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Low muscle mass and body composition analysis in a group of postmenopausal women affected by primary Sjögren's syndrome

Suhel Gabriele Al Khayyat, Paolo Falsetti, Edoardo Conticini, Roberto D'Alessandro, Francesca Bellisai, Stefano Gentileschi, Caterina Baldi, Marco Bardelli, Luca Cantarini, Bruno Frediani

Department of Medicine, Surgery and Neurosciences, Rheumatology Unit, University of Siena, Italy

Abstract

Objectives: Sarcopenia is the pathological reduction of skeletal muscle mass and strength. This condition is often underestimated in clinical practice, particularly in connective tissue diseases. The purpose of this study is to evaluate the prevalence of low muscle mass in primary Sjögren's syndrome (pSS) and to explore the relationships linking muscles and bone tissue.

Material and methods: Twenty-eight postmenopausal pSS patients were matched with 30 healthy controls and their body composition analysis was performed by dual-energy X-ray absorptiometry to investigate for sarcopenia considering appendicular lean mass (ALM) and the skeletal muscle mass index (SMI) as references. Bone mineral density analysis of lumbar spine (L1–L4), whole femur, femoral neck and whole body was also performed. Linear regression was used to assess the relationship between body composition and bone mineralization.

Results: Low muscle mass was significantly higher in the pSS group compared to controls whether expressed as ALM, SMI [odds ratio (OR) = 18.40, confidence interval (CI): 4.84–72.08, $p < 0.0001$] or considering total body lean masses. Lean masses appeared to be the best estimators of bone mineralization: total lean body mass (TLBM) lumbar spine $R^2 = 0.72$, $p < 0.0001$; TLBM femoral neck $R^2 = 0.36$, $p < 0.004$; lean mass of upper limbs lumbar spine $R^2 = 0.70$, $p < 0.0001$; femoral neck $R^2 = 0.66$; lean mass of lower limbs lumbar spine $R^2 = 0.66$, $p < 0.0001$; femoral neck $R^2 = 0.44$, $p = 0.008$). Primary Sjögren's syndrome patients had a significantly higher android/gynoid fat ratio compared to controls.

Conclusions: Female pSS patients have lower muscle mass compared to healthy controls and are exposed to a higher risk of developing sarcopenia than healthy subjects. Our research demonstrates that the amount of lean tissue is the main predictor of bone mineralization in pSS.

Key words: bone mineral density, osteoporosis, sarcopenia, primary Sjögren's syndrome.

Introduction

Sarcopenia is a clinical condition defined by the European Consensus Group [1] as the reduction of skeletal muscle mass, associated with poor physical performance measured as low grip strength and gait speed. Low muscle mass represents a transversal problem in medicine, mainly affecting geriatric patients simultaneously suffering from many disorders such as chronic obstructive pulmonary disease (COPD), chronic heart failure and neurodegenerative diseases, defining the classic phenotype of the complex patient.

In recent decades, scientific attention has gradually increased towards sarcopenia in relation to several rheumatological conditions: the most robust evidence has been reported for rheumatoid arthritis (RA) [2] and systemic sclerosis [3], while contraddictory evidences have been reported for spondyloarthropaties (SA) [4].

Address for correspondence:
Suhel Gabriele Al Khayyat, Department of Medical Sciences, Surgery and Neurosciences, Rheumatology Unit, University of Siena, Azienda Ospedaliero-Universitaria Senese, Viale Bracci 16, 53100 Siena, Italy, e-mail: alkhayyatsuhelg@gmail.com
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A high inflammatory load, partly supported by the rheumatological diseases themselves and assisted by an obesity-prone body composition, has been linked to higher prevalence of skeletal muscle loss [5].

No data exist about the prevalence of sarcopenia in patients affected by primary Sjögren's syndrome (pSS): in this regard, we aimed to assess whether patients suffering from this condition may display a reduced muscle mass and whether any correlation exists between body composition and bone mineral density.

