

Changes in body composition and bone mineral density in postmenopausal women with psoriatic arthritis

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Abstract

Objective: Prolonged inflammation status due to psoriatic arthritis (PsA) may contribute to the loss of muscle mass, extending from muscle weakness, and increased risk of falls and fractures. The risk of fractures and their complications increases with concomitant osteoporosis.

Material and methods: The study included 95 women aged 50–75 years. The presence of sarcopenia was evaluated in a group of 51 women with PsA, and 44 controls (without inflammatory joint disease). Measurements of muscle mass and lean body mass were made using the method of bioimpedance assessing ALM (Appendicular Lean Mass) index and SMI (Skeletal Muscle Index). The diagnosis of sarcopenia was made in women with low muscle mass and concomitant reduction of the efficiency of the assessed functional test Timed Up and Go (TUG). Bone density measurement was done by densitometry in the femoral neck and lumbar spine. (Ethics statement OIL 625/16/Bioet).

Results: Sarcopenia, using ALM index and SMI, was diagnosed in 13.7% and 43.1% of PsA women, and in healthy women in 9% and 20.4%, respectively. In the group of PsA, sarcopenia was associated with a significant increase in the occurrence of disorders of bone mineralisation (72.7% vs. 41.3% in patients without a decrease in muscle mass). There was no correlation between the loss of muscle mass, bone density, and activity of PsA.

Conclusions: The prevalence of sarcopenia in postmenopausal women suffering from PsA is associated with the occurrence of osteoporosis.

Key words: psoriatic arthritis, sarcopenia, measurement of body composition.

Introduction

Psoriatic arthritis (PsA), beyond joint involvement, is associated with many extra-articular complications. In a few available publications that analyzed body composition in patients with chronic inflammatory joint, the attention has mainly been focused on rheumatoid arthritis (RA), where the loss of muscle mass may involve up to two thirds of all patients and is often not associated with a reduction in fat mass or total body weight [1]. It seems that patients with PsA can also be exposed to muscle weakness and reduced mobility, which in turn can lead to imbalances and increase the risk of falls. Under phys-

iological conditions, loss of muscle tissue is associated with the aging of the body [2]. In the case of the disease a decrease in muscle mass can contribute to: chronic increase in the concentration of inflammatory mediators, prolonged bed rest, reduced physical activity, nutritional deficiencies, and disorders of innervation [3]. Recently, certain similarities between sarcopenia and osteoporosis were noticed, because in both cases increased risk of fractures, disability, and mortality were observed. It was even suggested that sarcopenia and osteoporosis should be treated as one disorder called “sarcoosteoporosis” or “sarcoporosis” [4]. Early diagnosis of sarcopenia or osteoporosis can accelerate the decisions to implement the

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appropriate prophylactic or therapeutic medications in patients vulnerable to fractures.

In order to evaluate sarcopenia a variety of diagnostic methods including computed tomography (CT), magnetic resonance imaging (MRI), dual-energy X-ray absorptiometry (DEXA), and bioelectrical impedance analysis (BIA) are used [5, 6]. The most accurate measurement methods are still highly expensive, such as computed tomography and magnetic resonance imaging. An accurate and much cheaper method is DEXA; however, like computed tomography, it involves the exposure of patients to X-ray radiation. The method of bioimpedance is based on measurements of electrical resistivity in different tissues of the human body, which conventionally are divided into parts (five cylinders – four limbs and the trunk). The advantages of this method are: non-invasive, low-cost test, convenience in use of the equipment, and high reproducibility of the results of research carried out under comparable conditions [7]. According to the recommendations of the European Working Group on Sarcopenia in Older People (EWGSOP), diagnosis of sarcopenia should relate to people with low muscle mass associated with impaired muscle strength and reduced physical mobility [6].

The primary diagnostic method used to identify individuals at risk of osteoporotic fractures is still DEXA. It is a method based on the simultaneous measurement of X-ray absorption of high and low energy levels. The huge difference, when changing the degree of absorption of radiation energy in the area to the bone as opposed to soft tissue, allows evaluation of the dose absorbed by the bone, and calculation of the absolute mineral mass and bone mineral density [8].

