



OSTEOPOROSIS: ETIOLOGY, CLINICAL PRESENTATION, AND DIAGNOSTIC APPROACHES

Nabiyeva F. S.

Senior Lecturer of the Department of Clinical Laboratory Diagnostics,
Samarkand State Medical University

Yakubova D. M.

Assistant of the Department of Clinical Laboratory Diagnostics,
Samarkand State Medical University

Hafizov S. I.

Cadet of the Department of Clinical Laboratory Diagnostics,
Samarkand State Medical University

Abstract

Osteoporosis is a systemic skeletal disease characterized by reduced bone mineral density and a deterioration of bone microarchitecture, resulting in an increased susceptibility to fractures. This condition is highly prevalent in postmenopausal women and the elderly. The etiology is multifactorial, encompassing hormonal, genetic, nutritional, and environmental factors. Early diagnosis and intervention are critical in preventing fractures and improving quality of life. This paper explores the etiology, clinical presentation, and diagnostic approaches to osteoporosis, with a focus on evidence-based criteria and guidelines for clinical practice.

Keywords: osteoporosis, bone mineral density, etiology of osteoporosis, calcium deficiency, vitamin D deficiency, parathyroid hormone.

The pathophysiology of osteoporosis involves a disruption in the balance between bone resorption and formation. Several factors contribute to this imbalance, including hormonal changes, genetic predisposition, nutritional deficiencies, and lifestyle choices [4].



The most significant hormonal factor in osteoporosis is estrogen deficiency, which is particularly evident in postmenopausal women. Estrogen has a direct inhibitory effect on osteoclast-mediated bone resorption, and its reduction accelerates bone loss. In men, testosterone deficiency similarly contributes to bone resorption, though its role is less pronounced compared to estrogen in women. Moreover, hyperparathyroidism, characterized by elevated levels of parathyroid hormone PTH, increases osteoclastic activity, thereby exacerbating bone loss [3,6].

Genetic predisposition plays a crucial role in determining susceptibility to osteoporosis. Family history of the disease significantly increases the risk, with specific genes related to bone metabolism, such as the COL1A1 gene (encoding for collagen type I), being implicated in bone density regulation. Variations in the VDR gene, which encodes the vitamin D receptor, also contribute to osteoporosis susceptibility.

Calcium and vitamin D are vital for bone health, and deficiencies in either can lead to osteopenia and osteoporosis. Calcium is essential for bone mineralization, while vitamin D promotes calcium absorption in the intestines. A deficiency in vitamin D impairs calcium homeostasis and reduces bone mineral density, increasing fracture risk [2,6].

Physical inactivity, particularly the lack of weight-bearing exercise, accelerates bone loss. Weight-bearing activities, such as walking or resistance training, stimulate osteoblastic bone formation and help maintain bone density. Additionally, smoking and excessive alcohol consumption have been identified as significant risk factors for osteoporosis. Smoking impairs osteoblastic function and reduces calcium absorption, while alcohol disrupts bone metabolism and impairs osteogenesis.

Clinical presentation. Osteoporosis is often asymptomatic until a fracture occurs, which is the most common presenting feature. The clinical manifestations may include back pain, postural changes, and a decrease in height, especially in more advanced stages.

Fractures, particularly of the vertebrae, hip, and wrist, are the hallmark of osteoporosis. These fractures commonly occur with minimal trauma, such as falls from standing height. Vertebral fractures, in particular, may be clinically silent or present with mild to moderate back pain and a reduction in height.



Compression fractures of the spine often lead to chronic back pain, which may worsen with movement or prolonged standing. In some cases, these fractures result in kyphosis, an abnormal forward curvature of the thoracic spine, commonly referred to as a "dowager's hump". This postural deformity can severely affect the quality of life by impairing mobility and increasing pain.

Height loss, often subtle, may be detected over time as a consequence of vertebral compression fractures. This phenomenon reflects the cumulative effect of multiple fractures that lead to a progressive reduction in spinal height.

Diagnostic approaches. Early diagnosis of osteoporosis is essential for preventing fractures. Several diagnostic tools and criteria have been developed to identify individuals at risk and assess bone health [1,5].

Bone mineral density BMD measurement. The Dual-Energy X-ray Absorptiometry DEXA scan is the gold standard for diagnosing osteoporosis. This technique measures BMD at key skeletal sites, including the lumbar spine, hip, and forearm. According to the World Health Organization (WHO), a T-score of -2.5 or lower at any skeletal site is diagnostic of osteoporosis. The T-score compares the patient's BMD to that of a young, healthy adult, while the Z-score compares it to an age- and sex-matched control population [4].

The FRAX tool, developed by the WHO, is a widely used method for estimating an individual's 10-year probability of sustaining a major osteoporotic fracture. This tool incorporates both clinical risk factors, e.g., age, gender, smoking history, alcohol consumption and BMD measurements to stratify patients based on fracture risk.

While X-rays are not routinely used for osteoporosis screening, they are valuable in detecting vertebral compression fractures. Radiographs can reveal fractures that are asymptomatic or missed during clinical examination. However, the sensitivity of X-rays for detecting early bone loss is limited, and they are generally not recommended for routine screening [2,3].

Laboratory tests are often employed to rule out secondary causes of osteoporosis or to evaluate nutritional status. Serum calcium and vitamin D levels should be assessed to detect deficiencies that may contribute to bone loss. Additionally, parathyroid hormone (PTH) levels may be measured to evaluate for hyperparathyroidism, a condition that accelerates bone resorption. In some cases, bone turnover markers-



osteocalcin, N-telopeptide are used to assess the rate of bone resorption and formation.

Conclusion

Osteoporosis is a prevalent and underdiagnosed disease that leads to significant morbidity due to fractures. Its etiology is multifactorial, involving hormonal, genetic, nutritional, and environmental factors. The disease often progresses silently until a fracture occurs, making early diagnosis crucial. Current diagnostic guidelines emphasize the use of BMD measurement through DEXA scans, fracture risk assessment via tools such as FRAX, and appropriate laboratory tests to identify secondary causes. Early intervention, including lifestyle modifications, pharmacologic treatment, and fall prevention strategies, can significantly reduce fracture risk and improve outcomes for individuals with osteoporosis.

References

1. Brown J. P. et al. Current use of bone turnover markers in the management of osteoporosis //Clinical biochemistry. – 2022. – T. 109. – C. 1-10.
2. Lewiecki E. M. Evaluating patients for secondary causes of osteoporosis //Current Osteoporosis Reports. – 2022. – C. 1-12.
3. Sabri S. A. et al. Osteoporosis: an update on screening, diagnosis, evaluation, and treatment //Orthopedics. – 2023. – T. 46. – №. 1. – C. e20-e26.
4. Umarova S. S., Mukhamadiyeva L. A., Nabiyeveva F. S. The pathogenesis of rheumatic fever //Journal of new century innovations. – 2023. – T. 29. – №. 4. – C. 164-169.
5. Vescini F. et al. Management of osteoporosis in men: a narrative review //International Journal of Molecular Sciences. – 2021. – T. 22. – №. 24. – C. 13640.
6. Даминов Ф. А., Набиева Ф. С., Очилов О. Ш. Биологическая роль кальция в организме человека //Research Focus. – 2023. – Т. 2. – №. 7. – С. 56-58.