Women's Health: Osteoporosis

Introductory Remarks

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Synopsis

The cause of osteoporosis, a condition in which bone mass is decreased to a point where structural failure may occur, is unknown; many factors that contribute to the development of osteoporosis are known.

Bone mass increases until the late twenties, the time when people attain peak bone mass. For a time after peak bone mass is reached, bone loss and formation are approximately equal. Soon after that, probably in the early thirties, an uncoupling of bone synthesis and bone resorption occurs, and a net loss of bone mass begins, a process that can ultimately result in osteoporosis.

Bone loss occurs most rapidly in white females immediately after menopause. The bone most affected is the spongy, trabecular bone of the vertebrae and pelvis and the ends of long bones.

Osteoporosis is classified into two syndromes, which are not distinct but have overlapping features and may have the same pathogenetic mechanism. Type I, or postmenopausal osteoporosis, is associated with estrogen deficiency and is characterized by loss of trabecular bone in the vertebrae and the distal radius (the wrist). Vertebral fractures and wrist fractures (also called Colles' fractures) result. Type II, or senile osteoporosis, is age-related, occurs in men as well as women, and is characterized by fractures of the hip and humerus. There is a loss of cortical as well as trabecular bone.

Estrogen deficiency, increasing age, smoking, high alcohol intake, large amounts of caffeine and protein, and lean body mass favor decreased bone density, whereas estrogen replacement after menopause, adequate dietary calcium, and moderate amounts of weight-bearing exercise tend to favor increased bone density.

Standard X-rays, computed tomography, single and dual photon absorptiometry, and neutron activation analysis are noninvasive techniques used in evaluating osteoporosis.

Calcium, estrogen, and calcitonin are approved drug therapies for osteoporosis.

OSTEOPOROSIS IS A CONDITION in which bone mass is decreased to a point where structural failure may occur. Epidemiologic surveys indicate that 10 percent of women over 50 years of age have bone loss severe enough to result in vertebral, long bone, or hip fracture (*1*). As the population ages and the proportion of women increases, osteoporosis becomes an evermore pressing public health concern. The cost of osteoporosis and its complications is estimated to be as high as $6.1 billion annually (*2*).

The cause of osteoporosis is unknown, but many factors are known that contribute to the development of the condition.

During and after puberty, skeletal growth results in an increase in bone density until the late twenties, when peak bone mass is attained. Peak bone mass is higher in males than females, and this may contribute to the lower prevalance of osteoporosis in men.

Bone is a dynamic tissue, constantly lost and formed in discrete units called remodeling units. The cells responsible for resorption of bone are osteoclasts, and those that synthesize bone matrix protein are osteoblasts. Osteoblastic and osteoclastic activity are coupled, probably by locally synthesized factors such as peptides or prostaglandins, which are in turn influenced by systemic steroid and peptide hormones (*3, 4*). For a time after peak bone mass is reached, bone loss and bone formation are approximately equal. Soon after that, probably in the early thirties, an uncoupling of bone synthesis and bone resorption occurs, and a net loss of bone mass begins, a process that can ultimately result in osteoporosis (*5*).

Bone loss occurs most rapidly in white females immediately after menopause. The bone most affected is the spongy, trabecular bone of the vertebrae and pelvis and the ends of long bones. There are two main types of bone: spongy bone, called cancellous or trabecular bone; and compact, or cortical bone. All bones have an outer shell of compact bone around a central mass of trabecular bone. Trabecular bone consists of slender irregular plates, or trabeculae, of cortical bone that branch and unite to form spaces, giving the bone a spongy appearance. The vertebrae are composed of about 60 percent trabecular bone, the hip about 50 percent trabecular and 50 percent cortical bone, and the midportion of the radius about 95 percent cortical bone.

