

Osteoporosis

Definition and Clinical Presentation

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Osteoporosis is a skeletal condition characterized by decreased density (mass/volume) of normally mineralized bone. The reduced bone density leads to decreased mechanical strength, thus making the skeleton more likely to fracture. Postmenopausal osteoporosis (Type I) and age-related osteoporosis (Type II) are the most common primary forms of bone loss seen in clinical practice. Secondary causes of osteoporosis include hypercortisolism, hyperthyroidism, hyperparathyroidism, alcohol abuse, and immobilization. In the development of osteoporosis, there is often a long latent period before the appearance of the main clinical manifestation, pathologic fractures. The earliest symptom of osteoporosis is often an episode of acute back pain caused by a pathologic vertebral compression fracture, or an episode of groin or thigh pain caused by a pathologic hip fracture. In the diagnostic process, the extent and severity of bone loss are evaluated and secondary forms of bone loss are excluded. A careful diagnostic work-up that includes clinical history, physical examination, laboratory evaluation, bone densitometry, and radiographic imaging will allow the clinician to determine the cause of osteoporosis and to institute medical interventions that will stabilize and even reverse this frequently preventable condition. [Key words: osteopenia, osteoporosis, postmenopausal] *Spine* 1997;22:12S-16S

In this definition, it is recognized that there is a strong association between bone mineral density and the likelihood of fracture.^{4,22,28} According to the criteria, approximately 0.6% of young women have osteoporosis and approximately 16% have low bone mass. By age 75, an estimated 38% of white women will have osteoporosis and 94% will have low bone mass.^{20,21,25,26} Although the definition is useful for establishing the prevalence of osteoporosis, it is inadequate as a guide to treatment, because other factors influence bone quality and fracture risk. Treatment should be determined for each patient after consideration of these other factors in addition to bone density. Because the risk of osteoporotic fracture during the remaining lifetime of many elderly patients is sufficiently low, aggressive treatment is usually not needed. Conversely, many patients who do not meet the World Health Organization's criteria for osteoporosis might have other risk factors and circumstances that justify treatment. Therefore, the World Health Organization's criteria has made physicians aware of the prevalence of osteoporosis but should not be used to dictate absolute thresholds for diagnosis and treatment.

Osteoporosis has been classified into two categories, primary and secondary. Primary osteoporosis is further divided into three types—postmenopausal osteoporosis (Type I), age-related osteoporosis (Type II), and idiopathic osteoporosis. Postmenopausal (Type I) osteoporosis develops in women who have estrogen deficiency, whereas age-related (Type II) osteoporosis occurs in men and women as their bone density decreases with aging. Secondary osteoporosis refers to those patients in whom a causative factor or disease process is identifiable.¹⁸

Osteoporosis is less common in men than in women, probably reflecting that men have greater bone mass than women at all ages, and experience no physiologic equivalent of menopause.²⁴ Nonetheless, severe Type II (age-related) and idiopathic osteoporosis occurs in men. Secondary osteoporosis caused by excessive alcohol intake, hypogonadism, hypercortisolism, and hyperthyroidism also occur in men and can lead to varying degrees of clinically significant osteoporosis.^{7,9,12,13,16}

■ Clinical Signs and Symptoms

In osteoporosis, as in hypertension, there is often a long latent period before clinical symptoms or complications develop. The most prevalent sequelae are compression

Osteoporosis is a skeletal condition characterized by decreased density (mass/unit volume) of normally mineralized bone. The reduced density impairs the mechanical strength of the bone, thus making it more vulnerable to fracture.

The World Health Organization has established diagnostic criteria for osteoporosis that are based on bone density measurements determined by dual-energy x-ray absorptiometry (DXA). A patient is classified as having low bone mass if the bone mineral density measures between 1 and 2.5 standard deviations below the mean value in a young reference population. The diagnosis of osteoporosis is made if a patient's bone density is 2.5 standard deviations or more below the mean for young normal people² (Table 1).

