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Original Research Article

Prevalence and impact of adiposity and sarcopenia during rheumatoid arthritis: rapid and non-invasive evaluation in Sub-Saharan African women

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ABSTRACT

Background: Body composition plays a fundamental role in the occurrence of complications in rheumatoid arthritis. Authors conducted this study, which aimed to determine body composition and its effects on physiological status in African sub-Saharan polyarthritis women.
Methods: The anthropometric parameters were measured after an interview and a complete physical examination. The body composition was evaluated using a Tanita® brand bioimpedance meter. Finally, all the patients had a dosage of certain biochemical parameters.
Results: An excess of percent fat mass was noted in more than half of women (59.52%) without loss of muscle mass. At the same time, 30% of women had a significant decrease in the percentage of body water. The BMI did not appear to be an adequate proxy for these changes. Visceral fat level was elevated just in 16% of women, however it would be a determinant of physiological aging of subjects. Dual therapy methotrexate and corticosteroid would have varying effects depending on the duration and the dose of treatment. The basic metabolism in polyarthritic subjects would be dependent on two parameters namely muscle mass and inflammatory state.
Conclusions: Evaluating changes in body composition quickly, non-invasively and inexpensively is possible. It could be useful in the follow-up of rheumatoid arthritis. Managing these changes can reduce cardiovascular morbidity and mortality in rheumatoid arthritis.

Keywords: Adiposity, Metabolic age, Rheumatoid arthritis, Sarcopenia, Sub-Saharan African women

INTRODUCTION

Rheumatoid arthritis (RA) is the most common chronic inflammatory rheumatism in adults.[1] It's a systemic autoimmune disease characterized by several consequences in short and long term, including an increased risk of comorbidities.[2] It's, therefore, characterized by a decrease in life expectancy.[3] Studies

have shown that cardiovascular events (CVs) dominate RA related morbidity and mortality and account for about 50% of excess mortality in RA.[4-5] Thus, authors consider RA as a cardiovascular risk factor of the same weight as diabetes.[6] These cardiovascular events would be attributable to classical cardiovascular risk factors but also to elements specific to RA such as the existence of a low-grade chronic systemic inflammatory state.[7] Also characterized by extra-articular manifestations, a change in body composition (BC) is frequently reported during RA and is associated with major complications of the disease especially cardiovascular morbidity and mortality.[8,9] Studies in patients followed for old systemic inflammatory diseases have shown significant changes in BC.[10]

They showed that at equal body weight, the proportion of lean body mass is lowered and that of body fat is elevated in RA patients compared to healthy subjects.[9] It therefore seems crucial to verify these findings in a Sub-Saharan African population but also to show the importance of considering changes in the body parameters in the overall management of cardiovascular risk during RA. Thus, authors conducted this study to determine, in a fast and non-invasive way, changes in anthropometric parameters and body composition as well as their impact on the physiological state of Senegalese women during RA.

METHODS

Subjects and protocol

It was a prospective, cross-sectional study. It was carried out in the human physiology and functional exploration department of the Faculty of Medicine, Pharmacy and Odontology (FMPO) at the Cheikh Anta Diop University (UCAD) in Dakar, Senegal.

Authors collected forty-two (42) women with rheumatoid arthritis and followed at the rheumatology unit of Aristide LeDantec university hospital in Dakar. Records of all patients aged between 18 and 50 years were studied and only patients who met the criteria of American College of Rheumatology 1980 (ACR) modified by Liao were included.

Authors didn't include subjects whose RA was associated with other systemic autoimmune diseases, or RA was severely complicated (ischemia, gangrene) but also lactating and pregnant women. The existence of cardiovascular risk factors was sought: treated or untreated arterial hypertension, history of obesity, treated or untreated hypercholesterolemia, active or weaned smoking, type I or II diabetes.

The study protocol is in line with the ethical principles set out in the 1975 Helsinki Declaration and has been approved by UCAD's FMPO Ethics Committee. All the subjects recruited were informed of the interest of this work and all gave their oral and written consent.

