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

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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 osteoarthrits (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/m²) and did not differ in mean BMD at the lumbar spine (1.0±0.2 vs. 1.1±0.2 g/cm²; 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/cm²; 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,

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BRIEF PAPER

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

### 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/m²). 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/cm² at the lumbar spine (T-score: -0.6±1.3) and 0.7±0.12 g/cm² at the femoral neck (T-score: -1.5±1.1). In controls, mean BMD was 1.1±0.2 g/cm² at the lumbar spine (T-score: -0.8±1.5) and 0.8±0.2 g/cm² at the femoral neck (T-score: -1.4±1.4) (Table II). Similar results were found when BMD was compared after adjustment for 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.

### 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 subchondral insufficiency fracture of the femoral head. A preliminary report. Arthritis Rheum 2000; 43: 2423-7.