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Acknowledgment date: June 17, 1997.

Acceptance date: June 17, 1997.

Device status category: 1.

Table 1. World Health Organization Diagnostic Criteria for Osteoporosis

Group	Diagnostic Criteria
Normal	Bone mineral density within 1 SD of the mean of a young adult reference population
Osteopenia—low bone mass	Bone mineral density between 1.0 and 2.5 SD below the mean of a young adult reference population
Osteoporosis	Bone mineral density 2.5 or more SD below the mean of a young adult reference population
Severe osteoporosis	Osteoporosis with one or more fragility fractures

fractures of the vertebral bodies and fractures of the ribs, proximal femurs, humeri, and distal radiuses.

Pathologic fractures are among the most obvious clinical manifestations of osteoporosis.^{14,17} In patients with osteoporosis, as well as in older persons, fractures are often the result of a fall.^{11,29} Results reported in recent studies on falls in the elderly have identified numerous predisposing factors. Intrinsic causes include neurologic and musculoskeletal disorders, cardiovascular disorders, and visual disturbances, all common in this population. Extrinsic factors that increase the risk of falls are the use of sedatives, excessive use of prescription medications, dim lighting, cluttered floors, and various other obstructions (scatter rugs, curbs, and stairs).^{21,23,29–31} Reduced resistance to trauma caused by a decrease in soft tissue padding that can help absorb and deflect the kinetic energy at sites of impact, as well as low bone mass, may contribute to the high incidence of fracture. Hip fractures in thin patients are probably related to decreased resistance and low bone mass. Therefore, persons with ample body fat or well-developed muscles are less likely to sustain a fracture during a fall.¹⁴

Vertebral Compression Fractures

The earliest symptom of osteoporosis is often an episode of acute back pain occurring when the person is at rest or during such routine activity as bending, standing from a seated position, lifting a heavy object, or opening a window. Although most compression fractures are painless, pain can occur suddenly. Most patients can recall the exact moment the pain began but may have difficulty identifying the vertebral site involved. Spinal movement is severely restricted, with flexion reduced more than extension. Pain intensifies with sitting or standing and is relieved by bed rest in the fully recumbent position. Coughing, sneezing, and straining to move the bowels can exacerbate the pain. Sitting or standing for prolonged periods may be impossible because of severe pain. The patient walks slowly, but the gait is otherwise normal.

Anterior compression fractures in the thoracic spine may cause thoracic kyphosis (dowager's hump), the stooped posture characteristic of osteoporosis. Loss of

vertebral height is usually insidious and painless and is accompanied by loss in height of the intervertebral discs. Involvement of the lumbar spine may lead to progressive loss of the normal lumbar lordosis. Axial height decreases after each fracture, and there is a discrepancy between the standing height and arm span. Patients with severe, progressive spinal compression may have an acquired short trunk and short stature. This is easily identified with the patient in the standing position. Normally the finger tips should come to the mid thigh. In advanced osteoporosis, with loss of axial height, the finger tips come to the lower thigh or knee when the patient is standing. Once the spine has collapsed to the point at which the lower ribs rest on the iliac crest, height remains stable, although bone density may continue to diminish.

After acute vertebral fractures, spasms of the paravertebral muscles are palpable and often visible. The spine and paravertebral muscles may be tender to deep palpation and to percussion at the level of the fracture.

Acute fractures are usually not associated with abnormal neurologic findings, in that they are usually stable injuries. When present, radiculopathy, can cause unilateral or bilateral pain that radiates along the costal margin of the affected spinal nerve. Involvement of the spinal cord or cauda equina is extremely uncommon, and should suggest other conditions, including infection, metastatic or primary bone tumors, myeloma, Paget's disease, or lymphoma.

During intervals between compression fractures, most patients remain pain free. However, some patients continue to be plagued by dull, aching back pain, especially with prolonged standing. This pain can often be relieved with intermittent bed rest throughout the day.