Investigative methods

The parameters required for this study were notified in a single medical visit. The patients were summoned at 8 o'clock in the morning. Sociodemographic information and the history of their illness were collected using a questionnaire. After the interview, all patients underwent a complete clinical examination.

Clinical evaluation

Each subject had a complete physical examination including taking anthropometric parameters and clinical constants necessary for the study.

Measurement of arterial pressure and pulse was performed by an Omron® electronic sphygmomanometer with cuff adapted to the arm, meeting the required conditions. According to the WHO, arterial hypertension (HTA) was defined when systolic blood pressure (SBP) was ≥140mmHg and/or diastolic blood pressure (DBP) was ≥90mmHg. Mean arterial pressure (MAP) was calculated according to the formula of Messai E. Ed. Arnette Blackwell (Paris) 1995: MAP=(SBP+2 DBP)/3.

Measurement of size, waist circumference (WC) and hip circumference (HC) was performed using a tape measure to the nearest cm. Then authors calculated the ratio WC/HC (RWH).

WC was considered high when it was greater than 80 cm and RWH was considered high when it was greater than 0.8.[11]

Measurement of weight to the nearest kg, muscle mass (MM) to the nearest kg, BMI, percentage fat mass (PFM), percentage body water (PBW), visceral fat level (VFL), metabolic age (MA) and basal metabolism (BM) was performed using a TANITA® brand a level 3 bio-impedance meter (model BC 601) that allows for a global and segmental evaluation of body composition (BC).

BMI (kg/m^2), was classified according to WHO standards proposed in 2000. Leanness ≤18.49; Normal BMI from 18.5 to 24.99, overweight from 25 to 29.99 and obesity ≥30.

MM was considered low when the absolute value was <30 kg and/or the muscle mass index (MMI) was less than 6.42 kg/m^2.[12] VFL was considered normal between 1 and 9, high between 10 and 14 and very high above 14.[13]

PBW was considered normal for values between 45% and 65%, as low if the value is less than 45% and high if it's greater than 65%.[14]

The PFM was allocated to class according to Table 1 below. The BM must be greater than or equal to 1,600 kcal/day.[16] The metabolic age is equivalent to the

physiological age of the subject and it's compared with the actual age of the patient.

Table 1: Body fat level in women according age (year) (d'après les recommendations de Gallagher et al).[15]

Ages	Lean	Normal	Overweight	Obese
20-39	5 21	21-33	33-38	>38
40-59	5-23	23-34	34-40	>40
60-75	5-24	24-36	36-42	>42

Evaluation of biological parameters

The biological parameters were measured the same day in the biochemistry laboratory of the FMPO. Samples were taken before the interviews and the patients were fasting for at least 12 hours.

Venous blood was collected at the fold of the elbow of the non-dominant arm. Thus, for each patient, the blood collected was distributed in a fluoride tube for the determination of fasting blood glucose, in a heparin tube for the measurement of lipids (Total cholesterol, HDL cholesterol, LDL cholesterol and Triglycerides) and the evaluation of renal function (Uremia and Creatininemia). By enzymatic method, Authors realized the measurement of all these parameters.

Statistical analysis

All variables were saved in an Excel table. Quantitative variables were described using mean±standard deviation (SD) and qualitative variables using absolute values and percentages.

The Student's T-test was used to compare the mean of the quantitative variables. Correlation tests and linear regression tests were carried out to find associations between the different parameters studied. The results are considered significant when p <5%. The exploitation of the data was carried out by SPSS software version 16.0

RESULTS

Descriptive results

Characteristics of the subjects and the rheumatoid arthritis

There was no smoker or former smoker. There was no diabetic subject, but they were all sedentary subjects. Authors noted that the mean values of all the variables studied were normal except for the high DBP and C-Reactive Protein (CRP) (Table 2).

Table 2: Characteristics of Patients and Rheumatoid Arthritis.

