Calcif Tissue Int (1992) 50:5–6

Calcified Tissue International

Vertebral Fracture or Vertebral Deformity?

Received November 2, 1990, and in revised form December 28, 1990

In our recently completed sodium fluoride (NaF) clinical trial [1] we observed that only about 25% of vertebral deformities, documented by change in vertebral morphometry on radiographs, were associated with corresponding areas of increased isotope uptake on radionuclide bone scans. This was an unexpected finding. In the initial trial design, we had anticipated that the scan would be more sensitive than radiographs and would detect "fractures" that were not documented radiographically. The study format included annual radiographs and semiannual scans in the hope that a higher vertebral fracture rate by scintigraphy would increase the power of the study. The failure of the bone scan in this regard, which was independent of treatment assignment (placebo or NaF), has several plausible explanations, the most compelling of which is that changes in vertebral morphometry in spinal osteoporosis can occur by mechanisms other than fracture. If correct, that hypothesis would have major impact on epidemiologic and clinical studies of spinal osteoporosis.

Most fractures in the appendicular skeleton are all-or-none events and the expected outcome is that the affected bone will heal completely with full restoration of original shape and function. Every attempt is made to avoid permanent postfracture deformity of a long bone. Postfracture deformity is easy to recognize clinically and radiographically when the involved bone is anywhere but in the spine, and there is rarely any doubt as to the cause of the deformity.

In sharp contrast, fractures of the vertebral bodies are graded in their severity, and permanent deformity is the expected outcome. It is accepted that certain vertebral deformities develop by mechanisms other than fracture (e.g., Scheuermann's juvenile kyphosis). Yet, most clinicians and investigators in this field operate under the assumption that a *change* in vertebral morphology *must* represent fracture occurrence. The findings of our fluoride trial raise the possibility that not every new vertebral deformity is the result of a fracture.

Harrison et al. [2] reported recently that isolated midthoracic vertebral deformity was not associated with significant osteopenia and cannot be regarded as diagnostic of osteoporotic fracture, confirming an earlier observation of Nordin et al. [3]. This distinction between fracture and deformity is crucial for the proper conduct of cross-sectional epidemiologic studies in spinal osteoporosis. This may also be the case for longitudinal studies including therapeutic trials in this disease, and affects the choice of methods to be employed.

The prevalence of vertebral "fractures" in a population actually represents the prevalence of vertebral deformity which includes some unknown proportion of true fractures. Unless an investigator can confirm by history and review of medical records that a particular episode of acute back pain was temporally associated with radiographic detection of a newly deformed vertebra or a bone scan abnormality close to the site of the pain, it cannot be stated with confidence that the deformity resulted from a fracture. Using this definition of vertebral fracture should minimize potential differences between studies attempting to define the prevalence of vertebral fractures in a given group. There will still be differences in the extent to which a fractured vertebra is deformed but these differences would become largely irrelevant to fracture epidemiology studies.

We and others [4–7] have stated that many vertebral fractures are asymptomatic, and it could be argued that the above rigid criterion for vertebral fracture documentation would underestimate the true prevalence of spinal osteoporotic fractures. We would now counter that it is an unproven assumption that many fractures are asymptomatic, and that the onus is now on those of us who have made such statements to justify them. As many vertebral deformities are not clinically significant events, it is possible that some of these represent gradual shape changes that result from internal changes in bone architecture with bone loss. In fact, the data of Harrison et al. [2] points out that abnormal vertebral morphometry resembling midthoracic vertebral fractures can be seen even in the absence of osteopenia.

Why is the distinction between vertebral fracture and vertebral deformity important for epidemiologic studies? In prevalence studies addressing the morbidity from spinal osteoporosis, the distinction may in fact not be relevant. The prevalence of true vertebral fractures defined as an acute symptomatic event is probably quite small and associated with limited morbidity (i.e., acute pain of perhaps 4–6 weeks duration) and probably zero mortality. The prevalence of vertebral deformity is much greater and includes vertebral fracture. It is the consequences of the deformities, rather than the fractures per se, that lead to substantial long-term morbidity affecting quality of life [7]. This morbidity includes progressive thoracic kyphosis, loss of stature, and chronic pain, or may relate to the altered body image of patients with progressive painless spinal deformity. Often it is a combination of both.

The situation is different with regard to the incidence of vertebral deformities, some of which may be identified as true fractures or events when accompanied by an episode of acute pain, as documented in the history, or by an area of increased isotope uptake on a bone scan. The data from our clinical trial of NaF therapy in postmenopausal osteoporosis, and from other clinical trials, that new vertebral deformities are not always accompanied by acute pain, suggest that some of these deformities represent gradual shape changes, or plastic deformities, rather than clinically significant or true fractures. There is a clear need for prospective studies in osteoporosis directly addressing the relationship between episodes of acute back pain and the concurrent documentation of new vertebral deformities or fractures.

