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Joint Bone Spine 70 (2002) 46–51
JOINT BONE SPINE
www.elsevier.com/locate/bonsoi

Original article

Is ultrasound of bone relevant for corticosteroid-treated patients? A comparative study with bone densitometry measured by DEXA

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Received 25 March 2001; accepted 8 March 2002

Abstract

Corticosteroid treatment diminishes bone mass and alters bone quality. *Objective*: to evaluate bone in corticosteroid-treated patients and controls and in fractured and non-fractured patients treated with corticosteroids using both X-ray densitometry (DEXA) and ultrasound. We evaluated 34 women aged 58 ± 14 years (X ± SD), who had been on long-term low dose prednisone therapy for at least 6 months, and who had never received specific treatment for osteoporosis. Bone mineral density of total skeleton (TS), lumbar spine (LS), femoral neck (FN), and vertebral morphometry (MXA) were measured by DEXA. Speed of sound (SOS), broadband ultrasound attenuation (BUA) and Stiffness were measured using an Achilles Plus system. Forty-two healthy women served as controls. Both densitometric and ultrasound parameters in the patients were significantly diminished compared with controls: TS: $P < 0.002$, LS: $P < 0.025$, FS: $P < 0.005$, Stiffness: $P < 0.001$, BUA: $P < 0.002$ and SOS: $P < 0.002$. The percentage of patients with a Z score below –2 was higher in Stiffness and BUA: 38% and 47%, respectively, compared with a range of 16–24% in the other parameters ($P < 0.05$ BUA vs. DEXA measurements). Eleven patients with previous bone fracture had values lower than the non-fractured patients, both according to DEXA and ultrasound measurements, but the difference was only significant for BUA ($P < 0.02$). BUA of the calcaneus was more effective in detecting the specific skeletal alterations and fracture risk of the group of patients receiving chronic corticosteroid treatment.

Keywords: Corticosteroids; Osteoporosis; X-ray densitometry; Ultrasound

Mots clés : Corticostéroïdes ; Ostéoporose ; Rayon X Dentisometrie ; Ultrasons

1. Introduction

Corticosteroid treatment diminishes bone mass [1–4], and patients under chronic steroid treatment, even during the early stages of treatment, have a higher risk of fractures than the control population [1,5,6]. Most studies have used dual photon X-ray absorptiometry (DEXA) to measure bone density [1–4,6]. However, bone strength, which is mostly dependent on bone mass, is also influenced by qualitative factors (such as architecture and physical properties). In vitro studies have suggested that ultrasound (US) methods could provide information about bone architecture in addition to bone density [7–9]. Bone quality may be more affected than bone mass since the frequency of vertebral fractures reported in corticosteroid-treated patients proved higher than expected for their bone mineral density (BMD) [10,11]. This concept has recently been reinforced by a recent communication showing that women taking corticosteroids have an elevated fracture risk even after adjustment for BMD [6]. Other authors have reported no difference in fracture threshold between patients receiving oral corticosteroids and patients with primary osteoporosis [12].

The aim of the present study was to evaluate bone in corticosteroid-treated patients and controls and in fractured

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(Fx) and non-fractured (non-Fx) patients treated with corticosteroids using both DEXA and US, in order to compare the efficiency of these two methods to detect bone alterations.

2. Patients and methods

2.1. Patients

Patients were recruited at the outpatient service of the Centro de Osteopatías Médicas. The protocol used in this study was approved by the Ethics Committee of the Institution.

Forty-two women receiving long-term low-dose prednisone therapy (<10 mg/d) for at least 6 months and who had received no specific treatment for osteoporosis (bisphosphonates, hormone replacement, fluoride, vitamin D analogs or calcitonin) were asked to participate in the study. Thirty-six patients agreed to participate, two of whom were excluded from the study due to lack of information on previous treatments and doses. All 34 patients gave informed consent. The patients' diagnoses were: rheumatoid arthritis (n = 24), polymyalgia rheumatica (n = 7) and systemic lupus erythematosus (n = 3). All the subjects had received an average dose of 6.4 ± 2.7 mg/d (X ± SD) of prednisone during the previous year. The cumulative dose of corticosteroids was: median (interquartile range) 14500 mg (4750–39300). Eleven patients presented fractures during the corticosteroid treatment period. A total of 14 fractures were reported in pelvis: 2, elbow: 2, metatarsus: 2, knee: 1, humerus: 1, ankle: 1, and vertebral abnormalities: 5 (three deformed, two fractured) detected by MXA (see Section 2.2). In the analysis of Fx patients vs. non-Fx, all the above-mentioned patients were included as Fx, including those with non-typical osteoporotic fractures such as elbow, knee and ankle because the fractures occurred during corticosteroid treatment.