Characteristics of patients and of rheumatoid arthritis	Number	Percentage
Known hypertensive subjects	18	42.86
Subjects with a family history of RA	12	28.57
Presence rheumatoid factor	26	61.90
Presence of anti-citrullinated peptide antibodies	42	100
	Mean±SD	**Min-Max**
Mean age (years)	43.88±11.04	26-65
Duration of RA evolution (years)	4.27±4.3	1-15
Duration of corticosteroid therapy (months)	18.74±17.17	2-100
Corticosteroid dose (mg/day)	9.40±1.64	5-10
Duration methotrexate treatment (months)	17.76±13.59	2-60
Methotrexate dose (mg/week)	11.53±1.31	8-12
Characteristics of clinical and biochemical parameters	**Mean±SD**	**Min-Max**
Weight (kg)	66.85±13.12	42-95
Size (cm)	164.67±6.59	150-185
SBP (mmHg)	122.93±3.17	90-160
DBP (mmHg)	82.44±1.87	70-100
MBP (mmHg)	96.75±2.25	80-123
Total cholesterol (UI)	2.12±0.06	1.50-3.23
HDL-cholesterol (UI)	0.63±0.02	0.33-0.9
Triglycerides (UI)	0.86±0.07	0.36-3.34
LDL-cholesterol (UI)	1.18±0.04	0.60-1.69
Fasting blood glucose (g/l)	0.92±0.05	0.69-1.34
Creatininemia (mg/l)	6.83±1.60	4.80-11.06

Study of anthropometric parameters, body composition and basic metabolism

Based on BMI, PFM, PBW and MM

According to BMI and PFM, the excess weight was respectively 40.48% and 59.52% of women with rheumatoid arthritis, as detailed in Table 3.

The PBW was low in 30.95% of subjects. Both MM and MMI were normal in all individuals in the study. Their mean values were respectively 41.37 kg ±6.38 and 15.33 kg/m 2±2.65.

Table 3: Distribution of subjects based to BMI and PFM.

Variables	BMI N (%)	PFM N (%)
Lean	2 (4.76)	7 (16.67)
Normal	23 (54.76)	10 (23.81)
Overweight	12 (28.57)	7 (16.67)
Obese	5 (11.90)	18 (42.86)

Based on WC, RWH and VFL

An abnormally high WC accounted for more than half of the women (64.29%), whereas the VFL was high in 7 women (16.67%). The WHR was high in 19 women (45.24%). Authors found that parameters that varied significantly with PFM were VFL and PBW. VFL increased with PFM while PBW decreased with PFM. See Figure 1.

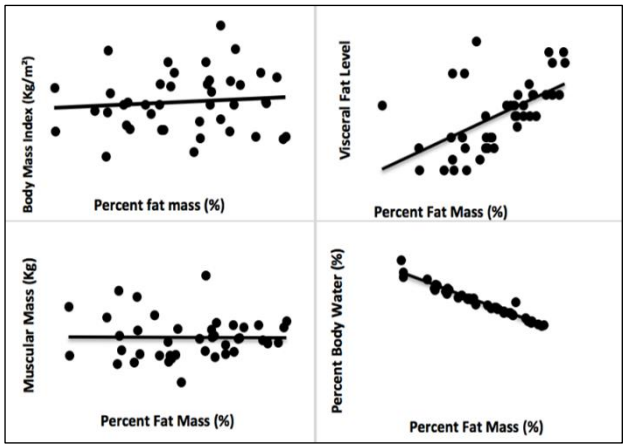

Figure 1: Distribution of body composition parameters as a PFM.

Evaluation of the BM and the MA of the subjects

BM was normal in all women in the study. Of the women in the study, 69.05% had a MA older than their actual age.

Analytical results

Bivariate analyzes

Authors found that the parameters that were significantly different according to the importance of PFM were PBW, VFL and MA. See Table 4.

Table 4: Comparison between subjects with excess PFM and subjects with normal PFM.

