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Effect of bone mineral density on lumbar discs in young adults

A case–control study

Nese Keser, MD[a,*], Arzu Atici, MD[b], Erhan Celikoglu, MD[a], Pinar Akpinar, MD[b], Ali F. Ramazanoglu, MD[a], İlknur Aktas, MD[b]

Abstract

Bone mineral density (BMD) might be a risk factor for lumbar disc herniation (LDH) in young adults, but there is not enough data concerning this effect. Several studies have been performed on elderly and osteoporotic patients. Thus, we aimed to investigate the effect of BMD on the etiopathogenesis of LDH in young adults.

One hundred patients (mean age: 38.45 ± 8.92 years; 50 men and 50 women) were enrolled this case–control study and classified into 2 groups, as follows. The case group (G-I) included 50 patients with symptomatic LDH who were hospitalized in the Physical Medicine and Rehabilitation Clinic, and the control group (G-II) included 50 patients with lower back pain but no finding of LDH detected using magnetic resonance imaging. Patients in the G-II were recruited among those admitted to the outpatient clinic at the time of the study, and whose age and sex were matched to those of the study group. Women in menopause were excluded from the study. BMD analysis by dual energy x-ray absorptiometry was performed in both groups.

The mean values of the femur neck and lumbar spine BMD were 1.02 ± 0.13 and 1.19 ± 0.14 g/cm^2, respectively. There was no statistically significant relationship between BMD and LDH in this population.

This result may mean that in a normal range, BMD does not exert a compressive load on the lumbar discs in young adults and therefore may not lead to LDH by this mechanism and the load.

Abbreviations: ALP = alkaline phosphatase, BMD = bone mineral density, BMI = body mass index, Ca = calcium, DD = disc degeneration, DDD = degenerative disc disease, DH = disc herniation, DM = diabetes mellitus, DXA = dual energy x-ray absorptiometry, G-I = case group, G-II = control group, IVD = intervertebral disc, IVDD = intervertebral disc degeneration, LDH = lumbar disc herniation, MRI = magnetic resonance imaging, OP = osteoporosis, P = phosphorus, PTH = parathormone, WC = waist circumference, WHO = World Health organization.

Keywords: bone mineral density, dual-energy x-ray absorptiometry, intervertebral disc degeneration, lumbago, osteoporosis

1. Introduction

Lumbar disc herniation (LDH) is one of the most important causes of lower back and leg pain, with a reported incidence of 1% to 2%.[1] Disc degeneration (DD), a cause of LDH, is a continuous lifelong process that starts in the third decade of life.[2] Genetic factors, smoking, aging, autoimmune and metabolic disorders, such as diabetes, as well as mechanical disorders that disrupt disc nutrition, such as compressive stress, are significant etiological factors.[3–7]

The correlation between bone mineral density (BMD) and degenerative disc disease (DDD) is controversial. Several studies have investigated the effect of BMD on lumbar DDD; however, many of these studies were conducted on only one sex or in osteoporotic patients more than 60 years old. Thus, no consensus has been reached.[8–18]

To our knowledge, no previous study in the literature has evaluated this issue in a specific group of young adults. Therefore, we aimed to investigate the effect of BMD on the etiopathogenesis of LDH among premenopausal women and men younger than 60 years.

2. Materials and methods

2.1. Selection of the study population

One hundred patients (50 men, 50 women) were enrolled in this case–control study and classified into the following 2 groups. Since we analyzed patients retrospectively, ethical approval was not necessary for our study. The case group (G-I) included 50 patients (mean age 40.28 ± 9.08 years) with symptomatic LDH hospitalized in the Physical Medicine and Rehabilitation Clinic, between February 2015 and March 2016. Whereas, the control group (G-II) included 50 patients (mean age 36.62 ± 8.46 years)

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[a] Neurosurgery Department, [b] Physical Medicine and Rehabilitation Department, TR University of Health Sciences, Istanbul Fatih Sultan Mehmet Education and Research Hospital, Istanbul, Turkey.

[*] Correspondence: Nese Keser, Neurosurgery Department, TR University of Health Sciences, Istanbul Fatih Sultan Mehmet Education and Research Hospital, E-5 Karayolu Uzeri Icerenkoy, 34752 Atasehir, Istanbul, Turkey (e-mail: nskeser@gmail.com).

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with lower back pain but no finding of LDH detected on magnetic resonance imaging (MRI) scans. Patients in the G-II group had been admitted to the outpatient clinic at the time of the study, and their age and sex were matched to those in the G-I group. Previously recorded MRI scans of patients were reevaluated by the same doctor, and those who met the eligibility criteria for LDH[19] were included in the analysis. Exclusion criteria were women in menopause; patients with conditions that would affect BMD, such as malignancy, inflammatory joint diseases, hyperthyroidism, hyperparathyroidism, chronic liver diseases, type 1 diabetes mellitus (DM), osteogenesis imperfecta, hypogonadism, chronic malnutrition, malabsorption spinal stenosis, spondylolisthesis, or compression fractures; and patients who previously received treatment for osteoporosis (OP) or those currently being treated with cortisone, antiepileptic drugs, or thyroid replacement medications.

