Rehabilitation of Orthopedic and Rheumatologic Disorders.  
1. Osteoporosis

Peter A.C. Lim, MD  
Baylor College of Medicine and Veterans Affairs Medical Center, Houston, TX 77030

Victoria A. Brander, MD  
Northwestern University Medical School, Chicago, IL 60611

Darryl L. Kaelin, MD  
Indiana University, Indianapolis, IN 46202

Terry H. Oh, MD  
Mayo Clinic, Rochester, MN 55905


This self-directed learning module reviews and summarizes recent literature on osteoporosis. It is part of the chapter on rehabilitation of orthopedic and rheumatologic disorders in the Self-Directed Physiatric Education Program for practitioners and trainees in physical medicine and rehabilitation. The areas covered include pathophysiology of primary and secondary osteoporosis, effects of various pharmacologic treatments on bone metabolism, and the utility of available diagnostic tests. Management strategies for perimenopausal women as compared with postmenopausal women with established osteoporosis are discussed. This is followed by an evaluation and management plan for the older man with acute osteoporotic fracture.

1. Objective.—Relate the risk factors for the development of osteoporosis to its pathophysiology.

Osteoporosis is a disease of impaired mineralization of bone (see table 1). It is classified as primary—type I, postmenopausal, and type II, age related—or secondary, caused by drugs and by conditions such as endocrine disease, malabsorption, metastatic cancer, connective tissue disorders, immobilization, or alcoholism.

Adult bone mass is determined by the rate of bone loss after acquisition of peak bone mass. After cessation of linear growth, bone remodeling is the principal physiologic process. Calcium homeostasis is maintained and bone structural integrity is preserved through resorption by osteoclasts or by collagenous osteoid secretion by osteoblasts which is then mineralized with hydroxyapatite. Because of the effect of androgens on the skeleton and a predisposition for thicker bones, men achieve a much higher average bone density than women. Estrogen deficiency increases the activation rate of new bone remodeling sites, where resorption exceeds formation.

Age-related bone loss of 0.5% to 1% per year begins in both sexes at about age 35 with a slight but persistent elevation in bone resorption. Perimenopausal loss of estrogen triggers accelerated bone resorption at 3% to 5% per year with preferential loss of trabecular bone which leads to increased risk of spine and distal radius fracture. From the fourth decade, decreased intestinal absorption of calcium, reduced 1,25-hydroxyvitamin D levels, and increased parathyroid hormone secretion contribute to imbalances in bone remodeling. After estrogen-dependent bone loss ceases, age-related loss of cortical and trabecular bone mass (with increased hip fractures) continues until death.

Genetics (race, sex, build, skin type), genetic disease (eg, Turner’s syndrome, osteogenesis imperfecta, homocystinuria, Ehler-Danlos syndrome), calcium intake, lack of physical activity, late menarche, and hypogonadism (exercise, anorexia nervosa, hyperprolactinemia, idiopathic) affect peak bone mass. Enumeration of risk factors for osteoporosis has been outlined elsewhere.

1.2 Objective. —Describe the effects on bone metabolism of pharmacologic treatments for osteoporosis.

TYPES OF TREATMENT

1. Calcium

Calcium is mostly found in bone; 1% appears in other tissues, extracellular fluid, and blood. Calcium is absorbed in the upper intestine by a vitamin D-dependent mechanism, passive diffusion, and kidney reabsorption. Calcium is excreted by the kidneys, with excess calcium being eliminated in the feces. Bone resorption occurs during calcium deprivation. Sufficient dietary calcium intake is critical during skeletal growth; the risk of adverse events, such as nephrothiasis, is low. Pharmaceutical calcium should be used when dietary intake is inadequate. Recommendations for daily calcium intake are 1200mg for premenopausal women, 1500mg for postmenopausal women, 1500mg during pregnancy, and 1500mg during growth years (ages 11 to 20 years).

2. Vitamin D

Vitamin D increases calcium absorption in the gastrointestinal tract. Deficiency of vitamin D may lead to secondary hyperparathyroidism, increased bone resorption, and fractures. For most persons, 400 to 800IU of vitamin D a day is...
Therapy should be monitored with regular screening mammography, breast examination, and endometrial surveillance. In doses of 30 to 150mg/day, it increases bone mineral density, decreases bone turnover, and lowers serum total and low-density lipoprotein cholesterol without endometrial stimulation in early postmenopausal women. It is not believed to increase the risk of endometrial pathology, and it has cardioprotective effects.

### Fluoride

Fluoride is the most potent stimulator of osteoblastic bone formation known. It increases spinal bone mass 5% to 10% yearly. Sodium fluoride may have a role in the treatment of established osteoporosis, and a slow-release version may be effective in reducing fractures, with fewer side effects (gastrointestinal and musculoskeletal toxicities) than earlier forms. Intermittent therapy with a break of 2 months every 6 months may be advantageous. However, further studies are necessary.

