Inverse Relationship of Interface Between Osteoporosis and Osteoarthritis

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ABSTRACT. The inverse relationship between osteoporosis and osteoarthritis (OA) was first noticed 20 years ago. The subject, however, remains controversial. Recent observations on bone mineral density (BMD) in large epidemiological population surveys confirmed that cases with generalized OA had significantly increased bone mass at the axial and peripheral skeleton compared to age and sex matched controls corrected for body weight. Because OA cases have significantly increased bone width, BMD at the forearm is not significantly increased in some studies. The association of moderate to severe OA with increased appendicular and axial BMD is comparable to that of other determinants of bone mass, including 10 years of age and 10 kg of body weight, and thus may confer protection against fracture, especially of hip fracture. (*J Rheumatol 1997;24:795–8*)

Key Indexing Terms:
OSTEOPOROSIS OSTEOARTHRITIS BONE MINERAL DENSITY

Although osteoporosis and osteoarthritis (OA) are both common conditions with high prevalence in the older age group, they are not purely due to simple aging and affect anthropometrically distinct populations. Typical patients with OA tend to be mesomorph, whereas the patients with type I and type II osteoporosis tend to be ectomorph[1].

The most impressive clinical observations are the general absence of OA in the head of the femur in cases with femoral neck fracture and the rarity of atraumatic hip or spine fractures in generalized OA cases[2-6].

In Figure 1, radiographs of a case with hip fracture show the joint space is well preserved and a case with arthritis of the hip in which the bone is well preserved while the cartilage is gone.

This inverse relationship between osteoporosis and OA has been contested[7]. It is true that in clinical practice a combination of osteoporosis and OA is coincidentally encountered, in particular in the very elderly. In our experience, if subjects with OA develop osteoporotic fracture, they do so at a later age, suggesting that OA or a related factor (obesity) might have a protective effect on the progression of osteoporosis[8]. Osteoporosis as well as OA can be secondary to other causes, such as corticosteroid treatment, trauma, dysplasia, sports injuries, etc. The inverse relationship between osteoporosis and OA is therefore seen in cases of primary osteoporosis and generalized OA.

The interface between osteoporosis and OA from a diagnostic and outcome point of view is of particular importance in the evaluation of vertebral deformity. In both conditions, OA and osteoporosis, thoracic kyphosis is a prominent feature. The tendency to include so called asymptomatic vertebral osteoporosis (wedging), measured by vertebral radiographic morphometry in prevalence studies of osteoporosis, may lead to overdiagnosis if the cases of thoracic OA are included. In OA of the spine, wedging is not a consequence of fracture but of remodeling of the shape to match the lumbar lordosis[9].

Figure 2 illustrates typical roentgenograms of an osteoarthritic and an osteoporotic case.

Because of the advent of precise and accurate measurement of bone mass and density, a number of large epidemiological studies have been published, confirming the observations made 20 years ago by Foss and Byers[10] and our group[11-13]. In a recent literature survey of the inverse relationship of OA/osteoporosis and bone mineral density (BMD), we traced 36 publications from 16 countries, covering 37,774 subjects, 11,137 OA cases and 26,637 controls[14]. In 28 studies, a significant increase in bone mass or BMD was found in the OA cases compared to age and sex matched controls, and in most studies a correction for anthropometric characteristics, in particular body weight, did not change the results.

Table 1 shows the period of publications and the number of publications with positive or negative findings concerning the inverse relationship of OA/osteoporosis.

Table 2 summarizes the positive and negative results concerning bone mass in OA compared to matched controls according to a technique used to evaluate BMD (details, see Reference 14).