A careful history was taken to determine the patient's current and past smoking status. Smoking was categorized as follows: 0, a lifelong non-smoker or quit ≥5 years prior; and 1, current smoker. Data on occupation and daily activities were collected, but objective conclusions could not be reached because patients made personal comments concerning these factors.

Waist circumference (WC) and body mass index (BMI) were measured. WC was measured with the patient in a standing position and the measuring tape at the level of the umbilicus, just above the hip bones. BMI was calculated by dividing the patient's weight by height squared and reported as kg/m^2. BMI was classified as follows: 25 to 30 kg/m^2, overweight; 30 to 40 kg/m^2, obese; and >40 kg/m^2, morbidly obese, according to the World Health Organization (WHO) definitions. In both groups, biochemical parameters, including the levels of serum 25(OH) vitamin D_3, parathormone (PTH), calcium (Ca), phosphorus (P), and alkaline phosphatase (ALP), were measured. The serum 25(OH) vitamin D_3 level was assessed using the chemiluminescent microparticle immunoassay method with Architect System Kits on the i2000SR immunological analyzer (Abbott Laboratories, Abbott Park, IL). According to the Endocrine Society Clinical Practice Guidelines, a 25(OH) vitamin D_3 level less than 20 ng/mL is defined as vitamin D deficiency and that between 21 and 29 ng/mL is defined as vitamin D insufficiency.[20]

BMD analysis by dual energy x-ray absorptiometry (DXA) was performed in both groups. BMD measurements of the lumbar spine (L1–4 levels) and femoral neck were determined using DXA (Lunar HVPS7681; General Electric, Madison, WI), and MRI scans were obtained using a 1.5 Tesla MRI machine (450W; General Electric). We defined OP according to WHO guidelines[21]: normal BMD is within 1 standard deviation (+1 or −1) of the young adult mean.

2.2. Statistical methods

The IBM SPSS Statistics 22 program (IBM SPSS, Istanbul, Turkey) was used to perform statistical analysis. The Shapiro–Wilks test was used to determine the conformity of the parameters to a normal distribution. All parameters were found to have a normal distribution. Student *t* test was used to compare parameters between the 2 groups. Continuity (Yates) correction was used to assess qualitative data. A *P*-value < .05 was considered statistically significant.

3. Results

The G-I group included 23 premenopausal women and 27 men with a history of lower back pain or unilateral radicular leg pain for at least 2 months, and single-level disc herniation (DH) detected on MRI scans. The G-II group included 50 patients in the same age group with lower back pain but no finding of DH detected on MRI scans.

There was no statistically significant difference between the groups regarding general characteristics and laboratory parameters, including sex distribution, age, height, weight, BMI, and levels of 25(OH) vitamin D_3, PTH, Ca, P, and ALP ($P > .05$). Demographic data are shown in Table 1. When the BMD measurements (*T* scores and *Z* scores) were evaluated, there were no significant differences in the L1–4 and femoral neck scores between the 2 groups ($P > .05$) (Table 2).

4. Discussion

Although it is still controversial, the general belief is that DD is the main cause of DHs,[22,23] and avoidable factors such as

Table 1

Evaluating the operating parameters according to the study (G-I) and control (G-II) groups in all cases.

	G-I	G-II		
	Mean ± SD	Mean ± SD	95% Confidence interval of the difference	*P*
Age	40.28 ± 9.08	36.62 ± 8.46	0.18 to 7.14	†.040*
Gender, n (%)				
Male	27 (54%)	23 (46%)	—	‡.549
Female	23 (46%)	27 (54%)		
BMI, kg/m^2	27.44 ± 4.34	26.36 ± 4.09	−0.6 to 2.75	†.206
WC, cm	96.02 ± 9.88	93.14 ± 7.33	−0.57 to 6.33	†.101
Smoking, n (%)	17 (34%)	26 (52%)	—	‡.106
25 (OH) vitamin D_3	16.06 ± 7.26	15.33 ± 6.43	−1.99 to 3.45	†.597
PTH	63.88 ± 30.52	72.88 ± 36.31	−22.31 to 4.32	†.183
Ca	9.64 ± 0.33	9.50 ± 0.47	−0.03 to 0.3	†.100
P	3.57 ± 0.49	3.64 ± 0.61	−0.29 to 0.15	†.507
ALP	69.42 ± 14.8	67.16 ± 20.61	−4.86 to 9.38	†.530

ALP = alkaline phosphatase, BMI = body mass index, Ca = calcium; P = phosphorus, PTH = parathormone, SD = standard deviation, WC = waist circumference.

* Significant $P < .05$.

