

Postmenopausal Osteoporosis: Causes, Mechanisms, Risks, Diagnosis, Treatment, and Prevention Guide

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DESCRIPTION

Postmenopausal osteoporosis is a common metabolic bone disorder that affects millions of women worldwide following menopause. It is characterized by decreased Bone Mineral Density (BMD) and deterioration of bone microarchitecture, resulting in increased bone fragility and susceptibility to fractures. The primary underlying cause is the decline in estrogen levels that occurs naturally during menopause, which accelerates bone resorption and impairs the balance of bone remodeling. Understanding the mechanisms, risk factors, clinical implications, and management of postmenopausal osteoporosis is essential for early intervention and prevention of complications such as fractures and loss of independence.

Bone is a dynamic tissue that continuously undergoes remodeling through two opposing processes: bone resorption by osteoclasts and bone formation by osteoblasts. During reproductive years, these processes are balanced, maintaining bone strength. Estrogen plays a vital role in maintaining this balance by suppressing osteoclast activity, promoting osteoclast apoptosis, and enhancing osteoblast survival. At menopause, when estrogen production sharply declines, this balance is disrupted. Bone resorption outpaces formation, leading to rapid bone loss. This phase of accelerated bone loss is most intense during the first five to ten years after menopause.

Postmenopausal osteoporosis primarily affects trabecular bone, which has a higher metabolic rate and is more sensitive to hormonal changes than cortical bone. This makes bones with a higher proportion of trabecular tissue, such as vertebrae, femoral neck, and the distal radius, especially vulnerable. As bone mass declines, the microarchitecture deteriorates, and bones become porous and brittle, increasing the risk of fragility fractures. These fractures often occur with minimal trauma, such as a fall from standing height or even during daily activities.

The clinical consequences of postmenopausal osteoporosis are significant. Vertebral compression fractures may present silently or with sudden back pain, loss of height, and spinal deformities such as kyphosis. Hip fractures are associated with high morbidity and mortality, often requiring surgery and prolonged

rehabilitation. Wrist fractures can impair hand function and independence. Beyond physical effects, these injuries can lead to psychological impacts, including fear of falling, reduced mobility, and depression.

Multiple risk factors contribute to the development of postmenopausal osteoporosis. In addition to estrogen deficiency, genetic predisposition plays a significant role. Women with a family history of osteoporosis or hip fractures are at greater risk. Low body weight or a small, thin frame increases vulnerability due to lower baseline bone mass. Nutritional deficiencies, particularly inadequate intake of calcium and vitamin D, further weaken bones. A sedentary lifestyle, smoking, excessive alcohol intake, and certain medications such as corticosteroids also contribute to bone loss.

Diagnosis of postmenopausal osteoporosis relies primarily on bone mineral density testing using Dual-Energy X-ray Absorptiometry (DEXA or DXA). The results are expressed as T-scores, comparing the patient's BMD to that of a young healthy reference population. A T-score of -1.0 to -2.5 indicates osteopenia, while a score of -2.5 or lower confirms osteoporosis. In addition to BMD, clinical assessment includes evaluation of fracture history, risk factors, and use of tools like FRAX (Fracture Risk Assessment Tool), which estimates the 10-year probability of hip and major osteoporotic fractures.

Management of postmenopausal osteoporosis involves lifestyle changes, nutritional support, and pharmacological intervention. Adequate calcium and vitamin D intake is essential. Postmenopausal women should consume 1,200 mg of calcium daily, preferably through diet and supplements if needed, and 800-1,000 IU of vitamin D to support calcium absorption and bone health. Regular weight-bearing and muscle-strengthening exercises improve bone density, balance, and muscle strength, reducing the risk of falls.

Pharmacologic treatments aim to reduce bone resorption or stimulate bone formation. Bisphosphonates, such as alendronate and risedronate, are the most commonly prescribed antiresorptive drugs and have proven efficacy in reducing vertebral and hip fractures. Denosumab, a monoclonal antibody

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Received: 18-Feb-2025, Manuscript No. JOPA-25-38088; **Editor assigned:** 20-Feb-2025, PreQC No. JOPA-25-38088 (PQ); **Reviewed:** 06-Mar-2025, QC No. JOPA-25-38088; **Revised:** 13-Mar-2025, Manuscript No. JOPA-25-38088(R); **Published:** 20-Mar-2025, DOI: 10.35841/2329-9509.25.13.450

Citation: Lin J (2025). Postmenopausal Osteoporosis: Causes, Mechanisms, Risks, Diagnosis, Treatment, and Prevention Guided. J Osteopor Phys Act.13:450.

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that inhibits RANKL, is another effective antiresorptive agent. Selective Estrogen Receptor Modulators (SERMs) like raloxifene mimic estrogen's protective effects on bone without stimulating breast or uterine tissue. Hormone Replacement Therapy (HRT) may be considered in early postmenopausal women, especially if they have menopausal symptoms, but it carries risks such as cardiovascular disease and breast cancer. Anabolic agents like teriparatide and romosozumab are reserved for severe osteoporosis or for those who fail other treatments, as they stimulate new bone formation.

Fall prevention is also a crucial component of managing postmenopausal osteoporosis. Measures include improving home safety, using assistive devices when needed, correcting vision problems, and avoiding sedative medications that impair balance. Regular physical activity enhances strength and coordination, lowering fall risk. Periodic reassessment of bone density and treatment efficacy ensures that therapeutic goals are being met and helps guide adjustments in management strategies.

Preventing postmenopausal osteoporosis begins before menopause. Building strong bones through adequate nutrition,

exercise, and healthy habits during adolescence and early adulthood helps achieve higher peak bone mass, providing a buffer against future bone loss. Educating women about the importance of bone health and encouraging lifestyle modifications early can significantly reduce the risk of osteoporosis and related fractures later in life.

CONCLUSION

Postmenopausal osteoporosis is a major public health issue with significant physical, emotional, and economic consequences. It results primarily from estrogen deficiency, which disrupts the normal balance of bone remodeling. Early identification of at-risk individuals, combined with preventive strategies and effective treatment, can reduce fracture risk and preserve quality of life. By addressing both modifiable and non-modifiable risk factors and promoting bone-healthy behaviors, healthcare providers and individuals can work together to manage and prevent this potentially debilitating condition.