact test or the chi-square test for categorical variables, and the unpaired t-test or Mann–Whitney test for numerical variables, as appropriate. Correlation analyses were performed using Pearson's test (parametric) or Spearman's test (non-parametric). A significance level of 5% was adopted for all analyses. Statistical calculations were performed using GraphPad Prism version 8.0.0 for Windows (GraphPad Software, San Diego, California, USA; www.graphpad.com).

RESULTS:

a) Description of studied sample and comparison with controls.

The RA sample had 54 females with mean age at diagnosis of 47,5±10.7 years old; about 66.6% were positive for rheumatoid factor. Patients and controls were paired for age (p=0.60) and exposure to tobacco (p=0.85).

The main characteristics of RA sample is on Table 1 that also shows the comparison between first and second evaluation.

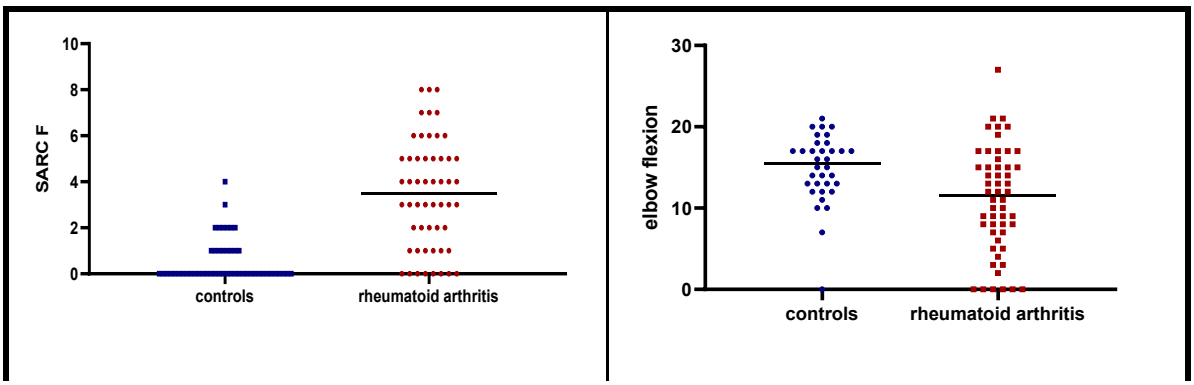
Table 1- Main characteristics of RA studied sample

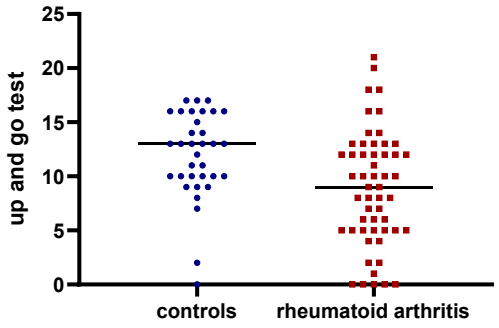
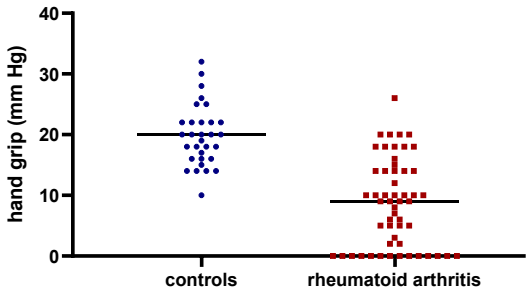
	First evaluation		Second evaluation		P
Number (n)	54		52		
Comorbidities (n)					
Arterial hypertension	23/54 – 42.5%				
Dislipidemia	17/54 – 31.4%				
Diabetes	8/54 -14.8%				
Hipotireoidismo	13/54 – 24.0%				
Fibromialgia	7/54 – 12.9%				
Osteoporose	10/54 – 18.5%				
Treatment (n)					
Antimalarials	6/54	11.1%	4/52	7.6%	0.74
Prednisone	19/54	35.1%	20/52	38.4%	0.72
Leflunomide	27/54	50.0%	26/52	50.0%	>0.99
Methotrexate	27/54	50.0%	23/52	44.2%	0.55
Anti TNF-α	6/54	11.1%	6/52	11.5%	0.94
Jak inhibitor	9/54	16.6%	8/52	15.3%	0.85
Rituximab	1/54	1.8%	0	0	N too small
Disease activity indexes					
SDAI – median (IQR)	7.40 (2.97-11.8)		4.70 (1.73-13.9)		0.94
CDAI - median (IQR)	5.0 (1.0-10.5)		4.0 (1.0-11.0)		0.74
ESR - median (IQR)- mm	29,0 (19.0-40.0)		25.0 (16.0-39.0)		0.61
CRP - median (IQR) – mg/dL	3,0 (1.42- 10.2)		4.0 (1.0-11.7)		0.80
Hemoglobine – mediana (IQR)- g/dL	13,0 (12,3-13,4)		13,0 (12.4-14.0)		0.38

SDAI – simplified disease activity index; CDAI – clinical disease activity index; ESR - erythrocyte sedimentation rate; CRP- C reactive protein; IQR= interquartile range.

