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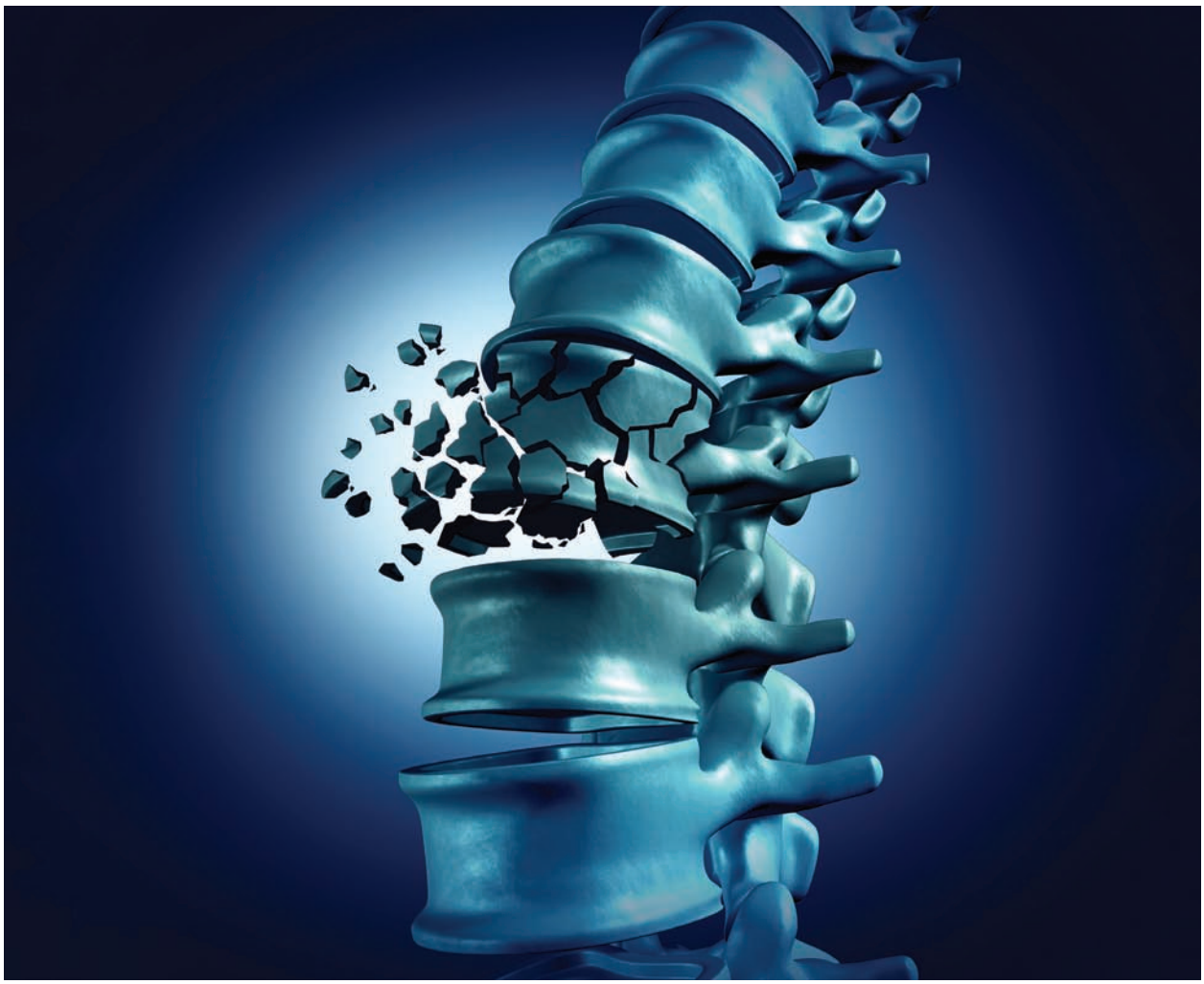

SHUTTERSTOCK/LIGHTSPRING

Osteoporosis: Diagnosis and management updates

BY ANNETTE M. PEACOCK-JOHNSON, DNP, RN AND PATRICIA KERESZTES, PhD, RN

Abstract: Osteoporosis, a common chronic bone disease, results in reduced bone mineral density and heightened fracture risk, particularly vertebral fractures. This article provides an overview of the condition's diagnosis and management updates.

