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Prevalence of Frailty in Geriatric People with Rheumatoid Arthritis and its Relationship with Disease Activity

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ABSTRACT

Background: Frailty, a condition of reduced physiological resilience, is common in older adults and is particularly prevalent among patients with rheumatoid arthritis (RA). RA's chronic inflammation can exacerbate frailty, leading to worse outcomes in the geriatric population.

Aim of the study: This study aims to assess the prevalence of frailty in geriatric patients with RA and investigate its relationship with RA disease activity, measured by the Disease Activity Score in 28 joints (DAS28), as well as inflammatory markers such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and rheumatoid factor (RF).

Methods: A cross-sectional study was conducted involving geriatric RA patients. Frailty was measured using a frailty index, while RA disease activity was assessed using DAS28. Clinical markers including CRP, ESR, and RF were also analyzed. According to frailty status, patients were divided into Robust (24) and prefrail/frail (76). Data was compared between subgroups.

Results: The study found a significant prevalence of frailty among the geriatric RA population (76.0%) and there was female sex predilection. All patients were in normal or overweight groups according to their body mass index (BMI), and the majority of them were widows. In addition, drug therapy, grip strength score and MMSE were comparable between Robust and prefrail/frail subgroups. Higher RA disease activity (DAS28) (β=0.379, P = 0.001) and disease duration (β= 0.232, p = 0.008) were strongly associated with increased frailty Additionally, elevated levels of CRP and ESR were correlated with frailty, highlighting the link between inflammation and frailty in these patients.

Conclusion: Disease activity and duration are key drivers of frailty in patients with RA. Managing RA effectively, particularly by achieving and maintaining low disease activity or remission, could significantly reduce the risk of frailty.

Keywords: Frailty; Frailty index; Geriatric; Rheumatoid arthritis

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INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, systemic autoimmune disease primarily affecting joints, causing pain, swelling, stiffness, and progressive disability [(1)]. The prevalence of RA increases with age, and it significantly impacts the quality of life of older adults due to its long-term effects on physical function and mobility. Geriatric patients with RA often experience more severe disease manifestations due to exacerbating comorbidities commonly associated with aging [(2)].

One of the critical issues in the aging population is frailty, a clinical syndrome characterized by decreased physiological reserves, increased vulnerability to stressors, and a higher risk of adverse outcomes such as falls, hospitalizations, and mortality [(3)]. Frailty is more prevalent in individuals with chronic inflammatory diseases like RA, where the inflammatory burden can accelerate the decline in physical and functional health.[4] The interplay between RA and frailty is of growing concern, as both conditions independently contribute to disability, reduced quality of life, and poor prognosis. Frailty has been identified as a potential marker for predicting outcomes in RA patients, particularly in the geriatric population. Disease activity in RA, often measured using the Disease Activity Score in 28 joints (DAS28), is a reliable indicator of the severity of joint inflammation [(5,6)]. High disease activity in RA is associated with worse clinical outcomes, including physical decline and increased mortality, both of which are critical components of frailty [(6)].

The relationship between RA disease activity and frailty has not been thoroughly investigated, particularly in geriatric patients. Understanding this relationship is crucial for optimizing treatment strategies and improving long-term outcomes for this vulnerable population. Early identification of frailty and its association with high disease activity may enable healthcare providers to implement targeted interventions aimed at minimizing disease progression and enhancing the overall well-being of geriatric patients with RA.

PATIENTS AND METHODS

Study Participants This cross-sectional study included geriatric patients diagnosed with rheumatoid arthritis (RA) who were recruited from outpatient Geriatric, and Rheumatology clinics, Al-Azhar University hospitals, Cairo, Egypt. Eligible participants were aged 60 years or older, had a confirmed diagnosis of RA based on the 2010 ACR/EULAR classification criteria, and were able to provide informed consent. Patients with cognitive impairments, terminal illnesses, or other conditions severely limiting their ability to participate were excluded from the study. A total of 100 patients met the inclusion criteria and were enrolled.
Frailty Assessment Frailty was assessed using the Frailty Index (FI), which measures the accumulation of health deficits across multiple domains, including physical function, comorbidities, cognitive status, and laboratory results. The FI was calculated by dividing the number of deficits present by the total number of possible deficits assessed. Patients were categorized as frail if their FI score was greater than or equal to 0.25, indicating a higher burden of health deficits, prefrail with scores between 0.10 and 0.24, and robust if the score was below 0.10. This approach provided a comprehensive evaluation of the participants' overall health and frailty status.

