Effect of spinal osteophytosis on bone mineral density measurements in vertebral osteoporosis

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Bone mineral density measurements are used increasingly to assess the future risk of fracture and to monitor the response to treatment. The lumbar region, which is usually used for these measurements (L1-L4), is the most reproducible. It is also commonly affected by degenerative spinal disease, including osteophytosis, the prevalence of which increases dramatically with age.[1] Other small studies on whether lumbar bone mineral density measurements are affected by osteophytes have been contradictory.[2,4] We have investigated to what extent varying degrees of spinal osteophytosis can affect bone mineral density measurements in the common clinical setting of women presenting with vertebral fractures.

Subjects, methods, and results

Ninety three postmenopausal women (aged 46-93) who presented to the osteoporosis clinic with at least one vertebral fracture were studied. We defined vertebral fracture as a 20% or greater reduction in vertebral height in lateral spine radiographs. Dual *x* ray absorptiometry (quantitative digital radiography 1000/whole body; QDR 1000/W) was used to measure lumbar spine bone density (L1-L4) and femoral neck bone density. The lateral spine radiographs were graded independently for the severity of osteophytosis by the method of Orwoll *et al* (OA0=none, OA1=mild, OA2=moderate, OA3=severe).[2] The presence or absence of vascular calcification in the aorta was also noted. The groups were compared by analysis of covariance (SAS software).

The mean lumbar spine bone density measurements adjusted for age and weight in all three groups with osteophytosis (OA1-OA3) were significantly higher than in the group with no osteophytosis (OA0; $p < 0.001$) (table). The most severe osteophytosis group (OA3) had an adjusted mean lumbar spine bone density 32·1% higher (95% confidence interval 23·5 to 40·7), and densities in the OA1 and OA2 groups were increased by smaller amounts—21·1% (13·0 to 29·2) and 20·2% (12·7 to 27·7) respectively. Overall the presence of even mild osteophytosis produced on average a 24·0% (17·7 to 30·3) increase in lumbar spine bone density.

The adjusted mean femoral neck bone density was higher in the osteophytosis groups (OA1-OA3) compared with the no osteophytosis group (OA0). However, the increases, ranging from a non-significant 1·8% (95% confidence interval −3·6 to 7·2) in the OA1 group to 11·1% (5·3 to 16·9) in the OA3 group, were smaller than those seen in the lumbar spine. When the osteophyte groups were combined (OA1+OA2+OA3) the resulting adjusted mean femoral neck bone density was 8·2% higher (4·1 to 12·3) than in the no osteophytosis (OA0) group ($p=0.021$).

The relative contribution of osteophytes to the variance in lumbar spine bone density was assessed by multiple regression. Osteophytes had the greatest effect on bone density, explaining 27% of the variance (r^2), whereas adding age and weight to the model explained only a further 13%, producing a total r^2 of 0·40.

Forty subjects (43%) had evidence of vascular calcification. The mean lumbar spine bone density measurements adjusted for age and osteophytosis grade for the vascular calcification and no calcification groups were 0·772 g/cm^2 and 0·812 g/cm^2 respectively. There was no significant difference in adjusted mean lumbar spine bone density between the two groups ($p=0.24$).

Comment

These data show that in postmenopausal women with fractures even mild osteophytosis can lead to falsely increased lumbar spine bone mineral density measurements. As osteophytosis is common in older women the presence of osteophytes is likely to cause problems in interpreting lumbar spine bone density results. Although cross sectional, our data also suggest caution in interpreting changes in lumbar spine bone density in prospective studies or trials unless changes in osteophytes are also noted.

Our results suggest that spinal osteophytosis does not affect femoral neck bone density to the same extent as lumbar spine bone density. Although in elderly people neck scans are often considered more difficult owing to poor positioning and poor patient mobility, these data imply that in the presence of spinal osteophytosis the femoral neck is a more reliable site for estimating bone mineral density. Our finding of a small (8·2%) increase in femoral neck bone density in women with spinal osteophytosis is consistent with the hypothesis of an inverse relation between osteoarthritis and osteoporosis.[5]

In conclusion our study shows that in postmenopausal women with fractures the common finding of mild spinal osteophytosis can lead to misleadingly high lumbar spine bone density readings and may mask the degree of underlying spinal osteoporosis. Our data suggest that, in older patients with vertebral fracture, unless the spine looks clear of osteophytes radiologically the femoral neck should be used to assess bone mineral density.