Variables	Subjects with excess PBF	Subjects with normal PBF	p-value
Age (years)	45.64±12.04	41.29±9.10	NS
Weight (kg)	67.84±13.06	62.41±13.48	NS
Size (cm)	164.48±7.92	164.94±4.12	NS
BMI (kg/m2)	25.09±4.83	23.98±4.80	NS
WC (cm)	82.76±11.79	80.71±11.36	NS
WHR	0.81±0.07	0.78±0.07	NS
MM (kg)	40.38±3.71	42.82±8.93	NS
PBW (%)	45.07±0.54	54.91±0.47	<0.0001
VFL	7.04±2.73	4.65±3.55	0.01
MA	50.52±14.01	33.65±16.44	0.001
BM (Kcal/j)	2061.76±192.45	2088.59±372.31	NS
SBP (mmHg)	124±20	120±20.76	NS
DBP (mmHg)	83.20±11.44	81.18±12.69	NS
MBP (mmHg)	98.13±13.95	94.31±14.99	NS
Fasting blood glucose (g/l)	0.97±0.50	0.89±0.08	NS
C-Reactive Protein (mg/l)	11.91±13.85	13.01±13.22	NS
LDL-Cholesterol (g/l)	1.20±0.33	1.17±0.28	NS
Triglycerides (g/l)	0.81±0.60	0.89±0.33	NS
HDL-Cholesterol (g/l)	0.60±0.11	0.67±0.12	NS
Cholesterol Total (g/l)	2.13±0.34	2.13±0.40	NS
Creatinemia (mg/l)	6.60±1.52	7.16±1.69	NS

Authors found that BMI was positively correlated with SBP (p=0.03 r=0.43). At the same time WC was positively correlated with SBP (p=0.000 r=0.56), DBP (p=0.004 r=0.41) and MBP (p=0.001 r=0.48).

With regard to MM, a strong positive association with BM was observed (p <0.0001, r=0.97). MM was negatively correlated with duration of corticosteroid therapy (p=0.04, r=-0.45) and duration of methotrexate therapy (p=0.03, r=-35). At the same time, BM was negatively correlated with duration of glucocorticoid therapy (p=0.04, r=-0.45) and methotrexate's dose (p=0.03, r=-0.44) and positively correlated with CRP (p=0.02, r=0.33).

MA was positively correlated with VFL (p <0.0001, r=0.79) and negatively correlated with BMI (p=0.005 r=-0.45), WC (p=0.004, r=-0.41) and the PBW (p<0.0001, r=-0.6).

Multivariate analyzes

After linear regression tests authors found that BM was only associated with MM (p <0.0001), VFL was associated with MA (p <0.0001) and PWB (p <0.0001). The PBW was also associated with LDL-cholesterol (p=0.01).

DISCUSSION

This study was to this knowledge, the first to describe changes in BC during RA in the Sub-Saharan African subject.

Authors noted in this study, an increase in the PBF that is greater than shown by BMI. The abdominal location of the fat is more subcutaneous than perivisceral. Abdominal obesity, especially by elevating the VFL, is more associated with cardiovascular risk factors than BMI. The basic metabolism depends on the muscle mass which in turn varies depending on the duration and dose of treatment with corticosteroids and MTX. The increase in the level of visceral fat promotes the precocity of physiological aging during RA.

An excessive development of PFM has been noted in the subjects of this series. These results go in the same direction as those found in literature. An increase in fat mass during RA is reported by many authors.[17] Systemic inflammation may result in changes in the lipid profile and, consequently, lead to adiposity in RA patients.[18]

Authors didn't note a decrease in MM in this subjects despite the overgrowth of PFM. In fact, during RA, a decrease in MM is observed in 20 to 30% of patients.[19,20] This means that the loss of MM is not constant during RA, although it can affect up to two-thirds of people with rheumatoid arthritis.[9] In this series just 4.76% of the subjects were lean according to the BMI or the changes of the BC during the RA are more marked in the patients having a weak BMI and they attenuate progressively with the increase of the BMI to disappear in obese polyarthritis subjects.[9] The loss of MM in RA would be more closely related to systemic inflammation than to increased PBF.[21,22] The inflammatory response increases BM and consequently reduces MM.[9] Authors noted in this series a strong association between MM and BM. Muscle is the major consumer of energy and contributes to the rate of basal body metabolism.[23] Activation of the immune system consumes a large amount of energy; inducing a hypermetabolic and catabolic state accompanied by an increase in BM.[21,24] In this series, the combination of the anti-inflammatory effects of glucocorticoids and MTX is thought to be the cause of the decrease in BM inducing the conservation of MM. However, the duration and the cumulative dose of treatment, especially with corticosteroids, should be strictly monitored and revised downwards or stopped as soon as stable and long-term remission of RA activity is achieved to avoid deterioration in quantity and especially in quality muscular. Authors have shown that muscle composition in PR patients treated with corticosteroids presents reduced muscle density and increased intramuscular fat leading to impaired muscle quality. [20,25]

