Osteoporosis: The Role of the Orthopaedist

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Abstract

Osteoporosis is one of the most prevalent musculoskeletal disorders encountered in orthopaedic practice today. This review provides an update on the pathophysiology of bone metabolism leading to osteoporosis, describes the latest methodology in the diagnostic workup of patients with low bone mass, and summarizes the current status of osteoporosis treatment regimens. The special needs of the osteoporotic fracture patient are also addressed. In general, load-sharing devices and sliding nail-plate constructs are preferred over rigid internal-fixation systems. Prolonged immobilization should be avoided.

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Osteoporosis ranks as a major health problem affecting more than 25 million Americans and leading to more than 1.5 million fractures each year. One of every two women over the age of 50 years will have an osteoporosis-related fracture, and one in every three men over the age of 75 years will be affected by this disease. A single hip fracture is estimated to cost between $26,000 and $30,000, and the overall cost of acute and long-term care associated with osteoporosis exceeds $10 billion annually.[1]

Because a substantial number of patients will encounter an orthopaedist for an osteoporosis-related problem, an understanding of the pathophysiology, diagnostic approach, and medical and surgical treatment options is essential. This article will provide a summary update for each of these issues, as well as a discussion of preventive strategies that the orthopaedist can offer to patients who may be at risk for developing this disease.

Defining the Problem

Osteoporosis is a disease characterized by low bone mass, microarchitectural deterioration of bone tissue leading to bone fragility, and a consequent increase in fracture risk. Although fractures of the spine, hip, and wrist are most typical of this condition, fractures of other bones, such as the ribs, humerus, and pelvis, are not uncommon.[1]

Two categories of osteoporosis have been identified: primary and secondary. Primary osteoporosis is by far the more common form of the disease and includes postmenopausal osteoporosis (type I); age-associated osteoporosis (type II), previously termed senile osteoporosis, which affects a majority of individuals over the age of 70 to 80 years; and idiopathic osteoporosis, a disorder of unknown cause that affects premenopausal women and men who are middle-aged or younger. In secondary osteoporosis, loss of bone is caused by an identifiable agent or disease process, such as an inflammatory disorder, a disorder of bone marrow cellularity, corticosteroid use, or a disorder of endocrine control of bone remodeling (Table 1).[2] It is important to recognize that the type I and type II variants of primary osteoporosis are not mutually exclusive. On the contrary, patients who have one type of osteoporotic fracture are likely to have another osteoporotic fracture of a different type.[2]

Osteoporosis reflects the inadequate accumulation of bone tissue during growth and maturation, excessive losses thereafter, or both. Since residual bone density is the net result of these factors, and since there are no safe, effective ways to rebuild the osteoporotic skeleton, prevention emerges as the crucial strategy.[1] Consequently, a knowledge of preventive approaches is essential, including awareness of the efficacy and safety of estrogen and progestin therapy, intake of calcium and other nutrients, exercise, calcitonin, bisphosphonates, and other modalities on the horizon. Prevention also requires an understanding of predictive factors, so that the likelihood of osteoporosis can be judged and an awareness of indications for estimating bone density can be developed (Table 2).

Bone Metabolism and Osteoporosis

Regulation of bone metabolism depends on the delicate balance

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Table 1
Types of Involutional Osteoporosis

Feature	Type I (Postmenopausal)	Type II (Age-Associated)
Age, yr	51–75	>70
Sex ratio (F:M)	6:1	2:1
Type of bone loss	Mainly trabecular	Trabecular and cortical
Fracture site	Vertebrae (crushed), distal radius, hip (mainly intertrochanteric)	Vertebrae (multiple wedged), hip (mainly femoral neck)
Main causes	Factors related to menopause	Factors related to aging

between the functions of several endocrine organs and their effects on the cell types found in bone (osteoblasts, osteoclasts, and osteocytes). Endocrine organs that are important to bone metabolism include the skin, parathyroid glands, liver, kidneys, gonads, adrenals, and thyroid. In addition, in certain pathologic states, pituitary and hypothalamic function also affect bone physiology. The activities of the endocrine system as they apply to bone are to maintain normal serum calcium levels.

