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Bone density, ultrasound measurements and body composition in early ankylosing spondylitis

E. Toussirot, F. Michel and D. Wendling

Department of Rheumatology, University Hospital Jean Minjoz, Bd A. Fleming, F-25030 Besançon cédex, France

Abstract

Objectives. In this cross-sectional study, we evaluated bone density using both dual-energy X-ray absorptiometry (DEXA) and quantitative ultrasound (QUS) techniques and examined the changes in body composition in patients with ankylosing spondylitis (AS).

Methods. Seventy-one patients were compared with seventy-one sex- and age-matched controls. Bone mineral density (BMD) was evaluated at the lumbar spine and femoral neck with a Lunar device. Total body measurements were also performed, giving BMD and bone mineral content (BMC) of the whole body, and fat and lean masses. Broadband ultrasound attenuation (BUA), speed of sound and stiffness were measured at the calcaneus using an Achilles ultrasound device.

Results. The patients had significantly lower lumbar spine, femoral neck and total body BMD as compared with controls (all $P < 0.05$). Total body BMC was also decreased in AS ($P = 0.002$). On the contrary, fat and lean masses did not differ between patients and controls as observed for QUS values. Mild to good correlations were found between BMD and QUS parameters (r ranging from 0.22 to 0.53; all $P \leqslant 0.01$). When applying the World Health Organization (WHO) definition for osteoporosis, we found that 46.5% of patients had lumbar spine osteopenia and/or osteoporosis, while 26.8% had femoral neck osteopenia and/or osteoporosis (controls: 23.9 and 10%; $P = 0.001$ and 0.08, respectively). No relationships between disease activity (as evaluated by erythrocyte sedimentation rate, serum C-reactive protein levels and BASDAI, a clinical index of disease activity) and BMD measurements were found and only femoral neck BMD correlated with disease duration ($r = -0.25$; $P = 0.04$). Finally, the presence of talalgia in AS did not influence the QUS values.

Conclusion. These results confirm that AS patients have decreased BMD values at both the spine and femur, and also in total body measurements, reflecting a generalized bone loss. On the contrary, soft tissue composition does not seem to be influenced by the disease. QUS parameters were found to be similar between patients and controls, suggesting that the QUS method did not provide additive information to DEXA. As it is thought that QUS provides information about qualitative properties of bone, the normal results of QUS values in our patient series argue against modifications in AS bone micro-architecture.

KEY WORDS: Bone mineral density, Ultrasound measurements, DEXA, Ankylosing spondylitis, Body composition.

Ankylosing spondylitis (AS) is an inflammatory rheumatic disease with spine and sacroiliac joint involvement that mainly affects young male subjects. Typical clinical features include sacroiliac joint pain and backaches, and progressively, the patient may develop dorsal kyphosis. Specific spine ossifications or syndesmophytes are considered to be a hallmark of the disease, reflecting a process of bone formation. However, AS may also be characterized by a reduction in bone formation [1]. Indeed, several reports have described osteoporosis as a complication of AS. In this regard, patients with established AS had a higher incidence of vertebral crush fracture [1, 2], bone mineral density (BMD) at the spine and the femoral neck was decreased and this bone loss was even observed in early stages of the disease without syndesmophytes [3]. However, the pathogenesis of this osteoporosis still remains unclear. The reduced range of movement of the spine in ankylosed patients, the treatment given [non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroids] and the inflammatory cytokines could be involved in this bone loss [4]. Additionally, recent reports suggested increased bone turnover in AS. Indeed, urinary excretion of markers of collagen breakdown or pyridinium cross-links was

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Correspondence to: E. Toussirot.

found to be increased in some AS patients, mainly those with active disease and elevated levels of acute-phase reactants [5].

Several measurement techniques have been developed to assess BMD [6]. The most popular is dual-energy X-ray absorptiometry (DEXA) and this accurate technique can measure BMD at specific fracture-related skeletal sites such as the spine, hip and radius. Recently, quantitative ultrasound (QUS) has generated widespread interest [6, 7]. Indeed, this method offers several advantages over DEXA: it is more simple to use than DEXA, the test is rapid and radiation free, and it provides information about the quality of bone. Indeed, DEXA only measures bone density which accounts for about 70% of bone strength. However, bone fragility and consequently fractures also depend on bone micro-architecture, which is not assessed by conventional DEXA [6]. Qualitative aspects of bone, such as elasticity, and micro-architectural characteristics could be assessed by QUS. Correlations between bone mechanical indices and ultrasound parameters have been found in *in vitro* experiments and QUS is able to discriminate between patients with osteoporotic fractures and non-fractured subjects [7, 8]. In AS, BMD has mainly been evaluated using DEXA, and QUS has never been used. Thus, no qualitative bone data are yet available.