Aim

There are very few reports that attempt to evaluate the prevalence sarcopenia in patients with chronic arthritis, and almost all published studies are related with rheumatoid arthritis. Sarcopenia with decrease in muscle strength and mobility is associated with increased risk of falls and frequent fractures, particularly in postmenopausal women. The aim of the study was to estimate the prevalence of sarcopenia and osteoporosis among postmenopausal women with PsA and to compare results to women without inflammatory joint diseases.

Material and methods

The study included 51 women diagnosed with peripheral subtype of PsA, treated at the Rheumatology Clinic and Dermatology Clinic of the Municipal Hospital in Olsztyn, Poland. The control group was 44 women

without any inflammatory joint diseases. The diagnosis of PsA was determined based on the CASPAR criteria [9]. The age of respondents ranged from 50 to 75 years. At least 12 months had passed since the last menstrual period in all women.

The mass of muscle tissue was evaluated using the method of the bioimpedance analyzer InBody 170 (Bio-space, South Korea). Analysis of skeletal muscle mass, lean mass limb, total weight, and height were tested. For the evaluation of the loss of muscle mass the Appendicular Lean Mass (ALM) index was used – calculated as the ratio of the sum of lean limb mass to the square of body height (unit kg/m²), and the Skeletal Muscle Index (SMI) – representing the ratio of the total skeletal muscle mass to the mass of the body expressed as a percentage.

Using the standardised Timed Up and Go (TUG) test the physical mobility of the women was rated. TUG test included the following tasks: changing from a sitting position on a chair with a back to the standing position, then walking three metres, rotating 180 degrees, returning to the chair, and sitting. The assumed time of this test was less than 14 seconds [10].

The diagnosis of sarcopenia in surveyed women was determined in accordance with the recommendations of the European Working Group on Sarcopenia in Older People based on the definition of Baumgartner et al.: ALM index less than 5.45 kg/m² [11] and, in accordance with the approach Janssen et al., SMI index less than 27.6% [12]. In the case of using the definition of Janssen et al., a group of sarcopenia was divided into two groups: the first class of sarcopenia (mild sarcopenia) with SMI index greater than 22.1% and less than 27.6% and class II sarcopenia (severe sarcopenia) with SMI ≤ 22.1% [11].

Bone density measurement was done by densitometry of the left femoral neck and lumbar spine. For the measurement of bone density dual energy X-ray absorptiometry – DEXA was used. The diagnosis of osteopenia was determined on the value of T score in a range between –1.0 and –2.5 SD, osteoporosis with values of T score below –2.5 SD [8].

Disease activity was assessed using the Disease Activity Score 28 (DAS28). Markers of inflammation were measured by two standard laboratory parameters: erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP).

We excluded patients who were treated with corticosteroids currently or in the past two years, suffering from hyperthyroidism and hypothyroidism, after implantation of a pacemaker, after total joint implant of the intervertebral disc, and with the presence of other

metallic elements in the body due to contraindications to perform the study BIA.

All study subjects gave written, informed consent to participate in the study. The study was approved by the Bioethics Committee of the Warmia and Mazury Chamber of Physicians (Ethics statement OIL 625/16/Bioet; December 21, 2016).

Statistical analysis

Statistical analysis was performed using STATISTICA 12.5. Results were presented as means and standard deviations (SD). The normality of the distribution of variables was rated with Shapiro-Wilk test. In the case of normal distribution, to compare two unrelated groups the t-test was used, and in the case of significant difference in variance the Cochran-Cox test was applied. In the case of lack of normal distribution, the U Mann-Whitney test was used, and for comparing more than two groups the Kruskal-Wallis test was used. The level statistically significant was $p < 0.05$.

Results

Ninety-five women aged 50–75 years (51 women with PsA and 44 women without inflammatory joint disease) were assessed.

Study and control groups were not significantly different in terms of age, height, weight, BMI, or duration of menopause. Between the group of patients with PsA and the group without joint symptoms, significant differences related to muscle mass, lean limb mass, and fat were observed. Values of ALM index were not statistically different between the groups. SMI index was significantly lower in women with PsA than in subjects in the control group. Test TUG was longer in patients with PsA (Table I).

