



# Sarcopenia in Spondyloarthritis

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## ABSTRACT

Sarcopenia, the age-related loss of skeletal muscle mass and function, has increasingly been recognized as a significant health issue in various rheumatic diseases. However, sarcopenia is commonly overlooked and undertreated in mainstream practice in patients with spondyloarthritis (SpA), likely due to the complexity of determining what variables to measure, how to measure them, what cut-off points best guide diagnosis and treatment, and how to best evaluate effects of therapeutic interventions. This review aims to explore the current understanding of sarcopenia in SpA focusing on its prevalence, pathogenesis, and clinical implications. We also discuss potential strategies for diagnosis, prevention, and management of sarcopenia in SpA patients.

**Keywords:** Sarcopenia; Spondyloarthritis; Ankylosing spondylitis; Rheumatic diseases

## INTRODUCTION

Sarcopenia is a natural phenomenon that decreases muscle mass and function with aging. In the seventh and eighth decade of life, muscle strength declines by 20-40% and the degree of reduction increases gradually [1]. The word sarcopenia or "leanness of the flesh" comes from ancient Greece (Greek "sarco" [meat] + "penia" [lack of]). Today, up to 52% of older adults suffer from sarcopenia and lose approximately 1% of muscle mass each year [2]. The introduction of the ICD-10 code for sarcopenia in 2016 underscores the growing societal recognition of the importance of this disorder. This Code can now be used to bill for care in some countries [3]. The prevalence of sarcopenia will continue to increase as the number of people over the age of 65 is expected to double over the next three decades. However, the term sarcopenia now, is used to describe all kinds of loss of muscle tissue and function whatever the cause is (aging, chronic diseases, or low protein-energy intake and physical inactivity). As early as in the third century B.C., Hippocrates described the wasting syndrome associated to chronic diseases. Indeed, several metabolic abnormalities secondary to chronic diseases of multifactorial origin are observed. Moreover, the spectrum of body composition changes in disease states varies widely from a minimal weight loss related to skeletal muscle wasting to an extreme state of loss of fat and muscle in refractory cachexia (as in cancer), including the particular case of the normal or high BMI of sarcopenic obesity, that combines high muscle loss with increased fat mass (as reported in rheumatoid arthritis) [4]. Sarcopenia, as defined by muscle loss and dysfunction, is a common feature of all chronic inflammatory diseases and involves impairment of either contractile, metabolic and endocrine functions

of skeletal muscle [5]. It is, thus important to define these different concepts of muscle wasting and sarcopenia-like conditions that are commonly used in the literature (Table 1). Cachexia, which is derived from the Greek words kakós (bad) and héxis (condition), is a syndrome of multifactorial origins characterized by severe body weight, fat and muscle loss and increased protein catabolism due to underlying disease(s) [6]. Among the contributory factors to the onset of cachexia we can list anorexia and metabolic alterations, i.e. increased inflammatory status, increased muscle proteolysis, impaired carbohydrate, protein and lipid metabolism. Inflammation does play a crucial role in its pathogenesis and its presence allows for cachexia identification.

Spondyloarthritis (SpA) is a group of chronic inflammatory diseases that primarily affect the joints and entheses, which are the points where tendons and ligaments attach to the bones [7]. The most common form of spondyloarthritis is ankylosing spondylitis (AS), which mainly impacts the spine, causing pain, stiffness, and reduced mobility. Other forms of SpA include psoriatic arthritis, reactive arthritis, enteropathic arthritis associated with inflamma-

Table 1: Définitions.

Sarcopenia	Syndrome characterized by low muscle strength with the presence of low muscle mass with/without low physical performance.
Cachexia	<ul style="list-style-type: none"> <li>• Loss of lean tissue mass, with a weight loss &gt;5% of body weight in 12 months; Or</li> <li>• BMI lowers than 20, plus three of the following: decreased muscle strength, fatigue, anorexia, low fat-free mass index, increase of inflammation markers such as CRP or IL-6, anemia, and low serum albumin.</li> </ul>
Sarcopenic Obesity	An extreme situation that combines high muscle loss with increased fat mass and normal or high BMI.