The role of corticosteroid therapy in chronic inflammatory diseases is complex. On the one hand, it can control inflammation, which can fight against glucose intolerance and dyslipidemia thus decreasing cardiovascular risk, on the other hand, their long-term and high-dose use is responsible for secondary effects, namely a disruption of lipid metabolism, hyperglycemia, arterial hypertension and central obesity, which will contribute collectively to the development of the metabolic syndrome.[20,26] But some authors have not found a relationship between corticosteroid therapy and changes in BC during RA.[1]

Authors noted that the cumulative dose of MTX would protect against an increase in BM. The mechanism of action of MTX in this study could be attributed to an anti-inflammatory effect leading to a decrease in protein hypercatabolism. Chronic inflammation during RA is thought to be responsible for an imbalance between protein degradation and protein synthesis in favor of protein degradation with loss of muscle mass and accumulation of body fat.[27]

This suggests that MTX would protect against the development of adiposity during RA. This would confirm the remarks of Toms et al. who demonstrated in their study that non-taking or lower use of MTX is an independent risk factor for adiposity in RA.[28]

This association has been found also by other authors.[29,30] However, some authors have not established a relationship between the presence of adiposity and non-use of MTX in their studies.[31,32]

These findings reveal that the pathophysiology of BC alterations during RA is complex and multifactorial. In addition to systemic inflammation, several factors would be involved in modifying the BC during RA such as treatment, the clean elements of the diseases and environmental factors. Authors therefore have an increase in PFM without a decrease in MM which would favor a non-sarcopenic adiposity. This result do not corroborate data from the literature for which studies in patients treated for RA have generally described significant changes in BC with increased total PFM and decreased MM, resulting in sarcopenic adiposity.[10,20,22] In addition, the deterioration of walking, quality of life and loss of autonomy in RA would be all the more marked if there is a sarcopenic adiposity that seems to be the earliest deterioration induced by the immuno-inflammatory response.[9,33]

Authors have reported that in most polyarthritis patients, overall body weight and BMI are conserved, and muscle loss is compensated for by increased body fat. In this series more than half of the subjects have normal muscle mass and the PFM has increased. On the other hand, a significant decrease of the total PBW was noted in this subject which could explain the compensation of the gain in fat mass. These results confirm the findings of some authors who believe that BMI is particularly inappropriate as an indirect indicator of BC in RA.[34] In their study, Elkan AC et al, found that BMI failed to detect rheumatoid cachexia assessed by dual-energy X-Ray Absorptiometry.[10] Other authors had also reported normal BMI in subjects with both a low non-fat mass index and a high or very high body fat index.[35] But despite these limitations, BMI has a prognostic value in RA, and lean BMI patients have the highest CV risk as well as the highest rates of erosive disease.[36,37]

An increase in VFL would cause a decrease in PBW and this would promote an increase in blood LDL-cholesterol. In view of these findings, VFL and WC would be more associated with CV risk factors than BMI in RA.

