Bone mineral density in patients with rapidly destructive or common hip osteoarthritis

P. Richette[1], E. Vicaut[2], M.-C. de Vernejoul[1], P. Orcel[1], T. Bardin[1]

[1]Fédération de Rhumatologie, Hôpital Lariboisière, and [2]Unité de Recherche Clinique, Hôpital Fernand Widal, UFR médicale, Assistance Publique-Hôpitaux de Paris, Université Paris 7, 75475 Paris Cedex 10, France.

Pascal Richette, MD, PhD
Eric Vicaut, MD, PhD
Marie-Christine de Vernejoul, MD, PhD
Philippe Orcel, MD, PhD
Thomas Bardin, MD

This work was funded by the Association Rhumatisme et Travail (Centre Viggo Petersen, Hôpital Lariboisière, service de Rhumatologie).

Please address correspondence to:
Dr. Pascal Richette,
Fédération de Rhumatologie,
Hôpital Lariboisière,
2 Rue Ambroise Paré,
75475 Paris Cedex 10, France.
E-mail: pascal.richette@lrb.aphp.fr

Received on May 26, 2008; accepted in revised form on November 28, 2008.

© Copyright CLINICAL AND EXPERIMENTAL RHEUMATOLOGY 2009.

Key words: Osteoarthritis, hip, bone mineral density, T-score, rapidly destructive.

Competing interests: none declared.

ABSTRACT

Background. *Recent findings that subchondral insufficiency fracture in the femoral head may precede rapid chondrolysis suggest a role for systemic low bone mass in the genesis of rapidly destructive hip osteoarthritis (RDHOA).*

Objective. *To compare bone mineral density (BMD) in females with RDHOA and those with common hip osteoarthritis (OA).*

Methods. *This prospective case-control study involved 26 females with RDHOA recruited from our institution between March 2000 and November 2006. BMD was measured at the femoral neck and lumbar spine (L1-L4) by dual-energy x-ray absorptiometry. For comparison, BMD was measured in 33 women with common hip OA who were scheduled for primary total hip arthroplasty.*

Results. *Patients with RDHOA and those with common hip OA were similar in age (74.9±9.9 vs. 74.7±8.8 years) and BMI (26.3±4.3 vs. 26.3±5 g/m^2) and did not differ in mean BMD at the lumbar spine (1.0±0.2 vs. 1.1±0.2 g/cm^2; mean T-score: -0.6±1.3 vs. -0.8±1.5) or at the femoral neck (0.7±0.1 vs. 0.8±0.2 g/cm^2; mean T-score: -1.5±1.1 vs. -1.4±1.4).*

Conclusion. *The results of this study do not suggest a role for systemic low bone mass in the pathophysiology of RDHOA.*

Introduction

Cartilage destruction in common hip osteoarthritis (OA) is a slow process, which might be related to alteration in type 2 collagen metabolism (1). Rapidly destructive hip osteoarthritis (RDHOA) is considered an uncommon form of hip OA largely encountered in elderly postmenopausal women. RDHOA is characterized by rapid chondrolysis within the osteoarthritic coxo-femoral joint, with no evidence for other types of rapidly destructive arthropathy (2). The mechanisms underlying the rapid chondrolysis are still elusive, despite numerous attempts to elucidate the pathophysiology of the disease.

Recent reports of subchondral insufficiency fracture of the femoral head triggering rapid hip chondrolysis suggest a role for subchondral bone in the pathophysiology of RDHOA (3). Patients with subchondral insufficiency fracture of the femoral head have been reported to be mostly osteopenic or osteoporotic (4, 5). These data led us to hypothesize that systemic low bone mass could play a role in the genesis of RDHOA.

We aimed to compare bone mineral density (BMD) in female patients with RDHOA and those with common hip OA.

Subjects and methods

Study design

This prospective cross-sectional case-control study involved 59 women who were recruited in our tertiary care hospital. All patients provided appropriate consent.