Material and methods

Study population

The authors retrospectively evaluated body composition and bone mineral density (BMD) of 28 postmenopausal women diagnosed in our department with pSS according to American College of Rheumatology/European League against Rheumatisms (ACR/EULAR) 2016 classification criteria [6].

The authors considered only women whose densitometry analysis was performed within 6.5 months (SD ±9.1) from pSS diagnosis. The exclusion criterion was the presence of any demineralizing condition, such as heparin therapy, corticosteroid use, COPD, chronic heart failure, malignancies, or cachexia.

Control group

Thirty postmenopausal women who had been evaluated with dual-energy X-ray absorptiometry (DXA) in our department for age-related issues, matched for age and gender, were randomly chosen from our records. None of them reported a major condition unbalancing bone homeostasis.

Methods

Detailed medical history, weight, height, and BMI were collected from patients and their sera were tested for autoimmunity with standard techniques: anti-nucleus antibodies (ANA) were detected by indirect immunofluorescence on HEp-2 cells (Euroimmun, Lübeck, Germany), anti-extractable nuclear antigens (ENA) using fluorescent enzyme immunoassay (FEIA – Thermo Fisher).

Minor salivary glands biopsy was performed according to Guevara-Gutiérrez technique [7] and samples were evaluated using the focus score (FS) and the Chisholm and Mason (CM) grading system. Samples were considered positive for pSS when FS ≥ 1 and CM ≥ 3. Bone mineral density and body composition were assessed with DXA using a Lunar Expert version 1.72®.

According to the manufacturer, before each examination routine quality control (QC) was performed. The following regions were evaluated:

- whole body,
- whole left femur,
- neck left femur,
- lumbar spine (L1–L4).

Bone mineral density was expressed in g/cm^2. A diagnosis of osteoporosis was made when the *t*-score of patients deviated below –2.5 standard deviations (SD) compared with the reference population according to the World Health Organization (WHO) criteria.

Body composition of pSS patients and controls was compared by fat and lean masses of total body, upper and lower limb regions. Appendicular lean mass (ALM, kg/m^2) was calculated as the sum of fat-free mass minus bone mineral content of lower and upper limbs, skeletal muscle mass index (SMI, kg/m^2) was calculated as ALM divided by height squared according to Baumgartner's criteria [8], fat mass index FMI (kg/m^2) as total fat mass divided by height squared, and fat-free mass index (FFMI kg/m^2) as fat-free tissue divided by height squared. A low muscle mass was identified for women when it was found to be 2 SD below the mean of young adults (women: < 5.5 kg/m^2) [8].

This study was conducted retrospectively and in accordance with the Declaration of Helsinki, all patients provided written consent to undergo clinical routine examination.

Statistical analysis

Statistical analysis was performed using GraphPad Prism version 8.4.2 for Windows, GraphPad Software, San Diego, California USA, www.graphpad.com. Mean total lean mass (kg), ALM (kg/m^2), skeletal muscle mass index (SMI, kg/m^2), fat-free tissue (kg), fat-free mass index (FFMI, kg/m^2), total fat mass (kg), body fat percentage (BF, %), fat mass index (FMI, kg/m^2), and total skeletal muscle mass (kg), calculated according to Kim et al. [9] were compared between pSS and controls using the *t*-test for parametric data or the Mann-Whitney *U* test.

Subsequently, Fisher's exact test was carried out to establish sarcopenia's prevalence in the studied population. Pearson's and Spearman's tests were used to evidence any correlation between the considered variables of body composition and BMD of the studied regions.

A bivariate regression analysis was conducted using BMD of the lumbar spine and femoral neck as the dependent variables, while total skeletal muscle mass, total lean mass, SMI, total fat mass, total body fat percentage, upper lean mass, lower limbs lean mass, upper limbs fat mass, lower limbs fat mass, upper limbs fat free mass, lower limbs fat free mass, android fat percentage, gynoid

fat percentage, A/G ratio, BMI, weight, FFMI and FMI were identified as the independent variables.

A multivariate regression analysis was performed to identify the best estimators of BMD considering age as an exogenous variable and introducing it in a stepwise manner. Lastly, we checked which model acted as the best predictor using the extra sum-of-squares *f*-test.

