

Annals of the
**RHEUMATIC
 DISEASES**

Leader

Bone mineral density and osteoarthritis

Bone mineral density and osteoarthritis—this title prompts two questions: firstly, can bone mineral density be measured reliably in areas where osteophytes are present; and secondly, is osteoarthritis associated with an increased bone mineral density? This paper will try to address these questions.

Single photon absorptiometry measures bone mineral density in the distal forearm where cortical bone predominates. This site is distant from and therefore unaffected by areas of osteoarthritis and osteophytosis but it has the disadvantage of correlating relatively poorly with early trabecular bone loss in the hip and vertebrae. Dual photon and dual x ray absorptiometry can measure bone mineral density in the axial skeleton. These methods give minimal radiation exposure and have a high precision (<2%).¹ The usual region of interest measured for assessment of lumbar spine bone mineral density (L2–L4) is, however, also commonly affected by degenerative spinal disease including osteophytosis. Osteoporosis and osteophytosis of the spine may occur together. Therefore it is important to know whether or not osteophytosis in the L2–L4 region interferes with bone mineral density measurements. A number of workers have addressed this problem. Orwoll *et al*² measured bone mineral density in 129 white men (71 admitted to hospital and 58 control subjects from the community) and also obtained corresponding lateral lumbar spine radiographs. An observer who was blinded to the bone mineral density result graded the amount of osteophytic or vascular calcification present in the L2–L4 region. Fifty five of these subjects were found to have osteophytic calcification. This had an effect on the spinal bone mineral density: a significant correlation was found between osteophyte index and spinal bone mineral density. (In contrast, they did not find that vascular calcification had any demonstrable effect on bone mineral density). Reid *et al*³ used the calcification scoring system described by Orwoll *et al* to assess whether a similar effect could be seen in a group of 130 postmenopausal women. Spinal bone mineral density was measured using dual energy x ray absorptiometry. They did not find any correlation between spinal bone mineral density and either osteophytic or vascular calcification. When they proceeded to multiple regression analysis, however, which also included age and body weight, they found a small but significant effect of the osteophyte score. It is possible that osteophytosis is more marked in men. Dawson-Hughes and Dallal⁴ looked at the effect of osteophytosis on the measurement of the annual adjusted rate of bone loss in 293 postmenopausal women. They measured spinal and distal forearm bone mineral density using dual and single photon

absorptiometry respectively. Seven of these women were found to have osteophytosis and 31 had calcification of the aorta on lateral lumbar spinal radiographs; five women had other miscellaneous (unspecified) abnormalities seen radiographically. The annual adjusted rate of bone loss in the spine was less in those with abnormal spinal radiographs compared with the normal group whereas there was no difference between the two groups with respect to the adjusted bone loss occurring at the wrist. This again suggests that osteophytic calcification can interfere with the assessment of lumbar spine bone mineral density. In each of these studies the frequency of osteophytosis was found to increase with age. The effect of osteoarthritis of the facet joints on bone mineral density was not addressed by these workers. Laitinen *et al*⁵ assessed the effect of osteophytosis and osteoarthritis of the lumbar spine on spinal bone mineral density in 72 women over the age of 50. They found that osteoarthritis or osteophytosis, or both, was associated with an increase in spinal bone mineral density; however, they did not analyse separately the effect of osteoarthritis of the facet joints alone on bone mineral density.

These findings indicate that spinal osteophytosis in the L2–L4 region can lead to falsely increased lumbar spine bone mineral density measurements and could potentially impair the recognition of coincident spinal osteoporosis.

Measurement of femoral bone mineral density is possible using dual energy x ray absorptiometry and dual photon absorptiometry; higher precision can be achieved using dual energy x ray absorptiometry. As the regions of interest usually studied (femoral neck, Ward's region, trochanteric region) are distal to the sites of femoral osteophytosis this does not pose a direct problem to the measurement of bone mineral density in these regions. Indirectly, however, there are still potential pitfalls in measuring femoral bone mineral density in a patient with osteoarthritis of the hip. This is because patient positioning is critical.⁶ Accurate positioning may be technically more difficult in patients with severe osteoarthritis of the hip.