Patients with RDHOA

RDHOA was defined by the following criteria: severe hip pain, symptom onset within the past 2 years, annual rate of joint space loss >1 mm and erythrocyte sedimentation rate <20 mm/h, according to the criteria proposed and used in previous publications (6, 7). Patients were excluded if they had a history of other conditions that might result in destructive hip arthropathy, including septic arthritis, inflammatory arthritis, osteonecrosis with secondary OA, pyrophosphate-associated arthropathy and neuropathic osteoarthropathy. Joint space width was measured manually on repeated x-rays, at the narrowest site.

During the 6 years of the study, 26 women received a diagnosis of RDHOA in our rheumatological department and were enrolled.

Controls

The control population consisted of 33 consecutive female who met the American College of Rheumatology criteria for primary hip OA, and who were scheduled for primary total hip arthroplasty.

Bone mineral density measurements

All participants underwent BMD measurements at the femoral neck of the OA hip and at the lumbar spine (L1-L4,

anterior-posterior view) by use of the same dual-energy x-ray absorptiometry (DEXA) equipment (Hologic®). BMD measurements were performed at time of diagnosis for RDHOA patients and before prosthesis surgery for controls. In agreement with the World Health Organization criteria, osteopenia was defined as a T-score between -1 and -2.5 and osteoporosis a T-score <-2.5. Z-score, the standard deviation from the mean value of age-matched normal women, was used to explore any difference in BMD between patients with hip OA and a reference population of the manufacturer's database.

Statistical analysis

All data were tested for normality by the Kolmigorov-Smirnov test. Demographic data and DEXA values were normally distributed and are presented as mean ± SD. Comparisons between the two groups involved the Student's *t*-test and Fisher's exact probability test for binary variables. Two-sided significance was fixed at 5%. All tests involved use of SAS v9.13 (SAS Inst.; Gary, IN).

Results

The characteristics of patients and controls are summarized in Table I. Patients with RDHOA and controls did not differ in age (mean 74.9±9.9 *vs*. 74.7±8.8 years) or BMI (mean 26.3±4.3 *vs*. 26.3±5 kg/m^2). However, age at menopause was slightly inferior in the RDHOA group as compared with the control group (mean 47.0 ±4.3 *vs*. 49.3±3.2 years, p=0.03).

BMD did not differ between the two groups. In patients with RDHOA, mean BMD was 1.0±0.2 g/cm^2 at the lumbar spine (T-score: -0.6±1.3) and 0.7±0.1 g/cm^2 at the femoral neck (T-score: -1.5±1.1). In controls, mean BMD was 1.1±0.2 g/cm^2 at the lumbar spine (T-score: -0.8±1.5) and 0.8±0.2 g/cm^2 at the femoral neck (T-score: -1.4±1.4) (Table II). Similar results were found when BMD was compared after adjustment on confounding factors such as age at menopause, smoking status, use of estrogen replacement therapy, use of bisphosphonates and previous fractures (p=0.6 and p=0.1 for ANCOVA of BMD at lumbar spine and at femoral neck, respectively).

The prevalence of osteoporosis and osteopenia was similar in patients and controls: 15% (n=4/26) *vs*. 15% (n=5/33) and 35% (n=9/26) *vs*. 39% (n=13/33), respectively. Finally, BMD at the femoral neck or lumbar spine did not differ between the RDHOA and control groups on normalization of data by the Z score.

Table I. Characteristics of patients and controls.

	Rapidly destructive hip OA (n=26 women)	Slowly progressive hip OA (n=33 women)	*p*-value
Age (years)	74.9 ± 9.9	74.7 ± 8.8	0.92
Body mass index (g/cm^2)	26.3 ± 4.3	26.3 ± 5.0	0.97
Age at menopause (years)	47.0 ± 4.3	49.3 ± 3.2	0.03
Current smokers (%)	3 (11.5)	5 (15.6)	0.72
Previous fractures (%)	5 (19.2)	5 (15.6)	0.74
Current users of ERT (%)	3 (11.5)	4 (12.5)	0.99
Current users of bisphosphonates (%)	2 (7.7)	4 (12.5)	0.68

Results are means ± SD or n. (percentages). ERT: estrogen replacement therapy. *P*-values correspond to *t*-test for continuous variables and Fisher's exact probability test for binary variables.