It is now possible to evaluate bone mass at different skeletal sites using total body DEXA measurements [9]. With this technique, the soft tissue composition and the ratio of fat mass/lean mass could be determined. Thus, this new method provides information concerning bone mass at specific skeletal sites and the influence of a disease or its treatment on bone and soft tissue. For instance, rheumatoid arthritis is characterized by generalized osteoporosis, decreased lean mass and abdominal shift of fat mass [10]. Moreover, fat and fat-free masses could influence bone mass. Bone loss has been well reported in AS, but the influence of the disease and its treatments on total body and soft tissue composition have not yet been studied.

In this study we examined bone density using both DEXA and QUS techniques in order to determine the usefulness of each method; we also aimed to determine the influence of AS on body composition.

Patients and methods

Patients

A cross-sectional study was conducted. Between 1997 and 1999, 71 White out-patients consecutively seen in our department were enrolled. Each patient responded to the modified New York criteria for AS [11]. Clinical assessment included demographic data: age, sex, weight, height, body mass index (BMI: weight/height^2; kg/m^2) and disease duration. Back pain, vertebral stiffness, peripheral involvement, extra-articular manifestation (uveitis) and history of talalgia (heel pain) were also recorded. The patients were assessed by two physicians for Schober's test measurement (ET, DW). This study group was analysed for sacroiliac joint changes (sacroiliitis, grades according to the New York scale [11]) and for the presence of syndesmophytes on postero-anterior and lateral dorsal and lumbar spine standard X-rays. The presence of vertebral fracture (defined as 20% reduction in body vertebral height at any edge [12]) was also examined. A clinical index of disease activity (BASDAI; Bath Ankylosing Spondylitis Activity Index) was also evaluated [13]. Laboratory activity was assessed by the Westergren erythrocyte sedimentation rate (ESR) and acute-phase reactants [serum C-reactive protein (CRP) levels]. Biological assessment also included HLA B antigen determination for the presence of HLA-B27. Patients excluded from this study corresponded to post-menopausal women, and those with a condition which might alter bone mineral content (BMC) and/or metabolism (alcoholism, history of habitual smoking, liver and kidney disease, Paget's disease, hypogonadism, hyperthyroidism, hyperparathyroidism, ongoing corticosteroid therapy, thyroxine and anti-convulsants). In this study, only AS patients without an associated condition were assessed. Consequently, no patient had psoriasis, history of reactive arthritis or inflammatory bowel disease.

Controls

The control group corresponded to 71 healthy White subjects (hospital staff) without a history of inflammatory rheumatic disease or a condition responsible for bone loss. The exclusion criteria were the same as the patient group. The controls were age and sex matched to the patients.

Methods

BMD. Measurements of BMD of the L2–L4 lumbar spine and the left femoral neck were carried out using a Lunar DPX-IQ (Lunar, Madison, WI, USA). The results were given as BMD (g/cm^2) and T score which corresponded to the number of standard deviations (S.D.) from any result from the peak bone mass-related population (the normal ranges were provided by the manufacturers of the bone densitometer). The precision error was 1% for the lumbar spine and 1.5% for the femoral neck. According to the World Health Organization (WHO), osteopenia was defined as a T score between -1 and -2.5 S.D. and osteoporosis as a T score below -2.5 S.D. [14].

Body composition. A total body scan was performed using the same Lunar densitometer, evaluating BMD and BMC (g). Measurements were given for body composition from the total body scan with lean mass (g) and fat mass (g). The reproducibility for total body measurements was 0.7%.

