Increased Bone Mineral Content But Not Bone Mineral Density in the Hip in Surgically Treated Knee and Hip Osteoarthritis

LORENZO SANDINI, JARI P.A. AROKOSKI, JUKKA S. JURVELIN, and HEIKKI KRÖGER

ABSTRACT. ***Objective.*** The inverse relationship between the occurrence of osteoarthritis (OA) and osteoporosis is controversial. Some investigators have found higher bone mineral density (BMD) in the hips, lumbar spine, and other skeletal sites of patients with OA; others have not. We investigated the relationship between BMD and OA.

Methods. We compared the BMD, bone mineral content (BMC), and projected area of the femoral neck (FN) and trochanter (TR) of 99 women with a validated diagnosis of primary OA from the Kuopio Osteoporosis Risk Factor and Prevention Study (OSTPRE) cohort, with 2012 controls. The measurements were made twice in women aged 47–59 years in 1989-91, and then repeated in 1994-98.

Results. After correction for age, body mass index (BMI), menopausal status, and hormone replacement therapy use before inclusion, we found no significant difference in femoral BMD of the OA patients compared with controls at baseline and at 5-year followup (FN +2.7%, +4.6%, respectively; nonsignificant). However, the BMC was significantly higher in all regions of interest in OA patients at baseline [FN +8.3% ($p = 0.004$); TR +13.3% ($p = 0.017$)]. The projected area of FN was also significantly higher at baseline and followup in OA patients (FN +3.7%, +3.9%, respectively; $p < 0.001$). The projected area of the bones increased in all subjects over the followup period. The BMD decrease rate was higher in OA patients for all regions of interest during followup.

Conclusion. Hip BMD of women treated surgically for hip or knee OA was not different from that of healthy controls when measured twice with a 5-year interval. However, at 5-year followup, OA can be accompanied by an increase in bone size or changes in shape, and faster loss of BMD. (J Rheumatol 2005;32:1951–7)

Key Indexing Terms:
BONE MINERAL DENSITY BONE DENSITOMETRY HIP KNEE
DUAL ENERGY X-RAY ABSORPTIOMETRY OSTEOARTHRITIS

Bone changes are potentially important in the pathogenesis of osteoarthritis (OA). One of the hypothesized consequences of cartilage degeneration is the thickening and stiffening of the subchondral bone. These changes in subchondral bone can appear in response to repetitive impact loading of the joint, leading to microfractures and local repair[1]. Less dense osteoporotic bone absorbs load more efficiently, reducing stress on the overlying cartilage[2]. Whether the pathogenesis of OA is related to primary bone or cartilage changes is still an unresolved question[3,4]. However, OA is a heterogeneous syndrome and the various mechanisms underlying its development are present in the different forms of the disease.

Evidence from cross-sectional studies suggests that patients with hip[5-9] or spine[10-12] OA show a higher local bone mineral density (BMD) than healthy, age and sex matched controls. Arokoski, *et al*[13] combined dual energy x-ray absorptiometry (DEXA) and magnetic resonance imaging (MRI) to estimate volumetric BMD of the femoral neck in men, and found that the increased bone mineral content (BMC) of the femoral neck was associated with an increase in the size of the bone, but that neither areal BMD nor volumetric BMD differed from the age and weight matched healthy controls. Other investigators also found no difference in the BMD between OA subjects and healthy controls after correction for body weight[12,14].

From the Department of Surgery and Applied Physics, University of Kuopio; Departments of Physical and Rehabilitation Medicine and Clinical Physiology and Nuclear Medicine, Kuopio University Hospital; Bone and Cartilage Research Unit, Mediteknia, University of Kuopio, Kuopio; and Department of Internal Medicine, Central Hospital of Kymenlaakso, Kotka, Finland.

