METABOLIC BONE DISEASE†

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ABSTRACT

Metabolic bone diseases are frequently seen in the geriatric population. The pain which often accompanies the disorder is a major cause of morbidity, frequent visits to the physician, health resource utilization, and even mortality. Successful prevention, detection and management of metabolic bone disease are essential components of optimal geriatric health care.

OSTEOPOROSIS

Introduction

Osteoporosis refers to a condition in which there is widespread reduction in bone mass to a degree that may compromise the function of bone as a support system. Although numerous potential etiologies exist, osteoporosis is most frequently noted in older patients, particularly postmenopausal women (1-5). In fact, radiologic evidence of osteoporosis is present in approximately 18 percent of women between 45-49 years old, 66 percent of women by age 64 and almost 90 percent of women by age 75 (1, 3, 6).

The impact of osteoporosis on the population is considerable. It has been estimated that approximately 50 million Americans have some degree of osteoporosis. Seventy percent of hip fractures among individuals over the age of 45 years are a direct result of this problem (1, 3, 6). Aside from evident morbidity, the financial outlay for this complication of osteoporosis has been estimated at about 4 billion dollars per year. Approximately 15,000 yearly deaths are directly related to the 135,000 yearly hip fractures incurred among elderly persons. There is an associated mortality of 12-20 percent within six months and loss of the ability to pursue independent living in 50 percent of persons affected (1, 6-9). Only 25 percent successfully resume their premorbid life-style, and 15-25 percent will require a period of long term care. Prognosis worsens with age. During their lifetime, 15 percent of white women and 5 percent of black women will suffer a hip fracture. Similar statistics pertain to the risk of wrist fracture (1, 8). White women over 80 years of age have a 2 percent chance of hip fracture each remaining year of their lives (8). Data for vertebral compression fractures are equally impressive. One in twenty white women suffers a compression fracture by age 70, and virtually 100 percent of white women over age 80.

Pathology

The basic pathology of osteoporosis is a reduction in bone mass (1, 4). The rate of bone resorption is thought to exceed the rate of bone formation. There is a decrease in the number and size of trabeculae with concomitant facilitation of microfractures secondary to the stress forces of daily activity (5, 10).

Metabolic Factors in Osteoporosis

Calcium and organic matrix represent the major elements of bone loss in osteoporosis. Matrix forms the substrate for skeletal deposition and may be affected by several factors (1, 11). The activity of osteoblasts may be inhibited by such factors as chronic disease, alcoholism, malnutrition, lack of weight bearing activity, and hypercortisolism. In addition, bone mass may be decreased by activation of osteoclasts. This may result from decreased calcium intake; excess circulating levels of parathyroid hormone, cortisol, or thyroid hormone; deficient estrogen or testosterone; acidosis; humoral factors produced in the presence of neoplasias; and decreased physical or weight bearing activity. Detection and prevention of these potential sources of metabolic imbalance constitute a principle approach to the prevention of significant osteopenia and eventual incapacitating osteoporosis (12).

Risk Factors

While several risk factors for the development of osteoporosis are essentially endogenous, others may be subject to modification during life (4, 13). Individuals with small stature, low muscle mass, small peak adult bone mass, fair skin and family history of compression fractures or loss of height.* To whom all correspondence should be addressed.† This is the 4th paper presented in the symposium, Pathogenesis and Management of Pain in the Elderly, presented during the 16th Annual Meeting of AGE in Washington, D.C. on 9/25/86.
have significant predisposition toward the development of osteoporosis (1, 4, 8). Early menopause, either natural or surgically induced, has been associated with more significant bone disease, as has the nulliparous state. Patients should attempt to modify those detrimental factors which are within their power, such as low dietary calcium intake, sedentary life style, smoking, excess intake of protein, fiber, phosphate, alcohol and caffeine (1, 4, 5, 8).