Since the classic paper by Foss and Byers⁷ the question has often been posed 'Is bone mineral density increased in association with osteoarthritis?'. Foss and Byers⁷ observed that primary osteoarthritis of the hip is rarely found together with fracture of the neck of femur. This may suggest that patients with osteoarthritis have increased bone mineral density, which might be generalised throughout the skeleton or local (confined to the region adjacent to the osteoarthritic joint). Foss and Byers,⁷ Carlsson *et al*,⁸ and Solomon *et al*⁹ studied bone mineral density in patients with

primary osteoarthritis of the hip; the sites of measurement were distant from the hip. Foss and Byers⁷ assessed the bone mineral density of the second metacarpal joint using radiographs of the hand. They observed an 'abnormally high' bone mineral density in the patients with osteoarthritis compared with control subjects. Using similar methods Solomon *et al*⁹ found that the bone mineral density at the second metacarpal joint was not increased in women with osteoarthritis of the hip compared with normal control subjects but did tend to be higher in older men with osteoarthritis of the hip compared with control subjects. Formal statistical comparisons were not made in either of these studies. Carlsson *et al*⁸ used single photon absorptiometry to compare the bone mineral density at the forearm in patients with osteoarthritis of the hip with that of normal control subjects. When a proximal site was chosen (representing predominantly cortical bone) there was no significant difference between patients and control subjects. When a distal site was measured (representing predominantly trabecular bone), however, the bone mineral density was significantly greater in women with osteoarthritis of the hip compared with control subjects. Overall these studies provide little evidence to support the theory that there is a generalised increase in bone mineral density in patients with primary osteoarthritis of the hip.

Some patients do not have osteoarthritis confined to one or two joints but have what is termed 'generalised osteoarthritis'. Generalised osteoarthritis was defined by Kellgren and Moore¹⁰ as the presence of radiological evidence of osteoarthritis in six or more groups of joints which were most likely to include the first carpometacarpals, proximal interphalangeals, apophysial joints of the spine, the knees, and the first tarsometatarsals. Dequeker and coworkers^{11, 12} have observed several anthropometric differences between postmenopausal women with osteoporosis and those with generalised osteoarthritis. The bone mineral density of such patients has also been studied.^{13, 14} Price *et al*¹³ measured bone mineral density of trabecular and cortical bone at the radius in 40 women with generalised osteoarthritis and compared the results with normal values obtained from prediction equations. Trabecular bone mineral density was measured at the distal radius, and cortical bone mineral density was measured at the midshaft of the radius. Two sets of prediction equations were used. No difference between patients or control subjects was seen with respect to cortical bone mineral density. Using prediction equations which were based on age alone the patients were found to have a significantly higher trabecular bone mineral density. When prediction equations which also took into account weight and height were used, however, the trabecular bone mineral density of patients with generalised osteoarthritis was not significantly higher than predicted. Once again this study measured bone mineral density at a site distant from osteoarthritic joints.

A different approach was used by Reid *et al*¹⁴ who measured total body calcium by *in vivo* neutron activation analysis. This gives a measure a total bone mass rather than bone mineral density. They studied 15 women with generalised osteoarthritis and compared their results with those from 12 healthy control subjects matched for age, menopausal status, and skeletal size. No significant difference was found. The small sample size does not exclude the possibility that small increases in bone mass might be present in generalised osteoarthritis. The results of the studies by Reid *et al*¹⁴ and Price *et al*¹³ suggest that generalised osteoarthritis is not associated with an increased bone mineral density throughout the skeleton but it is unclear whether or not these studies had the power to detect small but potentially important differences between the osteoarthritic subjects and controls.

We have addressed the possibility that Foss and Byers' observations could be explained by an increase in bone mineral density in the proximity of the hip joint. We measured femoral bone mineral density in 50 patients with primary osteoarthritis of the hip using dual energy x ray absorptiometry and found that the bone mineral density was significantly increased at the femoral neck and Ward's region compared with predicted control values. This suggests that bone mineral density may indeed be increased in the region adjacent to an osteoarthritic joint. Such an increase might be a consequence of the osteoarthritic process—perhaps induced by changes in blood flow—or might antedate the osteoarthritic degeneration. A change in bone mineral density is likely to be associated with changes in the mechanical properties of bone. Radin and Paul¹⁵ found that the maximum intra-articular pressure experienced by a joint subjected to impulsive loading was dependent on the properties of the capsule and bone (rather than cartilage and synovial fluid). This led them to suggest that cartilage degeneration may be caused by alterations in the mechanical properties of the underlying bone. Therefore an increase in bone mineral density might affect the progress of the osteoarthritis. Thus it is of more than academic importance to determine whether bone mineral density is increased in the region adjacent to all osteoarthritic joints, and if so by what mechanism.

In summary, caution is needed in interpreting spinal bone mineral density in patients with osteophytosis—indeed, it is possible that coexisting osteoporosis could be missed in such patients. There is no convincing evidence that patients with osteoarthritis have a generalised increase in bone mineral density but our data suggest that bone mineral density may be increased in the region of an osteoarthritic joint. This finding warrants further investigation.

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