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tory bowel disease, and undifferentiated spondyloarthritis. These conditions can also involve peripheral joints, such as hips, knees, and shoulders, as well as extra-articular manifestations affecting the eyes, skin, and gastrointestinal system. The exact cause of spondyloarthritis remains unclear, but genetic factors, particularly the HLA-B27 gene, are known to play a significant role in its development. Management of SpA typically involves a combination of medication, physical therapy, and exercise to alleviate symptoms, reduce inflammation, and maintain function and quality of life [8,9].

## LITERATURE REVIEW

This review aims to provide an overview of the current understanding of sarcopenia in SpA, focusing on its prevalence, pathogenesis, and clinical implications.

## Prevalence of sarcopenia in spondyloarthritis

The prevalence of sarcopenia in SpA patients is reported to be higher than in age-matched healthy controls [10] (Table 2). However, sarcopenia is commonly overlooked and undertreated in mainstream practice, likely due to the complexity of determining what variables to measure, how to measure them, what cut-off points best guide diagnosis and treatment, and how to best evaluate effects of therapeutic interventions.

Several definitions of sarcopenia have been proposed. The most widely used definition is from the European Working Group on Sarcopenia in Older People (EWGSOP), first published in 2010 [11] and then revised in 2019 [12]. All of sarcopenia definitions include criteria for low muscle quantity, assessed by low muscle

**Table 2:** Main studies assessing sarcopenia in patients with spondyloarthritis.

Author	N	Sex M (%)	Study design	Study population	Prevalence of sarcopenia	Definition of sarcopenia	Muscle mass assessment	Muscle strength assessment	Muscle performance assessment	Conclusion
Marcora S [19]	19	19 (100)	Case control	Long-standing AS	12% reduction in arms and legs lean mass	Comparison to a control group	DXA	Isokinetic knee extension and grip strength	30 s arm curl and chair sit-to-stand tests	Cachexia is a functionally relevant systemic complication of AS, particularly in patients with long-standing disease and radiological changes.
Ibanez VS [20]	70	42 (60)	Cohort	AxSpA anti-TNF naive	Lower FFMI than the reference (37 <sup>th</sup> percentile)	Comparison to a reference population	DXA	-	-	ASDAS CRP >3.5 was related to lower fat free mass content
Roren NK [36]	10	10 (100)	Case-control	AS	Lower appendicular LBM	Comparison to a control group	DXA	Musculus quadriceps femoris strength	-	A significantly lower appendicular LBM, muscle fiber type II CSA, and muscle strength in SpA patients compared to healthy controls.
El Maghraoui A [21]	67	67 (100)	Case-control	AS	34.50%	EWGSOP definition	DXA	Grip strength	Timed Get-Up-And-Go test	Sarcopenia and cachexia were significant associated with higher disease activity (Bath Ankylosing Spondylitis Disease Activity Index [BASDAI]) and lower BMD
Barone M [22]	22	14 (63)	Cohort	AS	20%	EWGSOP	BIA	Grip strength	-	Age ≥60 years and the presence of a disability were associated with a significantly increased risk of sarcopenia
Kim SC [17]	30	30 (100)	Cohort	AS	No significant reduction in skeletal muscle mass	Comparison to a reference population	BIA	Grip strength	-	No significant reduction in skeletal muscle mass in patients with AS compared to the general population.
Younis M [29]	50	47 (94)	Cohort	AS	10%	EWGSOP2	DXA	Grip strength	Gait speed	Lower BMI and longer disease duration increase the risk of sarcopenia, while differences in gender, smoking and the use of anti-TNF do not influence the risk.
Merle B [27]	103	51(49)	Case-control	SpA	21%	EWGOP 2	DXA	Grip strenght	Gait speed	Probable sarcopenia was associated with higher inflammation and disease activity, impaired muscle performance, and quality of life. These results suggest that muscle strength may be a salient hallmark in SpA.