QUS. QUS measurements of the right calcaneus were performed using an Achilles + device (Lunar). The left heel was evaluated in the case of unilateral right foot pathology (ankle oedema, trauma or fracture, reflex sympathic dystrophy). The patient's heel was positioned in a small temperature-controlled warm

bath (37°C) to avoid attenuation of ultrasound by air. The device uses a transmitting transducer, with a central frequency of 0.5 MHz, which is electrically excited to produce a broadband spectrum. The ultrasonic wave is transmitted through the heel, and detected by a receiving transducer. Three parameters were measured: (1) broadband ultrasound attenuation (BUA; dB/MHz) which corresponds to the frequency-dependent attenuation of the ultrasonic wave as it passes through the heel; (2) speed of sound (SOS; m/s), the velocity of the ultrasonic wave as it passes through the heel; and (3) stiffness, a combination of the two previous parameters and calculated as follows: 0.67 BUA + 0.28 SOS − 420 (this parameter does not reflect the homonymous mechanical property). This index was established by Lunar. The coefficient of variation (CV), calculated in 10 healthy volunteers measured on five occasions consecutively was 6.7% for BUA, 3.5% for SOS. The CV values given by the manufacturer were: BUA 1.7% and SOS 0.3%.

Statistical analysis

The results were given as mean ± S.D. Statistical significance between patients and controls was estimated by Student's *t*-test. Qualitative data were analysed by the χ^2 test. Simple linear regression was used to study the relationships between DEXA and QUS parameters and between BMD, BMC, ultrasonic values and indices of clinical (BASDAI) or laboratory activity (ESR and CRP). Correlation coefficients were also obtained between BMD measurements and disease duration. The data were stratified for lumbar spine and femoral neck BMD according to the WHO definition and each ultrasound measurement was compared between the three groups of patients (i.e. normal, osteopenia, osteoporosis) using one-way analysis of variance (ANOVA). AS patients could have talalgia that could modify the QUS measurements. Thus, ultrasonic parameters were analysed after stratification of patients according to the presence or the absence of heel pain. ANOVA was also applied between these patient groups and the controls. The significant level was 0.05 and the Statview software (Alsyd SAS, Meylan, France) was used for these statistical tests.

Results

The demographic, clinical and radiological characteristics of the patient group are listed in Table 1. The demographic variables (age, sex ratio, weight, BMI) were similar between patients and controls ($P > 0.05$), except height (AS *vs* controls: 169.1 ± 9.3 *vs* 172.5 ± 7.8 cm; $P = 0.02$; Table 2). All patients had radiological evidence of sacroiliitis and dorsolumbar X-rays showed that only 19 (26.7%) had syndesmophytes. Because the AS patients had a mean disease duration of approximately 10 yr and most of them had no syndesmophyte formation, this cohort could be considered as reflecting early AS. Only one patient (age: 49 yr; disease duration: 14 yr) had had a vertebral fracture (transdiscal fracture after falling). His lumbar spine T score was 0.7, while his femoral neck T score was −2.4. Twenty-four patients (33.8%) had AS with peripheral arthritis at the time of examination.

The lumbar spine and femoral neck BMD were lower in the patients compared with the controls (AS *vs* controls: all $P \leqslant 0.01$; Table 3). The corresponding T scores were also decreased in the patients (AS *vs* controls: all $P < 0.01$; Table 3).

The QUS parameters were found to be slightly decreased in the patients, but the results did not reach significance (all $P > 0.1$; Table 3).

Total body measurements (BMD and BMC) were also lower in the patient group (AS *vs* controls: $P = 0.03$ and 0.002, respectively). By contrast, no difference was found between patients and controls for lean and fat masses (Table 3).

When applying the WHO definition of osteoporosis for lumbar spine BMD, we found that 53.5% of patients had normal values, 32.4% had osteopenia and 14.1% had osteoporosis (control series: normal results = 76.1%; osteopenia = 23.9%; osteoporosis = 0%; $P = 0.001$). At the femoral neck, a similar trend was observed, but without significance (AS *vs* controls:

TABLE 1. Clinical, biological and radiological characteristics of AS patients ($n = 71$)