Keywords: bone mineral density, bone remodeling, fractures, osteoporosis, risk factors

Osteoporosis, the most common chronic bone disease nationally, is estimated to affect over 10 million persons at least 50 years old.[1] It is characterized by decreased bone mineral density, leading to an increased fracture risk.[1-5] Osteoporosis is commonly undiagnosed until a fracture occurs.[6,7]

Vertebral fractures are the most common type of osteoporotic fracture.[2,4] Once one vertebrae has fractured, there is a greater risk of additional vertebral and other fractures.[2,4]

Bone physiology

Bone physiology balances bone formation (mineralization) and destruc-

tion (resorption). Osteoblasts are cells responsible for bone formation and mineralization; osteoclasts are cells responsible for bone resorption or breakdown. A third type of cells, osteocytes, are involved in both the modeling and remodeling of bone.[8] Osteocytes regulate bone modeling and remodeling and produce sclerostin, inhibiting bone formation.[4,8] Bone remodeling, or the ability of the skeleton to renew itself completely, begins in fetal life and continues throughout the lifespan. This is necessary to repair damaged bone due to constant physical loading.[9]

There are five phases in the bone remodeling cycle: activation, resorption, reversal, formation, and mineralization.[9] Activation and resorption involve the start of bone resorption when osteoclast precursors arrive at the bone area to be remodeled. During the reversal phase, the osteoclasts disappear and are replaced by osteoblasts, which begin to function and start the phase of actual bone formation. During the last phase, the newly developed bone mineralizes with calcium phosphate.[9] Outside of this cycle, bones are considered to be in a resting state. Bone remodeling is stimulated by the breakdown of old bone by the osteoclasts to preserve bone strength, in the presence of fractures and the skeleton's ability to respond to mechanical use, and takes 2 to 4 weeks.[10] The bone absorption and growth cycle takes 4 to 6 months.[10] While bone remodeling occurs throughout a human's life, peak bone mass is achieved between ages 30 and 35. Bone mineral density (BMD) is the mineral content per unit volume of bone.[11]

Pathophysiology of osteoporosis

Osteoporosis is an imbalance between the activity of the osteoclasts and osteoblasts.[7,12] In this bone disease, the normal cycle of bone remodeling is not completed. Bone is resorbed by osteoclasts but not remineralized by the osteoblasts. Factors influencing this imbalance include calcium intake, vitamin D receptors, physical activity or mechanical stress, hormones (parathyroid, estrogen, testosterone), and receptor activators of nuclear factor-kappa-B ligand (RANKL). Released by osteoblasts and bone marrow stem cells, RANKL binds to a receptor that activates osteoclasts, causing bone resorption.[12] Calcium is essential for mineralization and subsequent bone strength, while vitamin D is essential for the intestinal absorption of calcium and thus influences bone strength. Physical activity and exercise stimulate osteoblasts and mineralization, increasing BMD. Additionally, exercise helps regulate estrogens, parathyroid hormone, and glucocorticoids.[9] Estrogens, especially 17-estradiol, inhibit bone resorption through their effects on cytokines and their involvement in calcium homeostasis and vitamin D metabolism required for bone maintenance.[12] Parathyroid hormone influences bone maintenance through its effect on the gastrointestinal and renal absorption of both calcium and phosphate.[12] Androgens, predominantly testosterone, have been shown to suppress bone resorption.[12] RANKL signaling is involved in regulating osteoclastic bone resorption.[10]

Nurses are essential in educating individuals on osteoporosis risk factors and prevention.