Increased BMD in OA has been found in the Chingford Study (UK)[15], the Framingham Study (USA)[16], the Rotterdam Study (The Netherlands)[17], the Montpellier Epidemiology Osteoporosis Study (France)[18], the San Francisco Osteoporosis Study (USA)[19], and the Dubbo Osteoporosis Epidemiology Study (Australia)[20]. Increase in BMD was found not only in the spine but also at sites where there is no influence of osteophytes or subchondral sclerosis

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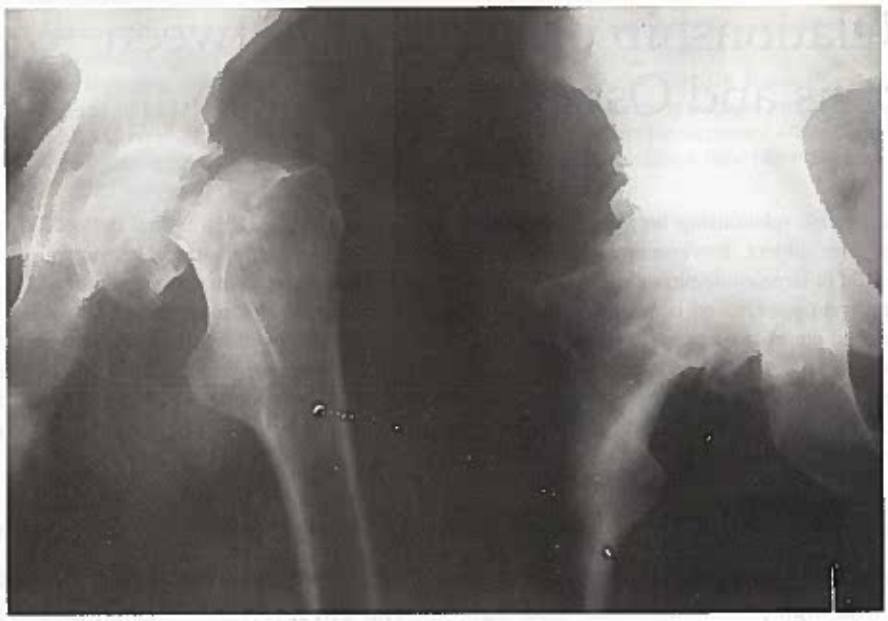

Figure 1. Radiographs of a case with hip fracture in which joint space is well preserved, and a case of arthritis of the hip in which bone is well preserved but the cartilage is gone.

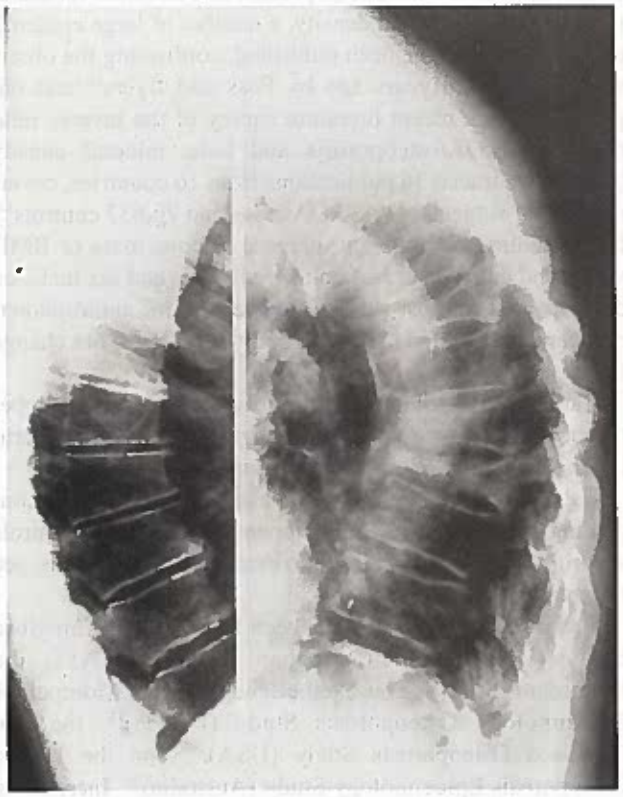

Figure 2. Typical roentgenograms of a case of osteoarthritis (L) and of osteoporosis (R).

Table 1. Period and number of publications with positive or negative findings concerning the inverse relationship of osteoarthritis/osteoporosis.