Table 2
Osteoporosis Risk Factors

Genetic and biologic
Family history
Fair skin and hair
Northern European background
Scoliosis
Osteogenesis imperfecta
Early menopause
Slender body build
Behavioral and environmental
Excessive alcohol use
Cigarette smoking
Inactivity
Malnutrition
Low calcium intake
Exercise-induced amenorrhea
High-fiber diet
High-phosphate diet
High-protein diet

Vitamin D

Vitamin D modulates calcium homeostasis, either directly or by affecting various calcium-regulating cell systems. In Caucasian persons, 15 minutes of exposure to bright sunlight on the hands and face per day produces enough vitamin D_3 (cholecalciferol) to satisfy the minimum requirement (10 mg) of this hormone. Dark-skinned persons may require longer exposure. The major source of vitamin D is the diet, which provides vitamin D_2 (ergocalciferol). All vitamin D metabolites are fat-soluble vitamins. Because some individuals may lack sufficient exposure to sunlight as well as dietary exposure to foods naturally containing vitamin D, most milk in the United States is supplemented with vitamin D_2. The only significant natural source of vitamin D is cod liver oil.[3]

In vitamin D metabolism, precursor molecules are converted to the active form. After formation in the skin, cholecalciferol circulates to the liver, where it is hydroxylated to produce the major circulating prohormone, 25-hydroxycholecalciferol (calcifediol). Conditions that affect hepatic function and drugs that induce P-450 microsomal enzymes (e.g., phenytoin) will interrupt this conversion pathway and lead to the production of inactive polar metabolites of cholecalciferol.[3] These conditions can increase the risk of osteoporosis and, if severe, can lead to various forms of osteomalacia.

The next step in the metabolism of vitamin D is the 1α-hydroxylation of 25-hydroxycholecalciferol to form 1,25-dihydroxycholecalciferol (calcitriol)—the physiologically active form of the vitamin. The enzyme for this reaction, located in the mitochondria of renal tubular cells, is activated by parathyroid hormone. Although parathyroid hormone is the major molecule that controls 1α-hydroxylase function, phosphate, ionized calcium, and specific levels of 1,25-dihydroxycholecalciferol itself can regulate this activity.[4]

The major target tissues of 1,25-dihydroxycholecalciferol are bone, kidney, and intestine. In the kidney, it increases proximal tubular reabsorption of phosphate. It also acts as a feedback regulator of its own formation. In the intestine, calcitriol induces production of the critical calcium-binding protein responsible for active calcium transport.[3]

The physiologic role of vitamin D is less well understood. At pharmacologic doses, it accelerates bone resorption by increasing the activity and number of osteoclasts. However, vitamin D probably modulates bone physiology by acting on the osteoblast. The osteoblast then influences the osteoclast via cytokines acting as regional second messengers.[3]

Parathyroid Hormone

Parathyroid hormone and vitamin D together form a parathyroid hormone–1,25-dihydroxycholecalciferol axis, which is the major metabolic regulator of calcium and phosphate fluxes in the body.[4] The three major target organs of parathyroid hormone are bone, kidneys, and intestines.

In bone, parathyroid hormone is generally regarded as a bone-resorbing hormone. However, receptors for parathyroid hormone

are found, not on osteoclasts, but on osteoblasts, osteoblast precursors, and very early osteoclast precursors. Parathyroid hormone causes osteoblasts to (1) stimulate the release of neutral proteases, which degrade surface osteoid and initiate the bone remodeling cycle; (2) stimulate the release of unknown factors from osteoblasts, which stimulate osteoclasts to resorb bone; and (3) stimulate osteoblasts to synthesize osteoid and form bone.

