CASE STUDY

79 OSTEOPOROSIS

For the Patient Case for this case study, see the printed book.

DISEASE SUMMARY

Definitions

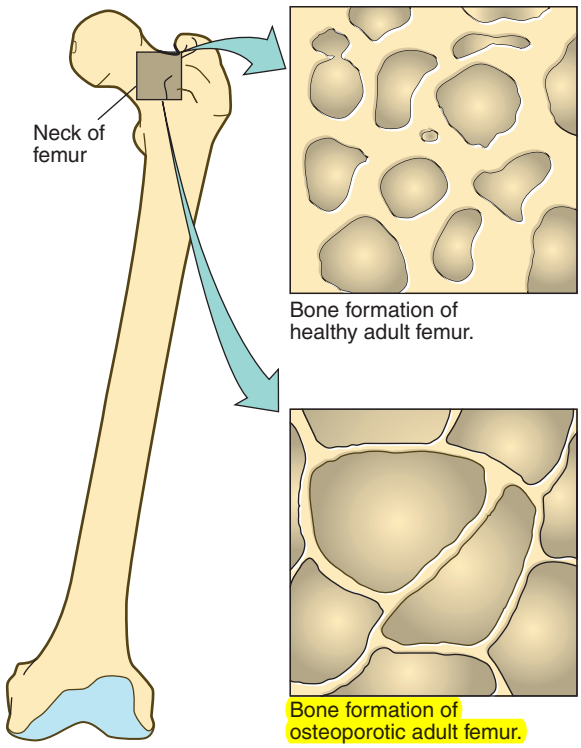
Osteoporosis has been defined in both qualitative and quantitative terms. The term *metabolic bone disease* denotes those conditions characterized by decreased bone density or mass (i.e., *osteopenia*) and diminished bone strength. *Osteoporosis* is a common, chronic, progressive type of metabolic bone disease of multifactorial etiology in which bone tissue is normally mineralized but the density of bone is decreased, causing bones to become thin and brittle and making them more likely to break. The pathology of osteoporosis is illustrated in Disease Summary Figure 79.1. The disease can be *generalized*, involving major portions of the skeleton, or *regional*, involving only one segment. Although osteoporosis-related fractures can occur in any skeletal bone region, the spine, hips, and wrists are the most common sites as illustrated in Disease Summary Figure 79.2. Bones affected by osteoporosis can fracture with only minor injury that normally would not cause a bone fracture. Even routine activities, such as bending over or coughing, can result in a fracture. Fractures can result from cracking (as with a hip fracture) or collapsing (as with a compression fracture of the vertebrae). Osteoporosis is a "silent disease" because there are few symptoms in the early stages of the disease and the condition generally comes to attention only after a bone has been broken.

The World Health Organization (WHO) has defined osteoporosis in quantitative terms based on bone density. Whereas normal bone density is >833 mg/cm^2, osteopenia occurs when bone density is 648–833 mg/cm^2 and ***osteoporosis is present when bone density is <648 mg/cm^2.*** In more clinical terms, osteoporosis has developed when bone mineral density T-scores established by dual-energy x-ray absorptiometry (DEXA or DXA) are less than –2.5.

Although this bone disease typically becomes symptomatic in the middle and later years of life, susceptibility to osteoporosis begins early in life. Because peak bone density is reached at approximately 25 years of age, it is important to build strong bones by the third decade of life so that bones will remain strong later in life. Adequate calcium intake is an essential part of building strong bones.

Prevalence

According to the International Osteoporosis Foundation, osteoporosis affects approximately one in three women and one in eight men worldwide. Currently, an estimated 24–28 million Americans have abnormally low bone mass. ***Eighty percent of all patients with osteoporosis are women.***



DISEASE SUMMARY FIGURE 79.1
Illustration showing the pathology of osteoporosis. This bone disorder involves a significant loss in the amount of bone with an increase in the size of the small pores normally found in healthy bone. (Reprinted with permission from Kamen G. Foundations of Exercise Science. Baltimore: Lippincott Williams & Wilkins, 2001.)

Because bones become weaker with aging, those individuals older than age 50 years are at greatest risk for developing osteoporosis. An estimated 10 million Americans older than age 50 have osteoporosis and another 34 million are osteopenic and considered high risk for developing the disease. This amounts to 55% of the U.S. population 50 years of age and older. Furthermore, osteoporosis affects approximately one in two women and one in five men older than age 60 years. This bone disease is not necessarily an inevitable consequence of the normal aging process, however, because some elderly people retain strong, relatively dense bones.

