

Assessment of nutritional status by dual X-Ray absorptiometry in women with rheumatoid arthritis

A case–control study

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Abstract

Rheumatoid arthritis (RA) has been related to an impairment of the nutritional status. Body mass index (BMI) has been used but questions arise about how to properly evaluate nutritional status in RA patients. Few studies have evaluated it by dual-energy X-ray absorptiometry.

In women with RA, to analyze:

- (1) their nutritional status compared to that of a control population,
- (2) differences between the prevalence of impairment of nutritional status measured by dual energy X ray absorptiometry (DXA) and by classical methods used in clinical care,
- (3) determinants of nutritional status,
- (4) prevalence of sarcopenia.

Case–control study including 89 women with RA. The control group was composed by 100 patients affected by non-inflammatory rheumatic disorders. Study variables included age, RA duration, history, activity and disability, and in relation to nutritional status: BMI, serum albumin (ALB), whole body DXA assessment, and skeletal muscle index (SMI).

Mean age of patients was 62 ± 8 years, mean duration of RA was 14 ± 9 years, mean disease activity score (DAS28) was 3.7 ± 1.4 and mean Health Assessment Questionnaire was 0.88 ± 0.77 . BMI was $27.43 \pm 5.16 \text{ Kg/m}^2$ in patients and $27.78 \pm 3.98 \text{ Kg/m}^2$ in controls (P : ns). ALB was within normal range in all patients.

By whole body DXA, RA patients presented a statistically significant lower lean mass in all locations and lower fat mass in limbs than controls. Patients had a redistribution of fat mass to trunk. Lean mass directly correlated with fat mass.

Neither BMI nor ALB correlated with DXA parameters.

BMI, appendicular lean mass and SMI correlated inversely with disease duration. Trunk lean mass correlated inversely, and fat mass directly, with RA disability parameters.

RA patients fulfilled criteria of sarcopenia in 44% of cases versus 19% of controls ($P < .001$). In RA patients, regarding SMI, BMI showed a high specificity to detect sarcopenia (94% of the patients with low BMI had sarcopenia) but low sensitivity (47% of the patients with normal BMI or overweight had sarcopenia).

RA patients have an impairment of nutritional status associated to disease duration that looks like sarcopenia and that is not predicted by BMI.

Abbreviations: ACPA = anti-citrullinated protein antibodies, ALB = albumin, BMI = body mass index, DXA = dual-energy X-ray absorptiometry, MAC = midarm muscular circumference, RA = rheumatoid arthritis, SMI = skeletal muscle index, TSF = triceps skinfold, WBC = whole body composition.

Keywords: arthritis, nutritional, rheumatoid, sarcopenia, status

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1. Introduction

Rheumatoid arthritis (RA) is a chronic inflammatory process that involves the joints. It is the most common type of inflammatory arthritis in adults, affecting 0.5% [0.3–0.9] of the population in Spain^[1] and has been an important cause of disability and premature mortality.

RA has been related to an impairment of the nutritional status of multifactorial etiology. The old term “rheumatoid cachexia” originally described by James Paget in the 19th century^[2] and representative of a severe degree of malnutrition is nowadays almost in disuse. The tight control of inflammatory activity in the treat to target strategy applied currently in RA patients avoids the appearance of an evident nutritional impairment. However, the

nutritional impairment in rheumatic patients is still now a relevant comorbidity that we have to consider.

Nutritional status is the result of the balance between the dietary supply and the nutritional requirements necessary to go on with physiological activities, to compensate losses and to maintain reserves. Nutritional status is a good predictor of health.^[3] Many efforts have been made to diagnose malnutrition,^[4] nevertheless there is no unequivocal definition. It is generally accepted that it results in a redistribution of fat tissue, muscle mass and the appearance of hypoproteinemia.

Traditionally, antropometrical measurements, and biochemical parameters have been used to evaluate malnutrition.

There are several anthropometric methods for the study of the composition and quantification of body fat and muscle mass. The most commonly used are body mass index (BMI), midarm muscular circumference (MAC)^[5] and triceps skinfold (TSF)^[6]; it is generally accepted that BMI and TSF basically evaluate fat mass and MAC evaluates skeletal appendicular muscle mass.

