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Osteoporosis in Dentistry

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Abstract

The osteoporosis is a disease characterized by a low osseous mass and a deterioration of the architecture of the bone leading to an increased osseous brittleness and an increase in the risk of fracture whose incidence increases with the age, approximately 40% of the women arriving at 50 years age risk at least an osteoporotic fracture. The frequency of these fractures is in constant increase, primarily because of the continuous aging of the population. One can from now on precociously identify the disease thanks to the osteodensitometry which measures the osseous mineral density. The age and menopause are the two principal determinants of the osseous loss. The factors of risks are mainly the family antecedents and the oestrogenic deficiency. The purpose of the preventive treatments of osteoporosis is to prevent the osseous loss by slowing down the osseous replanning which accelerates after the menopause. The curative treatments seek to increase the osseous mass and to prevent new fractures without deteriorating the quality of the bone. The Dentist must take into account the existence of an osseous pathology that affects the supporting structures of the tooth and the resistance of the jawbones.

Subject Areas

Dentistry

Keywords

Osteoporosis, Menopause, Odontology, Maxillary

1. Introduction

In recent years, osteoporosis has seen considerable advances in diagnostic and

therapeutic management, benefiting from the interest it receives from the scientific community.

The concept of bone fragility is currently based on both a quantitative and qualitative approach. Understanding it requires a reminder of its current pathophysiological and epidemiological importance.

Aside from secondary osteoporosis, which responds to its own pathophysiological mechanisms, postmenopausal osteoporosis has seen increased interest in recent years, both from the medical world and the general public. Age and menopause are the two main, but not exclusive, determinants of bone loss leading to osteoporosis.

Osteoporosis will have consequences in each of the disciplines that make up our practice, from surgery to prosthetic reconstruction, from periodontology to dentofacial orthopedics, but especially in disciplines related to bone structures.

2. General Information

Osteoporosis is a general skeletal disease characterized by low bone mass and alterations in the microarchitecture of bone tissue, leading to increased bone fragility with risk of fracture.

He demonstrated [1] that there is a statistically significant difference between the average number of missing teeth in osteoporotic patients [2] [3] compared to normal patients [4] [5]. She also reported that osteoporotic patients have lower bone mass and density, as well as a thinner cortex at the gonion. She also demonstrated that there is a significant relationship between osteoporosis and the resorption of the alveolar ridges. At the mandibular level, this induces the appearance of a knife-edge ridge with the presence of the mental foramen on the ridge as well as the impression of a long ascending ramus due to the reduction in height of the horizontal ramus. Alveolar bone has a dental specificity, but its aging contributes to the general process of physiological osteopenia, resulting in a loss of cortical and cancellous bone tissue. With age and metabolic impairment, there appears to be a reduction in the bone turnover process, compromising bone healing.

3. Definitions of Osteoporosis

The clinical identification of osteoporosis rests on a series of complementary definitions, from its fundamental etymological meaning to its anatomical characterization and its definitive densitometric assessment.

3.1. Etymological

Osteoporosis means: Porous bone.

3.2. Anatomical

According to the Hong Kong Consensus Conference (1993), osteoporosis is defined as:

"a diffuse skeletal disease characterized by low bone mass and microarchitectural alterations of bone tissue leading to increased bone fragility and susceptibility to fractures." [1] [6]-[8]

This definition suggests that there are two components to bone fragility: bone quantity and bone quality:

-Bone microarchitecture depends on the quality of collagen and crystals and the ability to repair microfractures. It cannot be measured using a non-invasive technique. Therefore, a bone biopsy is required to assess it.

-Bone mass can be assessed using several measurement techniques that have been developed in recent years, allowing for accurate, reliable, and reproducible estimation [9]-[13].

3.3. Bone Densitometry

In 1994, a WHO group proposed a definition that takes into account the contribution of bone densitometry, which primarily allows for the definition and diagnosis of osteoporosis at different stages of the condition, particularly at an asymptomatic anatomical stage (**Table 1**) [9].

Table 1. Classification of the bone densitometric diagnosis of osteoporosis proposed by the WHO [9].