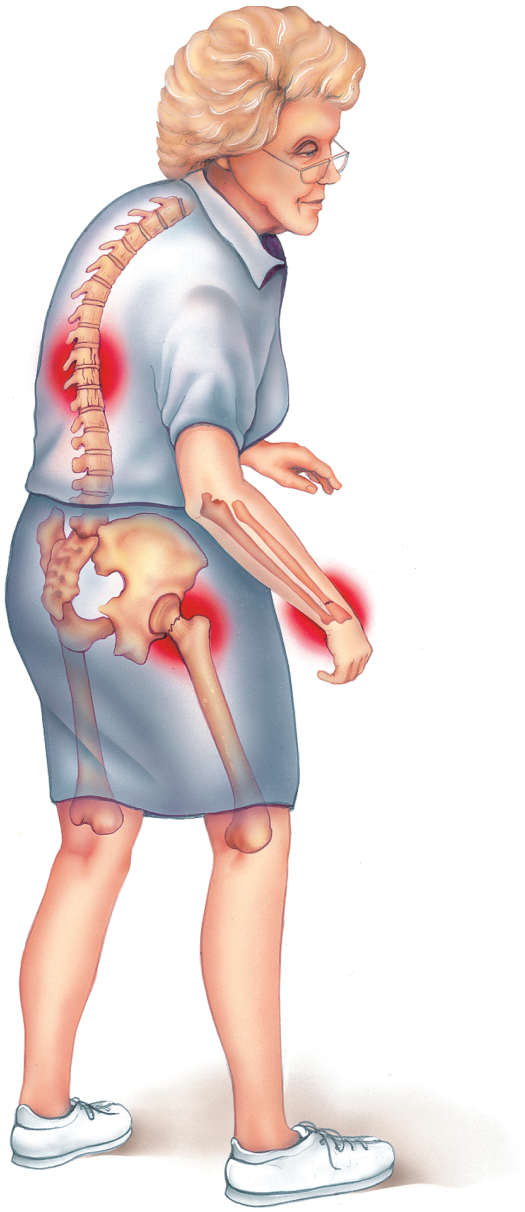
Although people from all ethnic backgrounds can develop the disorder, non-Hispanic whites (especially those of northern European descent) and southeastern Asians are at greatest risk.

According to the WHO, the prevalence of osteoporosis among U.S. white women past menopause is estimated to be 14% in those aged 50–59 years, 22% in those aged 60–69 years, 39% in those aged 70–79 years, and 70% in those aged 80 years and older.

Significance

Fractures are the most frequent and serious complications of osteoporosis. They often occur in the spine or hips—bones that directly support weight. One in two women and one in six men sustain a bone fracture from osteoporosis after age 50 years, more than 1½ million Americans experience fractures related to osteoporosis each year, and an estimated 37,500 people die from fracture-related complications. Furthermore, osteoporosis-related fractures are responsible for considerable pain, muscle wasting, spasms of back muscles, difficulty bending over, and impaired breathing from deformities of the spine and rib cage. Fractures result in decreased quality of life, lost work days, and permanent disability.

Approximately half of all broken bones caused by osteoporosis are vertebrae in the spine, so-called spinal or compression fractures, which often require a long recovery period. Once a person has experienced a spinal fracture, the risk is very high for suffering another such fracture within the next few years. Approximately 20% of postmenopausal women who



DISEASE SUMMARY FIGURE 79.2
Illustration of common fracture sites that result from osteoporosis. (Image provided by the Anatomical Chart Company.)

experience a compression fracture suffer a new spinal fracture during the following year. Fractures of the spine can cause severe "band-like" pain that radiates from the back to the side of the body. Over years, repeated spinal fractures can cause chronic lower back pain as well as loss of height and curvature of the spine (i.e., *kyphosis*). Kyphosis gives the individual a hunched-back appearance, often referred to as a "dowager hump."

