

Bone Mineral Content versus Bone Density in a Population with Osteoarthritis: A New Twist to the Controversy?



It is well recognized that osteoarthritis (OA) and osteoporosis are common musculoskeletal disorders that frequently coexist in the same patient population. However, what is more controversial and less well understood is the potential relationship between these 2 processes in a given individual. For 3 decades studies have been exploring possible relationships. Although most findings suggest an inverse relationship between the presence of OA and osteoporosis¹⁻³, other data indicate that the 2 processes are not mutually exclusive and that the prevalence of osteoporosis in the OA population is virtually identical to that seen in the "normal" population^{4,5}.

Although local factors such as osteophyte formation and subchondral sclerosis can alter/increase the measured values from bone density assessments done by central dual energy x-ray absorptiometry (DEXA) of the spine and hip, the Study of Osteoporotic Fractures has provided data that bone mineral density (BMD) measurements at remote sites such as proximal and distal sites of the radius and the calcaneus in women with severe hip OA were increased relative to individuals without OA¹. Additionally, the Framingham data² also reveal that in those older individuals with OA, BMD measured at sites away from the arthritic process was increased, thus suggesting a lower occurrence of osteoporosis in the osteoarthritic population, at least as defined by densitometric criteria. Interestingly, this seemed to hold true in individuals with osteophytosis rather than in those with isolated joint space narrowing. Similarly, the Chingford study³ looked at older women with knee OA and found that those with new onset disease, as defined by osteophyte formation, had higher baseline axial BMD than those without incident disease. The increase in BMD did not appear to correlate with patient characteristics of body weight, age, physical activity, or medication use⁴. Additionally, of the 834 women who were followed in the Chingford study, about one-quarter sustained a distal forearm and/or vertebral frac-

ture during the study period, and when analyzed for incident OA, this group had a reduced risk of developing OA during the study.

In contrast to these data, Antoniadou, *et al* noted in their study of female twins over 40 years of age that higher bone density was noted in the affected osteoarthritic hip when osteophytes were present, but not an affected hip with isolated joint space narrowing; moreover, "no clear association was found between hip OA and BMD at the contralateral site, lumbar spine, or total body."⁵ Drees, *et al* have recently reviewed 117 patients with documented OA and found that 23% of the women and 20% of the men in this group had osteoporosis as defined by standard criteria⁶. They report that this is very similar to the prevalence of osteoporosis in their general, age-matched female population and is somewhat higher than expected in their male cohort. They conclude that the presence of OA is not "protective" against the occurrence of osteoporosis.

Recognizing that osteoporosis as defined by low BMD scores on DEXA is clinically important because of the well documented predictive relationship of low BMD to osteoporotic fractures, it is important to explore the potential interrelationship of OA and fracture. Although intuitively, one might expect that the increased BMD noted in the OA population would be associated with a decrease in fragility fractures, the data are conflicting. The Mediterranean Osteoporosis Study (MEDOS), among others, has data to support a significant protective effect of preexisting OA on new hip fracture occurrence⁷, and Hart's data revealed that women who had sustained a recent fracture had a diminished chance of developing radiographic OA³. On the other hand, Australian data suggest that fracture incidence was not lowered by the presence of OA despite the associated increase in BMD⁸. Arden, *et al* confirmed these findings^{9,10}. They also noted that there was increased risk of fracture in those individuals with hip OA and hypothesized

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that this might be due to changes in patient mobility and fall potential.

In this issue of *The Journal*, Sandini, *et al* add a further dimension to the conflicting data regarding the relationship of OA and BMD¹¹. Their findings support their construct that changes in hip geometry and bone mineral content are responsible for the apparent relationship between OA and the occurrence of higher BMD. They assess a study population of subjects with primary OA using a population database obtained from OSTPRE (the Osteoporosis Risk Factor and Prevention Study), conducted in Finland. Diagnosis of primary OA was made by medical record validation of patients' self-report of hip and/or knee OA, focusing on those with severe enough disease to require orthopedic intervention. Bone mass was determined by looking at DEXA in its component parts, where the actual measurements include estimates of the bone mineral content and projected area of the target region, from which calculation of the BMD is made. The authors' findings suggest that bone mineral content is increased in the setting of geometric alterations in hip size and shape, which seems to occur in the OA population. As BMD is calculated from bone mineral content divided by the target area, they found that, overall, the BMD in the OA population was not increased significantly. Their findings that bone mineral content and hip area are increased but that hip BMD is unchanged in the OA population add yet another dimension to the controversy.

Their data give rise to additional questions: in particular, why are we interested in the bone density of our patients, particularly, in this case, those with OA?

If we are concerned about fracture potential as part of an overall assessment of fracture risk, DEXA would be considered a component of the evaluation; therefore, understanding potential confounders that a disease state may confer on a patient or a population of patients would be important. Given the lack of consensus on interpretation of the data, for each individual patient, results of bone density assessment should be placed in the context of their other risk factors for low bone mass and fracture (as with any other patient) when making therapeutic decisions.

However, the discrepant results amassed thus far give pause to consider that the DEXA data are telling us something different. The overall observation may provide clues into the pathogenesis of OA; moreover, by exploring further

the influences of various biochemical, cellular, and structural forces on bone metabolism and remodeling, we may increase our understanding of the causes of OA, and subsequently increase our therapeutic repertoire for this common arthritic process.

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