### Calcitonin

Calcitonin inhibits osteoclastic activity and thus decreases bone resorption. By stimulating endogenous opioids, calcitonin may reduce the pain of acute osteoporotic fractures. It may reduce postmenopausal bone loss and slowly increase bone density. It is used as an alternative for patients who are unable to take hormone replacement therapy or alendronate. Development of autoantibodies may limit its use to periods of 6 to 12 months.

### Bisphosphonates

Bisphosphonates such as etidronate and alendronate bind permanently to mineralized bone surfaces and inhibit subsequent osteoclastic activity. They are bone specific, have no known carcinogenesis, and have antiresorptive efficacy equivalent to or greater than that of estrogens. Gastrointestinal side effects, including esophageal erosions, limit its use in persons with known gastroesophageal (particularly reflux) disease. Alendronate increases bone density by 5% to 9% a year, reduces fracture risk regardless of age or bone mass, and is an alternative for women who cannot or are unwilling to undergo hormone replacement therapy. It can reduce osteoporotic fracture risk at the spine, hip, and wrist by 50%. Alendronate also increases bone mineral density in patients with, or at risk for, glucocorticoid-induced osteoporosis.

### Table 1: World Health Organization Classification of Osteoporosis

| Bone mineral density (BMD) readings from lumbar spine or femoral neck BMD for young adult | Normal: Not more than 1 SD below | Osteopenia: Between 1 and 2.5 SDs below (2-fold fracture risk increase) | Osteoporosis: More than 2.5 SDs below (4- to 5-fold fracture risk increase) | Severe osteoporosis: More than 2.5 SDs below, in presence of one or more fragility fractures (20-fold fracture risk increase) |

**Types of Diagnostic Tests**

**Plain Radiography**

A loss of mineralized bone of 30% to 50% is necessary before osteopenia becomes apparent on radiographs. Plain radiography is useful for differentiating other and potentially
misleading abnormalities, such as osteoarthritis and aortic calcification.

**Single Photon Absorptiometry (SPA) and Dual Photon Absorptiometry (DPA)**

These tests are based on differential absorption of photons through bone compared with adjacent soft tissue. They do not accurately predict bone mass and are no longer routinely used.

**Dual-Energy X-ray Absorptiometry (DEXA or DXA Scan)**

DEXA utilizes an x-ray source of photons and is the most widely used and accepted method of investigation for osteoporosis in clinical practice. Its advantages include a high degree of accuracy and reproducibility, adequate sensitivity for use in yearly follow-up, low radiation dose comparable to DPA, and accessibility. However, it does not distinguish trabecular from cortical bone, and abnormal spine anatomy/extraspinal calcifications may falsely elevate measurements. DEXA scans are recommended for measuring bone mineral density at the lumbar spine and femoral neck every 2 to 4 years for patients receiving hormone replacement therapy, and every 1 to 2 years for those taking bisphosphonates. The cost of this procedure is now reimbursed by Medicare.

**Quantitative Computed Tomography (QCT)**

QCT assesses bone density by comparing the trabecular bone center of the vertebral body on axial computed tomography (CT) images with the CT density of a phantom with known hydroxyapatite concentration. It differentiates trabecular (initial loss in postmenopausal period, hence a means of detecting early osteoporosis) and cortical bone. Operator dependence, radiation dose, and cost limit its use.

**Ultrasonography**

Quantitative ultrasonography of bone is a recent radiation-free technique that measures bone mass and may also be useful for assessing bone micro-architecture. Low BUA (broadband ultrasound attenuation) of the calcaneus predicts increased risk for hip and all non-spine fractures. Ultrasonography is appealing for its lower cost, portability, and lack of ionizing radiation.

### 1.4 Objective

Differentiate management strategies for a 50-year-old woman concerned about osteoporosis prevention from treatment in a 75-year-old woman with osteoporosis of the spine and kyphosis.

All women should receive prophylactic measures to prevent bone loss. The recommendations are for calcium, 400 to 800IU a day of vitamin D, and regular exercise. Women should be counseled to eliminate smoking, reduce use of alcohol and possibly caffeine, and minimize use of medications (eg, steroids, aluminum, excessive thyroxine) that contribute to bone loss. Hormone replacement therapy for at least 7 years after menopause is recommended unless contraindications exist. Alendronate, 5mg a day, is an alternative for osteoporosis prevention in women who are unwilling or unable to use hormone replacement therapy. Exercise is an important prevention and treatment strategy. Increasing bone mass early in life will reduce osteoporosis later. Thirty minutes or more of physical activity of moderate intensity on most days of the week is recommended. Weight bearing (eg, walking, jogging, dancing, sports) and muscle-strengthening (including weight-lifting) exercises are effective for osteoporosis prevention and overall health at all ages.

Treating established osteoporosis in a 75-year-old woman includes all of the above and also measures to prevent osteoporotic fractures, incorporating physiatric therapeutics such as flexibility, strengthening, various exercises, modalities, and fall-management strategies. The standard pharmacologic management is similar to that for osteoporosis prevention, except that use of alendronate is recommended at a dosage of 10mg daily.