The incidence of sarcopenia depended on the method used for the evaluation. Using the ALM index allowed diagnosis of sarcopenia in seven women with PsA (13.7%) and in four healthy women (9%). TUG test was longer than 14 seconds in all women with ALM index lower than 5.45 kg/m². Sarcopenia was diagnosed much more frequently with use of SMI. SMI was lower

Table I. Age, anthropometric data, and clinical characteristic of the women studied

Parameter	PsA (n = 51)	Control (n = 44)	p
Age (years)	65.6 ±5.9	61.3 ±8.7	NS
Weight (kg)	72.4 ±9.4	68.2 ±11.8	NS
Height (m)	1.61 ±0.07	1.58 ±0.05	NS
BMI	30.1 ±5.8	28.1 ±6.9	NS
DAS28	3.64 ±1.4	–	–
Duration of psoriasis (years)	22.9 ±12.9	–	NS
Duration of psoriatic arthritis (years)	11.1 ±8.9	–	–
Duration of menopause (years)	15.6 ±11.2	13.9 ±10.3	NS
Muscle mass (kg)	21.1 ± 2.3	23.3 ±2.9	< 0.01
Appendicular lean mass (kg)	15.7 ±2.6	16.9 ±3.7	< 0.01
Total fat mass (kg)	47.2 ±8.1	38.5 ±7.2	< 0.001
Total lean mass (kg)	38.9 ±4.4	43.1 ±7.1	< 0.01
Appendicular Lean Mass Index (kg/m ²)	6.44 ±0.72	6.75 ±0.61	NS
Skeletal Muscle Index (%)	28.6 ±3.9	39.2 ±3.6	< 0.001
Time Get-Up and Go (s)	10.8 ±3.8	9.4 ±5.6	< 0.01
Lumbar spine BMD (g/cm ³)	0.921 ±0.169	0.943 ±0.188	NS
Lumbar spine T-score	–1.28 ±1.34	–1.18 ±1.27	NS
Lumbar spine Z-score	0.44 ±1.54	0.39 ±1.61	NS
Femoral neck BMD (g/cm ³)	0.747 ±0.257	0.804 ±0.209	NS
Femoral neck T-score	–1.09 ±1.31	–0.99 ±1.45	NS
Femoral neck Z-score	0.31 ±1.32	0.38 ±1.51	NS
Total hip BMD (g/cm ³)	0.869 ±0.149	0.894 ±0.167	NS
Total hip T-score	–0.92 ±0.157	–0.94 ±0.141	NS
Total hip Z-score	0.61 ±1.29	0.56 ±1.43	NS

BMI – body mass index; DAS – disease activity score; BMD – bone mineral density

Table II. Relationship between sarcopenia and bone mineral density in women with PsA

Parameter	PsA sarcopenia (+) (n = 22)	PsA sarcopenia (-) (n = 29)	p
Osteoporosis (any site) n (%)	9 (40.9%)	5 (17.2%)	< 0.01
Osteopenia (any site) n (%)	7 (31.8%)	7 (24.1%)	NS
Lumbar spine BMD (g/cm ³)	0.826 ±0.181	0.984 ±0.122	< 0.01
Lumbar spine T-score	-1.43 ±1.42	-1.21 ±1.19	< 0.05
Lumbar spine Z-score	0.39 ±1.61	0.46 ±1.44	< 0.05
Femoral neck BMD (g/cm ³)	0.729 ±0.265	0.804 ±0.187	< 0.05
Femoral neck T-score	-1.18 ±1.39	-0.98 ±1.53	< 0.05
Femoral neck Z-score	0.28 ±1.42	0.43 ±1.46	< 0.05
Total hip BMD (g/cm ³)	0.842 ±0.132	0.958 ±0.187	< 0.05
Total hip T-score	-1.07 ±0.171	-0.7 ±0.127	< 0.01
Total hip Z-score	0.46 ±1.45	0.68 ±0.16	< 0.05

BMD – bone mineral density

than 27.6% in 25 female patients with PsA (48%), including less than or equal to 22.1% of women with PsA 4 (7.8%). Based on the SMI and the results of the TUG test sarcopenia was diagnosed in 22 women with PsA (43.1%), and mild and severe sarcopenia were found in 14 patients (27.4%) and eight patients (15.6%), respectively. Assessing a group of women without inflammatory diseases of the joints, SMI index revealed a decrease in muscle mass in 15 women (34%). Loss of mobility with reduction of SMI allowed diagnosis of sarcopenia in nine women (20.4%). Mild sarcopenia occurred in six women (13.6%) and severe sarcopenia in three women (4.5%) without inflammatory joint disease. In both groups with SMI index lower than or equal to 22.1%, the test TUG was longer than 14 seconds.