The rate of synthesis and release of parathyroid hormone by the cell is related to the extracellular ionized calcium concentration. Increased levels of parathyroid hormone have been noted in the elderly, possibly because of a decrease in fractional calcium absorption in the intestine. These findings support the conclusion that the parathyroid hormone–1,25-dihydroxycholecalciferol axis may aggravate the progressive loss of bone mass in the aged.[4]

Calcitonin

Calcitonin is an important calcium-regulating hormone, the exact physiologic role of which remains controversial. It does not regulate directly the functions of parathyroid hormone or vitamin D metabolites, but its ability to modulate serum calcium and phosphate levels is significant. Calcitonin is produced and secreted by the C cells (parafollicular cells) of the thyroid gland. The major target tissues for calcitonin seem to be bone, kidney, and the gastrointestinal tract. In bone, the major defined action is the inhibition of osteoclastic bone resorption.[5]

Estrogens and Corticosteroids

The association between bone loss, fracture risk, and a postmenopausal state (naturally occurring or surgically induced) is well known. Many studies have shown that bone loss is accelerated after menopause; when ovarian hormone production ceases and circulating levels fall to 20% of previous levels, this bone loss can be reversed only by administration of estrogen.[6] Although estrogens are known to inhibit bone resorption, the mechanisms responsible for this effect are not understood. Only recently has the presence of specific estrogen receptors in osteoblast-like cells been confirmed.[7] Although the level of such receptors is very low, the fact that they appear to be active in osteoblasts and osteoblast-like cells provides the first real evidence that bone is a target tissue for estrogen action. Preliminary evidence also suggests that osteoclasts possess estrogen receptors. If this is true, it is possible that estrogen may exert direct control over both bone formation and resorption.

Both men and women experience age-related bone loss, particularly from cortical bone. In women, the rate of trabecular bone loss increases in the first few years after menopause, associated with a decrease in endogenous estrogens. Not only does estrogen replacement block this bone loss in the early postmenopausal years (years 3 to 6), but a decrease in fracture rates in the appendicular skeleton has also been documented. When used alone, 0.625 mg of conjugated estrogen per day is the lowest effective dose for retarding bone loss. Some studies have suggested that a lower dose may be effective when combined with calcium supplementation. Patients who undergo bilateral oophorectomy before natural menopause also respond to estrogen therapy. To obtain maximal benefit from estrogen replacement therapy, it should be started as soon as possible after surgical or natural menopause.[6]

It is well accepted that any factor that increases a patient's exposure to estrogen (early menarche, late menopause, estrogen replacement therapy) can increase the risk of breast or endometrial cancer. Combined cyclical estrogen-progestin therapy is believed to decrease the occurrence of endometrial, but not breast, cancer. In patients who have undergone hysterectomy, unopposed estrogen treatment (i.e., without the use of a progestational agent) is indicated.[6]

The most important factors to consider in determining whether a patient should take estrogen is the relative risk-benefit ratio. In general, patients who have a strong family history of breast cancer or endometrial cancer may be at increased risk of developing cancer or stroke as a result of estrogen treatment. Any form of estrogen is contraindicated in patients with hypertension or a history of congestive heart failure, because its effect on the renin-angiotensin axis increases sodium retention.[6] In addition, the use of estrogen is known to exacerbate benign breast diseases and cholecystitis. Estrogen is strongly beneficial not only in the prevention of osteoporosis and hip fractures but also in the prevention of heart disease in women.

Corticosteroids can cause bone loss by directly inhibiting calcium absorption, increasing renal calcium excretion, and indirectly stimulating secondary hyperparathyroidism. Their principal effects are to decrease production of the intestinal binding proteins required for calcium absorption. Very high doses of steroids decrease both bone formation and resorption. Even with doses as low as 10 mg of prednisone per day, significant bone loss occurs.[8]

Thyroid Hormones

Patients with hyperthyroidism and those who are receiving exogenous thyroid treatment may develop osteoporotic bone disease. Both bone resorption and formation are stimulated, but resorption seems to occur at

a slightly faster rate than formation. Patients with hyperthyroidism and those who take thyroid supplements for the treatment of a hypothyroid condition are also at increased risk for sustaining a hip fracture independent of bone density. Hence, thyroid hormone may have an effect on bone quality as well as bone mass.[9]

Diagnosis

Any patient over the age of 50 who presents to an orthopaedist with a hip, distal radial, or vertebral compression fracture should be evaluated for the presence of osteoporosis. A comprehensive medical evaluation should seek potential causes of secondary osteoporosis, such as hyperthyroidism, Cushing's disease, disuse, or the use of drugs known to be associated with osteoporosis (e.g., glucocorticoids, thyroid hormone supplements, phenytoin, immunosuppressants). The extent of bone loss and fractures should be assessed, and baseline biochemical data should be obtained. A careful history should include notation of the chronology, location, type, and severity of back pain (if back pain is a symptom); previous treatment; age at onset and type of menopause (natural or surgical); family history of osteoporosis; amount of tobacco or alcohol used; level of physical activity; and amount of habitual calcium intake.