Historically, biochemical evaluation for the assessment of the nutritional status had included serum proteins such as albumin (ALB), a protein that reflects the severity and the prognosis of malnutrition.^[7] No other biomarker has proved superiority over ALB for the evaluation of nutritional status.^[8]

Bioelectrical impedance has also been used for the assessment of the nutritional status.^[9] It is based on the application of an electric current of low potential and intensity at different frequencies that are transmitted in a different way through lean and adipose tissues. It is useful for determining the fat-free mass.

Finally, there is a growing consensus^[10] that whole body composition (WBC) analysis by dual-energy X-ray absorptiometry (DXA) is the gold standard to evaluate nutritional status. DXA measures body composition with great reproducibility and is sensitive to small changes. While different studies have generated curves that can be used as reference for different populations,^[11] there is still a lot of work to do in this area to determine universal cutoff points for healthy people.

There are very few data in the literature about nutritional status measured by DXA in RA patients. In this study, we have analysed the WBC by DXA in a group of Spanish patients with RA in order to improve the knowledge in this area.

2. Patients and methods

2.1. Study design

An observational case-control study was carried out.

2.2. Subjects recruitment

The study included women diagnosed with RA (along with 1987 ACR criteria),^[12] and most of them fulfilled also ACR 2010 criteria.^[13] Patients were recruited consecutively as they were routinely visited from the outpatient clinics in the rheumatology service of a university tertiary hospital in which around 900 RA are regularly evaluated. At inclusion, none of the patients had been recently hospitalized by a flare of the disease. Selected patients were not affected by any potentially consuming disease as neoplasms, cardiac or respiratory insufficiency or chronic liver or kidney diseases, and also other debilitating disorders such as VIH, eating disorders, or psychiatric disorders were excluded. Controls were recruited, age-matched to patients, consecutively, from the outpatient clinics during the same period of time. They were women with mild soft tissue rheumatism as tendonitis or osteoarthritis; the exclusion criteria were the same as in patients.

All patients and controls belonged to the same geographical area and came from a similar social status. The recruitment period took 6 months. All patients and controls gave their oral consent to participate in the study and the local evaluating committee gave the conformity for it, which means that an official and a written approval from the local Institutional Review Board was obtained.

3. Study variables

All the evaluations were made simultaneously.

3.1. Sociodemographic data and RA evaluation

- Sociodemographic data: date at birth

Evaluation of:

- (1) RA history: date at onset, disease duration, rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPA),
- (2) RA activity: tender joint count (TJC), swollen joint count (SJC), visual analog scale (VAS), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and disease activity score (DAS28), and
- (3) RA disability: Health Assessment Questionnaire (HAQ).

3.2. Nutritional status assessment

- Anthropometric data: BMI was calculated in both patients and controls. BMI is the ratio of human body weight to squared height expressed in kg/m². It has been categorized as follows: <18.5 kg/m² is considered underweight; from 18.5 to 25 kg/m², normal range; from 25 to 30 kg/m², overweight or moderately obese; and >30 kg/m², severely obese.
- Biochemical data: serum ALB was determined only in patients.
- WBC by DXA assessment was determined in patients and controls.
- The examinations were made with a densitometer Hologic QDR 4500 (Hologic Inc, Bedford, MA), recording fat and lean mass in arms, trunk, and legs. Quality controls are made weekly with a step phantom to guarantee the proper functioning of the technique. The estimated room occupancy time for a single exam is 8 to 10 minutes. This includes patient placement, image acquisition and generation of the analysis. The patient is placed supine, centered at the table with arms stretched to the sides of the body, hands looking at the legs without touching them and the thumbs upwards.
- Skeletal muscle index (SMI) was calculated in patients and controls to measure sarcopenia. SMI is established by the formula: appendicular skeletal muscle mass/height². According to Janssen et al,^[14] sarcopenia is defined as a relative SMI ≤5.75 Kg/m² in women and <8.50 Kg/m² in men.