Risk factors

Primary osteoporosis is associated with age and gender. The two most significant risk factors include advanced age and female gender.[6] Bone loss in later life is associated with estrogen deficiency resulting from menopause, a possible explanation for osteoporosis being three times greater in females than males.[13] Additionally, a body mass index (BMI) less than 21, genetics, a history of smoking, and excessive alcohol are associated with low BMD.[14] Women who smoke demonstrate an earlier menopause with lower serum estradiol levels.[14] Consuming more than one serving of alcohol daily for females and three servings daily for males is associated with an increased osteoporotic risk.[3]

Secondary osteoporosis can occur from multiple endocrine and immune diseases that affect bone metabolism (see *Causes of secondary osteoporosis*).[6,14-16] Diabetes mellitus is the most common secondary cause of osteoporosis, followed by medication-induced osteoporosis.[16] Adults with type 1 diabetes tend to have a higher risk of fracture than those with type 2.[16]

Clinical manifestations

The first sign of osteoporosis is a fragility fracture that occurs from a fall from, at most, standing height. Fractures commonly occur in the spine and the hip. The initial symptom is the onset of acute pain, described as sharp or dull and presents over the affected area. Pain may decrease or limit the patient's range of motion. Because of the risk of vertebral

Causes of secondary osteoporosis[16]

Cause	Rationale
Dietary deficiencies of calcium and vitamin D	Adequate vitamin D is necessary for intestinal absorption of calcium needed for bone formation
Hyperparathyroidism	Parathyroid hormone indirectly stimulates osteoclasts to increase bone resorption
Chronic kidney disease	Hyperphosphatemia contributes to hypocalcemia and the development of secondary hyperparathyroidism
Hyperthyroidism	Severe hyperthyroidism increases bone turnover contributing to decreased bone density
Hypogonadism	Reduced testosterone in men inhibits osteoblasts decreasing bone density
Antidepressants, opioids, antipsychotics, antihypertensives	Adverse central nervous system effects may contribute to an increased fall and fracture risk
Diabetes mellitus	Increases bone porosity and impairs bone quality
Adverse drug reactions	• Aromatase inhibitors decrease estrogen levels thereby increasing bone resorption • Phenytoin impairs vitamin D metabolism • Glucocorticoids inhibit bone formation • Anticoagulants including heparin and warfarin have been associated with increased osteoporotic fractures
Inflammatory conditions: rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease	Proinflammatory cytokines stimulate increased production of osteoclasts with increased bone resorption

fractures, patients may develop either cervical lordosis or thoracic kyphosis and a decrease in height by 2 to 3 cm with aging.[17]

Diagnosis

Fracture prevention should begin with a screening and a comprehensive assessment of risk factors. BMD testing should be conducted to determine the risk of fractures in males and females 70 years and older and in postmenopausal females between 50 and 70 years with known risk factors.[2]

Lab evaluation includes a biochemistry profile (especially serum calcium, phosphorus, albumin, total protein, creatinine, liver enzymes including alkaline phosphatase, and electrolytes) and 25-hydroxyvitamin D levels. Despite food sources and sunlight, most populations around the world are deficient in vitamin D.[18] Additional lab tests may be indicated to identify underlying secondary causes of osteoporosis and may include a complete blood cell count, C-reactive protein, calcitonin or parathyroid hormone, and erythrocyte sedimentation rate. A mildly elevated alkaline phosphatase can occur in individuals with fractures or osteomalacia.[14-18]

The definitive diagnostic study for osteoporosis is the dual-energy X-ray absorptiometry (DXA) scan.[13,14,18] A noninvasive DXA scan of the lumbar spine and proximal femur is the preferred standard for assessing bone density. DXA scan results are expressed as a T-score (see *DXA results*).[4,17]