Physical examination should include an accurate measurement of height and a thorough investigation to rule out systemic disease. In all patients, a complete blood cell count, differential count, and blood chemistry profile should be performed (Table 3). Thyroid function should also be assessed. In patients who are receiving thyroid hormone supplements, determination of the thyroid-stimulating hormone level is useful to be certain that thyroid replacement is not excessive. Since primary osteoporosis generally presents with a normal serum biochemical profile, abnormalities in any of these studies suggest that osteoporosis may be secondary to an underlying disease. Serum protein electrophoresis should be performed on all potentially osteoporotic patients at initial evaluation. A normal pattern excludes the presence of multiple myeloma or a related lymphoproliferative disorder in 90% of patients.

An analysis of urinary calcium excretion, normalized for creatinine (24-hour collection), and the level of urinary pyridinium cross-links (2-hour fasting sample) is considered to be part of the state-of-the-art approach to diagnosing and managing an actively resorbing osteoporotic process. (Pyridinium cross-links are specific components of the types of collagens found in bone and cartilage tissues.) In the case of collagen breakdown, the measurement of hydroxyproline excretion has been essentially replaced by the measurement of pyridinium cross-links. In addition, since osteoblastic bone formation follows osteoclastic resorption, states of high bone turnover are accompanied by increased osteoblastic activity as well. In those instances, analysis of the serum for osteocalcin, a specific osteoblast product, is another way to ascertain bone metabolic activity.

Table 3
Laboratory Tests

Routine
Complete blood cell count
Sedimentation rate
Electrolytes
Creatinine
Blood urea nitrogen
Calcium
Phosphorus
Protein
Albumin
Alkaline phosphatase
Liver enzymes
24-hour urine calcium
Serum protein electrophoresis
Special
25-Hydroxycholecalciferol
1,25-Dihydroxycholecalciferol
Osteocalcin
Urine pyridinium cross-links
Recommended for further workup based on initial history
Gastrointestinal malabsorption
Serum carotene
Thyroid function
Plasma cortisol
Serum testosterone (men)
Urine immunoelectrophoresis
Bence Jones protein

Radiography

The most characteristic feature of osteoporosis is decreased radiodensity. The apparent radiodensity, however, may vary by up to 30% because of differences in several factors, such as film development, patient weight, and the amount of x-ray exposure. A lateral radiograph is the best way to image an osteoporotic spinal deformity. The usual findings are vertebral collapse (reduction of anterior and posterior height), anterior wedging (reduction in anterior height), and biconcave compression of the end plates ("ballooning"), which usually occurs in the lumbar spinal column. The nucleus pulposus also may herniate into the vertebral body (Schmorl's node).

Bone Densitometry

The most effective way of screening for osteoporosis and then following the results of treatment is by the measurement of bone density. Several methods exist for assessing skeletal density, all of which offer a dramatic improvement over previously available methods, such as standard radiography (Table 4).[10] Although measurements of bone density in different parts of the skeleton may correlate, it is generally believed that the direct measurement of bone density at the actual site of a fracture is of the greatest clinical interest.

Table 4
Techniques for the Measurement of Bone Mass

Technique	Site	Precision,* %	Accuracy,† %	Examination Time, min	Dose of Radiation, mrem	Approximate Cost, $
Single-photon absorptiometry	Proximal and distal radius, calcaneus	1-3	5	15	10-20	75-100
Dual-photon absorptiometry	Spine, hip, total body	2-4	4-10	20-40	5	150-200
Dual-energy x-ray absorptiometry	Spine, distal radius, hip, total body	0.5-2.0	3-5	3-7	1-3	150-200
Quantitative computed tomography	Spine	2-5	5-20	10-15	100-1,000	150-200
Radiographic absorptiometry	Phalanges	1-2	4	2	100	75-100

* Precision is the coefficient of variation for repeated measurements over a short period of time in young, healthy persons.
† Accuracy is the coefficient of variation for measurements in a specimen the mineral content of which has been determined by other means.