Decreased bone mineral density (BMD)	Diagnosis
1- BMD less than 1 SD lower than that of a young adult (T-score > −1)	Normal
2- BMD between −1 and −2.5 SD compared to a young adult (−1 < T-score > −2.5)	Osteopenia
3- BMD less than −2.5 SD compared to a young adult	Osteoporosis
4- BMD less than −2.5 SD compared to a young adult in the presence of one or more fragility fractures.	Severe osteoporosis

SD (standard deviation): standard deviation of the normal distribution in young adults. The mean +/− includes approximately 68% of individuals; the mean +/− 2SD, approximately 95% of individuals.

Studies have confirmed that fracture risk is closely correlated with bone mineral density (BMD).

Two methods of presenting the results are used:

- The Z score, which indicates the difference between the patient's value and the mean value of normal subjects of the same age and sex.
- The T score, which reflects the difference between the patient's value and the mean value of young adults of the same sex, is again expressed as a number of standard deviations. It is this latter index, an age-related index, that was chosen by experts at the World Health Organization (WHO) to define normal, osteogenic, and osteoporosis in postmenopausal women.

The IOF (International Osteoporosis Foundation), an assessment conducted in

the United States [14], modified these criteria by deciding to take the femoral neck measurement as the reference measurement site.

The WHO definition of osteoporosis is characterized by its simplicity, but certain precautions are required, particularly regarding the measurement site, the technique used, and the reference curves, which will be detailed in the diagnosis chapter.

4. Epidemiology

The descriptive epidemiology of osteoporosis is based on its most obvious clinical manifestation: fracture.

Osteoporosis is the main cause of all forms of fractures. Three skeletal sites are frequently affected:

- The upper end of the femur,
- The vertebrae,
- The distal end of the forearm [14] [15].

Due to the morbidity and mortality caused by its complications, particularly femoral neck fractures, osteoporosis is a major public health problem. No epidemiological studies are available in Morocco.

A European Union report by Deardorff *et al.* 2022 [14] estimates an increase in the number of hip fractures from 414,000 to 972,000 over the next 50 years, due to demographics (an aging population). A European study, Dargent P Breart G 1993 on vertebral fractures showed that by the age of 65, roughly 10% to 20% of women and men have already developed a vertebral fracture in Europe and that the incidence of new vertebral fractures is 1% per year for women aged 65 and 0.6% for men.

Currently, osteoporosis affects more than 10 million people in the United States; it is expected that osteoporosis will affect approximately 14 million adults over the age of 50 by the year 2020. Approximately 200 million women worldwide suffer from osteoporosis. Although the development of osteoporosis is currently significant in North America and Europe, it may continue to increase in these developed countries as the elderly population continues to rise. Numerous studies conducted in different regions of the world and among major ethnic groups have led to the following conclusions:

The white race is more at risk than the black race. Deardorff *et al.* 2022 [14] showed a prevalence of femoral neck fractures 10 to 20 times higher in Caucasian or Asian ethnic groups than in black people. The difference is considerable when the two races coexist (the United States, South Africa).

However, disparities within the white race are significant:

- Disparity along a North-South axis: Europeans, for example, are much more likely to be affected than Mediterraneans.
- Disparity within the same region: urban areas are more at risk than rural areas.

Various factors are assumed to be involved: Physical activity is more intense in the countryside, and lifestyle is more likely to expose people to risks in cities

(nutrition, alcohol, tobacco). The risk is thought to be higher in women of short stature, thinness, and a low body mass index. Obesity is considered a protective factor.

5. Clinical Forms of Osteoporosis

Osteoporosis is a rare and weakening osteopathy whose main determinants are age and menopause.

Thus, we can distinguish:

- Primary or common osteoporosis.
- Secondary osteoporosis.

5.1. Primitive Osteoporosis

Primary osteoporosis is not linked to an underlying pathology but to a physiological depletion of bone mass caused by menopause and aging. We distinguish:

- Osteoporosis type I.
- Osteoporosis type II.

5.1.1. Type I Osteoporosis

Type I osteoporosis occurs primarily in postmenopausal women (6 women for every 1 man). It is called classic osteoporosis or postmenopausal osteoporosis and occurs between the ages of 50 and 75.

