

ENTITLEMENT ELIGIBILITY GUIDELINES OSTEOPOROSIS

MPC 01303
ICD-9 733.0

DEFINITION

Osteoporosis is a disease of bone characterized by a reduction in bone density while maintaining the normal ratio of mineral to organic content. This decreased bone density leads to an increased risk of fracture.

Osteoporosis, by World Health Organization (WHO) consensus, is considered to be present when bone mineral density is equal to or greater than 2.5 standard deviations below the mean bone density of young adult sex matched controls, or there is radiological evidence of osteoporotic fracture and reduced bone density in the region of the fracture.

If the result is less than 2.5 standard deviations below the mean, a diagnosis of osteoporosis is not present.

“Osteopenia” is a generic term for a general reduction in bone mass. It includes the conditions of osteoporosis and osteomalacia (softening of the bones due to impaired mineralization, with pain, tenderness, muscular weakness, anorexia, and loss of weight).

Where osteoporosis is secondary to a primary disease state, it may be claimed as part of the primary disease state or claimed as consequential to the disease state, depending on the circumstances.

DIAGNOSTIC STANDARD

Diagnosis by a qualified medical practitioner is required. Bone densitometry and/or x-ray showing osteoporotic compression/ fracture must be provided.

ANATOMY AND PHYSIOLOGY

A distinguishing characteristic of osteoporosis is loss of bone mineralization and bone matrix resulting in thinner bone.

The skeleton functions to provide structural support for muscles and organs, and to serve as a depot for the body's calcium and other essential minerals. The skeleton contains two types of bone: cortical bone (80%) which is concentrated in the appendicular skeleton and femoral neck, and the more metabolically active trabecular bone (20%), which is located in the spine, epiphysis, and pelvis. Bone consists of organic matrix and an inorganic mineral component (calcium and phosphate).

Bone is constantly being broken down and reformed in order for growth, repair, and maintenance of the body to occur. Bone resorption and production is associated with the delicate balance of the actions of 2 types of cells found in body tissues. Osteoclast cells mediate bone resorption and are activated by concentrations of calcium, phosphorus, vitamin D and parathyroid hormone, as well as other systemic hormones. Estrogen, estrogen receptor modulators, calcitonin, and biphosphonates are factors that inhibit bone resorption. Osteoblast cells make up the part of bone that undergoes mineralization. Under steady-state physiologic circumstances, the rate of osteoclast-mediated bone resorption is equal and coupled with osteoblast-stimulated bone formation.

The development of, or resistance to, osteoporosis is determined by this balance between bone resorption and formation. Local factors in bone that affect this balance include prostaglandins, insulin-like growth factors, interleukins (IL-1, IL-6, and IL-11), tumor necrosis factor (TNF), and transforming growth factor. Osteoporosis is developed when there is a reduction in the amount of bone mass. Cortical bone becomes more porous and thin, while the structure of trabecular bone is impaired. This makes the bone weaker and more likely to fracture.

The two **primary types** of osteoporosis for pension purposes are Type I and Type II. Type I, or high-turnover osteoporosis, occurs in some women between the ages of 50 and 75 because of sudden postmenopausal decreases in estrogen levels. This results in a rapid depletion of calcium from the skeleton. Type II, or low-turnover osteoporosis, results when the process of resorption and formation of bones is no longer coordinated and bone breakdown overcomes bone-building. This occurs with age in everyone to some degree. The determining factor for the diagnoses of either Type I or Type II osteoporosis is the amount of calcium left in the bone.

A third type of osteoporosis is known as **secondary osteoporosis** and occurs secondary to certain diseases (e.g. chronic liver failure, hyperthyroidism, and renal failure) and to certain medications (e.g. corticosteroids, chronic heparin therapy).

CLINICAL FEATURES

Bone density normally reaches its peak after puberty and into the third decade of life. At menopause, there is an acceleration of bone loss. Factors that seem to confer the greatest risk of osteoporosis include low body mass index, previous fracture at less than 50 years of age, family history of osteoporosis, and current smoking. Other determinants related to the development of osteoporosis include non-modifiable risk factors such as advanced age, white race, and dementia.

Modifiable risk factors include, but are not limited to, low calcium intake, eating disorder, low testosterone levels (men), premenopausal estrogen deficiency, amenorrhea for more than 1 year or menopause at less than 45 years, excessive alcohol intake, frail or poor health, physical inactivity, neurologic disorders, and lack of sunlight exposure.

Osteoporosis usually becomes advanced before symptoms appear. It may present as a fracture of a vertebra, hip, forearm, or any bony site if sufficient bone mass is lost. Fractures frequently occur after apparently minor trauma such as bending over, lifting, jumping, or falling from the standing position. In the later stages of the disease, pain, disfigurement and debilitation are common. Early spinal compression fractures may go undetected for a long time, but after a large percentage of calcium has been lost the vertebrae in the spine start to collapse. This gradually causes a stooped posture called kyphosis or “dowager’s hump”. As much as 6 inches in height may be lost.