Deficient dietary calcium is a prominent factor in the development of osteoporosis in women (1, 4, 12, 14, 15). Matkovic et al. (14) examined the rate of fracture of the proximal femur in individuals from areas of high or low calcium intake and found a direct relationship between low calcium intake and increased rate of hip fracture among patients at or beyond age of menopause. Since calcium can only be deposited within the skeleton on trabeculae, and since loss of trabeculae is one of the major pathologic events of osteoporosis, late supplementation of calcium cannot replace this organic matrix and, therefore, cannot always overcome harm previously done to bone structure (4, 10). It may help, however, retard the progression of osteopenia.

Additional factors which may contribute to reduction in bone mass and potentiate osteoporosis include chronic renal disease, collagen vascular disease, and use of steroids, heparin and antacids containing aluminum (4). The latter have the potential for also facilitating osteomalacia. Increased protein intake augments the excretion of calcium in the urine and may contribute, in some cases, to osteoporosis (16, 17). Phosphate overload may also be problematic.

Presentation

Osteoporosis usually presents clinically with bone pain related to pathologic fractures and reduction in height (1, 2, 4). Pathologic fractures mostly affect the femoral neck, distal forearms, and thoracic and lumbar spine. Fractures among vertebral bodies in the spine result in height reduction and are not always accompanied by pain (1, 2, 4, 6). Wedge-shaped collapse of vertebrae is the main mechanism for formation of the so-called "Dowager’s hump" and its accompanying reduction in height (5, 18).

Radiologic Findings

The major radiological sign of osteoporosis is a decrease in bone density with a loss of normal trabeculation (1, 4, 11, 19). Since thirty percent of bone mass must be lost to permit such visualization, osteopenia is a relatively late finding. It usually first presents as endosteal resorption with patchy osteopenia and cortical thinning which become more diffuse. A relative increase in periarticular cortical density may be perceived since the latter area tends to be less resorbed than nonperiarticular bone (4). The vertebral endplates become indented. The normal turgor of the intervertebral disk overcomes the resisting pressure of the endplate and the decreased supporting trabeculae. The disk widens and the vertebral endplates become indented in a smooth, concave manner (1, 4, 19). As osteopenia progresses, microfractures may occur and vertebrae may collapse in a wedge, often preserving posterior components and height (1, 4, 10, 19). This image may be best visualized on a plain lateral roentgenogram.

While detection of this osteopenia by x-ray may be quite common, one must consider other potential causes of osteopenia, some of which may respond to therapeutic intervention. Rheumatoid arthritis is associated with periarticular osteopenia. Reflex sympathetic dystrophy may mimic osteoporosis, with the exception of lack of accentuation of periarticular cortical bone. Hyperthyroidism produces some additional changes, including erosive disease, subperiosteal tunneling, and loss of terminal tufts. The major differential diagnoses for osteopenia resulting from osteoporosis include renal osteodystrophy, osteomalacia and Paget’s disease of bone.

Evaluation of Osteoporosis

The majority of cases of osteoporosis are considered involutional or primary and not very responsive to intervention at the time of diagnosis (18). It is for this reason that prevention is of prime importance. Approximately 20 percent of osteoporosis is considered secondary and may be amenable to intervention (1, 5, 7). Therefore, further evaluation of selected patients is appropriate to both seek a cause of the osteoporosis and assess its severity. The history should include an estimate of daily or weekly exercise or activity as well as a dietary history, including intake of calcium, phosphate, fiber, protein, caffeine and alcohol (1, 4, 12). The physical exam should note any signs of kyphosis, pelvic tilt, leg length discrepancy and loss in height as well as symptoms or findings which might suggest an underlying cause for the osteoporosis.

Laboratory assessment in primary osteoporosis usually reveals normal serum calcium, phosphorus and alkaline phosphatase. The latter may be elevated in osteomalacia, Paget’s disease of the bone or osteoporosis associated with fractures. Malignancy must be ruled out. Serum or urine protein electrophoresis usually helps rule out multiple myeloma. Additional tests commonly used to rule out disease of the thyroid, parathyroid or kidney may include tests of thyroid function, serum creatinine, urinalysis and 24 hour excretion of calcium (1, 4). Measurement of 25-OH vitamin D levels assesses the storage form of vitamin D. Measurement of serum immunoreactive para-