Neto A [28]	27	18 (67)	Case control	AxSpA	0	EWGOP2	BIA	Isometric muscle strength of trunk, upper and lower limbs	Gait speed	Young axSpA patients with a relatively short disease duration presented similar segmental muscle physical properties as the controls and had no sarcopenia.
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mass using various imaging modalities, and low muscle quality, evaluated by diminished strength and function. Functional testing is commonly measured by gait speed and/or grip strength.

### Muscle mass assessment

Assessment of muscle mass can be done using several techniques such as anthropometry, bioimpedance analysis (BIA), dual energy X-ray absorptiometry (DXA), ultrasound, computed tomography (CT) scan and magnetic resonance imaging (MRI) [13,14]. Although CT and MRI are considered as gold standards for estimating muscle mass in research, DXA is the preferred alternative method for research and clinical use for body composition assessment. BIA is a useful technique for assessing body composition both in healthy individuals and in patients with chronic conditions that do not show major disturbances of their water distribution. This method has seen widespread use due to its simplicity and low cost and the results obtained with BIA strongly correlate with the data obtained with DXA [15].

A small number of studies have used BIA to assess the body composition of AS patients. A study revealed that AS patients and controls had similar amounts of total body water and fat-free mass index [16]. Another study by Kim, et al. [17] in 30 young patients with AS (mean age 30 years) found no significant reduction in muscle mass assessed using BIA in patients with AS compared to the general population.

DXA is a simple, non-invasive technique with a little amount of radiation that has a variety of clinical uses in this area. Lean mass can be distinguished from bone and fat mass using distinct X-ray tissue attenuations [18]. Because to its inexpensive cost and low radiation exposure, DXA is the method that has been employed the most frequently in published studies. The advantages of DXA compared with other methods such as CT or MRI are the lower radiation dose and the lower costs 5. However, DXA measures muscle mass but not quality, in contrast to CT and MRI. Appendicular lean mass, which is the total of lean mass in both the lower and upper extremities, is the muscle metric for sarcopenia that is most frequently employed with DXA.

Marcora, et al. [19] showed in AS patients with a short disease duration (8–11 years) and mild structural changes at the spine ( $\leq 1$  syndesmophyte) a non-significant reduction on the LM compared to controls ( $\pm 3$ kg; 4.5–5.8%). On the other hand, a significant reduction in LM ( $\pm 6$  kg; 12%) was found in long-standing AS patients (mean disease duration of 19 years) with severe radiological changes ( $\geq 1$  syndesmophytes in 84% of the patients). Moreover, they showed a correlation between a decline in LM and disease activity levels. A cross-sectional by Ibáñez, et al. [20] observed a decrease in muscle mass (and fat mass) associated with disease activity in male patients with AS, but not in women with AS. In both men and women, disease activity [Ankylosing Spondylitis Disease Activity Score (ASDAS) CRP] correlated negatively with fat mass. In a cross-sectional study of 10 patients with AS and 10 healthy controls, Røren, et al. observed significantly lower appendicular

lean body mass (but no total mass), lower muscle strength, and a reduced number of type II muscle fibres in patients with AS. In a cross-sectional study, which included 67 males with AS and 67 healthy controls, we observed lower muscle mass in patients with AS [21]. Prevalence of pre-sarcopenia, sarcopenia (defined by EWGSOP), and cachexia in patients with AS were 50.4%, 34.3%, and 11.9%, respectively. Sarcopenia and cachexia were significant associated with higher disease activity (Bath Ankylosing Spondylitis Disease Activity Index [BASDAI]) and lower BMD. Finally, in a cross-sectional study, Barone, et al. [22] also observed a high prevalence of sarcopenia among patients with AS, around 20%.

It would be hypothesized that TNF inhibitors (TNFi) would alleviate or prevent muscle loss in AS given the putative role of TNF in the disease process. Unfortunately, there are just a few studies looking into this matter, and the information that is currently available is not conclusive. Among AS patients receiving TNFi treatment or not, we have not discovered any appreciable variations in muscle size, strength, or functionality. Interestingly, patients taking TNFi have significantly higher FM and fat mass index (FMI) values [21]. A 2-year prospective study in TNFi-treated SpA patients documented an increase in body weight at 1 year and 2 years of treatment, mostly due to a gain in FM but also due to a significant increase in LM and in bone mineral density (BMD) [23]. These findings open up new directions for future studies in SpA patients to elucidate the impact of medications like TNFi or other therapies on muscle and body composition.

