

# THE “SILENT KILLER” OSTEOPOROSIS. HOW TO PREVENT IT AND HOW TO LIMIT ITS COMPLICATIONS?

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**Abstract:** Osteoporosis is a disease affecting millions of people all over the world which had been defined as a “disease characterised by low bone mass and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk”. This definition has been modified. Now it is defined as a skeletal disorder characterized by compromised bone strength which predisposes a person to an increased risk of fracture, where clinical bone strength is estimated by an assessment of bone mineral density /BMD/. The prevalence of osteoporosis is very high and varies by different regions. According to reliable sources osteoporosis causes more than 9 million fractures annually worldwide where most occur in the Americas and Europe. There are a lot of well-recognized osteoporosis causes and risk factors. This knowledge will aid in developing an effective prevention strategy. In this review the current data about silent killer are discussed.

**Key words:** osteoporosis, causes and risk factors, prevalence, complications.

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## Introduction

Osteoporosis has become one of the major public problems and is very often called: *the silent killer, the silent thief or the silent epidemic* [1]. Recently, we can observe the dramatic increase in the average life expectancy, and with this fact, there is abundant evidence which shows [2] that the prevalence of osteoporosis increases with age, and that the age-related bone loss is significantly greater in women than in men. In women, some hormonal alterations mostly during the changes occurring at menopause contribute to many of the major factors leading to osteoporosis. Ovarian aging is a biological fact, and it results in a rapid and significant decrease in 17 $\beta$ -estradiol secretion and consequently an increase in the secretion of cytokines which activate osteoclasts finally leading to bone loss and microarchitectural deteriorations [3]. In many cases osteoporosis, literally “porous bones”, has no clinical manifestations until there is a fracture. The data presented in the report from 2015 have shown that in 2010 in Poland, there were 2,247,000 osteoporotic fractures in women and 463,000 in men. The same report reveals that the cost of treatment of osteoporosis and osteoporotic fractures was very high. Therefore, the introduction of a preventive strategy as well as effective treatment of osteoporosis is of great significance [4, 5].

## Pathophysiology of osteoporosis

Health care physicians and nurses need to understand the basic mechanisms of bone physiology and pathophysiology

in order to both prevent and treat this devastating disorder [6–8]. Bone mass measured as bone mass density /BMD/ increases rapidly during childhood and adolescence. To understand properly the pathophysiology of osteoporosis medical care specialists should know that a bone is living tissue and what is more important that its strength depends upon the normal functioning of three bone cells. It consists of osteoblasts, osteoclasts and osteocytes. Osteoblasts and osteoclasts compose the functional multicellular unit responsible for bone reconstruction and remodelling. With advancing age the time needed for osteoclasts to resorb bone is measured in weeks, while the time needed for osteoblasts to form bone is long measured in months. The age-related bone loss is greater in women than in men. In women the rapid bone loss takes place during the first years after the menopause. In men the peak bone mass (BMD), as measured by dual energy X-ray absorptiometry (DEXA), is measured at 40 years and then bone loss accelerates exponentially after 70. This fall in bone mass is the result of functional activities of osteoclasts and osteoblasts [9–11]. In recent years, the molecular mechanisms which regulate these cells have been discovered and described, but in clinical practice the recognition of risk factors and the efforts to prevent osteoporosis ought to be the main goal [12]. We should remember that osteoporosis has no clinical manifestations until there is a fracture. Hence, the disease is described as the silent thief or silent killer.

## Risk factors of osteoporosis

There are many well-recognized factors that can contribute to a greater possibility of the development of osteoporosis. These factors are divided into two large groups: not modifiable which are out of our control and modifiable which should be the subject of our prophylactic activity [13].

Not modifiable risk factors, including:

- women** – are much more likely to develop osteoporosis than men;
- age** – the risk of osteoporosis is higher in older subjects;
- race** – white women are at greatest risk of osteoporosis;
- family history** – parents with osteoporosis increase an offspring's risk of developing the disease;
- body frame size** – it concerns people with small body frames.