The control group included 42 healthy women who volunteered for the study. Volunteers under 60 years of age were recruited among hospital staff, and those between 60 and 80 were relatives of the volunteer staff members of our Section members. Control patients were free of osteoporosis, hyperparathyroidism, osteomalacia, thyroid dysfunction, and other diseases affecting bone metabolism. None of the control subjects was receiving corticosteroids, thyroid hormone, bisphosphonates or fluoride, nor did they exhibit vertebral fractures on plain radiographs of the spine. The number of premenopausal women was similar in both groups (n = 5 in each one). Features of both groups are described in Table 1.

Table 1
Clinical features of patients receiving corticosteroids (CS) and of the control group. Bone mineral density (X ± SD) (g/cm^2) of total skeleton, lumbar spine, femoral neck, and ultrasound parameters: Stiffness (%), broadband ultrasound attenuation (BUA, dB/MHZ) and speed of sound (SOS, m/s) in both groups

	CS group	Control group	P
N	34	42	
Age (years)	58.1 ± 14.2	60.5 ± 5.8	NS
Weight (kg)	63.3 ± 12.3	66.6 ± 8.8	NS
Height (cm)	157.0 ± 5.9	157.1 ± 5.8	NS
Premenopausal (n)	5	5	NS
BMI (kg/cm^2)	25.7 ± 4.9	26.9 ± 3.4	NS
Total Skeleton	0.977 ± 0.09	1.048 ± 0.09	0.002
Lumbar Spine	0.965 ± 0.13	1.051 ± 0.16	0.025
Femoral Neck	0.764 ± 0.16	0.854 ± 0.13	0.005
Stiffness	64.0 ± 15.7	77.2 ± 15.6	0.001
BUA	94.3 ± 13.7	104.2 ± 14.9	0.002
SOS	1504 ± 29	1526 ± 28	0.002

2.2. Methods

2.2.1. Densitometric and ultrasound measurements

BMD of the lumbar spine (LS), femoral neck (FN) and total skeleton (TS) was measured using DEXA (Lunar Expert, Madison W). Calibration was performed by measuring a phantom everyday to assess long-term reproducibility for each system. Long-term precision in vivo was determined as a percentage coefficient of variation (CV) of repeated US and DEXA measurements in 15 normal subjects, three times over one month period. The in vivo CV obtained from each area was: LS: 1.0%, FN: 1.8%, and TS: 0.9 %. US measurements were performed on the calcaneus using the Achilles plus system (Lunar Madison W). This system allows measuring broadband ultrasound attenuation (BUA) in dB/Mhz, speed of sound (SOS) in m/s, and a third parameter derived from the combination of BUA and SOS: the Stiffness Index. The CVs for the Achilles Plus were: 2.6% for BUA, 0.4% for SOS and 3.1% for Stiffness [7]. The Z score of the US and DEXA measurements was calculated for each patient using the following formula:

$$\text{Z score} = \frac{\text{patient value} - (\text{X value for sex and age})^*}{\text{SD for sex and age}} \quad (1)$$

*The mean value was obtained from a previously reported reference population [13,14].

2.2.2. Vertebral morphometry (MXA)

The presence of deformed or fractured vertebrae was evaluated by MXA, a recently developed method to determine vertebral deformities. The Expert XL uses a high resolution array detector coupled to an X-ray tube in fan-beam geometry. Scanning time from T4 to L4 is less than 1 min and low radiation is used. In all the patients, it was possible to visualize at least from T6 downward. The software (version 1.62) automatically places the points in the superior, middle, and inferior endplate of the vertebrae. Additionally, the anterior/posterior ratio, middle/posterior ratio and the average of the three heights (anterior, middle and posterior) are automatically calculated by the software [16]. The expected heights are normalized in each case for total height of the L2–L4 sequence. The Z score was calculated using the reference values for heights and ratios [15]. Vertebrae were con-

Table 2
Correlation coefficients between DEXA and ultrasound parameters in the total study population (patients treated with corticosteroids and controls). TS: total skeleton, LS: lumbar spine, FN: femoral neck, BUA: broadband ultrasound attenuation, SOS: speed of sound, STIFF: Stiffness

	STIFF	BUA	SOS
TS	0.73*	0.61*	0.69*
LS	0.53*	0.52*	0.43*
FN	0.69*	0.58*	0.65*

Spearman's * P< 0.01.

sidered abnormal according to the following criteria: "deformed" if at least one of the ratios or the average height was between –3 and –4 SD and "fractured" if those values were less than –4 SD compared to the database. Average CV values were obtained and published previously and were between 2.2% and 4.6% [16].