4. Statistical analysis

To calculate the sample number, we considered the incidence of undernutrition in our general population and the calculated risk of undernutrition in RA patients (from the study of Helliwell et al).^[15] Accepting an alfa risk of 0.05 and a beta risk of 0, the sample needed was 80 patients per arm.

All the results were recorded in an Access 2003 database. The statistical software package used was Windows SPSS 15.0.

The results are presented as absolute numbers (percentage) or as mean ± standard deviation. Differences between groups were

Table 1
Clinical characteristics of RA patients (n: 89).

Disease duration (years)	13.7 ± 9.5 (1–42)
RF positive	65 (73%)
ACPA positive	66 (75%)
TJC	4.2 ± 6.3 (0–28)
SJC	1.2 ± 2.2 (0–10)
VAS general health patient	48.4 ± 25.9 (0–100)
ESR, mm/h	24.24 ± 15.65 (3–103)
CRP, mg/L	8.25 ± 10.96 (0.2–83)
DAS28	3.7 ± 1.39 (1.5–6.7)
<2.6 (remission)	21 (24%)
2.6–3.2 (low activity disease)	11 (12%)
>3.2 (moderate activity disease)	37 (41%)
>5.1 (high activity disease)	20 (23%)
HAQ	0.88 ± 0.74 (0–2.75)

ACPA=anti-citrullinated peptide antibodies, CRP=C-reactive protein, DAS28=disease activity score, ESR=erythrocyte sedimentation rate, HAQ=health assessment questionnaire, RA=rheumatoid arthritis, RF=rheumatoid factor, SJC=swollen joint count, TJC=tender joint count, VAS=visual analogue scale.

calculated by JI squared or analysis of variance. Spearman correlations were calculated where indicated. Statistical significance was established at *P* < .05.

5. Results

Eighty-nine patients and 100 controls were included in the study. Mean age of the patients was 62 ± 8 years without differences with controls (64 ± 9 years). The clinical characteristics of the patients are shown in Table 1. Most patients were seropositive

regarding RF and ACPA and had a moderate activity of the disease.

The results of nutritional assessment are detailed in Table 2. BMI was 27.43 ± 5.16 Kg/m² in the patients and 27.78 ± 3.98 Kg/m² in controls (*P*: ns). Eight percent of the patients versus 18% of controls were underweighted (*P* < .05).

All RA patients had normal levels of serum ALB. Whole body DXA results showed a decrease of lean mass in all locations and a decrease of fat mass in limbs in RA patients. Fat mass redistributed to trunk (*P* < .01) in RA patients although absolute trunk fat mass was not different between patients and controls.

Forty-four percent of the patients with RA and 19% of the controls had sarcopenia (*P* < .001).

BMI was very specific to detect sarcopenia in patients with RA (94% of patients with low BMI had sarcopenia), but not very sensitive (47% of patients with normal BMI or overweight had sarcopenia).

There were more patients than controls with sarcopenia evaluated by SMI, and the overweight subgroup assessed by BMI was significantly higher in patients than in controls. This group could correspond to patients with sarcopenic obesity.

In Table 3, we present the correlations between nutritional and RA variables. Appendicular lean mass and SMI correlated inversely with disease duration. Trunk lean mass correlated inversely, and fat mass directly, with RA disability parameters.

6. Discussion

In this study, we have analyzed by DXA the nutritional status of a group of Spanish women with RA representative of the RA population in a tertiary hospital, evaluating 2 body compart-

Table 2
Assessment of nutritional status in RA patients and controls.