Single-photon absorptiometry is a useful method for determining the amount of bone substance present at the distal radius, forearm, and calcaneus. It is relatively inexpensive and takes only about 15 minutes to perform. It results in a relatively low dose of radiation to the patient.

Dual-photon absorptiometry (DPA) uses transmission scanning with photons from a radioisotope source, such as gadolinium 153, that emits two energy peaks, thus allowing bone density to be measured independently from soft-tissue density. It allows measurement of the spine, hip, and total body and requires approximately 20 to 40 minutes to perform. Systems for performing DPA are no longer being manufactured because they have been replaced by the more accurate dual-energy x-ray absorptiometry (DXA) apparatus.

Dual-energy x-ray absorptiometry is an x-ray-based scanning procedure that is often used to detect bone loss in the spine, distal radius, hip, or total body. This technique is rapid, taking only 3 to 7 minutes, and delivers a radiation dose that is so low (1 to 2 mrem) as to be equivalent to approximately 5% of the radiation dose of one chest radiograph. Precision and accuracy estimates for DXA are excellent. Currently, this may be the preferred method for assessing bone loss clinically.

Quantitative computed tomography (QCT) is a sophisticated procedure that makes it possible to measure the trabecular bone compartment only, thus allowing targeted analysis of trabecular bone loss. However, it exposes the patient to a radiation dose equivalent to that of several radiographs. This may make this technique less acceptable for use in repeated bone-mass measurements.

Radiographic absorptiometry is a method of noninvasive measurement of bone mineral from radiographs of the hands. In this method, radiographs taken with standard x-ray equipment are subjected to computer-controlled analysis.

Presently, the Health Care Financing Administration (the federal agency that administers Medicare) recognizes only single-photon absorptiometry and radiographic absorptiometry as reimbursable health care costs. This agency is currently reassessing its coverage policy for these tests, as well as considering reimbursement for DPA, DXA, and QCT. In addition, third-party payers, such as Blue Cross/Blue Shield, are reassessing their coverage policies on bone-mass measurement. Charges for DPA, DXA, and QCT may be reimbursed by some insurers, but orthopaedists should advise their patients that reimbursement is not guaranteed. Since the monetary issues surrounding health care are in a state of evolution, physicians and patients must check the local and federal reimbursement policies to determine the coverage status of these relatively expensive tests. The American Academy of Orthopaedic Surgeons and the National Osteoporosis Foundation are working with federal regulatory agencies, congressional policy makers, and private insurers to develop strategies that will make these tests available to patients who need them (Table 5).

Table 5
Indications for Bone-Mass Measurement

In estrogen-deficient women, to make decisions about estrogen replacement therapy
In patients with spinal osteopenia, to diagnose osteoporosis and make decisions about further workup and treatment
In patients on long-term steroid treatment, to diagnose decreased bone mass in order to adjust dose
In patients with asymptomatic primary hyperparathyroidism, to identify need for surgical parathyroidectomy

Prevention

Prevention of osteoporosis is of primary importance, since there are no safe and effective methods for restoring healthy bone tissue and normal bone architecture once they have been lost. Preventive approaches include ensuring maximal accumulation of bone during skeletal growth and maturation and reducing or eliminating bone loss after the skeleton matures. In addition, good nutrition, modifications of lifestyle (e.g., moderation in use of alcohol and cessation of cigarette smoking), and regular physical activity are important adjuncts to any prevention and treatment program. Because most orthopaedists are exposed to a cross section of patients with respect to age, playing a proactive role in osteoporosis prevention is possible.

Adolescence and Young Adulthood

Adequate calcium nutrition during growth and maturation are key determinants of adult bone mass. Weight-bearing exercise, such as walking, jogging, and dancing, for 3 to 4 hours per week is also recommended. Skeletal integrity may be jeopardized by entities associated with premenopausal estrogen deficiency, such as anorexia, bulimia, excessive athleticism, prolactinoma, and hyperthyroidism, and by taking drugs that impair skeletal metabolism, such as glucocorticoids and antiepileptic agents. It is important for the orthopaedist to recognize these risks and to initiate preventive measures where possible.