Indeed, the decrease and then cessation of estrogen secretion induces osteoclastic hyperactivity, leading to increased bone resorption and, at the same time, an activation of bone remodeling.

However, the increase in resorption is greater than bone formation, creating an imbalance and therefore bone loss, both cortical and trabecular.

Added to this is the age-related decrease in bone formation: physiological osteopenia [6] [14] [15].

Thus, during menopause, bone loss is approximately 1% to 2% per year, reaching, in extreme cases, 12% over two years. Type 1 osteoporosis primarily affects cancellous or trabecular bone, with not only thinning of the bone trabeculae, but also a disappearance of the horizontal trabeculae, leading to destabilization of the vertical trabeculae, which often results in vertebral collapse [16].

5.1.2. Type II Osteoporosis

Type II osteoporosis affects both sexes at a later age, beyond 70 years (the average age of onset of a femoral neck fracture is 78 years). This is referred to as senile osteoporosis.

It results from age-related bone loss that continues in a linear fashion and affects both trabecular and cortical bone.

It predominates at the cortical level. Cortical thickness decreases with age, while cortical porosity increases. The separation of the bone trabeculae is more significant. Vitamin D deficiency is an additional aggravating factor, particularly through secondary hyperparathyroidism [17]-[19].

5.1.3. Osteoporotic Bone Marrow Defect

The etiopathogenesis remains hypothetical, with a possible bone development disorder (embryonic remnant not subject to bone marrow remodeling) being suggested. However, it is unlikely that bone marrow hyperplasia is due to a local factor, trauma (tooth extraction), or a systemic factor (anemia, for example).

Clinical signs are lacking in most cases. Pain or swelling is rare. The vitality of the teeth implanted in the lesion is always preserved.

Radiological signs are sometimes troubling; radiolucency is most often poorly delineated, with fine or barely visible trabeculation (**Figure 1**).

According to Wang and Xu S [20] [21], the anterior limits of luceracy are clearly defined, while the posterior limits are blurred and imprecise. The lesion does not exceed 15 mm in size. Its differential diagnosis, often difficult, must be made with osteolytic, infectious, or benign and malignant tumoral bone lesions [20] [22].

The diagnosis is primarily histological, based on the curettage product, which is also curative [21].

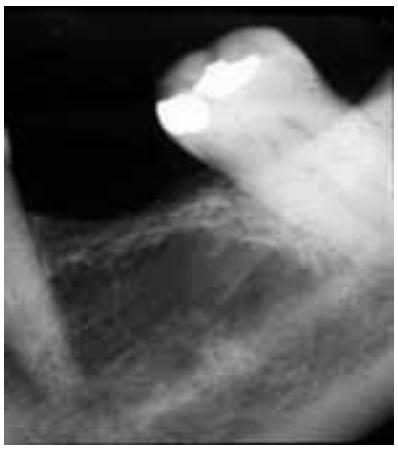

Figure 1. Periapical radiology of a patient with osteoporotic bone marrow defect [23].

5.2. Secondary Osteoporosis

These osteoporosis cases are called secondary because they result from a clearly identified pathological process.

The list of etiologies that may have a causal relationship continues to grow; several etiologies are distinguished: [20]-[22].

5.2.1. Endocrine Causes

The following section details the primary endocrine pathologies that lead to secondary osteoporosis.

1) Cortisone-induced osteoporosis

Corticosteroid-induced osteoporosis is the most common secondary osteoporosis. It is linked to corticosteroid excess.

Corticosteroid excess can result from Cushing's syndrome or disease or, more commonly, from long-term corticosteroid therapy for a chronic condition such as chronic inflammatory rheumatism (rheumatoid arthritis, etc.) or inflammatory

enteropathy (Cru's disease, ulcerative colitis).

Its incidence varies, due to factors related to the patient's condition, the condition that indicated treatment, its duration, the corticosteroid derivative used and dosage, and even individual susceptibility.