The comparison of strength data on RA patients and controls is on **Figure 1** that shows significant difference between RA patients and controls

FIGURE 1. Comparison of data on strength between RA (rheumatoid arthritis) patients and controls



RA=3.5 (1.0-5.0); controls 0 (0-1.0); p<0.0001	RA=11.5 (6.7-15.2); controls 15.5 (12.7-17.2); p=0.002
	
RA=9.0 (5.0-12.2); controls =13.0 (10.0-16.0) p= 0.002	RA=9.0 (0-14.0) mm Hg; controls=20.0 (16.0-22.0) mm Hg. p<0.0001

Considering the first evaluation, 43 out of 54 (79.6%) patients had a palmar grip strength below 16 mmHg; 34 out of 54 (62.9%) had a sit-to-stand test score below 12; 53 of them (98.1%) had an elbow flexion test score below 22 repetitions, and 35 patients (64.8%) scored ≥4 on the SARC-F questionnaire.

Table 2 shows the strength data in the first and second evaluation.

TABLE 2 - Description of strength parameters in the first and second evaluation

Strength Data	First evaluation	Second evaluation	P
SARC-F -median (IQR)	3.5 (1.0-5.0)	3.0 (1.0-5.2)	0.94
Elbow flexion test- mean(SD)	10.9 ±6.5	9.6±5.2	0.25
Sit -to-stand test– mean (SD)	8.8±5.3	8.9±4.7	0.89
Handgrip – median (IQR)	9.0 (0-14.0)	10.0 (5.0-15.0)	0.31

SARC-F= Simple Questionnaire to Rapidly Diagnose Sarcopenia) questionnaire; IQR= interquartile range; SD= standard deviation

2- Correlation studies of inflammatory indexes with strength parameters.

Table 3 shows the results of correlation studies with disease activities index in first and second visits.

TABLE -3 – Correlation studies of disease activity indexes with strength tests.

SARC-F STUDIES - FIRST VISIT			
SDAI	0.32	0.04 to 0.55	**0.01**
CDAI	0.47	0.22 to 0.66	**0.0003**
ESR	0.13	-0.15 to 0.39	0.35
CRP	-0.20	-0.45 to 0.07	0.13

SARC-F STUDIES - SECOND VISIT			
SDAI	0.29	0.002 to 0.54	**0.03**
CDAI	0.30	0.02 to 0.54	**0.04**
ESR	0.19	-0.09 to 0.43	0.16
CRP	-0.02	-0.30 to 0.25	0.84
SIT-TO-STAND TEST -FIRST VISIT			
SDAI	-0.10	-0.27 to 0.18	0.48
CDAI	-0.23	-0.27 to 0.05	0.09
ESR	-0.28	-0.51 to -0.0003	**0.04**
CRP	0.11	-0.16 to 0.38	0,39
SIT-TO-STAND TEST – SECOND VISIT			
SDAI	-0,46	-0,66 to -0.19	**0.001**
CDAI	-0,38	-0,60 to -0,11	**0.005**
ESR	-0.15	-0.41 to 0,12	0,28
CRP	0.04	0,24 to 0.32	0.74
ELBOW FLEXION TEST – FIRST VISIT			
SDAI	-0.21	-0.46 to 0.77	0.13
CDAI	-0.31	-0.54 to -0.04	0**.02**
ESR	-0.08	0.35 to 0.20	0.56
CRP	0.05	-0.22 to 0.32	0.68
ELBOW FLEXION TEST – SECOND VISIT			
SDAI	-0.41	-0.63 to -0.14	**0.003**
CDAI	-0,38	-0.60 to -0.10	**0.006**
ESR	-0.03	-0,31 to 0.24	0.81
CRP	0.07	-0.22 to 0.34	0.62
HANDGRIP TEST- FIRST EVALUATION			
SDAI	-0.27	-0.51 to 0.01	0.053
CDAI	-0.34	-0.57 to -0.07	**0.01**
ESR	-0.05	-0.32 to 0.22	0.68
CRP	-0.09	-0.36 to 0.18	0.48
HAND GRIP TEST- SECOND EVALUATION			
SDAI	-0.24	-0.50 to 0.05	0.09
CDAI	-0.29	-0.54 to -0.009	**0.03**
ESR	-0.009	-0.36 to 0.19	0.53
CRP	-0.05	-0.33 to 0.23	0.71

SARC-F= Simple Questionnaire to Rapidly Diagnose Sarcopenia) questionnaire; SDAI – simplified disease activity index; CDAI – clinical disease activity index; ESR - erythrocyte sedimentation rate;CRP- C reactive protein

3- Correlation studies of chance in inflammatory parameters with change in strength evaluation.

