Prevention of Osteoporosis: The Role of Primary Physicians

Nelson B. Watts, MD
Atlanta, Georgia

There has been a rapid escalation of knowledge about osteoporosis over the past several years. We now have a clear understanding of some of the general pathophysiological mechanisms, tools such as bone mineral density measurements for assessment, and effective modalities for prevention and treatment.[1,2] Therefore, it is appropriate to consider what primary physicians should do to identify patients who are at risk for developing osteoporosis and to institute appropriate preventive measures.

In this issue of the Journal, Bourguet, Hamrick, and Gilchrist[3] report on their record review of patients from a community-based, university-affiliated family medicine program. They conclude that documentation of risk factors for osteoporosis is infrequent and that modification of these risk factors is rarely attempted. Another recent study indicates that risk-factor data obtained by patient interview is quite often questionable and incomplete.[4] It is my view that the risk-factor approach to osteoporosis is unnecessarily complex and cumbersome; the situation is much simpler than it may initially appear.

I reserve the term *osteoporosis* for patients who have had fractures with little or no trauma. I use *osteopenia* to describe individuals with low bone mass (more than two standard deviations below the mean level of peak bone mass) who have not had fractures. Low bone mass is essentially a prerequisite for osteoporosis, but it is not synonymous with osteoporosis; many patients with osteopenia will never have fractures. Low bone mass is clearly the most important risk factor for osteoporosis, and it is really osteopenia that the physician seeks when assessing other risk factors. The issue is not so much the patient's risk for osteoporosis, but rather the patient's risk for developing or already having osteopenia.

No test or risk factor, alone or in combination, will accurately forecast which patients will or will not experience osteoporotic fractures. A number of clinical risk factors for osteopenia can be identified from history and physical examination. Some of these risk factors are potentially remediable, but some are not. Nonremediable risk factors include ethnic origin (Asian or white), advancing age, family history of osteoporosis, small frame, and low body weight.

Consider the potentially remediable risk factors for osteopenia. Cigarette smoking, heavy alcohol use, and sedentary lifestyle are risk factors for multiple diseases, not just osteopenia, and should be identified and targeted for correction regardless of whether the individual is at risk for osteopenia. Good medical practice requires the use of all medications judiciously and in the lowest effective dosages; this includes medications that predispose to bone loss such as glucocorticoids, anticonvulsants, thyroid hormones, furosemide, and tetracycline. With the exception of menopause and a low calcium diet, the other "risk factors for osteopenia" are really risk factors for poor health and should be dealt with uniformly.

Management of menopause (specifically, whether to prescribe estrogen therapy, with or without progesterone) should be addressed in every woman, not just those at risk for osteopenia. The decision to use estrogen should be individualized and based on multiple factors, including the presence or absence of symptoms and the potential benefits of estrogen therapy on coronary disease, stroke, and osteopenia. The drawbacks and risks of estrogen treatment should also be taken into consideration, including withdrawal or breakthrough bleeding, endometrial carcinoma, thrombotic disorders, aggravation of fibrocystic breast disease, and others. Finally, the patient's other risk factors for osteopenia should enter into the decision to prescribe estrogen. Bone densitometry may be helpful in resolving the matter of estrogen therapy if other considerations are equivocal.

The only potentially remedial risk factor for osteopenia that remains to be addressed is low calcium intake.

Submitted December 18, 1990.

From Emory University School of Medicine, Atlanta, Georgia. Requests for reprints should be addressed to Nelson B. Watts, MD, Section of Internal Medicine, The Emory Clinic, 1365 Clifton Rd, NE, Atlanta, GA 30322.

ISSN 0094-3509

This can be handled by having information readily available regarding the recommended level of calcium intake, dietary sources of calcium, and calcium supplements, and by encouraging all patients to optimize their calcium intake.

Having dealt with these remediable risk factors for osteopenia, we must know if clinical assessment is adequate to determine which patients actually have osteopenia, since it is patients with osteopenia who would be candidates for therapy to increase bone mass and reduce their future risk of osteoporotic fractures. Evidence suggests that clinical assessment is both insensitive and nonspecific for identifying these patients. In a study of community volunteers, all of whom were perimenopausal women, Slemenda and colleagues[5] performed bone density measurements and then examined various combinations of clinical risk factors to determine whether patients with osteopenia could be identified. No combination of risk factors permitted identification of more than 70% of women with low bone mass. The authors conclude that bone density measurements, not clinical risk factors, should be used to make decisions about treatment, either with estrogen or with other medication. Other investigators have reached the same conclusion.[6,7]

Who should have bone mass measurements? It has previously been stated that routine bone mineral density screening of perimenopausal women should not be done.[8–10] This recommendation was based primarily on the lack of specific interventions. Now that effective measures are available for both prevention and treatment, it appears that it may be cost-effective to perform bone mass determinations in all perimenopausal women to identify those in whom specific measures should be taken. Some of the same authorities who previously recommended against bone mineral density screening at menopause[11] are now in favor of it.[12] I believe that baseline bone mass measurements will soon become a routine part of the care of all postmenopausal women. At present, it seems desirable to perform these measurements for women who are particularly concerned about their own risk, and for women who are at high risk based on clinical assessment. This brings us full circle back to clinical risk factors, but now the risk factors are used to decide whether bone densitometry should be done, rather than to determine treatment. By taking a selective approach to ordering bone densitometry studies, we will be missing at least 30% of women with osteopenia, but it may be argued that these asymptomatic and unconcerned women are less likely to comply with interventions involving lifestyle changes and medication.