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L. Sandini, MD, Department of Internal Medicine, Central Hospital of Kymenlaakso, and Department of Surgery, University of Kuopio; J.P.A. Arokoski, MD, PhD, Department of Surgery, University of Kuopio, and Department of Physical and Rehabilitation Medicine, Kuopio University Hospital; J.S. Jurvelin, PhD, Professor, Department of Clinical Physiology and Nuclear Medicine, Kuopio University Hospital, and Department of Applied Physics, University of Kuopio; H. Kröger, MD, Professor, Department of Surgery, University of Kuopio, Bone and Cartilage Research Unit, Mediteknia, University of Kuopio.

Address reprint requests to Dr. L. Sandini, Department of Internal Medicine, Central Hospital of Kymenlaakso, Kotkantie 41, 48210 Kotko, Finland. E-mail: lorenzo.sandini@kymshp.fi.

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Various studies have compared BMD at different skeletal sites and peripheral radiographic OA findings[10,12,15-20]. Again, results are controversial, and while some authors report higher BMD of the metacarpals, forearm, and total body in patients with hip, knee, or spine OA[15,17,21,22], some others found no association between the OA findings and the BMD values. It has also been suggested that OA is associated with an increase in trabecular but not cortical BMD[16]. Overall, in the majority of previous studies it seems that BMD is increased in patients with OA at the sites measured away from the OA-affected joint.

There is strong evidence of a protective effect of OA against hip fracture[23]. However, in some instances the relative risk of fractures has been found to be higher in OA patients, and this was thought to be the result of patients' decreased mobility and increased number of falls[24].

We tested the hypothesis that the femoral neck BMD, BMC, and projected area in DEXA measurements of menopausal women with a diagnosis of primary OA are not different from those of healthy controls. We used a population database from the Kuopio Osteoporosis Risk Factor and Prevention Study (OSTPRE), which contains 2950 pairs of DEXA measurements of the hip from women aged 48–59 years at baseline, and which were repeated 5 years later[25].

MATERIALS AND METHODS

We analyzed differences in DEXA measures of the hip among participants of the OSTPRE study, according to the presence of a diagnosis of primary OA. DEXA measurements were carried out at baseline in 1989-91 and at 5-year followup in 1994-98. The diagnosis of primary OA was assessed from the medical records of women with retrospective self-report of hip/knee surgery for OA in the 10-year followup questionnaire in 1999. The year of first diagnosis of primary OA in the medical records was recorded when available. We cross-sectionally compared DEXA measures of patients with OA and controls at baseline and at 5-year followup, and we also compared the absolute changes in DEXA findings between the 2 measurements.

Questionnaires and diagnosis of OA. In 1989, a postal enquiry was sent to all 14,220 women aged 47–56 years living in the district of Kuopio, eastern Finland[26]. The OSTPRE study aimed to detect medical and lifestyle factors associated with the occurrence of osteoporotic fractures. A 5-year followup questionnaire was sent in 1994, and a 10-year followup questionnaire was sent in 1999 to all survivors (n = 12,562) of the initial cohort, even if the 5-year questionnaire had not been returned. The response rate was high (n = 11,538, 91.8%).

All questionnaires contained questions about lifestyle, medications, diseases, falls, and gynecologic history including past and present hormone replacement therapy (HRT) use. Body mass index (BMI, kg/m^2) was determined at baseline from the participants' self-reported height and measured weight. At the 5-year followup, height and weight were measured in standard conditions. Self-reported HRT use 6 months or more before inclusion was regarded as "previous use." For the period between measurements as well as for the analysis of the 5-year DEXA measurements, the women were categorized as "never-users" or "ever-users."

In 1999, a 10-year followup questionnaire was sent to all surviving participants (n = 12,562) of the OSTPRE study, asking about medical history, fractures, and current diagnoses. Participants were asked whether they had experienced a hip/knee replacement or lower limb axis correction surgery (osteotomy), performed as a treatment for OA. From the 193 women who stated having had such surgery, data were immediately available in the medical records for 144 women who had been operated on in the Kuopio University Hospital. To the other 49, we sent a complementary questionnaire asking to confirm the original answer, and to indicate where the operation had been performed. Forty-four questionnaires were returned, allowing retrieval and verification of medical records for 37 more cases. From the 176 verified records, 132 women (75%) had confirmed primary OA (as a radiographic finding, arthroscopy finding, or per-operative finding during joint surgery), 41 (23.3%) had secondary OA (trauma, femoral head necrosis, inflammatory arthritis, congenital or acquired deformation), 12 cases could not be determined based on the available data and were removed from the analysis, and 8 had no OA (no mention in records, or corrected answer in confirmation questionnaire) and were moved to the control group. Only the cases of primary OA were retained for analysis; all other cases including anatomical deformations were excluded.