It is important to distinguish chronic back pain from the incapacitating pain of temporally clustered fractures. For a significant number of patients with cluster fractures, the severe pain initiated by the first vertebral compression fracture barely subsides before the occurrence of equally severe pain with the next fracture. Typically, these patients will have multiple fractures in a period of months, followed by gradual recovery. Such patients are able to recall each exacerbation and tend to have more severe pain of longer duration than those with isolated compression fractures. When cluster fractures are suspected in a patient, evaluation for secondary causes of osteopenia is warranted. Exacerbation of a preexisting chronic illness in a severely osteopenic, steroid-dependent patient, or an increase in the glucocorticoid medication often precipitates temporal clustering of fractures.¹⁵

Some permanent side effects of progressive vertebral compression fractures are related to decreases in the size of the thoracic and abdominal cavities. Postural changes diminish exercise tolerance. After ingesting even small amounts of food, the patient often feels full and bloated. Severe vertebral collapse in the lumbar spine causes the abdomen to protrude. Circumferential pachydermal skin folds may develop at the rib and pelvic margins as the spinal deformity progresses.

Table 2. Results of Common Laboratory Studies Used for the Diagnosis of Osteoporosis and Other Metabolic Bone Disorders

Disorder	Ca ²⁺	PO ₄ ⁻³	PTH	AP	25-(OH) Vitamin D ₃	1,25-(OH) ₂ Vitamin D ₃	Urinary Calcium
Early postmenopausal osteoporosis	N	N	N, ↓	N	N	N	↑
Age-related osteoporosis	N	N	N, ↑	N	N	N, ↓	N
Primary hyperparathyroidism	↑	↓	↑↑	↑, N	N	↑, N	↑
Hyperthyroidism	N, ↑	N	N, ↓	N	N	N	↑
Glucocorticoid-induced osteoporosis	N	N	↑, N	N	N	N	↑
Multiple myeloma	↑	↑, N	↓	N	N	N, ↓	↑↑
Metastatic disease	↑	↑, N	N, ↓	↑, N	N	N, ↓	↑↑↑
Vitamin D-resistant osteomalacia	N	↓	N	↑	N	N, ↓	N
Severe vitamin D-deficiency osteomalacia	↓	↓	↑↑↑	↑	↓↓	↓	↓
Severe disuse osteoporosis	N, ↑	N, ↑	N, ↓	N	N	↓	↑↑
Hypophosphatasia	↑	↑	N, ↓	↓↓	N	N	↑

AP = alkaline phosphatase; N = normal.

Appendicular Fractures

In some persons, osteoporosis is first manifested by a pathologic fracture of the proximal femur or distal radius, sustained after a fall. The incidence of fractures of the femoral neck increase with age.¹⁹ Fractures of the proximal femur are among the most feared complications of osteoporosis and are solely responsible for catapulting the disease into the category of a life-threatening disorder. These fractures often occur in patients with several preexisting comorbidities that contribute to more complicated postoperative recovery, including pneumonia, deep vein thrombosis, and fat embolus syndrome. Although reduced bone density is a critical component leading to a fractured hip, other intrinsic and extrinsic factors—cardiac disease, neurologic disorders, and medications that cause dizziness—may be equally important.

Patients typically complain of hip pain and the hip's inability to bear weight. Physical examination reveals a shortened, externally rotated leg. In cases of occult fractures, the patient complains of severe pain when the hip is in a weight-bearing position. Occult hip fractures can be observed in patients who have risk factors for osteoporosis and tend to be more active. Magnetic resonance imaging or a bone scan is often useful in diagnosing occult fractures.

■ Diagnostic Evaluation

The diagnostic work-up of osteoporosis focuses on evaluating the cause and magnitude of bone loss and on excluding secondary causes of bone loss. In many patients, the diagnosis of osteoporosis is made only after a pathologic fracture has occurred. To avoid the potentially devastating effects of osteoporosis, it may be clinically warranted and cost-effective to assess bone density in patients at high risk before fractures or deformities occur. These low-cost, usually available techniques are valuable diagnostic tools. Serial bone density measurements are extremely useful for monitoring the effectiveness of therapy or preventive interventions.