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Description of lumbar spine bone density and femoral neck bone density in subjects with vertebral fracture by osteophyte status

Osteophyte status	Mean age (years) (SE)	Mean weight (kg) (SE)	Mean lumbar spine bone density (g/cm^2) (SE)	Adjusted mean lumbar spine bone density* (g/cm^2) (SE)	% Difference in lumbar spine bone density* from OA0 (95% confidence interval)	Mean femoral spine bone density (g/cm^2) (SE)	Adjusted mean femoral neck bone density* (g/cm^2) (SE)	% Difference in femoral neck bone density* from OA0 (95% confidence interval)
OA0 (n=34)	66·6 (1·6)	58·2 (2·1)	0·638 (0·020)	0·634 (0·025)		0·559 (0·018)	0·560 (0·017)	
OA1 (n=18)	64·7 (2·5)	60·1 (2·8)	0·819 (0·040)	0·804 (0·033)	21·1 (13·0 to 29·2)	0·589 (0·028)	0·570 (0·022)	1·8 (−3·6 to 7·2)
OA2 (n=25)	69·6 (1·9)	61·6 (2·1)	0·780 (0·027)	0·794 (0·029)	20·2 (12·7 to 27·7)	0·617 (0·023)	0·620 (0·019)	9·7 (4·7 to 14·7)
OA3 (n=16)	70·3 (2·1)	63·4 (3·8)	0·934 (0·057)	0·934 (0·036)	32·1 (23·5 to 40·7)	0·617 (0·035)	0·630 (0·024)	11·1 (5·3 to 16·9)
Combined group (OA1+OA2+OA3) (n=59)	68·2 (1·3)	61·6 (1·6)	0·833 (0·024)	0·834 (0·020)	24·0 (17·7 to 30·3)	0·608 (0·016)	0·610 (0·012)	8·2 (4·1 to 12·3)

*Adjusted for age and weight.

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1 Lawrence JS. Disc degeneration: its frequency and relationship to symptoms. *Ann Rheum Dis* 1969;**28**:121-38.
2 Orwoll ES, Oviatt SK, Mann T. The impact of osteophytic and vascular calcifications on vertebral mineral density measurements in men. *J Clin Endocrinol Metab* 1990;**70**:1202-7.
3 Reid IR, Evans MC, Ames R, Wattie DJ. The influence of osteophytes and aortic calcification on spinal mineral density in postmenopausal women. *J Clin Endocrinol Metab* 1992;**72**:1372-4.
4 Dawson-Hughes B, Dallal GE. Effect of radiographic abnormalities on rate of bone loss from the spine. *Calcif Tissue Int* 1990;**46**:280-1.
5 Dequeker J. The relationship between osteoporosis and osteoarthritis. *Clin Rheum Dis* 1985;**11**:271-95.

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Late cardiac manifestation of infection with *Borrelia burgdorferi* (Lyme disease)

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The whole range of cardiac complications of Lyme disease, which is caused by the tick borne spirochaete *Borrelia burgdorferi*, is not yet clear. We report a case of a very late debut of Lyme carditis, which has not been reported before.

Case report

A female patient, born in 1923, lived in a rural area of western Norway, where Lyme disease is endemic. In the summer of 1972 she developed a rash typical of erythema migrans on her right flank. She did not recall any tick bite. The lesion disappeared after some weeks. Thereafter she developed a transient burning pain on the right side of the body and a transient right sided Bell's palsy followed by a contralateral Bell's palsy. Afterwards she constantly had migratory myalgias, arthralgias, and malaise consistent with chronic borrelia infection. She was not given antibacterial treatment.