Corticosteroid impairs bone by stimulating resorption and inhibiting bone formation. Indeed, they are responsible for a deficit in bone formation inducing relative hyper resorption, maximum during the first year of treatment. It is related to a direct action on osteoblasts and their protein synthesis capacity, but also to an indirect action through humoral mediation (parathyroid hormone, estrogen, testosterone, growth factors), and to a lesser degree with an action on osteoblasts [23].

Furthermore, corticosteroids inhibit intestinal calcium absorption by competing with vitamin D (whose structure is similar to that of corticosteroids) at nuclear receptors. This decrease induces hyperparathyroidism, which also contributes to bone loss.

Corticosteroids also compete with sex hormones, especially estrogens, at their receptors.

- Pathophysiological characteristics:

Bone loss, predominantly in the cancellous bone, is rapid during the first year of treatment, around 5%, and continues in subsequent years, at approximately 2% per year.

Trabecular bone, due to its higher bone turnover, appears more sensitive to corticosteroid therapy than cortical bone. It presents a microarchitectural alteration specific to corticosteroid-induced osteoporosis, characterized by a significant overall decrease in the thickness of the bone bases, combined with a reduction in the degree of mineralization.

These qualitative abnormalities explain why, for equal bone mass, the fracture risk is higher in postmenopausal osteoporosis.

Thus, the risk is recognized for a T-score < −1.5 DS in corticosteroid-induced osteoporosis (T-score < −2.5 DS in postmenopausal osteoporosis).

While high doses are particularly harmful in the initial phase of treatment, a harmful effect is also possible for predatory doses below 7.5 mg per day.

The progression of bone loss after discontinuation of corticosteroid therapy is still poorly understood; however, recovery from Cushing's syndrome appears to be accompanied by an improvement in skeletal density. Growing children and postmenopausal women are most susceptible [24].

- Clinical particularities:

In adults, the disease is often revealed by repeated vertebral compression fractures, occurring a few months after the initiation of corticosteroid therapy. Vertebral fractures, rib fractures, and fragility fractures of the long bones are also observed.

In children, growth retardation may be observed.

Osteoporosis is relatively painless due to the analgesic action of corticosteroids [25].

2) Hypogonadism

Osteoblasts, responsible for bone formation, possess androgen receptors and also numerous estrogen receptors that participate in controlling bone cell activity. These cells are also capable of converting testosterone into estradiol by aromatization. It is therefore likely that androgens act not only directly on bone tissue but also through estrogen.

In male osteoporosis, hypogonadism is found in 20% of men with vertebral collapse.

Androgens act directly on bone through the presence of receptors on osteoblastic cells, inducing their replication.

Androgen deficiency, most often linked to antiandrogen therapy (for prostate cancer), induces accelerated bone remodeling, which primarily predominates at the vertebral level.

The most common etiologies of male hypogonadism:

- Klinefelter syndrome.
- Hypogonadism of pituitary origin.
- Hyperprolactinemia.
- Sequelae of orchitis (mumps).
- Genetic hemochromatosis.
- Treatment with Gn Rh (Gonadotropin-releasing hormone) agonists [26].

The consequences on the skeleton appear to depend on the stage of life in which this hormone deficiency occurs. Before puberty, hypogonadism is responsible for delayed bone mass acquisition, with a marked deficit at the cortical level.

In adult men, androgen deficiency is responsible for more pronounced bone loss in the cancellous area.

In women, estrogen deficiency, outside of menopause, can be linked to:

- Early menopause (often familial) or induced menopause (surgery or chemotherapy).
- Primary idiopathic amenorrhea.
- Amenorrhea related to anorexia nervosa.
- Hyperprolactinemia
- Treatment for endometriosis with a Gn Rh analogue.

The mechanism of bone loss associated with hypogonadism is a rapid acceleration of bone remodeling, which initially predominates at the vertebral level [27].

3) Hyperthyroidism

Thyroid osteoporosis is a corticotrabecular osteopathy due to accelerated bone remodeling through the direct effect of thyroid hormones on bone tissue. Thyroid hormones accelerate bone remodeling by stimulating osteoclasts and reducing intestinal calcium absorption.

In young people, the increase in resorption is accompanied by a roughly equivalent increase in bone formation, and bone loss is minimal.