History and Laboratory Studies

Postmenopausal osteoporosis in women and age-related osteoporosis in men and women are the most common forms of symptomatic bone loss seen in clinical practice. A detailed history, however, may suggest that the low bone density is secondary to hyperthyroidism, primary hyperparathyroidism, hypercortisolism, myeloma, or osteomalacia (Tables 2 and 3).

Risk factors for low bone density have limited value in estimating a person's actual bone density.²⁷ However, determining risk factors for fracture can be useful in identifying those at high risk, and treatment can be initiated to reduce the risk.⁶ In women, several common, important, and clinically useful risk factors have been identified recently in the Study of Osteoporotic Fractures. These include low bone mineral density; history of a fracture after age 40; history of a fracture of the hip, wrist, or vertebra in a first-degree relative; or current cigarette smoking.⁵

Obtaining a thorough history facilitates selection of appropriate baseline tests. Routine laboratory tests include complete blood count with leukocyte differential measurement; a 24-hour urine collection to measure calcium and creatinine excretion; and determination of serum levels of calcium, albumin, phosphorus, alkaline phosphatase, blood urea nitrogen, and creatinine (Table 2). In asymptomatic postmenopausal osteoporosis, results of routine laboratory tests are normal and do not assess the extent or rate of bone loss or indicate the prognosis. Even in severe postmenopausal osteoporosis, the serum levels of calcium, inorganic phosphorus, and alkaline phosphatase are usually normal, although alkaline phosphatase levels may rise transiently for several weeks after a fracture. Measurement of biochemical markers appears helpful in assessing bone turnover and aids in monitoring therapy. Total alkaline phosphatase, osteocalcin, Type I collagen propeptides, urinary collagen cross-links, and collagen telopeptides are several

Table 3. Clinical Evaluation for Common Causes of Secondary Osteoporosis

Suspected Disease Process	Clinical Clues	Important Laboratory Studies
Hyperthyroidism	Tachycardia, tremor, proximal weakness, pathologic fractures, goiter	↓ TSH, ↑ FTI, TRH stimulation test can be used only if other tests are inconclusive
Primary hyperparathyroidism	↑ thirst, ↑ urination, lethargy, depression, bone pain, weakness, abdominal pain, kidney stones, pathologic fractures	↑ PTH, ↑ Ca ²⁺ , ↓ PO ₄ ³⁻
Hypogonadism	Abnormal sexual and menstrual history, secondary sex characteristics, body habitus, anemia, pathologic fractures	Testosterone, FSH, LH, estrogen, estrogen receptors
Multiple myeloma	Bone pain, ill appearing, pathologic fractures	Anemia, ↑ Ca ²⁺ , proteinuria, ↑ Cr/BUN, protein electrophoresis (M-spike); Bence-Jones proteins in urine
Excessive alcohol intake	Jaundice, spider angiomas, "CAGE" questions, ⁷ tremors, pathologic fractures	LFTs, ↑ PT, ↓ albumin level
Hypercortisolism	Hypogonadism, truncal obesity, ↑ skin pigmentation, ↑ bruising, pathologic fractures	Dexamethasone suppression test, ↑ ACTH, ↓ K, ↓ Cl, ↑ Glu
Osteomalacia	Chronic renal failure, malabsorption, bone pain, tenderness, myopathy, symmetric pathologic fractures	25-hydroxy vitamin D, 1,25-dihydroxy vitamin D, product of Ca ²⁺ and phosphate <25

Ca²⁺ = calcium; Cr = creatinine; BUN = blood urea nitrogen; LFTs = transaminases (ALT, AST, LDH, GGTP), alkaline phosphatase; K = potassium; Cl = chloride; FSH = follicle stimulating hormone; Glu = glucose; FTI = free thyroxine index; ACTH = adrenocorticotropic hormone; LH = luteinizing hormone; TRH = thyrotropin-releasing hormone; PT = prothrombin time; PTH = pituitary hormone; PO₄³⁻ = inorganic phosphate.