	Mean	Range	Median	S.D.
Age (yr)	39.1	20–67	38	11.5
Sex	49 male, 22 female			
Disease duration (yr)	10.6	1–29	7	8.3
Schober test (cm) (0–6)	2.9	0–6	3	1.6
Sacroiliitis	Grade 2: 43 (60.5%) Grade 3 or 4: 28 (39.5%)			
Dorsolumbar syndesmophytes	19/71 (26.7%)			
ESR (mm/h)	27.7	1–98	20	26.0
CRP (mg/l)	23.7	0–126	12.5	28.1
HLA-B27	60/71 (84.5%)			
BASDAI (0–10)	5.2	0–9.5	4.9	2.2
Axial/peripheral disease	47/24			
Talalgia	21/71 (29.5%)			
Uveitis	18/71 (25.3%)			

normal: 73.2 *vs* 85.9%; osteopenia: 22.5 *vs* 14.1%; osteoporosis: 4.3 *vs* 0%; $P = 0.08$). In the patient group, the values for ultrasound measurements at each stratum of BMD for the lumbar spine and femoral neck are shown in Table 4. Apart from the BUA values in the lumbar spine BMD groups (normal, osteopenia and osteoporosis), the ultrasound data were generally lower in the osteoporotic patient group (all $P \leqslant 0.01$).

We also examined the relationships between DEXA measurements and QUS values. Mild to good correlations were found between lumbar spine, femoral neck and total body BMD and the different QUS variables (r ranging from 0.22 to 0.53 with all $P \leqslant 0.01$; Table 5). Conversely, there were no clear statistically significant correlations between measured BMD at any site (lumbar spine, femoral neck and total body) and variables of disease activity including ESR, serum CRP levels and BASDAI (data not shown, all $P > 0.05$). The correlation coefficients between BMD values and disease duration were also obtained and only a correlation between femoral neck BMD and disease duration was found ($r = -0.25$; $P = 0.04$).

We finally studied the influence of talalgia on QUS measurements: there was no difference in the QUS values between the patient subsets and the controls (all $P > 0.05$; Table 6).

Table 2. Comparative demographic data of AS patients and controls

	AS ($n = 71$)	Controls ($n = 71$)	P
Age (yr)	39.1 ± 11.5	37.3 ± 10.5	NS[a]
Sex (male/female)	49/22	49/22	NS[b]
Weight (kg)	68.6 ± 13.6	71.2 ± 13.5	NS[a]
Height (cm)	169.1 ± 9.3	172.5 ± 7.8	0.02[a]
BMI (kg/m^2)	23.8 ± 3.8	23.8 ± 3.7	NS[a]

[a]Student's t-test; [b]χ^2 test.

Discussion

This study was undertaken on a large series of patients with early AS in order to determine bone density using two methods, DEXA and QUS. At the same time, body composition was also evaluated.

Our results confirm previous DEXA measurements in AS [1–5] and show that BMD was decreased at both the lumbar spine and the femoral neck. Only a few patients had syndesmophytes (26.7%) which could falsely

Table 3. BMD, BMC and ultrasonic measurements in AS patients and controls

	AS 2 ($n = 71$)	Controls ($n = 71$)	P[a]
DEXA			
Lumbar spine			
BMD (g/cm^2)	1.08 ± 0.17	1.18 ± 0.13	0.0002
T score	−0.91 ± 1.42	−0.06 ± 1.05	<0.0001
Femoral neck			
BMD (g/cm^2)	0.97 ± 0.16	1.04 ± 0.13	0.01
T score	−0.31 ± 1.25	0.23 ± 1.04	0.006
Total body			
BMD (g/cm^2)	1.21 ± 0.11	1.25 ± 0.1	0.03
BMC (g)	2884.7 ± 536.7	3183.6 ± 550.9	0.002
Lean mass (g)	65 400.4 ± 13 633.1	68 434.2 ± 12 913.0	NS
Fat mass (g)	17 884.1 ± 6901.3	17 746.7 ± 6279.2	NS
Ultrasonic parameters			
BUA (dB/MHz)	122.2 ± 12.8	123.9 ± 12.6	NS
SOS (m/s)	1556.9 ± 40.6	1565.6 ± 36.4	NS
Stiffness	97.3 ± 17.7	100.9 ± 17.2	NS

[a]Student's t-test.