The correlation studies of change in the strength test (Δ strength tests) with changes in the inflammatory measurements (Δ inflammatory indexes) is on **Table 4**. No correlations were found but for CRP.

Table 4 – Correlation studies of changes (Δ) in inflammatory indexes and strength measurement tests;

Δ SDAI	-0,16	-0.16 to 0.43	0.14
Δ CDAI	-0.10	-0.37 to 0.18	0.48
Δ ESR	-0.06	-0.34 to 0.22	0.64
Δ CRP	-0.28	-0.52 to -0.001	**0.04**

SDAI – simplified disease activity index; CDAI – clinical disease activity index; ESR - erythrocyte sedimentation rate; CRP- C reactive protein

Discussion

Our findings indicate that the majority of patients with rheumatoid arthritis (RA) are at risk of sarcopenia, with nearly 65% screening positive using the SARC-F tool. The SARC-F, developed by Malmstrom et al., [11] is a simple, self-administered questionnaire designed for the clinical detection of sarcopenia. According to its authors, it should be integrated into the initial assessment of this condition. This instrument evaluates perceived difficulty in five functional domains—strength, assistance with walking, rising from a chair, climbing stairs, and falls—thereby allowing clinicians to monitor changes in health status associated with sarcopenia [12] .

In addition to the SARC-F, we employed the elbow flexion and sit-to-stand tests to assess upper and lower limb strength, respectively. Handgrip strength, commonly used as a surrogate marker of overall muscle force, was also evaluated. However, in the context of RA, its interpretation is limited. While Wind et al[13] demonstrated a strong correlation between handgrip and total muscle strength in children, adolescents, and young adults, this relationship may not hold in RA due to inflammation, pain, stiffness, and joint deformities affecting the hands. These factors can lead to an underestimation of true muscular capacity.

The cutoff values used for tests such as handgrip strength or chair rise are generally based on T-scores derived from healthy young adults, defining weakness relative to normative data [3]. Notably, in RA, handgrip strength follows a markedly different trajectory from that of the general population, declining in parallel with disease duration. Remarkably, the handgrip strength observed at the time of RA diagnosis has been reported to correspond to that of healthy individuals in their ninth or tenth decade of life, irrespective of the patient's actual age [3].

Several mechanisms contribute to muscle mass loss in RA, including aging, dietary factors, physical inactivity, metabolic disturbances, hormonal dysregulation, and the action of pro-inflammatory cytokines[14]. In our cohort, composite indices of disease activity (CDAI and SDAI) correlated with muscle strength, underscoring the impact of inflammation in this context.

RA, being an immune-mediated inflammatory disorder, is characterized by the activity of cytokines such as tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β), interleukin-6 (IL-6), interferon gamma (IFN-γ), and transforming growth factor beta (TGF-β). These mediators not only drive joint destruction but also influence systemic

protein and energy metabolism [15]. IL-6, for instance, functions as both a cytokine and a myokine—produced by skeletal muscle during exercise, where it promotes myogenesis and myofiber hypertrophy. However, chronic IL-6 elevation, particularly in the inflammatory milieu of RA, promotes muscle catabolism. Myostatin, another myokine that induces skeletal muscle atrophy, has been linked to RA disease activity regardless of muscle mass, suggesting a potential role of myokines in sustaining synovitis and systemic inflammation [3]. TNF-α, a key cytokine in RA pathophysiology, contributes to muscle dysfunction by inducing insulin resistance and promoting cachexia through reduced peripheral insulin sensitivity and attenuation of its anticatabolic effects. Moreover, muscle protein degradation is further mediated by IFN-γ signaling and activation of the nuclear factor kappa B (NF-κB) transcription pathway[16].

In our study, we did not observe a significant correlation between changes in inflammatory indices and changes in muscle strength. Several hypotheses may explain this finding: (1) the interval between assessments may have been too short to capture meaningful variations, or (2) disease activity control alone may be insufficient to reverse sarcopenia, requiring concurrent muscle-strengthening interventions to restore functional capacity.

Attention to muscle function in RA is crucial, as it plays a central role in maintaining mobility, preventing falls and fractures, and preserving patient autonomy.

This study is limited by its small sample size and short follow-up period. Larger and longer-term studies are warranted to clarify the relationship between disease activity and sarcopenia in RA.

In conclusion, most RA patients exhibit a significant degree of sarcopenia compared with healthy controls. Muscle mass measurements were associated with inflammatory activity; however, we were unable to demonstrate that changes in inflammation translate into measurable changes in muscle strength.

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