By what technique and at what anatomic site should bone density be measured? Vertebral fractures due to loss of trabecular bone are the most common complication of osteoporosis, so it is desirable to measure trabecular bone in the spine to identify patients at risk for these fractures. In theory and in rigorously controlled research studies, quantitative computed tomography (QCT) seems best suited for this purpose,[13] but QCT involves fairly high radiation exposure and is too imprecise to be useful for sequential studies in the same patient over time. Anterior-posterior spine measurements with dual energy techniques[14] (dual-photon absorptiometry [DPA] and dual-energy x-ray absorptiometry [DEXA]) are not as sensitive as QCT for the detection of osteopenia because they measure the entire vertebral body (cortical bone and trabecular bone) plus the posterior elements. These techniques are preferred for sequential follow-up studies in the same patient, however, because they are more precise than QCT and involve less radiation exposure.[13] Lateral spine measurements using DEXA should be generally available soon, and appear to provide satisfactory diagnostic sensitivity for detection of osteopenia, as well as adequate precision for follow-up studies.[15] Most physicians will have access only to a single method for bone densitometry, but until there is consensus regarding the optimal technique, almost any bone density measurement using almost any technique will provide potentially useful information.

Management of patients with osteopenia requires identification and modification of risk factors. Estrogen is the only agent approved by the United States Food and Drug Administration (FDA) for prevention of osteoporosis, and should be strongly considered for all women with documented osteopenia who are perimenopausal or 10 years or less postmenopausal. Bisphosphonates and calcitonin hold promise for prevention, but the effectiveness of these agents for this purpose has not yet been proven in clinical trials. Although only one drug, salmon calcitonin, now has FDA approval for treatment of patients with established osteoporosis, intermittent cyclical etidronate therapy[16,17] is under consideration by the FDA and is currently approved in some European countries. Parathyroid hormone, sodium fluoride, and vitamin D metabolites are still experimental, but may also prove useful. There are sufficient therapeutic options so that every person with osteopenia or osteoporosis should be offered something to prevent or reverse bone loss.

Osteoporotic fractures are preceded by decades of gradually progressive bone loss, but not by any other warning. Even with effective modification of clinical risk factors, some women will continue to lose bone and develop osteoporosis. Truly effective prevention of osteoporosis will be possible only with systematic application of bone densitometry to identify women who have significant osteopenia without apparent clinical risk factors.

References

1. Watts NB. Osteoporosis. Am Fam Physician 1988; 38:193–207.
2. Watts NB, Chesnut CH III, Heaney RP, Miller PD. Osteoporosis: its prevention, diagnosis, and treatment. Med Times 1990; 118: 39–45.
3. Bourguet CC, Hamrick GA, Gilchrist VJ. The prevalence of osteoporosis risk factors and probability of intervention in a family practice setting. J Fam Pract 1991; 32:265–271.
4. Beard CM, Melton LJ III, Cedel SL, et al. Ascertainment of risk factors for osteoporosis: comparison of interview data with medical record review. J Bone Mineral Res 1990; 5:691–8.
5. Slemenda CW, Hui SL, Longcope C, et al. Predictors of bone mass in perimenopausal women: a prospective study of clinical data using photon absorptiometry. Ann Intern Med 1990; 112: 96–101.
6. Davis MR. Screening for postmenopausal osteoporosis. Am J Obstet Gynecol 1987; 156:1–5.
7. van Hemert AM, Vandenbroucke JP, Birkenhager JC, Valkenburg HA. Prediction of osteoporotic fractures in the general population by fracture risk scores: a 9-year follow-up among middle-aged women. Am J Epidemiol 1990; 132:123–35.
8. Ott SM. Should women get screening bone mass measurements? Ann Intern Med 1989; 110:267–74.
9. Hall FM, Davis MA, Baran DT. Bone mineral screening for osteoporosis. N Engl J Med 1987; 316:212–4.
10. Melton LJ III, Eddy DM, Johnston CC Jr. Screening for osteoporosis. Ann Intern Med 1990; 112:516–28.
11. Cummings SR, Black D. Should perimenopausal women be screened for osteoporosis? Ann Intern Med 1986; 104:817–23.
12. Cummings SR, Browner WS, Ettinger B. Should prescription of postmenopausal hormone therapy be based on the results of bone densitometry? Ann Intern Med 1990; 113:565–6.
13. Pacifici R, Rupich R, Griffin M, et al. Dual energy radiography versus quantitative computer tomography for the diagnosis of osteoporosis. J Clin Endocrinol Metab 1990; 70:705–10.
14. Wahner HW, Dunn WL, Brown ML, et al. Comparison of dual-energy x-ray absorptiometry and dual photon absorptiometry for bone mineral measurements of the lumbar spine. Mayo Clin Proc 1988; 63:1075–84.
15. Rupich R, Pacifici R, Griffin M, et al. Lateral dual energy radiography: a new method for measuring vertebral bone density: a preliminary study. J Clin Endocrinol Metab 1990; 70:1768–70.
16. Storm T, Thamsborg G, Steiniche T, et al. Effect of intermittent cyclical etidronate therapy on bone mass and fracture rate in women with postmenopausal osteoporosis. N Engl J Med 1990; 322:1265–71.
17. Watts NB, Harris ST, Genant HK, et al. Intermittent cyclical etidronate treatment of postmenopausal osteoporosis. N Engl J Med 1990; 323:73–9.