2.2.3. Statistical methods

Results are reported as mean values ± 1 standard deviation. Continuous variables were compared by the Mann Whitney U-test or Student's *t*-test according to data distribution. Categorical variables were analyzed by Chi Square Test or Fisher's exact test; $P < 0.05$ was considered significant. Spearman correlation coefficients were calculated.

3. Results

Both DEXA and US measurements were significantly diminished in the corticosteroid-treated patients compared to the control group (Table 2). We have analyzed US parameters by adjusting weight (using robust regression) and BUA, SOS and Stiffness continued being significantly different between control and corticosteroid-treated patients.

Fig. 1A shows the average Z score for each determination in corticosteroids-treated patients. BUA and Stiffness were

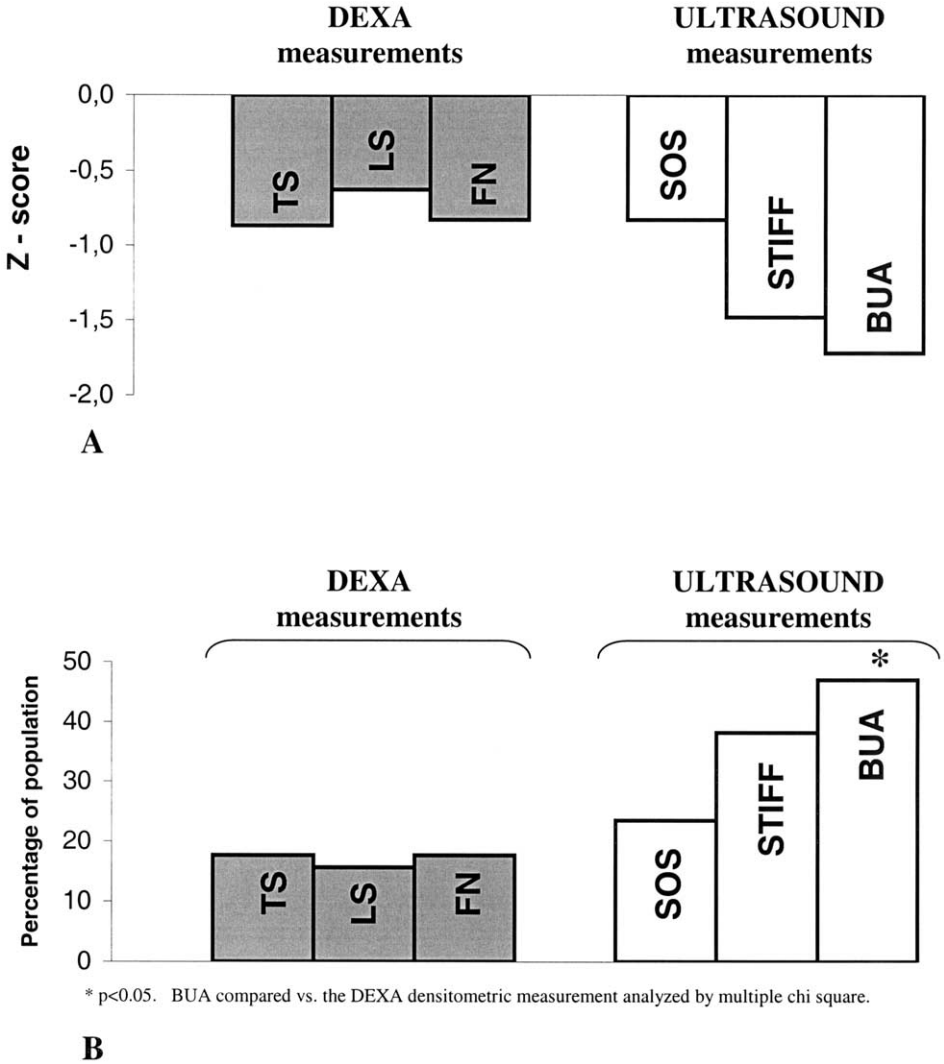

Fig. 1. (A) Average Z score of DEXA measurements: total skeleton (TS), lumbar spine (LS) and femoral neck (FN); and of ultrasound measurements: Stiffness, broadband ultrasound attenuation (BUA) and speed of sound (SOS) in patients receiving chronic corticosteroid treatment. (B) Percentage of patients receiving chronic corticosteroid treatment with DEXA and ultrasound Z scores below –2.