In this study, more than half of the subjects (54.76%) had an MA greater than their actual age. Which would mean early physiological aging. In addition, an increase in BMI, WC and PBW would protect against this early physiological aging, which would be favored by excessive development of VFL. This results would fuel the conclusions of some authors who said that being fat is bad for the heart but good for the joints but provided that it's not accompanied by an excessive development of the VFL because in this case the cardiovascular risk would be similar to that of the general population with insulin resistance and increased cardiometabolic risks.[1,9]

Excess truncal and abdominal body fat has been suggested as the main factor responsible for the increased prevalence of insulin resistance and metabolic syndrome in RA, and visceral fat is more predictive of insulin resistance than subcutaneous fat.[38]

In authors study, authors were able to show in sub-Saharan African women with RA, an excess of development of the PFM without loss of MM. In contrast, a low PBW was noted in several subjects. In addition, authors were able to show early physiological aging during RA which would be favored by a high VFL. The study was carried out quickly, simply, non-invasively, non-dependent operator and especially inexpensive using a bio-impedance meter. It is a portable material and a highly acceptable methodology.[39] Most studies of BC in RA have as a measurement tool the dual-energy X-Ray Absorptiometry (DEXA), which until now has been the reference tool for evaluation of the BC. However, the major disadvantage of this method is its high cost. In this series, authors could have similar results by using a bio-impedance meter that allows to evaluate in a global and segmental way the muscular, fatty and bone compartment. Unlike the first bio-impedance meters, which admittedly made it possible to obtain an immediate mass non-fat but underestimated lean mass did not directly assess the muscle compartment or bone compartment and the results depended on the hydration status of the subject. Thus, they were not validated in chronic pathologies. Currently, impedance measurement could be an inexpensive alternative to DEXA. It could make the measurement of adiposity and sarcopenia feasible in the context of the management of cardiovascular risk factors in chronic inflammatory diseases at the occurrence of RA.

The limitations of this study lie in the fact that this cohort was reduced and that the duration of progression of RA in this patient was variable.

Adiposity and sarcopenia in RA are thought to be related to chronic inflammation induced by the disease itself.[21] The adipocytes themselves represent a potential source of pro-inflammatory cytokines aggravating systemic inflammation.[40] In addition, pro-inflammatory cytokines lead to peripheral insulin resistance that diverts nutrients to adipose tissue, which increases adiposity.

A peripheral distribution of this adiposity would be beneficial during the RA but its central location would be a source of cardiovascular morbidity and mortality and with a risk of early physiological aging. Activation of the immune system consumes a large amount of energy; inducing a hyper-metabolic and catabolic protein state accompanied by an increase in resting energy expenditure to the detriment of muscle mass despite appropriate food intake.[1,9,21,24] This results in a chronic consumption of muscle mass leading to a sarcopenic or even cachectic state.

Further studies are needed to determine during RA, on the one hand the effects of dual therapy corticosteroid and MTX on the excessive development of adipose tissue and on the occurrence of sarcopenia or even cachexia rheumatoid and secondly the implication of the loss of water mass on the decrease of the lean mass.

CONCLUSION

The evolution of RA can be characterized by modifications of the BC. From a practical point of view, the measurement of these changes would be useful in the follow-up of arthritis patients. Evaluation of BC is currently possible in clinical practice using a bio-impedance meter. Since impaired BC is a reflection of chronic metabolic disturbances, the BC study can facilitate the prevention of disability, cardiovascular risk factors, and mortality during RA. More interesting, treatments targeting BC could help reduce morbidity and mortality during RA. A study on a larger cohort would better anchor this result.