In its 2018 definition, EWGSOP2 [12] 0055ses low muscle strength as the primary parameter of sarcopenia; muscle strength is presently the most reliable measure of muscle function. To summarize, sarcopenia is probable when low muscle strength is detected, but the diagnosis of sarcopenia is confirmed by the presence of low muscle quantity or quality. When low muscle strength, low muscle quantity/quality and low physical performance are all detected, sarcopenia is considered severe. Muscle strength can be measured reliably using the handheld dynamometer in upper extremities while physical performance can be assessed using several tests such as the gait speed, the Timed Get-Up-and-Go and the Short Physical Performance Battery. The latter includes standing balance, gait speed, and chair rises (sit-to-stand).

### Patient population and data collection

A retrospective chart review was conducted of older ( $\geq 80$  years of age) patients, presenting to the ED, at a single level-one trauma center (Memorial Regional Hospital, Hollywood, Florida), following a fall between April 2016 and January 2017. Only 1.5% of the patients reported falling from a height (ladder or stool), the rest fell from standing, sitting (wheelchair), or supine (bed) positions. No patients were excluded and complete two-year follow up data was available. The Memorial Healthcare System Institutional Review Board approved the study with a waiver of consent.

All study subjects underwent a standard trauma evaluation with appropriate imaging. Upon arrival to the ED, patients were

seen, evaluated, and treated by the ED staff including physicians, nurses, respiratory therapists, and pharmacists. Patients received basic x-rays of chest, pelvis, or injured extremities. Computer tomography (CT) scans and expert consultation from trauma, neurosurgery, and orthopaedic sub-specialists was based on sustained injuries and their severity. Hemodynamically unstable patients were treated (e.g. intubated, transfused) before proceeding to imaging or surgery. Injured patients were either admitted to the intensive care unit (ICU) or floor, while minimally injured were discharged upon evaluation by ED social workers. Patients were considered to be AC if they were receiving anti-factor (e.g. warfarin, apixaban) and/or anti-platelet (e.g. aspirin, clopidogrel) medications regardless of dosing or level of anticoagulation. Of note, only one patient received anti-factor medications other than warfarin, rivaroxaban, or enoxaparin.

Baseline demographics, date and location of hospital admission and discharge, co-morbidities, treatment with AC, need for transfusion or mechanical ventilation, site of traumatic injury, in- and out-of-hospital complications, including short- (in-hospital,  $\leq 30$ ,  $\leq 90$  day) and long-term mortality (90 days – 2 years), were entered into a secure, password-protected computer database. Patients marked as ‘moribund’ on discharge, were either deceased or discharged to hospice with expected short-term mortality, although not everyone died within 30 days post discharge. Of note,  $<30$  and  $<90$ -day mortality included patients who died in the hospital. Two years of long-term follow-up was used to evaluate re-admission and mortality, including deaths from any cause occurring prior to or following hospital discharge. When our electronic medical records (EMR) were insufficient, phone calls were made to home/institutions in order to acquire complete long-term data.

### Muscle strength assessment

A handgrip strength test performed with a handheld dynamometer is regarded as a trustworthy surrogate for more sophisticated measurements of arm and leg muscle strength. The method is regarded as being low cost, as dynamometers are easily accessible and simple to use. It is frequently used in both therapeutic settings and academic research. Only a few research, nevertheless, have used this instrument to quantify muscular strength in the setting of SpA. A reduction in handgrip strength was documented in AS patients in a study by Carter, et al. [24] but not in our study 21 neither In the one by Marcora, et al. [19] However, in the latter study, both knee extensor strength and all of the functional strength test scores (sit-to-stand test and arm curl test) were significantly lower in AS

patients. In patients with long-standing disease and radiological changes, the handgrip strength test correlates positively with aerobic power, and a general reduction in muscle strength has been found to be significantly associated with muscle wasting. When the measurement of grip is not possible due to hand disability (e.g., with advanced arthritis or stroke), the chair stand test (also called the sit-to-stand test) can be used as a proxy for the strength of leg muscles. This test measures not only strength but also endurance. The sit-to-stand test scores of patients with AS have been found to be significantly worse than those of healthy control subjects and correlated with appendicular lean mass [19].