However, in older people, where bone formation is reduced, the increase in resorption can lead to significant bone loss.

In postmenopausal hyperthyroid women, bone loss can become very significant due to the proliferation of resorption sites that are incompletely filled by bone apposition. Hyperthyroidism causes cortical and trabecular bone loss of varying severity, likely depending on the severity of the hyperthyroidism and its duration.

Prescribing thyroid hormone replacement doses in cases of hypothyroidism would not result in bone loss, as the ultrasensitive TSH assay allows for precise dosage adjustment. If replacement therapy is used, free T4 and TSH should be maintained at normal serum levels.

In the case of suppressive therapy, TSH should be maintained at a subnormal level [28].

4) Diabetes

In type 1 diabetes, studies, such as Calciolari *et al.* 2015 [29], have shown a decrease in vertebral and hip BMD. This decrease is thought to be correlated with a decrease in IGF and its transporters 1, 3, and 43.

Moreover, in type 2 diabetes, a decrease in BMD at the hip has been observed, correlated with proinsulin levels.

5.2.2. Drug-Induced Osteoporosis

- Alcoholism and chronic smoking can be responsible for osteoporosis. We will explore their mechanisms in the risk factors.
- Long-term treatment with anticonvulsants, particularly phenobarbital, interferes with vitamin D metabolism and can promote deficiencies in cases of insufficient intake. However, numerous studies, such as Paulo Insalaco, 2001 [6] have demonstrated the association between disturbances in the phosphocalcic balance and long-term anticonvulsant treatment, essentially leading to hypocalcemia and, more controversially, hypovitaminosis D.

This drop in serum calcium is associated with an elevation in PTH and a decrease in bone mineral density despite subnormal 25(OH) D levels.

- High-dose thyroid hormones stimulate bone remodeling and lead to bone loss.
- Long-term treatment with heparin can induce osteoporosis. The mechanism(s) responsible do not seem to be fully understood.
- Studies conducted by Gulsahi, 2015 [9] [30] have shown that heparin was responsible in vitro for an increase in bone resorption.
- Other drugs with an established role.
  + Aluminum salts
  + Lithium
  + Hypervitaminosis A

5.2.3. Idiopathic Hypercalciuria

It is defined by a calciuria level greater than 0.7 m.mol/24h in humans on a free-cholesterol diet on 3 occasions in the absence of hypercalcemia.

Bone mineral density is thought to be decreased primarily by a leak leading to an increase in parathyroid hormone. This abnormality is thought to be located primarily in the proximal renal tubule.

Idiopathic hypercalciuria is very often associated with osteopenia, which predominates in the spine [31].

5.2.4. Immobilization-Induced Osteoporosis

Several studies, including Sobh *et al.* 2022 [6], have demonstrated that immobilization leads to increased resorption and decreased bone formation, with subsequent stabilization, which occurs earlier in terms of formation. It primarily affects weight-bearing bones.

5.2.5. Digestive Causes

An increase in the prevalence of osteoporosis in these intestinal diseases is now documented, and recent studies have shown an increased risk of fracture in these young patients.

Links link chronic liver disease to bone, and may be common (initially vitamin D metabolism, more recently cytokines) or more specific to each type of liver disease: bilirubin, glucocorticoid use or dopamine: bilirubin, glucocorticoid or immunosuppressant use, alcohol. The pathogenesis remains controversial, most often multifactorial. The prevalence of osteoporosis in chronic liver disease ranges from 20% to 100% [32].

5.2.6. Ankylosing Spondylitis

It has recently been demonstrated, Sobh *et al.* 2022 [6], the existence of diffuse osteoporosis weakening the bone, mainly affecting the vertebrae and which can cause, later in the disease's progression, vertebral fractures that can lead to rare but significant mortality and morbidity.

6. Risk Factors

Osteoporosis is a multifactorial disease. There are genetic, medical, and behavioral risk factors.