Disease Activity Assessment: Rheumatoid arthritis disease activity was evaluated using the Disease Activity Score in 28 joints (DAS28), which includes a count of 28 tender and swollen joints, the patient's global health assessment (on a visual analog scale), and inflammatory markers (either C-reactive protein [CRP] or erythrocyte sedimentation rate [ESR]). DAS28 scores were used to categorize disease activity into four levels: remission (DAS28 $\leq$ 2.6), low disease activity (DAS28 > 2.6 to $\leq$ 3.2), moderate disease activity (DAS28 > 3.2 to $\leq$ 5.1), and high disease activity (DAS28 > 5.1). This standardized method allowed for consistent classification of RA severity across the study population.

Analysis: The data were analyzed using the Statistical Program for Social Science (SPSS) version 24. Qualitative data were presented as frequencies and percentages. Quantitative data were presented as mean ± SD. The mean (average) is the center value of a discrete collection of numbers, calculated as the sum of values divided by the number of values. The standard deviation (SD) is a measure of how dispersed a group of data is. A low SD implies that the values are close to the set's mean, whereas a high SD shows that the values are scattered throughout a wider range. The independent sample T test (T) is used to compare two sets of normally distributed data. A chi-square test was used to compare non-parametric data. P-values < 0.05 were deemed significant. P-values < 0.001 were regarded as highly significant. P-values greater than 0.05 were deemed insignificant.

RESULTS

The study included a total of 100 participants, of which 24 were classified as robust (non-frail), 53 as pre-frail and 23 as frail. Among the robust group, 37.5% were males and 62.5% were females, while in the prefrail/frail group, 28.9% were males and 71.1% were female. The difference in sex distribution between the groups was not statistically significant (p = 0.430). The mean age of all participants was 64.1 years (SD = 2.5). The mean age in the robust group was 64.33 years (SD = 2.408), while in the prefrail/frail group it was 64.00 years (SD = 2.582). There was no significant difference in age between the two groups (p = 0.416). The median BMI was 25 kg/m² (IQR = 21-27) for the whole sample. The robust group had a median BMI of 21 kg/m² (IQR = 19-23), while the prefrail/frail group had a significantly higher median BMI of 25 kg/m² (IQR = 23-27). This difference in BMI was statistically significant (p < 0.001). The marital status distribution showed no significant differences between groups (p = 0.839). In the robust group, 37.5% were married, 8.3% single, 50% widowed, and 4.2% divorced. In the prefrail/frail group, 36.8% were married, 10.5% single, 43.4% widowed, and 9.2% divorced (Table 1). There was a significant difference in DAS categories between the robust and prefrail/frail groups (p = 0.013). In the robust group, 16.7% were in remission, while only 5.3% of the prefrail/frail group were in remission. Low disease activity was observed in 54.2% of the robust group and 30.3% of the

prefrail/frail group. Medium and high disease activity were more common in the prefrail/frail group (52.6% and 11.8%, respectively) compared to the robust group (29.2% and 0%). The median ESR was significantly higher in the prefrail/frail group (56 mg/dl, IQR 42.3-69.8) compared to the robust group (41.5 mg/dl, IQR 21.9-61.1), with a p-value of 0.012. CRP (C-Reactive Protein): Median CRP levels were also significantly higher in the prefrail/frail group (34 mg/dl, IQR 15.5-52.5) than in the robust group (12 mg/dl, IQR 0.4-23.6), with a p-value < 0.001. RF (Rheumatoid Factor): Median RF levels were significantly elevated in the prefrail/frail group (80 IU/ml, IQR 63.8-96.3) compared to the robust group (50.5 IU/ml, IQR 23.6-77.4), with a p-value < 0.001. Participants in the prefrail/frail group had a longer median disease duration of 7 years (IQR 5.5-8.5) compared to 4 years (IQR 2.5-5.5) in the robust group (p < 0.001). There was no significant difference between the groups regarding the use of conventional DMARDs, biological agents, steroids, or NSAIDs (p= 0.209). The median MMSE scores were similar between the groups (20.5 in the robust group and 22 in the prefrail/frail group), with no significant difference (p = 0.942). Median grip strength was higher in the robust group (24.5 kg, IQR 18.6-30.4) than in the prefrail/frail group (22 kg, IQR 17.8-26.3), but this difference was not statistically significant (p = 0.185) (Table 2)