	RA patients (n: 89)	Controls (n: 100)	<i>P</i>
Weight, Kg	65.51 ± 10.98	66.36 ± 14.76	ns
Height, cm	155.41 ± 6.35	156.9 ± 9.81	ns
BMI, Kg/cm ²	27.78 ± 3.98	27.42 ± 5.16	ns
<18.5, underweight	8%	18%	<.05
18.5–25, normal	15%	20%	
25–30, overweight	54%	33%	
>30, severely obese	23%	29%	
ALB, g/dL	40.44 ± 4 (28–48)	—	—
Fat left arm	2117 ± 899	2416 ± 966	<.01
Fat right arm	2434 ± 1187	2561 ± 1083	ns
Fat arms	4552 ± 2036	5146 ± 1982	<.05
Fat trunk	12016 ± 3793	11567 ± 4309	ns
Fat left leg	3962 ± 1303	4351 ± 1102	<.05
Fat right leg	4021 ± 1321	4453 ± 1173	<.05
Fat legs	7992 ± 2601	8865 ± 2183	<.05
Fat extremities	12561 ± 4231	13999 ± 3777	<.05
Fat total	25616 ± 7308	26424 ± 7467	ns
Lean left arm	1641 ± 360	1988 ± 400	<.001
Lean right arm	1911 ± 455	2222 ± 487	<.001
Lean arms	3552 ± 761	4219 ± 791	<.001
Lean trunk	18966 ± 2505	20557 ± 3677	<.01
Lean left leg	5277 ± 897	5987 ± 1076	<.001
Lean right leg	5455 ± 855	6081 ± 1076	<.001
Lean legs	10733 ± 1690	11913 ± 1700	<.001
Lean extremities	14286 ± 2301	16126 ± 2327	<.001
Lean total	37153 ± 4723	40943 ± 6419	<.001
SMI, Kg/m ²	5.93 ± 0.93	6.55 ± 0.9	<.001

ALB=albumin, BMI=body mass index, RA=rheumatoid arthritis, SMI=skeletal muscle index.

Table 3**Correlations between nutritional and RA variables in RA patients.**

	Lean extremities	Lean trunk	Fat extremities	Fat trunk	BMI	ALB	SMI
Age	ns	ns	ns	r: 0.20 <i>P</i> < .05	ns	ns	Ns
Disease duration	r: -0.27 <i>P</i> < .05	ns	ns	ns	ns	ns	r: -0.27 <i>P</i> < .05
TJC	ns	ns	ns	ns	ns	r: -0.37 <i>P</i> < .05	Ns
SJC	r: 0.22 <i>P</i> < .05	r: 0.26 <i>P</i> < .05	ns	ns	ns	ns	Ns
HAQ	ns	r: -0.28 <i>P</i> < .01	r: 0.31 <i>P</i> < .01	r: 0.28 <i>P</i> < .01	r: 0.44 <i>P</i> < .05	ns	r: 0.31 <i>P</i> < .05
CRP	ns	ns	ns	ns	ns	ns	Ns
ESR	ns	ns	ns	ns	ns	r: -0.37 <i>P</i> < .05	Ns
VAS	ns	ns	ns	ns	ns	ns	ns
DAS28	ns	ns	ns	ns	ns	ns	ns

ALB=serum albumin, BMI=body mass index, CRP=C-reactive protein, DAS28=disease activity score, ESR=erythrocyte sedimentation rate, HAQ=health assessment questionnaire, SJC=swollen joint count, SMI=skeletal muscle index, TJC=tender joint count, VAS=visual analogue scale.

ments: fat and lean mass. RA patients had a decrease of lean mass in all locations and fat mass in limbs with a redistribution of fat mass to trunk. The assessment of BMI or ALB was not useful to detect these alterations.

RA is a chronic disease characterized by a high inflammatory burden. Inflammation, secondary to an excess of production of inflammatory cytokines as tumor necrosis factor- α , interleukins 1 and 6 and others, accelerates protein catabolism. While the recent and widespread use of more effective therapies and a tight control of inflammation in RA have nearly extinguished rheumatoid cachexia, some degree of impairment of nutritional status is still present in RA patients and more studies are needed to address the problem.^[16,17]

In our study, we have found that RA disability is inversely correlated to lean mass and directly to fat trunk; also, that RA time of evolution correlates inversely with lean mass in limbs and SMI. More long and aggressive disease provokes a loss of muscle mass. In our cohort, we did not find correlation between inflammatory activity and nutritional parameters, but this is not contradictory because, in cross-sectional studies, parameters of inflammation represent a punctual moment and nutritional status, the burden of years of disease.