Hip fractures are the second most common type of osteoporosis-related fractures and often require hospitalization. An estimated 17% of white females and 6% of white males break a hip sometime after age 50 years. By the age of 90, 32% of females and 17% of males have suffered a hip fracture. Among women who sustain a hip fracture, approximately half spend time in a nursing home while recovering and nearly one third require long-term nursing care. About half of all patients are unable to walk independently and only one third return to their pre-fracture level of function. Patients incur a diminished quality of life and decreased independence in daily living. Although most people do relatively well with modern surgical treatment, hip fractures may be slow to heal after surgical repair because of poor bone quality. Furthermore, approximately 20% of patients die from surgical complications or

other complications in the year following the fracture. Elderly patients may develop pneumonia and blood clots in the leg veins that can travel to the lungs (i.e., *pulmonary thromboembolism*) from prolonged bedrest after a hip fracture. Other fatal complications of fractures include fat embolism, hemorrhage, and shock.

Osteoporosis is the most common disease that affects bone and a significant health problem both in the United States and around the world. Unfortunately, because of an increase in sedentary lifestyles, a continued increase in life expectancy, and an aging "baby boomer" population, the incidence of osteoporosis may reach epidemic proportions in the coming decades. The extent of pain and suffering and the economic costs from this disease are expected to become enormous.

Causes and Risk Factors

Osteoporosis results from a broad and complex combination of aging, sex hormone deficiency, and genetic and environmental factors that affect peak bone mass (PBM), rate of new bone formation, and rate of bone breakdown (i.e., *resorption*). Based on cause, there are three types of osteoporosis. *Type 1* (or postmenopausal) *osteoporosis*—which is three times more common in women than men and most typically occurs in people ages 50–70 years—is believed to result from gonadal hormone (i.e., estrogen or testosterone) deficiency. Sex hormone deficiency, regardless of age of occurrence, results in accelerated bone loss and is the ***single leading cause of osteoporosis.*** *Type 2* (or senile) *osteoporosis*—which occurs twice as commonly in women and usually affects people age 70 years and older—is the result of decreased α-1-hydroxylase activity in the kidney, decreased renal production of the biologically active form of vitamin D, and decreased formation of new bone. *Type 3 osteoporosis*—which affects both genders equally and can occur at any age—is caused by a variety of medications and medical conditions that increase bone loss by multiple mechanisms. ***Corticosteroid use is the most common cause of medication- induced osteoporosis today.*** Other causes of type 3 osteoporosis and their pathogenic mechanisms are shown in Disease Summary Table 79.1.

Osteoporosis runs in families. A positive family history of osteoporosis or an osteoporosis-related fracture in a parent or sibling is a major risk factor. A tendency for lower PBM may be passed on from parent to child. Furthermore, having a mother with an osteoporosis-related hip fracture doubles the risk for a hip fracture.

Osteogenesis imperfecta is a genetic bone disorder caused by a major mutation in the gene that encodes for type I collagen, the major collagen constituent of bone. This causes severe osteoporosis and spontaneous fractures in utero and during childhood. Mutations in genes that encode for type I collagen are common, particularly in Caucasians, resulting in abnormal collagen organization.

Other risk factors include older age (starting in the mid-30s and increasing after age 50), small body frame/bone structure, physiologic or surgically induced menopause, and physical inactivity/sedentary lifestyle. Stress placed on bone from exercise and weightbearing stimulates new bone growth as a result of changes in electrical charges on the surface of bone.

Pathophysiology

Whatever the cause, osteoporosis develops when there is an imbalance between new bone formation and old bone resorption. The body may fail to form sufficient new bone, too much bone may be resorbed, or, in many cases, both processes occur simultaneously. Furthermore, mineral and protein matrix components in bone decrease. Cancellous or spongy bone (normally present in the interior of many bones where they are filled with marrow) is lost faster than cortical bone (i.e., compact bone that is >70% mineralized). As a result, fractures tend to occur earlier in cancellous bone (e.g., vertebrae) than in cortical bone (e.g., hip).

Bone is living tissue that is in a constant state of regeneration—new bone is made and old bone is broken down—a process known as *remodeling* or bone turnover. The strength of bone does not depend on bone mass alone but also on the micro-architecture of bone. Other important factors include amounts of calcium, phosphorus, and other trace elements (e.g., zinc, copper, manganese), brittleness, vitality of bone cells, structure of bone proteins, and the ability to repair micro-fractures. If any of these factors are defective or lacking, bones weaken and eventually lose their internal supporting structure.