A baseline DXA scan for persons at risk is essential. The National Osteoporosis Foundation recommends repeat DXA scans every 2 years as indicated to evaluate BMD related to disease progression or treatment response.[18]

The Fracture Risk Assessment Tool (FRAX) estimates the 10-year probability of hip fracture and major osteoporotic fracture (hip, spine, proximal humerus, or forearm) for untreated patients.[2,5,6,13,16] The FRAX tool screens for risk factors, including older age, gender, height, and weight. In addition, the questionnaire assesses secondary causes of osteoporosis and personal risk factors, such as smoking, prolonged glucocorticoid use beyond 3 months, three or more daily alcoholic beverages, rheumatoid arthritis, history of a parent with a hip fracture, and a previous fracture involving the wrist, spine, or hip.

Lifestyle management

Nurses are essential in educating individuals on osteoporosis risk factors and prevention. The primary approach for osteoporosis prevention is the recommendation of lifestyle adjustments. Recommendations provided by the nurse include instructions on weight management, dietary guidelines, physical activity, smoking cessation, limiting alcohol intake, and fall prevention.

DXA results

Interpretation	T-score
Normal	≥-1.0
Osteopenia	Between -1.0 and -2.5
Osteoporosis	≤-2.5
Established osteoporosis	≤-2.5 and fragility fracture

Weight management

Maintaining a healthy weight for an individual's height is recommended since body weight is the load on bones.[19] A body weight of less than 127 lb (57.6 kg) places females in the lower quartile of weight for US females 65 years and older and is a risk factor for low BMD and osteoporosis.[14,20] Low body weight may also be an indicator of malnutrition.[19]

Nutrition

A well-balanced diet high in calcium-rich foods is a primary dietary approach to preventing and managing osteoporosis. Green leafy vegetables, tofu, and calcium water are alternate sources of calcium.[20] Calcium supplements are recommended for persons unable to achieve adequate dietary calcium intake of 1,000 mg to 1,200 mg daily.[14] Additionally, vitamin D3 supplements are recommended when vitamin D-fortified foods and sun exposure are insufficient to achieve therapeutic serum vitamin D levels.[15] Dietary recommendations should include alcohol cessation as alcohol directly decreases osteoblastic activity.[16]

Physical activity

Weight-bearing activities like walking, low-impact aerobics, and stair climbing can help prevent mineral loss and fragility fractures.[20] Exercise may influence cytokines, hormones, and signaling pathways promoting bone formation while minimizing bone resorption. While weight-bearing activities will not reverse bone loss, they may minimize further bone loss and the risk of fractures. A minimum of 30- to 40-minute weight-bearing sessions three times weekly is recommended.[15,20]

Resistance exercise, balance training, and postural exercises are also encouraged. Resistance exercises using elastic exercise bands, weights, and weight machines increase muscle strength.[4] Improved muscle strength exerts a greater force on bone than gravity; decreased muscle force is associated with bone loss.[21]

Pharmacologic treatment of osteoporosis must begin by assessing calcium and vitamin D levels and providing supplementation.

A systematic review on the effects of different exercise regimens on bone health found that all exercise interventions were effective in preventing bone loss compared with no exercise program, while mind-body exercises were most effective at minimizing vertebral BMD loss.[22] The latter exercises are characterized by slow, gentle, and coordinated movements with breathing suitable for older adults. Mind-body exercises incorporate upper extremity exercises, promote postural stability, and increase the secretion of hormones or hormone-like substances, all indirectly promoting bone metabolism and improving vertebral bone density.[22]

Fall prevention

Falls are a significant contributor to fractures. One-third of females 65 years and older fall one or more times yearly.[23] Multicomponent exercise programs that emphasize balance and muscle strength in addition to weight-bearing activities are advisable for preventing falls and subsequent fractures.[23,24]

Fall prevention should include screening for factors that increase the risk of falls.[4] A comprehensive medication review should identify medications that may potentially contribute to falls from neurologic or cardiac effects, such as lightheadedness or sedation. Additionally, a thorough home assessment should be done to detect safety hazards, such as poor lighting, clutter in walkways, and throw rugs.