The diagnosis of OA was based on information in medical records. When available, the first mention of chronic localized joint pain/stiffness, radiographic findings of OA, or arthroscopy was used as a starting date for the disorder. When no precise mention of the beginning of symptoms was found, the year of the first operation for OA was retained. The affected side was recorded, as well as bilateral involvement, for hips and/or knees. Of all the identified patients with primary OA, 21% were diagnosed at the hip, 71% at the knee, and the rest had involvement of both joints.

In the 26 cases where an arthroscopy was the primary surgical intervention, 14 total joint replacements (TJR) or partial joint replacements (PJR) were subsequently done, and 11 osteotomies. In 25 cases, osteotomies were done first, with 10 subsequent TJR or PJR. Finally, TJR or PJR was the first operation in 60 cases. Of 115 patients with surgery once, 63 were reoperated on a second time, 25 had a third operation, and 5 had a fourth operation. We think all our patients with primary OA finally had severe OA, leading to osteotomies or joint replacements in a matter of years.

The homogeneity of the group can be established based on the number of surgical interventions, even if the OA diagnosis was made on the basis of a knee radiograph and subsequent knee TJR, with no further information on the measured hip. The radiological diagnostic evidence of primary OA was not available for all patients undergoing DEXA measurements, and that is why we consider all patients with "lower limb primary OA" as one group. The lack of systematic radiological assessment of presence of OA in our subjects before DEXA measurement is due to the retrospective population-based study design. We wanted to investigate the controversial issue of OA and BMD at a population level. This approach does not allow detailed study of individual subjects as in a more experimental study design.

We merged data from the baseline and 5-year followup DEXA measurements and from the 10-year questionnaires. We obtained 99 repeated BMD measurements in patients with primary OA, and 2012 repeated measurements in controls without OA, after exclusion of women with self-reported OA in the 5-year followup questionnaire (1994) from the control group. BMD was measured on the left hip in all OA patients and controls. From all measurements, 22 women had hip implants, but they all belonged to the secondary OA group, and thus did not interfere with our analysis.

Densitometry. Out of the 13,100 questionnaires that were completed and returned at baseline, 11,055 women (92.1%) were willing to undergo femoral neck and lumbar densitometry. A random sample of 3686 women was invited for a clinical evaluation and BMD measurements, and between 1989 and 1991, 3222 DEXA measurements were made. Of those, 2950 measurements were eligible for analysis, after elimination of measurements not satisfying quality control criteria (deformation, artefacts, positioning error). These women were invited about 5 years later in 1994-97 for a followup visit, and 2942 femoral neck DEXA measurements were repeated. We again excluded the hip measurements failing the quality controls, biased by disease or artefacts. Two regions of interest were analyzed: the femoral neck (FN) and trochanteric region (TR).

We used the DEXA reference values for the femoral neck determined in Finnish women aged 20–40 years using the Lunar DPX densitometer[27]. A femoral neck BMD < 0.68 g/cm^2 (–2.5 SD) defines osteoporosis, while BMD ≥ 0.86 g/cm^2 (–1.0 SD) is in the normal range. Women with BMD values between these limits are considered osteopenic.

The control group consisted of women without a validated diagnosis of primary OA or a suspicion of OA based on self-reported data. We excluded women who mentioned a diagnosis of OA in the 5-year questionnaire in 1994, even if this diagnosis was not validated. A simple self-report diagnosis of OA is probably not enough to ensure diagnosis, but by doing this we excluded the women who most probably had chronic joint pain that could be due to OA. Among these women, 3 had hip BMD measurements that failed DEXA quality controls because of local sclerosis of bone secondary to OA, with T scores of +0.28 to 1.78 SD on the femoral neck. No woman in the OA group failed the DEXA quality controls because of sclerosis or deformation.