A full cycle of bone remodeling takes approximately 2–3 months. During childhood, adolescence, and early adulthood, the body makes new bone faster than it breaks down old bone

Disease Summary Table 79.1 Causes and Pathogenic Mechanisms of Type 3 Osteoporosis

Cause of Type 3 Osteoporosis	Examples	Mechanism(s)
Heparin, high-dose or long-term therapy		↓ collagen synthesis
Anticonvulsants	Phenytoin, carbamazepine	↓ vitamin D synthesis ↑ renal calcium excretion
Corticosteroids	Prednisone, prednisolone	↓ intestinal absorption of calcium ↑ renal excretion of calcium ↓ sex hormone production ↓ collagen synthesis and new bone formation
Diuretics	Furosemide, bumetanide	↑ renal excretion of calcium
Aluminum-containing antacids		↑ renal excretion of calcium ↓ phosphate absorption ↓ bone mineralization
Cushing disease or Cushing syndrome		↑ circulating corticosteroids ↓ collagen synthesis and new bone formation
Thyrotoxicosis (hyperthyroid disease)	Graves disease, high doses of thyroid medication	↑ renal excretion of calcium ↑ bone resorption
Alcohol		↓ osteoblast activity and new bone formation Nutritional deficiency
Smoking		↑ hepatic metabolism of estrogen Direct toxic effect on bone
Prolonged immobilization	Paralysis after stroke or spinal cord injury	↑ bone resorption from activated osteoclasts
Cancer	Multiple myeloma	↑ bone resorption from activated osteoclasts
Calcium deficiency		↓ new bone formation ↑ bone resorption from ↑ parathyroid hormone activity
Vitamin D deficiency		↓ intestinal absorption of calcium and new bone formation
Gastrointestinal malabsorption disease	Crohn disease, celiac disease, cystic fibrosis	↓ intestinal absorption of calcium and new bone formation
Eating disorders	Anorexia nervosa, bulimia	Nutritional deficiency
β-thalassemia		Compensatory red cell hyperplasia in bone marrow with thinning of adjacent bone
Chronic renal disease	Diabetes mellitus, hypertension, systemic lupus erythematosus	Vitamin D deficiency ↓ renal absorption of calcium
Vitamin C deficiency	Scurvy	↓ collagen synthesis and new bone formation
Protein deficiency	Kwashiorkor	↓ collagen synthesis and new bone formation
Copper deficiency		↓ collagen synthesis and new bone formation
Hyperparathyroid disease	Benign tumor of parathyroid glands	↑ parathyroid hormone activity ↑ bone resorption
Excessive consumption of phosphates	Cola soft drinks and "junk" foods	Interference with calcium-phosphate balance
Breast cancer chemotherapy drugs	Anastrozole, letrozole	↓ estrogen concentrations

and bone mass/density increases. Peak bone mass is reached in the mid-30s. As a natural part of aging, the balance between bone resorption and bone formation shifts and more old bone is resorbed than new bone is formed. After age 35, both men and women normally lose 0.3–0.5% of their bone density each year. The pathophysiology of age-related bone loss remains unclear, but it is known that decreased serum growth hormone and insulin-like growth factor occur with increasing age. Both affect osteoblast and osteoclast function. A reduction in physical activity may also be a major factor because preservation of bone mass depends on mechanical stress through muscle contraction and weightbearing.