6.1. Genetic Factors

Heredity

This role of heredity has been demonstrated primarily through family studies, particularly by comparing the variance in bone mass between pairs of homozygous and heterozygous twins. This latter comparison suggests that 80% to 90% of the variability in peak bone mass can be explained by genetic factors. There is currently a trend toward polygenic involvement; the genes studied are: the genes coding for IL1ra.

Differences in the incidence of osteoporotic fractures are also observed between white, black, and yellow races [33] [34].

Black women have a bone mass 5% to 15% higher than that of white women.

6.2. Estrogen Deficiency

While hereditary factors are primarily involved in the acquisition of peak bone

mass, the predominant factor in bone loss is estrogen deficiency occurring in women at menopause, as well as menstrual abnormalities during the period preceding menopause. Estrogen deficiency is responsible for increased bone remodeling, which explains bone loss [35].

6.3. Nutritional Factors

Nutritional factors are involved in the occurrence of fractures primarily through their influence on bone density, the main component of bone strength. These factors include calcium, phosphorus, protein, and vitamins C, D, and K [33] [34] [36] [37].

Calcium:

Throughout the skeletal system's lifespan, a deficiency in calcium and vitamin D can be a risk factor for osteoporosis.

This risk becomes particularly significant in older age, particularly in elderly people suffering from malnutrition and lacking sun exposure. Nutrition, particularly low calcium intake, appears to play a role at the extremes of life.

- During the growth period, a decrease in dietary intake is responsible for a decrease in peak bone mass.
- In older adults, a classic deficiency frequently occurs, resulting from a decrease in dietary intake and impaired intestinal absorption due to an associated vitamin D deficiency.
- This dual deficiency is responsible for secondary hyperparathyroidism and cortical bone loss, which leads to femoral neck fractures.

Vitamin D:

- Vitamin D, among other actions, promotes the active absorption of calcium present in the digestive tract. This action is particularly important in cases of low dietary calcium intake.
- Vitamin D stores are generally lower in the elderly than in young adults.
- This vitamin D deficiency in the elderly is due to insufficient exposure to sunlight, which leads to a reduction in the synthesis of this vitamin.
- Vitamin D requirements therefore increase with age.

Vitamin K:

- Vitamin K is necessary for the gamma-carboxylation of glutamic acid residues in many proteins, the best known of which are involved in coagulation pathways.
- In the context of osteoporosis, two proteins are involved in a possible vitamin K deficiency: osteocalcin and nephrocalcin.
- Bone metabolism is dependent on numerous vitamin K-dependent proteins (osteocalcin, proteins). Vitamin K levels are also frequently lowered in the serum of older adults. In 2022, chondrodysplasia puneta, or fetal warfarin syndrome, was described [38], a bone dysplasia observed in the children of women taking vitamin K antagonists. However, observational studies in adults taking vitamin K antagonists have shown conflicting results (Kinalski *et al.* 2022)

[38], either with no effect on bone mass or a decrease in bone mass. More recent supplement studies have shown that vitamin K delays postmenopausal bone loss [38].

- Vitamin K2 supplementation may increase or preserve bone density in postmenopausal women; it is believed that this vitamin may activate certain proteins involved in bone mass structure [39].
- Vitamin K is a cofactor in many biological pathways. Carboxylation reactions, in particular, are dependent on vitamin K. This is the case for the carboxylation of osteocalcin, a bone protein containing gamma-carboxyglutamic acid. This is also the case for a large number of other calcium-binding proteins, such as calbindin. These proteins are involved in calcium absorption and bone mineralization. Osteocalcin is synthesized in osteoblasts in bone tissue. When osteocalcin is not carboxylated, it cannot bind to hydroxyapatite. Its serum levels are a good marker of metabolic bone turnover.

Vitamin K is therefore closely linked to osteocalcin and bone health:

- Low blood and dietary levels of vitamin K are associated with low bone mineral density in women.
- Taking vitamin K supplements appears to stimulate the bone-building process by increasing calcium's attraction to bone tissue and improving bone density.
- Vitamin K supplementation also reduces the amount of calcium lost in the urine, meaning that more of this mineral is available for bone building. Vitamin K2 may slow the breakdown of bone tissue.
- Vitamin K also helps the body make a protein called matrix protein G1a, another substance that promotes bone building [40] [41].