Table (3) presents the results of a regression analysis examining variables associated with the frailty index (FI). The model explains 63.2% of the variability of the frailty index (R^2 = 0.632, p < 0.001), indicating a strong overall fit. Disease Activity Score (DAS) had the strongest association with the frailty index, showing a standardized beta value of 0.379 and a highly significant p-value of 0.001. This suggests that higher disease activity is strongly linked with greater frailty. Disease Duration also had a significant positive association with frailty, with a standardized beta value of 0.232 and a p-value of 0.008, indicating that longer disease duration contributes to higher frailty levels. Other variables, such as age (β = 0.068, p = 0.313), sex (β = 0.009, p = 0.900), RF (β = 0.050, p = 0.655), grip strength (β = -0.045, p = 0.526), BMI (β = -0.012, p = 0.868), and the inflammatory marker ESR (β = -0.019, p = 0.848), did not show significant associations with frailty in this analysis. Notably, the MMSE (cognitive function) had a negative but borderline insignificant association with frailty (β= -0.142, p= 0.069), suggesting a potential, though not statistically significant relationship between cognitive function and frailty. DAS and disease duration were the most significant predictors of frailty in this analysis, with higher disease activity and longer disease duration strongly contributing to increased frailty. Other factors like age, sex, and inflammatory markers did not show significant associations with frailty in this model.

Table (1): Participant characteristics of participants stratified by frailty status.

		All n=100	Robust n=24	Prefrail/ Frail n=76	P-value
Sex	**Male (%)**	**31%**	**37.5%**	**28.9%**	**0.430**
	Female (%)	**69%**	**62.5%**	**71.1%**	
Age (years); mean (SD)		**64.1 (2.5)**	**64.33 (2.4)**	**64.00 (2.6)**	**0.416**
BMI (kg/m2); median (IQR)		**25 (21-27)**	**21 (19-23)**	**25 (23-27)**	**<0.001**
Marital status	**Married**	**37**	**37.5%**	**36.8%**	**0.839**
	Single	**10**	**8.3%**	**10.5%**	
	Widow	**45**	**50.0%**	**43.4%**	
	Divorced	**8**	**4.2%**	**9.2%**	

Table (2): Disease-describing factors of the whole sample and stratified by frailty status.

		All n=100	Robust n=24	Prefrail/Frail n=76	P-value
DAS	**Remission**	8.0%	16.7%	5.3%	
	Low	36.0%	54.2%	30.3%	**0.013**
	Medium	47.0%	29.2%	52.6%	
	High	9.0%	0.0%	11.8%	
Inflammatory parameters	**ESR (mg/dl): median (IQR)**	55 (40-70)	41.50 (21.9-61.1)	56 (42.3-69.8)	**0.012**
	CRP (mg/dl); median (IQR)	26 (11-39)	12 (0.4-23.6)	34 (15.5-52.5)	**<0.001**
RF (IU/ml); median (IQR)		77 (55.8-99)	50.5 (23.6-77.4)	80 (63.8-96.3)	**<0.001**
Median years (IQR)		6 (4-8)	4 (2.5-5.5)	7 (5.5-8.5)	**<0.001**
Drug therapy	**cDMARDs**	51	54.2%	50.0%	
	Biological	7	8.3%	6.6%	0.209
	Steroid	28	33.3%	26.3%	
	NSAIDs	14	4.2%	17.1%	
MMSE; median (IQR)		22 (15-26)	20.5 (14.3-26.8)	22 (17-27)	0.942
Grip strength (kg); median (IQR)		22 (17-26)	24.5 (18.6-30.4)	22 (17.8-26.3)	0.185

Table (3): Variables associated with the frailty index

R^2, p-value	Included independent variable	Standardized beta-value	p-value
FI **0.632, p<0.001**	Age	0.068	0.313
	Sex	0.009	0.900
	DAS	**0.379**	**0.001**
	RF	0.050	0.655
	Grip Strength	-0.045	0.526
	BMI	-0.012	0.868
	Disease duration	**0.232**	**0.008**
	MMSE	-0.142	0.069
	ESR	-0.019	0.848

DISCUSSION

Our results show that frailty is present in 15% of RA geriatric patients. Even more alarming is that an additional 30% are found to be prefrail.

The prevalence of frailty is consistent with the results of the study of **Andrews *et al.*** [7] who investigated the frailty prevalence in younger RA patients with a mean age of 58.0 (SD 10.8) years. However, the percentage of prefrail patients, at 69%, is much higher in their sample. This could be because the individuals in the Andrews et al. study had a longer disease duration of 19.2 (SD: 10.6) years and a lower handgrip strength of 17.4 (SD: 9.3) kg. Another reason could be that the different methods of assessing frailty status: Anderson et al.[7] measured frailty with the Fried phenotype, and in our study we used the Frailty index.

Comparing these numbers to the prevalence in community-dwelling people above 65 years in Austria (frail: 10.8%; prefrail: 40.7%) [8] and compared to the prevalence of frailty in geriatric individuals in nursing homes (frail: 24.7%; prefrail: 61.4%) [9], these numbers for rheumatoid arthritis patients are concerning. Furthermore, these numbers are even higher than those in Canadian cancer patients [10] and stable chronic obstructive pulmonary disease patients [11].