Measurements were performed at the Kuopio University Hospital by trained personnel. To avoid possible errors caused by malrotation of the hip, all measurements were made using a stabilization apparatus for the foot, which keeps the foot in the same position between subjects. The short term reproducibility (coefficient of variation, CV %) of the method has been shown to be 1.5% and 2.2% for FN and TR BMD measurements, respectively[27,28]. Longterm reproducibility (CV %) of the DEXA instrument for BMD during the study period as determined by a regular phantom measurement (n = 60) was 0.4%[29].

Menopausal status and hormone replacement therapy. We excluded from the study all women with uncertain menopausal status because they had started HRT before natural menopause occurred. These represented about 10% of the cases in both groups. The women were categorized as postmenopausal if ≥ 6 months had passed since the last self-reported natural menstrual bleeding. We then categorized women according to their HRT use before the baseline measurement (ever/never) and before the 5-year followup measurement (ever/never). For the analysis of variation of DEXA values between measurements, we classified the women according to their HRT use during this period as ever/never users.

Time onset of OA. We divided patients with primary OA (with no missing data for other covariates) into 3 categories: (1) diagnosis > 5 years before baseline DEXA measurement (n = 16); (2) diagnosis < 5 years before baseline DEXA (n = 13); and (3) diagnosis after baseline DEXA (n = 34) and data that were missing for the others (n = 39). For each subgroup, we checked the BMD, BMC, and projected surface differences at baseline and at 5 years, taking 2215 controls at baseline and 1985 at 5 years.

Statistical methods. The Mann-Whitney nonparametric U test was used to compare age and BMI between the OA and control groups, and the duration of the interval between the 2 measurements. The distribution of age and BMI was close to, but not normal, in the OA group. After verification of the normal distribution, BMD, BMC, and projection area were compared between the OA and the control group using a general linear model (covariance analysis), with adjustments for age, BMI, menopausal status, and HRT use. The absolute changes in DEXA parameters (BMD, BMC, and projected area) were compared between the OA and control groups with a general linear model (ANCOVA), with adjustment for baseline DEXA measures, age, BMI, menopausal status at the first measurement, HRT use between the measurements (in years), and followup duration. The analyses were performed using the Statistical Package for Social Sciences, version 10 (SPSS v.10.0.7; SPSS Inc., Chicago, IL, USA).

RESULTS

The diagnosis of primary OA was made for 34% of women before 1989 and 66% between 1990 and 1999. The OA group was slightly but significantly older than the control group, and the BMI was higher in the OA group (Table 1). There was a slight difference in the interval between the DEXA measurements (median duration in controls 5.8 yrs, range 3.5–8.0 yrs; in OA patients 5.9 yrs, range 4.8–7.9 yrs). While DEXA was performed on the left hip for all subjects, the diagnosis of OA was made on the right hip only for 31.3% of cases; all others were of the left hip or both sides. There were no differences in DEXA parameters of the left hip when the results were grouped by affected side (left, right, or bilateral; data not shown). Regardless of the site (knee, hip, or both), bilateral involvement was observed at the time of surgery in 51 (51.5%) patients. The right side was involved in 31 patients, the left side in 17 patients. For the involved site, 71 knees, 19 hips, and 9 hip-knee cases were included. Analysis of the distribution of osteoporotic, osteopenic, and normal women based on the reference values for the FN in Finnish women aged 20–40 years showed a difference at baseline that disappeared at the followup (Table 2).

Table 1. Baseline characteristics of subjects.

	OA, n = 99 median (range)	Controls, n = 2012 Median (range)	p*
Age, yrs	54.5 (48.3–59.3)	53.1 (48.0–59.6)	< 0.001
BMI	28.2 (20.4–46.9)	25.3 (16.3–52.9)	< 0.001
Followup, yrs	5.9 (4.8–7.9)	5.8 (3.5–8.0)	0.011

* Mann-Whitney U test. BMI: body mass index.