Results

Epidemiological features and body composition characteristics of the studied populations are summarized in Table I.

Statistically significant differences were found between pSS and the control group for ALM, SMI, total skeletal muscle mass and android/gynoid fat distribution. Appendicular lean mass and SMI were significantly lower in pSS compared to the control group, as well as total skeletal muscle mass.

No difference in postmenopausal age was evidenced between cases and controls. Conversely, A/G ratio was identified as significantly higher in our patients.

The Mann-Whitney *U* test or Student's *t*-test was used to compare appendicular lean mass, SMI and total skeletal muscle mass, evidencing a statistically significant lower muscle mass in pSS patients compared to the control group. Subsequently Fisher's exact test evidenced that 80% of pSS patients had a low muscle mass expressed as SMI < 5.5 kg/m^2 compared to healthy individuals (20% of low muscle mass) [odds ratio (OR) = 18.40 confidence interval (CI): 4.84–72.08, $p < 0.0001$].

Significant correlations between body composition variables and bone mineral density were found in pSS patients: higher correlations were identified for lean masses considered as total body (TLBM), lean mass of upper limbs (LMUL) and lean mass of lower limbs (LMLL).

Fat-free masses, which are the lean masses plus bone mineral content, displayed a high correlation with bone mineral densities of nearly every studied region. Appendicular lean mass and SMI showed a correlation only for femoral BMDs. Weight and BMI were highly correlated with bone mineralization, although the latter only with femoral BMDs. Fat masses did not display any correlation with BMD. A/G ratio correlated with lumbar spine and whole-body BMDs (Table II).

From the bivariate regression analysis, we concluded that lean masses had higher goodness of fit as bone mineral density predictors; although other variables, particularly weight and BMI, acted in a statistically significant fashion as feasible estimators, their R^2 did not reach significant values. Appendicular lean mass and SMI acted as poor predictors of femoral BMD and they did not reach statistical significance as lumbar spine BMD estimators (Table III).

Table I. Epidemiological and body composition features of primary Sjögren's syndrome patients and controls

Parameter	pSS patients	Controls
Number	28	30
Gender	F	F
Age	64.31 ±9.7	59.90 ±3.8
Weight [kg]	65.81 ±12.6	67.38 ±16.9
Height [cm]	162.5 ±7.43	162.3 ±6.03
Body mass index [BMI, kg/m^2]	24.86 ±4.1	25.03 ±6.1
Postmenopausal age	51.09 ±3.19	50.22 ±3.04
Body composition analysis		
Total lean body mass [TLBM, kg]	38.88 ±6.73	39.10 ±4.83
Body fat [BF, %]	36.91 ±8.68	39.35 ±8.99
Appendicular lean mass [ALM, kg/m^2]	**11.87 ±3.42****	**16.84 ±2.96****
Skeletal muscle mass index [SMI, kg/m^2]	**4.47 ±1.18****	**6.38 ±0.96****
Fat-free tissue [kg]	39.92 ±6.66	42.17 ±5.51
Fat-free mass index [FFMI, kg/m^2]	14.58 ±3.33	15.65 ±1.60
Total fat mass [kg]	23.99 ±9.82	26.98 ±13.12
Body fat percentage [BF, %]	36.91 ±8.68	39.35 ±8.99
Fat mass index [FMI, kg/m^2]	9.07 ±3.53	10.31 ±4.81
Total skeletal muscle mass [kg]	**13.10 ±3.94****	**16.76 ±3.29****
Android fat [%]	4.61 ±11.58	43.48 ±43.88
Gynoid fat [%]	41.91 ±7.25	43.91 ±7.31
Android/gynoid fat ratio [A/G ratio]	**1.8 ±0.47****	**1 ±0.39****

*pSS – primary Sjögren's syndrome, **p < 0.005.*

Subsequently, a multivariate model including age as an independent variable was built and a comparison between the two regression models was made using the extra sum-of-squares *f*-test, concluding that the multivariate model including age acted as the best BMD predictor for every studied region ($p < 0.05$; the null hypothesis where H0 = the simpler model is correct was rejected).