Table 4. Ultrasonic values in AS according to the lumbar spine and femoral neck BMD and WHO definition of osteoporosis

Lumbar spine T score	Normal ($n = 38$)	Osteopenia ($n = 23$)	Osteoporosis ($n = 10$)	P[a]
BUA (dB/MHz)	124.3 ± 12.7	118.7 ± 15	120.6 ± 13.8	NS
SOS (m/s)	1572.5 ± 38.4	1542.1 ± 37.9	1529.3 ± 26.8	0.001
Stiffness	103.0 ± 16.1	90.9 ± 17.8	88.5 ± 16.3	0.009
Femoral neck T score	**Normal ($n = 52$)**	**Osteopenia ($n = 16$)**	**Osteoporosis ($n = 3$)**	**P[a]**
BUA (dB/MHz)	125.2 ± 11.4	117.4 ± 13.0	100.6 ± 9.1	0.0008
SOS (m/s)	1567.1 ± 37.5	1534.8 ± 39.9	1516.7 ± 26.0	0.004
Stiffness	102.1 ± 15.4	88.1 ± 18.1	71.7 ± 11.5	0.0004

[a]ANOVA.
Normal, T score > -1 S.D.; osteopenia, T score -2.5 to -1 S.D.; osteoporosis, T score < -2.5 S.D.

Table 5. Correlations between BMD and ultrasound parameters in patients and controls

BMD	BUA	SOS	Stiffness
Lumbar spine	0.22 ($P = 0.01$)	0.34 ($P < 0.0001$)	0.31 ($P = 0.0002$)
Femoral neck	0.44 ($P < 0.0001$)	0.51 ($P < 0.0001$)	0.53 ($P < 0.0001$)
Total body	0.44 ($P < 0.0001$)	0.49 ($P < 0.0001$)	0.51 ($P < 0.0001$)

Table 6. QUS measurements in patients with and without talalgia and controls

Ultrasonic parameters	AS with talalgia ($n = 50$)	AS without talalgia ($n = 21$)	Controls ($n = 71$)	P[a]
BUA (dB/MHz)	122.7 ± 12.4	120.9 ± 13.9	123.9 ± 12.6	NS
SOS (m/s)	1561.9 ± 40.4	1544.9 ± 39.3	1565.6 ± 36.4	NS
Stiffness	99.1 ± 17.3	93.1 ± 18.2	100.8 ± 17.2	NS

[a]ANOVA.

increase the lumbar spine BMD, but this was not observed in our series. A similar decrease in BMD and BMC was found in total body evaluations.

The WHO criteria for osteoporosis may only be applied to White women [14] and AS is a disease mainly involving male patients. Despite this limitation, we applied these criteria and observed that a moderate proportion of our patients had lumbar spine and femoral neck osteopenia and/or osteoporosis (46.5 and 26.8%, respectively), but difference with the control group was only significant for the lumbar spine. These data highlight the need for a control population when evaluating BMD in a disease population such as AS.

Only one patient had a history of fracture (transdiscal fracture). No patient had vertebral deformity suggestive of vertebral fracture and, thus, the vertebral fracture prevalence in our series could be estimated to be 1.4%. However, only the dorsolumbar spine radiographs were analysed in this study and appendicular skeleton fractures were not systematically examined and, thus, were not excluded. Our patients were smaller than the controls, but this could not be seen as an indirect reflection of vertebral deformity, but rather connected to the dorsal kyphosis of the disease.

Total DEXA scans in AS patients clearly showed that BMD was also decreased in the total body. The BMC results also showed the influence of the disease over the total body. These results suggest that the bone loss may be considered as generalized and not restricted to the spine or the femoral region. Conversely, the lean and fat masses were found to be equivalent in the studied groups, suggesting that the disease has no influence on soft tissue composition. Because fat mass was not impaired in our patients, it could not be considered as a contributing factor in the bone impairment of the disease. However, our series did not include patients with criteria of severe disease such as severe extra-articular manifestations. Thus, a possible loss of lean and/or fat mass could not be excluded in such patients with severe disease.