6.4. Lifestyle Factors

- Tobacco:

It appears well established that tobacco causes a decrease in bone mass in older adult smokers. This decrease is greater in women than in men. It depends on the number of packs smoked per year. The toxic effects of tobacco may persist after smoking cessation. The effects of tobacco on bone are both direct and indirect [42] [43] [44]:

- Direct effects decrease collagen synthesis by embryonic cells and have a deleterious effect on osteoblast proliferation.
- Indirect effects decrease gastrointestinal calcium absorption, resulting in hyperbone resorption.
- Tobacco also appears to have an antiestrogenic effect, manifesting as a decrease in peripheral estrogen production, an increase in FSH and LH, accompanied by an increase in the destruction of exogenous estrogen. Tobacco reduces the effectiveness of hormone replacement therapy in postmenopausal women [45].

- Alcohol:

Overall, alcohol consumption does not appear to be a significant predictive

factor. However, it can reduce bone mass and increase the frequency, especially in men. The duration of intoxication, rather than the dose, appears to be important.

However, studies conducted by Sobh *et al.* 2022 [6] have shown the absence of bone damage, particularly when consumption is moderate, and even an increase in bone density, particularly during (social) drinking.

The alcohol-tobacco cocktail triples the risk of osteoporosis in young men.

- Coffee:

Coffee is considered a significant risk factor for femoral neck fractures.

Caffeine, which modifies the AMPE level involved in the resorptive response to PTH, is not always recognized as a risk factor. It appears to act primarily on cortical bone and less so on trabecular bone.

-Physical Exercise:

Prolonged immobilization accelerates bone remodeling and depresses osteoblast activity, resulting in very rapid bone loss.

Conversely, sufficient physical activity has a beneficial effect on bone mass.

Indeed, bone loss as a consequence of intensive training has been demonstrated in a few studies [9] [46].

6.5. Drug Factors

At the limit of risk factors and associated diseases, certain conditions and drug treatments are responsible for bone loss, and therefore likely to aggravate or unmask so-called secondary osteoporosis.

The main osteopenic drugs are:

- Cortisone derivatives, which induce very early and rapid bone loss, which subsequently slows down. They rank first in terms of their frequency of prescription and the magnitude of the risk.
- Thyroid hormones, administered in excessive doses, which stimulate bone remodeling and cause bone loss.
- GnRH analogs used in the treatment of endometriosis, the impact of which remains unknown, especially since bone loss appears to be reversible.
- Prolonged heparin therapy can promote osteopenia, the mechanism of which is uncertain [47].

7. Diagnosis

The diagnostic process for osteoporosis relies on multiple criteria, proceeding from clinical evaluation to confirmatory testing.

7.1. Positive Diagnosis

The diagnosis of osteoporosis must be made early, before fractures occur. It is therefore important to consider it in multiple circumstances [6] [48]-[51].

7.1.1. Interview

- Personal and family history.

- Conditions of fracture onset.
- The nature of the pain and its progression.

7.1.2. Clinical Examination

This must be comprehensive, focusing on the skin, neurological, hepato-digestive, and endocrine status.

This examination will also include regular and careful height measurement and assessment of kyphosis.

The thoroughness of this examination often avoids several unnecessary investigations.

7.1.3. Laboratory Assessment

The laboratory assessment must always include:

- Inflammatory parameters: ESR, CRP, a complete blood count, and serum protein electrophoresis.
- Assessment of renal function (blood urea nitrogen and plasma creatinine) and a 24-hour urine protein measurement.
- Serum iron measurement.
- Gamma-GT or 5'-nucleotidase measurement.
- Thyroid hormone measurement; in men, serum testosterone and LH should be systematically measured.
- Phospho-calcium assessment should include blood measurements of total serum calcium, serum phosphorus, alkaline phosphate levels, and 24-hour urine measurements of urinary calciuria, phosphaturia, and creatinine [49]-[51].

7.2. Radiological Examination

Standard X-rays:

These will include a frontal and lateral view of the thoracic and lumbar spine in the supine position. A lateral view centered on the thoracolumbar junction, a pelvic view, and a frontal and lateral view of the skull will be added, as well as frontal X-rays of both hands.