Pharmacotherapy

In addition to lifestyle recommendations, pharmacologic intervention is indicated in patients with a significantly increased risk of fractures (see *Medications for osteoporosis*).[25] The pharmacologic treatment of osteoporosis aims to reduce fracture risk and improve quality of life.[9] The Bone Health and Osteoporosis Foundation recommends pharmacologic treatment for primary fracture prevention in postmenopausal females and males 50 years of age and older with a T-score of -2.5 or less at the lumbar spine, femoral neck, or total hip by DXA.[4] Pharmacologic treatment is also recommended for secondary fracture prevention in patients with a past hip or vertebral fracture or fracture of the humerus, pelvis, or distal forearm with a T-score between -1.0 and -2.5.[4] Treatment plans must be individualized.

Calcium and vitamin D

Pharmacologic treatment of osteoporosis must begin by assessing calcium and vitamin D levels and providing supplementation as needed. Commonly used calcium supplements include calcium carbonate and calcium citrate.[12] The recommendation for daily intake of calcium

is 1,000 mg/day for males ages 50 to 70 and 1,200 mg/day for females 51 years or older and men 71 years of age or older.[4] Vitamin D is given as cholecalciferol (vitamin D3). Levels of vitamin D should be maintained between 30 ng/mL and 50 ng/mL. Vitamin D supplements are given in doses of 800-1,000 units/day up to as much as 4,000 units/day as needed to achieve an adequate vitamin D level.[4]

Medications for osteoporosis[17]

Medication	Classification	Adverse reactions	Nursing implications
Calcium carbonate Calcium citrate	Supplement	• Constipation • Nausea • Anorexia	• Take calcium carbonate with food. • Calcium citrate most easily absorbed. • High doses may cause kidney stones.
Vitamin D3	Supplement	• Nausea • Confusion	• Monitor for signs and symptoms of vitamin D toxicity, including hypercalcemia, hypercalcuria, confusion, psychosis, tremor, calcification of soft tissues, nausea, and weakness.
Ibandronate Alendronate Risedronate Zoledronic acid (I.V. only)	Bisphosphonates	• Upper respiratory infection • Back pain • Dyspepsia • Diarrhea • Esophagitis	• Take on an empty stomach with a full glass of water. • Remain upright for 30-60 minutes after taking. • May cause hypocalcemia. • May cause osteonecrosis of the jaw or atypical femoral fracture with prolonged use. • No data on use in pregnancy.
Raloxifene Bazedoxifene	Selective estrogen-receptor modulators	• Flulike symptoms • Deep vein thrombosis (DVT) • Muscle spasm • Arthralgia • Infection • Insomnia • Hot flashes	• Not for use in men. • Taken with or without food. • Teach patients signs and symptoms of DVT and pulmonary embolism.
Denosumab	RANKL inhibitor	• Skin infections • Back pain • Extremity pain • Asthenia • Hypophosphatemia • Dyspepsia • Hypocalcemia • Cough • Headache	• Given subcutaneous every 6 months. • Not recommended in those under 18 years. • May cause hypocalcemia. • Drug holiday not recommended.
Calcitonin	Hormone	• Rhinitis • Epistaxis	• Given as intranasal spray once daily.
Romosozumab	Antisclerostin monoclonal antibody	• Angioedema • Dermatitis	• Given as two subcutaneous injections once a month for 12 months. • Limit use to 1 year. • Boxed warnings for an increased risk of cardiovascular events. • Teach patients signs and symptoms of myocardial infarction and stroke.
Teriparatide	Parathyroid hormone analogue	• Pain • Arthralgia • Rhinitis • Asthenia • Headache • Hypertension • Pharyngitis	• Administered daily by subcutaneous injection. • Limited use of 1-2 years but may continue longer with severe fracture risk.

