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FİZİKSEL TIP

LUMBAR AND FEMORAL BONE MINERAL DENSITY IN PATIENTS WITH ANKYLOSING SPONDYLITIS

ANKİLOZAN SPONDİLİTLİ HASTALARDA LOMBER VE FEMORAL KEMİK MİNERAL YOĞUNLUKLARI

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SUMMARY

This study was carried out to determine the bone mineral density (BMD) values of the lumbar spine and femoral neck in ankylosing spondylitis (AS) patients. Eighteen outpatients who fulfilled the modified New York criteria for AS and also 18 healthy controls were consecutively included in the study. BMD of lumbar spine and femoral neck was evaluated by dual energy x-ray absorptiometry (DEXA). Laboratory parameters included erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). The demographic variables such as age,-sex-and BMI were similar between patients and controls (p>0.05). The biochemical parameters ESR and CRP were found to be different between the patient and control groups (p<0.001 for both).

BMD values of lumbar and femoral regions in AS patients were 0.98 ± 0.2 gr /cm² and 0.87 ± 0.1 gr /cm². BMD values of lumbar and femoral area in control subjects were 1.02 ± 0.13 gr /cm² and 0.97 ± 0.12 gr /cm². Patients with AS had reduced BMD in their lumbar spine and femoral neck regions (p<0.05, p<0.01 respectively). Femoral measurements exhibited greater severity of reduced BMD than lumbar values when average BMD scores were compared. Consequently, related to the structural possible changes seen in the lumbar area, the lumbar region BMD measurements can be misleading when evaluating the extent of bone mass loss in AS patients. Therefore, alternative sites or the femoral region should be used to evaluate bone mass in AS patients.

Key words: *Ankylosing spondylitis, bone mineral density*

ÖZET

Bu çalışma ankilozan spondilitli (AS) hastalarda lomber ve femoral bölge kemik mineral yoğunluklarını (KMY) saptamak amacıyla yapıldı. Çalışmaya modifiye New York kriterlerine göre AS tanısı konmuş 18 hasta alındı. KMY ölçümleri dual energy x-ray absorptiometry (DEXA) ile değerlendirildi. Eritrosit sedimentasyon hızı (ESR) ve C-reaktif protein (CRP) takip edilen laboratuar parametreleriydi. Demografik veriler açısından (yaş, cinsiyet ve vücut kitle indexi (VKI)) hasta ve kontrol grubu arasında anlamlı fark yoktu (p>0.05). Grupların ESR ve CRP değerleri arasında istatistiksel olarak anlamlı fark saptandı (p<0.001). AS'li hastalarda lomber bölge KMY değerleri 0.98 ± 0.2 gr/cm², femoral bölge KMY değerleri 0.87 ± 0.1 gr/cm² idi. Kontrol vakalarında KMY değerleri lomber bölgede 1.02 ± 0.13 gr /cm², femoral bölgede ise 0.97 ± 0.12 gr /cm² idi. AS'li hastaların lomber ve femoral bölgelerine ait KMY değerleri kontrol grubuna göre daha düşük saptandı (sırasıyla p<0.05, p<0.01). Ortalama BMD skorları karşılaştırıldığında ise, femoral ölçümler lomber BMD değerlerine göre daha fazla azalma sergiliyordu.

Sonuç olarak, AS'li hastaların lomber bölgelerinde görülen olası yapısal değişikliklere bağlı olarak objektif KMY değerleri maskelenebilmektedir. Bu nedenle, AS'li hastalarda KMY'larının izlenmesinde femoral bölge veya başka alternatif alanlar göz önünde bulundurulmalıdır.

Anahtar sözcükler: *Ankilozan spondilit, kemik mineral yoğunluğu.*

INTRODUCTION

Ankylosing Spondylitis (AS) is a chronic and progressive inflammatory disease of the spine. It is characterized by early sacroiliac joint involvement followed by hardening of the anulus fibrosus and surrounding connective tissue along with arthritic changes in the facet joints. It is characterized by mild or moderate flares of active spondylitis alternating with periods of almost or totally inactive inflammation (1, 2). The cause of AS is not known, but all of the spondylarthropathies share a common genetic marker, called HLA-B27, in most affected individuals (3).

Osteoporosis (OP) is frequently associated with AS and is a well recognized complication of this disease (4). The physiopathogenesis of bone mass loss found in AS patients has been completely elucidated. However, various factors such as disease activity, treatment, hormonal disorders, decreased mobi-

lity and physical activity or mineralization defects due to subclinical gut involvement may contribute to the development of decreased bone mineral density (BMD) in AS patients (5, 6). Recent reports suggested increased bone turnover (7) or decreased bone formation (8) in AS. BMD decreased predominantly in patients with active AS. It has been suggested that local and systemic inflammatory cytokine release might be implicated in bone loss. Particularly, cytokines such as TNF-alpha and IL-6 may play an important part in the pathogenesis of osteoporosis in early AS (9). OP in patients with AS is largely confined to the axial skeleton, in contrast to the pattern of OP seen in rheumatoid arthritis. The most precise method currently available to quantify bone mineral content is dual energy x-ray absorptiometry (DEXA). Several authors have investigated the bone loss related to AS using bone densitometry techniques and have shown varying prevalence and degree of osteopenia or osteoporosis (10-12).

This study was carried out to determine the BMD values of the lumbar spine and femoral neck in AS patients.