Height, weight, and BMI all changed significantly ($p > 0.001$) in the OA and control groups from the baseline to 5-year measurements. Height loss was more important in the OA group (–1.8 ± 1.9 cm) than in the controls (–1.1 ± 1.8 cm; $p < 0.001$). Weight increase was similar between groups (OA, +4.6 ± 7.5 kg; controls, +4.5 ± 5.8 kg; $p = 0.497$). BMI changes were also similar between groups (OA, +2.5 ± 2.9 kg/m^2; controls, +2.0 ± 2.4 kg/m^2; $p = 0.086$).

Analysis of DEXA measures at baseline and at 5 years. After correction for age, BMI at the time of DEXA measurement, menopausal status, and HRT use before inclusion, we found no significant difference in the femoral BMD of the OA patients compared with the controls at baseline and 5-year followup (FN, +2.7%, +4.6%; nonsignificant). However, the BMC was significantly higher in all regions of interest in OA patients at baseline [FN, +8.3% ($p = 0.004$); TR, +13.3% ($p = 0.017$)]. The projected area of FN was also significantly greater at baseline and followup in OA patients

Table 2. Distribution of osteoporotic, osteopenic, and normal women according to their femoral neck BMD measurements, in patients with OA and controls.

	Baseline DEXA Measurement 1989–91		5 yr Followup DEXA Measurement 1994–97	
	Controls, n = 2012 (%)	OA, n = 99 (%)	Controls, n = 2012 (%)	OA, n = 99 (%)
Normal	68.6	82.2	60.0	62.6
Osteopenic	29.5	17.8	37.4	32.7
Osteoporotic	1.9	—	2.6	4.7
	p = 0.008*		p = 0.394*	

* Pearson chi-square test.

(FN, +3.7%, +3.9%; $p < 0.001$; Tables 3 and 4). We could not confirm that the date of first diagnosis had an influence on the differences in BMD, BMC, and area observed between the patients with primary OA and controls.

When the unadjusted means were examined, we found almost identical results. Similarly, when applying correction for height and weight separately instead of for BMI, we found the same results, except for the disappearance of the apparent difference in the trochanteric BMC at baseline ($p = 0.109$ instead of 0.017).

Between-group changes in DEXA parameters during followup. The BMD, BMC, and projected area of the regions of interest of the hip changed significantly between the 2 measurements, in both the OA group and the controls. The changes were different in the OA and control groups, though: the BMD loss was greater at all 3 regions of interest in the OA group. There were no differences in the rates of modification of the projected area or measured BMC, except for the trochanteric region, where the increase of BMC was greater in the control group (Table 5).

DISCUSSION

Our analysis of the hips of women with OA showed significant differences in the DEXA measures compared to controls. The BMC of all 3 regions of interest were higher in OA patients, as was the projected area of the FN. There were no statistically significant differences in BMD at those sites, although a trend for increased values in the OA group was observed. At the 5-year followup measurements, the projected area of the FN was greater in the OA group. There was no difference in the projected area of the TR, or in the BMC of the FN or TR. When the unadjusted means for the various DEXA measures were analyzed, only small differences appeared in the BMD of the TR region at baseline. That those become nonsignificant after adjustment for height and weight, and for the BMI, shows that those regions composed mainly of trabecular bone might be more easily influenced by variations in body measurements.

Between the 2 measurements, we observed a BMD loss at all regions of interest within both groups, except at the trochanteric region in the control group. At this site, the projected area increased less than the BMC, resulting in a small but significant increase in BMD. The projected areas increased at all measurement sites. The observed BMD loss was due to the combined effect of the increase in the projected area and the decrease in BMC. While there was no

Table 3. BMD, BMC, and projected area of the femoral neck and trochanter at baseline in OA patients and controls.