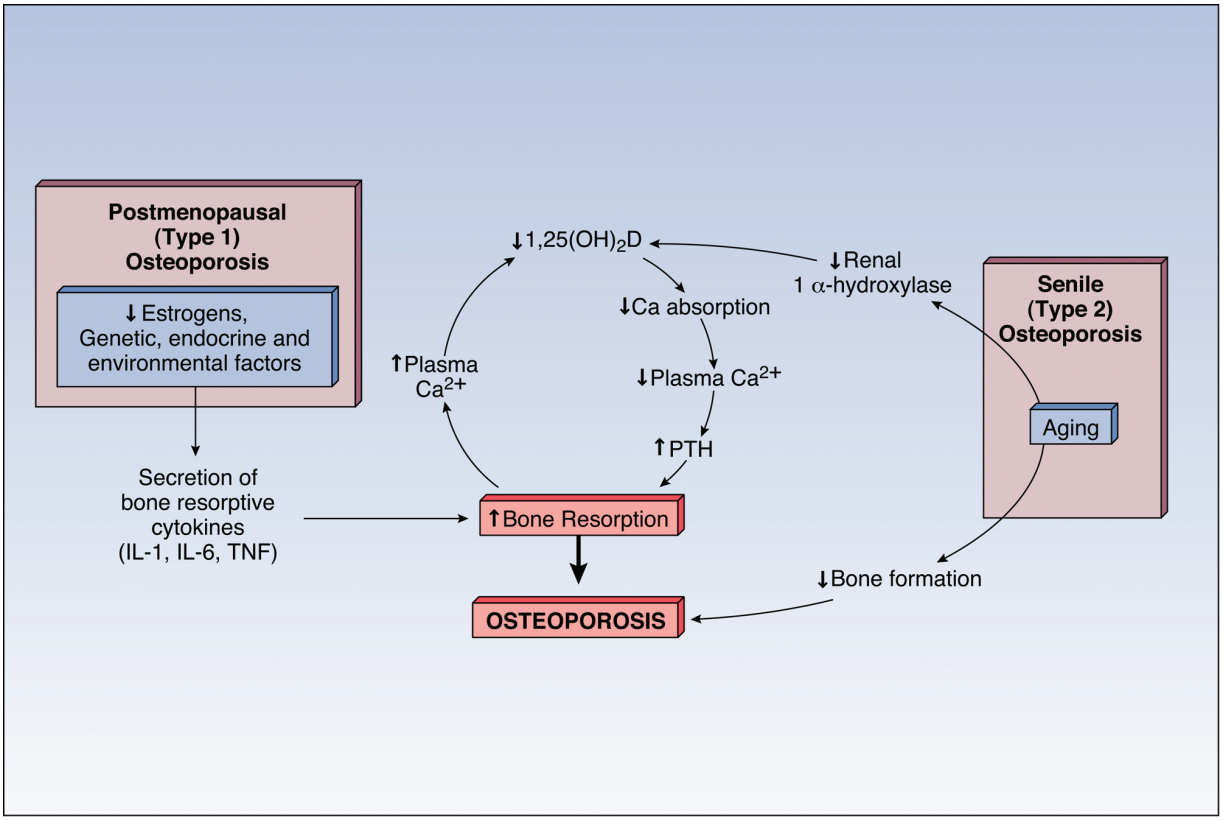
The risk for developing osteoporosis depends partly on how much bone mass is attained by 35 years of age and how rapidly it is lost later on. The greater the PBM, the more bone that is in reserve, and the less likely one will develop osteoporosis as they age. Insufficient dietary vitamin D and calcium tends to cause a lower PBM and an increased risk for developing osteoporosis.

At menopause, when the ovaries atrophy and circulating estrogen concentrations decrease, bone loss in women increases dramatically. Women may experience an accelerated bone loss up to 4% per year for the first 5–7 years. Although many factors contribute to bone loss, the leading cause in women is decreased estrogen production. Because men typically have 30% more PBM than do women, they tend to develop osteoporosis later in life. In addition, men show a more gradual loss of sex hormones than women, thereby maintaining their bone mass longer. Whereas women often become symptomatic in their 50s and 60s, men often do not present until their 70s.

The pathophysiologic mechanisms of bone loss caused by sex hormone deficiency are numerous. Ultimately, however, *an* ***increased recruitment and responsiveness of bone-resorbing osteoclast precursors, a decrease in osteoclast apoptosis (i.e., cell death), and an increase in bone resorption that outpaces bone formation occur.*** Scientific evidence also indicates that estrogen deficiency causes bone to become more sensitive to the effects of parathyroid hormone, leading to an increase in the release of calcium from bone. Osteoclasts are also activated by cytokines, such as tumor necrosis factor-alpha and interleukins 1 and 6, which are produced by mononuclear cells in times of sex hormone deficiencies. Furthermore, recent data has revealed that sex hormones protect bone-forming osteoblasts from apoptosis but induce apoptosis in osteoclasts—a scenario that is reversed when hormone levels are low.

Regional osteoporosis is often associated with immobilization of a limb as a result of a fracture or paralysis. A negative calcium balance develops early and continues throughout the period of immobilization. Significant osteoporosis will often develop within 8 weeks.