Table II. Bone mineral density (BMD) of females with rapidly destructive hip osteoarthritis (OA) and those with slowly progressive hip osteoarthritis.

	Rapidly destructive hip OA (n=26 women)	Slowly progressive hip OA (n=33 women)	*p*-value
Femoral neck			
Bone mineral density (g/cm^2)	0.7 ± 0.1	0.8 ± 0.2	0.4
T-Score*	-1.5 ± 1.1	-1.4 ± 1.4	0.86
Z-Score	0.4 ± 1.1	0.8 ± 1.3	0.29
Lumbar spine			
Bone mineral density (g/cm^2)	1.0 ± 0.2	1.1 ± 0.2	0.67
T-Score*	-0.6 ± 1.3	-0.8 ± 1.5	0.62
Z-Score	0.7 ± 1.4	1.0 ± 1.4	0.52

Results are means ± SD. BMD: bone mineral density.
*World Health Organization T-score for osteopenia (between -1 and -2.5) and osteoporosis (T-score <-2.5).

Discussion

We compared female patients with RDHOA and those with common hip OA and found no difference in BMD between the two groups. The prevalence of osteopenia and osteoporosis were similar between the groups and to those found in previous studies evaluating BMD in women with hip OA scheduled for total joint replacement (8). Moreover, our results are in agreement with those failing to demonstrate a difference in levels of bone metabolic markers between patients with RDHOA and those with slow-progressing hip OA (6, 9). Elevated levels of serum type I collagen C-telopeptide were found in patients with RDHOA but likely originated from focal degradation of the affected hip joint rather than from other skeletal sources (9).

BMD measurements were performed at femoral neck and lumbar spine in both groups in order to study the putative role of systemic low bone mass in the genesis of RDHOA. We did not evaluate the BMD in the subchondral area of the hip for several reasons. First, subchondral area is well known to be difficult for the measurement of BMD because of the acetabular coverage. Second, the severity of the subchondral bone destruction frequently encountered in RDHOA at time of diagnosis could lead to higher BMD values, and could not reflect the true pathophysiology of RDHOA.

The importance of subchondral bone in the pathogenesis of OA (10) has been recently emphasized by MRI studies showing bone marrow edema, which may represent histologic microfractures, associated with pain and cartilage loss (11). Yamamoto *et al*. (3) and others (12) have showed that subchondral insufficiency fracture of the femoral head, a recently recognized condition which occurs predominantly in osteopenic or osteoporotic patients (4, 5, 13), is one of the important factors involved in the pathogenesis of RDHOA. These data highlight the role of the femoral subchondral bone in hip OA and raise the question of the influence of systemic low bone mass in the pathogenesis of this disease, not found in our study.

The relation between subchondral bone changes in OA and a more generalized alteration in skeletal bone mass remains elusive, although a weak correlation was found between low BMD at the hip and progression of OA knee (14). Data from cross-sectional studies suggested that the incidence of osteoporosis was inversely associated with incidence of OA (15). This matter is controversial (16), and recent data have found hip OA not associated with increased BMD at the femoral neck, in a comparison with healthy controls (17, 18). To our knowledge, this is the first work that evaluated BMD in patients with RDHOA. Nevertheless, we are aware that one limitation of our study may be the small number of subjects, due to the very low incidence of RDHOA. This might have reduced the statistical power to detect a difference in BMD.

In conclusion, these data suggest that systemic low bone mass does not play a role in the genesis of RDHOA in females.

Acknowledgments

We thank Dr. S. Lamy and Prof. F. Lioté for their help with the data collection.