Bone resorption inhibitors

Bisphosphonates

Treatment is typically initiated with antiresorptives, which can reduce the risk of fracture by up to 50%.[25] It is recommended that calcium and vitamin D levels are adequate before initiating pharmacologic therapy and are usually given as supplements along with the pharmacologic agents.[9]

Bisphosphonates inhibit bone resorption by inhibiting osteoclasts.[10] Bisphosphonates are indicated for postmenopausal females, males with osteoporosis, and in glucocorticoid-induced osteoporosis.[4,17] The common bisphosphonates prescribed include ibandronate, alendronate, and risedronate. The I.V. option is zoledronic acid.

Bisphosphonates are contraindicated in patients with a glomerular filtration rate less than 35 mL/min/1.73 m^2.[25]

Alendronate has been shown to reduce the rate of spine, hip, and wrist fractures by 50%.[13]

Zoledronic acid is given as a once-yearly infusion and is considered the most potent bisphosphonate.[17] It has been shown to increase BMD of the spine by 4% to 5% and the hip by 3.1% to 3.5%.[17] Zoledronic acid has also been shown to be effective in treating osteoporosis in men.[17]

A rare adverse event associated with bisphosphonates is osteonecrosis of the jaw and atypical femoral fracture.[25] Therefore, it is recommended that patients see a dentist before initiating therapy. Additionally, because of these risks, the Bone Health and Osteoporosis Foundation recommends evaluating patients and their BMD scores after 5 years of oral and 3 years of I.V. bisphosphonates for a temporary drug suspension in patients who are no longer at high risk of fracture (T-score of –2.5 or more, no new fractures).[4]

The common bisphosphonates prescribed include ibandronate, alendronate, and risedronate.

Selective estrogen receptor modulators (SERMs)

Raloxifene and bazedoxifene are classified as estrogen receptor agonists and reduce postmenopausal bone loss.[25] While also used for breast cancer prevention, these medications decrease bone resorption by affecting estrogen receptors.[17] The SERMs mainly reduce the risk of vertebral fractures.[25] Raloxifene is indicated for use in postmenopausal women.[17] Raloxifene is administered daily in oral pill form.

RANKL antibody

Approved by the FDA in 2010, denosumab is a monoclonal antibody that binds and inhibits the effects of RANKL, thereby inhibiting bone resorption through the reduction of osteoclast formation.[9,17,25] There is an increase in BMD in both the hip and lumbar spine.[25] Baseline calcium and vitamin D levels must be obtained before initiating therapy as denosumab reduces serum calcium levels.[25] Denosumab is administered subcutaneously every 6 months.[17]

Calcitonin

Released by the thyroid gland, calcitonin inhibits osteoclasts, thereby reducing bone resorption. Calcitonin is recommended in females who are 5 years postmenopausal with low bone density.[17] Its use is reserved for those who cannot tolerate bisphosphonate or when bisphosphonates are contraindicated.[17] Calcitonin is typically given with calcium and vitamin D as a subcutaneous injection or intranasal spray once daily.[17]

Romosozumab

Romosozumab is classified as an antisclerostin monoclonal antibody that increases bone formation and decreases bone resorption.[17,25] It is recommended for postmenopausal females at high risk for fractures and severe osteoporosis.[17,25] It is effective in improving bone density in the spine and hip.[25] Romosozumab is contraindicated in patients with a history of myocardial infarction or stroke within the preceding year and carries a boxed warning for

Case study: Baseline DXA

Category	T-score	Interpretation
Spine	+0.6	Normal
Total hip	-0.6	Normal
Femoral neck	-1.0	Osteopenia
FRAX assessment		Major osteoporotic fracture risk: 6.5% Hip fracture risk of 0.4%