MATERIAL AND METHOD

Eighteen outpatients who fulfilled modified New York criteria for AS (13) were consecutively included in the study and all of them showed positive HLA B27. Baseline clinical assessment included demographic data: age, sex, body mass index (BMI: weight / height^2; kg /m^2) and disease duration. In the patient group (n=18), there were 15 men and 3 women (mean age: 33.3 ± 8.7, range: 20–45). The mean disease duration was 12.5 ± 7.6 years (range 2–25). The control group was formed by twenty healthy volunteers without any evidence of diseases, matched in age and sex with AS patients (15 men and 3 women, mean age: 35.2 ± 7.7, range: 22–50). Thirteen patients were taking a combination of NSAIDs and sulfasalazine and five patients were only taking NSAIDs. Corticosteroids were not administered. Exclusion criteria included liver and kidney diseases, renal stones, diabetes, alcoholism, thyroid and parathyroid diseases, previous and current anti-osteoporotic treatments, hematological, lymphoproliferative and other malignant diseases. The exclusion criteria for the control group were the same as for the AS group.

BMD of lumbar spine and femoral neck was evaluated in 18 consecutive patients by DEXA (Hologic QDR 2000). The axial BMD was measured in the lumbar spine (L1-L4) and the appendicular BMD was measured in the total hip. The results were expressed as gr/cm^2. Eighteen patients were compared with eighteen sex- and age-matched controls. Laboratory parameters included ESR and CRP in peripheral blood as inflammation markers. ESR was determined according to the Westergren method and CRP by a nephelometric method (Beckman Array Protein System, USA).

Data were processed using the SPSS package programme. Laboratory results were given as mean ± standard deviation (SD). Differences between groups were analyzed using the Mann-Whitney U test. P values less than 0. 05 were considered to be statistically significant.

RESULTS

The demographic and clinical characteristics of patient and control groups are listed in Table I. The demographic variables such as age, sex and BMI were similar between patients and controls ($p>0.05$). The acute phase reactants (ESR and CRP) were found to be statistically different between the patient and control groups ($p<0.001$ for both) (Table I).

Table I. The clinical and laboratory features of the patients with AS and healthy controls

	Patient group	Control group	p-value
Sex (male/female)	15/3	15/3	ns
Age (years)	33.3 ± 8.7	35.2 ± 7.7	ns
Disease duration (years)	12.5 ± 7.6	—	
BMI (kg/m^2)	26.5 ± 3.85	27.0 ± 3.51	ns
ESR (mm/h)	29.25 ± 15.84	12.6 ± 6.51	<0.001
CRP (mg/L)	1.06 ± 0.79	0.44 ± 0.30	<0.001

ns: not significant

Table II. Lumbar spine and femoral neck BMD values of the patients with AS and control subjects

	Patient group	Control group	p-value
Lumbar BMD (gr/cm^2)	0.98 ± 0.2	1.02 ± 0.13	p<0.05
Femoral BMD (gr/cm^2)	0.87 ± 0.1	0.97 ± 0.12	P<0.01

Eighteen control subjects and patients with AS had lumbar and femoral BMD studies using DEXA. BMD measurements of the lumbar spine and femoral neck are shown in Table II. Patients with AS have reduced BMD in their lumbar spine and femoral neck compared BMD values of control subjects ($p<0.05$, $p<0.01$ respectively). Femoral BMD measurements indicated greater distinction than in the lumbar area when average BMD scores were compared.

DISCUSSION

Our data suggest that bone loss can occur in AS patients. The etiology of bone mass loss in AS patients remains controversial. It has been suggested that local or systemic inflammatory cytokines release might be involved in bone loss (14). There have been various studies related to osteoporosis reported in literature.

This study demonstrated a reduction of BMD in the lumbar spine and femoral neck in patients with AS. Will et al (15) and Donnelly et al (16) found significant decreases in the mean BMD of the lumbar spine and the femoral neck in AS patients. Femoral measurements exhibited greater severity of reduced BMD than lumbar values when average BMD scores were compared. The results are consistent with previous findings of Capaci et al, Singh et al and Will et al (17-19). It is known that trabecular bone loss is more prominent than cortical bone loss in osteoporosis. Therefore, it is expected that BMD might be lower in the lumbar region which is rich of trabecular bone when compared to the femoral area. But, we found lower BMD values for the femoral region. This discrepancy might be due to new bone formation in lumbar spine area such as syndesmophytes, interapophyseal joint and interpedicular ankylosis rather than differences in bone remodeling between these two sites (20). Pathological changes in AS patients occur predominantly in the spine. Enthesopathies and new bone formation such as syndesmophytes and ligament calcifications in the lumbar region can increase the spinal bone mineral content (21, 22). BMD used as a measure of bone mass loss of lumbar spine in AS patients is unreliable probably as a consequence of syndesmophyte formation or apophyseal joint fusion. For this reason; the lumbar measurements are insensitive and inappropriate. The femoral neck measurements provide a more reliable indication of the presence and severity of reduced BMD in patients with AS (16, 23).

Consequently, the antero-posterior lumbar region DEXA measurements can be misleading when evaluating the extent of bone mass loss in AS patients. Therefore, alternative sites or the total hip should be used to evaluate bone mass in AS patients.

The disparity between lumbar spine and femoral neck BMD values in patients with AS needs further evaluation.

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