	OA, n = 99	Controls, n = 2012	Difference Between Group Means, %	p*
Femoral neck				
BMD (g/cm^2)	0.968 ± 0.129†	0.925 ± 0.126	4.64	0.158
BMC (g)	4.85 ± 0.70	4.48 ± 0.72	8.26	0.004
Area (cm^2)	5.02 ± 0.44	4.84 ± 0.35	3.72	0.001
Trochanter				
BMD (g/cm^2)	0.901 ± 0.137	0.841 ± 0.133	7.1	0.102
BMC (g)	12.01 ± 3.01	10.60 ± 2.61	13.3	0.017
Area (cm^2)	13.22 ± 2.07	12.54 ± 1.89	5.4	0.086

† Mean ± SD. * ANCOVA with adjustment for age and body mass index, menopausal status at baseline, and previous HRT use. BMD: areal bone mineral density. BMC: bone mineral content.

Table 4. BMD, BMC, and projected area of the femoral neck and trochanter at 5-year followup in OA patients and controls.

	OA, n = 99	Controls, n = 2012	Difference,%	p*
Femoral neck				
BMD (g/cm^2)	0.919 ± 0.124†	0.895 ± 0.125	2.7	0.947
BMC (g)	4.66 ± 0.73	4.37 ± 0.71	6.6	0.079
Area (cm^2)	5.07 ± 0.41	4.88 ± 0.33	3.9	< 0.001
Trochanter				
BMD (g/cm^2)	0.880 ± 0.139	0.847 ± 0.138	3.9	0.378
BMC (g)	12.36 ± 2.875	11.47 ± 2.87	7.8	0.736
Area (cm^2)	13.95 ± 2.05	13.43 ± 1.97	3.9	0.717

† Mean ± SD. * ANCOVA with adjustment for age and body mass index, menopausal status at the time of the 5-year measurement, and previous HRT use. BMD: areal bone mineral density. BMC: bone mineral content.

Table 5. Absolute changes in DEXA measures in OA subjects and controls between the baseline and 5-year followup measurements.

	OA, n = 99	Controls, n = 2012	p*
Femoral neck			
BMD (g/cm^2)	–0.05	–0.03	0.002
BMC (g)	–0.20	–0.11	0.138
Area (cm^2)	0.04	0.03	0.054
Trochanter			
BMD (g/cm^2)	–0.02	0.01	< 0.001
BMC (g)	0.37	0.86	0.001
Area (cm^2)	0.76	0.90	0.429

* ANCOVA with adjustment for baseline BMD (respectively BMC, area), followup duration, age, body mass index, menopausal status at baseline, and HRT use during followup.

difference in the accrual rate of the projected area between the OA group and controls, the BMD loss was greater in the OA group for every region of interest.

These observations illustrate a concept that is somewhat obscured by the usual DEXA terminology, where the expression "bone mineral density" is commonly used. The areal BMD values, expressed in g/cm^2, in T score (standard deviations from the mean of a young reference population), or in Z score (standard deviations from the mean of the age matched population), have been chosen as the gold standard for the diagnosis of osteoporosis, and BMD measurements at a variety of skeletal sites consistently show that the probability of fracture can be estimated in this way[25,30-33]. However, it is important to respect the limitations of the DEXA technique, namely the uncertainty introduced by projecting a volume onto a surface, losing the ability to account for depth (shape or size) in the interpretation of the measurements. Moreover, it has been shown that technical issues may interfere with the accuracy of measurements of changes in DEXA parameters; in particular, a dependency of the measured bone projected area on the BMC has been observed in spine measurements, and this could also apply to hip measurements, although this has not been confirmed[28].

Previous cross-sectional studies have found greater BMD in the proximal femur of OA patients[5-10] compared with age matched controls. We could not confirm these results, either at baseline or at the 5-year measurement. Bruno, *et al*[6] observed that BMD of the femur was increased in the early stages of OA (Kellgren-Lawrence scores of 1–2), but decreased in the later stages. Our cases with primary OA were selected according to the indication for surgical replacement or osteotomy, and might represent more advanced situations, with BMD rapidly decreasing because of immobility. The faster rate of BMD loss we observed in the OA group supports this hypothesis.