Table 3
Bone mineral density (X ± SD) (g/cm^2) of total skeleton (TS), lumbar spine (LS), femoral neck (FN), and ultrasound parameters: Stiffness (STIFF, %), broadband ultrasound attenuation (BUA, dB/MHZ) and speed of sound (SOS, m/s) in fractured (Fx) and non-fractured (Non-Fx) patients

	Fx	Non-Fx	P
n	11	23	
TS	0.939 ± 0.14	0.996 ± 0.06	NS
LS	0.914 ± 0.19	0.980 ± 0.11	NS
FN	0.730 ± 0.21	0.779 ± 0.13	NS
STIFF	56.18 ± 12.36	66.90 ± 15.19	NS
BUA	85.36 ± 11.46	97.48 ± 11.73	0.02
SOS	1496.82 ± 26.12	1507.10 ± 30.48	NS

found to be most affected: average Z scores between –1.4 and –1.7 vs. an average –0.9 Z score for the remaining parameters. The percentage of patients presenting a Z score below –2 was highest in Stiffness and BUA, (38% and 47%, respectively, compared to the remaining parameters): TS 18%, LS 16%, FN 18%, SOS 24% ($P < 0.05$ BUA vs. DEXA measurements, analyzed by multiple chi square).

A positive correlation was observed among DEXA measurements (r between 0.60 and 0.85, $P < 0.001$). Correlations among DEXA and US parameters in the corticosteroid group were similar to those observed in the control group. For this reason, the correlations among the parameters of both methods were calculated in the study population as a whole (Table 2). The correlations between DEXA and US parameters ranged between 0.43 and 0.73. There was no significant correlation between the US or DEXA parameters and the accumulated corticosteroid dose, the dose of the previous year, and the length of the course of the disease.

A comparison between Fx patients is shown in Table 3 The 11 Fx patients had lower DEXA and US values. However, the differences were not statistically significant except for BUA. Stiffness and BUA showed the most marked decrease (16% and 12%, respectively) compared to TS, LS, and FN (approximately 6%).

4. Discussion

Corticosteroid induced osteoporosis is associated with decreased bone mass, a marked decrease in bone formation and altered bone architecture with thinning and even disrupted trabeculae [1,2,4,17–19].

More recent studies have demonstrated corticosteroids to be associated with apoptosis of osteoblasts and osteocytes. The latter would act as sensors responsible for repairing microdamage and for bone remodeling. Since these cells are affected during the early stages of corticosteroid treatment, they may contribute to the observed structural alterations [17]. The combination of these factors leads to increased fracture risk even during the first months of corticosteroid treatment, maintaining risk above that of the control population throughout corticosteroid treatment [1,5,20,21]. The most common risk is of vertebral fractures, although risk of non-vertebral fractures is also increased [5,21,22]. Such was the case in our study, in which the fractured patients presented vertebral and peripheral osteoporotic fractures.

The characteristic alteration in bone architecture associated with corticosteroid induced osteoporosis is the destruction of horizontal and vertical trabeculae, whereas it is primarily the horizontal trabeculae that are affected by primary osteoporosis [19]. The different alterations in bone architecture observed in corticosteroid and primary osteoporosis may affect the relation between bone density and fracture risk. Moreover, fracture threshold was found to be higher in corticosteroid-treated than in post-menopausal osteoporotic patients [6,10]. In theory, there could be differences between the results obtained by DEXA and US, since DEXA only evaluates bone mass and US studies provide data on bone architecture and density [7,9].

The patients receiving moderate corticosteroid treatment (approximately 6 mg/d of prednisone) presented a significant decrease in all the parameters measured by DEXA and by US. Mean BMD of the LS, FN, and TS showed a similar decrease of approximately 1 SD in all the three areas.

Although the LS region was found to present the most marked decrease in BMD, the patients under chronic corticosteroid treatment also exhibited a diminution in the FN region which was similar to, or even greater than, that observed in the LS [11,21,23,24].