PENSION CONSIDERATIONS

A. CAUSES AND/OR AGGRAVATION

THE TIMELINES CITED BELOW ARE NOT BINDING. EACH CASE SHOULD BE ADJUDICATED ON THE EVIDENCE PROVIDED AND ITS OWN MERITS.

1. PRIMARY OSTEOPOROSIS

While the causes of primary osteoporosis are currently unknown, i.e. idiopathic, the following are considered contributing factors:

a) Smoking

Smoking must occur prior to clinical onset or aggravation, and be in the amount of at least 10 pack-years* of cigarettes, or the equivalent in other tobacco products.

*One pack-year means 7,300 cigarettes, or 1,460 cigars, or 7.3 kg of pipe tobacco.

b) Alcohol consumption

Alcohol consumption is measured using 10 grams absolute alcohol per standard drink. 150 kg of alcohol is approximately equivalent to 4 standard drinks per day for 10 years. A standard drink is 175 cc wine, 350 cc/beer or 30 cc liquor.

Alcohol consumption, for VAC pension purposes, differs for men and women as follows:

MEN - consumption of at least 150 kg of alcohol over any 10 year period within approximately 20 years prior to clinical onset or aggravation;

WOMEN - consumption of at least 75 kg of alcohol over any 10 year period within the 20 years prior to clinical onset or aggravation.

2. **SECONDARY OSTEOPOROSIS**

Secondary causes and/or aggravation factors of osteoporosis should be claimed as consequential to the primary disease state:

a) Chronic renal failure or chronic cholestatic liver disease at the time of clinical onset or aggravation

Chronic renal failure is a clinical condition resulting from a multitude of pathologic processes that lead to derangement and insufficiency of renal excretory and regulatory function.

Chronic cholestatic liver disease is any chronic intrahepatic or extrahepatic disease which inhibits bile secretion into the gastrointestinal tract, allowing accumulation of biliary substances in the plasma and usually results in jaundice.

b) Use of a specified drug prior to clinical onset or aggravation

A *specified drug* includes, but is not limited to, the following drugs taken as follows:

- *phenytoin* - daily for a minimum of 2 continuous months within approximately 2 years prior to clinical onset or aggravation. Phenytoin interferes with the metabolism of vitamin D.
- *glucocorticoids** (intravenous, intramuscular, oral) - daily or every second day for a minimum of 6 continuous months within approximately 2 years prior to clinical onset or aggravation. Examples would include prednisone, prednisolone and cortisone.
- *inhaled glucocorticoids** - dosages greater than 1200 ug/day of beclomethasone or its equivalent for a minimum of 12 continuous months within approximately 2 years prior to clinical onset or aggravation.

*Corticosteroids are either glucocorticoids or mineralcorticoids. The majority of corticosteroids are glucocorticoids, and are used to treat asthma and other conditions. The Compendium of Pharmaceuticals and Specialities (CPS) may assist in determining if a drug is a glucocorticoid.

- *corticotrophins* - at least weekly dose for a minimum of 12 continuous months within approximately 2 years prior to clinical onset or aggravation. Corticotrophins stimulate the secretion of corticosteroids. They are also known as adrenocorticotrophins (ACTH).
- *heparin* (intravenous, subcutaneous) - at least 20,000 units daily (a therapeutic dose) for a minimum of 3 continuous months within approximately 12 months prior to clinical onset or aggravation. The osteoporotic effect is felt to be reversible within several months upon discontinuation of heparin.
- *thyroid hormone/throxine* (oral) - any supraphysiologic dose for a minimum of 6 months within approximately 2 years prior to clinical onset or aggravation. Supraphysiologic dosages of thyroid hormone are an excessive quantity of thyroid hormone resulting from:
 - (1) overproduction by the thyroid gland (e.g. Graves' disease)
 - (2) ingestion of greater than therapeutic dosages of thyroxine medication

(3) loss of storage function and leakage from the thyroid gland.

- *immunosuppressive drugs* - drugs such as cyclosporine used in solid organ transplantation (e.g. heart, lung, kidney), and used for a minimum of 6 months.

c) Endocrine disease prior to clinical onset or aggravation

For endocrine disease to cause or aggravate Osteoporosis, endocrine disease must have existed for at least 1 year within the 10 year period prior to clinical onset or aggravation of Osteoporosis.

Endocrine disease includes, but is not limited to, the following:

- hypogonadism - This condition may be primary (associated with retardation of growth and sexual development) or secondary and may be the result of disease, trauma, surgical excision, chemical castration techniques, decreased estrogen, and decreased testosterone;
- hyperprolactinemia - This is associated with amenorrhea and galactorrhea in women;
- Cushing's syndrome;
- thyrotoxicosis or treatment for thyrotoxicosis;
- hyperparathyroidism;
- hypercalcuria.

Loss of gonadal function may occur in men with hemacromatosis and thalassemia, which are associated with loss of bone density.

d) Rheumatoid Arthritis or Ankylosing Spondylitis at the time of clinical onset or aggravation

Rheumatoid Arthritis is a chronic multisystem disease primarily involving the joints. It is characterized by inflammatory synovitis, joint involvement, muscle atrophy, and bone destruction.