If the long-term morbidity from spinal osteoporosis results from the permanent vertebral deformities, then the use

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of surrogate measures of deformity in epidemiologic studies or clinical trials may be appropriate and perhaps even more efficient than radiographic approaches. One such surrogate measure would be standing height or stature, which can be measured with a short-term precision of ±0.15% [8]. Height loss with age may occur for reasons other than change in vertebral morphology such as disk deterioration, weakening of muscle groups, or postural change [9, 10], but these were taken into account by Cline et al. [11] who reported that healthy women of mean age 65 lost height at an average rate of 0.12 cm/year. In our fluoride trial where stature was measured every 3 months, the average annual height loss in those women in whom no changes in vertebral morphometry were detected was also 0.12 cm/year [8]. In contrast, the average loss of stature in those in whom new vertebral deformities were detected was 0.59 cm/year ($P < 0.05$). In a cross-sectional study in which serial measurements of stature in individuals are not available, height loss can be calculated from other anthropometric estimates of peak adult stature (e.g., knee height [12]) compared with current stature.

Physicians who regularly care for patients with spinal osteoporosis recognize that progressive height loss and spinal deformity accounts for most of the morbidity in this disease and that most patients will complain of chronic back pain. Though patients may from time to time report acute exacerbation of back pain, it is most likely that this results from acute fracture in only a minority of the cases. A well-structured physical therapy/rehabilitation program focused on the activities of daily living with instructions on the correct methods of bending, lifting, reaching, etc. is unlikely to have any effect on spinal bone mass or quality. It is likely, however, to reduce the occurrence of new vertebral fractures. A therapeutic approach focused only on stabilizing or improving spinal bone mass or quality may well halt the progression of vertebral deformities, but fractures resulting from such activities as heavy lifting may continue to occur.

We are advocating that the distinction between vertebral fracture (that appears to always result in deformity) and vertebral deformity (that may not always result from a fracture) is necessary before meaningful approaches to spinal osteoporosis can be accomplished. If true fractures can be shown to result only from an acute mechanical overload, then major emphasis must be placed on ergonomic factors in the prevention of these fractures. This is of course not unique to spinal osteoporosis but mirrors the approach that most are advocating with respect to the even greater community health problem of proximal femur fractures. One thing is clear, however: In order to determine the etiology and significance of vertebral fractures and vertebral deformities, and to plan approaches to prophylaxis or therapy, we must use appropriate, uniform methods to document these phenomena.

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References

1. Kleerekoper M, Peterson E, Nelson DA, Phillips E, Schork MA, Parfitt AM, Tilley BC (1991) A randomized trial of sodium fluoride as a treatment for postmenopausal osteoporosis. Osteoporosis Int, in press
2. Harrison JE, Patt N, Muller C, Bayley TA, Budden FH, Josse RG, Murray TM, Sturtridge WC, Strauss A, Goodwin S (1990) Bone mineral mass associated with postmenopausal vertebral deformities. Bone Miner 10:243–251
3. Nordin BEC, Crilly RG, Smith DA (1984) Osteoporosis. In: Nordin BEC (ed) Metabolic bone and stone disease. Churchill Livingstone, New York
4. Kleerekoper M, Parfitt AM, Ellis BI (1984) Measurement of vertebral fracture rates in osteoporosis. In: Christiansen C, Arnaud CD, Nordin BEC, Parfitt AM, Peck WA, Riggs BL (eds) Osteoporosis. Aalberg Stiftsbogtrykkeri, Copenhagen
5. Kleerekoper M, Avioli LV (1990) Evaluation and treatment of postmenopausal osteoporosis. In: Favus MJ (ed) Primer on the metabolic bone diseases and disorders of mineral metabolism. American Society for Bone and Mineral Research, Kelseyville, CA
6. Cooper C (1989) Osteoporosis—an epidemiological perspective: a review. J Roy Soc Med 82:753–757
7. Leidig G, Minne HW, Sauer P, Wuster C, Wuster J, Lojen M, Raue F, Ziegler R (1990) A study of complaints and their relation to vertebral destruction in patients with osteoporosis. Bone Miner 8:217–229
8. Kleerekoper M, Nelson DA, Peterson E, Tilley BC (1991) Outcome variables in osteoporosis trials. Bone, in press
9. Trotter M, Gleser G (1951) The effect of aging on stature. Am J Phys Anthropol 9:311–324
10. Rogers SL (1982) The aging skeleton. Charles C. Thomas, Springfield, IL
11. Cline MG, Meredith KE, Boyer JT, Burrows B (1989) Decline of height with age in adults in a general population sample: estimating maximum height and distinguishing birth cohort effects from actual loss of stature with aging. Hum Biol 61:415–425
12. Frisancho AR (1990) Anthropometric standards for the assessment of growth and nutritional status. The University of Michigan Press, Ann Arbor, MI