Perimenopause and Postmenopause

Prevention of bone loss in the postmenopausal period is of the utmost importance for women at risk for osteoporosis. A strong family history of osteoporosis or a medical and social history that suggests an increased risk of osteoporosis (Table 2) should lead to the performance of a bone-density examination. If low bone mass is detected, a high calcium intake alone will not significantly mitigate the accelerated spinal loss of the postmenopausal period. Estrogen is the therapy of choice. While the best exercise regimen to promote skeletal health has not yet been determined, evidence indicates that weight-bearing exercise can reduce bone loss in this group. Preliminary studies suggest that injectable calcitonin is effective in reducing postmenopausal bone loss; however, it has not been approved by the Food and Drug Administration (FDA) for this indication.

Advancing Age

Patients who do not experience rapid bone loss at menopause but present with moderate to severe osteoporosis beginning in the seventh decade of life (type II osteoporosis) can still benefit from prophylactic measures. Appropriate calcium, vitamin D, and exercise are necessary, and cigarette smoking and excessive alcohol intake should be avoided.

Treatment

The treatment of patients who have sustained osteoporotic fractures includes maintaining their quality of life, encouraging mobilization, controlling pain safely, and promoting social interaction. Prolonged bed rest, inadequate attention to nutrition, and social isolation are avoidable pitfalls. Drugs that impair motor function, such as sedatives, tranquilizers, and hypnotic agents, should be avoided, since they may predispose to falls and fractures.

For the patient who has low bone mass or a typical osteoporotic fracture, a complete history and physical examination are necessary, and a thorough laboratory workup should be ordered to exclude common medical disorders known to cause bone loss. Osteomalacia, which can masquerade as osteoporosis, must be excluded. Treatment mainstays include adequate calcium intake, mild weight-bearing exercise, and the use of calcitonin, etidronate (Didronel), or estrogen in selected patients. The indications for bone biopsy are few and are limited to those situations in which histologic examination of bone is the only means by which osteomalacia, hyperparathyroidism, or neoplasia can be excluded with certainty. The routine use of bone biopsy in patients with osteoporosis is not recommended except when patients are being followed up as part of an experimental protocol.

Calcium

Adequate calcium in the diet is required during growth because the body does not make calcium. It continues to be an essential nutrient after full skeletal growth has been achieved because the body loses calcium every day through

shedding of skin, nails, and hair, as well as in sweat, urine, and feces. When the diet does not contain enough calcium to offset these losses, bone is catabolized in order to scavenge calcium. The current recommended dietary allowance in the United States is 1,200 mg/day in adolescence through age 24 and 800 mg/day for older adults. It is recommended that men and premenopausal women ingest 1,000 mg/day and that postmenopausal women not receiving estrogen ingest 1,500 mg/day. As already mentioned, high calcium intake will not protect a woman against bone loss caused by estrogen deficiency (type I osteoporosis), physical inactivity, alcohol abuse, smoking, or various medical disorders and treatments.[11,12]

Calcitonin

Calcitonin has been repeatedly shown to decrease osteoclast activity. It may also have an analgesic effect; the mechanism causing this pain relief is unclear. Calcitonin is inherently safe. It is available in the United States only as an intramuscular or a subcutaneous injection. Use of the injectable form may be associated with nausea, vomiting, a flushing sensation over the face, and irritation at the injection site. Injectable salmon calcitonin is approved by the FDA for treating established osteoporosis at a dosage of 100 IU daily. Lower dosages are, however, commonly utilized in practice. Human calcitonin is not FDA approved for the treatment of osteoporosis, but it is approved for the treatment of Paget's disease. A nasal spray form of calcitonin is under investigation. Patients should be advised that the cost of calcitonin treatment is high, averaging approximately $120 per month.