In the spring of 1990 she developed a persistent stabbing pain centrally in the chest, burning sensations on the right side of the body, an increasing exertional dyspnoea, feelings of cold, and a growing malaise. On admission to hospital early in June 1990, she had dyspnoea with moderate exertion. Clinical examination revealed a sequela on the left side after Bell's palsy. Her erythrocyte sedimentation rate was 80 mm/h. Serum protein electrophoresis showed a transient acute phase response. Tests for cryoglobulins, circulatory immune complexes, syphilis, leptospirosis, cytomegalovirus, coxsackievirus B, autoantibodies, and antibodies against cardiolipin were all negative. Serological analysis gave no indication of recent infection with streptococcus or Epstein-Barr virus. Haematological and biochemical values were either normal or without actual significance.

Electrocardiograms showed initially fluctuating diffuse T wave inversions. Radionuclide ventriculography revealed a left ventricular ejection fraction of 55%. Echocardiography showed little pericardial effusion and increased left ventricular dimensions. A chest roentgenogram showed considerable cardiomegaly and slightly distended pulmonary veins whereas previous chest roentgenograms, the latest taken in 1987, had been normal.

The table shows the results of different assays for borrelia antibodies in cerebrospinal fluid and serum samples. The specific antibody ratio index was calculated from tests for borrelia antibodies by indirect haemagglutination.[1] This index reflects the relative specific antibody load in cerebrospinal fluid compared to that of serum. The calculated index of 42·5 was high,

Results of tests for borrelia antibodies on cerebrospinal fluid and serum samples taken from a patient at different times

	Time when sample collected			
	June 1990		October 1990 serum	December 1991 serum
	Spinal fluid	Serum		
Indirect haemagglutination (titre)*	40	400	400	<200
Specific antibody ratio index	42·5			
Enzyme immunoassays (%):†				
Borrelia flagella‡				
IgG		462	669	475
IgM		<100	<100	<100
Sonicated *Borrelia*§				
IgG			<100	
IgM			<100	
Total immunoglobulin‖			279	
Western blotting¶				
IgG			Positive**	
IgM			Negative	

*Lymag, Diagast Laboratoires, Lille, France.
†Results related to cut off (100%) so that values > 100 are positive, < 100 are negative.
‡Lyme borreliosis ELISA kit, Dako, Glostrup, Denmark.
§Test based on sonicated whole cells of *B burgdorferi*.
‖Human Lyme EIA diagnostic test, Cambridge BioScience, Worcester, USA.
¶Cambridge BioScience, Worcester, USA.
**Borrelia protein antigens of 41, 21, 56, and 75 kDa showed reactivity.

indicating intrathecal production of borrelia antibodies and therefore past or present neuroborreliosis.

From the middle of June the patient was treated intravenously with 3 g benzylpenicillin three times daily for three weeks. During treatment her symptoms rapidly faded, and she felt well for the first time since her erythema migrans in 1972. Her erythrocyte sedimentation rate had fallen to 12 mm/h in August 1990, and her echocardiogram and chest roentgenogram were unremarkable. She was still well two years after the antibiotic treatment.

Comment

The typical medical history, positive assays for borrelia antibodies, and quick and lasting response to adequate antibiotic treatment form the basis for the diagnosis of Lyme carditis in the patient, which appeared 17-18 years after the primary infection. The patient received no treatment except benzylpenicillin which could have contributed to the remission. The medical history and laboratory findings gave no indication of any other disease that could explain the cardiac manifestations. A recent retrospective study of patients with dilating cardiomyopathy suggests that this condition could be a late manifestation of Lyme borreliosis.[2] If this is correct our patient could have been in an early phase of a progressive dilating cardiomyopathy. Antibiotic treatment was effective in this case, which suggests that such treatment at an early stage of an assumed progressive dilating cardiomyopathy may halt or cure the condition.

1 Hofstad H. Serologisk diagnostikk av Borrelia burgdorferi infeksjon. *Tidsskr Nor Lægeforen* 1987;**107**:3038-40.
2 Stanek G, Klein J, Bittner R, Glogar D. Borrelia burgdorferi as an etiologic agent in chronic heart failure? *Scand J Infect Dis* 1991;77:85-7.

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