An explosion of new information has recently impacted the fields of bone biology and bone pathophysiology. Many new concepts are becoming available that will help to better explain the pathophysiology of osteoporosis in the near future. A current and detailed description of important cytokines and cytokine receptors that play key roles in the development of osteoporosis is provided by Crowther and McCance (2006). Important pathophysiologic mechanisms involved in types 1 and 2 osteoporosis are summarized in the flowchart provided in Disease Summary Figure 79.3.



Disease Summary Figure 79.3

Flowchart showing the pathophysiology of types 1 and 2 osteoporosis. (Reprinted with permission from Rubin E, Farber JL. Pathology. 3rd Ed. Philadelphia: Lippincott Williams & Wilkins, 1999.)

Diagnosis: Clinical Manifestations and Laboratory Tests

Often the first manifestations of osteoporosis are those that accompany a fracture. The disease may initially present as a backache or neck pain of varying degrees of severity, as a fracture from minimal trauma, or as collapse of a vertebra. Patients may not recall a fall or other trauma that caused the broken bone. Forward falls often result in forearm fractures near the wrist, while backward falls typically cause a hip fracture. Rib fractures are often the result of osteoporosis from corticosteroid use or Cushing disease/syndrome. Pain may become worse with movement or activity that places weight on the fractured bone. Sometimes, back pain radiates around to the abdomen.

Once osteoporosis is suspected, a carefully gathered patient history and physical examination are performed. The patient history often includes information about lifestyle factors, medications, current medical conditions, early menopause, and family history. On physical examination, the patient may show signs of a fracture of the radius near the wrist, a femoral or hip fracture, or a spinal fracture. A decrease in height is common. Obvious kyphosis of the thoracic spine may be present and the patient may complain of back spasms, difficulty bending over, or difficulty breathing. Systemic manifestations, such as weakness and a recent, significant, and unintentional weight loss, suggest that osteoporosis might be caused by another medical condition (i.e., type 3 osteoporosis is present).

Serum calcium, phosphate, and parathyroid hormone concentrations are usually normal. The serum alkaline phosphatase level is typically normal but may be elevated for several months after a fracture. Serum osteocalcin (i.e., a marker of bone turnover) concentrations are elevated. Once a diagnosis of osteoporosis has been established, further testing for Cushing disease/syndrome, hyperthyroid disease, vitamin D deficiency, celiac disease, or sex hormone deficiency may be required. Vitamin D deficiency is extremely common, and a serum 25-hydroxyvitamin D level should be obtained from any individual with abnormally low bone density.

Routine x-ray films and quantitative CT (i.e., computed tomography) scans often show increased radiolucency of bones (i.e., x-rays penetrate easily with minimal absorption), sparse cancellous bone, thinning of cortical bone, abnormally shaped and deformed thoracic and lumbar vertebrae, and signs of both old and recent fractures. Unfortunately, however, by the time abnormalities can be detected by radiographic examination, as much as 30% of total bone may have been lost. Furthermore, CT scanning can only determine bone density in the spine and is associated with significant exposure to radiation at high cost.

An important advance in methods used for the diagnosis of osteoporosis has been the use of bone mineral density assessment. Common areas of marked demineralization in patients with osteoporosis are the vertebrae and pelvis, especially the head and neck of the femur. Demineralization is less pronounced in the skull and limbs. ***The clinical method of choice*** (recommended by both the National Osteoporosis Foundation and the American Medical Association) ***for bone density studies is dual-energy x-ray absorptiometry (DEXA or DXA) of the lumbar spine and hip.*** DEXA can determine the density of any bone with accuracy, is fast, non-invasive, painless, and delivers far less radiation (only 1–10%) than does a standard radiograph. Whenever possible, the first four vertebrae are assessed and the site with the lowest score is used to make the diagnosis. The DEXA test finding is compared against a bone mineral density reference standard developed from young, healthy individuals of the same gender and race, resulting in a clinical value known as a *T-score*. A T-score $>$ –1.0 is normal. T-scores between –1.0 and –2.5 are an index of osteopenia. If the T-score $\leq$ –2.5, osteoporosis is present by definition.

Type 1 osteoporosis is associated with estrogen or testosterone deficiency, loss of cancellous bone, and fractures of the vertebrae and radius near the wrist. Type 2 osteoporosis is associated with calcium deficiency, loss of both cancellous and cortical bone, and hip fractures.