Estrogens and Hormone Replacement

Loss of estrogen production at any age results in increased bone remodeling, which is associated with loss of bone tissue. In patients with an intact uterus, estrogen can increase the risk of endometrial cancer unless either intermittent or continuous progestin therapy is given to prevent this complication. Estrogen replacement therapy returns bone remodeling to the level seen in premenopausal women, prevents bone loss, and reduces fracture risk. Estrogen replacement therapy, if recommended by an orthopaedist, should be used in conjunction with the consultation of an obstetrician-gynecologist or endocrinologist. Patients should be monitored for uterine response and followed yearly with mammography. There may be a small increase in the risk of breast cancer, particularly with long-term use (more than 10 years) and high doses.[13]

Bisphosphonates

The bisphosphonates, originally called diphosphonates, are a group of synthesized chemical compounds with structures similar to that of pyrophosphate. This property renders them chemically attractive to bone mineral surfaces. Once bound to bone mineral, bisphosphonates inhibit bone resorption. A number of bisphosphonates are involved in ongoing research protocols.

Published double-blind controlled studies utilizing the bisphosphonate etidronate, given 2 weeks of every 3-month period, demonstrated increased spinal bone mass and a possible decrease in the number of spinal fractures.[14] However, etidronate, if administered continuously, will cause a mineralization defect with an adverse effect on bone. Orthopaedists who prescribe this drug should advise patients that it is experimental and not FDA approved for the treatment of osteoporosis. If this experimental form of therapy is chosen, etidronate should be administered in a dose of 400 mg/day and should be taken on an empty stomach with a glass of water only. Food should not be ingested for at least an hour, because of the poor absorbability of bisphosphonates from the gastrointestinal tract. It is important to administer this drug in a noncontinuous cyclical pattern (e.g., 2 weeks on, 10 to 13 weeks off, 2 weeks on, and so on) to avoid the mineralization defect associated with continuous use. Long-term studies are required to determine the ultimate utility of this cyclical therapy.

Fluoride

Although fluoride has been used for approximately 30 years, it remains an experimental drug for the treatment of osteoporosis. Recent data suggest that fluoride may increase spinal bone mass but without a reduction in vertebral fracture rate. Of greater concern is the fact that an increased incidence of appendicular fractures may occur in certain patients. The fracture incidence may be due to the toxicity of sodium fluoride in the dosage used.[15] At present, there are no data to determine whether lower doses will be safe and effective. Until such data are available, fluoride administration should be considered highly experimental. On the basis of published reports and a careful prospective analysis of a cohort of patients, the senior author (T.A.E.) has discontinued using this drug.

Vitamin D

Most multivitamin supplements contain 400 IU of vitamin D, and milk contains 100 IU per cup. It seems reasonable for elderly persons to take a multivitamin with 400 IU of vitamin D. More than 800 IU of

vitamin D per day is not recommended because of its potential toxic side effects. Although an increase in bone mineral content has been reported in patients receiving active forms of vitamin D, it is still considered experimental in the treatment or prevention of osteoporosis.[16]

Evolving Therapies

Several drugs are currently in clinical trials to test their safety and efficacy in the treatment of osteoporosis. These include a variety of new bisphosphonates, nasal spray calcitonin, and active 1,25-dihydroxycholecalciferol. In the future, growth factors and other recombinant peptides may be shown to be safe and effective in restoring bone mass. Exercise remains a potentially important form of therapy that has been insufficiently studied. It is conceivable that the appropriate type, intensity, and frequency of exercise therapy will be found effective in preventing bone loss and increasing bone mass. Biophysical modalities such as electromagnetic stimulation and ultrasound are currently under study. While none of these is recommended for use at this time, the orthopaedist should remain aware of these investigations, since patients frequently ask their doctors about emerging technologies that may benefit them.

Rehabilitation

Back pain is frequently reported by patients with spinal osteoporosis. In many cases, the symptoms are produced by compression fractures in the thoracic and lumbar spine. Microfractures can also occur in trabeculae even when the vertebrae appear architecturally normal. Regardless of whether a macrofracture or a microfracture exists, muscle spasm is often the major cause of the patient's symptoms. To address these problems, a comprehensive spinal rehabilitation program should be developed.

In terms of prevention, patients should be instructed in the proper techniques of posture and body mechanics. They should avoid lifting heavy objects and should learn proper bending motions.[17] The use of a cane often provides the patient with better balance and reduces the possibility of falls. Patients should also be instructed in pectoral stretching, deep breathing, and back extension exercises.[17] Swimming and bicycling are excellent means of maintaining aerobic fitness and do not place undue stresses on the vertebral column.