### Physical performance

Physical performance has been described as a whole-body function associated with locomotion that may be objectively measured. This is a multidimensional concept that includes equilibrium as well as central and peripheral nervous system function in addition to muscles [25]. Gait speed, the Short Physical Performance Battery (SPPB), and the Timed-Up and Go test (TUG), among other measures, can all be used to measure physical performance [26]. Table 3 summarizes the EWGSOP2 cut-offs [12].

A recent study by Merle, et al. [27] using the recent EWGSOP2 definition showed a higher prevalence of sarcopenia in SpA patients compared to controls. A total of 103 patients (51% women) with SpA, mean age  $47.1 \pm 13.7$  years, were included and compared to 103 age- and sex-matched controls. Twenty-two SpA patients (21%) versus 7 controls (7%) had a low grip strength, i.e., probable sarcopenia ( $p < 0.01$ ), 15 SpA (15%) patients and 7 controls (7%) had low Skeletal Muscle mass Index (SMI), respectively, and 5 and 2% of SpA patients and controls had low grip strength and low SMI, i.e., confirmed sarcopenia. All the sarcopenic SpA patients had a low gait speed, i.e., severe sarcopenia. Finally, probable sarcopenic SpA patients had significantly higher C-Reactive Protein and Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), lower gait speed, and SarQoL® score (a quality-of-life score) than SpA patients with normal grip strength.

In another recent study by Neto, et al. [28] in young patients with axial SpA (mean age 36.5 (SD 7.5) years, 67% males, mean disease duration 6.5 (3.2) years), no significant difference in segmental muscle stiffness, tone or elasticity, compared with the controls, despite showing a slight numerically higher lower lumbar (L3-L4) stiffness. No participants presented sarcopenia even though patients with axial SpA, compared to the healthy controls, had lower total strength, as well as lower strength in the upper and lower limbs

Table 3: EWGSOP2 sarcopenia cut-offs points.

Test	Cut-off points for men	Cut-off points for women
<b>Low strength</b>		
Grip strength	<27 kg	<16 kg
Chair stand	>15 s for five rises	
<b>Low muscle quantity</b>		
ASM	<20 kg	<15 kg
ASM/height <sup>2</sup>	<7.0 Kg/m <sup>2</sup>	<5.5 K/m <sup>2</sup>
<b>Low performance</b>		
Gait speed		$\leq 0.8 \mu/\sigma$
SPPB		$\leq 8 \text{ } \mu\text{oi}\nu\tau \text{ } \sigma\chi\text{op}\epsilon$
TUG		$\geq 20\sigma$
400m walk test		Non-completion or $\geq 6$ min for completion

ASM: Appendicular Skeletal Muscle Mass; TUG : Timed Get-Up-and-Go; SPPB: Short Physical Performance Battery

independently of muscle physical properties. Patients had also significantly lower gait speed adjusted for muscle mass, strength and muscle physical properties.

Younis, et al. [29] in a cross-sectional study from Iraq studied 50 patients with AS. The prevalence of presarcopenia was 6%, while sarcopenia was documented in 10% of patients. The vast majority of the sarcopenic group (80%) were physically inactive which was statistically significant compared with the non-sarcopenic group. No significant association of treatment with anti-TNF or its duration was found between sarcopenia and non-sarcopenia groups. Both lean mass (LM) and handgrip showed fair validity to differentiate between AS patients. Lower BMI and longer disease duration increased the risk of sarcopenia, while differences in gender, smoking and the use of anti-TNF did not influence the risk.