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REFERENCES

1. Nassar K, Janani S, Rachidi W, Mkinsi O. The assessment of muscle function and fall risk in rheumatoid arthritis: assessment tools and treatment effects. Rev Mar Rhum. 2013;25:20-7
2. Maradit-Kremers H, Nicola PJ, Crowson CS, Ballman KV, Gabriel SE. Cardiovascular death in rheumatoid arthritis: a population based study. Arthritis Rheum. 2005;52(3):722-32.
3. Doran MF, Pond GR, Crowson CS, O’Fallon WM, Gabriel SE. Trends in incidence and mortality in rheumatoid arthritis in Rochester, Minnesota, over a forty-year period. Arthritis Rheum. 2002;46(3):625-31.
4. Daïen CI, Fesler P. Rheumatoid arthritis: a cardiovascular disease?. Ann Cardio Angio. 2012;61(2):111-7.
5. Watson DJ, Rhodes T, Guess HA. All-cause mortality and vascular events among patients with rheumatoid arthritis, osteoarthritis, or no arthritis in the UK General Practice Research Database. J Rheumatol. 2003;30(6):11962-202.
6. Soubrier M, Barber Chamoux N, Tatar Z. Cardiovascular risk in rheumatoid arthritis. Joint Bone Spine. 2014;81(4):298-302.
7. Solomon DH, Curhan GC, Rimm EB, Cannuscio CC, Karlson EW. Cardiovascular risks in women with and without rheumatoid arthritis. Arthritis Rheum. 2004;50(11):3344-9.
8. Tantayakom P, Koolvisoot A, Arromdee E, Chiowchanwisawakit P, Muangchan C, Katchamart W. Metabolic syndrome is associated with disease activity in patients with rheumatoid arthritis. Joint Bone Spine. 2016;83(5):563-7.
9. Challal S, Minichiello E, Boissier MC, Semerano L. Cachexia and adiposity in rheumatoid arthritis. Relevance for disease management and clinical outcomes. Joint Bone Spine. 2016;83(2):127-33.
10. Elkan AC, Engvall IL, Cederholm T, Hafström I. Rheumatoid cachexia, central obesity and malnutrition in patients with low-active rheumatoid arthritis: feasibility of anthropometry, Mini Nutritional Assessment and body composition techniques. Eur J Nutr. 2009;48(5):315-22.
11. Fox KA, Després JP, Richard AJ, Bretté S, Deanfield JE. Does abdominal obesity have a similar impact on cardiovascular disease and diabetes?: a study of 91,246 ambulant patients in 27 European countries. Euro Heart J. 2009;30(24):3055-63.
12. Janssen I, Baumgartner R, Ross R, Rosenberg IH, Roubenoff R. Skeletal muscle cut points associated with elevated physical disability risk in older men and women. Am J Epidemiol. 2004;159(4):413-21.
13. Hume R, Weyers E. Relationship between total body water and surface area in normal and obese subjects. J Clin Pathol. 1971;24(3):234-8.
14. Ottaviani S. Obesity and rheumatoid arthritis. J Rheumat Monographs. 2016;83:29-33.
15. Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. Am J Clin Nutr. 2000;72(3):694-701.
16. Fantuzzi G. Adipose tissue, adipokines, and inflammation. J Allergy Clin Immunol. 2005;115(5):911-9.
17. Prado CM, Lieffers JR, McCargar LJ, Reiman T, Sawyer MB, Martin L, et al. Prevalence and clinical implications of sarcopenic obesity in patients with solid tumours of the respiratory and gastrointestinal tracts: a population-based study. Lancet Oncol. 2008;9(7):629-35.
18. Chung CP, Oeser A, Solus JF, Avalos I, Gebretsadik T, Shintani A, et al. Prevalence of the metabolic syndrome is increased in rheumatoid arthritis and is associated with coronary atherosclerosis. Atheroscl. 2008;196(2):756-63.
19. Tournadre A, Pereira B, Dutheil F, Giraud C, Courteix D, Sapin V, et al. Changes in body composition and metabolic profile during IL6 inhibition in rheumatoid arthritis. J Cachexia Sarcopenia Muscle. 2017;8(4):639-46.
20. Giles JT, Ling SM, Ferrucci L, Bartlett SJ, Andersen RE, Towns M, et al. Abnormal body composition phenotypes in older rheumatoid arthritis patients: association with disease characteristics and pharmacotherapies. Arthritis Care Res. 2008;59(6):807-15.
21. Roubenoff R, Roubenoff RA, Cannon JG, Kehayias JJ, Zhuang H, Dawson-Hughes B, et al. Rheumatoid cachexia: cytokine-driven hypermetabolism accompanying reduced body cell mass in chronic inflammation. J Clin Invest. 1994;93(6):2379-86.