Osteoporosis is classified into two syndromes that are not distinct but have overlapping features and may have the same pathogenetic mechanism (*6*). Type I, or postmenopausal osteoporosis, is associated with estrogen deficiency such as occurs in postmenopausal women or in young women with amenorrhea. This type of osteoporosis is characterized by loss of trabecular bone in the vertebrae and the distal radius. Vertebral fractures and wrist fractures, also called Colles' fractures, result. Vertebral fracture results in loss of height and deforming of the spine, producing the kyphotic "dowager's hump". Estrogen therapy after menopause slows bone loss in Type I osteoporosis (*7-9*). Type II, or senile osteoporosis, is age-related and tends to occur later in life. This type of osteoporosis occurs in males as well as females, but to a lesser extent in males. There is a loss of cortical and trabecular bone in Type II osteoporosis, which results in fractures of the humerus and femoral neck. There is a major increase in hip fracture in women after age 65 and in men after age 75, although the rate is twice as high in women as in men.

Many factors influence the development of osteoporosis. Estrogen deficiency (*6*), increasing age (*10*), smoking (*11, 12*), high alcohol, caffeine, or protein intake, and lean body mass favor decreased bone density (*13*). Estrogen replacement after menopause (*14*) and moderate amounts of weight-bearing exercise (*15*) tend to maintain bone density. However, when young female atheletes exercise to the point that amenorrhea results, they have lower bone densities than those who still menstruate (*16*).

Dietary calcium plays a role in building and maintaining bone density, and some studies show that dietary calcium slows bone loss in women (*17-19*). The roles of dietary calcium and the calcium-regulating hormones 1,25-dihydroxycholecalciferol, parathyroid hormone, and calcitonin in the development of osteoporosis have not been completely defined. Two recent studies (*20, 21*) have failed to show a correlation between calcium intake and bone density.

Race is also a factor in the development of osteoporosis. Blacks have a higher peak bone mass than whites or Asians (*22, 23*), which may account for the higher incidence of osteoporosis in whites than in blacks.

Other factors affect the development of osteoporosis or cause secondary osteoporosis; among these factors are hyperthyroidism, hyperadrenalcorticalism, and steroid therapy.

Methods for measuring bone density have aided in osteoporosis diagnosis and research. Standard X-rays have certain applications, such as measuring trabecular bone in the hip (the Singh index) (*24*), but are insensitive for assessing vertebral bone mass. Computed tomography is effective in determining vertebral bone mass. Single and dual photon absorptiometry are useful in measuring bone density in cortical and trabecular bone, respectively (*25*). The latter two techniques are relatively inexpensive and expose the patient to relatively low doses of radiation. Neutron activation analysis accurately estimates total body calcium, but the instrumentation is not widely available. Most of these methods have not been demonstrated to predict who will develop hip fracture, the fracture most responsible for mortality in osteoporosis (*26*).

Accepted drug therapies for osteoporosis are calcium supplements, postmenopausal estrogen, and calcitonin, all of which decrease bone resorption. It has been recommended that women with Type I osteoporosis receive calcium supplements, with the addition of estrogen in more advanced cases. When estrogen is contraindicated, calcitonin is suggested (*27*). These and other therapies have been discussed in a recent review (*28*).

Limited treatment is available for osteoporosis. More effective treatments, early intervention, and, ultimately, prevention are needed. Increased activity in osteoporosis research should expedite an understanding of the pathophysiology of this condition and help identify risk factors. Groups could then be selected for intervention efforts and assessment of effectiveness in practice, thus facilitating a solution to this major public health problem.

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Women's Health: Osteoporosis

Osteoporosis: Radiologic and Nuclear Medicine Procedures

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Synopsis

A number of radiographic and nuclear medicine techniques are available for the assessment of patients suspected of having osteoporosis or at risk for the development of osteoporosis.

Spinal radiographs are insensitive indicators of osteoporosis. They can document the presence of metastatic tumors or other lesions that may produce compression fracture.

The Singh index assesses the trabecular pattern of the proximal femur. As bone loss occurs, the trabeculae disappear in a definite sequence.

Radiogrammetry refers to the measurement of bone and cortical widths in the peripheral skeleton, usually the second metacarpal. The method is low cost and