In this study, we also evaluated the QUS parameters. A slight decrease in all the ultrasonic parameters was observed, but without significance. This could be interpreted as a lack of sensitivity of this method to discriminate between patients with osteopenia and/or osteoporosis and non-osteoporotic patients. We previously checked the relationships between DEXA measurements and QUS variables and found strong correlations between these parameters at all sites (lumbar spine, femoral neck and total body). This is in keeping with previous published data on the relationships between DEXA and ultrasonic measurements [7, 15]. Similarly, a strong correlation exists between BMD at the calcaneus and ultrasonic variables, and lesser but good correlations have usually been found between lumbar spine, femoral neck BMD and QUS parameters [7]. In our study, we did not have available measurements of BMD of the calcaneus that might have given us better knowledge about the relationship between QUS and BMD measurements. At the present time, QUS generates widespread interest as it gives a quick evaluation of bone and it is believed to provide some information concerning the structural organization of bone [6, 7]. Indeed, experimental studies on human cancellous bone specimens have shown that BUA is influenced by structural factors such as pore size and numbers, and that SOS is determined by bone density and elasticity and bone architecture [7]. There were several studies which determined the usefulness of QUS measurements in predicting the risk of fractures in elderly women and it is also known that QUS variables decline with age [7, 16, 17]. However, the definition of osteoporosis and/or osteopenia using ultrasonic variables is still lacking and the diagnosis of osteoporosis and the monitoring of skeletal changes are other areas of research for the clinical use of QUS. Thus, it is thought that QUS can evaluate bone architecture and thus gives more information than DEXA. However, it has been shown to be a suitable method for predicting risk fractures and was important in patients with established osteoporosis and fractures. Conversely, in postmenopausal women without fractures, this method did not give additional information about patterns of bone loss [16]. Therefore, QUS assessment seems interesting for screening patients with advanced bone loss. Our patient population was young (mean age: 39.1 yr) and

had never had fractures (except one patient), and this probably explains the lack of difference in QUS values between AS and controls. However, all the ultrasound values were lower in the osteoporotic groups (except BUA in the lumbar spine osteoporotic group). This could be related to the good correlation coefficients between BMD measurements and QUS values (r between 0.22 and 0.53) and thus, could only reflect the decreased BMD of the patients. Additionally, because QUS parameters were similar between patients and controls, it is tempting to speculate about the absence of bone architecture modifications in AS. However, an increased fracture risk is admitted in AS [1, 2] and, thus, the normal findings of calcaneal ultrasound measurements in AS suggest that this higher fracture risk could only be related to the decline in BMD.

There are few data about histomorphometric changes in AS. One study evaluated the histomorphometric variables of 16 White men with AS and found osteopenia and mineralization defects [18]. In the static variables were noted significantly reduced trabecular wall thickness, trabecular plates and wall thickness, some changes suggestive of micro-architecture alterations. However, Hans *et al.* [19] failed to find any difference between histomorphometric and ultrasound parameters, apart from a relationship with bone quantity.

AS patients could present talalgia in the course of their disease, reflecting inflammation of the enthese structures. As a consequence, a localized bone sclerosis could occur and could be an artefact for QUS measurements. In this study, we did not perform heel X-rays in each patient, but the history of talalgia was recorded. We thus examined the QUS parameters between patients with and without talalgia, and controls, and failed to find a difference, ruling out an effect of heel inflammation on BUA and SOS values.

As previously observed, we did not find a correlation between indices of disease activity (ESR, CRP levels and BASDAI) and BMD measurements at all sites [4, 5]. Most studies which have investigated a relationship between bone loss and inflammatory activity in AS have yielded negative results and this could be explained by the characteristics of the evaluated variables: inflammation parameters are assessed at the moment of the study while BMD is a longitudinal variable [4]. On the contrary, bone remodelling markers such as pyridinium cross-links reflect the bone degradation at the time of assessment and the urinary excretion of these collagen compounds has been found to correlate with disease activity [5].

The relationship between BMD and disease duration in our series showed that only the femoral neck was related to the chronicity of the disease. However, it has been reported that AS patients with short disease duration could present bone loss, suggesting that the chronicity of the disease is probably not involved in this bone loss [3].

On this large series of AS patients, we can conclude that patients had lowered lumbar spine, femoral and also total body BMD. Conversely, soft tissue composition did not seem to be involved in the disease process. It has been hypothesized that inflammatory cytokines [interleukin-6, tumour necrosis factor α (TNFα) or interleukin-1] may play a role in the inflammatory process of AS and they are probably involved in this bone loss [4, 20]. By contrast, these inflammatory cytokines do not probably play an important role in soft tissue composition, as no loss of lean/fat mass was observed in our study. According to the WHO definition, a moderate proportion of patients had osteopenia more frequently than osteoporosis and, thus, the bone loss in AS should be considered as mild with probably a moderate risk for fracture. However, the exact prevalence of fracture in AS requires further longitudinal studies. Our results also suggest that the QUS method did not provide additive information to DEXA and this could be related to the absence of bone architecture changes in this inflammatory rheumatic disease.

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