Mobility TUG test results in women suffering from PsA were lengthened, with an increase in disease activity using the DAS28 ($p < 0.05$), and with increasing rates of inflammation. In the control group the TUG test was not associated with the ESR and CRP. There were no statistically significant differences in the length of mobility test subjects divided in relation to BMI groups of normal weight, overweight, and obesity.

There was no correlation between ALM, SMI, and increased activity of the assessed markers of inflammation in both groups. The activity of PsA as measured by DAS28 also was not associated with the indicators of muscle mass. The results of ALM and SMI indices did not depend on BMI.

Among women suffering from PsA, SMI index negatively correlated with both the duration of arthritis ($r = -0.335$, $p = 0.028$) and psoriasis ($r = -0.389$, $p = 0.017$). The duration of diseases of the joints and the skin does not affect the height of the ALM index in women with PsA. In all of the women ALM and SMI were reduced with age.

In both groups bone mineral density in the femoral neck and lumbar spine were rated. The incidence of osteoporosis and osteopenia were not significantly different between groups with PsA and without arthritis. Osteoporosis was diagnosed in 14 patients with PsA (27.4%) and 11 (25%) women with no joint symptoms, then osteopenia in 14 patients with PsA (27.4%) and in 10 controls (22.7%). In female patients suffering from PsA without sarcopenia, no significant differences were found in the incidence of osteoporosis. Among PsA patients with sarcopenia osteoporosis occurred twice as often as compared with patients without loss of muscle mass (Table II). In the case of women without inflammatory joint disease there were no similar differences. Osteoporosis in this group was diagnosed in two (22.2%) people with sarcopenia and in eight (22.8%) with normal muscle mass. Osteopenia occurred slightly more often among the patients with PsA with concomitant sarcopenia, but this was not a statistical relationship ($p = 0.059$). The incidence of osteopenia in the control group did not differ significantly among patients with sarcopenia or without sarcopenia, and was 22.8% and 25.7%, respectively.

In our study, we observed a statistically significant correlation between the duration of psoriasis and BMD measured at the femoral neck ($r = -0.362$; $p = 0.019$) and the lumbar spine ($r = -0.384$; $p = 0.041$). The duration of PsA negatively correlated with the mineral density of the femoral neck ($r = -0.402$; $p = 0.044$) and lumbar spine ($r = -0.374$; $p = 0.027$).

We observed no association between decreased BMD and the activity of PsA assessed with DAS28 and the value of CRP and ESR.

Discussion

The pathogenesis of loss of muscle mass and muscle weakness in PsA is complex. It may include, in addition

to aging patients, decreased physical activity secondary to stiffness and pain, hormonal changes, disturbances in the metabolism of protein, and associated with the chronic inflammation process (increase level of proinflammatory cytokines and C-reactive protein) [13].

Our research showed that occurrence of sarcopenia varies depending on the used marker of muscle mass. Sarcopenia recognised with SMI was twice as often presented in the group of women suffering from PsA compared to the control group (43.1%), while with ALM only in 13.7% patients with PsA. The vast majority of the available studies of sarcopenia due to chronic arthritis focused on patients with RA. Only a few articles have been published so far on the prevalence of sarcopenia in patients with PsA. In a small study of 60 patients, including men and women with ankylosing spondylitis and PsA, Aguiar et al. [14] observed a significant decrease in muscle mass measured by the MMI (muscle mass index) in as many as 61.7% of surveyed patients with spondyloarthropathies. This study did not include muscle strength or physical activity, and the adopted methods of measuring muscle were less accurate anthropometric methods [14]. Giles et al. [1], using the method of measuring body composition with DEXA, evaluated the risk of sarcopenia in a group of 117 women with RA at the age of 45–84 years. A significant loss of muscle mass was assessed by the ratio of the sum of lean limb mass to the square of the body height less than 5.75 kg/m², and sarcopenia was diagnosed in 21.4% of patients compared with 7.7% in the group of healthy women [1]. In other research using this method of measurement, Ceyhan Doğan et al. [15] recognised sarcopenia in 43.3% of women with RA aged 35–50 years.