However, the greatest difference between corticosteroid-treated patients and controls was observed by BUA (mean Z score: –1.7). It would seem that this parameter best reflected bone density and architecture, whereas SOS (which analyzes bone density and elasticity) and DEXA rendered similar results.

This observation is further supported by the fact that the percentage of patients presenting values below –2 SD (below normal range) was approximately 40% according to BUA but around 20% according to DEXA and SOS determinations.

A comparison between Fx and non-Fx patients revealed that Stiffness and BUA parameters exhibited a greater decrease in Fx patients than the remaining parameters. This decrease only reached statistical significance in BUA. Therefore, BUA measurement would seem to be considerably more effective to evaluate the specific deleterious effect of corticosteroids on the skeleton and fracture risk.

A high correlation was observed among DEXA measurements (0.60–0.85), whereas correlation between DEXA and US was more variable (0.43–0.73). This observation suggests that DEXA and US measure different properties of bone. The other possibility is that BUA has a high correlation with DEXA of calcaneus, may be reflecting a marked loss of trabecular density of the calcaneus that is a bone composed approximately of 90% of trabecular bone [25].

Few studies have investigated bone alterations in patients receiving corticosteroids using both US and DEXA. Daens et al. [26] performed US (BUA and SOS) determinations of the calcaneus and measured LS BMD by DEXA in patients presenting rheumatoid diseases with and without chronic

corticosteroid therapy, and in a control group (fracture percentages were 56%, 33%, and 47%, respectively). The group of patients receiving corticosteroid treatment exhibited the greatest decrease in both US and DEXA measurements. The authors found that the relation between BUA and BMD of LS in the total population varied depending on the administration of corticosteroid treatment. They concluded that US was not only useful to evaluate bone loss associated with corticosteroid therapy, but could also provide additional information about bone structure alteration caused by corticosteroid treatment.

Wünster et al. [27] evaluated US of phalanges and DEXA of LS in patients presenting primary osteoporosis, in patients who had received corticosteroid treatment for 6–12 months, and in control subjects. The author observed that DEXA allowed discriminating between osteoporotic and healthy patients, whereas US differentiated healthy subjects from the group of patients under short-term corticosteroid treatment. He concluded that US was more accurate than DEXA to evaluate bone alterations associated with corticosteroids treatment.

Contrary to the above authors and the findings reported herein, in his study on a population of control subjects and patients with primary and corticosteroid induced osteoporosis, Blanckaert et al. [28] concluded that US and DEXA were equally useful to evaluate bone alterations occurring in these two forms of osteoporosis. A possible explanation for these different findings may be that 55% of Blanckaert's patients had received ethidronate, fluoride, or hormone replacement therapy which could have affected the primary alteration caused by corticosteroid treatment. Our patients received no treatment for osteoporosis prior to the study, and can, therefore, be thought to exhibit the specific effect of corticosteroids on the skeleton.

A group of patients with Cushing Syndrome was evaluated using DEXA and US [29]. The measurements showed diminution of LS and FN BMD as well in BUA and Stiffness. No diminution was observed in SOS. Comparison among the methods showed DEXA of the LS to be most sensitive. One possible explanation that may account for the difference observed with our results is that the mechanisms leading to bone loss in endogenous glucocorticoid excess are different from those involved in the more chronic, less severe exogenous glucocorticoid excess.

US, mainly BUA, has also proven to be substantially more sensitive for differential diagnosis of other diseases. For example, studies comparing a group of patients with anorexia nervosa to a group of controls showed a marked decrease in BUA values, which was greater than the diminution observed in the spine and femur by DEXA, whereas SOS showed no decrease [30,31]. Similarly, patients with primary hyperparathyroidism subjected to US determinations with different machines and to DEXA measurements of the hands and LS exhibited a significant diminution in all BUA values as well as DEXA values of the hands, but not in DEXA values of the LS or SOS determinations [32]. Our observations in corticosteroid-treated patients are in keeping with these reports, indicating the efficiency of BUA to discriminate bone alterations in a variety of diseases.

To conclude, BUA of the calcaneus was more effective in detecting the specific skeletal alterations and fracture risk of the group of patients receiving chronic corticosteroid treatment included in this study. US measurements, especially BUA, can, therefore, be considered the choice method to evaluate bone alterations in which bone density determinations alone may prove insufficient.

Acknowledgements

The authors wish to thank Dr. J. Hofman, Dr. G. Tate, and Dr. P. Maid for recruiting the patients. This study was supported by the Fundación Argentina de Osteología.

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