Management of acute and chronic pain can be more difficult. Extended bed rest is not recommended in a comprehensive treatment program for osteoporotic patients. A properly fitted back support is occasionally appreciated, although these braces should be discarded as soon as symptoms improve. Management of chronic pain secondary to microfractures and kyphotic or scoliotic changes in the spine requires a program of back extension exercises and specific physical therapy tailored to the patient's needs.

Osteoporotic Fractures

The treatment of fractures in patients who have osteoporosis requires special care and attention because of the special problems associated with bone with deficient mechanical properties and fractures that are excessively comminuted. Fracture healing does not seem to be impaired in elderly persons or in patients with idiopathic osteoporosis. Hence, once an acceptable reduction and an appropriate degree of stabilization of the fragments have been achieved, fracture healing should progress normally.

Fractures to the spinal column in osteoporotic patients generally occur within the bodies of the vertebrae and usually do not affect the posterior elements. Thus, the vast majority of these fractures are stable and rarely require surgical stabilization. The temporary use of a low-profile corset or polypropylene brace may reduce muscle spasm and symptoms. The orthosis should be constructed so that it does not compromise chest expansion and pulmonary function. In most cases, patients do not require a brace in order to become comfortable.

In rare cases, unstable fractures do occur in the osteoporotic skeleton, and these may require surgical intervention (e.g., when there is neurologic compromise). The major problem in treating these unstable fractures is gaining adequate purchase for implants in osteoporotic bone.

The majority of fractures of the long bones in elderly osteoporotic patients are best managed by early surgical stabilization. Surgery should be kept simple to minimize operative time, blood loss, and physiologic stress. The goal of operative intervention is to achieve early weight-bearing status for the lower extremity and rapid restoration of functional capacity in the upper extremity.

Fracture-fixation devices that allow compaction of fracture fragments into stable patterns, minimize stresses at bone-implant interfaces, and reduce stress shielding are preferred. Because of the inability of the skeleton to hold plates and screws securely, sliding nail-plate devices, intramedullary rods, and tension-band wire constructs that share loads between implants and bone are preferred. Methylmethacrylate can be used to enhance the stability of screws in plate-fixation systems if necessary. Several manufacturers are attempting to develop new and improved fracture grout materials that not only will serve to stabilize orthopaedic implants but also may be osteoconductive and potentially resorbable.

Prolonged immobilization associated with "conservative fracture management" places the patient at risk for

medical complications. Pneumonia, congestive heart failure, thromboembolic disease, decubitus ulceration, and further generalized musculoskeletal deterioration are frequent complications in bedridden elderly patients. In addition, the delicate, poor-quality skin of many elderly patients is prone to sloughing, particularly when there is a peripheral neuropathy or vascular disease. This can lead to serious complications when casts are applied, particularly to the lower extremities. In these instances, particular attention should be paid, with well-padded casts being used.

One of the problems commonly associated with osteoporosis is the occurrence of stress fractures leading to pain, angular deformity, and, in many cases, complete fractures of the vertebrae or long bones. Although the question of stress fractures is beyond the scope of this report, it is important for the orthopaedist to recognize that osteoporotic patients who describe pain at specific skeletal sites may be experiencing a stress fracture even when the radiographs appear normal. A bone scan, CT scan, or MR imaging study may be required to make the definitive diagnosis. When stress fractures occur in parts of the skeleton that experience significant loads, prophylactic internal fixation may be required to avoid a catastrophic event, such as a displaced femoral neck fracture.

Conclusion

Unless the orthopaedist is subspecialized in an area of musculoskeletal medicine that deals strictly with young patients, it is likely that osteoporosis will become part of the day-to-day clinical experience. A comprehensive working knowledge of diagnostic modalities, medical therapeutics, and the special needs of the osteoporotic surgical patient will become more important as the population continues to age. Despite our best efforts at large-scale osteoporosis prevention, one can anticipate that the consequences of osteoporosis will affect orthopaedic surgical practice well into the 21st century.

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