Age always acted as negatively related to bone mineral densities. Absolute *t*-values for lean masses of body composition were higher than those of the age variable, suggesting a greater impact of lean masses in BMD prediction compared to age (Table IV).

Table II. Body mass composition correlations with bone mineral density of studied regions among our cohort of primary Sjögren's syndrome patients

Parameter	Whole femur BMD	Femoral neck BMD	Lumbar spine BMD	Whole body BMD
Whole body BF [%]	0.29	0.09	0.18	0.10
Total mass whole body [kg]	**0.48***	**0.54****	**0.56****	**0.62****
Fat [kg]	0.32	0.24	0.42	0.32
Lean total body [kg]	**0.51****	**0.46***	**0.64****	**0.51****
Fat free tissue total body [kg]	**0.42***	**0.46***	**0.63****	**0.63****
Android BF [%]	**0.50***	**0.36**	**0.35**	**0.22**
Gynoid BF [%]	0	–0.13	–0.17	–0.12
Torso BF [%]	**0.48***	**0.45***	**0.26**	**0.21**
Total mass torso [kg]	0.21	0.33	0.38	**0.49****
Fat torso [kg]	0.37	0.32	0.37	**0.42***
Lean torso [kg]	0.14	0.35	**0.53***	**0.50****
Fat free tissue torso [kg]	0.16	0.26	**0.59****	**0.42***
Lower limbs total mass [kg]	0.37	**0.55****	0.36	**0.46***
Lower limbs BF [%]	0.28	0.14	0.02	–0.12
Fat lower limbs [kg]	0.18	0.06	0.15	0.13
Lean lower limbs [kg]	0.38	**0.52****	**0.58****	0.58
Fat free tissue lower limbs [kg]	0.39	**0.53****	**0.59****	**0.60****
Upper limbs BF [%]	0.06	–0.11	0.09	0.01
Upper limbs total mass [kg]	**0.43***	**0.50****	0.26	0.03
Fat upper limbs [kg]	0.32	0.14	0.21	0.33
Lean upper limbs [kg]	**0.60****	**0.62****	**0.69****	**0.59****
Fat-free tissue upper limbs [kg]	**0.63****	**0.63****	**0.52***	**0.74****
ALM [kg/m^2]	**0.54****	**0.53****	0.25	0.39
SMI [kg/m^2]	**0.51****	**0.47***	0.37	0.29
FMI [kg/m^2]	0.28	**0.47***	0.35	0.22
FFMI [kg/m^2]	0.50*	**0.51****	0.22	**0.46***
A/G ratio	0.19	0.33	**0.60****	**0.55****
TSMM	**0.57***	**0.55****	**0.46***	**0.40***
BMI [kg/m^2]	**0.40***	**0.43***	**0.41 (p = 0.06)**	**0.32**
Weight (kg)	**0.42***	**0.48***	**0.53***	**0.48***

*A/G ratio – android/gynoid fat ratio, ALM – appendicular lean mass, BMD – bone mineral density, BMI – body mass index, FFMI – fat-free mass index, FMI – fat mass index, SMI – skeletal muscle mass index, TSMM – total skeletal muscle mass; *$p < 0.05$, **$p < 0.005$.*

Discussion

Primary Sjögren's syndrome is a chronic inflammatory autoimmune connective tissue disease of unknown etiology which is characterized by CD4+ T-helper, B-cell, macrophage, and dendritic cell infiltration of target organs, in which the dysfunction of the immune and cytokine system plays a pivotal role in supporting disease activity.

Many rheumatological diseases characterized by a high inflammatory load have been linked to a reduction of BMD and lean masses. To date, no study has investigated the association between low muscle mass and pSS.