Modifiable risk factors, including:

- excessive alcohol consumption** – more than two drinks a day;
- tobacco use** – has been shown to diminish bone endurance;
- daily physical activity** – simple non-pharmacological intervention [14].

Other groups of risk factors must not be overlooked, such as low calcium intake, eating disorders or gastrointestinal surgeries, administering corticosteroids as well as numerous other medical conditions.

Some endocrine factors can have an enormous impact. They concern increased or diminished concentrations of certain hormones. Low oestrogen levels are the most dangerous risk factor found mostly in women at menopause. Also, high levels of thyroid gland hormones as well as overactive parathyroid and adrenal glands increase the risk of osteoporosis [15].

It has been well-established that sex hormones play a significant role in bone breakdown. To limit the progression of *the silent killer* we ought to be aware that irregular periods in young women may be a warning sign of hormonal disturbances that could increase the risk of osteoporosis. Another situation, primary ovarian insufficiency, is not common, but it is an important cause of ovarian hormone deficiency. There are data that suggest that the number of pregnancies have a negative effect on bone mass and later on may lead to osteoporosis and fractures. Prolonged breast feeding can also influence the development of postmenopausal osteoporosis. Hormonal replacement therapy, especially with oestrogens, has a favourable impact on female bone health, but such treatment is not advised as the main goal of the treatment. In clinical practice the awareness of the risk factors of osteoporosis is the best way to

eliminate or prevent the disease. There are four very important areas that require special attention i.e. to provide sufficient calcium and vitamin D support, to advise patients to quit cigarette smoking and to reduce alcohol consumption and to do regular exercise [16].

## Diagnosis of osteoporosis

The precise diagnosis of osteoporosis should offer guidance for the bone loss and fragility fractures. Nowadays, both instrumental and biochemical tests are available. The instrumental method for this purpose is a dual energy X-ray absorptiometry (DEXA) which has become the most widely used technique. DEXA allows the assessment of BMD (bone mass density, the amount of bone mass g/cm<sup>2</sup>) for the whole skeleton or for specific parts of the body. DEXA is usually performed at the level, of lumbar spine or proximal femur [17,18]. We must remember that bone strength is also influenced by bone quality and tissue properties, and these parameters are currently evaluated by of some other available radiological techniques [19]. The International Osteoporosis Foundation recommends the use as markers of bone formation different biological substances, but their estimations are usually performed in specialized centres.

## The assessment of fracture risk

From a practical point of view the proper assessment of fracture risk allows the clinician to identify individuals who need specific medication and to screen out those who do not. The group of WHO experts prepared a report in which they distinguished the most significant risk factors of fractures: previous fracture, a parent's fracture hip, smoking cigarettes, treatment with Glucocorticoid, rheumatoid arthritis, secondary osteoporosis, alcohol consumption exceeding three units per day and femoral neck BMD T score of -2.5 or less. It has resulted in the establishment of an algorithm to function as a FRAX calculator (WHO Fracture Risk Assessment Tool). In Poland, a team from Cracow has added a modification called the hand held FRAX calculator [20]. By using this devices we can calculate the 10-year fracture risk and for practical purposes assign patients to one of three groups: group 1 – patients with the high risk of fracture, who need immediate treatment; group 2 – intermediate group-patients who need densitometric verification if the Frax score was calculated on the basis of BMI; group 3, – low risk group of patients who need neither treatment nor further diagnosis [20]. There are a lot of publications which state that the FRAX tool is not an ideal one, but by its simplicity it currently is becoming the most popular option [21,22]. In Poland, it is commercially available at a low cost.

### Disclosure

The author reports no conflict of interest.