Postural training is particularly important in patients with compression fractures in the thoracic spine, kyphosis, back and shoulder pain, protuberant abdomen, and sleep difficulty. Education in proper sitting and standing posture, proper biomechanics during activities of daily living, and use of extension orthoses and training devices such as a postural training support may be effective. When vertebral compression fractures are present, flexion exercises should be avoided. Gentle back extension, abdominal and lower body muscle-strengthening exercises, and pelvic tilts should be done regularly.

Tinetti and colleagues have associated falls with the use of sedatives, cognitive impairment, disability of lower extremities, presence of a palpmoment reflux, balance and gait dysfunction, and foot problems. Environmental and other lifestyle hazards should be assessed. Low-impact forms of aerobic exercise (eg, walking, bicycling, and swimming) improve endurance and cardiovascular fitness. Exercises that improve balance, such as Tai Chi and the Tinetti group’s progressive competency-based balance and strengthening exercises, have been reported to reduce falls.
Ambulation aids may improve safety and balance. Use of reachers and long-handled equipment minimizes forward flexion during activities of daily living and subsequent falls.4

1.5 Objective.—Devise an evaluation and management plan for a 65-year-old man with an acute T12 compression fracture.24

Osteoporosis in men is not uncommon. One-seventh of all osteoporotic vertebral compression fractures occur in men. The differential diagnosis of male osteopenia is shown in table 3.

A complete history and physical examination are mandatory in men who present with osteoporosis or osteopenia. Long-standing testosterone deficiency is common in men presenting with spinal osteoporosis in their 6th decade. Levels of serum testosterone and luteinizing hormone levels should be obtained in the workup of men with osteopenia or osteoporosis. Hypocalcemia, hypophosphatemia, and an elevated alkaline phosphatase level may be present in men with osteomalacia. Investigation of the serum 1, 25-hydroxyvitamin D level is indicated if vitamin D malabsorption is suspected. Vertebral radiographs, magnetic resonance imaging, or bone scan can exclude focal tumor masked as a compression factor. Serum protein electrophoresis will exclude multiple myeloma in most cases. Liver and renal function tests, complete blood count, and erythrocyte sedimentation rate exclude the presence of chronic systemic disease. Thyroid-stimulating hormone assay will rule out hyperthyroidism. Prostate-specific antigen testing should be performed if it was not obtained within the previous year.

Treatment includes calcium supplementation at 1500mg daily, increasing daily vitamin D intake to 800IU (or higher if vitamin D deficiency is present), and supplementing testosterone if testosterone deficiency is identified. Alendronate therapy is indicated, although its efficacy in men has not been established. Calcitonin (nasal or injectable) may be useful to reduce pain from a vertebral fracture. After an acute spinal compression fracture, nonsteroidal anti-inflammatory drugs, narcotic analgesics, and muscle relaxants should be used to relieve pain. Thoracic orthoses, such as the CASH hyperextension orthosis, an abdominal binder, or a light-weight molded polyethylene orthosis (eg, "warm and form" brace), may also help relieve pain.

Physical modalities such as ice or heat are helpful. The patient should be instructed to remain fairly sedentary until the pain begins to improve. Early physical therapy focuses on patient education regarding comfortable and safe positions in which to rest or sit, modalities to help alleviate pain, and a program of gradually increasing back-extension exercises. An ongoing, preferentially weight-bearing, exercise program will contribute to restoration of bone mass.

References


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**Table 3: Differential Diagnosis of Male Osteoporosis**

<table>
<thead>
<tr>
<th>Category</th>
<th>Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endocrinopathies</td>
<td>Hypogonadism, Cushing’s disorders, hyperthyroidism, primary hyperparathyroidism, hyperprolactinemia, acromegaly, hypercalcemia</td>
</tr>
<tr>
<td>Osteomalacia</td>
<td>Vitamin D deficiency, phosphate wasting syndromes, metabolic acidosis, inhibitors of mineralization</td>
</tr>
<tr>
<td>Neoplastic Diseases</td>
<td>Multiple myeloma, systemic mastocytosis, diffuse bony metastasis, myelo- and lymphoproliferative disorders</td>
</tr>
<tr>
<td>Drug-Induced</td>
<td>Glucocorticoids, ethanol, excessive thyroid hormone, hepatic, anti-convulsants, tobacco</td>
</tr>
<tr>
<td>Hereditary Disorders</td>
<td>Osteogenesis imperfecta, Ehlers-Danlos and Marfan syndromes, homocystinuria</td>
</tr>
<tr>
<td>Other Conditions</td>
<td>Immobilization, chronic diseases, malnutrition, skeletal sarcoïdosis, Gaucher’s disease, hemoglobinopathies, hypophosphatasia</td>
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<tr>
<td>Idiopathic</td>
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