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Postmenopausal Osteoporosis

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Postmenopausal Osteoporosis

INTRODUCTION

Postmenopausal osteoporosis is a systemic skeletal disease associated with decreased bone mineral density (BMD) and microarchitectural deterioration within bone, which result in increased risk of fracture with minimal trauma in women after menopause. Osteoporosis is closely related to osteopenia, a less severe form of reduction in bone mass below the normal level. Although patients suffering from osteoporosis have an increased risk for developing fragility fractures,¹ this definition of osteoporosis does not require a fracture to be present for diagnosis.

The current definition of osteoporosis relies on non-invasive measurement of BMD by a variety of techniques. These techniques were developed because quantitation of bone strength and microarchitectural deterioration of the skeleton is not possible in a living patient at present without an iliac crest bone biopsy. Future noninvasive technologies may be able to quantify bone strength and microarchitectural deterioration of the skeleton better than current technologies.

It is estimated that 28 million individuals in the United States are affected by low bone density, with about one third of these meeting current World Health Organization (WHO) criteria for osteoporosis and about two thirds meeting criteria for osteopenia.² The average 50-year-old Caucasian woman in the United States has a 40% risk of a clinical fracture in her remaining lifetime years, but this figure increases to 50% if asymptomatic vertebral collapse fractures are included in the risk estimate.³ Clearly, osteoporosis is a public health problem in the United States, but because it tends to be clinically silent until fractures occur, busy clinicians focused on more pressing medical problems may easily overlook it.

The consequences of ignoring osteoporosis until fractures occur often are severe for patients who sustain hip fractures. Twenty percent more patients die in the first year after a hip fracture than would otherwise be expected for age, commonly due to comorbidities such as deep venous thrombosis (DVT) and pulmonary embolism resulting from immobilization associated with the fracture.⁴ Of those who survive a hip fracture, 50% fail to regain full functional capacity, and many patients require extended supervised care.⁵ Patients with wrist, vertebral, or other osteoporotic fractures experience

pain and temporary disability, and those with vertebral fractures may experience subsequent chronic back pain, loss of height, and consequent loss of self-esteem.

This case-based review describes risk factors for postmenopausal osteoporosis and osteoporotic fracture. Attention is given to the diagnostic evaluation of the patient with postmenopausal osteoporosis, including use of bone mineral densitometry and T scores. Finally, nonpharmacologic and pharmacologic approaches to management of postmenopausal osteoporosis are discussed in detail.

CAUSES AND RISK FACTORS

CASE PRESENTATION

Initial Presentation

A 74-year-old Caucasian woman comes to the office of her primary care physician complaining of acute onset of severe low back pain 2 nights ago.

History

The patient claims to have heard and felt at least 1 bone break in her low back while lifting a pot roast out of her oven 2 nights ago. She was seen at her local emergency trauma unit that evening for severe lumbar pain, at which time a new L2 vertebral compression fracture was confirmed on spine films. She was treated with an intramuscular nonsteroidal pain medication and prescribed an oral narcotic medication for home use as needed. A soft foam rubber lumbar support brace also was prescribed. Since then, the patient's low back pain has subsided to a dull ache. She has been on estrogen replacement therapy (ERT) for at least 15 years, without any other antiresorptive therapy. She wonders whether she has osteoporosis, and if so, how this could have developed while taking estrogen replacement. She claims to have drunk at least 2 glasses of milk every day for her entire adult life.

The patient has a 10-year history of mild essential hypertension treated with hydrochlorothiazide (25 mg/day) and a 15-year history of osteoarthritis causing low back pain treated with ibuprofen (200 mg 3 times daily or as needed). She has taken conjugated equine estrogen without complication since undergoing total abdominal hysterectomy and bilateral