### Literature

- [1] Cathleen S., Colon E., Saag K.G. Osteoporosis fractures in older adults. *Best Practice & Research: Clinical Rheumatology*, (20):695, 2006.
- [2] NIH publication no 11-7737, October 2011-Global Health Aging.
- [3] Bord S., Ireland D. C., Beavan S. R. B. et al. The effect of estrogen on osteoprotegerin, RANKL and estrgen expression in human osteoblasts. *Bone*, (32):136–141, 2003.
- [4] Szulc P., Bouxsein M. L. *Overview of osteoporosis epidemiology and clinical management*. International Osteoporosis Foundation., 2011.
- [5] Czerwiński E., Synder M. *Raport. Osteoporoza-cicha epidemia w Polsce*. Europejska Fundacja Osteoporozy i Chorób Mięśniowo-Szkieletowych Polskie Towarzystwo Ortopedyczne i Traumatologiczne, Kraków, 2015.
- [6] Heaney R.P. Pathophysiology of osteoporosis. *Endocrinology Metabolism Clinics*, (27):255–265, 1998.
- [7] Sipos W., Pietschmann P. et al. Pathophysiology of osteoporosis. *Wiener Medizinische Wochenschrift*, 159(9-10):230–234, 2009.
- [8] Armas L.A., Recker R.R. Pathophysiology of osteoporosis:new mechanistic insights. *Endocrinology and Metabolism Clinics of North America*, 41(3):475–486, 2012.
- [9] Khosia S. Pathogenesis of age-related bone loss in humans. *The Journals of Gerontology. Series A, Biological Sciences*, 68(10):1226–1235, 2013.
- [10] Carson J. A., Manolagas S.C. Effects of sex steroids on bones and muscles: similarities, parallels and putative interaction in health and disease. *Bone*, (80):67–78, 2015.
- [11] Almeida M., Laurent M.R. et al. Estrogens and androgens in skeletal physiology and pathophysiology. *Physiological Reviews*, (1):135–187, 2017.
- [12] Capozzi A., Lello S., Pontecorvi A. The inhibition RANKL-ligand in the management of postmenopausal osteoporosis and related fractures: the role of denosumab. *Gynecological Endocrinology*, 30(6):403–481, 2014.
- [13] Snelling A.M., Crespo C.J. et al. Modifiable and Nonmodifiable Factors associated with osteoporosis in postmenopausal women:results from the third national Health and nutrition survey, 1988-1994. *Journal of Women Healths, G-B Medicine*, 10(1), 2001.
- [14] Abrahamsen B., Brask-Linderman D. et al. A review of lifestyle, smoking and other modifiable risk factors for osteoporotic fractures. *Bonekey Reports*, (3):574, 2014.
- [15] Liu W., Yang L.H. et al. Meta-analysis of osteoporosis: fracture risks, medication and treatment. *Minerva Medica*, 106(4):203–214, 2015.
- [16] Szamatowicz M. How can gynecologists cope with the silent killer-osteoporosis. *Menopause Review*, 15(4):189–192, 2016.
- [17] Dasher L.G., Newton C.D., Lenchik L. Dual X-ray absorptiometry in today’s clinical practice. *Radiologic Clinics of North America*, (48):541–560, 2010.
- [18] Rossini M., Adami S., Bertholdo F. et al. Guidelines for the diagnosis, prevention and management of osteoporosis. *Reumatismo*, (68):1–39, 2016.
- [19] Bauer J.S., Link T.M. Advances in osteoporosis imaging. *European Journal of Radiology*, (71):440–449, 2009.
- [20] Czerwiński E., Osieleniec J., Kumorek A. Nowe narzędzie w diagnostyce osteoporozy. *Ortopedia Traumatologia Rehabilitacja*, (11 (suppl. 2)):72, 2009.
- [21] Amarowicz J., Bolisega D., Rutkowski J. et al. Cost effectiveness analysis of treatment of osteoporosis fractures in relations to FRAX algorithm in sample of Polish population. *Ortopedia Traumatologia Rehabilitacja*, (17):59–69, 2015.
- [22] Rubin K.H., Kris-Holmberg T., Herman A.P. et al. Risk assesment tools to identify women with increased risk of osteoporotic fracture. Complexity or simplicity? A systematic review. *Journal of Bone and Mineral Research*, (28):1707–1717, 2013.

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