One underlying reason for the high prevalence of prefrailty and frailty could be the high amount of people suffering from exhaustion, which is a common phenomenon in rheumatoid arthritis patients [12-14].

According to the available literature, exhaustion has been reported in 40–80% of RA patients, and has been shown to have a multifactorial cause [12]. Mentioned reasons are pain, physical inactivity, depression and sleep disturbance [15].

The Disease Activity Score (DAS) was found to be the most significant predictor of frailty ($\beta = 0.379$, $p = 0.001$). This strong association highlights that active RA, characterized by ongoing inflammation and joint damage, is a major contributor to frailty. High disease activity likely leads to physical disability, pain, fatigue, and muscle weakness, all of which increase vulnerability to frailty. These findings are consistent with previous studies that demonstrate a link between uncontrolled RA and worsened physical function. Effective management of disease activity through aggressive treatment strategies, including disease-modifying antirheumatic drugs (DMARDs) and biologics, may reduce the frailty burden in RA patients.

The significant relationship between disease duration and frailty ($\beta = 0.232$, $p = 0.008$) suggests that long-term RA increases the risk of frailty. Prolonged inflammation and joint degradation over time are likely to contribute to physical decline. Additionally, long disease duration may be associated with cumulative damage from both the disease and its treatment (e.g., corticosteroids), which can accelerate frailty. Early diagnosis and treatment of RA, aiming for sustained remission, could help in preventing frailty by minimizing joint damage and maintaining physical function over time.

While CRP and ESR did not show significant associations, the trend for CRP supports the idea that systemic inflammation plays a role in frailty among RA patients.

In this context, it should be mentioned that **Cooney *et al.*** [16] identified the excessive production of proinflammatory cytokines as being responsible for cachexia in RA patients. In addition, in older people, studies have shown that high inflammatory markers are associated with lower muscle mass and lower muscle strength, and are also related to self-reported functional disabilities [4,17]. These inflammatory markers, and the glucocorticoid treatment, might lead to reduced myocyte protein synthesis and increased protein degradation [18,19].

As we saw a significant difference between people who were assessed as prefrail/frail and the robust individuals, we assume that inflammatory markers and cytokines, in combination with the low level of physical activity, might contribute to the high prevalence of frailty in our data set. Interestingly, the MMSE showed a borderline association with frailty, suggesting that cognitive impairment could be a factor in RA-related frailty. RA patients with cognitive decline may have difficulty managing their disease and adhering to treatment, which could exacerbate physical frailty. Cognitive assessments may become important in comprehensive RA care, particularly for frail patients.

We expected an association between disease activity and handgrip strength, but this was refuted by the data. Nonetheless, the non-association is in line with the available literature, which describes only a limited association [20].

A further study, made up of patients with a disease duration of at most two years, saw a stronger association, and these participants even reached normal handgrip strength when the disease was in remission [21].

Contrary to expectations, variables such as age, sex, BMI, and grip strength did not significantly correlate with frailty in this cohort. This finding suggests that in RA patients, frailty may be more heavily driven by disease-specific factors (e.g., inflammation, disease activity) than by traditional demographic or physical health indicators. For example, BMI was not a significant predictor, despite its usual association with frailty in the general population. This emphasizes the unique mechanisms driving frailty in RA, particularly the role of chronic disease management.

Interventions to prevent or reduce frailty include physical training, nutritional interventions and medication to lower inflammation [22].

For RA patients, the Centers for Disease Control and Prevention (CDC) and the American College of Sports Medicine (ACSM) have recommended a combination of endurance, strength and flexibility exercises [23,24].

Apart from the general effects of physical training (e.g. cardiovascular, musculoskeletal effects, effects on bones and overall function), the training also has specific health benefits in RA patients; for example, it positively influences the rheumatoid cachexia [16].

Exercise also decreases functional disabilities and improves functional capacity and joint count. This positive impact has been shown to be clinically relevant [25]. However, to date, no direct association between diet and RA has been proven [26,27].

Considering that cardiovascular diseases are common in this population and considering that 24% subjectively report food as having an influence on their RA symptoms, it is recommended that national nutritional guidelines are followed [28].

For the prevention of frailty, energy and protein intake are of special relevance [29,30]

Conclusion:

The findings from our study emphasize the importance of disease activity and disease duration as key drivers of frailty in patients with RA. Managing RA effectively, particularly by achieving and maintaining low disease activity or remission, could significantly reduce the risk of frailty. This study highlights the need for targeted interventions to reduce frailty in RA patients, particularly focusing on early, aggressive management of disease activity. Future research should explore the role of inflammation and cognitive function further and identify strategies to improve outcomes for frail RA patients.

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