In our study, increased levels of CRP and ESR were associated with prolonged test TUG in patients with PsA. Other researchers observed muscle weakness dependent on CRP increase. Visser et al. [16], in a research project that included more than 3000 men and women aged 70–79 years, using measurements of muscle strength and knee extensor isometric force measurement handshake, described the relationship in the concentration of CRP with reduced muscle strength. In another large study on elderly people (average age 74.6 years, SD ±6.2), Schaap et al. [17] observed elevated levels of CRP with twice the decrease in muscle strength measured by isometric handshake.

In both study groups, we found no relationship between the activity of inflammation and the reduction in muscle mass. In contrast to our results, the occurrence of sarcopenia in persons without inflammatory joint disease associated with an increase in CRP concentration was described by Schaap et al. [17]. Similarly, Visser et al. [16] observed decreased muscle mass, measured with

DEXA and CT, associated with an increase in CRP among the elderly. Among patients with RA, Munro et al. [18] found negative correlation with CRP and ESR and muscle mass. Ceyhan Doğan et al. [15], revealed a link between the loss of muscle mass and an increase in CRP, among a group of women with RA, but not with an increased rate of DAS28. The increase in activity of RA was evaluated by DAS28 and it was linked with a reduction in muscle mass, in the study of Walsmith et al. [19]. Dao et al. [20] described the relationship of disease activity with loss of muscle mass and the occurrence of a specific type of sarcopenic obesity in women with seropositive RA, lasting less than three years. On the other hand, Giles et al. [1], in a study of 189 patients with RA and 189 controls without RA, did not find a significant relationship in body composition with increases of DAS28. Moreover, the recently published article by Lemney et al. [21], assessing the impact of disease activity and therapeutic effect to achieve low disease activity, showed no effect of treatment “for the purpose” (treat-to-target – T2T) and did not cause halting of the loss of muscle mass in patients with RA. Aguiar et al. [14] did not observe a statistical relationship between DAS28 for peripheral spondyloarthropathies and axial subtype index BASDAI (Bath Ankylosing Spondylitis Disease Activity Index) in a group of women. Reducing the MMI was associated with an increased rate of BASDAI only in men [14].

In our study, we found no relationship between the loss of muscle mass and BMI in any of the studied groups. The study by Ceyhan Doğan et al. [15] revealed that sarcopenia was more frequent in patients with normal or excess body weight. Among the surveyed women with RA, Giles et al. observed obesity accompanied by sarcopenia more often than in the control group, although the prevalence of sarcopenia showed a downward trend with BMI [1]. Stavropoulos-Kalinoğlu et al. [22] showed similar changes in body composition in patients with normal body weight, especially in women.

Current literature on the prevalence of osteoporosis in PsA provides ambiguous, often divergent data. Due to physiological differences in the incidence of osteoporosis in women and men, our study included only postmenopausal women.

In both groups, the prevalence of osteoporosis and osteopenia did not differ significantly. Impaired mineralisation in the form of osteopenia and osteoporosis occurred in more than 50% of patients with PsA and almost 50% of women from the control group. Interesting results were obtained after the division of the treated groups, because of sarcopenia. Among patients with PsA and sarcopenia, osteoporosis occurred in 40%, and impaired mineralisation included over 70% of pa-