The analysis of these views should include a careful examination of the shape of the bony parts, a search for signs of fracture, signs of osteolysis suspicious of malignancy, and changes in the homogeneous or non-homogeneous framework.

A standard X-ray of the thoracolumbar spine (frontal and lateral) should be requested when a vertebral fracture is suspected:

- In the presence of acute spinal pain.
- In the presence of a decrease in the patient's height (an important element in the diagnosis and therapeutic monitoring of osteoporotic fractures).

A decrease in height of 6 cm is pathognomonic for vertebral compression [52]-[55].

Other imaging methods:

Bone scintigraphy, CT scan, and/or magnetic resonance imaging are sometimes essential in the presence of suspicious-looking vertebral collapse to rule out vertebral

metastasis or myeloma.

In particular, sagittal slices taken during an MRI scan allow for a comprehensive study of the spine and thus the search for changes in the bone marrow signal in uncollapsed vertebrae.

Computed tomography, which allows for detailed analysis of bone contours and sometimes detects osteolysis or tumor infiltration within a vertebral body.

Bone densitometry:

Bone densitometry is the gold standard for diagnosing osteoporosis and its severity. Measuring bone mineral content, *i.e.*, the calcium density of bones, provides a relatively accurate reflection of bone strength. Osteoporosis is assessed by comparing the results obtained with those of a young adult of the same sex and a subject in the same age group [52] [54] [55].

Specifying the extent of bone loss. Densitometry provides guidelines for treatment.

7.3. Laboratory Examination

Iliac bone biopsy:

Performed after double tetracycline staining, this allows for the histomorphometric study of bone remodeling as well as the search for abnormal cells within the hematopoietic marrow. It should only be performed if necessary.

8. Differential Diagnosis

In this section, the following conditions were included in the differential diagnosis.

8.1. Malignant Osteopathy

The absence of a history of cancer or blood disease, the mechanical nature of spinal pain, the preservation of general condition, the normality of the complete clinical examination and laboratory parameters, the absence of suspicious lytic or condensing radiographic findings, vertebral compression located above D4, and posterior wall recession must be ruled out. These are all arguments that allow the following to be ruled out:

- Metastatic bone cancer.
- Myeloma.
- Lymphoma-myeloproliferative syndrome [55] [56].

8.2. Renal Osteodystrophy and Osteomalacia

Renal osteodystrophy is quickly ruled out by measuring plasma creatinine, urea nitrogen, serum calcium, and plasma parathyroid hormone. While the diagnosis of osteomalacia is not problematic in the obvious forms that continue to be frequently observed in our country, it is more difficult in the more subtle forms, at which stage the diagnosis should be made.

Osteomalacia is defined as a rarefied osteopathy in adults, characterized by a defect in the mineralization of a portion of the bone's protein framework (this is not the case with osteoporosis). Most often linked to vitamin D deficiency, or sometimes to

resistance to its action in tissues, it is similar to childhood rickets [55] [57].

8.3. Primary Hyperparathyroidism

A history of recurrent renal lithiasis or, more rarely, the presence of chondrocalcinosis may be found. Currently, it is laboratory tests that allow for diagnosis: hypercalcemia, hypophosphatemia, hypercalciuria, and assessment of plasma parathyroid hormone levels [57] [58].

8.4. Physiological Osteopenia

It results from bone loss linked to the normal tissue aging process of senescence. This loss is clinically latent. Radiologically, it manifests as a change in texture without deformation of the bone, with increased transparency.

There is also a thinning of the cortical bone wall. A decrease in bone volume of around 15% to 20% is observed. This osteopenia does not cause any pain; it begins after reaching peak bone mass, present in the third decade of life [59].

9. Conclusions

Osteoporosis is a major public health problem in several countries (United States, Germany, Japan, etc.). In this study, we attempted to demonstrate the different aspects of the disease.

The aim of this study is to define osteoporosis, study the different etiological forms, the risk factors for the disease, and the various diagnostic methods.

Conflicts of Interest

The authors declare no conflicts of interest.

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