In our patients we documented a higher frequency of low SMI. We could speculate that classical risk factors for sarcopenia such as reduced physical activity [10], hormonal imbalance, high disease burden, altered body composition, inflammation [11], low vitamin D levels [12] and insulin resistance [13] are involved in reducing lean mass in pSS patients, alongside an altered

Table III. Results of multiple simple linear bivariate regression of body mass composition analysis conducted by DXA as feasible predictors of bone mineral density of lumbar spine and femoral neck

X Independent variable	Y Dependent variable	Equation	*p*-value	r^2	Std. err. of the Y-intercept	Number of observed values
Lean total body mass [kg]	Lumbar spine BMD [g/cm^2]	Y = 0.03235*X – 0.1980	0.0002	0.52	0.2644	28
Lean total body mass [kg]	Femoral neck BMD [g/cm^2]	Y = 0.009076*X + 0.5052	0.035	0.17	0.1561	28
Total body fat [%]	Lumbar spine BMD [g/cm^2]	Y = 0.003605*X + 0.8602	0.4191	0.03	0.1680	28
Total body fat [%]	Femoral neck BMD [g/cm^2]	Y = 0.005983*X + 0.6373	0.0697	0.12	0.1197	28
Total body fat mass [kg]	Lumbar spine BMD [g/cm^2]	Y = 0.007294*X + 0.8231	0.054 ns	0.18	0.2644	28
Total body fat mass [kg]	Femoral neck BMD [g/cm^2]	Y = 0.007947*X + 0.6674	0.005	0.27	0.06704	28
SMI [kg/m^2]	Lumbar spine BMD [g/cm^2]	Y = 0.04188*X + 0.8126	0.0932 ns	0.14	0.1098	28
SMI [kg/m^2]	Femoral neck BMD [g/cm^2]	Y = 0.04818*X + 0.6506	0.0131	0.22	0.08185	28
ALM [kg/m^2]	Lumbar spine BMD [g/cm^2]	Y = 0.02117*X + 0.7451	0.06 ns	0.19	0.1278	28
ALM [kg/m^2]	Femoral neck BMD [g/cm^2]	Y = 0.02643*X + 0.5443	0.001	0.36	0.08185	28
TSMM	Lumbar spine BMD [g/cm2]	Y = 0.01967*X + 0.7373	0.003	0.21	0.1246	28
TSMM	Femoral neck BMD [g/cm^2]	Y = 0.02391*X + 0.5451	0.0008	0.38	0.08458	28
FMI [kg/m^2]	Lumbar spine BMD [g/cm^2]	Y = 0.01672*X + 0.8453	0.1162	0.12	0.09854	28
FMI [kg/m^2]	Femoral neck BMD [g/cm^2)	Y = 0.01966*X + 0.6797	0.0125	0.22	0.07117	28
FFMI [kg/m^2]	Lumbar spine BMD [g/cm^2]	Y = 0.04188*X + 0.8126	0.0932 ns	0.14	0.1098	28
FFMI [kg/m^2]	Femoral neck BMD [g/cm^2]	Y = 0.04818*X + 0.6506	0.0131	0.22	0.08185	28
Total body fat-free tissue [kg]	Lumbar spine BMD [g/cm^2]	Y = 0.02817*X – 0.1073	0.001	0.44	0.2882	28
Total body fat-free tissue [kg]	Femoral neck BMD [g/cm^2]	Y = 0.01131*X + 0.3956	0.004	0.28	0.1471	28
Lean mass upper limbs [kg]	Lumbar spine BMD [g/cm^2]	Y = 0.1506*X + 0.3733	0.0007	0.48	0.1557	28
Lean mass upper limbs (kg)	Femoral neck BMD [g/cm^2]	Y = 0.1161*X + 0.3592	0.0001	0.52	0.09894	28
Lean mass lower limbs [kg]	Lumbar spine BMD [g/cm^2]	Y = 0.07724*X + 0.1056	0.0006	0.46	0.2188	28
Lean mass lower limbs [kg]	Femoral neck BMD [g/cm^2]	Y = 0.03545*X + 0.4358	0.0020	0.32	0.1246	28
Fat free tissue upper limbs [kg]	Lumbar spine BMD [g/cm^2]	Y = 0.1487*X + 0.3348	0.0002	0.51	0.1485	28
Fat free tissue upper limbs [kg]	Femoral neck BMD [g/cm^2]	Y = 0.1090*X + 0.3590	< 0.0001	0.52	0.09629	28

Table III. Cont.