Ankylosing Spondylitis can result in low bone density and fractures.
(See Entitlement Eligibility Guidelines on Rheumatoid Arthritis and Ankylosing Spondylitis)

e) Infiltrative process of bone marrow at the time of clinical onset or aggravation

Infiltrative processes include, but are not limited to, the following:
multiple myeloma;

systemic mastocytosis;
Gaucher's disease (an accumulation of glucocerebrosides in macrophages in the spleen, liver and bone marrow);
leukemia.

f) A chronic disease causing gastrointestinal malabsorption prior to clinical onset or aggravation

For a chronic disease causing gastrointestinal malabsorption to cause or aggravate Osteoporosis, the chronic disease causing the gastrointestinal malabsorption must have existed for 1 year within the 10 year period prior to clinical onset or aggravation of Osteoporosis.

Gastrointestinal malabsorption is a medical condition which may be caused by a range of diseases, all of which are characterized by faulty absorption from the intestine of essential food stuffs such as fat, vitamins and mineral salts, including the following:

total or partial gastrectomy;
total or partial small or large bowel resection;
pancreatic insufficiency, including cystic fibrosis or chronic pancreatitis;
celiac disease;
primary biliary cirrhosis .

g) Crohn's Disease or Ulcerative Colitis prior to clinical onset or aggravation

For Crohn's Disease or Ulcerative Colitis to cause or aggravate Osteoporosis, Crohn's Disease or Ulcerative Colitis must have existed for 1 year within the 10 year period prior to clinical onset or aggravation of Osteoporosis.

Crohn's Disease is a type of inflammatory bowel disease affecting any part of the gastrointestinal tract, which is characterized by chronic inflammation. It may extend through all layers of the gastrointestinal tract wall, and may also be known as Regional Enteritis.

Ulcerative Colitis is a chronic inflammation of the gastrointestinal tract, which primarily affects the large bowel and is usually limited to the mucosa and submucosa.

h) Anorexia Nervosa at the time of clinical onset or aggravation

Anorexia Nervosa is a psychiatric disorder defined in DSM-IV, which occurs predominantly in females, and is, usually, of adolescent onset. It is characterized by refusal to maintain a normal minimal body weight, intense fear of becoming

obese that is undiminished by weight loss, disturbance of self body image (resulting in a feeling of being fat even when extremely emaciated), and amenorrhea (in females).

i) Immobility prior to clinical onset or aggravation

For immobility to cause or aggravate Osteoporosis, immobility must exist for a continuous period of approximately 60 days within the year immediately prior to clinical onset or aggravation of Osteoporosis.

Immobility means continuous restriction to a lying or sitting position as directed by a physician, including prolonged hospitalization with strict bed rest, quadriplegia and prolonged spinal traction.

j) An inheritable connective tissue disorder at the time of clinical onset or aggravation

An inheritable connective tissue disorder includes, but is not limited to, the following:

Ehlers-Danlos syndrome - characterized by hyperelasticity of the skin and hypermobile joints;

Homocystinuria - which results in defects in the cross-linking of bone collagen;

Osteogenesis imperfecta - which is characterized by bone fragility, blue sclerae, and premature deafness. Strictly speaking, the bone metabolism defect in osteogenesis imperfecta is osteoporosis.

B. MEDICAL CONDITIONS WHICH ARE TO BE INCLUDED IN ENTITLEMENT/ASSESSMENT

- osteomalacia

C. COMMON MEDICAL CONDITIONS WHICH MAY RESULT IN WHOLE OR IN PART FROM OSTEOPOROSIS AND/OR ITS TREATMENT

- fractures at the site of the osteoporotic bone

REFERENCES FOR OSTEOPOROSIS

1. Amin, S. and D.T. Felson. *Osteoporosis in Men*. Rheumatic Disease Clinics of North America, vol 27, no 1. Toronto: W.B. Saunders, February 2001.
2. Australia. Department of Veterans Affairs. Statement of Principles Concerning Osteoporosis [Instrument 61 of 1997].
3. Fauci, Anthony S. and Eugene Braunwald, et al, eds. *Harrison's Principles of Internal Medicine*. 14th ed. Montreal: McGraw-Hill, 1998.
4. Gray, Jean D., ed. January - February 1998. An Overview of the Pharmacological Management of Osteoporosis. *Drugs and Therapeutics for Maritime Practitioners*, vol 21, no 1.
5. Goroll, Allan H. and Albert G. Mulley, Jr., et al. *Primary Care Medicine, Office Evaluation and Management of the Adult Patient*. 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2000.
6. Repchinsky, Carol, editor-in-chief, et al. *CPS (Compendium of Pharmaceuticals and Specialties)*. 36th ed. Ottawa: Canadian Pharmacists Association, 2001.
7. Ruddy, Shaun, et al, eds. *Kelley's Textbook of Rheumatology*. 6th ed. Toronto: W.B. Saunders, 2001.
8. U.S. Preventative Services Task Force, Guidelines from Guide to Clinical Preventative Services. 2nd ed. [N. p.]: Williams and Wilkins, 1996.
9. Wilson, Jean D., Daniel W. Foster, et al. *Williams Textbook of Endocrinology*. 9th ed. Toronto: W.B. Saunders, 1998.