markers that may be useful. A complete description of these tests is summarized by Erye in this issue.^{1,3,8,10}

Additional tests are warranted if bone loss caused by conditions other than aging and menopause is suspected. Diagnosis of primary or iatrogenic hyperthyroidism requires measurement of serum triiodothyronine resin uptake (T₃RU), and thyroxine (T₄) levels, and calculation of the free thyroxine index (FTI). Serum thyroid-stimulating hormone (TSH) levels are markedly suppressed in all forms of hyperparathyroidism and are a sensitive indicator of thyroid status. Serum protein electrophoresis, urinary immunoelectrophoresis and bone marrow aspirate may be needed to detect multiple myeloma. In patients who have hypercalcemia, parathyroid hormone levels must be determined. The serum level of 25-hydroxyvitamin D, an excellent indicator of total body reserves of vitamin D, may be measured to evaluate a possible vitamin D deficiency, the most common biochemical abnormality associated with hip fractures. All men with osteopenia or osteoporosis should have an evaluation of serum testosterone level, in that asymptomatic hypogonadism is a common cause of osteopenia in men.

Differential Diagnosis

Osteoporosis and osteomalacia are commonly confused osteopenic conditions in adults. Whereas osteoporosis is characterized by a decreased density of normally mineralized bone matrix, osteomalacia is a qualitative rather than a quantitative disorder of bone metabolism. In osteomalacia, bone density may be increased, normal, or (most commonly) decreased, and bone matrix is insufficiently mineralized.

Unlike its more easily recognized childhood counterpart, rickets, adult osteomalacia may be difficult to diagnose clinically. The incidence is evenly distributed throughout all age groups. The most common causes are

chronic renal failure, malabsorption, vitamin D deficiency, abnormalities of the vitamin D pathway, and hypophosphatemic syndromes. Rarer causes are renal tubular acidosis, aluminum intoxication, and hypophosphatasia.

In contrast to osteoporosis, which does not become evident until fractures occur, osteomalacia may cause generalized bone pain, tenderness, and generalized myopathy. Osteomalacia caused by vitamin D deficiency is suggested by bone pain or pathologic fracture in a patient taking anticonvulsants, by a history of malabsorption syndrome in a patient, or by a femoral neck fracture in an older patient. The levels of 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D must be determined when osteomalacia is suspected. The diagnosis can be confirmed with fluorescent microscopic examination of non-decalcified trabecular bone tissue obtained by transiliac bone biopsy after administration of time-separated double tetracycline labeling.

Although the radiographic features of osteoporosis and osteomalacia may be similar, axial changes predominate in osteoporosis, whereas appendicular changes predominate in osteomalacia. Osteomalacia is suggested by symmetric pathologic fractures and traumatic fractures. Pseudofractures (Looser zones) are characteristic of osteomalacia. These small, incomplete cortical fractures develop perpendicular to the long axis of a bone and are often bilaterally symmetric. Common areas of involvement include medial borders of the scapulas, ribs, ischiopubic rami, femoral necks, lateral borders of the femur, and distal radius.

Results of routine laboratory studies, typically normal in osteoporosis, may be abnormal in osteomalacia. Osteomalacia should be suspected when the product of the serum calcium level and serum phosphate level is chron-

ically below 25 (with normal serum albumin), especially if accompanied by an elevated bone-specific alkaline phosphatase level and a urinary calcium excretion of less than 50 mg per 24 hours.

■ Conclusions

The effectiveness of current treatment methods for osteoporosis relies on the accurate diagnosis and classification of the disease process that results in low bone density and fractures. A careful diagnosis that is based on clinical history, physical examination laboratory evaluation, bone densitometry, and radiographic imaging will allow the clinician to enact preventive measures and medical interventions that can even reverse this frequently preventable disorder.

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