References

1. CONROZIER T, POOLE AR, FERRAND F *et al*.: Serum concentrations of type II collagen biomarkers (C2C, C1, 2C and CPII) suggest different pathophysiologies in patients with hip osteoarthritis. *Clin Exp Rheumatol* 2008; 26: 430-5.
2. LEQUESNE M, RAY G: La coxopathie destructrice rapide idiopathique. *Rev Rhum Engl Ed* 1989; 56: 115-19.
3. YAMAMOTO T, BULLOUGH PG: The role of subchondral insufficiency fracture in rapid destruction of the hip joint: a preliminary report. *Arthritis Rheum* 2000; 43: 2423-7.
4. YAMAMOTO T, SCHNEIDER R, BULLOUGH PG: Insufficiency subchondral fracture of the femoral head. *Am J Surg Pathol* 2000; 24: 464-8.
5. YAMAMOTO T, BULLOUGH PG: Subchondral insufficiency fracture of the femoral head: a differential diagnosis in acute onset of coxarthrosis in the elderly. *Arthritis Rheum* 1999; 42: 2719-23.
6. GARNERO P, CONROZIER T, CHRISTGAU S, MATHIEU P, DELMAS PD, VIGNON E: Urinary type II collagen C-telopeptide levels are increased in patients with rapidly destructive hip osteoarthritis. *Ann Rheum Dis* 2003; 62: 939-43.
7. GARNERO P, CHARNI N, JUILLET F, CONROZIER T, VIGNON E: Increased urinary type II collagen helical and C telopeptide levels are independently associated with a rapidly destructive hip osteoarthritis. *Ann Rheum Dis* 2006; 65: 1639-44.
8. MAKINEN TJ, ALM JJ, LAINE H, SVEDSTROM E, ARO HT: The incidence of osteopenia and osteoporosis in women with hip osteoarthritis scheduled for cementless total joint replacement. Bone 2007; 40: 1041-7.
9. BERGER CE, KRONER A, STIEGLER H, LEITHA T, ENGEL A: Elevated levels of serum type I collagen C-telopeptide in patients with rapidly destructive osteoarthritis of the hip. *Int Orthop* 2005; 29: 1-5.
10. BURR DB: Increased biological activity of subchondral mineralized tissues underlies the progressive deterioration of articular cartilage in osteoarthritis. *J Rheumatol* 2005; 32: 1156-8; discussion 1158-9.
11. CONAGHAN PG, FELSON D, GOLD G, LOHMANDER S, TOTTERMAN S, ALTMAN R: MRI and non-cartilaginous structures in knee osteoarthritis. *Osteoarthritis Cartilage* 2006;14 (Suppl. A): A87-94.
12. WATANABE W, ITOI E, YAMADA S: Early MRI findings of rapidly destructive coxarthrosis. *Skeletal Radiol* 2002; 31: 35-8.
13. ELKAYAM O, PARAN D, FLUSSER G, WIGLER I, YARON M, CASPI D: Insufficiency fractures in rheumatic patients: misdiagnosis and underlying characteristics. *Clin Exp Rheumatol* 2000; 18: 369-74.
14. HART DJ, CRONIN C, DANIELS M, WORTHY T, DOYLE DV, SPECTOR TD: The relationship of bone density and fracture to incident and progressive radiographic osteoarthritis of the knee: the Chingford Study. *Arthritis Rheum* 2002; 46: 92-9.
15. MCDONALD BLUMER MH: Bone mineral content versus bone density in a population with osteoarthritis: a new twist to the controversy? *J Rheumatol* 2005; 32: 1868-9.
16. AMIN S: Osteoarthritis and bone mineral density: what is the relation and why does it matter? *J Rheumatol* 2002; 29: 1348-9.
17. AROKOSKI JP, AROKOSKI MH, JURVELIN JS, HELMINEN HJ, NIEMITUKIA LH, KROGER H: Increased bone mineral content and bone size in the femoral neck of men with hip osteo-arthritis. *Ann Rheum Dis* 2002; 61: 145-50.
18. SANDINI L, AROKOSKI JP, JURVELIN JS, KROGER H: Increased bone mineral content but not bone mineral density in the hip in surgically treated knee and hip osteoarthritis. *J Rheumatol* 2005; 32: 1951-7.