Case study: 2-year DXA

Category	T-score	Interpretation
Spine	-0.7	Normal
Total hip	-1.1	Osteopenia
Femoral neck	-1.2	Osteopenia
FRAX assessment		Major osteoporotic fracture risk: 6.9% Hip fracture risk of 0.5%

cardiovascular adverse events.[17] It is administered as a subcutaneous injection every month for a period of 12 months.[17]

Anabolic agents

Teriparatide

A synthetic form of parathyroid hormone, teriparatide stimulates bone formation. It is reserved for treating severe osteoporosis when patients continue to sustain fractures with other pharmacologic treatments or are intolerant of other medications.[25] Serum calcium and parathyroid hormone levels must be closely monitored while a patient receives teriparatide. Additionally, teriparatide is given for 2 years in a patient's lifetime but may be given longer if there is a continued fracture risk.[4] Teriparatide is given as a daily subcutaneous injection.

Case study

MB is a thin, White, 64-year-old, postmenopausal female diagnosed with invasive carcinoma of the right breast. The pathology report indicated that the breast cancer cells were positive for estrogen and progesterone and negative for HER2 protein receptors.

Postoperative management to prevent breast cancer recurrence included Letrozole (Femara), an aromatase inhibitor. While lowering cancer recurrence, aromatase inhibitors are a known risk factor for accelerated bone loss. Studies show that bone loss in individuals using long-term aromatase inhibitors is double that of normal postmenopausal women of the same age.[26]

Risk factors for osteoporosis in MB include gender, increased age, White race, diabetes mellitus type 2, low BMI, and a newly prescribed aromatase inhibitor. A baseline DXA scan during the cancer diagnosis demonstrated a normal BMD (see *Case study: Baseline DXA*).

Preventive osteoporosis treatment was initiated. Management included using calcium supplements (500 mg/day), calcium-rich foods, vitamin D3 (cholecalciferol 2,000 international units/day), and weight-bearing activities three to five times weekly.

Two years later, another DXA scan was completed (see *Case study: 2-year DXA*).

Despite activity, dietary interventions, and supplements, the DXA scan reported osteopenia with a significant decrease in BMD. As a result, Risedronate (Actonel) 150 mg, one tablet monthly, was prescribed. MB was educated on taking risedronate monthly, first thing in the morning at least 30 minutes before the first food or drink of the day, with 8 oz of water while remaining in an upright position for 30 to 60 minutes after taking it to help prevent the risk of upper gastrointestinal irritation.

A third DXA scan was completed 2 years later (see *Case study: 4-year DXA*).

The third DXA scan again showed osteopenia with a continued decrease in BMD. As a result, medical management included zoledronic acid 4 mg I.V. over a minimum of 15 minutes every 6 months.

The nurse reviewed preinfusion renal studies, which showed normal blood urea nitrogen, serum creatinine, and creatinine clearance results. The nurse taught MB the need for hydration, including using I.V. fluids and oral hydration during and after the infusion. The nurse also taught MB about the most common adverse reactions of zoledronic acid, including nausea, fatigue, anemia, bone pain, constipation, fever, vomiting, dyspnea, and the rare but serious adverse reaction of osteonecrosis of the jaw.

Following 2 years of zoledronic acid, the DXA scan will be repeated to determine treatment response before initiating further pharmacotherapy.

Conclusion

Osteoporosis is the most common bone disease globally, affecting both males and females. The leading initial symptom of osteoporosis is a fracture. The definitive diagnostic study for osteoporosis is the DXA scan. Treatment of osteoporosis involves lifestyle modifications and pharmacotherapy. Nurses are essential in educating patients on osteoporosis risk factors and prevention and lifestyle modifications. ■

Case study: 4-year DXA

Category	T-score	Interpretation
Spine	-0.7	Normal
Total hip	-1.5	Osteopenia
Femoral neck	-1.3	Osteopenia
FRAX assessment		Major osteoporotic fracture risk: 7.7% Hip fracture risk of 0.8%

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