Appropriate Therapy

The goals of treatment for osteoporosis are to slow down the rates of calcium and bone loss and prevent broken bones, stop progression of the disease, and, in some cases, reverse the disorder. A number of pharmacologic treatment options are available, including sex hormone replacement, selective estrogen receptor modulators (SERMs), bisphosphonates, calcitonin, vitamin D, calcium, teriparatide, and strontium. Treatment varies with cause.

Disease Summary Table 79.2 Comparison of the Benefits and Disadvantages of Estrogen and Raloxifene Treatments for Osteoporosis

Drug Effect	Estrogen	Raloxifene
Reduces LDL "bad cholesterol"	+	+
Increases HDL "good cholesterol"	+	–
Reduces hot flashes	+	–
Relieves vaginal dryness	+	–
May cause blood clots	+	+
May cause uterine bleeding	+	–
May cause endometrial cancer	+	–
May cause breast cancer	+	–
May cause breast soreness	+	–
Significantly reduces the risk of breast cancer	–	+
May cause birth defects when taken during pregnancy	–	+

Source: Fitzgerald PA. Osteoporosis. In: McPhee SJ, Papadakis MA, Tierney LM Jr., eds. 2007 Current Medical Diagnosis and Treatment. 46th Ed. New York: McGraw-Hill, 2007:1179–1183.

Women with osteoporosis and estrogen deficiency may be considered for replacement estrogen therapy (e.g., *conjugated estrogen*, *estradiol*), although this is not always the first therapeutic choice. While estrogen treatment increases bone mineral density by approximately 2% in 2 years in these patients, it has not been shown to significantly reduce the risk for fractures. The use of *phytoestrogens*, naturally occurring plant compounds, has gained popularity as an alternative for estrogens. However, information regarding their effect on bone health is conflicting and incomplete. Men with osteoporosis and androgen deficiency may be treated with *testosterone*. Testosterone replacement in men increases spinal, but not hip, bone mineral density.

SERMs have been developed to provide the positive effects of estrogen on bone while minimizing the negative effects on breast and endometrial tissues. *Raloxifene* (taken orally every day) is a SERM that can be used by postmenopausal women in place of estrogen to prevent the progression of osteoporosis. It is recommended for women who have infrequent hot flashes, are at low risk for cardiovascular disease, and are at moderate-to-high risk for breast cancer. Bone density increases approximately 1% over 2 years with this drug. It also reduces the risk of vertebral fractures by up to 50% but does not appear to reduce the risk of non-vertebral fractures. A brief comparison of the benefits and disadvantages of estrogen and raloxifene treatments is shown in Disease Summary Table 79.2.

Bisphosphonates are the most effective agents for treatment of osteoporosis and act by inhibiting osteoclast-induced resorption of bone. The most dramatic impact of these agents has been in the reduction of multiple spinal fractures, a finding that indicates that treatment can slow the progression of osteoporosis. Bisphosphonates may be given orally once weekly. This is more convenient than daily therapy and equally effective. Available oral preparations include *alendronate* and *risedronate*. Alendronate has been shown to reduce bone resorption and the prevalence of fractures of the spine and hip by approximately 50%. ***Alendronate is also the treatment of choice for prevention of glucocorticoid-induced osteoporosis in premenopausal women.*** Risedronate has been shown to reduce the prevalence of new vertebral fractures by 62% and multiple new vertebral fractures by 90% in postmenopausal women with osteoporosis. The risk for hip fractures is also markedly decreased. In March 2005, the U.S. Food and Drug Administration (FDA) approved *ibandronate* (taken orally once monthly) for both treatment and prevention of osteoporosis. In postmenopausal women, the drug reduces the risk for vertebral but not non-vertebral fractures. *Pamidronate* is a parenteral bisphosphonate that can be given by slow intravenous (IV) infusion in normal saline solution every 3 months for patients who cannot tolerate oral bisphosphonate preparations. *Zoledronic acid* is a third-generation bisphosphonate and a potent inhibitor of osteoclast-induced resorption. It can be given every 6–12 months IV over 15–30 minutes. Zoledronic acid is also especially useful for patients who cannot tolerate oral bisphosphonates.

Calcitonin can be used to decrease the activity of osteoclasts in patients in whom bisphosphonates and estrogen are contraindicated or not tolerated. A nasal spray of calcitonin is available, and nasal administration causes significantly less nausea and flushing than does

the parenteral preparation. Five years of therapy with calcitonin has been shown to increase bone density by 2–3% and reduce the frequency of vertebral fractures. However, a positive effect upon non-vertebral fractures has not been demonstrated.