In theory, if the femoral neck were a perfect cylinder, an increase in the external bone dimensions by a homogenous periosteal apposition would increase both the depth and the width of the cylinder in its flat projection. A linear increase in the projected bone size would be accompanied by a quadratic increase in the bone volume, and in the BMC. This also means that, in this hypothetical and ideal situation, a linear increase in the femoral neck radius would result in a quadratic increase of the femoral neck BMD. Obviously, the femoral neck is not an ideal cylinder[34], it is structurally not homogenous, and the dynamic changes in bone involve endosteal resorption as well as periosteal apposition. This explains our observation of larger bones and higher BMC, but similar BMD in the OA group compared with the healthy controls.

It has been shown that an approximation of the volumetric BMD, using a circular cross-section derived from the measured projected area, correlated positively with the volumetric density estimated using DEXA data supplemented by MRI-derived bone measurements, but led constantly to overestimation of the volume and to underestimation of the volumetric density[34]. The increased size of the bone might provide better resistance to fracture, as deficit of bone size has been associated with a higher fracture risk in men[35]. Using pelvis radiographs in anteroposterior projection, Michelotti, *et al* have shown that in women with hip fractures, the femoral neck cortex was thinner, and the femoral neck diameter was larger[36]. Similar results were found in another study, which described a deficit of femoral neck diameter in men, but not in women[37]. The diameter of the femoral neck in anteroposterior projection is greater than in mediolateral projection, revealing an elliptical cross-section. An increase in the bone size and BMC due to OA might remain invisible in the areal BMD measurements, although they could provide a significantly increased resistance to fracture.

Comparison of absolute changes in the projected area, BMC, or BMD between the 2 measurements, with adjustment for baseline DEXA values and followup duration, HRT use, menopausal status, BMI, and age, consistently indicated greater BMD loss in our patients with severe OA compared with the controls in all regions of interest. The reason for the difference is not obvious, but it supports the observation by Bruno, *et al*[6] that the later Kellgren-Lawrence stages of OA might be associated with lower and not higher BMD. Patients might spare the affected side by increasing the load on the opposite joint during walking, or mobility might be decreased sufficiently to induce immobility related BMD loss. There might be an effect of OA related pain on mobility outdoors and sunlight exposure. We hypothesize that there might also be an association between higher BMI and the number of comorbidities, different diet, and pain medications that could influence the BMD measurements, but these were not assessed in this study.

We think that the date of first diagnosis, based on med-

ical records, is misleading. Perhaps the women without data for the date of first diagnosis all had primary OA long before this study, which would explain why all the differences were in this group. At least we could not demonstrate, based on the available data, that the date of first diagnosis influenced the differences in BMD, BMC, and area observed between patients with primary OA and controls.

Finally, we compared the distribution of osteopenic, osteoporotic, and normal women at baseline and at the 5-year measurements, in the OA and control groups. A significant number of expected osteoporosis cases were found in the control group, while there were no osteoporosis cases in the OA group. We found no difference in the prevalence of osteoporosis either at baseline or at the 5-year followup measurement. When the diagnosis of osteopenia was taken into account, the diagnoses of osteoporosis or osteopenia were less frequent at baseline in the OA group compared to controls, while the difference disappeared at followup.

Patients with hip OA often suffer pain on internal rotation of the hip, and this is very likely to affect positioning for bone density estimation. It has been shown in several studies that malrotation of the hip is an important confounding factor when interpreting serial BMD values, and that proper positioning of the femur during a scan can improve precision significantly[38-41]. In our study, all measurements were carried out by trained personnel. To avoid possible errors caused by malrotation of the hip, all our measurements were made using a stabilization apparatus for the foot, which keeps the foot in the same position between subjects.

We conclude that BMD at the hip of women treated surgically for hip or knee OA was not different from that of healthy controls when measured twice with a 5-year interval. However, at a 5-year followup, OA can be accompanied by an increase in bone size or changes in shape, and faster loss in BMD.

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