X Independent variable	Y Dependent variable	Equation	*p*-value	r^2	Std. err. of the Y-intercept	Number of observed values
Fat free tissue lower limbs [kg]	Lumbar spine BMD [g/cm^2]	Y = 0.07304*X + 0.09543	0.0004	0.49	0.2094	28
Fat free tissue lower limbs [kg]	Femoral neck BMD [g/cm^2]	Y = 0.03436*X + 0.4204	0.0013	0.34	0.1227	28
Android fat [%]	Lumbar spine BMD [g/cm^2]	Y = 0.002914*X + 0.8788	0.3632 ns	0.04	0.1309	28
Android fat [%]	Femoral neck BMD [g/cm^2]	Y = 0.005888*X + 0.6190	0.0145	0.21	0.09462	28
Gynoid fat [%]	Lumbar spine BMD [g/cm^2]	Y = 0.004454*X + 1.188	0.5433 ns	0.01	0.3147	28
Gynoid fat [%]	Femoral neck BMD [g/cm^2]	Y = 0.0005586*X + 0.8815	0.8911 ns	0.00	0.1718	28
A/G ratio	Lumbar spine BMD [g/cm^2]	Y = 0.1838*X + 0.6658	0.0827 ns	0.15	0.1836	28
A/G ratio	Femoral neck BMD [g/cm^2]	Y = 0.08419*X + 0.7015	0.1694 ns	0.07	0.1142	28
BMI (kg/m^2)	Lumbar spine BMD [g/cm^2]	Y = 0.01844*X + 0.5447	0.06	0.17	0.2287	28
BMI (kg/m^2)	Femoral neck BMD [g/cm^2]	Y = 0.01572*X + 0.4672	0.02	0.19	0.1634	28
Weight (kg)	Lumbar spine BMD [g/cm^2]	Y = 0.007824*X + 0.4931	0.01	0.28	0.1860	28
Weight (kg)	Femoral neck BMD [g/cm^2]	Y = 0.005803*X + 0.5228	0.02	0.23	0.1384	28

*A/G ratio – android/gynoid fat ratio, ALM – appendicular lean mass, BMD – bone mineral density, BMI – body mass index, FFMI – fat-free mass index, FMI – fat mass index, SMI – skeletal muscle mass index, TSMM – total skeletal muscle mass; ns – not significant,*p < 0.05.*

gut microbiota, an impaired nutritional status [14] and altered glandular functions [15], which may occur in Sjögren's disease.

Indeed, many of our patients displayed low vitamin D levels (data not shown), and this could partially explain the reduction of lean tissue in pSS. Phenotypical changes may reflect pathological states that act simultaneously on bone, muscle and fat tissue where the bone-muscle unit cross-talk is bidirectional and one tissue influences the other [16].

The fat mass acts as an endocrinological tissue that may influence BMD, muscles, and body composition by secreting adipokines: the most studied are leptin and adiponectin; the former is known as an appetite suppressor and, even if its role in bone is not thoroughly defined yet [17], it seems to enhance energy consumption in skeletal muscle [18]. The latter has been negatively correlated with BMD [17], while its deficiency was linked to lower insulin sensitivity of muscle tissue [19].

The results derived by body composition analysis evidenced that, as already reported in the literature [20], lean tissues may act as good predictors of BMD, while a similar association was not found for BMI and patients' weight.

Although lean masses carried higher absolute *t*-values, adding age as an independent variable significantly ameliorated our models' prediction capabilities, allowing us to hypothesize that muscle tissue have a greater influence on BMD than BMI or weight and to speculate that, independently from age, physical activity may act as the best non-pharmacological measure against muscle and bone loss, in accordance with what is reported [21].

Moreover, 6 out of our patients (21%) could be classified in the osteosarcopenic-obesity phenotype, i.e. BF% > 40% for women [22], BMD (expressed as *t*-score at any site) lower than –1 SD in accordance with the WHO definition and SMI < 5.45 kg/m^2 [23].