Rheumatoid cachexia or its current equivalent to a much lesser degree, loss of lean body mass, is under-recognized in clinical practice. Usually, it runs in parallel with an increased body fat mass, resulting in a normal BMI.^[18] This also involves an under-diagnosis of obesity when using the traditional values of BMI in well controlled RA patients, compared to DXA parameters.^[19] Stravroupulos-Kalinoglu et al pointed out that BMI could be an inexact tool to categorize the nutritional status in RA and that the standard cut-offs point should be modified.^[20] In the same sense, we have found that they don't always detect sarcopenia: BMI was very specific but not very sensitive to detect sarcopenia in patients with RA.

We found that controls and patients had globally a similar BMI, but comparing the subgroups there were remarkable differences in this nutritional parameter in the underweight and the overweight groups: controls had more underweight than RA patients (8% in RA, 18% in controls) and RA patients had more overweight than controls (54% in RA, 33% in controls).

In a previous study of our group, hypoalbuminemia was present and closely related to RA activity parameters,^[21] but this

was only seen in the patients with worse functionality and BMI, it does not seem useful in patients without clear clinical malnutrition.

Studies in the literature on nutritional status in RA are heterogeneous. The majority of them only measure either anthropometric or biochemical parameters, and very few assess whole body DXA. Furthermore, lately, they are centered more in obesity as an additional cardiovascular risk factor for RA patients or as a contributor, through the production of proinflammatory adipokines, to disease severity than in undernutrition.

Elkan et al evaluated body composition and nutritional status using DXA and bioelectrical impedance in 80 patients with RA (they were about 60 years old and had a mean disease duration of 6 years) and concluded that rheumatoid cachexia and central obesity were common by DXA and bioelectrical impedance and that BMI was unable to detect them. Cachexia, defined as fat-free mass index below the 25th percentile and fat mass index above the 50th percentile of a reference population, was not related to diet fat intake, but associated with high levels of LDL cholesterol and a high frequency of hypertension.^[22]

Popescu et al found that 107 RA women (they were about 56 years old and had a mean disease duration of 10 years) had a higher prevalence of normal weight obesity than controls. Disease duration and severity were associated with higher whole body and regional adiposity.^[23]

In a recent review^[24] in search of an association between obesity and RA activity, in 6 of 11 studies there was a significant direct correlation between clinical activity and BMI. The studies did not provide enough data to assess the relationship between body composition and clinical activity. In the only study in which DXA was used, the analysis was centered in obesity and associated cardiovascular risk.^[25]

Ngeuleu et al evaluated the prevalence of sarcopenia (defined as a SMI < 5.5 Kg/m² in women) in 123 patients with RA and the influence of sarcopenia on disease activity. Sarcopenia was prevalent and related to age, bone erosion, normal or overweight BMI, and high cardiometabolic risk according to waist circumference but not with disease activity.^[26]

Rheumatologist may need to assess nutritional status among other comorbidities in RA patients because chronic inflammation increases the metabolic index and nutritional requirements. When malnutrition is clinically evident is very difficult to correct

it, that is why is so important to have new methods to establish an early diagnose, obviously the sooner we can make the diagnosis, the better we can apply therapeutic strategies. Nevertheless, nowadays, is not common to see a high chronic inflammatory state that provokes an evident malnutrition in RA patients. Hence, the identification of mild impairment of nutritional status could be beneficial to avoid proinflammatory state associated to obesity and to reduce cardiovascular risk associated to RA, obesity, and sarcopenia.

A clear weakness of the study is that we did not analyzed how the different treatments that RA patients were receiving (as glucocorticoids or biological therapies) could have influenced in the nutritional status. Likewise, another weakness is the absence of an analysis of the exercise that both groups in the study were doing.

The usual methods used to evaluate nutritional status do not seem to be always suitable in RA patients: more specific measures of lean mass like whole body DXA are needed. Simple identification of patients eligible for DXA analysis is still needed to improve cost-effectiveness. Although measurement of body composition using DXA is clearly feasible, more studies are needed to fully understand the clinical benefits of this technique.

Author contributions

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