	+	−	±
1970–1979	5	3	
1980–1989	2	2	1
1990–	20	3	1
Total 37	27	8	2

Table 2. Summary of the positive and negative results concerning bone mass in osteoarthritis compared to matched controls according to technique to evaluate bone mineral density.

Technique	Increased	No Difference	Decreased
Radiogrammetry MC	5	5	
SPA			
Radius trabecular	5		
Radius cortical	5	5	
Femur shaft		1	
Calcaneus	1		
pQCT			
Radius trabecular	1		
Radius cortical		1	
Singh index	2		
NAA		1	
Iliac crest histomorphometry	3		1
QCT L1	2		
DXA or DPA			
Spine	14		
Femur	12		
Total body	3		
Total	53	13	1

MC: metacarpal; NAA: neutron activation analysis; SPA: single photon absorptiometry; DPA: dual photon absorptiometry; DXA: dual energy X-ray absorptiometry; QCT: quantitative computed tomography; pQCT: peripheral quantitative computed tomography.

(e.g., the femoral neck). In the Baltimore Study (USA) of the aged, no consistent relationship was found at the forearm, and metacarpal BMD in cases with knee OA[21]. This can be explained by the fact that bone width plays a more important role in the upper extremities than at other sites. OA cases have a greater bone width and therefore a lower BMD. When the bone mass is expressed as absolute bone mass, then male OA cases have also more bone than those without OA[22].

Figure 3 illustrates BMD at the spine and femoral neck in the Chingford Study (UK)[15] according to site of OA. The increase in BMD is higher in the spine, as expected, because of osteophytes, but significant increases are also observed at the femoral neck.

The association of moderate to severe OA with increased appendicular and axial BMD is comparable to that of other important determinants of bone mass, including 10 years of age and 10 kg of body weight, and thus may confer protection against fracture, especially hip fracture[19].

Generalized OA could be a good negative indicator for selecting patients at risk for osteoporosis. The generalized increase in apparent and real BMD in OA indicates that this disease might initially be a subchondral bone disease rather than a cartilage disorder[23].

In studies of greater depth, we found bone mass in OA cases increased; we also found that qualitative bone changes occur in OA with increased pure BMD and increased contents of such anabolic growth factors as insulin growth factor (IGF-I), IGF-II, transforming growth factor beta, and osteocalcin, indicating better repair mechanisms[24].

Furthermore, the above observations may also provide information about pathophysiological mechanisms underlying diseases such as OA and osteoporosis. In particular for OA, subchondral bone alterations may change because of increased stiffness, shock absorption, with negative consequences for the cartilage.

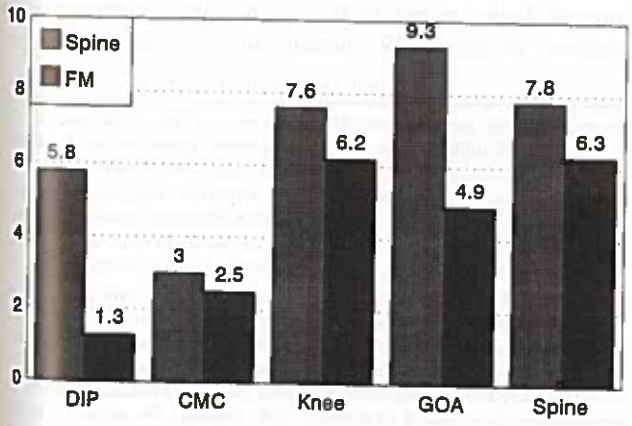

Figure 3. Bone mineral density at the spine and femoral neck (FM) according to the site of OA (Chingford Study[15]). Subjects are women aged 40–65 years, adjusted for age and BMI.

Thus, the inverse relationship between OA and osteoporosis might influence outcome measurements in OA and osteoporosis and should be studied further. Will drugs that increase BMD induce OA, or inversely, can drugs that affect bone improve the outcome of OA?

The OA and osteoporosis studies mentioned here are based on Caucasian populations. There is a need to study BMD and fracture occurrence in OA in Asian and African populations.

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