22. Engvall IL, Elkan AC, Tengstrand B, Tengstrand B, Cederholm T, Brismar K, et al. Cachexia in rheumatoid arthritis is associated with inflammatory activity, physical disability and low bioavailable insulin-like growth factor. Scand J Rheumatol. 2008;37(5):321-8.
23. Butler RN. Did you say sarcopenia?. Geriatrics. 1993;48(2):11-2.
24. Hotamisligil GS. Inflammation and metabolic disorders. Nature. 2006;444(7121):860-7.
25. Kramer HR, Fontaine KR, Bathon JM, Giles JT. Muscle density in rheumatoid arthritis: associations with disease features and functional outcomes. Arthritis Rheum. 2012;64(8):2438-50.
26. Cojocarua M, Cojocaru IM, Silosi I, Vrabie CD. Metabolic Syndrome in Rheumatoid Arthritis. J Clin Med. 2012;7(2):148-52.
27. El Maghraoui A. Denutrition, cachexia and osteoporosis. Rev Rhum Monographique. 2013;80:100-4.
28. Toms TE, Panoulas VF, John H, Douglas KMJ, Kitas GD. Methotrexate therapy associates with reduced prevalence of the metabolic syndrome in rheumatoid arthritis patients over the age of 60 more than just an anti-inflammatory effect? A cross sectional study. Arthritis Res Ther. 2009;11(4):R110
29. Zonana-Nacach A, Santana-Sahagun E, Jimenez-Balderas FJ, Camargo-Coronel A. Prevalence and Factors Associated With Metabolic Syndrome in Patients With Rheumatoid Arthritis and Systemic Lupus Erythematosus. J Clin Rheumatol. 2008;14(2):74-7.
30. Dao HH, Do QT, Sakamoto J. Increased frequency of metabolic syndrome among Vietnamese women with early rheumatoid arthritis: a cross-sectional study. Arthritis Res Ther. 2010;12(6):R218
31. Crowson CS, Myasoedova E, Davis JM, Matteson EL, Roger VL, Therneau TM, et al. Increased Prevalence of Metabolic Syndrome Associated with Rheumatoid Arthritis in Patients without Clinical Cardiovascular Disease. J Rheumatol. 2011;38(1):29-35.
32. Raterman HG, Voskuy AE, Dijkmans BA, Nurmohamed MT. Use of methotrexate therapy is not associated with decreased prevalence of metabolic syndrome. Arthritis Res Ther. 2009;11(5):413
33. Lusa AL, Amigues I, Kramer HR, Dam TT, Giles JT. Indicators of walking speed in rheumatoid arthritis: relative influence of articular, psychosocial, and body composition characteristics. Arthritis Care Res. 2015;67(1):21-31.
34. Stavropoulos-Kalinoglou A, Metsios GS, Koutedakis Y, Nevill AM, Douglas KM, Jamurtas A, et al. Redefining overweight and obesity in rheumatoid arthritis patients. Ann Rheum Dis. 2007;66(10):1316-21.
35. Konijn NP, van Tuyl LH, Bultink IE, Lems WF, Earthman CP, van Bokhorst-de van der Schueren MA. Making the invisible visible: bioelectrical impedance analysis demonstrates unfavourable body composition in rheumatoid arthritis patients in clinical practice. Scand J Rheumatol. 2014;43(4):273-8.
36. Kremers HM, Nicola PJ, Crowson CS, Ballman KV, Gabriel SE. Prognostic importance of low body mass index in relation to cardiovascular mortality in rheumatoid arthritis. Arthritis Rheum. 2004;50(11):3450-7.
37. Kaufmann J, Kielstein V, Kilian S, Stein G, Hein G. Relation between body mass index and radiological progression in patients with rheumatoid arthritis. J Rheumatol. 2003;30(11):2350-5.
38. Stefan N, Haring HU. The metabolically benign and malignant fatty liver. Diabetes. 2011;60(8):2011-7.
39. Baumgartner RN, Chumlea WC, Roche AF. Bioelectric impedance phase angle and body composition. Am J Clin Nutr. 1988;48(1):16-23.
40. Escalante A, Haas RW, del Rincon I. Paradoxical effect of body mass index on survival in rheumatoid arthritis: role of comorbidity and systemic inflammation. Arch Intern Med. 2005;165(14):1624-9.

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