Vitamin D supplementation is useful for preventing the progression of postmenopausal osteoporosis because it optimizes intestinal calcium absorption and inhibits parathyroid hormone secretion, which stimulates calcium resorption from bone. It reduces the incidence of vertebral fractures and may slightly decrease the frequency of non-vertebral fractures. Vitamin D_2 (ergocalciferol) is typically given daily. High doses may be required when serum levels of 25-hydroxyvitamin D are <20 ng/mL and in those patients with intestinal malabsorption.

It is well accepted that oral calcium intake sufficient to maintain normal calcium balance is necessary during adolescence to ensure maximum PBM and that calcium-deficient diets can aggravate bone loss associated with menopause and aging. However, *calcium* supplementation alone has only a minor effect on the progression of osteoporosis and has not yet been established to significantly reduce the risk of fractures. More clinical trials that account for potential confounding factors (e.g., smoking and level of physical activity) are necessary to test the effects of dietary calcium or calcium supplements on bone loss. Nevertheless, calcium (1.0–1.5 g/day) is recommended for patients with established osteoporosis and calcium supplements may also reduce the risk of colon cancer. Calcium can be given as calcium citrate (0.4–0.7 g elemental calcium daily) or as calcium carbonate (1.0–1.5 g elemental calcium/day). Studies have shown that taking calcium plus vitamin D significantly reduces the risk of hip and other fractures in women who have gone through menopause.

Teriparatide is a powerful new biotechnological analog of parathyroid hormone that works primarily to stimulate new bone formation by increasing both the number and activity of osteoblasts. When administered to patients with osteoporosis subcutaneously daily for 2 years, the drug dramatically improves bone density in all bones except the radius. Teriparatide is approved only for a 2-year course of treatment.

Strontium ranelate (taken orally every day) can be given to women with osteoporosis and vertebral fractures. Strontium reduces fracture incidence by 40% and increases bone density in the lumbar spine and neck of the femur.

Soy products may help reduce the risk of broken bones due to osteoporosis, but there is not enough evidence to show if other natural products are effective.

The diet should be rich in protein, total calories, and calcium. Regular, moderate weight-bearing exercise, aerobics, and resistance exercises can slow down bone loss and, in some cases, reverse demineralization, because the mechanical stress of exercise stimulates bone formation. Walking increases bone mineral density and increases strength in both the spine and hips in postmenopausal women. High-impact physical activity (e.g., jogging) significantly increases bone density in both men and women. Stair-climbing increases bone density in women. Patients who cannot exercise vigorously are encouraged to engage in other exercise regularly, thereby increasing strength and reducing the risk of falling. Weight training is also helpful to increase muscle strength and bone density. Measures should be taken to avoid falls at home (e.g., adequate lighting, handrails on stairs and in bathrooms). Patients who are weak or have balance problems must use a cane or walker. Rolling walkers should have a brake mechanism. Balance exercises (e.g., tai chi) can reduce the risk of falls. Patients should keep active. Bedridden patients should be given active or passive exercise routines.

Some health professionals recommend the use of hip protectors for persons at risk for falling. Hip protectors look like a girdle or underwear with pads on both sides of the hips to reduce the force of a fall. When hip protectors are used 24 hours/day, the frequency of hip fractures is greatly reduced.

Smoking is strongly discouraged and alcohol use should be limited to no more than one drink each day. For pain and muscle spasms, analgesics (e.g., non-steroidal anti-inflammatory agents such as ibuprofen and naproxen, acetaminophen, or narcotics such as codeine and morphine) and muscle relaxants may be necessary.

Serious Complications and Prognosis

The most serious complications of osteoporosis are hip fractures and compression fractures of the spine—both of which may lead to chronic pain and long-term disability—and impaired breathing from deformities of the spine and rib cage.

With adequate treatment, the progression of osteoporosis can be slowed, stopped, or reversed. Nevertheless, some people become severely disabled as a result of weakened bones and require long-term nursing care. Hip fractures are a frequent complication and leave about half of those who break a hip unable to walk independently again. Hip fractures can also contribute to significant mortality (e.g., from life-threatening pneumonia or blood clots). Only one third of patients are able to return to their pre-fracture level of function. Patients often incur a diminished quality of life and decreased independence in daily living.

Suggested Readings

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