### Thoracic sarcopenia in ankylosis spondylitis

Ankylosing spondylitis (AS) is a condition that affects the axial skeleton and dorsal kyphosis is its hallmark. The role of sarcopenia in the progression of kyphotic deformities in AS is of particular interest [30]. Multiple mechanisms have been proposed. In AS patients, when paraspinal muscle atrophy, the psoas muscles typically maintain their volumes [31]. Due to restricted lumbar mobility, the biomechanical imbalance between flexors and extensors may cause disuse atrophy. It has been proposed that a possible explanation may be the denervation atrophy brought on by the osteophytic bone trapping of dorsal nerve roots. Last but not least, paraspinal muscles in AS patients have unevenly distributed collagen buildup on a histological level [32]. These elements taken together may provide an explanation for how sarcopenic paraspinal muscles affect spinal kyphosis in AS patients. One study followed paraspinal muscle volumes on MRI in patients with AS [31]. Patients with spinal deformity had lower paraspinal muscle volumes compared to AS patients without spinal deformity. The latter cohort showed lower paraspinal muscle volumes compared to non-AS patients with chronic back pain. These findings suggest that lower paraspinal muscle volumes are seen in early stages of AS, before the kyphotic deformity is observed. In addition to decreased muscle volumes, increased fatty degeneration of the paraspinal muscles has also been reported in patients with AS compared to those with non-radiographic axial SpA [33].

### Pathogenesis of sarcopenia in spondyloarthritis

The pathogenesis of sarcopenia in SpA is complex and multifactorial, involving both systemic inflammation and local factors. Key mechanisms include:

- Chronic inflammation: Pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF- $\alpha$ ) and interleukin-6 (IL-6), are elevated in SpA patients and contribute to muscle catabolism and protein degradation, leading to sarcopenia.
- Reduced physical activity: Pain, stiffness, and fatigue in SpA patients can limit physical activity, resulting in muscle disuse atrophy.
- Nutritional deficiencies: Malabsorption, decreased appetite, and altered metabolism due to inflammation can contribute to inadequate nutrient intake and muscle wasting in SpA patients.
- Hormonal changes: SpA patients may have alterations in insulin-like growth factor-1 (IGF-1), testosterone, and cortisol levels, which can influence muscle mass and strength.

### Clinical implications of sarcopenia in spondyloarthritis

Sarcopenia in SpA patients is associated with several adverse outcomes, including increased functional impairment (sarcopenic SpA patients show worse functional status and higher disability scores compared to non-sarcopenic patients); reduced quality of life (sarcopenia is associated with lower health-related quality of life in SpA patients); and increased risk of falls and fractures (the loss of muscle mass and strength in sarcopenia can lead to impaired balance, increased risk of falls, and subsequently, fractures).

However, despite its high prevalence and potential consequences, it is usually under-diagnosed. As the muscle mass and muscle strength in younger individuals is high before it is affected by this disorder, secondary muscle mass and muscle strength loss is usually thought to be functionally less relevant. However, some studies suggest that assessing rheumatic patients for sarcopenia is important because it may help identify patients with a poorer prognosis as it has been associated with progression of the disease, more frequent systemic symptoms, and more rapid decline in physical function.

### Management of sarcopenia in spondyloarthritis

Early identification and management of sarcopenia in SpA patients are crucial to minimize its impact on clinical outcomes. Management strategies may include:

- Physical activity: regular aerobic and resistance exercise can help preserve muscle mass, strength, and function in SpA patients [34];
- Nutritional interventions: Adequate protein intake, calcium and vitamin D supplementation, and the adoption of an anti-inflammatory diet [35].
- Pharmacological interventions: controlling inflammation through the use of nonsteroidal anti-inflammatory drugs (NSAIDs), disease-modifying antirheumatic drugs (DMARDs), and biologic agents when needed may help alleviate sarcopenia in SpA patients [36].

### DISCUSSION AND CONCLUSION

Sarcopenia is a prevalent and clinically significant comorbidity in spondyloarthritis patients. Understanding its pathogenesis and early identification can help inform targeted prevention and management strategies. Future research should focus on elucidating the molecular mechanisms underlying sarcopenia in SpA and identifying novel therapeutic targets to improve patient outcomes.

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Authors have nothing to disclose.

### CONFLICT OF INTEREST

No conflicts of interest.

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