tients. On the other hand, in patients with PsA without the loss of muscle mass, osteoporosis and impaired mineralisation was recognised in approximately 17% and 40% of patients, respectively. Frediani et al. [23] observed mineralisation disorder in at least one of the regions of the skeletal system in 67% of premenopausal women and in 100% of postmenopausal women, using fan-beam densitometry techniques together with ultrasound densitometry measurements of the heel. In patients with PsA an increased incidence of fracture compared with the general population and similar risk for any fracture in patients with PsA and severe psoriasis to RA were observed by Odgie et al. [24]. Borman et al. [25] recognised osteoporosis only in 5%, and osteopenia in 50% (the control group showed 27.5% of osteopenia) of patients with PsA (but not post-menopausal women). Some authors suggest a complete lack of relation between osteoporosis and PsA. Reid [26] and Nolla [27] found no significant differences in bone density between patients with peripheral subtype of PsA and a control group matched for gender, age, and menopausal status. No differences in BMD status between the PsA patients and the general population were found in a study by Busquets et al. [28]. In our study, bone mineral density, measured within the lumbar spine in the range L2–L4 vertebrae and in the femoral neck in women suffering from PsA, was significantly lower than in the control group. Similarly, a significant reduction in bone mineral density (BMD) in patients with PsA in the general population was described Frediani et al. [23]: lumbar spine in the range of 1.112 vs. 1.326, in terms of the femoral neck 0.870 vs. 1.006.

In this study, we observed the relationship between the duration of psoriasis and PsA, and reduction of bone mineral density measured in the femoral neck and spine. Borman et al. [25] also noted a relationship between the intensity of demineralisation and the duration of joint disease. Similarly, Dheda et al. [29] observed a marked reduction in BMD in patients with a duration of arthritis over nine years.

The activity of PsA assessed by DAS28 showed no association with decreased BMD. Borman et al. [25] have reported recently no evidence of mineralisation compound with the activity of PsA. Whereas Dheda et al. [29] showed the dependency of BMD number of involved joints. However, it should be noted that the method of measuring the activity of the disease – DAS28 for a peripheral PsA, is only transferred from RA and therefore does not cover the whole clinical picture of PsA. In our research, ESR and CRP were not associated with decreased bone mineral density. Similar results were achieved by Frediani et al. [23] and Grisar et al. [30].

Conclusions

The loss of muscle mass in patients with chronic inflammation of the joints, leading to muscle weakness and disability conditions, may deepen progressive destruction of joints, causing pain and stiffness. These results can cause an increased risk of falls and their complications, especially in the presence of osteoporosis. Our study showed a similar incidence of osteoporosis in women with PsA and without inflammatory disease, but in patients with PsA the sarcopenia caused more impaired bone mineralisation.

The authors declare no conflict of interest.

References

- Giles JT, Ling SM, Ferrucci L, et al. Abnormal body composition phenotypes in older rheumatoid arthritis patients: association with disease characteristics and pharmacotherapies. *Arthritis Rheum* 2008; 59: 807-815.
- Janssen I. Evolution of sarcopenia research. *Appl Physiol Nutr Metab* 2010; 35: 707-712.
- Greenlund LJS, Nair KS. Sarcopenia – consequences, mechanisms, and potential therapies. *Mech Ageing Dev* 2003; 124: 287-299.
- Binkley N, Buehring B. Beyond FRAX: It's time to consider "sarcopenia". *J Clin Densitom* 2009; 12: 413-416.
- Pahor M, Manini T, Cesari M. Sarcopenia. Clinical evaluation, biological markers and other evaluation tools. *J Nutr Health Aging* 2009; 13: 724-728.
- Cruz-Jentoft AJ, Baeyens JP, Bauer JM, et al. Sarcopenia: European consensus on definition and diagnosis. Report of the European Working Group on Sarcopenia in Older People. *Age Ageing* 2010; 39: 412-423.
- Kyle UG, Bosaeus I, De Lorenzo AD, et al. Bioelectrical impedance analysis – part I: review of principles and methods. *Clin Nutr* 2004; 23: 1226-1243.
- Ström O, Borgström F, Kanis JA, et al. Osteoporosis: burden, health care provision and opportunities in the EU. *Arch Osteoporos* 2011; 6: 59-155.
- Taylor W, Gladman D, Helliwell P, et al. CASPAR Study Group. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. *Arthritis Rheum* 2006; 54: 2665-2673.
- Baumgartner RN, Koehler KM, Gallagher D, et al. Epidemiology of sarcopenia among elderly in New Mexico. *Am J Epidemiol* 1998; 147: 755-763.
- Janssen I, Heymsfield B, Ross R. Low relative skeletal muscle mass (sarcopenia) in older person is associated with functional impairment and physical disability. *J Am Geriatr Soc* 2002; 50: 889-896.
- Rall LC, Roubenoff R. Rheumatoid cachexia: metabolic abnormalities, mechanisms and interventions. *Rheumatology* 2004; 43: 1219-1223.
- Roubenoff R, Roubenoff RA, Cannon JG, et al. Rheumatoid cachexia: cytokine-driven hypermetabolism accompanying