Though an increase in fracture risk is described for pSS [24], lean mass tissues have not been considered in these studies, and as suggested by Yu et al. [25], combining the presence of sarcopenia with FRAX could improve fracture risk prediction. The weight of low muscle mass in rheumatological diseases as a predictor of fractures has yet to be fully investigated.

Table IV. Results of the multivariate regression analysis showing the lean masses and age estimators of lumbar spine and femoral neck bone mineral density (g/cm^2)

X1	X2	Y	β0	β1	β2	*p*-value F	r^2	Std. err. β1	Std. err. β2	|t| β1	|t| β2	*n*
TLBM [kg]	Age (y)	Lumbar spine BMD [g/cm^2]	0.344	0.033	–0.0093	< 0.0001	0.72	0.005	0.002	4.5	2.0	28
TLBM [kg]	Age (y)	Femoral neck BMD [g/cm^2]	0.807	0.012	–0.007	0.0046	0.36	0.003	0.002	0.0	0.0	28
LMUL [kg]	Age (y)	Lumbar spine BMD [g/cm^2]	0.948	0.153	–0.0095	< 0.0001	0.70	0.028	0.002	4.5	2.0	28
LMUL [kg]	Age (y)	Femoral neck BMD [g/cm^2]	0.690	0.126	–0.0058	< 0.0001	0.66	0.019	0.001	5.1	1.2	28
LMLL [kg]	Age (y)	Lumbar spine BMD [g/cm^2]	0.647	0.078	–0.0090	< 0.0001	0.66	0.015	0.002	4.1	2.0	28
LMLL [kg]	Age (y)	Femoral neck BMD [g/cm^2]	0.734	0.039	–0.0054	0.0008	0.44	0.009	0.002	3.4	1.2	28
TSMM	Age (y)	Lumbar spine BMD [g/cm^2]	1.324	0.021	–0.0098	0.0076	0.00	0.007	0.003	2.2	0.0	28
TSMM	Age (y)	Femoral neck BMD [g/cm^2]	0.781	0.023	–0.0036 (ns)	0.0013	0.44	0.006	0.002	3.8	1.2	28

β0 – Y-intercept, β1/β2 – estimates of independent variables, Std. err – standard error, LTBM – lean total body mass, LMUL – lean mass of upper limbs, LMLL – lean mass of lower limbs, TSMM – total skeletal muscle mass, ns – not significant, |t| – t absolute value of the univariate analysis used to weight the impact of each independent variable on the model.

Altered body composition in RA has been correlated with the increase in IL-6 levels [26], insulin resistance and metabolic syndrome [27], thus partially accounting for the increased cardiovascular risk of these patients. The degree of inflammation in chronic inflammatory arthritis was directly correlated in several studies with obesity [28] and, as well as in RA, with prolongation of the QTc interval leading to malignant ventricular arrhythmias such as torsade de pointes [29].

Our patients displayed a statistically significantly higher A/G ratio compared to controls, and android fat distribution is positively correlated with a higher risk of coronary disease in the literature [30].

Except for the study of Gravani et al. [31], which reported myo-intimal thickening of the arteries of patients with pSS, no other papers correlated an altered body composition and an altered cytokine pool with an increased risk of cardiovascular events in pSS patients.

Conclusions

At the present time, muscle, fat and skeletal tissue should be no longer considered as entities functionally separated from each other but rather as a syncytium with endocrine and immunological function, given the close interconnection of these systems.

This is the first study to document a high prevalence of reduced skeletal muscle mass in postmenopausal women recently diagnosed with pSS. Such patients appear to be at least as much at risk of sarcopenic syndrome as those with RA and systemic sclerosis, albeit perhaps due to different pathophysiological mechanisms.

This paper has several limitations: the considered cohort is too small to allow generalization of the results to every postmenopausal woman affected by pSS; moreover, due to its retrospective design, it was not possible to stratify patients according to organ involvement and disease activity. Therefore, prospective studies involving larger cohorts are needed to obtain higher quality of evidence.

The authors declare no conflict of interest.

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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