† Student *t* test.

‡ Continuity (Yates) correction.

Table 2

Evaluating the BMD parameters according to the study (G-I) and control (G-II) groups in all cases.

	G-I	G-II		
	Mean ± SD	Mean ± SD	95% Confidence interval of the difference	*P*
L1–4 BMD	1.19 ± 0.14	1.17 ± 0.14	−0.03 to 0.08	.370
L1–4 *T* score	0.53 ± 1.06	0.22 ± 1.13	−0.13 to 0.74	.167
L1–4 *Z* score	0.4 ± 1.04	0.22 ± 1.14	−0.26 to 0.61	.417
Femur neck BMD	1.02 ± 0.13	0.98 ± 0.15	−0.01 to 0.1	.130
Femur neck *T* score	0.24 ± 1.08	−0.03 ± 1.13	−0.17 to 0.7	.231
Femur neck *Z* score	0.44 ± 0.89	0.17 ± 1.11	−0.13 to 0.67	.178

BMD = bone mineral density, SD = standard deviation.
Student *t* test.

trauma and smoking are known factors that facilitate the formation of DHs in combination with DD.[3,24,25]

Many studies have demonstrated that OP is associated with a reduced risk of DD and herniation.[8,10–13,15,18]The relationship between delayed DH and OP can be explained by 2 pathophysiological pathways: the mechanical pathway and vascular pathway.[8] In the mechanical pathway, vertebral stiffness decreases because of low vertebral BMD, and the relative stress on the disc is reduced because of the shock-absorbing characteristics of the intervertebral disc (IVD).[8,26]

In contrast, in nonosteoporotic patients, bones with sufficient mineral content and intact cortical and trabecular structures place mechanical stress on the IVDs. Increased endplate resistance will also affect endplate vascularization and facilitate DH by disrupting the diffusion of disc nutrients. There is insufficient data concerning the effect of increased BMD or the absence of OP on DH.[22] Therefore, in the present study, we investigated the effect of BMD on LDH in young adults.

With the DXA method, the presence of degenerative changes, such as aortic calcifications, vertebral osteophytes, facet joint degenerations, and sclerosis of the IVD, may lead to falsely increased lumbar BMD.[9] In the future, other methods such as diffusion-weighted MRI may detect BMD,[27,28] but in this study, we excluded patients aged more than 60 years, a population more likely to develop these confounding factors. Additionally, since a decrease in BMD values is observed in postmenopausal women, we included only premenopausal women.

Many studies have investigated the relationship between DD and BMD, and they demonstrated an association between lumbar DD and a high BMD.[8,10–12] However, some studies have also reported that a low BMD is associated with increased intervertebral disc degeneration (IVDD)[12] and that there is no relationship between IVDD and vertebral OP.[13] One explanation for the differences in these reported findings is that most studies were conducted in patients who were osteoporotic and hence of advanced age.[9,14–17] When we compared our enrolled patients with and without LDH, we did not detect any significant differences in any measure of bone health, not just BMD.

A previous study demonstrated that women have more DD than men and that less DD is observed in patients with a low lumbar vertebral BMD.[18] However, this study included patients in the advanced age group. In another study conducted only in female patients with IVDD, increased BMD was seen in the lumbar vertebra, radius, and calcaneus, and the DD score was found to be significantly higher in the postmenopausal group than in the premenopausal group.[11]

Even though the DD score increased in accordance with age in all premenopausal and postmenopausal groups, this score correlated with BMI in the premenopausal group.

On the basis of the observed relationship between a high BMD and degenerated discs in premenopausal patients, as well as the relationship between BMD of the radius, a bone that does not bear weight, and IVDD, the authors suggested that a high systemic BMD establishes ground for DH.[11]

The correlation between systemic BMD and DH suggests that different mechanisms may also play a role by alternate pathophysiological pathways, as mentioned in the introduction. In light of previous studies, it can be said that increased BMD may facilitate LDH formation. However, our result showed no relationship between LDH and BMD of the femoral neck, which we consider nonvertebral.

The strength of our study includes the standardized analysis of fasting blood samples and MRI scans of LDH. Additionally, we controlled for known effects of obesity and DM by excluding individuals with these diseases, and we used a homogeneous study sample, with equal distribution of men aged less than 60 years old and women who were not in menopause, thereby also controlling for effects of aging and menopause on bone health. However, the small sample size and retrospective analysis are limitations of our study.

The accumulation of knowledge through case–control studies and additional prospective studies involving more patients are essential to physicians' understanding of the relationship between bone health and LDH. Moreover, these studies will also guide physicians in detecting the BMD that leads to adjacent segment disease, which is very important for lumbar fusion operations.

In conclusion, we did not detect any statistically significant differences in any measure of bone health, not just BMD. This result may mean that healthy bone does not exert a compressive load on the lumbar discs in young adults; therefore, it may not lead to LDH by this mechanism.

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