- reduced body cell mass in chronic inflammation. *J Clin Invest* 1994; 93: 2379-2386.
14. Aguiar R, Sequeira J, Meirinhos T, et al. SARCOSPA – Sarcopenia in spondyloarthritis patients. *Acta Reumatol Port* 2014; 39: 322-326.
 15. Ceyhan Doğan S, Hizmetli S, Hayta E, et al. Sarcopenia in women with rheumatoid arthritis. *Eur J Rheumatol* 2015, 2: 57-61.
 16. Visser M, Pahor M, Taaffe DR, et al. Relationship of interleukin-6 and tumor necrosis factor-alpha with muscle mass and muscle strength in elderly men and women: the Health ABC Study. *J Gerontol A Biol Sci Med Sci* 2002; 57: 326-332.
 17. Schaap LA, Pluijm SM, Deeg DJ, et al. Inflammatory markers and loss of muscle mass (sarcopenia) and strength. *Am J Med* 2006; 119: 5269-5217.
 18. Cesari M, Kritchevsky S, Baumgartner RN, et al. Sarcopenia, obesity, and inflammation--results from the trial of angiotensin converting enzyme inhibition and novel cardiovascular risk factors study. *Am J Clin Nutr* 2005; 82: 428-434.
 19. Walsmith J, Abad L, Kehayias J, et al. Tumor necrosis factor- α production is associated with less body cell mass in women with rheumatoid arthritis. *J Rheumatol* 2004; 31: 23-29.
 20. Dao HH, Do QT, Sakamoto J. Abnormal body composition phenotypes in Vietnamese women with early rheumatoid arthritis. *Rheumatology (Oxford)* 2011; 50: 1250-1258.
 21. Lemmey AB, Wilkinson TJ, Clayton RJ, et al. Tight control of disease activity fails to improve body composition or physical function in rheumatoid arthritis patients. *Rheumatology (Oxford)* 2016; 55: 1736-1745.
 22. Stavropoulos-Kalinoglou A, Metsios GS, Koutedakis Y, et al. Redefining overweight and obesity in rheumatoid arthritis patients. *Ann Rheum Dis* 2007; 66: 1316-1321.
 23. Frediani B, Allegri A, Falsetti P, et al. Bone mineral density in patients with psoriatic arthritis. *J Rheumatol* 2001; 28: 138-143.
 24. Ogdie A, Harter L, Shin D, et al. The risk of fracture among patients with psoriatic arthritis and psoriasis: a population-based study. *Ann Rheum Dis* 2017; 76: 882-885.
 25. Borman P, Babaoğlu S, Gur G, et al. Bone mineral density and bone turnover in patients with psoriatic arthritis. *Clin Rheumatol* 2008; 27: 443-447.
 26. Reid DM, Kennedy NS, Nicoll J, et al. Total and peripheral bone mass in patients with psoriatic arthritis and rheumatoid arthritis. *Clin Rheumatol* 1986; 5: 372-378.
 27. Nolla JM, Fiter J, Rozadilla A, et al. Bone mineral density in patients with peripheral psoriatic arthritis. *Rev Rhum Engl Ed* 1999; 66: 657-661.
 28. Busquets N, Vaquero CG, Moreno J, et al. Bone mineral density status and frequency of osteoporosis and clinical fractures in 155 patients with psoriatic arthritis followed in a university hospital. *Rheumatol Clin* 2014; 10: 89-93.
 29. Dheda K, Cassim B, Patel N, et al. A comparison of bone mineral density in Indians with psoriatic polyarthritis and healthy Indian volunteers. *Clin Rheumatol* 2004; 23: 89.
 30. Grisar J, Bernecker PM, Aringer M, et al. Ankylosing spondylitis, psoriatic arthritis, and reactive arthritis show increased bone resorption, but differ with regard to bone formation. *J Rheumatol* 2002; 29: 1430-1436.